NUTRIPRO DEMO

Name: NUTRIPRO DEMO
Date of Birth: 01-01-1111
Gender: Male
Age: 01
Height:
Weight:
Fasting: FASTING

Telephone: 000-000-0000
Street Address:
Email:

FINAL REPORT

Accession ID: 2311080002

Provider Information

Practice Name: DEMO CLIENT, MD
Provider Name: DEMO CLIENT, MD
Phlebotomist: 0

Telephone: 000-000-0000
Address: 3521 Leonard Ct, Santa Clara, CA 95054

Report Information

Current Result | Previous Result | In Control | Moderate | Risk

Specimen Information

Sample Type	Collection Time	Received Time	Report	Final Report Date
Serum	2023-11-27 11:00 (PDT)	2023-11-28 11:49 (PDT)	NutriPro - P2	2023-12-13 18:11 (PDT)
TES	2023-11-27 11:00 (PDT)	2023-11-28 11:49 (PDT)	NutriPro - P2	2023-12-13 18:11 (PDT)
EDTA	2023-11-27 11:00 (PDT)	2023-11-28 11:49 (PDT)	NutriPro - P2	2023-12-13 18:11 (PDT)

VibrantWellness
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INTRODUCTION

Vibrant Wellness is pleased to present NutriPro to help you make healthy lifestyle, dietary and treatment choices in consultation with your healthcare provider. It is intended to be used as a tool to encourage a general state of health and well-being. The Vibrant NutriPro Panel is a test to measure various genetic mutations present in an individual's body to gauge the scope of predispositions an individual might have towards achieving optimum nutrition, along with this the actual nutrient value is also measured in serum, RBC and WBC as appropriate. Thus, this panel is designed to give a complete picture of the predispositions that could lead to various nutrient deficiencies or toxicities, along with the current levels of these nutrients enabling a good overview of the potential nutrient pitfalls and how the individual's nutrient values are currently. Based on the genetic predispositions, personalized diet and supplement suggestions are offered to enable the provider and patient to make informed decisions to optimize nutrition.

Methodology:

The Vibrant NutriPro Genetics panel uses real-time PCR methodology. DNA is extracted and purified from blood samples and a SNP (single nucleotide polymorphism) genotyping assay is performed using real-time PCR to detect the specific allele targets of each assay performed.

The Vibrant NutriPro panel uses tandem mass spectrometry methodology (LC-MS/MS) for quantitative detection of the Intracellular (RBC & WBC) and Extracellular (Serum) Micronutrients markers.

Interpretation of Report:

The NutriPro Summary provides concise information on the abnormal genetic mutations, serum and cellular values along with corresponding results from previous testing (if applicable). This is followed by a complete list of all analytes tested with quantitative results and genetic mutations tested to enable a full overview along with the corresponding reference ranges. Reference ranges have been established using a cohort of 1000 apparently healthy individuals. The mutation alleles are indicated with a + symbol and wild type alleles are indicated with a – symbol. The classification of Red indicates a result that is outside the reference range and the classification of Green denotes a result that is within the reference range.

Any changes based on the information provided should be made in consultation with the ordering provider, primary care provider, specialist, or geneticist as appropriate. Pediatric ranges have not been established for this test.

The Vibrant Wellness platform provides tools for you to track and analyze your general wellness profile. Testing for the NutriPro panel is performed by Vibrant America, a CLIA certified lab CLIA#:05D2078809 and Vibrant Genomics, a CLIA certified lab CLIA#: 05D2098445. Vibrant Wellness provides and makes available this report and any related services pursuant to the Terms of Use Agreement (the "Terms") on its website at www.vibrant-wellness.com. By accessing, browsing, or otherwise using the report or website or any services, you acknowledge that you have read, understood, and agree to be bound by these terms. If you do not agree to these terms, you shall not access, browse, or use the report or website. The statements in this report have not been evaluated by the Food and Drug Administration and are only meant to be lifestyle choices for potential risk mitigation. Please consult your physician for medication, treatment, diet, exercise, or lifestyle management as appropriate. This product is not intended to diagnose, treat, or cure any disease or condition.

Please note:
It is important that you discuss any modifications to your diet, exercise, and nutritional supplementation with your physician before making any changes. The Vibrant America Clinical Support team can only provide basic and generalized interpretation of hormone biomarkers and pathways.

NutriPro			⊕⊕ Homozygous Mutant	⊕⊖ Heterozygous	⊖⊖ Homozygous Wild				
Vitamins and Minerals	SNP ID	Your Mutation	Serum Current	Serum Previous	Serum Reference	Cellular Current	Cellular Previous	Cellular Reference	
Chromium, Cr 53		*No mutation tested*	0.06		0.1-0.7 (ng/mL)				

PHYSIOLOGICAL FUNCTION

Chromium is an essential nutrient used in trace amounts in humans that acts as a cofactor for chromodulin, a peptide that enhances the effect of insulin on target tissues, which aids in regulation of blood sugar and lipid metabolism.

HOW IT GETS DEPLETED

Deficiency is very rare, but can occur in patients receiving IV parenteral nutrition without supplemental chromium added, and individuals who regularly participate in endurance exercise.

CLINICAL MANIFESTATIONS OF DEPLETION

Chromium deficiency can contribute to the development of diabetes and metabolic syndrome. Even mild deficiencies of chromium can produce problems in blood sugar metabolism, and contribute to symptoms such as anxiety or fatigue.

FOOD SOURCES

Food sources of chromium include brewer's yeast, especially beer, broccoli, grape juice, meat and whole-grain products. Some fruits, vegetables, and spices provide chromium. Romaine lettuce, raw onions and ripe tomatoes are all good sources.

SUPPLEMENT OPTIONS

The AI for chromium is 35 µg/day for men and 25 µg/day for women. Increased needs may be present during pregnancy and lactation. Supplemental chromium is generally not needed as dietary consumption easily meets physiological needs. Supplementation is poorly studied and insufficient evidence exists to provide recommendations, but chromium picolinate is a form commonly used in treatment of insulin resistance and diabetes.

Fluoride	rs4284505	⊕⊖ A/G			*No nutrient tested*				

GENETIC MUTATIONS

rs4284505: The ESR1 gene is responsible for enamel formation of the tooth. Mutations in the ESR1 gene affects enamel mineralization in the presence of flouride leading to dental flurosis that softens tissues of the teeth causing decay.The G allele heterozygous carriers (partially abnormal) are moderately affected by dental fluorosis when exposed to excessive fluoride containing water. Susceptible individuals must reduce consumption of acidic, sugary foods and drinks and must avoid drinking water containing fluoride.

Iodine	rs225014	⊕⊖ C/T			*No nutrient tested*				

GENETIC MUTATIONS

rs225014: DIO2 gene produces the D2 enzyme that is responsible for maintaining T3 (thyroid hormone) levels when the body is deficient in iodine. Thyroid hormones are vital for normal bone development and maintenance. This function is impaired upon DIO2 mutation which can lead to diseases of the bone such as osteoarthritis.Homozygous mutant (abnormal) individuals carry 1.3 to 1.79 times increased risk of osteoarthritis due to reduced thyroid levels. Seafood, sea vegetables and iodized salt are recommended to be included in the diet. Multivitamin/mineral supplements are recommnded in pregnant and lactating women who msy be prone to iodine deficiency and carry the CC and CT genotypes.

NutriPro			⊕⊕ Homozygous Mutant	⊕⊖ Heterozygous	⊖⊖ Homozygous Wild			
Vitamins and Minerals	SNP ID	Your Mutation	Serum Current	Serum Previous	Serum Reference	Cellular Current	Cellular Previous	Cellular Reference
Iron, Fe 56	rs4820268	⊖⊖ A/A	*No nutrient risk*					

GENETIC MUTATIONS

rs4820268: The TMPRSS6 gene regulates a protein called hepcidin, which balances the iron content in the body. Mutation suppresses the production of the hormone hepcidin, required for iron transport which leads to iron deficiency (anemia), one of the most frequent disorders worldwide. Anemic individuals include gastrointestinal disturbances and impaired cognitive function, immune function, exercise or work performance, and body temperature regulation. In infants and children, it can result in psychomotor and cognitive abnormalities and learning difficulties and deficiencies early in life and persist through adulthood. Individuals with heterozygous (normal) genotypes are susceptible to decrease in hepcidin levels which affects iron transport thus reduced iron, hemoglobin. Foods rich in iron such as dark green leafy vegetables, beans, tofu, baked potatoes, lentils, cashews, whole grain and enriched breads are recommended.

Magnesium, Mg 24	rs4680	⊕⊖ A/G	*No nutrient risk*					

GENETIC MUTATIONS

rs4680: The COMT gene encodes an enzyme which breaks down dopamine (neurotransmitter- molecules that help transmit signals) in the brain. Magnesium enhances the COMT activity. Therefore, individuals with the abnormal allele may benefit from eating foods rich in magnesium, such as pumpkin seeds, almonds, spinach boiled, cashews, and peanuts.

Myo-Inositol	*No mutation tested*		15.1		20.5-60.7 (nmol/mL)			

PHYSIOLOGICAL FUNCTION

Inositol derivatives are used in the cellular signaling process after the insulin receptor is activated; it is crucial for the development of peripheral nerves, helps move fats out of the liver, promotes the production of lecithin, and is anti-arteriosclerotic, and anti-atherogenic. Inositol is also stored in the liver, spinal cord nerves, and in the brain and cerebral spinal fluid.

HOW IT GETS DEPLETED

Inositol can be released from phytate compounds via intestinal bacteria breaking phytate-degrading enzymes (Lactobacillus plantarum, Lactobacillus brevis, Lactobacillus curvatus, L. gasseri, B. subtilis and Saccharomyces cerevisiae). If many courses of antibiotics are used, there may be some depletion of inositol from microbiome conversion.

CLINICAL MANIFESTATIONS OF DEPLETION

There do not appear to be any clinical manifestations of depletion of inositol. Inositol can be synthesized in the human body from glucose-6-phosphate, a derivative of glucose, therefore, deficiency would be rare. Conditions associated with depletion of inositol, however, are depression, anxiety, PCOS, diabetes, CVD, and obesity.

FOOD SOURCES

Good dietary sources of inositol include: oranges, cantaloupe, prunes, navy beans, grapefruit, limes, blackberries, kiwis, rutabagas, fresh green beans, unrefined molasses, stone ground wheat, bran flakes, and pumpernickel.

SUPPLEMENT OPTIONS

There is currently no established RDA, AI, or UL for inositol. Myo-inositol is noted for its benefits to female fertility and insulin sensitivity, and is used often in treatment for PCOS in dosages of 2-4g/day. Higher doses of inositol are used to treat psychiatric conditions like depression and anxiety/OCD in much higher doses of 12-18 g/day; some mild gastrointestinal distress is noted with the higher doses and may need to be consumed in split doses. Lowering blood glucose can be seen with doses of inositol around 2-4 g/day. Current supplementation of inositol has shown some promise in treating Alzheimer's to reduce progression of fibrosis formation. Inositol may decrease LDL-C and ApoB in persons with metabolic syndrome with doses of 5-10 g/day. Doses of inositol of 4 g/day have been associated with improvement of all markers of glycemic control and insulin resistance in gestational diabetes.

NutriPro			⊕⊕ Homozygous Mutant	⊕⊖ Heterozygous	⊖⊖ Homozygous Wild			
Vitamins and Minerals	SNP ID	Your Mutation	Serum Current	Serum Previous	Serum Reference	Cellular Current	Cellular Previous	Cellular Reference
Phosphorus	rs4074995	⊕⊖ G/A	*No nutrient tested*					

GENETIC MUTATIONS

rs4074995: RGS14 gene encodes for the kidney-specific sodium phosphate transporter protein (Npt2a) involved in urinary phosphorus excretion. Excess of serum phosphorus leads to the removal of these Npt2a transporters resulting in reduced phosphorus re-absorption and increased excretion of phosphorus in the urine. A low serum phosphorus concentration enhances phosphorus re-absorption in the kidney. However, deletion of the Npt2a gene may result in urinary phosphorus wasting and impaired skeletal development. Heterozygous mutations in Npt2a may lead to formation of kidney stones (nephrolithiasis) and calcium stones due to urinary phosphorus leak.Homozygous mutant (abnormal) individuals may have altered serum phosphorus levels. Phosphorus rich foods such as meats, poultry, fish, nuts, beans, and dairy products should be consumed to compensate for the phosphorus leakage. Limiting consumption of sugar-sweetened foods and drinks may benefit people who form kidney stones. A healthy diet containing vegetables, fruits, whole grains, and low fat dairy products is recommended.

Potassium	rs4343	⊕⊖ A/G	*No nutrient risk*					

GENETIC MUTATIONS

rs4343: The ACE gene associated with angiotensin-converting enzyme (ACE) activity regulates blood pressure by relaxing the veins and maintaining body salts and fluids. Potassium levels in the body regulates the ACE activity. High levels of potassium decreases its activity relaxing the veins. Mutations in the gene impairs this process leading to high blood pressure.Heterozygous (partially abnormal) individuals have intermediate concentrations of ACE serum levels. Individuals with a GG genotype may be advised high dietary potassium intake as potassium levels could reduce ACE activity and prevent high blood pressure.

Selenium, Se 76	rs1050450	⊕⊕ T/T	*No nutrient risk*					
	rs3877899	⊖⊖ G/G						

GENETIC MUTATIONS

rs1050450: The GPX1 gene activity is sensitive to changes in selenium status in individuals with low to moderate intake. Mutations in the gene causes oxidative stress increasing the risk of acquiring several diseases. Homozygous mutant (abnormal) T allele carriers have a tendency for selenium deficiency. Iodine deficient individuals are recommended to have iodine rich food such as fish (cod and tuna), seaweed, shrimp, and other seafood, dairy products (such as milk, yogurt, and cheese). Brazil nuts, pork, beef, turkey, chicken, fish, shellfish, and eggs contain high amounts of selenium. American diet such as breads, grains, meat, poultry, fish, and eggs can increase selenium levels.

rs3877899: SEPP1 gene aids in the transport of selenium in the bloodstream to tissues of the brain, testes, and placenta. It has an antioxidant activity. Mutation in the gene reduces antioxidant activity, leading to increased incidence of age-related diseases.The G allele in homozygous mutant (abnormal) pregnant women tends to cause decreased levels of whole blood selenium levels. Selenium supplementation is recommended in pregnant women who have low selenium levels. Selenium rich foods such as nuts, lean meats, poultry, eggs, seafood, beans, peas, lentils and soy products are recommended to prevent selenium deficiency in pregnant women. High amounts of selenium are found in pork, beef, turkey, chicken, fish, shellfish, and eggs.

NutriPro			⊕⊕ Homozygous Mutant	⊕⊖ Heterozygous	⊖⊖ Homozygous Wild			
Vitamins and Minerals	SNP ID	Your Mutation	Serum Current	Serum Previous	Serum Reference	Cellular Current	Cellular Previous	Cellular Reference
Sodium	rs2304478	⊕⊕ G/G	*No nutrient risk*					
	rs7204044	⊕⊖ A/G						

GENETIC MUTATIONS

rs2304478: The SLC12A3 gene regulates chloride transport and sodium reabsorption from the urine in the kidneys. It ensures normal functioning of kidneys , body fluids and blood pressure. Mutation in the gene leads to its increased expression and elevates sodium reabsorption in the kidney contributing to hypertension.Homozygous mutant (abnormal) individuals have a tendency for increased sodium reabsorption. Leafy green vegetables help eliminate excess sodium from the kidneys hence, affected people must include them in their diet along with blue berries, oatmeal, skim milk and yoghurt.

rs7204044: The SLC12A3 gene regulates chloride transport and sodium reabsorption from the urine in the kidneys. It ensures normal functioning of kidneys , body fluids and blood pressure. Mutation in the gene leads to its increased expression and elevates sodium reabsorption in the kidney contributing to hypertension.Homozygous mutant (abnormal) individuals have a tendency for increased sodium reabsorption. Leafy green vegetables help eliminate excess sodium from the kidneys hence, affected people must include them in their diet along with blue berries, oatmeal, skim milk and yoghurt.

NutriPro			⊕⊕ Homozygous Mutant	⊕⊖ Heterozygous	⊖⊖ Homozygous Wild			
Vitamins and Minerals	SNP ID	Your Mutation	Current	Serum Previous	Reference	Current	Cellular Previous	Reference
Vitamin A (All-Trans-Retinol)	rs1667255	⊖⊖ A/A	*No nutrient risk*					
	rs6564851	⊕⊖ G/T						
	rs11645428	⊕⊖ A/G						
	rs7501331	⊕⊖ C/T						

GENETIC MUTATIONS

rs1667255: The TTR gene encodes for a protein that transports vitamin A (retinol) throughout the body. The TTR mutation impairs efficient transport which causes low serum retinol levels. Vitamin A is required to maintain good eyesight and boost immunity.Homozygous mutant (abnormal) individuals have decreased circulating retinol levels Affected individuals with the AA genotype are recommended to have vitamin A rich foods such as carrots, spinach, brocolli.

rs6564851: The BCMO1 gene converts precursors of vitamin A (beta carotene) in the small intestine. Mutation causes reduced BCMO1 activity, which results in accumulation of unconverted β-carotene and to a lesser extent, α-carotene, leading to vitamin A deficiency.Homozygous mutants (abnormal) individuals have higher beta carotene levels in the body and can face increased risk of vitamin A deficiency Foods rich in vitamin A such as persimmon, tangerine, beef liver, fish, and milk are recommended for individuals with GT and GG genotypes to overcome the vitamin A deficiency.

rs11645428: The SNP on the BCMO1 gene is a useful biomarker for predicting visual function. It encodes for an enzyme that helps the body convert carotenoids to vitamin A. Vitamin A is required to synthesize pigments in the eye. Mutation of this gene leads to reduced conversion efficiency leading to vitamin A deficiency. Deficiency may affect vision and sensitivty, a condition called as macular pigment optical density (MPOD). The BCMO1 gene produces an enzyme that converts dietary β-Carotene, precursors of vitamin A. Mutation leads to the failure of this function efficiently. Heterozygous (partially abnormal) individuals carry the G allele which is associated with lower beta carotene levels hence may have vitamin A deficiency. Individuals with AG and GG genotypes lack the precursors such as lutein and zeaxanthin that are required to form vitamin A. They should consume dark green leafy vegetables which will ensure lutein and zeaxanthin intake. These are also present in colorful fruits and vegetables, such as broccoli, orange peppers,corn, peas, persimmons and tangerines. Nutritional supplements are required to maintain good eye health. Affected individuals are advised to consume vitamin A-rich foods such as persimmon, tangerine, beef liver, fish, and milk.

rs7501331: This SNP on the BCO1 gene codes for an enzyme that enables the body to produce vitamin A1 (retinol) from beta carotenes required to boost immune cells functioning. Mutation leads to the reduced activity of the enzyme and affects vitamin A1 conversion leading to vitamin A deficiency. This impairs immunity and causes eyesight issues. In severe cases, it may be a cause of mortality in pregnant as well as lactating women and preschool children.Heterozygous (partially abnormal) individuals with TC genotypes possess reduced ability to convert beta-carotene to retinol. Individuals with TC and TT genotypes are recommended to consume animal derived vitamin A supplements which mainly include liver and fish oil. Other good sources are milk, dairy products, eggs, chicken, fish and meat.

Vitamin A (Beta-Carotene)	rs11645428	⊕⊖ A/G	*No nutrient tested*					

GENETIC MUTATIONS

rs11645428: The SNP on the BCMO1 gene is a useful biomarker for predicting visual function. It encodes for an enzyme that helps the body convert carotenoids to vitamin A. Vitamin A is required to synthesize pigments in the eye. Mutation of this gene leads to reduced conversion efficiency leading to vitamin A deficiency. Deficiency may affect vision and sensitivty, a condition called as macular pigment optical density (MPOD). The BCMO1 gene produces an enzyme that converts dietary β-Carotene, precursors of vitamin A. Mutation leads to the failure of this function efficiently. Heterozygous (partially abnormal) individuals carry the G allele which is associated with lower beta carotene levels hence may have vitamin A deficiency. Individuals with AG and GG genotypes lack the precursors such as lutein and zeaxanthin that are required to form vitamin A. They should consume dark green leafy vegetables which will ensure lutein and zeaxanthin intake. These are also present in colorful fruits and vegetables, such as broccoli, orange peppers,corn, peas, persimmons and tangerines. Nutritional supplements are required to maintain good eye health. Affected individuals are advised to consume vitamin A-rich foods such as persimmon, tangerine, beef liver, fish, and milk.

NutriPro			⊕⊕ Homozygous Mutant		⊕⊖ Heterozygous		⊖⊖ Homozygous Wild	
Vitamins and Minerals	SNP ID	Your Mutation	Serum Current	Serum Previous	Serum Reference	Cellular Current	Cellular Previous	Cellular Reference
Vitamin B1 (Thiamine diphosphate)	rs17514104	⊕⊕T/T				0.06		0.1-7.0 (pg/MM WBC)

GENETIC MUTATIONS

rs17514104: SLC35F3 gene encodes thiamine transporter protein which regulates vitamin B1 (thiamine diphosphate) uptake by the cells and mutation inhibit this process leading to thiamine deficiency.Homozygous mutant (abnormal) individuals with the risk allele (T) display decreased thiamine levels in the blood. Thiamine diphosphate or vitamin B1 supplementation is recommended in affected individuals. They are recommended to include thiamine rich foods such as whole grains, yogurt, lean meats, poultry, eggs, seafood, beans, peas, and lentils, nuts, seeds, and soy products. Pork, fish, and seafood are good sources of thiamine.

PHYSIOLOGICAL FUNCTION

Vitamin B1 aids in energy transformation and production of ATP. It acts as a coenzyme in the breakdown of carbohydrates, fats and proteins to produce energy.

HOW IT GETS DEPLETED

Thiamin can become depleted or deficient from frequent consumption of thiaminases present in higher amounts in raw fish and tannins/tannic acid (tea and coffee). Thiamin is vulnerable to loss during cooking. Can be depleted with excessive or chronic alcohol intake. There may be higher risk of depletion with gastric bypass surgery.

CLINICAL MANIFESTATIONS OF DEPLETION

Thiamin deficiency can lead to nervous system and cardiac abnormalities. The most severe form of thiamin deficiency is called beri beri, a condition commonly resulting in weakness, fatigue, confusion, irritability, weight loss, muscle wasting, and peripheral neuropathy.

FOOD SOURCES

Food sources of thiamin include: pork, organ meats, legumes, sweet potato, brown rice, brewer's yeast, pine nuts, sunflower seeds, enriched grains.

SUPPLEMENT OPTIONS

The RDA for thiamin is 1.0 mg/day for females and 1.2 mg/day for males. The RDA for thiamin during pregnancy/lactation is 1.4 mg/day. Therapeutic intake of thiamin is commonly 25-100mg/day. No UL for thiamin has been set. Thiamin is commonly found in multi-B vitamin products.

NutriPro			⊕⊕ Homozygous Mutant		⊕⊖ Heterozygous		⊖⊖ Homozygous Wild	
Vitamins and Minerals	SNP ID	Your Mutation	Serum Current	Serum Previous	Serum Reference	Cellular Current	Cellular Previous	Cellular Reference
Vitamin B12 (Cyanocobalamin)	rs602662	⊕⊕ G/G	>2000		232.0-1245.0 (pg/mL)			

GENETIC MUTATIONS

rs602662: Variation in the FUT2 gene ensure absorption of vitamin B12 responsible for the normal functioning of the brain, nervous system and the formation of red blood cells. It produces an enzyme which enhances vitamin B12 absorption. Mutations in the gene affects efficient absorption resulting in low levels of vitamin B12.Homozygous mutant (abnormal) individuals, adhering to a vegetarian diet have significantly lower levels of vitamin B(12). Good sources of vitamin B12 includes meat, salmon, milk, dairy products and eggs. Older individuals may be advised to take supplements.

PHYSIOLOGICAL FUNCTION

Vitamin B12 is an important coenzyme when in its active form of methylcobalamin. B12 facilitates the metabolism of folic acid through its primary role as a methyl donor. B12 requires intrinsic factor for absorption, which is calcium dependent. The role of vitamin B12 in the production of some neurotransmitters may also be evidenced by mood imbalance in susceptible individuals.

HOW IT GETS DEPLETED

Age is a risk factor for deficiency of B12 due to a natural decline in intrinsic factor. Chronic use of PPIs may reduce HCl and lead to sub-clinical deficiencies. Some genetic SNPs (such as MTHFR) may lead to deficiencies in active B12 (methylcobalamin).

CLINICAL MANIFESTATIONS OF DEPLETION

Deficiency of B12 can appear as pernicious anemia, usually due to lack of intrinsic factor. Another form of anemia associated with B12 deficiency is megaloblastic anemia, when folate is in excess and insufficient B12 is present, which creates a 'folate trap'. Another symptom of B12 deficiency is dementia due to degradation of myelin. In B12 deficiency, methylmalonyl CoA will be metabolized to methylmalonic acid (MMA), which is why MMA is considered the definitive marker for B12 deficiency. Achlorhydria (insufficient stomach acid) can lead to B12 deficiency because HCl is required to cleave B12 from intrinsic factor.

FOOD SOURCES

Vitamin B12 is synthesized by bacteria and exists in all animal foods. Vitamin B12 is only available from animal sources. The B12 synthesized by gut bacteria may not be a significant source for humans, as it is not absorbed in the colon.

SUPPLEMENT OPTIONS

The RDA for B12 is 2.6 mcg/day. Consider the upper limit of folate supplementation as a factor for the supplementation of B12, due to potential folate trap. Vitamin B12 is extremely safe. No toxicity from high doses of vitamin B12 has ever been reported. Intramuscular injection are often used, particularly in the elderly to bypass intrinsic factor. Humans store large amounts of B12 in the liver so larger doses can be given at 6 month intervals. Supplementation is highly encouraged on a vegan diet. Due to high storage capacity in the liver, it may take years to deplete the body of B12 after adopting a vegan diet. Consider MTHFR genetic, and methyl cobalamin supplementation, particularly with hyperhomocysteinemia. Methylcobalamin is the recommended form of supplementation, but may be poorly absorbed in people taking antacids or those with very poor absorption (celiac, intestinal permeability, etc). Cyanocobalamin is not recommended for patients with MTHFR mutations. Hydroxocobalamin is recommended for patients with autoimmune diseases and elevated nitric oxide levels. Glutathione is also required for methylcobalamin to be bound for transport adequately. Vitamin B12 supplementation may help manage anemia, asthma, fatigue, hepatitis, dementia, epilepsy, depression, psychosis, irritability, tinnitus, numbness, tingling, neuropathy, AIDS, multiple sclerosis, ataxia, and infertility. Supplemental B12 is commonly given in 1000 to 5000 mcg doses.

NutriPro			⊕⊕ Homozygous Mutant	⊕⊖ Heterozygous	⊖⊖ Homozygous Wild			
Vitamins and Minerals	SNP ID	Your Mutation	Serum Current	Serum Previous	Serum Reference	Cellular Current	Cellular Previous	Cellular Reference
Vitamin B2 (Riboflavin 5-Phosphate)	rs1799983	⊕⊕T/T				0.1		0.2-3.6 (pg/MM WBC)

GENETIC MUTATIONS

rs1799983: rs1799983, located on the NOS3 gene is associated with a ~2x risk of developing pre-eclampsia (pregnancy-induced hypertension). Vitamin B2, is required for the correct functioning of NOS3 gene. The gene product regulates blood pressure which upon mutation, impairs normal functioning thereby increasing the risk for pre-eclampsia.Individuals with a homozygous mutant (abnormal) T allele carriers have an increased risk of preeclampsia and ischemic heart disease. Preclampsia can reduce the amount of nutrients that baby is getting hence healthy foods like wholegrains, fish, nuts, legumes, fruit, vegetables and dairy products are recommended. Vitamin B2 supplementation may prove beneficial for those with high blood pressure and individuals with cardiovascular risk.

PHYSIOLOGICAL FUNCTION

Two very important coenzymes involved in energy metabolism are derived from riboflavin to participate in oxidation/reduction reactions. Riboflavin is also essential for NOS enzyme (nitric oxide synthase) and glutathione reductase which regenerates glutathione, and which is very important for antioxidation/detoxification.

HOW IT GETS DEPLETED

Riboflavin is commonly depleted by excessive or chronic alcohol consumption. Need for riboflavin is increased in the elderly.

CLINICAL MANIFESTATIONS OF DEPLETION

Frank deficiency of riboflavin is rare, however, marginal deficiency is common. Deficiency of riboflavin is associated with fatigue/weakness.

FOOD SOURCES

Food sources high in riboflavin include: organ meats, dairy foods, eggs, leafy greens (spinach), broccoli, and liver. *Enriched grains include riboflavin

SUPPLEMENT OPTIONS

The RDA for riboflavin is 1.7 mg/day. Common levels of therapeutic intake of riboflavin are 25-50 mg/day. No UL for riboflavin has been set.

NutriPro			⊕⊕ Homozygous Mutant			⊕⊖ Heterozygous			⊖⊖ Homozygous Wild
Vitamins and Minerals	SNP ID	Your Mutation	Serum Current	Serum Previous	Serum Reference	Cellular Current	Cellular Previous	Cellular Reference	
Vitamin B3 (Nicotinic acid)		*No mutation tested*	64.8		2.6-36.1 (ng/mL)				

PHYSIOLOGICAL FUNCTION

Niacin is extensively involved in metabolic reduction reactions through NAD-NADPH pathways. Over 200 enzymes in the human body require niacin. Other important major functions of niacin include: fatty acid synthesis, ATP synthesis, DNA repair, lower cholesterol/LDL, aids in circulation.

HOW IT GETS DEPLETED

Synthesized from tryptophan and uses iron, B6 and riboflavin as cofactors; deficiencies of these companion nutrients may be underlying causes. Can be depleted by oral contraceptives and statin drugs.

CLINICAL MANIFESTATIONS OF DEPLETION

Symptoms of niacin deficiency include: vomiting, constipation, red tongue, headache, fatigue, and depression. Severe deficiency of niacin is called pellagra. Pellagra is commonly accompanied by the following 4Ds: dermatitis, diarrhea, dementia, death.

FOOD SOURCES

The most concentrated sources of niacin are in animal products (pork), peanuts/peanut butter, tofu, and eggs. Also consider food sources high in tryptophan. *Enriched grains provide supplemental niacin.

SUPPLEMENT OPTIONS

The RDA for niacin is 20 mg/day. The UL for niacin is 35 mg/day, but oral administration up to 6g per day has been used without side effects. Niacin is often recommended therapeutically for lipid management. Niacin has been shown to lower LDL cholesterol, lipoprotein(a), triglyceride, and fibrinogen levels, while raising HDL levels. Flushing can occur at high doses. Aspirin may help reduce flushing. Time release niacin or no-flush niacin is not recommended for therapeutic treatment. Monitor liver function carefully with high dose Niacin supplementation.

Vitamin B5 (Pantothenic acid)		*No mutation tested*	9.7		22.7-429.2 (mcg/L)				

PHYSIOLOGICAL FUNCTION

Vitamin B5 is part of the structural component of coenzyme A. It is also important for synthesis of red blood cells, sex hormones, adrenal hormones, and vitamin D. Another significant function of B5 is to work with carnitine and CoQ10 for fatty acid oxidation/metabolism.

HOW IT GETS DEPLETED

It is possible to block absorption of B5 in the intestines by taking high doses of supplemental biotin.

CLINICAL MANIFESTATIONS OF DEPLETION

Deficiency of B5 is very rare, however, in a diet that is high in biotin, or if high dose biotin supplementation occurs, B5 may become conditionally deficient due to competition for the same uptake receptor in the intestine.

FOOD SOURCES

Food sources of B5 include: beef, pork, chicken, fish, egg yolks, whole grains, legumes, lentils.

SUPPLEMENT OPTIONS

There is currently no RDA established for B5. The AI for B5 is 5 mg/day in adults, 6 mg/day during pregnancy, and 7 mg/day during lactation. Because breakdown of B5 is metabolically slow, and deficiency is rare, there is probably no need for supplementation.

NutriPro	⊕⊕ Homozygous Mutant	⊕⊖ Heterozygous	⊖⊖ Homozygous Wild

Vitamins and Minerals	SNP ID	Your Mutation	Serum Current	Serum Previous	Serum Reference	Cellular Current	Cellular Previous	Cellular Reference
Vitamin B9 (Folate) (L-5-methyl tetrahydrofolate)	rs1801131	⊕⊖C/A	3.2		≥4.6 (ng/mL)			
	rs1801133	⊕⊖C/T						

GENETIC MUTATIONS

rs1801131: This SNP found on the MTHFR gene encodes for an enzyme that converts diet derived into active form. Mutation in this gene results in reduced which is required for the metabolism of homocysteine to make proteins. Heterozygous (partially abnormal) individuals have possibly impaired folate metabolism. Folate deficiency as well as over-supplementation can lead to impaired folate metabolism. The goal of folate supplementation should be optimal physiological levels.

rs1801133: The SNP in the MTHFR gene, encodes an enzyme involved in vitamin B9 (folic acid) metabolism. Mutation in this gene results in reduced vitamin B9 which is required for the metabolism of an amino acid called homocysteine to make proteins. Thus, unavailability of vitamin B9 increases homocysteine levels. This is linked to early development of heart disease, blood pressure (hypertension), blood clots and pregnancy loss.Individuals with heterozygous (partially abnormal) genotype can process vitamin B9 with 65% efficiency resulting in high homocysteine levels. Individuals with T allele have difficulty to convert dietary folate into the active form of Vitamin B9. Such individuals need to take methylated folate or supplements in the active form in order to avoid deficiency.

PHYSIOLOGICAL FUNCTION

Folate, especially in the form of L-5-methyltetrahydrofolate, is essential for DNA synthesis and repair, methylation reactions, and amino acid metabolism. It plays a key role in cellular division and growth, particularly important during pregnancy and infancy. Folate is vital for the production of red and white blood cells in the bone marrow and for converting carbohydrates into energy.

HOW IT GETS DEPLETED

Folate deficiency can occur due to inadequate dietary intake, malabsorption disorders, increased demand (e.g., pregnancy), or use of certain medications like anticonvulsants and some anti-inflammatory drugs. Alcohol abuse can also impair folate absorption and metabolism.

CLINICAL MANIFESTATIONS OF DEPLETION

Folate deficiency can lead to megaloblastic anemia, characterized by large, immature red blood cells. In pregnant women, it increases the risk of neural tube defects in the fetus. Other issues include elevated homocysteine levels, which is a risk factor for cardiovascular disease, and potential neurological disorders such as depression and cognitive impairments.

FOOD SOURCES

Rich dietary sources of folate include leafy green vegetables, legumes, nuts, seeds, liver, and fortified cereals. Citrus fruits and juices also contain significant amounts of folate. The bioavailability of folate varies depending on the food source, with liver and synthetic forms (e.g., in supplements and fortified foods) being highly bioavailable.

SUPPLEMENT OPTIONS

Folate supplements are available in various forms, including folic acid and L-5-methyltetrahydrofolate. Supplementation is particularly recommended for women of childbearing age to prevent neural tube defects. The recommended dietary allowance varies by age, pregnancy, and lactation status. Excess intake of folic acid, particularly from supplements, can mask vitamin B12 deficiency symptoms.

Vitamin C (L-Ascorbic Acid)	rs6596473	⊕⊕G/G	*No nutrient risk*

GENETIC MUTATIONS

rs6596473: The SLC23A1 gene encodes SVCT1, which transports vitamin C (ascorbic acid) for absorption in the liver and kidneys. Mutation may cause loss of function which hinders the active transport of vitamin C to the tissues. Vitamin C deficiency may cause aggressive periodontitis leading to tooth loss. Homozygous mutant (abnormal) individuals have lower vitamin C levels. Affected individuals are advised to take vitamin C supplements and eat citrus fruits, potatoes, broccoli and strawberries.

NutriPro	⊕⊕ Homozygous Mutant	⊕⊖ Heterozygous	⊖⊖ Homozygous Wild

Vitamins and Minerals	SNP ID	Your Mutation	Serum Current	Serum Previous	Serum Reference	Cellular Current	Cellular Previous	Cellular Reference
Vitamin D, 1-25 dihydroxy	rs4588	⊕⊖ A/C	*No nutrient tested*					

GENETIC MUTATIONS

rs4588: VDR gene encodes the vitamin D receptor that binds to 1,25(OH)2D to form a regulatory factor that modulates the processing of genes in many tissues in the body. Mutation in the gene disrupts the normal functioning leading to deficiency.Heterozygous (partially abnormal) individuals have intermediate levels of 25(OH)D, and 1,25(OH)2D. Vitamin D rich foods are recommended to affected individuals. Vitamin D sources include egg yolks, cheese, beef liver, fatty fish like tuna, mackeral and salmon, fortified foods. Foods rich in calcium and phopshorus (animal proteins) may reduce the 1,25(OH)2D levels, hence they should be avoided.

Vitamin D, 25-OH	rs10741657	⊕⊕ G/G	*No nutrient risk*					
	rs12785878	⊕⊖ G/T						
	rs2282679	⊕⊖ C/A						

GENETIC MUTATIONS

rs10741657: This CYP2R1 gene codes for an enzyme that regulates vit D metabolism and its formation in the liver. The SNP causes loss of function which is associated with lower levels of vitamin D3.Individuals with homozygous mutant (abnormal) genotype tend to have lower vitamin D levels. Individuals with low vitamin D deficiency may be recommended supplements. Foods rich in vit D such as oily fish (salmon sardines, mackeral, and herring), red meat, liver, egg yolks, mushrooms are recommended to individuals who are prone to have vitamin D deficiency.

rs12785878: The NADSYN1 gene functions to regulate and coordinate the basic activities of cells and is linked to vitamin D serum concentrations. Mutation impairs the function leading to vitamin D deficiency.Heterozygous (partially abnormal) genotypes carry a slightly increased risk for vitamin D insufficiency. Vitamin D rich foods are recommended to affected individuals. Vitamin D sources include egg yolks, cheese, beef liver, fatty fish like tuna, mackeral and salmon, fortified foods.

rs2282679: The SNP on the GC gene encodes for a vitamin D binding protein. Vitamin D-binding protein (DBP), reversibly binds and transports vitamin D3 metabolites to different target organs, influencing its bioavailability. Mutation in the gene affects vitamin D3 levels.Heterozygous (partially abnormal) genotypes carry lower vitamin D3 levels. Food rich in vitamin D(oily fish[salmon sardines, mackerel, and herring], red meat, liver, egg yolks, mushrooms) is recommended in individuals with CC and AC genotypes.

Vitamin D3 (Cholecalciferol)	rs10877012	⊖⊖ G/G	*No nutrient risk*					

GENETIC MUTATIONS

rs10877012: CYP27B1 produces an enzyme which activates vitamin D in the body. The rs10877012 mutation impairs this mechanism, leading to low levels of vitamin D in the blood. Homozygous mutant (abnormal) individuals may have low concentrations of vitamin D3. Supplementation of vitamin D or consuming foods rich in vitamin D (oily fish[salmon, sardines, mackerel, herring], red meat, liver, egg yolks, mushrooms) may help to elevate vitamin D levels in susceptible individuals.

NutriPro			⊕⊕ Homozygous Mutant			⊕⊖ Heterozygous			⊖⊖ Homozygous Wild
Vitamins and Minerals	SNP ID	Your Mutation	Serum Current	Serum Previous	Serum Reference	Cellular Current	Cellular Previous	Cellular Reference	
Vitamin E (Alpha-tocopherol)		*No mutation risk*	7.0		7.4-30.6 (mg/L)				

PHYSIOLOGICAL FUNCTION

Vitamin E is an important antioxidant that reduces the formation of reactive oxygen species (ROS) that result from oxidative damage. Vitamin E also regulates cell signaling, influences immune function, and inhibits coagulation.

HOW IT GETS DEPLETED

Vitamin E may become depleted or deficient due to intestinal malabsorption. Smoking also depletes the body's vitamin E stores.

CLINICAL MANIFESTATIONS OF DEPLETION

Vitamin E deficiency may result in peripheral neuropathy, ataxia, muscle weakness, skeletal myopathy, retinopathy, and increased risk of CVD, red blood cell destruction, prostate cancer, and cataracts.

FOOD SOURCES

Dietary sources of vitamin E include: plant seeds (such as sunflower seeds), walnuts, hazelnuts, olive oil, canola oil, wheat germ oil, sunflower oil, safflower oil, tomato, avocado, spinach, and Swiss chard.

SUPPLEMENT OPTIONS

The RDA for vitamin E is 15mg/day. The UL for vitamin E is set at 1000 mg/day in order to prevent interference in vitamin K clotting pathways. The only supplementary form of vitamin E that reverses deficiency symptoms is α-tocopherol. In addition, α-Lipoic acid is an important co-factor that can aid in restoring vitamin E levels when depleted.

NutriPro	⊕⊕ Homozygous Mutant	⊕⊖ Heterozygous	⊖⊖ Homozygous Wild

Vitamins and Minerals	SNP ID	Your Mutation	Serum Current	Serum Previous	Serum Reference	Cellular Current	Cellular Previous	Cellular Reference
Vitamin K2 (Menaquinone-MK-7)		*No mutation tested*				0.03		0.1-0.89 (pg/MM WBC)

PHYSIOLOGICAL FUNCTION

Vitamin K is a group of fat-soluble vitamins. This group of vitamins includes two natural vitamins: vitamin K1 and vitamin K2. Vitamin K2 is the main storage form of Vitamin K in animals. It has several forms, referred to as menaquinones. The nomenclature denoting vitamin K2 types will include an 'MK' to specify this is a menaquinone and the number following this denotes how many isoprenyl units are on the side chain of the molecule. The most common forms are MK-4 and MK-7. Bacteria in the colon can convert K1 (from plant-based foods) into vitamin K2. Vitamin K2 is necessary to prevent arterial calcification, which it does by activating matrix GLA protein (MGP). This matrix GLA protein is present in blood vessels and inhibits soft tissue calcification. Matrix GLA protein needs to be carboxylated to work properly and Vitamin K2-MK7 plays a major role in this carboxylation.

HOW IT GETS DEPLETED

Dietary deficiency of vitamin K1 is extremely rare unless there has been significant damage to the intestinal lining, such as in inflammatory bowel disorders (Crohn's, ulcerative colitis, etc), liver disease, cystic fibrosis, and fat malabsorption disorders. In addition, the use of oral blood-thinning medications and some antibiotics can interfere with vitamin K. Individuals with chronic kidney disease are at risk for vitamin K deficiency. Individuals with ApoE4 genotype may be at greater risk for low vitamin K.

CLINICAL MANIFESTATIONS OF DEPLETION

Inadequate levels of both Vitamin K1 and K2 will radically increase risk for heart disease and stroke. Chronically low vitamin K levels can lead to uncontrolled bleeding and chronic marginally low vitamin K levels are correlated in some studies with osteoporosis. Because vitamin K2 also assists in calcium homeostasis, low or deficient levels of vitamin K2 can lead to unregulated calcium repletion from bone tissue sources in the presence of vitamin D3 supplementation. Supplementation of vitamin D2 does not tend to lead to this, however. It is recommended that vitamin K2 be supplemented when vitamin D3 is supplemented. Levels of K2 are inversely related to cardiovascular disease and coronary calcification.

FOOD SOURCES

The best sources of vitamin K2 include some fermented foods predominantly natto and some rare fermented cheeses, and liver. There are minor amounts present in egg yolk and butter.

SUPPLEMENT OPTIONS

Studies suggest daily therapeutic doses of about 360-500 micrograms (mcg) of vitamin K2. Fermented foods contain a wide variety of different bacteria, and only certain ones—such as Bacillus subtilis—actually make vitamin K2. Dietary vitamin K2 intake is enhanced with regular consumption of fermented foods. You can make fermented foods yourself, by using a starter culture specifically designed to optimize K2. Vitamin K2 supplements come in 'MK' varieties and MK-4 is what all forms of vitamin K2 are converted into. If one takes an MK-7 variety, the body will convert to MK-4, however, MK-4 supplements can be found commercially to bypass activation after absorption.

Zinc, Zn 67	rs11126936	⊕⊖ C/A	*No nutrient risk*

GENETIC MUTATIONS

rs11126936: The SLC30A3 gene encodes the ZnT3 protein which influences serum concentrations of zinc. Zinc plays a significant role in the maintenance of nerve impulses, preventing cell damage due to unstable molecules, and promotes healthy aging. Mutations may lead to neurobiological implications.The serum zinc cnoncentration in heterozygous (partially normal) individuals was maintained. Consumption of zinc rich foods such as baked beans, oysters, and fortified breakfast cereals could help prevent oxidative damage to DNA, proteins and lipids due to ageing.

NutriPro	⊕⊕ Homozygous Mutant	⊕⊖ Heterozygous	⊖⊖ Homozygous Wild

Amino Acids	SNP ID	Your Mutation	Serum Current	Serum Previous	Serum Reference	Cellular Current	Cellular Previous	Cellular Reference
Choline	rs3733890	⊕⊕ G/G	*No nutrient risk*					
	rs7946	⊕⊕ T/T						

GENETIC MUTATIONS

rs3733890: BHMT is a zinc-dependent enzyme which plays a role in DNA methylation, a process significant in regulating gene expression during mammalian development. It is associated with folic acid and choline metabolism as well as maternal nutritional factors important in mammalian developmental including a decreased risk of orofacial cleft in the embryo. Mutation may impair this function leading to choline and folic acid deficiency. BHMT is a zinc-dependent enzyme which plays a role in DNA methylation, a process significant in regulating gene expression during mammalian development. It is associated with folic acid and choline metabolism as well as maternal nutritional factors important in mammalian developmental including a decreased risk of orofacial cleft in the embryo. Mutation may impair this function leading to choline and folic acid deficiency.Homozygous mutant (abnormal) individuals have reduced levels of folic acid and choline. Folic acid and choline supplementation are recommended to individuals with susceptible genotypes. Folic acid and choline supplementation are recommended to individuals with susceptible genotypes.

rs7946: The PEMT codes for an enzyme that helps remove fat from the liver. Mutation in the gene inhibits the process due to lack of choline in the body and may sometimes lead to fatty liver in affected individuals. Choline aids in the fat removal from the liver which in turn protects against inflammation and anxiety. Choline deficiency increases sensitivity to carcinogens. Homozygous mutant (abnormal) individuals may likely carry the risk of acquiring a fatty liver due to choline deficiency. This genotype slows the export of fat from liver and develops fatty livers in individuals who overeat. Individuals with TT genotype may be recommended to include lentils, nuts, soy products, fish, beef, poultry, eggs, lean meats; beans, peas to increase choline

Glutathione Oxidized	rs1695	⊕⊖ A/G	*No nutrient risk*					

GENETIC MUTATIONS

rs1695: The GSTP1 gene produces an enzyme that decreases damage in cells by catalyzing the modification of toxic compounds to glutathione. The cells contain glutathione, an antioxidant that helps combat free radicals. Mutation decreases enzyme activity resulting in glutathione deficiency that can damage the cells.Heterozygous (partially normal) individuals exposed to tobacco smoke may carry asthma rsk. Glutathione can protect cells from free radicals and detoxify pollutants hence, foods rich in glutathione (mushrooms, avocados, spinach, okra) may help affected individuals.

MMA (Methymalonic Acid)	rs291466	⊕⊖ C/T	*No nutrient risk*					

GENETIC MUTATIONS

rs291466: This SNP on the HICBH gene accounts for variation in MMA levels which is a compound that reacts with vitamin B12 to produce a coenzyme essential for normal cellular function. Vitamin B12 (cobalamin) deficiency leads to increase in MMA. Heterozygous individuals (partially normal) individuals have lower levels of MMA. Affected individuals may be recommended to restrict intact protein and supplement with amino acid-based formula.

NutriPro			⊕⊕ Homozygous Mutant			⊕⊖ Heterozygous			⊖⊖ Homozygous Wild
Fatty Acids	SNP ID	Your Mutation	Serum Current	Serum Previous	Serum Reference	Cellular Current	Cellular Previous	Cellular Reference	
AA/EPA		*No mutation tested*				58.2		2.5-10.9	

PHYSIOLOGICAL FUNCTION

The AA/EPA Ratio, also known as the Arachidonic Acid to Eicosapentaenoic Acid Ratio, is a measure that reflects the balance between two essential fatty acids in the body: arachidonic acid (AA) and eicosapentaenoic acid (EPA). These fatty acids play pivotal roles in various physiological functions. AA is a precursor to pro-inflammatory eicosanoids, while EPA is a precursor to anti-inflammatory eicosanoids. Maintaining an appropriate ratio is crucial for inflammation regulation and overall health.

HOW IT GETS DEPLETED

The AA/EPA Ratio can be influenced by dietary choices and the intake of foods rich in these fatty acids. A diet high in omega-6 fatty acids, found in vegetable oils like soybean oil and corn oil, can increase AA levels. Conversely, a diet rich in omega-3 fatty acids, especially EPA, can elevate EPA levels and favorably impact the ratio. Certain health conditions and medications can also influence the ratio.

CLINICAL MANIFESTATIONS OF DEPLETION

An imbalance in the AA/EPA Ratio, characterized by excessive AA relative to EPA, is associated with chronic inflammation and an increased risk of inflammatory diseases such as cardiovascular disease, arthritis, and neurodegenerative conditions. Maintaining a balanced ratio, with sufficient EPA, can help reduce inflammation and support overall health.

FOOD SOURCES

Dietary sources of arachidonic acid (AA) include animal products like meat and dairy. Eicosapentaenoic acid (EPA) is primarily found in fatty fish such as salmon, mackerel, and sardines. Balancing the intake of foods rich in AA and EPA is essential for managing the AA/EPA Ratio.

SUPPLEMENT OPTIONS

Supplements containing EPA (omega-3 supplements) can be considered to help balance the AA/EPA Ratio, especially for individuals with an imbalanced diet or specific health conditions. It's important to consult with a healthcare provider or nutritionist to determine the appropriate EPA supplementation. Reducing the consumption of excessive omega-6-rich foods and increasing omega-3-rich foods can also help maintain a healthy ratio.

NutriPro	⊕⊕ Homozygous Mutant	⊕⊖ Heterozygous	⊖⊖ Homozygous Wild

Fatty Acids	SNP ID	Your Mutation	Serum Current	Serum Previous	Serum Reference	Cellular Current	Cellular Previous	Cellular Reference
Omega-3 Index		*No mutation tested*				6.33		8.0-12.65 (%)

PHYSIOLOGICAL FUNCTION

Omega-3 Index is the sum of EPA & DHA % as measured in whole blood, and derived by validated calculations to yield the equivalent sum of EPA % and DHA % in red blood cell membranes. Please note the value is a percentage, with the denominator being the sum of all Fatty Acids measured in the blood and thus the index can vary based on fatty acid composition of the diet. The index can be used as an indicator of risk for sudden cardiac death and nonfatal cardiovascular events and as a therapeutic target. It can also be used to assess adherence to omega-3 therapy and/or success or failure of such therapy. Optimal omega-3 index positively impacts heart rate, blood pressure, triglyceride levels, myocardial efficiency, inflammatory responses, and endothelial function while also improving cognitive function.

HOW IT GETS DEPLETED

The Omega-3 Index is a validated biomarker of tissue membrane omega-3 (n-3) polyunsaturated fatty acid (PUFA) status. The ratio is expressed as a percentage where the denominator is the sum off all fatty acids measured in the blood. Thus, a decrease in the ratio can be caused by a low intake of omega-3 fatty acids and incorporation of those fatty acids into cell membranes; or due to a proportionally high intake of other dietary fatty acids (saturated fatty acids, mono-unsaturated fatty acids and omega-6's poly unsaturated fatty acids)

CLINICAL MANIFESTATIONS OF DEPLETION

Low levels of omega-3 index are associated with increased risk for cardiac death.

FOOD SOURCES

If omega-3 index is <8.0% it is advised to increase dietary sources of omega-3's (EPA and DHA) from both plant and animal sources. Because the omega-3 index is a relative ratio of omega-3 compared to all other fatty acids in the blood, it is also important to evaluate intake of all other dietary fatty acids (saturated fatty acids, mono-unsaturated fatty acids and omega-6's poly unsaturated fatty acids)

SUPPLEMENT OPTIONS

Currently, no official dietary intake recommendations have been established. Several official health organizations have proposed a minimum dietary intake level of 500 mg/day of EPA+DHA. Because the efficiency of conversion of ALA to DHA is low, supplementing DHA is generally recommended to meet therapeutic doses. The recommended minimum level of DHA supplementation in adults is 250 mg per day. Pregnant and lactating women are recommended to consume at least 200 mg DHA per day. Diabetic individuals may benefit from supplementing DHA (along with EPA) due to its triglyceride-lowering effects. High dose supplementation of omega-3 fatty acids (including DHA) has been shown to reduce the need for non-steroidal anti-inflammatory drugs (NSAIDs). Persons suffering from ulcerative colitis have been shown to need fewer corticosteroids when supplementing with high dose omega-3 fatty acids. Adverse side effects observed with high dose omega-3 fatty acids from supplement form include gastrointestinal upset and loose stools. Omega-3 supplements including EPA and DHA should be used with caution in persons with clotting disorders or on anti-clotting medication.

Blood Cell Count	Current	Previous	Result	Reference
WBC (x 10^3/µL)	8.50		1.01 — 8.99 — 30	9.0-30.0

NutriPro

NutriPro			⊕⊕ Homozygous Mutant		⊕⊖ Heterozygous		⊖⊖ Homozygous Wild	
Vitamins and Minerals	SNP ID	Your Mutation	Current	Serum Previous	Reference	Current	Cellular Previous	Reference
Calcium, Ca 44	rs4516035	⊕⊕T/T	9.8		8.9-10.6 (mg/dL)	43		15.0-120.0 (ng/MM WBC)
Chromium, Cr 53	*No mutation tested*		0.06		0.1-0.7 (ng/mL)			
Copper to Zinc Ratio	*No mutation tested*		1.2		0.9-2.6			
Copper, Cu 63	rs76151636	⊖⊖G/G	0.7		0.6-1.8 (mcg/mL)	14		2.0-15.0 (ng/MM WBC)
Fluoride	rs4284505	⊕⊖A/G	*No nutrient tested*					
Iodine	rs225014	⊕⊖C/T	*No nutrient tested*					
Iron, Fe 56	rs1799945	⊖⊖C/C	100		37.0-145.0 (ug/dL)	99.4		88.9-117.0 (mg/dL)
	rs1800562	⊖⊖G/G						
	rs3811647	⊕⊕G/G						
	rs4820268	⊖⊖A/A						
	rs855791	⊕⊕C/C						
Magnesium, Mg 24	rs4680	⊕⊖A/G	2.2		1.6-2.6 (mg/dL)	4.6		3.6-7.7 (mg/dL)
Manganese, Mn 55	rs13107325	⊖⊖C/C	0.5		0.3-2.0 (ng/mL)	23		2.0-75.0 (pg/MM WBC)
Molybdenum	rs594445	⊕⊕C/C	*No nutrient tested*					
Myo-Inositol	*No mutation tested*		15.1		20.5-60.7 (nmol/mL)	0.13		0.1-2.5 (ng/MM WBC)
Phosphorus	rs4074995	⊕⊖G/A	*No nutrient tested*					
Potassium	rs4343	⊕⊖A/G	4.7		3.5-5.5 (mmol/L)			
Selenium, Se 76	rs1050450	⊕⊕T/T	115.6		109.8-218.4 (ng/mL)	592		234.0-1050.0 (pg/MM WBC)
	rs3877899	⊖⊖G/G						
Sodium	rs2304478	⊕⊕G/G	139		136.0-145.0 (mmol/L)			
	rs7204044	⊕⊖A/G						
Tetrahydrobiopterin	rs5030853	⊖⊖G/G	*No nutrient tested*					
	rs8007267	⊖⊖C/C						

Patient Name: **DEMO DEMO**
Date of Birth: **01-01-1111** Accession ID: **2311080002**
Service Date: **2023-11-28 11:49 (PDT)**

NutriPro

NutriPro			⊕⊕ Homozygous Mutant			⊕⊖ Heterozygous			⊖⊖ Homozygous Wild
Vitamins and Minerals	SNP ID	Your Mutation	Current	Serum Previous	Reference	Current	Cellular Previous	Reference
Vitamin A (All-Trans-Retinol)	rs11645428	⊕⊖ A/G	72.3		40.8-154.5 (mcg/dL)	10.2		0.9-17.3 (pg/MM WBC)
	rs12934922	⊖⊖ A/A						
	rs1667255	⊖⊖ A/A						
	rs6564851	⊕⊖ G/T						
	rs7501331	⊕⊖ C/T						
Vitamin A (Beta-Carotene)	rs11645428	⊕⊖ A/G	*No nutrient tested*					
Vitamin B1 (Thiamine diphosphate)	rs17514104	⊕⊕ T/T	16.4		1.4-71.3 (nmol/L)	0.06		0.1-7.0 (pg/MM WBC)
Vitamin B12 (Cyanocobalamin)	rs492602	⊖⊖ T/T	>2000		232.0-1245.0 (pg/mL)	5.78		2.0-11.99
	rs526934	⊖⊖ A/A						
	rs602662	⊕⊕ G/G						
Vitamin B2 (Riboflavin 5-Phosphate)	rs1799983	⊕⊕ T/T	18.0		5.6-126.1 (mcg/L)	0.1		0.2-3.6 (pg/MM WBC)
	rs778479139	⊖⊖ G/G						
Vitamin B3 (Nicotinic acid)	*No mutation tested*		64.8		2.6-36.1 (ng/mL)	69.7		39.6-303.5 (pg/MM WBC)
Vitamin B5 (Pantothenic acid)	*No mutation tested*		9.7		22.7-429.2 (mcg/L)	18.5		2.5-32.8 (pg/MM WBC)
Vitamin B6, Pyridoxal 5-Phosphate	*No mutation tested*		65.1		2.8-76.2 (ng/mL)	5.5		0.5-9.7 (pg/MM WBC)
Vitamin B7 (Biotin)	rs13078881	⊕⊕ G/G	*No nutrient tested*					
Vitamin B9 (Folate) (L-5-methyl tetrahydrofolate)	rs1801131	⊕⊖ C/A	3.2		≥4.6 (ng/mL)			
	rs1801133	⊕⊖ C/T						
Vitamin C (L-Ascorbic Acid)	rs33972313	⊕⊕ G/G	0.2		0.2-1.1 (mg/dL)	3.9		0.5-9.7 (ng/MM WBC)
	rs4257763	⊖⊖ A/A						
	rs6139591	⊖⊖ C/C						
	rs6596473	⊕⊕ G/G						
Vitamin D, 1-25 dihydroxy	rs4588	⊕⊖ A/C	*No nutrient tested*					

Patient Name: DEMO DEMO
Date of Birth: 01-01-1111 Accession ID: 2311080002
Service Date: 2023-11-28 11:49 (PDT)

NutriPro

NutriPro			⊕⊕ Homozygous Mutant		⊕⊖ Heterozygous	⊖⊖ Homozygous Wild		
Vitamins and Minerals	**SNP ID**	**Your Mutation**	**Current**	**Serum Previous**	**Reference**	**Current**	**Cellular Previous**	**Reference**
Vitamin D, 25-OH	rs10741657	⊕⊕ G/G	39.1		30.0-108.0 (ng/mL)			
	rs12785878	⊕⊖ G/T						
	rs2282679	⊕⊖ C/A						
Vitamin D2 (Ergocalciferol)	rs10766197	⊖⊖ A/A	*No nutrient tested*					
Vitamin D3 (Cholecalciferol)	rs10877012	⊖⊖ G/G	0.7		0.4-1.8 (ng/mL)	104.1		25.9-246.6 (pg/MM WBC)
Vitamin E (Alpha-tocopherol)	rs12272004	⊕⊕ C/C	7.0		7.4-30.6 (mg/L)	212.8		18.4-1031.1 (pg/MM WBC)
Vitamin K1 (Phylloquinone)	rs2108622	⊖⊖ C/C	1.14		0.1-8.1 (ng/mL)	0.42		0.1-0.71 (pg/MM WBC)
Vitamin K2 (Menaquinone-MK-7)	*No mutation tested*		0.99		0.1-5.19 (ng/mL)	0.03		0.1-0.89 (pg/MM WBC)
Zinc, Zn 67	rs11126936	⊕⊖ C/A	0.6		0.5-1.0 (mcg/mL)	8		4.0-15.0 (ng/MM WBC)
Amino Acids	**SNP ID**	**Your Mutation**	**Current**	**Serum Previous**	**Reference**	**Current**	**Cellular Previous**	**Reference**
Choline	rs3733890	⊕⊕ G/G	22.9		6.8-31.0 (nmol/mL)	0.6		0.2-1.5 (ng/MM WBC)
	rs7946	⊕⊕ T/T						
Coenzyme Q10 (Ubiquinone + Ubiquinol), Total	rs775607037	⊖⊖ C/C	1.09		0.56-2.78 (µg/mL)	57.4		39.6-225.3 (pg/MM WBC)
	rs786204770	⊖⊖ A/A						
Free Carnitine	*No mutation tested*		28.5		11.6-43.4 (nmol/mL)	0.6		0.3-1.5 (ng/MM WBC)
Glutathione Oxidized	rs121909307	⊕⊕ G/G				339.4		98.7-1163.0 (pg/MM WBC)
	rs1695	⊕⊖ A/G						
L-Arginine	*No mutation tested*		84.8		81.6-249.0 (nmol/mL)			
L-Asparagine	*No mutation tested*		61.9		39.2-89.8 (nmol/mL)	1.0		0.5-2.8 (ng/MM WBC)
L-Citrulline	*No mutation tested*		43.0		18.7-47.5 (nmol/mL)			
L-Cysteine	*No mutation tested*		16.3		3.4-37.0 (nmol/mL)	226.0		60.0-565.0 (pg/MM WBC)
L-Glutamine	*No mutation tested*		502.2		393.5-699.3 (nmol/mL)	4.2		1.4-7.0 (ng/MM WBC)
L-Isoleucine	*No mutation tested*		42.2		25.5-158.9 (nmol/mL)			

Patient Name: DEMO DEMO
Date of Birth: 01-01-1111 Accession ID: 2311080002
Service Date: 2023-11-28 11:49 (PDT)

NutriPro

NutriPro

⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild

Amino Acids	SNP ID	Your Mutation	Current	Serum Previous	Reference	Current	Cellular Previous	Reference
L-Leucine		*No mutation tested*	116.8		101.2-249.3 (nmol/mL)			
L-Serine		*No mutation tested*	146.5		94.2-246.8 (nmol/mL)	3.3		1.8-19.8 (ng/MM WBC)
L-Valine		*No mutation tested*	255.1		155.9-368.0 (nmol/mL)			
MMA (Methymalonic Acid)	rs121918252	⊕⊕ G/G	0.20		0.1-0.5 (nmol/mL)			
	rs291466	⊕⊖ C/T						
Phenylalanine	rs5030853	⊖⊖ G/G	*No nutrient tested*					

Fatty Acids	SNP ID	Your Mutation	Current	Serum Previous	Reference	Current	Cellular Previous	Reference
AA (Arachidonic Acid)		*No mutation tested*				18.05		5.5-19.01 (%)
AA/EPA		*No mutation tested*				58.2		2.5-10.9
DHA (Docosahexaenoic Acid)		*No mutation tested*				6.02		2.42-10.52 (%)
DPA (Docosapentaenoic Acid)		*No mutation tested*				0.92		0.45-1.8 (%)
EPA (Eicosapentaenoic Acid)		*No mutation tested*				0.31		0.15-2.26 (%)
LA (Linoleic Acid)		*No mutation tested*				6.87		3.22-10.49 (%)
Omega-3 Index		*No mutation tested*				6.33		8.0-12.65 (%)
Omega-3 Total		*No mutation tested*				7.50		3.25-13.99 (%)
Omega-6 Total		*No mutation tested*				29.34		11.03-34.96 (%)

Blood Cell Count	Current	Previous	Result	Reference
Lymphocyte Count (x 10^3/µL)	2.88		0 0.69 7.3	0.7-7.3
Neutrophil Count (x 10^3/µL)	4.72		0 1.64 6.75	1.65-6.75
WBC (x 10^3/µL)	8.50		1.01 8.99 30	9.0-30.0

Risk and Limitations

This test has been developed and its performance characteristics determined and validated by Vibrant Genomics LLC., a CLIA certified lab. These assays have not been cleared or approved by the U.S. Food and Drug Administration. Vibrant Wellness provides additional contextual information on these tests and provides the report in a more descriptive fashion.

The Vibrant CardiaX Genetics panel does not demonstrate absolute positive and negative predictive values for any condition. Its clinical utility has not been fully established. Clinical history and current symptoms of the individual must be considered by the healthcare provider prior to any interventions. Test results should be used as one component of a physician's clinical assessment.

CardiaX Genetics testing is performed at Vibrant Genomics, a CLIA certified laboratory. Vibrant Genomics has effective procedures in place to protect against technical and operational problems. However, such problems may still occur. Examples include failure to obtain the result for a specific test due to circumstances beyond Vibrant's control. Vibrant may re-test a sample to obtain these results but upon re-testing the results may still not be obtained. As with all medical laboratory testing, there is a small chance that the laboratory could report incorrect results. A tested individual may wish to pursue further testing to verify any results.

Genetic testing is helpful in analyzing the risk of various diseases. However, it is important to note that Genetic risk determinants are neither necessary nor sufficient for the development of diseases. Environmental and lifestyle risk factors could also affect the risk of disease development. Results from genetic analysis should always be interpreted along with clinical findings on the individual. Genetic testing evaluates only for the genotypes indicated; it does not test for other genetic abnormalities found elsewhere in the genome. Different genetic variants can be tested by different genetic labs to evaluate the risk for a particular disease, depending on what is tested, genetic risk may not be comparable between labs. It should be realized that there are possible sources of error like any lab testing which include sample misidentification, trace contamination of PCR reactions, technical errors and rare genetic variants that may interfere with analysis.

Some individuals may feel anxious about getting their genetic test health results. If the potential user feels very anxious, such user should speak to his or her doctor or other health care professional prior to collection of a sample for testing. Users should consult with their doctor or other health care professional if they have any questions or concerns about the results of their test or their current state of health. Users of the test are also encouraged to discuss their test results with a genetic counselor, board-certified clinical molecular geneticist, or equivalent health care professional.

The information in this report is intended for educational purposes only. While every attempt has been made to provide current and accurate information, neither the author nor the publisher can be held accountable for any errors or omissions. Tested individuals may find their experience is not consistent with Vibrant's selected peer reviewed scientific research findings of relative improvement for study groups. The science in this area is still developing and many personal health factors affect diet and health. Since subjects in the scientific studies referenced in this report may have had personal health and other factors different from those of tested individuals, results from these studies may not be representative of the results experienced by tested individuals. Further, some recommendations may or may not be attainable, depending on the tested individual's physical ability or other personal health factors. A limitation of this testing is that many of these scientific studies may have been performed in selected populations only. The interpretations and recommendations are done in the context of these studies, but the results may or may not be relevant to tested individuals of different or mixed ethnicities.

Vibrant Wellness makes no claims as to the diagnostic or therapeutic use of its tests or other informational materials. Vibrant Wellness reports and other information do not constitute medical advice and are not a substitute for professional medical advice. Please consult your healthcare practitioner for questions regarding test results, or before beginning any course of medication, supplementation, or dietary changes.