SpectraCell Laboratories
Science + Health + Solutions

LABORATORY REPORT

Account Number: 283116

Michale Hartte, NNCP
1102 Long Ridge Drive
Kelowna, BC V1V 2W9
Canada

Name: **Michale Hartte**
Gender: Female DOB: 10/24/1967

Accession Number: S43004
Requisition Number: 1821141

Date of Collection: 06/20/2018
Date Received: 06/21/2018
Date Reported: 07/02/2018

Summary of Deficient Test Results

Testing determined the following functional deficiencies:

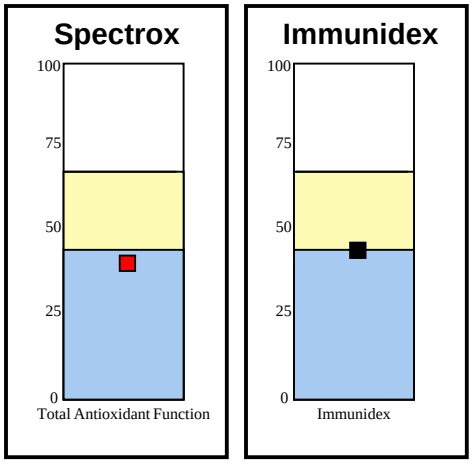
Vitamin B3	Asparagine	Oleic Acid	Insulin
Calcium	Coenzyme Q-10	Spectrox	

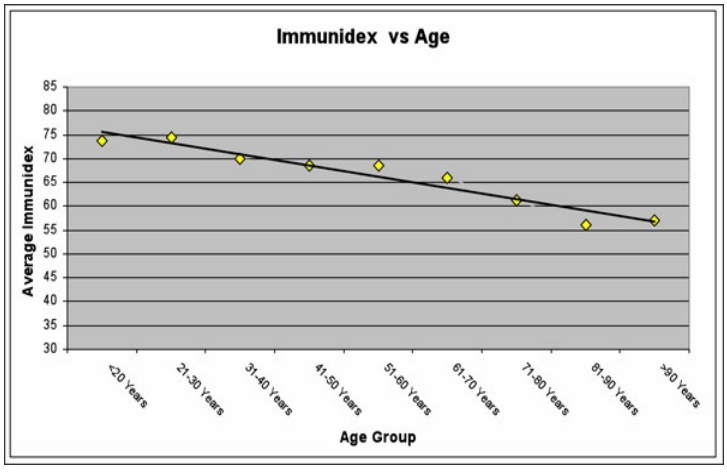
Borderline deficiencies include:

Folate	Serine	Carnitine	Immunidex

The assay for the CardioMetabolic Panel, Pre-Diabetes Panel, LPP Panel, Hormone Panel and Thyroid Panel require refrigerated serum from a centrifuged SST (red/black speckled serum separator tube). The SST received for this patient was not centrifuged; therefore the sample was not suitable for testing. Charges for the test(s) you requested requiring the SST have been canceled.

If repeat testing is desired, please collect an SST; allow it to clot for 30 minutes, then centrifuge for 20 minutes at 2200-2500 RPM. Thank you for your cooperation.





James W. Jacobson, 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, Alpha Lipoic Acid, and Vitamin C.

Repletion Suggestions

1. Vitamin B3 (Niacinamide)	100 mg b.i.d. (200 mg daily) of Niacin
2. Asparagine	500 mg t.i.d. (1500 mg daily) Take 30 minutes prior to protein intake.
3. Oleic Acid	2-3 tbsp olive oil daily for repletion of Oleic Acid. Deficiency of Oleic Acid suggests impaired synthesis of unsaturated long chain fatty acids. Take 600 mg b.i.d. (1.2 grams daily) of EPA and DHA in Omega-3 Fatty Acids.
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. Calcium	500 mg b.i.d. (1000 mg daily) as citrate, malate, ascorbate or glycinate
6. Total Antioxidant Function	Based on Spectrox and individual Antioxidant tests: * Glutathione: 600 mg daily of N-Acetylcysteine (NAC) * Cysteine: The daily dose of N-Acetylcysteine (NAC) listed for Glutathione is usually sufficient for Glutathione and/or Cysteine repletion. * Vitamin E: 200 IU daily of mixed tocopherols * Selenium: 50 mcg daily * Coenzyme Q10 Deficient: 50 mg t.i.d (150 mg daily) Take each dose with a meal * Lipoic Acid: 50 mg daily * Vitamin C: 250 mg 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)	84		>78%
Vitamin B2 (Riboflavin)	66		>53%
Vitamin B3 (Niacinamide)	74	Deficient	>80%
Vitamin B6 (Pyridoxine)	65		>54%
Vitamin B12 (Cobalamin)	23		>14%
Folate	36	Borderline	>32%
Pantothenate	23		>7%
Biotin	49		>34%
Amino Acids			
Serine	32	Borderline	>30%
Glutamine	55		>37%
Asparagine	37	Deficient	>39%
Metabolites			
Choline	33		>20%
Inositol	73		>58%
Carnitine	50	Borderline	>46%
Fatty Acids			
Oleic Acid	61	Deficient	>65%
Other Vitamins			
Vitamin D3 (Cholecalciferol)	73		>50%
Vitamin A (Retinol)	79		>70%
Vitamin K2	58		>30%
Minerals			
Calcium	33	Deficient	>38%
Manganese	71		>50%
Zinc	43		>37%
Copper	56		>42%
Magnesium	54		>37%
Carbohydrate Metabolism			
Glucose-Insulin Interaction	38	Deficient	>38%
Fructose Sensitivity	48		>34%
Chromium	52		>40%
Antioxidants			
Glutathione	50		>42%
Cysteine	53		>41%
Coenzyme Q-10	84	Deficient	>86%
Selenium	80		>74%
Vitamin E (A-tocopherol)	88		>84%
Alpha Lipoic Acid	88		>81%
Vitamin C	62		>40%
SPECTROX™			
Total Antioxidant Function	40	Deficient	>40%
Proliferation Index			
Immunidex	41	Borderline	>40%

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

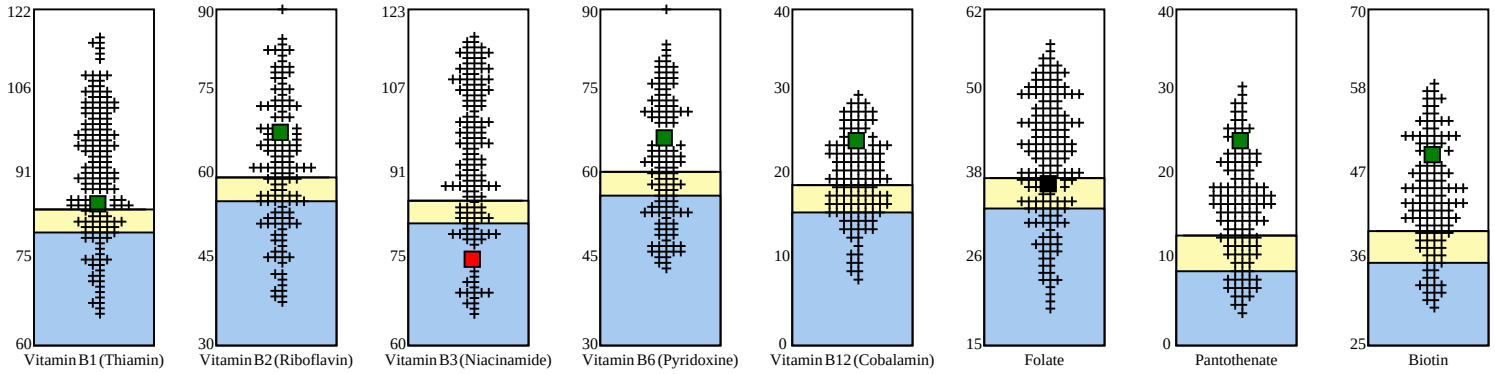
Adequate
Borderline
Deficient

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

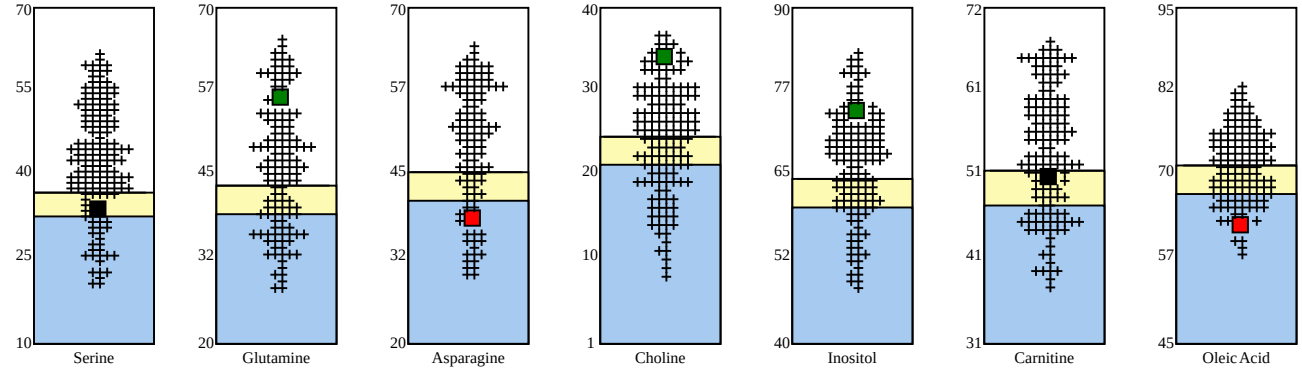
Borderline
Values in this area represent a borderline and may require nutrient repletion or dietary changes.

Accession Number: S43004
Michale Hartte

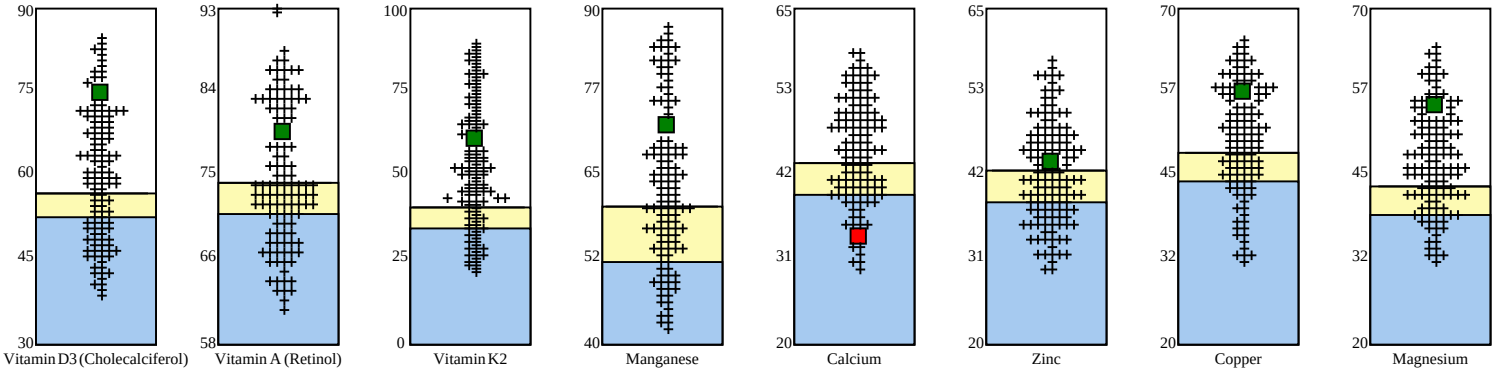
B Complex Vitamins



Amino Acids & Metabolites



Other Vitamins & Minerals



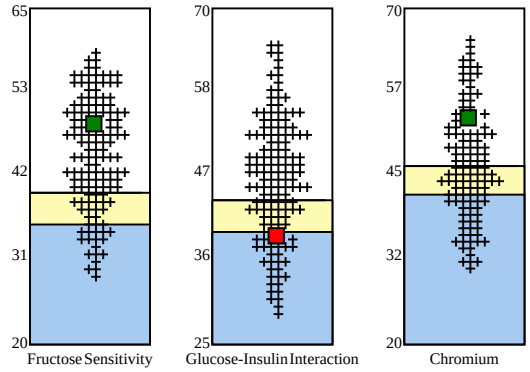
Adequate
Borderline
Deficient

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

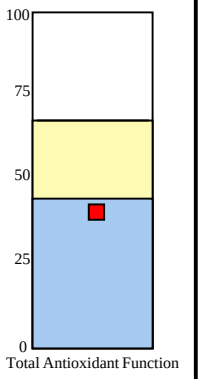
Borderline: Values in this area represent a borderline and may require nutrient repletion or dietary changes.

Accession Number: S43004
Michale Hartte

Carbohydrate Metabolism



Spectrox

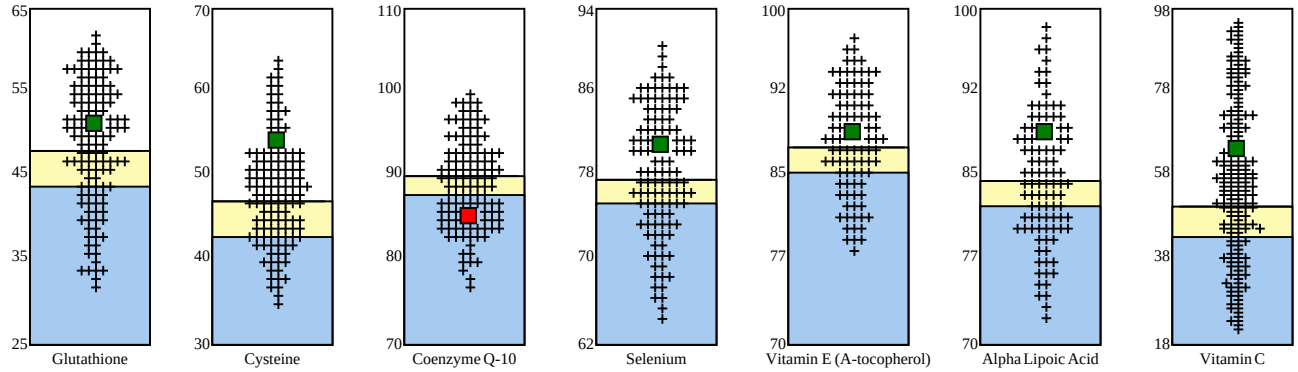


A Spectrox value above 65%-
indicates a desirable status for apparently healthy individuals. Since antioxidants are protective nutrients, the most desired status would be the greatest ability to resist oxidative stress.

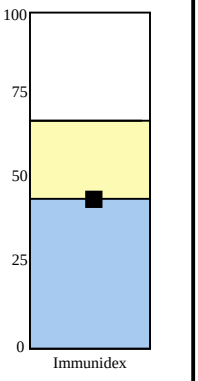
A Spectrox value between 40% and 65%-
indicates an average antioxidant function for apparently healthy individuals. An average status means the ability to resist oxidative stress similar to the majority of persons. However, average status is not ideal, nor is it clearly deficient.

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.

SpectraCell Laboratories
Science + Health + Solutions

SUPPLEMENTAL INFORMATION

Name: **Michale Hartte**
Gender: Female DOB: 10/24/1967
Accession Number: S43004

Date Received: 06/21/2018
Date Reported: 07/02/2018
Requisition Number: 1821141

Account Number: 283116
Michale Hartte, NNCP
1102 Long Ridge Drive
Kelowna, BC V1V 2W9
Canada

Vitamin B3 (Niacinamide)

Status:

The patient's lymphocytes have shown a deficient status for Vitamin B3 (Niacinamide, Nicotinamide, Niacin, Nicotinic Acid).

Function:

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

Deficiency Symptoms:

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

Repletion Information:

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

Nutritional Supplements	Nutritional Yeasts
Meats	Legumes including peanuts
Enriched Cereals	Potatoes

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

Asparagine

Status:

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

Function:

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

Deficiency Symptoms:

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

Repletion Information:

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

Oleic Acid

Status:

The patient's lymphocytes have shown a deficient status for Oleic Acid (long-chain, monounsaturated, fatty acid)

Function:

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

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

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

Deficiency Symptoms:

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

Repletion Information:

Dietary sources rich in Oleic Acid include:

Canola Oil	Olive Oil
Avocado Oil	Almond Oil
Avocados	High Oleic Safflower Oil

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

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

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

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.

Calcium

Status:

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

Function:

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

Deficiency Symptoms:

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

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

Repletion Information:

Dietary Sources richest in Calcium (per serving) are:

Calcium Supplements	Multiple Vitamin/Mineral Supplements with Calcium
Tofu	Milk and Dairy Products (milk, yogurt, cheeses)
Bone Meal	Canned Salmon & Sardines (with bones)

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

Coenzyme Q-10

Status:

The patient's lymphocytes have shown a deficient status for coenzyme Q-10

Function:

Coenzyme Q-10 belongs to a family of substances called ubiquinones. These compounds are lipophilic, water-insoluble substances involved in electron transport and energy production within the mitochondria. In this capacity, coenzyme Q-10 facilitates the conversion of the energy released through glycolysis into ATP (adenosine 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.

Repletion Information:

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

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

SPECTROX? (Total Antioxidant Function)

Function:

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

Oxidative Stress:

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

Cellular Antioxidants:

Protection of deleterious effects from free radicals is found in a diverse range of molecules termed *antioxidants*. Free radicals and their chain reaction byproducts can be neutralized and converted to less harmful products (quenched) by antioxidants. Antioxidants are enzymes (superoxide dismutase, catalase, glutathione peroxidase), essential nutrients (carotenoids, vitamin C, vitamin E, cysteine, selenium) or a wide variety of endogenous compounds (glutathione, sulfhydryl groups, thioredoxin, lipoic acid, coenzyme Q_{10}, 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 indicated 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.