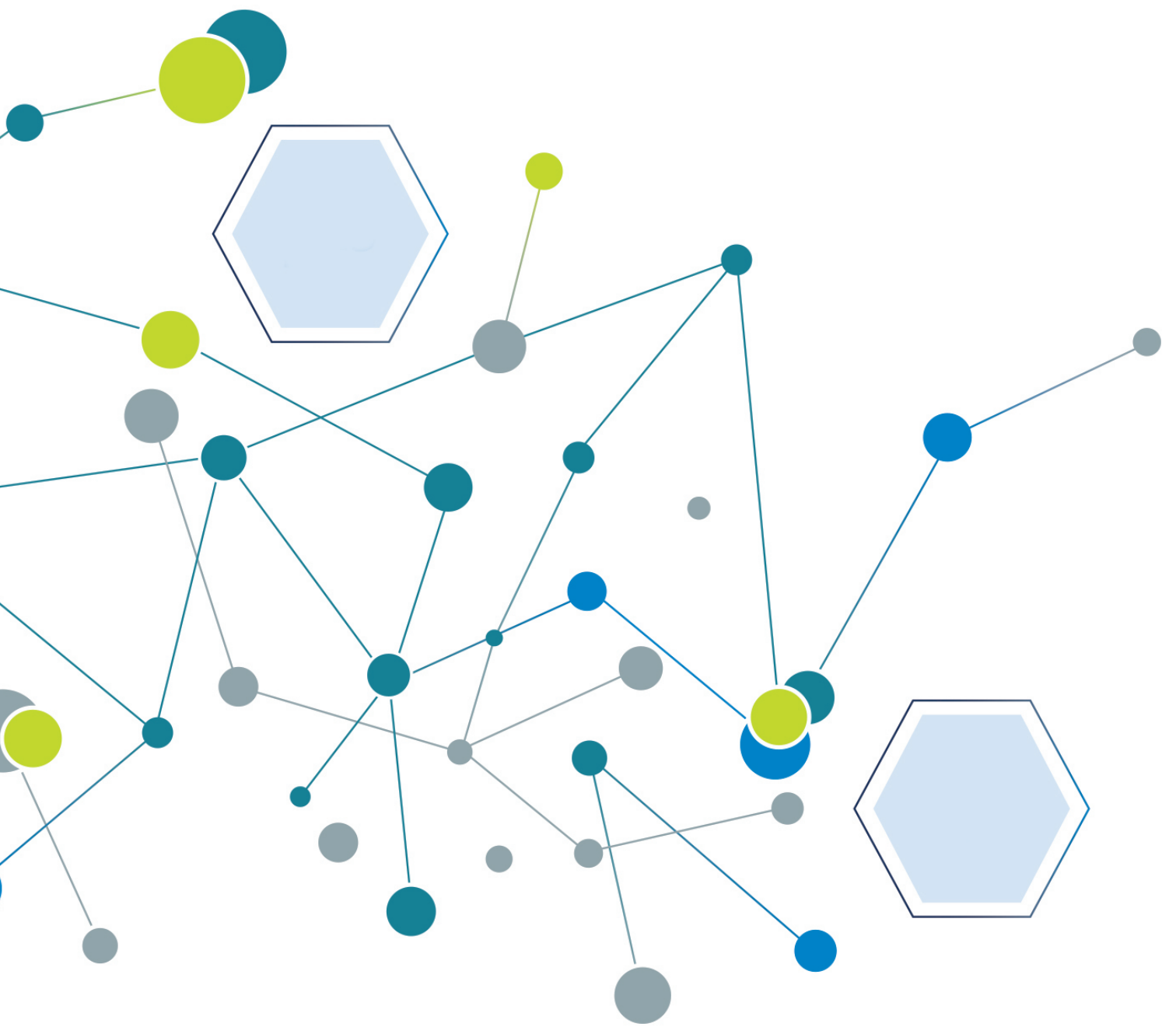
SpectraCell Laboratories

Science + Health + Solutions

SUPPLEMENTAL INFORMATION REFERENCE GUIDE

MICRONUTRIENT TEST



ALPHA LIPOIC ACID

PHYSIOLOGICAL 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 refen-ed 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.

FOOD SOURCES

Although lipoic acid is found in many plant and animal foods, quantitative data is limited. Animal sources rich in lipoic acid include organ meats (kidney, heart, liver) and plant sources include spinach, broccoli, tomatoes, peas and brussels sprouts.

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 dmg therapy may be necessary to avoid hypoglycemia. Doses of up to 600 mg/day have been well tolerated.

ASPARAGINE

PHYSIOLOGICAL FUNCTION

Asparagine is an 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.

FOOD SOURCES

Although quantitative data is limited, asparagine (an amino acid) is found in many high protein animal foods. It is also found in asparagus, nuts and legumes.

REPLETION INFORMATION

Asparagine supplementation appears safe in modest doses (up to 6 grams daily).

BIOTIN

PHYSIOLOGICAL 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 microflora from antibiotic therapy or altered gut motility, pregnant and lactating women, antiepileptic drng therapy, alcoholics, trauma (bums and surgery), elderly, malabsorption (especially achlorhydria).

FOOD SOURCES

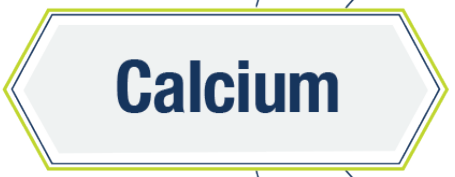
Food	Serving	(μg)
Liver	3 oz.	27
Egg (cooked)	1 large	25
Nutritional yeast	1 tbsp	14
Wheat bran	1 oz.	14
Salmon, wild	3 oz.	4

Food	Serving	(μg)
Cauliflower	1 cup	4
Avocado	1/2 fruit	3
Royal Jelly	1 gram	3
Sweet potato	1/2 cup	2
Raspberries	1 cup	2

REPLETION INFORMATION

Dietary intake of foods rich in Biotin should be increased. Do not eat raw egg whites. No adverse effects have been noted in humans ingesting up to 2000 mcg daily for long time periods.

CALCIUM



PHYSIOLOGICAL 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).

FOOD SOURCES

Food	Serving	(mg)
Tofu (raw)	1/2 cup	434
Yogurt, plain	8 oz.	415
Sardines	1 can	351
Cheese	1 1/2 oz.	303
Milk	8 oz.	300
Blackstrap molasses	1 tbsp	200

Food	Serving	(mg)
Salmon, canned	3 oz.	181
Spinach, cooked	1/2 cup	115
Turnip greens	1/2 cup	99
Bok choy, cooked	1/2 cup	79
Kale, cooked	1 cup	94
Kale, raw	1 cup	24

Calcium in dairy and the kale family (broccoli, bok choy, cabbage, turnip greens) tends to be easily absorbed. However, foods high in oxalate (spinach) will significantly inhibit calcium absorption. Phytates (in whole grains and beans) will also inhibit calcium absorption.

REPLETION INFORMATION

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.

CARNITINE

PHYSIOLOGICAL FUNCTION

L-camitine is an amino acid derivative of the essential amino acids L-lysine and methonine. The conversion to camitine 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. Camitine 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).

DEFICIENCY SYMPTOMS

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

FOOD SOURCES

Food	Serving	(mg)
Beef	3 oz.	80
Pork	3 oz.	24
Milk (whole)	1 cup	8
Fish (cod)	3 oz.	5

Food	Serving	(mg)
Chicken	3 oz.	3
Cheese	2 oz.	2
Avocado	1 medium	2
Asparagus	6 spears	0.2

REPLETION INFORMATION

There have been no reports of toxicity from L-camitine supplementation. The biologically active form of camitine is the L- isomer. DL-camitine should be avoided. Usual dosages found in capsules and tablets range from 250 to 1000 mg in a variety of chemical formulations: L acetylcamitine, L-camitine, and the HCl, tartrate and fumarate salts. Camitine, Coenzyme Ql0 and pantothenate (BS) appear to work synergistically

CHOLINE

PHYSIOLOGICAL 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 dietaty choline depletion in adults suggests that choline is an essential nutrient. 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.

FOOD SOURCES

Food	Serving	(mg)
Beef liver	3 oz.	350
Wheat germ	1 cup	200
Egg	1 large	147
Beef	3 oz.	70-110
Scallops	3 oz.	94
Cod	3 oz.	71

Food	Serving	(mg)
Potato	1 large	57
Kidney beans	1/2 cup	45
Milk	1 cup	38
Brussels sprouts	1/2 cup	32
Broccoli	1/2 cup	31
Peanuts	1/4 cup	24

REPLETION INFORMATION

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 oflecithin 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.

CHROMIUM

PHYSIOLOGICAL FUNCTION

Chromium is an essential trace mineral that plays an important role in optimizing insulin function and the regulation of blood glucose levels. Chromium may also be anti-atherogenic and assist in lowering cholesterol. Following food intake, 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. Chromium also plays a role in nucleic acid synthesis.

DEFICIENCY SYMPTOMS

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 U.S. Chromium deficiency may increase the likelihood of insulin resistance which can lead to elevated blood levels of insulin (hyperinsulinemia) and elevated blood levels of glucose, which can ultimately cause heart disease and/or diabetes. 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. These symptoms increase one's risk for heart disease. Low levels of chromium are also associated with an increased risk of coronary artery disease incidence and mortality. Chromium deficiency correlates with depressed nucleic acid synthesis. 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.

FOOD SOURCES*

Food	Serving	(μg)	Food	Serving	(μg)
Broccoli	1/2 cup	11.0	Beef	3 oz.	2.0
Grape Juice**	1 cup	7.5	Apple	1 whole	1.4
Garlic, dried	1 tsp	3.0	Green beans	1/2 cup	1.1
Potatoes	1 cup	2.7	Banana	1 whole	1.0
Basil, dried	1 cup	2.7	Red wine	5 oz.	1-13

*The chromium content has been measured accurately in relatively few foods.
**It is also important to note that foods high in simple sugars promote chromium loss.

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. However, people with liver or kidney disease may be more susceptible to adverse effects from excessive intake of chromium, and such individuals are cautioned to avoid taking more than 200 micrograms of chromium supplements per day. There is limited evidence to suggest that long term chromium picolinate supplementation at levels greater than 200 micrograms per day may also be hazardous to chromosome integrity and should be avoided.

COENZYME Q10

PHYSIOLOGICAL 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 triphospate). 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 contest 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.

FOOD SOURCES

Food	Serving	(mg)
Grass fed beef	3 oz.	2.6
Herring	3 oz.	2.3
Canola oil	1 tbsp	1.0
Trout	3 oz.	0.9
Peanuts	1 oz.	0.9

Food	Serving	(mg)
Sesame seeds	1 oz.	0.7
Pistachio nuts	1 oz.	0.6
Broccoli	1/2 cup	0.5
Salmon	3 oz.	0.4
Orange	1 whole	0.3

REPLETION INFORMATION

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.

COPPER

PHYSIOLOGICAL FUNCTION

Like most trace minerals, copper acts as an enzyme cofactor in several key metabolic processes in the body. Among its many functions, copper aids in the fonnation of bone, hemoglobin and red blood cells, therefore enabling the efficient transport of oxygen throughout the body. In addition, copper works in balance with vitamin C and zinc to manufacture elastin (skin protein) as well as collagen and other structural proteins in cartilage and tendons. It is also involved in the healing process, energy production, hair and skin coloring (production of melanin) and taste sensitivity.

Copper stimulates the absorption of iron through the copper transport protein ceruloplasmin. Copper also aids in the metabolism of several fatty acids and helps prevent oxidative damage by serving as a cofactor to superoxide dismutase. In addition, copper is needed for proper insulation (myelination) of nerve cells and serves as a cofactor for the synthesis of the neurotransmitter norepinephrine.

DEFICIENCY SYMPTOMS

Due to copper's role in the formation of collagen, copper deficiency can manifest as osteoporosis. Other possible signs of deficiency include anemia (due to its role in hemoglobin formation), baldness, diarrhea, general weakness, impaired respiratory function, myelopathy, decreased skin pigment, reduced resistance to infection and increased triglyceride levels. Evidence also links copper deficiency with increased oxidative damage to cell membranes.

FOOD SOURCES

Food	Serving	(μg)
Beef liver	1 oz.	1265
Oysters	1 medium	670
Cashews	1 oz.	629
Crab	3 oz.	624
Clams	3 oz.	585
Sunflower seeds	1 oz.	519

Food	Serving	(μg)
Beef	3 oz.	2.0
Apple	1 whole	1.4
Green beans	1/2 cup	1.1
Banana	1 whole	1.0
Red wine	5 oz.	1-13
Peanut butter	2 tbsp	165

Although quantitative data is limited, blackstrap molasses is also a good source of copper.

REPLETION INFORMATION

Pharmacologic doses of copper in scientific studies usually range from 2-4 mg per day. Ingesting amounts over 10 mg per day regularly can result in nausea, although toxicity will typically occur at much higher levels (200 times the RDA). Ingestion of excessive levels of zinc, vitamin C or fructose can cause copper deficiency.

CYSTEINE

PHYSIOLOGICAL FUNCTION

Cysteine is a sulfur-containing, conditionally-essential amino acid. The sulfur group (thiol or sulfhydryl group) in cysteine accounts for most of cysteine's functions. Cysteine can be oxidized with itself to form cystine. Cysteine has six major functions: 1) incorporation into amino acid sequences of proteins, where cysteine promotes protein structure by sulfhydryl bonding; 2) rate-limiting precursor for glutathione synthesis; 3) precursor for taurine (used in bile formation and nerve function); 4) source of sulfate for connective tissue synthesis; 5) source of pyruvate for energy or glucose production; and 6) neurotransmitter. As a component of glutathione, cysteine functions include being a powerful antioxidant, detoxification agent, component of some prostaglandins, and an amino acid transporter across membranes. Formation of cysteine from homocysteine is one pathway to reduce homocysteine levels.

DEFICIENCY SYMPTOMS

Cysteine deficiencies identified by inherited metabolic disorders or reduced levels in body fluid have been associated with: 1) impaired antioxidant defenses; 2) decreased ability to metabolize drugs or toxic compounds; 3) depressed immune functions; 4) some psycoses; and 5) homocystinemia. Patients with rheumatoid arthritis, hypertension, and smokers have reduced levels of cysteine in fluids and tissues. Clinical trials of cysteine supplementation have shown benefits for skin disorders, hair loss, asthma, bronchitis, allergies, cystic fibrosis, chronic obstructive pulmonary disease, heavy metal toxicity, iron deficiency, diabetes & diabetic nephropathy, seizure disorders, reducing cytoxic treatment side effects, HIV infection, and alcoholism.

FOOD SOURCES

Although quantitative data is limited, cysteine (an amino acid) is found in high protein animal foods such as fish, meats, egg and dairy. Unprocessed (raw) whey protein contains especially high levels of bioavailable cysteine. Plant sources include broccoli, brussels sprouts, cabbage, dates, garlic, ginseng, nuts, onions, lentils, peppers and sunflower seeds.

REPLETION INFORMATION

Cysteine is found in all proteins, and thus, is richest in high protein foods such as meats, yogurt, wheat germ, and eggs. However, some cysteine is oxidized to cystine and other compounds during cooking and storage, and is less available to the body. Regardless of dietary protein intake, cysteine supplementation with N-Acetyl-L-Cysteine has been found to be safe at doses up to 2000 mg daily. Supplementation with cysteine is not recommended as it is not well tolerated by many patients. In addition, it may be rapidly oxidized to cystine which is less available for utilization.

FOLATE

PHYSIOLOGICAL 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, intestinal lesions. However the neurological complications of vitamin B12 deficiency do not occur with folate deficiency. Thus, a regulatory limit on folate levels in dietary supplements of 400 mcg per unit is in effect, to prevent a potential missed diagnosis of Vitamin B12 deficiency.

Those at risk include individuals that: are Vitamin B12 deficient, malnourished, experience malabsorption, pregnant and lactating women, alcoholics, oral contraceptive users, elderly, infants, have inherited folate disorders, have an increased rate of cellular division (burns, trauma, malignancies, hemolytic anemias), use anti-convulsant therapy, folate antagonist therapy, tuberculosis therapy, and sulfasalzine therapy.

FOOD SOURCES

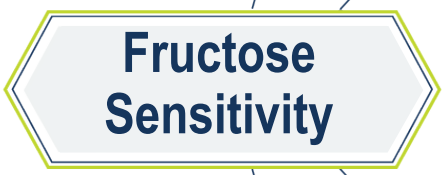
Food	Serving	(mcg)
Nutritional yeast	1 tbsp	270-600
Beef liver	1 cup	215
Lentils	1/4 cup	179
Garbanzo beans	1/2 cup	141
Spinach (cooked)	1/2 cup	130
Spinach (raw)	1 cup	58

Food	Serving	(mcg)
Asparagus	6 spears	130
Lima beans	1/2 cup	78
Brussel sprouts	1/2 cup	78
Romain lettuce	1 cup	64
Broccoli	1/2 cup	52
Orange	1 small	29

REPLETION INFORMATION

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.

FRUCTOSE SENSITIVITY



PHYSIOLOGICAL 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-l-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.

FOOD SOURCES

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 com syrup, com syrup, fruit juice concentrates and a large list of prepared foods containing sucrose and/or com syrup.

REPLETION INFORMATION

Reduction of excess dietary fructose intake by avoidance of foods very rich in fructose is suggested when fructose intolerance is exhibited

GLUCOSE-INSULINE INTERACTION

PHYSIOLOGICAL 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.

FOOD SOURCES

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.

REPLETION INFORMATION

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.

GLUTAMINE

PHYSIOLOGICAL 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 (bums, surgery), immune functions, and gastric ulcers. Glutamine may be useful in alcoholism and fatigue.

FOOD SOURCES

Although quantitative data is limited, glutamine (an amino acid) is found in many high protein foods, especially milk and meat.

REPLETION INFORMATION

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.

GLUTATHIONE

PHYSIOLOGICAL 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 such as aging, cancer, atherosclerosis, arthritis, viral infections, AIDS, cardiovascular, neurodegenerative diseases and pulmonary diseases may be produced, or made worse, by “free radicals”. 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 to decrease the formation of oxidized LDL, implicated in the development of atherosclerosis. T-lymphocytes become deficient in glutathione in the progression of AIDS which impairs immune function. Glutathione is also required for the synthesis of some prostaglandins from n-3 and n-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.

FOOD SOURCES

Unprocessed (raw) whey protein contains large amounts of the glutathione precursor, cysteine, in a form that is bioactively capable of being converted into functional glutathione.

Glutathione-rich foods include asparagus, avocado and walnuts. Glutathione is synthesized endogenously so foods that support glutathione function may raise intracellular levels. This includes the following:

- Cruciferous vegetables (cabbage, broccoli, brussels sprouts, cauliflower)
- Unprocessed citrus (lemon, lime, orange)
- Limonene-rich herbs (dill, caraway)

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 ofN-Acetyl-L-Cysteine appears safe. Supplementation with cysteine is not recommended as it may be poorly tolerated by many patients. In addition, it may be rapidly oxidized to L-cystine, a less usable form for the synthesis of glutathione.

IMMUNIDEX

PHYSIOLOGICAL FUNCTION

What Does the Immunidex Measure?
A patient's Immunidex score is one measurement to evaluate a person's cell-mediated immune system performance. Specifically, it measures T-cell lymphocyte proliferation. Since immune function is a systemic measure of general health, a higher Immunidex score is generally desired since it means a person can respond efficiently not only to exogenous threats such as pathogens or allergens, but also to endogenous threats like tumors. The immune system, comprised of both cell mediated (Th1) and humoral (Th2) components, when balanced and performing optimally, affords us critical protection and promotes health and wellness.

How is the Immunidex Performed?
A patient's lymphocytes are isolated from whole blood and introduced to a protein that stimulates growth. The protein mitogen used to trigger mitosis, or cell division, is PHA (phytohemagglutinin), which stimulates T-lymphocytes to proliferate. The proliferative response is measured by the incorporation of radioactive thymidine into newly synthesized DNA. Your patient's response is compared to responses of a reference population and results are reported to you as an Immunidex score.

What Affects the Immunidex Result?
Micronutrient deficiencies will undermine a person's immune function, and thus lower the Immunidex. Since the highly
complex immune system is dependent on the intracellular availability of vitamins, minerals and antioxidants, correcting specific micronutrient deficiencies typically raises the Immunidex and contributes to tangible clinical benefits, such as reduced infections and may assist in achieving Th1/Th2 balance.

How Does the Immunidex Correlate with Antioxidant Function?
In general, the higher the antioxidant score (Spectrox®), the higher the Immunidex score. Antioxidant function plays an important role in promoting optimal T-cell (lymphocyte) function. It is important to find out if a patient has deficiencies in specific antioxidant nutrients so they can supplement wisely. But it is also important to measure a total antioxidant function because the metabolic pathways in which antioxidants are involved are highly complex, sometimes redundant and often overlapping. Research confirms that taking excess antioxidants that are not needed (i.e. where no deficiency exists) can actually cause them to become pro-oxidants and decrease antioxidant function.

How is Immunidex Related to Aging?
As we age, our immune function typically decreases as seen in the figure below. Although many factors are involved in this complicated process of decline, the Immunidex is one of many relevant aging biomarkers since age diminishes the ability of a person's lymphocytes to respond to challenges. The effects of both good and poor antioxidant function on the Immunidex is shown and emphasizes the importance of testing for antioxidant function (Spectrox®) and individual antioxidant deficiencies.

How Do You Order Immunidex?
The Immunidex is part of SpectraCell's Micronutrient Testing panel. There is no additional charge for this calculated test result. Ordering instructions are the same – same kit, same blood draw instructions.

INOSITOL

PHYSIOLOGICAL 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).

FOOD SOURCES

Food	Serving	(mg)
Canteloupe	1/4	355
Orange	1 whole	207
Kidney beans*	1/2 cup	249
Green beans	1/2 cup	193
Grapefruit	1/2	199

Food	Serving	(mg)
Mango	1 whole	198
Lime	1 whole	194
Kiwi	1/2 cup	136
Peanut butter	2 tbsp	121
Nectarine	1 whole	121

*Beans and legumes may contain high levels of phytates which can impair absorption of nutrients on how they are prepared or processed.

REPLETION INFORMATION

Usual dietary intake is one gram per day. Oral doses of up to 1-2 grams daily are well tolerated.

MAGNESIUM

PHYSIOLOGICAL 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 (Trouseau and Chvostek signs, muscle spasms, tetany, cardiac arrythmias, 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.

FOOD SOURCES

Food	Serving	(mg)
Oat bran	1/2 cup	96
Brown rice	1 cup	86
Mackerel	3 oz.	82
Spinach, cooked	1/2 cup	78
Almonds	1 oz.	77
Swiss chard, cooked	1/2 cup	75

Food	Serving	(mg)
Lima beans	1/2 cup	63
Edamame	1/2 cup	50
Blackstrap molasses	1 tbsp	48
Potato, with skin	1 baked	43
Black eyed peas	1/2 cup	42
Banana	1 whole	34

REPLETION INFORMATION

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.

MANGANESE

PHYSIOLOGICAL FUNCTION

Manganese is a mineral element that is both nutritionally essential and has the potential to be very toxic. This fact is further complicated by the small range of dosage for clinical benefit and toxicity with serious consequences. Manganese is an important factor in many biochemical processes including antioxidant function. The principle antioxidant enzyme within the mitochondria (energy) is superoxide dismutase and the enzyme requires manganese for optimal performance. Manganese is also required for normal skeletal development and cartilage synthesis. Wound healing is also impacted by manganese, as the synthesis of collagen in skin cells is dependent on the presence of adequate manganese. Manganese is important functioning as a co-factor in the metabolism of carbohydrates, amino acids and cholesterol. Manganese is considered anti-osteoporotic and anti arthritic.

DEFICIENCY SYMPTOMS

Deficiency in manganese was once considered rare, but it is now estimated that up to 15% of the population may be deficient in manganese caused by improper diet and eating habits. Deficiency in manganese may lead to various health problems which could include bone malformation, eye and hearing problems, increased cholesterol, hypertension, infe1iility, cardiovascular issues, memory loss, hearing loss, muscle cramping and tremors. Other deficiency symptoms may include ataxia, fainting and carbohydrate intolerance (diabetes) Manganese deficiency has also been linked to myasthenia gravis.

MANGANESE TOXITY: Symptoms of manganese toxicity mimic those of Parkinson's Disease with permanent neurological damage. It may also precipitate hypertension in patients over 40, and significant rises in manganese are found in patients with hepatitis, cirrhosis, dialysis patients and victims of heart attack. Early signs of toxicity include loss of appetite, impaired memory, and mask-like facial expressions. Excess manganese will reduce iron absorption.

FOOD SOURCES

Food	Serving	(mg)
Blackstrap molasses	1 tbsp	1.53
Pineapple, raw	1/2 cup	1.28
Oatmeal	1 cup	1 oz.
Pecans	1 oz.	1.12
Brown rice	1/2 cup	0.88
Spinach	1/2 cup	0.84

Food	Serving	(mg)
Almonds	1 oz.	0.74
Peanuts	1 oz.	0.59
Sweet Potato	1/2 cup	0.55
Lima beans	1/2 cup	0.48
Tea, green	1 cup	0.41-1.6
Tea, black	1 cup	0.18-0.77

REPLETION INFORMATION

Estimated average dietary manganese intakes range from 2.0 to 3.0 mg/day for men and 1.6-1.8 mg/day for women. People eating vegetarian diets may have higher intakes. Foods high in phytic acid or oxalic acid may reduce manganese absorption.

OLEIC ACID

PHYSIOLOGICAL FUNCTION

- Most common monounsaturated fatty acid in human cells. It 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.
- Major energy source for cells. It is catabolized to acetyl groups used for energy (ATP) production and biosynthesis of many essential metabolites.
- 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).

FOOD SOURCES*

Source	**Oleic acid composition
High oleic safflower oil	84%
Peanut oil	71%
Avocado oil	70%
Almond oil	67%

Source	Oleic acid composition
Olive oil	66%
Canola oil	63%
Rice bran oil	43%
Sesame oil	42%

*The corresponding foods to the oils listed above (e.g. olives, avocados, almonds) are also good sources oleic acid.
** Despite the high content of oleic acid in listed oils, some also ocntain high levels of polysaturated fatty acids which may become pro-inflammatory due to oxidation that occurs during processing and/or cooking.

REPLETION INFORMATION

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 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.

PANTOTHENATE

PHYSIOLOGICAL 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.

FOOD SOURCES

Food	Serving	(mg)
Liver	3 oz.	5.6
Nutritional	1 tbsp	3.0
Sunflower seeds	1 oz.	2.0
Trout	3 oz.	1.9
Yogurt	8 oz.	1.6

Food	Serving	(mg)
Chicken	3 oz.	0.5
Avocado	1/2 fruit	0.5
Spinich, cooked	1 cup	0.4
Dried plums	1 cup	0.4
Hazelnuts	1 oz.	0.2

REPLETION INFORMATION

Oral administration of pantothenate has shown no toxicity in doses up to 10 gms daily. Higher doses may cause diarrhea.

SELENIUM

PHYSIOLOGICAL 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 prope1iies 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 mcg/day) increases the ratio ofHDL 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.

FOOD SOURCES

Food	Serving	(μg)
Brazil nuts	1 oz.	839
Tuna	3 oz.	92
Oysters	3 oz.	65
Clams	3 oz.	54
Halibut	3 oz.	47
Shrimp	3 oz.	42

Food	Serving	(μg)
Salmon	3 oz.	40
Crab	3 oz.	38
Brown rice	1 cup	19
Sunflower seeds	1/4 cup	18
Beef	3 oz.	17
Walnuts	1 oz.	5

Some plants including garlic, Brazil nuts and plants in the Brassica family (cruciferous vegetables such as cabbage and broccoli) tend to accumulate selenium if they are grown in selenium-rich soil, making them a potentially rich plant-based source of this mineral.

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.

SERINE

PHYSIOLOGICAL 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.

FOOD SOURCES

Although quantitative data is limited, serine (an amino acid) is found in high protein animal foods such as fish, meats, egg and dairy. Plant sources rich in serine include asparagus, beans, fenugreek, lentils, nuts, seaweed, spinach and watercress.

REPLETION INFORMATION

Doses of 1-2 grams daily of pure serine appear safe.

SPECTROX™ (TOTAL ANTIOXIDANT FUNCTION)

PHYSIOLOGICAL 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.

The clinical correlation of antioxidant status to health remains under investigation. Research evidence in humans has indicared that deficient intakes or levels of nutrient antioxidant 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. Encouraging studies have also shown that intervention with antioxidant nutrient supplements have therapeutic benefits in humans. Thus, strong scientific evidence illustrates that antioxidants help to prevent chronic degenerative diseases and may help to restore health.

VITAMIN A (Retinol)

PHYSIOLOGICAL FUNCTION

Vitamin A is a family of fat soluble compounds (carotenoids) 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. Retinal (Vitamin A) promotes healthy surface linings of the eyes and respiratory, urinary and instestinal tracts. Vitamin A also promotes healthy skin function and integrity. Retinal is the most active form of Vitamin A and is synthesized in the body by conversion of provitamin A, primarily beta carotene, into retinal. Lycopene, lutein and zeaxathin are carotenoids 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 hyperkeratinization, 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 pmiicularly 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.

FOOD SOURCES

Food	Serving	µg RAE*	Food	Serving	µg RAE*
Beef liver	3 oz.	6582	Butternut squash	1/2 cup	572
Cod liver oil	1 tbsp	4080	Spinach, cooked	1/2 cup	472
Sweet potato	1/2 cup	1136	Cantaloupe	1/2 melon	466
Pumpkin, canned	1/2 cup	953	Red peppers	1/2 cup	117
Carrots	1/2 cup	595	Apricot	1 medium	74

*µg RAE = micrograms of Retinol Activity Equivalents

REPLETION INFORMATION

ADEQUATE ZINC IS REQUIRED to synthesize retinal binding protein (RAP) 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.

VITAMIN B1 (Thiamin)

PHYSIOLOGICAL FUNCTION

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

DEFICIENCY SYMPTOMS

Early thiamin deficiency leads to clinical signs of:

- *Loss of Appetite*
- *Irritability*
- *Fatigue*
- *Constipation*
- *Mental Depression*
- *Nausea*
- *Peripheral Neuropathy*

Clinical signs of more severe thiamin deficiency (Wemicke-Korsafoff Syndrome):

- *Mental Confusion*
- *Loss of Eye Coordination*
- *Loss of Fine Motor Control*

Those at risk for thiamin deficiency include: Patients suffering from Malnutrition, Starvation or Malabsorption Syndromes

- *Alcoholics*
- *Patients on restricted diets*
- *Prolonged hemodialysis*
- *Gastric partitioning surgery*
- *Elderly*
- *Metabolic disorders*
- *Inherited Thiamin-Responsive*

FOOD SOURCES

Food	Serving	(mg)
Nutritional yeast	1 tbsp	3.0-6.0
Wheat germ	1 cup	1.9
Pork	3 oz.	0.74
Brazil nuts	1 oz.	0.28
Peas	1/2 cup	0.21
Brown rice	1 cup	0.19

Food	Serving	(mg)
Pecans	1 oz.	0.19
Lentils	1/2 cup	0.17
Orange	1 whole	0.11
Cantelope	1/2 melon	0.10
Spinach	1/2 cup	0.09

REPLETION INFORMATION

Excessive ingestion of certain raw fresh-water fish and shellfish, tea, coffee, blueberries and red cabbage should be avoided, as these foods may contain anti-thiamin factors. There is no evidence of thiamin toxicity form oral administration, except for development of sensitivity in very rare cases.

VITAMIN B2 (Riboflavin)

PHYSIOLOGICAL 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.

FOOD SOURCES

Food	Serving	(mg)
Nutritional yeast	1 tbsp	2.0-6.0
Milk	8 oz.	0.29
Almonds	1 oz.	0.26
Egg	1 large	0.26
Spinach	1/2 cup	0.21
Beef	3 oz.	0.15

Food	Serving	(mg)
Chicken (dark)	3 oz.	0.16
Chicken (light)	3 oz.	0.08
Asparagus	6 spears	0.13
Salmon	3 oz.	0.13
Broccoli	1/2 cup	0.10

REPLETION INFORMATION

There is no evidence of toxicity from oral administration of riboflavin, except for rare cases of sensitivity.

VITAMIN B3 (Niacinamide)

PHYSIOLOGICAL 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, diatThea (the “3 D’s of pellagra), tremors and sore (black) tongue. Deficiencies ofthiamin, riboflavin, and pyridoxine commonly accompany (or can cause) niacinamide deficiency.

FOOD SOURCES

Food	Serving	(mg)
Nutritional yeast	1 tbsp	11-38
Turkey	3 oz	10
Tuna (canned)	3 oz.	9
Chicken (light meat)	3 oz.	9
Salmon	1/2 cup	0.21

Food	Serving	(mg)
Beef	3 oz.	4
Peanuts	1 oz.	4
Lentils/peas	1 cup	2
Lima beans	1 cup	2
Potatoes	1 cup	2

REPLETION INFORMATION

Dietary sources of niacinamide are expressed as niacin equivalents, taking into account tryptophan’s contribution. Niacinamide has no observed toxicity for intakes up to 3-9 gms daily, and is the prefen-ed 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.

VITAMIN B6 (Pyridoxine)

PHYSIOLOGICAL FUNCTION

Vitamin B6 is needed to metabolize proteins and is important for a healthy immune system, nerves, bones and arteries. Vitamin B6 is a complex of three similar molecules: Pyridoxine, Pyridoxal and Pyridoxamine. All are present in foods and converted into to pyridoxal-5-phosphate, the most active coenzyme form. The primary functions of vitamin B6 are in protein metabolism, transferring amino acid and sulfur groups. Roles in synthesis of heme (for hemoglobin), niacin, neurotransmitters, connective tissues, eicosanoids, and sphingolipids in nerve sheaths are also essential. Vitamin B6 also participates in the utilization of glycogen and immune function.

DEFICIENCY SYMPTOMS

Early vitamin B6 deficiency symptoms are primarily peripheral neuropathy, weakness, irritability, depression, insomnia and anxiety. More severe deficiency leads to dermatitis, nausea, vomiting, and convulsions. Carpal tunnel syndrome, premenstrual tension syndrome, and atherosclerosis may also be related to vitamin B6 deficiency. Sideroblastic anemia is indicative of vitamin B6 deficiency. Homocysteine levels in serum may be elevated by a vitamin B6 deficiency.

FOOD SOURCES

Food	Serving	(mg)
Nutritional yeast	1 tbsp	1.5-6.0
Chickpeas	1 cup	1.1
Banana	1 cup	0.7
Potato, with skin	1 whole	0.7
Salmon, wild	1/2 cup	0.21

Food	Serving	(mg)
Chicken	3 oz.	0.5
Avocado	1/2 fruit	0.5
Spinich, cooked	1 cup	0.4
Dried plums	1 cup	0.4
Hazelnuts	1 oz.	0.2

REPLETION INFORMATION

Oral intakes of pyridoxine hydrochloride in excess of 1000 mg daily for long time periods have caused peripheral neuropathy. Doses between 200 and 1000 mg daily for long time periods have also been associated with peripheral neuropathy. In general, doses up to 100 mg daily have exhibited long-term safety. Persons with drug-induced neuritis may tolerate higher doses, while pyridoxine may diminish the effectiveness of the medication L-DOPA in patients with Parkinson's Disease.

VITAMIN B12 (Cobalamin)

PHYSIOLOGICAL FUNCTION

Vitamin B12 is required to form blood and immune cells, and support a healthy nervous system. A series of closely-related compounds known collectively as cobalamins or vitamin B12 are converted into active forms methylcobalamin or 5-deoxyadenosylcobalamin. Methylcobalamin interacts with folate metabolism, preventing folate derivatives from being trapped in unusable states. Adenosylcobalamin is involved in the metabolism of odd-chain fatty acids and branched chain amino acids.

DEFICIENCY SYMPTOMS

Deficiency symptoms of vitamin B12 are both hematological (pernicious anemia) and neurological. A megaloblastic anemia may occur because the effects of the vitamin B12 deficiency on folate metabolism. Shortness of breath, fatigue, weakness, itTitability, sore tongue, decrease in blood cell counts (red, white and platelets) are all clinical signs of a vitamin B12 deficiency. Neurological symptoms are manifested as a progressive neuropathy, with loss of position sense and ataxia. If vitamin B12 repletion 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. Vitamin B12 is necessary to prevent accumulation of homocysteine, a toxic metabolic byproduct linked to cardiovascular disease and connective tissue abnormalities. Hypochlorhydria and gastrointestinal disturbances are frequently associated with vitamin B12 deficiency.

FOOD SOURCES

Food	Serving	(mcg)
Clams	3 oz.	84
Beef liver	3 oz.	70
Mussels	3 oz.	20
Mackerel	3 oz.	16
Crab	3 oz.	10

Food	Serving	(mcg)
Trout, wild	3 oz.	5.4
Troup, farmed	3 oz.	3.5
Salmon, farmed	3 oz.	2.4
Beef	3 oz.	2.1
Egg	1 large	0.6

REPLETION INFORMATION

No toxic effects of oral vitamin B12 intake have been demonstrated, even in doses over 1000 ug daily. Since the absorption and intracellular activation of oral vitamin B12 are frequently difficult, consideration should be given to injectable forms of vitamin B12. Some patients may require more frequent or larger doses than usual before repletion occurs.

VITAMIN C (Ascorbate)

PHYSIOLOGICAL FUNCTION

Vitamin C is required for several metabolic functions in the body. One of its major roles is in the synthesis of collagen and elastin. It is also necessary in the production of several stress response hormones including adrenalin, noradrenalin, cortisol and histamine, and it is required in the synthesis of camitine. Vitamin C protects against heart disease by helping dissolve arterial plaque, reducing free radical oxidation of cholesterol, decreasing levels of lipoprotein Lp(a) and maintaining the elasticity of vascular walls. In addition to boosting immunity, vitamin C enhances iron absorption, promotes efficient wound healing, and detoxifies the body by binding to certain heavy metals so they can be eliminated from the body. The anti-cancer effects of vitamin C stem from its role as a potent water-soluble antioxidant in the plasma and cytoplasm. It also protects nucleic acids (DNA) from oxidative damage and inhibits the formation of nitrosamines. Additionally, it can regenerate vitamin E and works synergistically with other antioxidants such as beta-carotene and glutathione to increase their overall antioxidant effect.

DEFICIENCY SYMPTOMS

Deficiency symptoms include capillary fragility which often manifests clinically as bleeding gums, easy bruising, tender joints, muscle weakness and poor wound healing. Subclinical deficiency can also result in lowered immunity, anemia, and fatigue.

FOOD SOURCES

Food	Serving	(mg)
Rose hip	1 cup	541
Red bell peppers	1/2 cup	95
Strawberries	1 cup	82
Orang juice*	3/4 cup	75
Kiwi	1 medium	64

Food	Serving	(mg)
Green bell peppers	1/2 cup	60
Broccoli	1/2 cup	51
Brussels sprouts	1/2 cup	48
Grapefruit	1 whole	39
Tomato	1 medium	17

*Vitamin C is lost during heat processing so in most commercial citrus juices ascorbic acid (a form of vitamin C) is added after pasteurization.

REPLETION INFORMATION

Mega-doses of vitamin C have been used for specific conditions such as viral infections and cancer, although the therapeutic benefit of mega-doses is thought to occur due to a pro-oxidant effect of vitamin C on viral or cancer cells. Since excess vitamin C can actually increase free radical production, it is important to balance it with other antioxidants in order to not induce a pro-oxidant effect. Large doses may result in diarrhea. It is recommended to ingest therapeutic doses at various intervals throughout the day since the body readily excretes excess vitamin C. Physical or emotional stress will increase the body's requirement for vitamin C. In some patients, excess vitamin C can produce high amounts of oxalic acid, which are linked to kidney stones so patients with kidney disease or a history of kidney stones should exercise caution in taking mega-doses of vitamin C.

VITAMIN D (Ergocalciferol)

PHYSIOLOGICAL 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.

FOOD SOURCES

Natural Sources	Serving	(IU)
Salmon, wild	3 1/2 oz.	600-1000
Salmon, farm	3 1/2 oz.	100-250
Mackerel	3 1/2 oz.	250
Tuna	3 1/2 oz.	230
Cod liver oil	1 tsp	400
Shitake mushroom, fresh	3 1/2 oz.	100
Shitake mushroom, dried	3 1/2 oz.	1600

Fortified Sources	Serving	(IU)
Milk	8 oz.	100
Orange juice	8 oz.	100
Yogurt	8 oz.	100
Cheese	3 oz.	100
Butter	3 1/2 oz.	50

*Vitamin D is a fat-soluble vitamin so if a food is fat-free (e.g. skim milk, OJ, low-fat yogurt) absorption may be an issue even if it is fortified.

REPLETION INFORMATION

Supplemental vitamin D is available as vitamin D2 (ergocalciferol) or vitamin D3 (cholecalciferol). 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.

VITAMIN E (a-tocopherol)

PHYSIOLOGICAL 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 free radicals to less harmful derivatives, limiting formation of proinflammatory cytokines. In deficiencies of vitamin E, arachidonic acid is converted to proinflammatory 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).

FOOD SOURCES *

Food	Serving	(mg)
Wheat germ oil	1 tbsp	20.3
Cod liver oil	1 tbsp	20.1
Sunflower seeds	1 oz.	7.4
Almonds	1 oz.	7.3
Hazelnuts	1 oz.	4.3

Food	Serving	(mg)
Tomato sauce	1 cup	3.5
Peanut butter	2 tbsp	3.2
Dried apricots	1/2 cup	2.8
Avocado	1 whole	2.7
Olive oil	1 tbsp	1.9

* The form of vitamin E listed in this table is alpha-tocopherol.

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-a-tocopherol. Beta-tocopherol, gamma-tocopherol and the alpha- and delta tocoretinols have less than 50% of the biological activity than d-a-tocopherol.

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 LU. per day. Some studies report effective use of vitamin E at doses up to 3000 LU. per day without observed side effects over a two-year period.

VITAMIN K

PHYSIOLOGICAL FUNCTION

The primary function of vitamin K is to aid in the formation of clotting factors and bone proteins. It serves as a cofactor in the production of six proteins that regulate blood clotting, including prothrombin. In addition, it helps to form osteocalcin, a protein necessary for the mineralization of bone. Vitamin K also aids in the formation of glucose into glycogen for storage in the liver. In addition, it promotes the prevention and reversal of arterial calcification, plague progression and lipid peroxidation. Deficiency may increase the risk of calcification of arterial walls, particularly in individuals on vitamin D supplementation (Vitamin D promotes calcium absorption). Vitamin K exists in three forms: K,, a natural form found in plants (phylloquinone); K2, which is synthesized in the intestine (menaquinone); and K3, a synthetic form that must be activated in the liver (menadione).

DEFICIENCY SYMPTOMS

Excessive bleeding, a history of bruising, appearance of ruptured capillaries or menorrhagia (heavy periods) are the most common clinical symptoms of overt vitamin K deficiency, although subclinical deficiency may not affect clotting mechanisms. Due to its critical role in bone formation, long-term vitamin K deficiency may impair bone integrity and growth, eventually predisposing a person to osteoporosis. Anticoagulants such as Coumadin and other warfarins can deplete vitamin K by blocking the activation of prothrombin. Excess vitamin K will not adversely affect clotting function for patients. However, patients on warfarin or other blood anticoagulants should not supplement with vitamin K unless specifically recommended and approved by their physician. Other causes of deficiency include celiac disease, liver disease, certain medications (i.e. aspirin, Dilantin), very high doses of vitamins A and E (over 600 IU) and gastrointestinal disorders associated with the malabsorption of fats, such as bile duct obstruction, pancreatitis or inflammatory bowel disease.

FOOD SOURCES

Food	Serving	(µg)
Natto	3 oz.	850
Collard greens	1/2 cup	530
Kale, raw	1 cup	472
Turnip greens	1/2 cup	426
Swiss chard, raw	1 cup	299

Food	Serving	(µg)
Parsley, raw	1/4 cup	246
Broccoli	1 cup	220
Spinach, raw	1 cup	145
Watercress	1 cup	85
Lettuce	1 cup	46

REPLETION INFORMATION

The liver secures the amount of vitamin K required for the saturation of clotting factors. Supplementation with vitamin KI is recommended as it is the precursor of vitamin K2. As a result patients should receive benefits of both KI and K2. Vitamin K is a fat soluble vitamin so ingestion with fats or oils significantly increases absorption. Since up to 50% of the vitamin is manufactured by bacteria in the gut, the balance of intestinal microflora is important in maintaining adequate endogenous production of vitamin K. Antibiotic usage can upset this balance.

ZINC

PHYSIOLOGICAL 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.

FOOD SOURCES

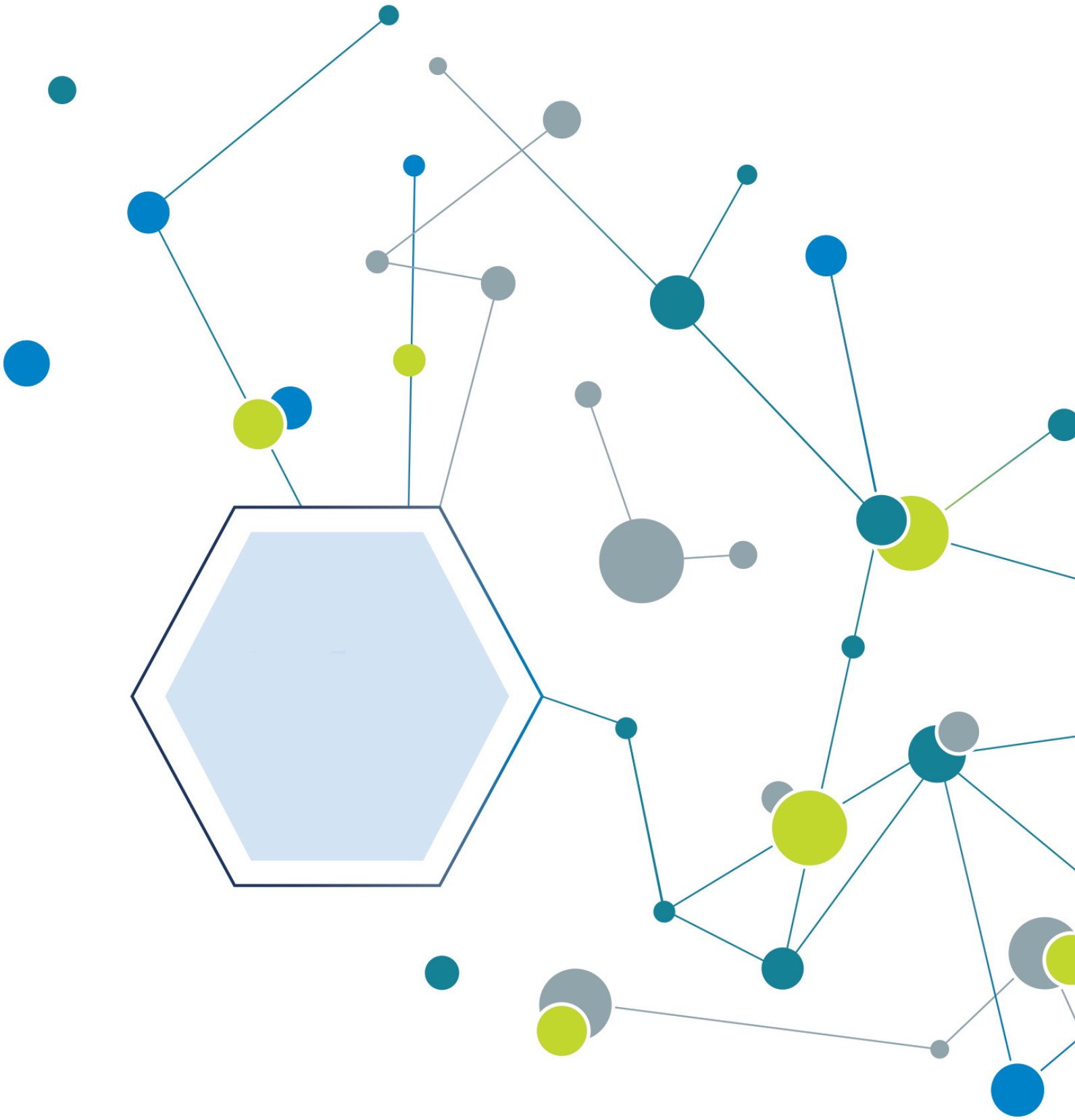
Food	Serving	(mg)
Oysters	3 oz.	74
Beef	3 oz.	6
Crab	3 oz.	5
Turkey	3 oz.	4
Chicken	2.4	2.4
Beans	1/2 cup	1.8

Food	Serving	(mg)
Yogurt	1 cup	1.8
Cahsews	1 oz.	1.6
Chickpeas	1/2 cup	1.3
Almonds	1 oz.	1.0
Milk	1 cup	1.0
Cheese	1 oz.	0.9

Phytates (in whole grains and beans) will inhibit zinc absorption.

REPLETION INFORMATION

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.



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