SPECTRACELL LABORATORIES

LABORATORY REPORT

Account Number: 186506

John Doe, M.D.
1234 Any Street
Suite 244
Anytown, TX 77581-1234
USA

Name: **Janet Doe**
Gender: Female DOB: 04/10/1971

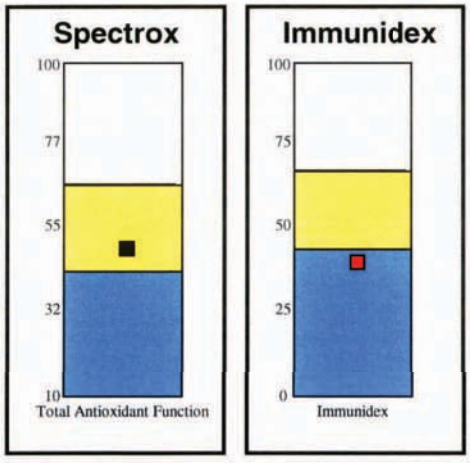
Accession Number: K88809
Requisition Number: 438507

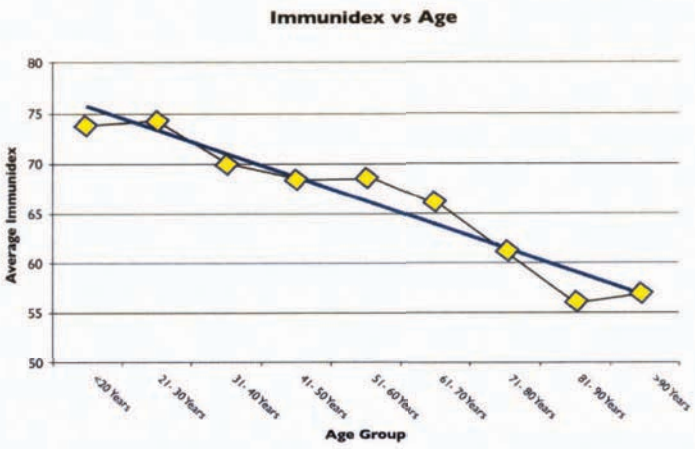
Date of Collection: 01/10/2012
Date Received: 01/11/2012
Date Reported: 01/20/2012

Summary of Deficient Test Results

Testing determined the following functional deficiencies:

Vitamin B1	Glutathione	Selenium	Vitamin E
Vitamin K2	Immunidex		





John F. Crawford, Ph.D.
Laboratory Director

CLIA# 45D0710715

OVERVIEW OF TEST PROCEDURE

1. A mixture of lymphocytes is isolated from the blood.
2. These cells are grown in a defined culture medium containing optimal levels of all essential. nutrients necessary to sustain their growth in cell culture.
3. The T-lymphocytes are stimulated to grow with a mitogen (phytohemagglutinin) and growth is measured by the incorporation of tritiated (radioactive) thymidine into the DNA of the cells.

The growth response under optimal conditions is defined as 100%, and all other growth rates are compared to this 100% level of growth.

For example – we remove vitamin B6 from the medium and stimulate the cells to grow by mitogen stimulation. Growth is measured by DNA synthesis and the rate of growth is dependent only upon the functional level of vitamin B6 available within the cells to support growth. For Vitamin B6 a growth rate of at least 55% of the growth rate observed in the optimal (100%) media is considered normal. Results less than 55% are considered to indicate a functional deficiency for Vitamin B6. Each nutrient has a different reference range that was established by assaying thousands of apparently healthy individuals.

BREAKING DOWN THE REPORT

1. TEST RESULT (% CONTROL)

This column represents the patient's growth response in the test media measured by DNA synthesis as compared to the optimal growth observed in the 100% media.

2. FUNCTIONAL ABNORMALS

An interpretation is provided for those nutrients found to be deficient.

3. REFERENCE RANGE

This column represents how this patient's result compares to thousands of patients previously tested. A patient's result is considered deficient when it is less than the reference range.

4. GRAPHS

The abnormal range of results is noted in the blue area. Abnormal results are indicated in red. The gray cross hatch area is a representation of the range of test results found in a random selection of subjects.

SPECTROX® – TOTAL ANTIOXIDANT FUNCTION

SPECTROX® is a measurement of overall antioxidant function. The patient's cells are grown in the optimal media, stimulated to grow, and then increasing amounts of a free radical generating system (H_2O_2) are added. The cell's ability to resist oxidative damage is determined. The increasing levels of peroxide will result in diminished growth rates in those patients with poor antioxidant function capacity.

INDIVIDUAL ANTIOXIDANT LEVELS

In the tests for individual antioxidants, it is determined which specific antioxidants may be deficient and thus affecting the SPECTROX® antioxidant function result. For these tests, the patient's cells are preincubated with one of the nutrient antioxidants, i.e. selenium, and then the Spectrox® test is repeated to determine if the addition of selenium improves the patient's antioxidant function. This process is repeated for each individual antioxidant.

Antioxidants tested with this process:
Glutathione, Cysteine, Coenzyme-Q10, Selenium, Vitamin E, and Alpha Lipoic Acid

Repletion Suggestions

1. Vitamin B1 (Thiamin)	50 mg daily
2. Glutathione	600 mg t.i.d. (1800 mg daily) of N-Acetylcysteine (NAC). Reduce to 1200 mg daily after 3 months and retest after 6 months. Take each dose with a meal
3. Selenium	200 mcg daliy of selenium glycinate or selenomethionine for 3 months and then reduce to 100 mcg daily
4. Vitamin E (A-tocopherol)	400 IU daily of mixed tocopherols
5. Vitamin K2	100 mcg vitamin K1 (K2 precursor) daily
6. Immunidex	Immunidex is an evaluation of the patient's T-lymphocyte's response to mitogen stimulation. Low responses may be related to the patient's levels of stress, nutritional deficiencies, or pathology. Improvement in the patient's Immunidex may be facilitated by correction of the functional deficiencies reported in this analysis.

Please note: Supplementation is usually required for four to six months to effect the repletion of a functional deficiency in lymphocytes

Suggestions for supplementation with specific micronutrients must be evaluated and approved by the attending physician. This decision should be based upon the clinical condition of the patient and the evaluation of the effects of supplementation on current treatment and medication of the patient.

Micronutrients	Patient Results (% Control)	Functional Abnormals	Reference Range (greater than)
B Complex Vitamins			
Vitamin B1 (Thiamin)	76	Deficient	>78%
Vitamin B2 (Riboflavin)	53		>53%
Vitamin B3 (Niacinamide)	86		>80%
Vitamin B6 (Pyridoxine)	61		>54%
Vitamin B12 (Cobalamin)	21		>14%
Folate	35		>32%
Pantothenate	9		>7%
Biotin	36		>34%
Amino Acids			
Serine	33		>30%
Glutamine	40		>37%
Asparagine	55		>39%
Metabolites			
Choline	30		>20%
Inositol	68		>58%
Carnitine	59		>46%
Fatty Acids			
Oleic Acid	76		>65%
Other Vitamins			
Vitamin D3 (Cholecalciferol)	65		>50%
Vitamin A (Retinol)	81		>70%
Vitamin K2	26	Deficient	>30%
Minerals			
Calcium	48		>38%
Manganese	69		>55%
Zinc	44		>37%
Copper	48		>42%
Magnesium	55		>37%
Carbohydrate Metabolism			
Glucose-Insulin Interaction	46		>38%
Fructose Sensitivity	52		>34%
Chromium	54		>40%
Antioxidants			
Glutathione	32	Deficient	>42%
Cysteine	43		>41%
Coenzyme Q-10	97		>86%
Selenium	74	Deficient	>74%
Vitamin E (A-tocopherol)	79	Deficient	>84%
Alpha Lipoic Acid	83		>81%
Vitamin C	50		>40%
SPECTROX™			
Total Antioxidant Function	44		>40%
Proliferation Index			
Immunidex	37	Deficient	>40%

The reference ranges listed in the above table are valid for male and female patients 12 years of age or older.

Adequate
Borderline
Deficient

Deficient
Values in this area represent a deficiency and may require nutrient repletion or dietary changes

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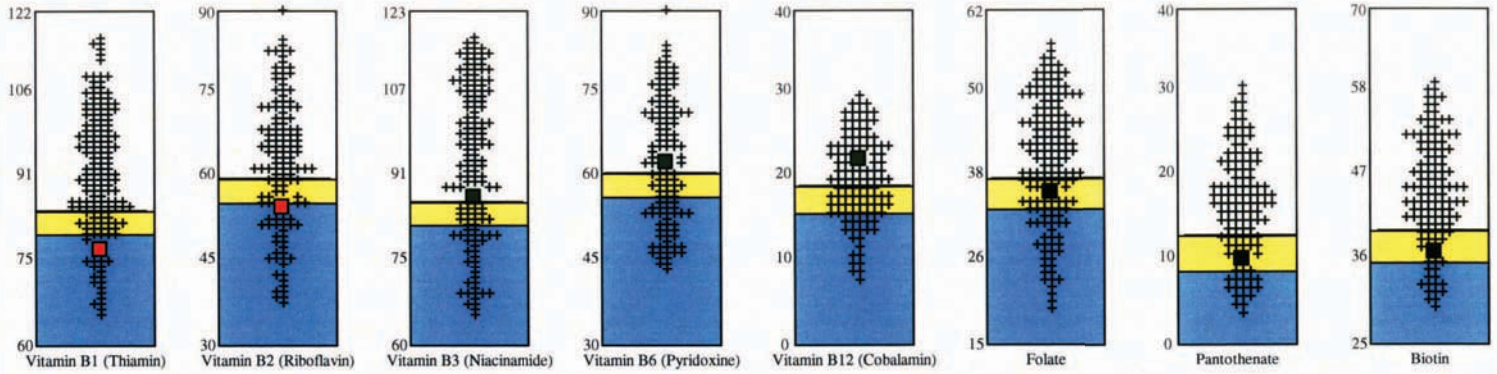
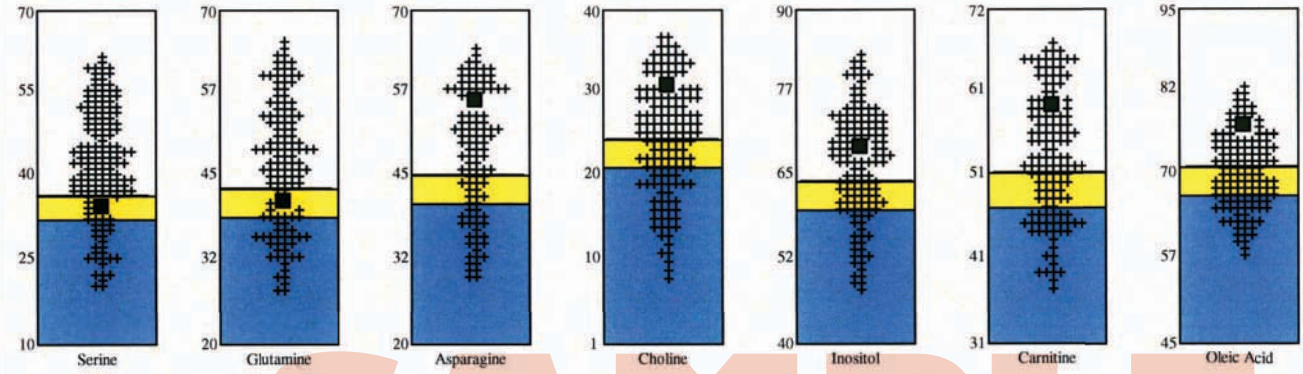
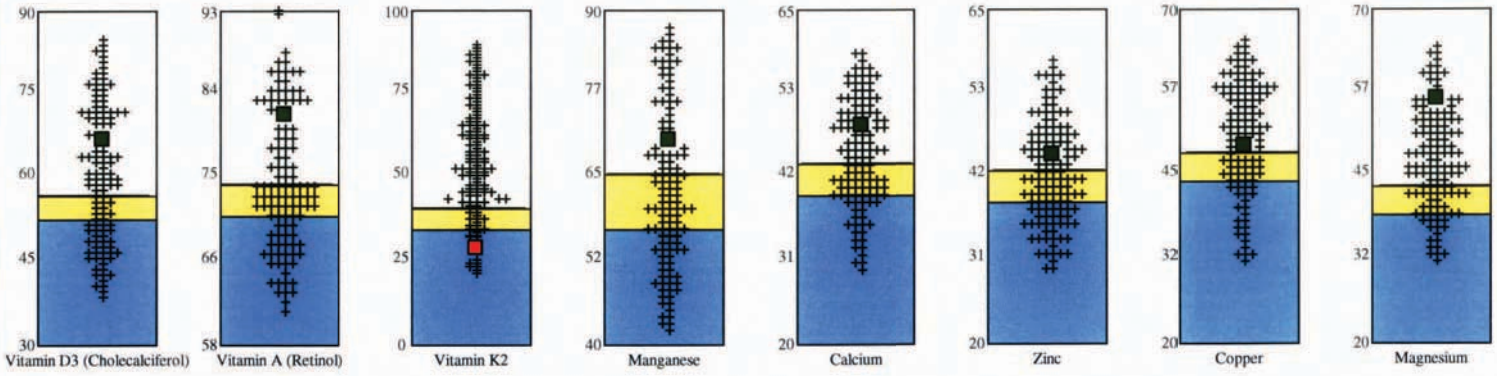
B Complex Vitamins



Amino Acids & Metabolites



Other Vitamins & Minerals



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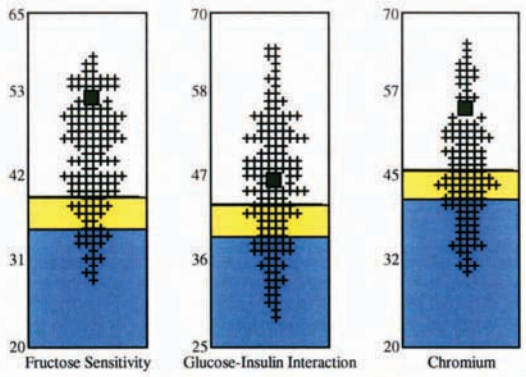
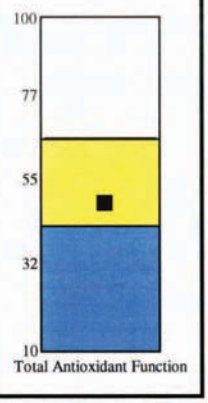
Carbohydrate Metabolism



Spectrox

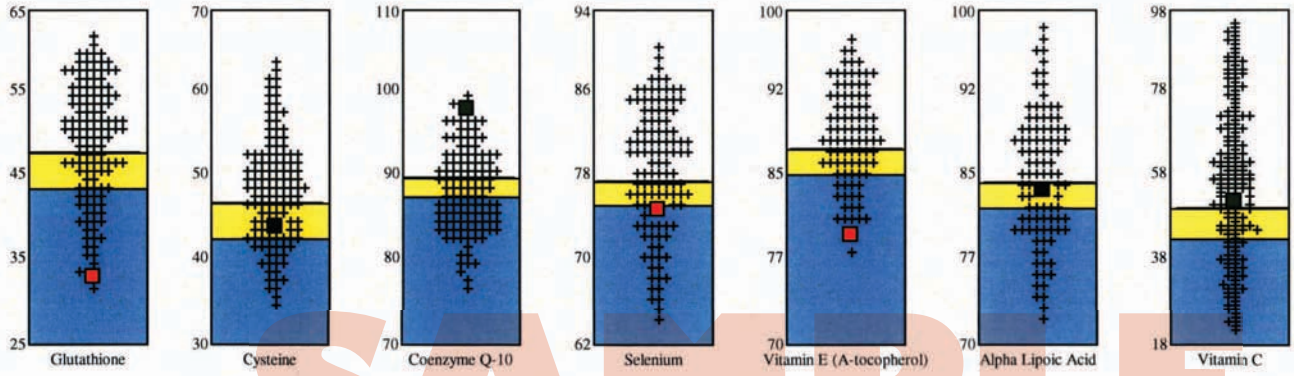


A Spectrox value above 65%-
indicates a desirable status for apparently healthy individuals. Since antioxidants are protective nutrients, the most desired status would be the greatest ability to resist oxidative stress.

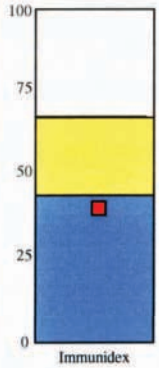
A Spectrox value between 40% and 65%-
indicates an average antioxidant function for apparently healthy individuals. An average status means the ability to resist oxidative stress similar to the majority of persons. However, average status is not ideal, nor is it clearly deficient.

A Spectrox value below 40%-
indicates a deficient antioxidant function resulting in a decreased ability to resist oxidative stress or an increased antioxidant load.

Individual Antioxidants



Immunidex



The Immunidex is an indication of the patient's T-Lymphoproliferative response to mitogen stimulation relative to the response of a control population. An average or weakened immune response may improve with correction of the nutritional deficiencies determined by the micronutrient testing.

An Immunidex above 65% indicates a strong response, a measurement of cell-mediated immune function.

An Immunidex between 40% and 65% - indicates an average response.

An Immunidex below 40% may indicate a weakened cell mediated immune response.

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SUPPLEMENTAL INFORMATION

Name: Janet Doe
Gender: Female DOB:
Accession Number: K88809

Date Received: 01/11/2012
Date Reported: 01/20/2012
Requisition Number:

Account Number:
Dr. Anyone
1234 Anywhere
City, State
USA

Accession Number:
Janet Doe

Vitamin B1 (Thiamin)

Status:

The patient's lymphocytes have shown a deficient status for Vitamin B1 (Thiamin)

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	Constipation	Nausea
Irritability	Mental Depression	Peripheral Neuropathy
Fatigue		

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

Mental Confusion	Loss of Fine Motor Control
Loss of Eye Coordination	Weakness

Those at risk for thiamin deficiency include:

Patients suffering from Malnutrition, Starvation or Malabsorption Syndromes

Alcoholics	Elderly
Patients on restricted diets	Patients with an increased metabolic rate
Prolonged hemodialysis (pregnancy, lactation, fever, infection, trauma)	
Gastric partitioning surgery	Inherited Thiamin-Responsive Metabolic Disorders

Repletion Information:

Dietary sources richest in B_1 (per serving) include:

Nutritional Supplements	Nutritional Yeasts
Rice Bran	Wheat Germ
Pork	Enriched Grain & Grain Products (cereals)
Legumes (beans, peas, soybeans, lentils)	

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. The 1989 RDA for thiamin is between 1.0-1.5 mg for adults. There is no evidence of thiamin toxicity form oral administration, cxccpt for development of sensitivity in very rare cases.

Glutathione

Status:

The patient's lymphocytes have shown a deficient status for Glutathione.

Function:

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

Deficiency Symptoms:

A wide range of human conditions 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.

Repletion Information:

Glutathione is poorly absorbed from the gastrointestinal tract and foods rich in glutathione do not appear to contribute to increases in intracellular glutathione levels. Cysteine appears to be the limiting amino acid in the intracellular synthesis of glutathione and supplementation with up to 2000 mg daily of N-Acetyl-L-Cysteine 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. Foods rich in cysteine are generally high protein foods such as meats, yogurt, wheat germ and eggs.

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Selenium

Status:

The patient's lymphocytes have shown a deficient status for selenium.

Function:

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

Deficiency Symptoms:

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

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

Repletion Information:

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

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

Wheat Germ	Bran
Brazil Nuts	Red Swiss Chard
Whole Wheat Bread	Oats
Brown Rice	Turnips

The adult RDA for selenium is 50 mcg/day.

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Vitamin E (a-tocopherol)

Status:

The patient's lymphocytes have shown a deficient status for vitamin E

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 pro-inflammatory cytokines. In deficiencies of vitamin E, arachidonic acid is converted to pro-inflammatory leukotrienes and cytokines. In neutralizing free radicals, vitamin E is oxidized to a free radical. Conversion back to the reduced form occurs by reaction with vitamin C (ascorbate).

Deficiency Symptoms:

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

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

Repletion Information:

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

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

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Vitamin K

Status:

The patient's lymphocytes have shown a deficient status for vitamin K2.

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_1, a natural form found in plants (phylloquinone); K_2, which is synthesized in the intestine (menaquinone); and K_3, 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.

Repletion Information:

The RDA for vitamin K is 1 microgram (mcg) per 2.2 pounds of body weight, with 80 mcg per day (males) and 65 mcg per day (females) being the officially recognized amount, although therapeutic doses range from 100 to 500 mcg per day. No Tolerable Upper Intake Level for vitamin K has been established. The liver secures the amount of vitamin K required for the saturation of clotting factors. Supplementation with vitamin K1 is recommended as it is the precursor of vitamin K2. As a result patients should receive benefits of both K1 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. Exogenous food sources particularly rich in vitamin K include kale, green tea, turnip greens, spinach, and broccoli. Other sources include lettuce, cabbage, beef liver, asparagus, watercress, cheese, oats, peas, and whole wheat.