SPECTRACELL LABORATORIES

LABORATORY REPORT

Account Number: 193113

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United Kingdom of Great B

Name: **Damien Blenkinsopp**
Gender: Male DOB: 11/13/1974

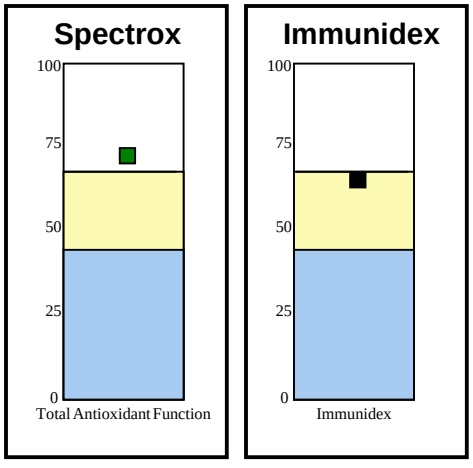
Accession Number: L37512
Requisition Number:

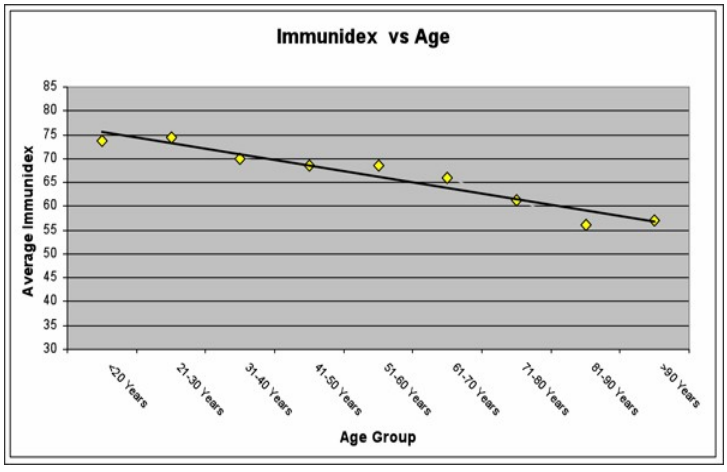
Date of Collection: 09/11/2012
Date Received: 09/12/2012
Date Reported: 09/21/2012

Summary of Deficient Test Results

Testing determined the following functional deficiencies:

Vitamin B12 Pantothenate Serine Insulin
Vitamin K2





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 B12 (Cobalamin)	300 mcg daily (methylcobalamin or adenosylcobalamin)
2. Pantothenate	500 mg b.i.d. (1000 mg daily)
3. Serine	1000 mg daily of L-serene Take 30 minutes prior to protein intake.
4. Glucose-Insulin Interaction	Replace intake of foods with high glycemic index (sugar, white flour) with whole foods (fruit, vegetables, whole grains, legumes). If chromium deficient, please see repletion for chromium.
5. Vitamin K2	100 mcg vitamin K1 (K2 precursor) daily

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)	92		>78%
Vitamin B2 (Riboflavin)	58		>53%
Vitamin B3 (Niacinamide)	91		>80%
Vitamin B6 (Pyridoxine)	66		>54%
Vitamin B12 (Cobalamin)	13	Deficient	>14%
Folate	45		>32%
Pantothenate	6	Deficient	>7%
Biotin	36		>34%
Amino Acids			
Serine	24	Deficient	>30%
Glutamine	41		>37%
Asparagine	56		>39%
Metabolites			
Choline	29		>20%
Inositol	72		>58%
Carnitine	53		>46%
Fatty Acids			
Oleic Acid	70		>65%
Other Vitamins			
Vitamin D3 (Cholecalciferol)	72		>50%
Vitamin A (Retinol)	81		>70%
Vitamin K2	27	Deficient	>30%
Minerals			
Calcium	52		>38%
Manganese	57		>50%
Zinc	48		>37%
Copper	54		>42%
Magnesium	56		>37%
Carbohydrate Metabolism			
Glucose-Insulin Interaction	37	Deficient	>38%
Fructose Sensitivity	42		>34%
Chromium	45		>40%
Antioxidants			
Glutathione	45		>42%
Cysteine	43		>41%
Coenzyme Q-10	96		>86%
Selenium	80		>74%
Vitamin E (A-tocopherol)	88		>84%
Alpha Lipoic Acid	86		>81%
Vitamin C	53		>40%
SPECTROX™			
Total Antioxidant Function	72		>40%
Proliferation Index			
Immunidex	62		>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

Borderline
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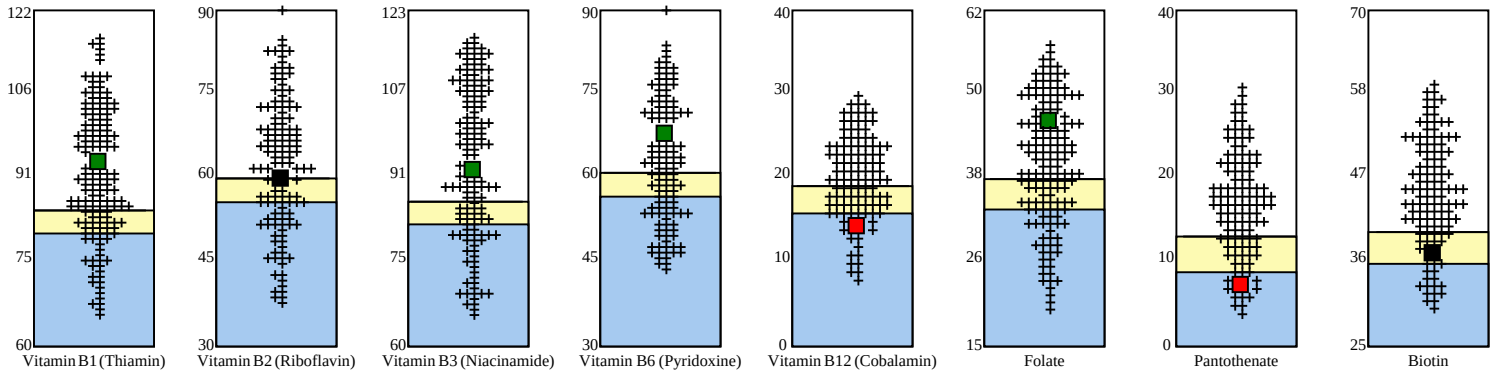
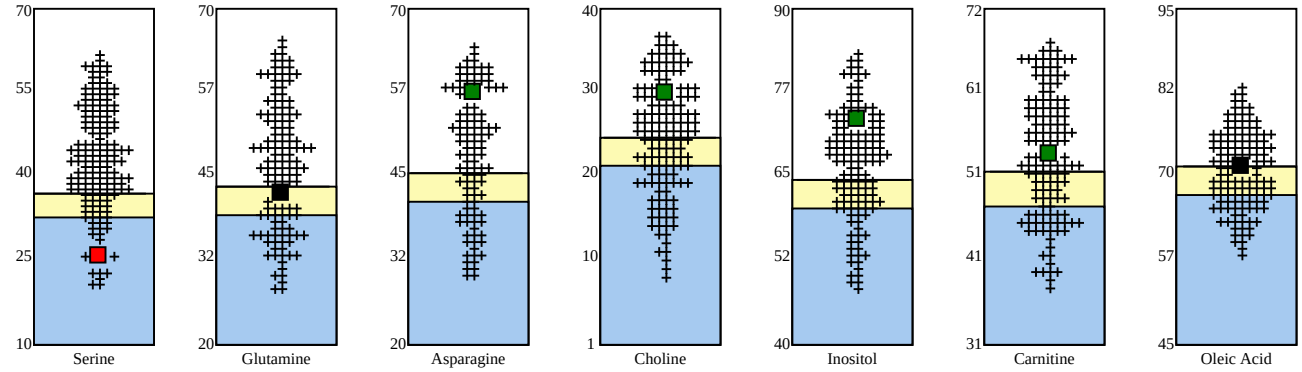
DamienBlenkinsopp

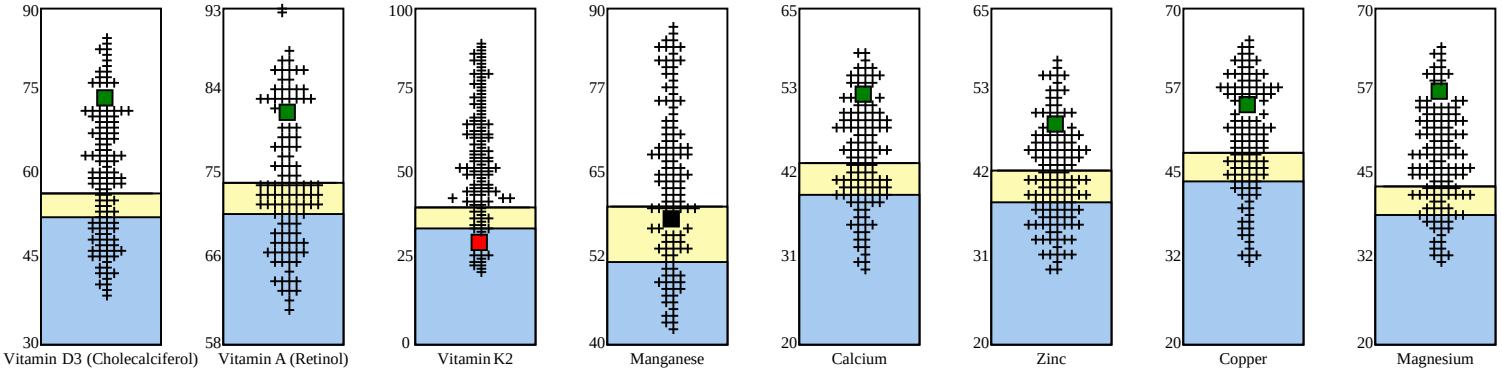
B Complex Vitamins



Amino Acids & Metabolites



Other Vitamins & Minerals



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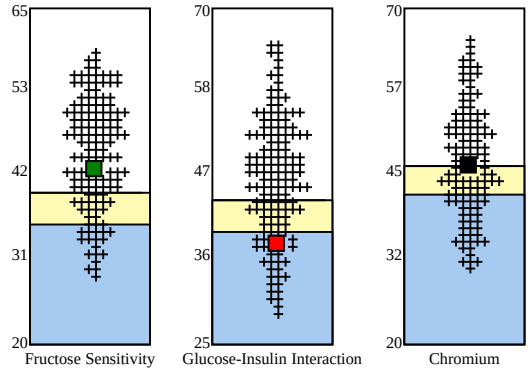
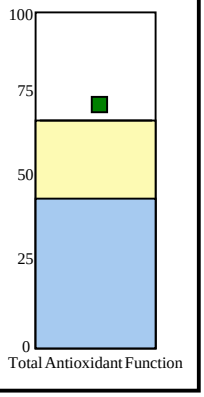
Carbohydrate Metabolism



Spectrox

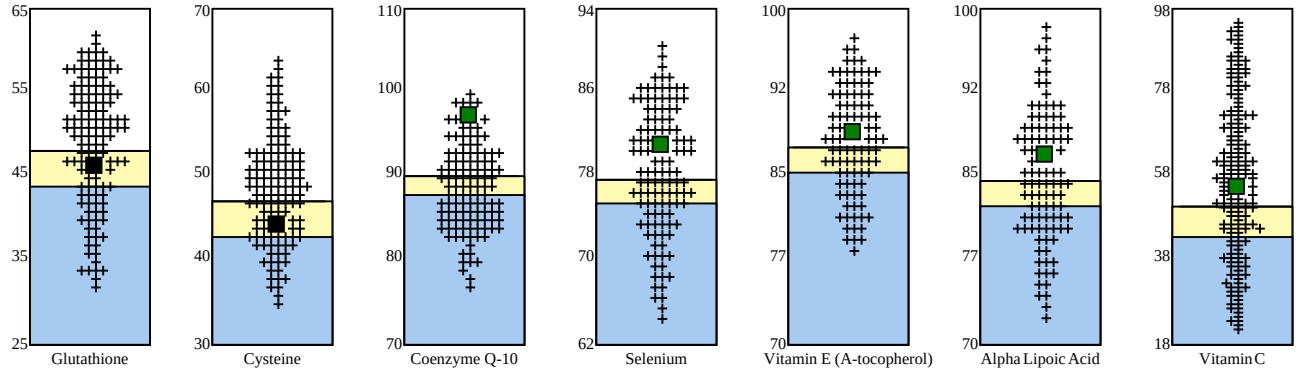


A Spectrox value above 65%-
indicates a desirable status for apparently healthy individuals. Since antioxidants are protective nutrients, the most desired status would be the greatest ability to resist oxidative stress.

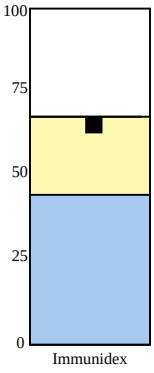
A Spectrox value between 40% and 65%-
indicates an average antioxidant function for apparently healthy individuals. An average status means the ability to resist oxidative stress similar to the majority of persons. However, average status is not ideal, nor is it clearly deficient.

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: **Damien Blenkinsopp**
Gender: Male DOB: 11/13/1974
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Requisition Number:

Account Number: 193113
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United Kingdom of Great B

Vitamin B12 (Cobalamin)

Status:

The patient's lymphocytes have shown a deficient status for vitamin B12 (Cobalamins).

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, irritability, 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.

Repletion information:

Dietary sources for cobalamins are strictly from animal foodstuffs. Vitamin B12 is not found in plant foodstuffs. Dietary supplements can also contain vitamin B12

The 1989 RDA for vitamin B12 is 2.0 ug for adults. 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.

Pantothenate

Status:

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

Function:

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

Deficiency Symptoms:

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

Repletion Information:

Dietary sources richest in Pantothenate (per serving) include:

Nutritional Supplements	Nutritional Yeasts
Meats	Legumes
Whole Grain Products	Wheat Germ
Vegetables	Nuts
Seeds	

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

Serine

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Status:

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

Function:

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

Deficiency Symptoms:

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

Repletion Information:

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

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Glucose-Insulin Interaction

Status:

The patient's lymphocytes have shown a deficient status for Glucose-Insulin Interaction.

Function:

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

Deficiency Symptoms:

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

Repletion Information:

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

Since chromium status is closely linked with insulin function and glucose tolerance, a chromium deficiency is one possible reason for abnormal Glucose-Insulin Interaction.

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Vitamin K

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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.