DEMO DEMO

FINAL REPORT

Accession ID: 2592152459

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Date of Birth: 03-24-1962
Biological Sex: Male
Age: 63
Height:
Weight:
Fasting: UNKNOWN

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Provider Name: DEMO CLIENT, MD
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Report Information

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

Specimen Information

Sample Type	Collection Time	Received Time	Report	Final Report Date
EDTA	2024-04-22 00:00 (PST)	2024-04-23 13:05 (PST)	Nutrients - P	2024-10-08 23:46 (PST)

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Nutrient Genetics

INTRODUCTION

Vibrant Wellness is pleased to present "Nutrient Genetics" 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 Nutrient Genetics Panel is a test to measure various genetic variants present in an individual's body to gauge the scope of predispositions an individual might have towards achieving optimum nutrition. This panel is designed to give a complete picture of the predispositions that could lead to various nutrient deficiencies or toxicities. 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.

The Nutrient Genetics report starts with a summary page which contains the summary page that lists the set of analytes with risk associated variants. Following this section is the complete list of the genetic markers measured in the panel. Elevated risk associated variants are indicated with red, partially elevated risk associated variants are indicated with yellow and alleles with no risk are indicated with green. All contents provided in the report are purely for informational purposes only and should not be considered medical advice. Any changes based on the information provided should be made in consultation with the clinical provider. The Vibrant Wellness platform provides tools for you to track and analyze your general wellness profile. Testing for the Nutrient Genetics panel is performed by Vibrant Genomics, a CLIA certified lab CLIA#: 05D2098445.

Methodology:

The Vibrant Nutrient 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.

Interpretation of Report:

Vibrant 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.

All laboratory testing is performed by CLIA-certified and CAP-accredited clinical laboratories upon the order of a licensed healthcare professional, using biological specimens obtained from patients by, or at the direction of, the ordering healthcare professional. This test has not been reviewed or approved by the U.S. Food and Drug Administration (FDA). The test is a laboratory-developed test (LDT) that has been designed, manufactured, and validated by a CLIA-certified and CAP-accredited clinical laboratory, and is performed in accordance with applicable federal and state laboratory regulations. While certain individual analytes within this test may be measured using FDA-cleared or FDA-approved assays.

Please note:

Consider all supplements in relation to medical history and symptoms. Not all recommended supplements are appropriate in all individual cases. It is important that you discuss any modifications to your diet, exercise, and nutritional supplementation with your healthcare provider before making any changes. Pediatric ranges have not been established for these tests. Pediatric ranges have not been established for this test. It is important that you discuss any modifications to your diet, exercise, and nutritional supplementation with your healthcare provider before making any changes.

Nutrient Genetics - Summary

Vitamins

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs11645428	BCMO1	Impaired beta carotene conversion	⊕⊕G/G	Elevated	A/A

Associated nutrient: **Vitamin A (All-Trans-Retinol)**. The BCMO1 gene encodes the enzyme beta-carotene 15,15'-dioxygenase. This enzyme converts the most abundant provitamin A carotenoid in the diet, beta-carotene, to retinol. The active form of vitamin A, retinol, is required by photoreceptors in the eye, stem cells, immune cells, red blood cells, and embryonic cells. It also affects the expression of the major extracellular matrix constituents, including collagen, laminin, entactin, fibronectin, elastin, and proteoglycans. The rs6564851 mutation inhibits the gene's expression, resulting in lower conversion of beta-carotene to retinol.Individuals with the GG genotype exhibit poor conversion efficiency and may have a reduced ability to convert beta-carotene to retinol.Susceptible individuals might benefit from a diet rich in retinol, the active form of vitamin A which be readily used by the body. Animal-sourced foods, such as oily fish, liver, cheese, butter, cheese, eggs, oily fish, fortified low-fat spreads, milk, and yogurt can all be included in the diet.

rs1667255	TTR	Impaired transportation of Vitamin A	⊕⊕A/A	Elevated	C/C

Associated nutrient: **Vitamin A (All-Trans-Retinol)**. Transthyretin is a protein that is produced by the TTR gene. The liver is the primary site of transthyretin production. This protein is produced in small amounts in the brain's choroid plexus and the light-sensitive tissue that lines the back of the eye (the retina). This protein is in charge of transporting vitamin A (retinol) throughout plasma and cerebrospinal fluid. Inside cells, vitamin A participates in the biosynthesis of various proteins, including those involved in the regulation of development and cell functioning or determining cell sensitivity to hormones and hormone-like factors. The mutation lowers protein levels and reduces gene expression, resulting in impaired retinol transport to various cells of the body. As a result, mutations cause low serum retinol levels and vitamin A deficiency.Individuals with the CC genotype have normal vitamin A transport, have vitamin A adequacy.Susceptible individuals might benefit from a diet rich in vitamin A. Animal-sourced foods, such as oily fish, liver, cheese, butter, cheese, eggs, oily fish, fortified low-fat spreads, milk, and yogurt can all be included in the diet. The diet can also include dietary supplements of the vitamin A precursors, beta-carotene, lutein, and zeaxanthin.

rs6564851	BCMO1	Impaired beta carotene conversion	⊕⊖G/T	Elevated	T/T

Associated nutrient: **Vitamin A (All-Trans-Retinol)**. The BCMO1 gene encodes the enzyme beta-carotene 15,15'-dioxygenase. This enzyme converts the most abundant provitamin A carotenoid in the diet, beta-carotene, to retinol. The active form of vitamin A, retinol, is required by photoreceptors in the eye, stem cells, immune cells, red blood cells, and embryonic cells. It also affects the expression of the major extracellular matrix constituents, including collagen, laminin, entactin, fibronectin, elastin, and proteoglycans. The rs6564851 mutation inhibits the gene's expression, resulting in lower conversion of beta-carotene to retinol.Individuals with the GT genotype exhibit poor conversion efficiency and may have a reduced ability to convert beta-carotene to retinol.Susceptible individuals might benefit from a diet rich in retinol, the active form of vitamin A which be readily used by the body. Animal-sourced foods, such as oily fish, liver, cheese, butter, cheese, eggs, oily fish, fortified low-fat spreads, milk, and yogurt can all be included in the diet.

rs7501331	BCO1	Impaired beta carotene conversion	⊕⊕T/T	Elevated	C/C

Associated nutrient: **Vitamin A (All-Trans-Retinol)**. The BCO1 gene encodes for an enzyme beta-carotene oxygenase 1. This enzyme converts the most abundant provitamin A carotenoid in the diet, beta-carotene, to retinol. The active form of vitamin A, retinol, is required by photoreceptors in the eye, stem cells, immune cells, red blood cells, and embryonic cells. It also affects the expression of the major extracellular matrix constituents, including collagen, laminin, entactin, fibronectin, elastin, and proteoglycans. The rs12934922 mutation inhibits the gene's expression, resulting in lower conversion of beta-carotene to retinol.Individuals with the TT genotype exhibit poor conversion efficiency and may have a reduced ability to convert beta-carotene to retinol.Susceptible individuals might benefit from a diet rich in retinol, the active form of vitamin A which be readily used by the body. Animal-sourced foods, such as oily fish, liver, cheese, butter, cheese, eggs, oily fish, fortified low-fat spreads, milk, and yogurt can all be included in the diet.

Nutrient Genetics - Summary

Vitamins

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs526934	TCN1	Impaired cellular uptake of vitamin B12	⊕⊕G/G	Elevated	A/A

Associated nutrient: **Vitamin B12 (Cyanocobalamin)**. The TCN1 gene encodes the glycoprotein transcobalamin I, which is made by salivary glands in the mouth. The glycoprotein is a vitamin B12-binding protein that is responsible for vitamin B12 transport in the body. Intracellularly, vitamin B12 is very important for DNA synthesis. It also functions as a cofactor for two enzymes, methionine synthase, and L-methylmalonyl-CoA mutase, which are involved in methionine and propionate metabolism, respectively. The mutation in the gene lower gene expression and reduces cellular uptake of vitamin B12. This lowers vitamin B12 in the cells and gives rise to vitamin B12 deficiency irrespective of its prevalence in serum. This can affect the above-mentioned cellular functions.Individuals with the GG genotype have have impaired cellular uptake of vitamin B12, have a vitamin B12 deficiency.Susceptible individuals might benefit from a diet rich in vitamin B12. Fish, meat, poultry, eggs, dairy products, salmon, and milk, can all be included in the diet. The diet can also include vitamin B12 dietary supplements.

rs602662	FUT2	Impaired cellular uptake of vitamin B12	⊕⊕G/G	Elevated	A/A

Associated nutrient: **Vitamin B12 (Cyanocobalamin)**. Galactoside 2-alpha-L-fucosyltransferase 2 is an enzyme that is encoded by the FUT2 gene in humans. The enzyme is involved in the attachment of the bacterium Helicobacter pylori to the gastric mucosa, which inhibits vitamin B12 absorption. A mutation in the FUT2 gene results in decreased gene expression and enzyme production. Thus, mutations ensure vitamin B12 absorption, which is required for normal brain and nervous system function, as well as the formation of red blood cells.Individuals with the AA genotype have impaired cellular uptake of vitamin 12, have a vitamin 12 deficiency.Susceptible individuals might benefit from a diet rich in vitamin B12. Fish, meat, poultry, eggs, dairy products, salmon, and milk, can all be included in the diet. The diet can also include vitamin B12 dietary supplements.

rs1799983	NOS3	Impaired NOS3 gene function	⊕⊕T/T	Elevated	G/G

Associated nutrient: **Vitamin B2 (Riboflavin 5-Phosphate)**. The NOS3 gene encodes for endothelial nitric oxide synthase (eNOS), an enzyme that facilitates the production of Nitric Oxide (NO). Vitamin B2 is required for the appropriate functioning of the NOS3 gene. The gene product regulates blood pressure which upon mutation, impairs normal functioning thereby increasing the risk for pre-eclampsia (high blood pressure that can occur during pregnancy).Individuals with a homozygous mutant (abnormal) individuals have altered gene function (mediated by vitamin B2) and have an increased risk of preeclampsia.Susceptible individuals might benefit from a diet rich in vitamin B2. Eggs, organ meats (such as kidneys and liver), lean meats, low-fat milk, mushrooms, spinach, fortified cereals, bread, and grain products can all be included in the diet. The diet can also include dietary vitamin B2 supplements.

rs13078881	BTD	Affected activity of the enzyme that is responsible for the reuse and recycle of biotin.	⊕⊕C/C	Elevated	G/G

Associated nutrient: **Vitamin B7 (Biotin)**. The BTD gene produces an enzyme called biotinidase that recycles vitamin B7 (biotin). Biotinidase separates biotin which is attached to proteins in food, allowing vitamin B7 to break down fats, proteins, and carbohydrates. Vitamin B7 promotes the appropriate function of the nervous system and is essential for liver metabolism as well. The mutation causes decreased gene expression and biotinidase deficiency wherein the body is unable to reuse and recycle vitamin B7. Thus, it leads to a deficiency of vitamin B7.Individuals with the CC genotype have decreased gene expression have a vitamin B7 deficiency.Susceptible individuals might benefit from a diet rich in vitamin B7. Beef liver, Eggs (cooked), Salmon, Avocados, Pork, Sweet potato, Nuts, and seeds can all be included in the diet. The diet can also include dietary vitamin B7 supplements.

Nutrient Genetics - Summary

Vitamins

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs1801131	MTHFR	Impaired methylation which is associated with the conversion of inactive to active folate	⊕⊕C/C	Elevated	A/A

Associated nutrient: **Vitamin B9 (Folate) (L-5-methyl tetrahydrofolate)**. The MTHFR gene produces an enzyme methylenetetrahydrofolate reductase. This enzyme converts the inactive folate (5,10 methylenetetrahydrofolate) to the active folate (5-methyltetrahydrofolate). Folate also known as vitamin B9 converts the amino acid, homocysteine to methionine, which is used to make proteins and other important compounds. It also helps to form DNA and RNA and is involved in protein metabolism. The mutation reduces gene expression, resulting in lower conversion efficiency, leading to the reduced ability to convert inactive folate to active folate. This gives rise to vitamin B9 deficiency which affects vitamin B9-associated functions in the body.Individuals with the CC genotype have lower conversion efficiency, cannot convert inactive folate to active folate and have a vitamin B9 deficiency.Susceptible individuals might benefit from a diet rich in folate. Dark green leafy vegetables (turnip greens, spinach, romaine lettuce, asparagus, brussels sprouts, broccoli), beans, peanuts, sunflower seeds, fresh fruits, fruit juices, whole grains, liver, seafood, eggs can all be included in the diet. Individuals can be supplemented with the methylated forms of folate and cobalamin supplements.

rs1801133	MTHFR	Impaired methylation which is associated with the conversion of inactive to active folate	⊕⊕T/T	Elevated	C/C

Associated nutrient: **Vitamin B9 (Folate) (L-5-methyl tetrahydrofolate)**. The MTHFR gene produces an enzyme methylenetetrahydrofolate reductase. This enzyme converts the inactive folate (5,10 methylenetetrahydrofolate) to the active folate (5-methyltetrahydrofolate). Folate also known as vitamin B9 converts the amino acid, homocysteine to methionine, which is used to make proteins and other important compounds. It also helps to form DNA and RNA and is involved in protein metabolism. The mutation reduces gene expression, resulting in lower conversion efficiency, leading to the reduced ability to convert inactive folate to active folate. This gives rise to vitamin B9 deficiency which affects vitamin B9-associated functions in the body.Individuals with the TT genotype have lower conversion efficiency, cannot convert inactive folate to active folate and have a vitamin B9 deficiency.Susceptible individuals might benefit from a diet rich in folate. Dark green leafy vegetables (turnip greens, spinach, romaine lettuce, asparagus, brussels sprouts, broccoli), beans, peanuts, sunflower seeds, fresh fruits, fruit juices, whole grains, liver, seafood, eggs can all be included in the diet. Individuals can be supplemented with the methylated forms of folate and cobalamin supplements.

rs33972313	SLC23A1	Impaired cellular uptake of vitamin C	⊕⊕A/A	Elevated	G/G

Associated nutrient: **Vitamin C (L-Ascorbic Acid)**. Solute carrier family 23 member 1 is a protein that in humans is encoded by the SLC23A1 gene. The encoded protein is involved in vitamin C transport across the cell uptake. Vitamin C serves as a reducing agent in cells (an electron donor). It functions as an antioxidant by donating its two highly energetic electrons to scavenge free radicals. In addition to helping with the biosynthesis of collagen, L-carnitine, and a few neurotransmitters. Vitamin C is also involved in protein metabolism. Mutations may cause loss of gene function which hinders vitamin C from being actively transported into cells. This can give rise to vitamin C deficiency in the cells irrespective of its prevalence in serum. Thus, an affect the above-mentioned cellular functionsIndividuals with the AA genotype have impaired cellular uptake of vitamin C, have a vitamin C deficiency.Susceptible individuals might benefit from a diet rich in vitamin C. Citrus fruits such as oranges, kiwi, lemon, and grapefruit, and cruciferous vegetables such as broccoli, Brussels sprouts, cabbage, and cauliflower can all be included in the diet. The diet can also include vitamin C dietary supplements.

Nutrient Genetics - Summary

Vitamins

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs4257763	SLC23A1	Impaired cellular uptake of vitamin C	⊕⊕G/G	Elevated	A/G, A/A

Associated nutrient: **Vitamin C (L-Ascorbic Acid)**. Solute carrier family 23 member 1 is a protein that in humans is encoded by the SLC23A1 gene. The encoded protein is involved in vitamin C transport across cell membrane. Vitamin C serves as a reducing agent in cells (an electron donor). It functions as an antioxidant by donating its two highly energetic electrons to scavenge free radicals. In addition to helping with the biosynthesis of collagen, L-carnitine, and a few neurotransmitters. Vitamin C is also involved in protein metabolism. Mutations may cause loss of gene function which hinders vitamin C from being actively transported into cells. This can give rise to vitamin C deficiency in the cells irrespective of its prevalence in serum. Thus, an affect the above-mentioned cellular functionsIndividuals with the GG genotype have impaired cellular uptake of vitamin C, have a vitamin C deficiency.Susceptible individuals might benefit from a diet rich in vitamin C. Citrus fruits such as oranges, kiwi, lemon, and grapefruit, and cruciferous vegetables such as broccoli, Brussels sprouts, cabbage, and cauliflower can all be included in the diet. The diet can also include vitamin C dietary supplements.

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs6596473	SLC23A1	Impaired cellular uptake of vitamin C	⊕⊕C/C	Elevated	G/G

Associated nutrient: **Vitamin C (L-Ascorbic Acid)**. Solute carrier family 23 member 1 is a protein that in humans is encoded by the SLC23A1 gene. The encoded protein is involved in vitamin C transport across the cell membrane. Vitamin C serves as a reducing agent in cells (an electron donor). It functions as an antioxidant by donating its two highly energetic electrons to scavenge free radicals. In addition to helping with the biosynthesis of collagen, L-carnitine, and a few neurotransmitters, vitamin C is also involved in protein metabolism. Mutations may cause loss of gene function which hinders vitamin C from being actively transported into cells. This can give rise to vitamin C deficiency in the cells irrespective of its prevalence in serum. Thus, affects the above-mentioned cellular functions.Individuals with the CC genotype have impaired cellular uptake of vitamin C, have a vitamin C deficiency.Susceptible individuals might benefit from a diet rich in vitamin C. Citrus fruits such as oranges, kiwi, lemon, and grapefruit, and cruciferous vegetables such as broccoli, Brussels sprouts, cabbage, and cauliflower can all be included in the diet. The diet can also include vitamin C dietary supplements.

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs10741657	CYP2R1	Impaired vitamin D conversion to its active form	⊕⊕G/G	Elevated	A/A

Associated nutrient: **Vitamin D, 25-OH**. Serum 25-hydroxyvitamin D (25(OH)D) is the main circulating metabolite of vitamin D and is a clinical biomarker of vitamin D. The CYP2R1 gene encodes the enzyme, 25-hydroxylase which catalyzes the conversion of vitamin D to 25(OH)D in the liver. Vitamin D is the major regulator of calcium homeostasis and protects the organism from calcium deficiency via effects on the intestine, kidney, parathyroid gland, and bone. Vitamin D is needed to keep bones, teeth, and muscles healthy. Mutations may cause a loss of gene function which hinders the conversion efficiency and can affect the circulating levels of 25(OH)D in the body, leading to a vitamin D deficiency.Individuals with the GG genotype have affected conversion of vitamin D leading to lower 25(OH)D levels associated with a vitamin D deficiency.Susceptible individuals might benefit from a diet rich in vitamin D. Vitamin D sources include egg yolks, cheese, beef liver, fatty fish like tuna, mackeral, salmon and fortified foods can all be included in the diet. Individuals can be supplemented with the hydroxylated form (25-hydroxyvitamin D) of vitamin D which will not have to undergo the conversion reaction and may actively help in increasing serum 25(OH)D levels.

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs10877012	CYP27B1	Impaired conversion of vitamin D to its active form	⊕⊕G/G	Elevated	T/T

Associated nutrient: **Vitamin D3 (Cholecalciferol)**. The CYP27B1 gene provides instructions for making an enzyme called 1-alpha-hydroxylase (1?-hydroxylase). This enzyme aids in one of the reactions involved in converting vitamin D to its active forms, 1,25-dihydroxyvitamin D3, also known as calcitriol. Vitamin D is the major regulator of calcium homeostasis and protects the organism from calcium deficiency via effects on the intestine, kidney, parathyroid gland, and bone. Vitamin D is needed to keep bones, teeth, and muscles healthy. Mutations may cause loss of gene function which hinders the enzyme from converting inactive vitamin D to its active form calcitriol. The mutation thus causes vitamin D3 deficiency.Homozygous mutant (abnormal) individuals who have lower conversion efficiency, cannot convert inactive vitamin D to its active form calcitriol and have a vitamin B9 deficiency.Susceptible individuals might benefit from a diet rich in vitamin D. Vitamin D sources include egg yolks, cheese, beef liver, fatty fish like tuna, mackerel, salmon and fortified foods, can all be included in the diet. Owing to impaired vitamin D conversion to calcitriol, individuals can directly be supplemented with 'calcitriol.'

Nutrient Genetics - Summary

Vitamins

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs2108622	CYP4F2	Affected gene activity associated with vitamin K function	⊕⊕T/T	Elevated	C/C

Associated nutrient: **Vitamin K1 (Phylloquinone)**. The CYP4F2 gene affects vitamin K activity and is a predictor of warfarin dose in individuals with cardiovascular diseases. Vitamin K1 ensures blood clotting to reduce bleeding from wounds. Warfarin is an oral anticoagulant and inhibits vitamin K associated enzymes required for the activation of clotting factors. For heart patients, prone to developing harmful blood clots, blood thinners such as warfarin is prescribed. Thus, high levels of vitamin K can decrease the effect of warfarin.Individuals with homozygous mutants (abnormal) TT genotype have higher vitamin K levels thus require higher warfarin dosing.Vitamin K1 supplements and foods rich in vitamin K1 (kale, broccoli, spinach, lettuce) should be avoided when patients are under warfarin dosage.

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs12934922	BCO1	Impaired beta carotene conversion	⊕⊖A/T	Partially elevated	A/A

Associated nutrient: **Vitamin A (All-Trans-Retinol)**. The BCO1 gene encodes for an enzyme beta-carotene oxygenase 1. This enzyme converts the most abundant provitamin A carotenoid in the diet, beta-carotene, to retinol. The active form of vitamin A, retinol, is required by photoreceptors in the eye, stem cells, immune cells, red blood cells, and embryonic cells. It also affects the expression of the major extracellular matrix constituents, including collagen, laminin, entactin, fibronectin, elastin, and proteoglycans. The rs12934922 mutation inhibits the gene's expression, resulting in lower conversion of beta-carotene to retinol.Individuals with the TA genotype exhibit poor conversion efficiency and may have a reduced ability to convert beta-carotene to retinol.Susceptible individuals might benefit from a diet rich in retinol, the active form of vitamin A which be readily used by the body. Animal-sourced foods, such as oily fish, liver, cheese, butter, cheese, eggs, oily fish, fortified low-fat spreads, milk, and yogurt can all be included in the diet.

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs11645428	BCMO1	Impaired beta carotene conversion	⊕⊖A/G	Partially elevated	A/A

Associated nutrient: **Vitamin A (Beta-Carotene)**. The BCMO1 gene encodes the enzyme beta-carotene 15,15'-dioxygenase. This enzyme converts the most abundant provitamin A carotenoid in the diet, beta-carotene, to retinol. The active form of vitamin A, retinol, is required by photoreceptors in the eye, stem cells, immune cells, red blood cells, and embryonic cells. It also affects the expression of the major extracellular matrix constituents, including collagen, laminin, entactin, fibronectin, elastin, and proteoglycans. The rs6564851 mutation inhibits the gene's expression, resulting in lower conversion of beta-carotene to retinol.Individuals with the AG genotype exhibit poor conversion efficiency and may have a reduced ability to convert beta-carotene to retinol.Susceptible individuals might benefit from a diet rich in retinol, the active form of vitamin A which be readily used by the body. Animal-sourced foods, such as oily fish, liver, cheese, butter, cheese, eggs, oily fish, fortified low-fat spreads, milk, and yogurt can all be included in the diet.

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs17514104	SLC35F3	Impaired thiamine cell-uptake protein function	⊕⊖C/T	Partially elevated	C/C

Associated nutrient: **Vitamin B1 (Thiamine diphosphate)**. SLC35F3 gene encodes for the protein solute carrier family 35 member F3. This protein is a thiamine transporter protein that causes the cellular uptake and secretion of thiamin. In the cell, thiamin also called vitamin B1 plays an important role in energy metabolism. It aids in the breakdown of carbohydrates into energy. It is also involved in many cellular processes and is necessary for the proper functioning of the nervous system. The mutation reduces the gene expression and impairs the cellular uptake of thiamine into the cells. This can give rise to vitamin B1 deficiency irrespective of its prevalence in serum. As a result, low intracellular levels of vitamin B1 can have implications on energy metabolism.Individuals with the CT genotype have impaired cellular uptake of vitamin B1, have a vitamin B1 deficiency.Susceptible individuals might benefit from a diet rich in vitamin B1. Cereals, beef, pork, nuts, whole grains, pulses, oranges, cauliflower, oranges, potatoes, asparagus, kale, liver, eggs, brewer's yeast, and blackstrap molasses can all be included in the diet. The diet can also include dietary vitamin B1 supplements.

Nutrient Genetics - Summary

Vitamins

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs4588	VDR	Affected activity of the vitamin D receptor gene that acts as a regulatory factor.	⊕⊖A/C	Partially elevated	C/C

Associated nutrient: **Vitamin D, 1-25 dihydroxy**. VDR gene encodes the vitamin D receptor that binds to vitamin 1,25(OH)2D to form a regulatory factor that modulates the processing of genes in many tissues in the body. Vitamin D is the major regulator of calcium homeostasis and protects the organism from calcium deficiency via effects on the intestine, and kidney, parathyroid gland, and bone. Vitamin D is needed to keep bones, teeth, and muscles healthy. Mutation in the gene disrupts the normal functioning leading to a vitamin D deficiency.Individuals with the AC genotype have intermediate levels of vitamin 25(OH)D and 1,25(OH)2D.Vitamin D -ich foods are recommended to affected individuals. Vitamin D sources include egg yolks, cheese, beef liver, fatty fish like tuna, mackerel 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.

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs12785878	NADSYN1	Affected activity of the gene which is linked to serum vitamin D concentrations.	⊕⊖G/T	Partially elevated	G/G

Associated nutrient: **Vitamin D, 25-OH**. Glutamine-dependent NAD(+) synthetase is an enzyme that in humans is encoded by the NADSYN1 gene. The NADSYN1 gene functions to regulate and coordinate the basic activities of cells and are linked to vitamin D serum concentrations. Vitamin D is the major regulator of calcium homeostasis and protects the organism from calcium deficiency via effects on the intestine, kidney, parathyroid gland, and bone. Vitamin D is needed to keep bones, teeth, and muscles healthy. Mutations may cause loss of gene function which decreases the vitamin D levels in the serum. This can give rise to vitamin D deficiency and thus, affects the above-mentioned serum functions.Individuals with the GT genotype have decreased vitamin D levels in the serum have a vitamin D deficiency.Susceptible individuals might benefit from a diet rich in vitamin D. Vitamin D sources include egg yolks, cheese, beef liver, fatty fish like tuna, mackerel, salmon and fortified foods, can all be included in the diet.

Minerals

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs4284505	ESR1	Affected enamel mineralization in the presence of fluoride leading to fluorosis	⊕⊕G/G	Elevated	unavailable

Associated nutrient: **Fluoride**. The ESR1 gene is responsible for enamel formation of the tooth. Fluoride prevents the development of dental caries by having a significant effect on dental tissue and plaque formation. However, excess fluoride intake can lead to dental fluorosis. 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.Individuals with the GG genotype have altered gene function and are at a high risk of dental fluorosis when exposed to excessive fluoride containing water.Susceptible individuals must reduce the consumption of acidic, sugary foods and drinks and must avoid drinking water containing fluoride. Dietary calcium and magnesium can reduce fluoride absorption.

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs225014	DIO2	Affected iodine-associated gene which is responsible for maintaining T3	⊕⊕C/C	Elevated	T/T

Associated nutrient: **Iodine**. Type I iodothyronine deiodinase is an enzyme that in humans is encoded by the DIO2 gene. The type I iodothyronine deiodinase catalyzes the removal of an iodine residue from the pro-hormone thyroxine (T4) molecule, thus producing either the active form triiodothyronine (T3; activation) or inactive metabolites (reverse T3; inactivation). The mutation leads to the downregulation of the gene affecting the conversion of T4 to T3 by the removal of iodine.Individuals with the CC genotype have an 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 may be prone to iodine deficiency and carry the CC and CT genotypes.

Nutrient Genetics - Summary

Minerals

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs1799945	HFE	Impaired absorption of iron	⊕⊕G/G	Elevated	C/C

Associated nutrient: **Iron, Fe 56**. The HFE gene codes for a protein that interacts with other proteins on the cell surface to detect and regulate the amount of iron in the body. This protein is also part of a signaling pathway that controls the levels of another protein called hepcidin. Hepcidin is secreted primarily by hepatocytes into the circulation, and there levels are generally less since it functions to inhibit iron absorption in the proximal small intestine. Mutation inhibits HFE protein from interacting with other proteins allowing excess absorption of iron in the body, a disorder called hemochromatosis. The H63D is a variant that indicates an iron metabolism disorder which accounts for a mild form of hereditary hemochromatosis (HH). The most common form is caused by mutations in the HFE gene, which are inherited recessively. Women may be less affected by hemochromatosis due to elimination of excess iron during menstruation, but may become affected after menopause.Individuals with the GG genotype are likely affected by mild form of hemochromatosis due to excess iron absorption.Affected individuals are recommended to reduce iron levels in the body. Heme iron is easier for the body to absorb than non-heme iron. Plant-based foods contain only non-heme iron, whereas meat, poultry, fish, and seafood contain both heme and non-heme iron. Calcium rich foods such as dairy products can inhibit the absorption of both non-heme and heme iron.

rs1800562	HFE	Impaired absorption of iron	⊕⊕A/A	Elevated	G/G

Associated nutrient: **Iron, Fe 56**. The HFE gene codes for a protein that interacts with other proteins on the cell surface to detect and regulate the amount of iron in the body. This protein is also part of a signaling pathway that controls the levels of another protein called hepcidin. Hepcidin is secreted primarily by hepatocytes into the circulation, and there levels are generally less since it functions to inhibit iron absorption in the proximal small intestine. Mutation inhibits HFE protein from interacting with other proteins allowing excess absorption of iron in the body, a disorder called hemochromatosis. The H63D is a variant that indicates an iron metabolism disorder which accounts for a mild form of hereditary hemochromatosis (HH). The most common form is caused by mutations in the HFE gene, which are inherited recessively. Women may be less affected by hemochromatosis due to elimination of excess iron during menstruation, but may become affected after menopause.Individuals with the AA genotype are likely affected by mild form of hemochromatosis due to excess iron absorption.The AA genotype women who are affected after menopause are advised to consume non-heme plant based foods containing phytates and polyphenols such as greens and legumes, lean proteins, eggs, tea and coffee which will help reduce iron absorption. Calcium rich foods such as dairy products can inhibit the absorption of iron. They should also avoid raw sea food, vitamin A and C rich foods, fortified foods, alcohol, iron vitamin C and multivitamin supplements.

rs3811647	TF	Impaired iron transportation	⊕⊕A/A	Elevated	G/G

Associated nutrient: **Iron, Fe 56**. TF gene encodes for glycoprotein transferrins. Transferrin is a transport protein that plays a central role in iron metabolism and is responsible for ferric-ion delivery. Transferrin functions as the most critical ferric pool in the body. It transports iron through the blood to various tissues such as the liver, spleen, and bone marrow. Iron is an essential component of hemoglobin, an RBC protein that transfers oxygen from the lungs to the tissues of the body. Iron is also involved in cellular functioning, the synthesis of some hormones, and muscle metabolism. RBC values of iron give insights into the iron stores of the body, which can affect nutrient values. Mutation reduces gene activity that leads to a reduction in iron transport to tissues, which can lead to lower levels of intracellular iron, thus affecting the above-mentioned functions.Individuals with the AA genotype have a reduced iron transport carry the risk of reduced iron levels in the cells.Susceptible individuals are advised to consume an iron-rich diet including cereals, beans, lentils, tofu, spinach, apricots, prunes, raisins, prune juice, enriched bread, broccoli, and nuts. The diet can also include dietary iron supplements.

rs4680	COMT	Lowered COMT gene activity resulting in increased dopamine levels in the brain. Magnesium can enhance COMT activity.	⊕⊕A/A	Elevated	G/G

Associated nutrient: **Magnesium, Mg 24**. The nutrient, magnesium, plays crucial roles in the nervous system. The COMT gene encodes an enzyme which breaks down dopamine (neurotransmitter- molecules that help transmit signals) in the brain. Magnesium enhances COMT activity. Mutation lowers the enzymatic activity resulting in increased dopamine levels in the brain, which can affect stress resiliency.Individuals with the AA genotype have altered gene function and reduced stress resiliency.Magnesium, a cofactor for COMT activity may help increase COMT levels, hence, foods rich in magnesium (pumpkin seeds, almonds, spinach boiled, cashews, peanuts) are recommended to affected individuals.

Nutrient Genetics - Summary

Minerals

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs13107325	SLC39A8	Impaired absorption of manganese	⊕⊕T/T	Elevated	C/C

Associated nutrient: **Manganese, Mn 55**. SLC39A8 gene encodes for the membrane transporter protein responsible for manganese absorption into the blood. Manganese contributes to many bodily functions, including bone formation, blood clotting, and reducing inflammation. It also plays a role in fat and carbohydrate metabolism, calcium absorption, and blood sugar regulation. Manganese is also necessary for normal brain and nerve function. The mutation leads to altered gene function resuting low or deficient levels of manganese.Individuals with the TT genotype have affected gene function leading to low manganese levels.Affected individuals are recommended to have manganese-rich foods such as, shellfish, nuts, seeds, whole grains, legumes, and leafy green vegetables.

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs594445	MOCOS	Affected molybdenum cofactor enzyme	⊕⊕A/A	Elevated	C/C

Associated nutrient: **Molybdenum**. The MOCOS gene provides instructions for making an enzyme called molybdenum cofactor sulfurase. Molybdenum cofactor sulfurase carries out a chemical reaction that adds sulfur to a molecule called the molybdenum cofactor. This molecule is required to turn on (activate) xanthine dehydrogenase. Xanthine dehydrogenase is involved in the conversion of xanthine to uric acid, a waste product that is normally excreted in urine and feces. Mutations in the gene alter the shape and function of the enzyme thus molybdenum cofactor sulfurase is unable to add sulfur to the molybdenum cofactor, and xanthine dehydrogenase is not activated. This lead to excess xanthine which can form tiny crystals and accumulate in the kidneys, occasionally leading to the formation of stones that can impair kidney function and ultimately cause kidney failure.Homoygous mutant (abnormal) individuals have altered genes with altered shape and function of the enzyme (molybdenum cofactor sulfurase), leading to a molybdenum cofactor deficiencyIndividuals with a non functional gene (AA genotypes) are recommended to consume molybdenum supplements.

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs4343	ACE	Impaired regulation of ACE activity by potassium	⊕⊕G/G	Elevated	A/A

Associated nutrient: **Potassium**. The ACE gene associated with angiotensin-converting enzyme (ACE) activity regulates blood pressure by relaxing the veins and maintaining body salts and uids. Potassium levels in the body regulates the ACE activity. High levels of potassium decreases its activity relaxing the veins. Mutations in the gene impairs its fuction leading to high blood pressure. Heterozygous individuals have intermediate concentrations of ACE serum levels, while homozygous mutant individuals have high ACE serum levels and is associated with blood pressure which can lead to hypertension.Homozygous mutant (abnormal) individuals have high ACE serum levels and is associated with blood pressure which can lead to hypertension, migraine and cardiovascular diseases.Individuals with a GG genotype may be advised high dietary potassium intake as potassium levels could reduce ACE activity and prevent high blood pressure.

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs1050450	GPX1	Affected gene associated with selenium concentration.	⊕⊕T/T	Elevated	C/C

Associated nutrient: **Selenium, Se 76**. Selenium is a trace element in the body and it is an essential component of various enzymes and proteins, called selenoproteins , that help in the synthesis of DNA and protect against cell damage and infections. Selenium also has antioxidant properties that help to break down peroxides, which can damage tissues and DNA, leading to inflammation and other health problems. The GPX1 gene activity is sensitive to changes in the selenium status in individuals with low to moderate intake. Mutations in the gene can alter the selenium status in the body which causes oxidative stress thus, increasing the risk of acquiring several diseases.Individuals with the TT allele have altered gene function leading to a selenium deficiency.Individuals susceptible to selenium deficiency are advised to consume a diet rich in selenium. High amounts of selenium are found in pork, beef, turkey, chicken, fish, shellfish, and eggs. The diet can also include dietary selenium supplements.

Nutrient Genetics - Summary

Minerals

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs3877899	SEPP1	Affected transport of selenium	⊕⊕G/G	Elevated	A/A

Associated nutrient: **Selenium, Se 76**. Selenoprotein P is a protein that in humans is encoded by the SEPP1 gene and is important for transporting selenium into the cells. In cells, selenium is part of enzymes and proteins, called selenoproteins, which aid in the production of DNA and defend against infections and cell damage. Additionally, selenium has potent antioxidant properties that help to protect cells from peroxides, organic hydroperoxides, and peroxynitrites by converting them to harmless forms. Mutation in the gene reduces gene activity and the levels of selenoprotein P. Thus mutations reduce the transport of selenium to various cells in the body. As a result, different organs may suffer from low levels of selenium due to impaired selenium transport.Individuals with the GG genotype have reduced transport of selenium to various cells of the body, have decreased selenium levels.Individuals susceptible to selenium deficiency are advised to consume a diet rich in selenium. High amounts of selenium are found in pork, beef, turkey, chicken, fish, shellfish, and eggs. The diet can also include dietary selenium supplements.

rs5030853	PAH	Affected production of tetrahydrobiopterin	⊕⊕T/T	Elevated	G/G

Associated nutrient: **Tetrahydrobiopterin**. The PAH gene provides instructions for making an enzyme called phenylalanine hydroxylase (PAH). The PAH is involved in the first step of metabolizing phenylalanine to tyrosine. Phenylalanine is involved in the production of the neurotransmitters norepinephrine and dopamine, which are essential for the proper functioning of the brain and nervous system. Phenylalanine also helps with the production of the pigment melanin, which gives color to the skin, hair, and eyes. The mutation decreases gene expression resulting in tetrahydrobiopterin deficiency resulting in the failure of phenylalanine metabolism. Accumulation of phenylalanine in the blood or other tissues disrupts brain functioning and the condition is called phenylketonuria or hyperphenylalaninemia.Homozygous mutant (abnormal) individuals who have tetrahydrobiopterin deficiency have failure of phenylalanine metabolism.Susceptible individuals are advised to consume a low-protein diet and must avoid protein-rich food such as meat, dairy and fish.

rs2304478	SLC12A3	Impaired sodium reabsorption	⊕⊖A/G	Partially elevated	A/A

Associated nutrient: **Sodium**. The SLC12A3 gene encodes a cotransporter protein NCC (Na-Cl cotransporter). Reabsorbing sodium and chloride ions from the tubular fluid into the cells of the distal convoluted tubule of the nephron is the primary function of NCC. It is part of the mechanism by which kidneys reabsorb salt (sodium chloride or NaCl) from the urine back into the bloodstream. Sodium reabsorption is needed because passive water reabsorption and sodium reabsorption are closely related, so when sodium moves, water also does. Water flow maintains extracellular body fluid volume by balancing the osmotic pressure within or across tubule walls. Mutation in the gene leads to its increased gene expression and increased NCC protein levels in the kidney. This elevates sodium reabsorption in the kidney leading to increased blood pressure.Heterozygous (partially abnormal) individuals who have increased gene expression have elevated sodium reabsorption in the kidney.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. Additionally, potassium supplementation also enhances sodium excretion and reduces sodium retention.

rs7204044	SLC12A3	Impaired sodium reabsorption	⊕⊖A/G	Partially elevated	A/A

Associated nutrient: **Sodium**. The SLC12A3 gene encodes a cotransporter protein NCC (Na-Cl cotransporter). Reabsorbing sodium and chloride ions from the tubular fluid into the cells of the distal convoluted tubule of the nephron is the primary function of NCC. It is part of the mechanism by which kidneys reabsorb salt (sodium chloride or NaCl) from the urine back into the bloodstream. Sodium reabsorption is needed because passive water reabsorption and sodium reabsorption are closely related, so when sodium moves, water also does. Water flow maintains extracellular body fluid volume by balancing the osmotic pressure within or across tubule walls. Mutation in the gene leads to its increased gene expression and increased NCC protein levels in the kidney. This elevates sodium reabsorption in the kidney leading to increased blood pressure.Heterozygous (partially abnormal) individuals who have increased gene expression have elevated sodium reabsorption in the kidney.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. Additionally, potassium supplementation also enhances sodium excretion and reduces sodium retention.

Nutrient Genetics - Summary

Amino Acids

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs7946	PEMT	Impaired conversion of phosphatidylethanolamine (PE) into phosphotidyl choline (PC)	⊕⊕T/T	Elevated	C/T, C/C

Associated nutrient: **Choline**. PEMT encodes an enzyme phosphatidylethanolamine N-methyltransferase. This enzyme converts phosphatidylethanolamine (PE) into phosphotidyl choline (PC), using S-adenosylmethionine (SAM-e) as a methyl group donor. PEMT is mainly expressed in the liver and accounts for 30% of liver PC production. PC can then release choline and, to a certain extent, compensate for the lack of this nutrient. This enzyme is crucial for maintaining liver choline levels. Choline is vital for maintenance of various key metabolic processes which play a role in the prevention or progression of various health impairments. Choline deficiency is associated with significant damage to DNA and with apoptosis in peripheral lymphocytes. Low levels of choline in WBCs can lead to its intracellular deficiency, thus affecting the above-mentioned functions. Mutation alters the gene activity and inhibits the process of converting PE to PC and thus the choline deficiency. Choline deficiency can thus affect the above-mentioned functions.Individuals with the TT genotype have inhibited conversion PE to PC, have a choline deficiency.Affected individuals are recommended to include lentils, nuts, soy products, fish, beef, poultry, eggs, lean meats, beans, peas to increase choline levels in the body.

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs775607037	COQ4	Impaired production of coenzyme Q10	⊕⊕T/T	Elevated	C/T, C/C

Associated nutrient: **Coenzyme Q10 (Ubiquinone + Ubiquinol), Total**. Coenzyme Q4 is a protein that in humans is encoded by the COQ4 gene. This protein is involved in the production of a molecule called coenzyme Q10. Coenzyme Q10 functions as one of the most significant lipid antioxidants in the cell that prevents the generation of free radicals and modifications of proteins, lipids, and DNA. It is also required for cellular growth and immune function. Coenzyme Q10 (CoQ10) is an essential cofactor in oxidative phosphorylation in mitochondria and is fundamentally important to cellular energy (ATP) production. Additionally, CoQ10 has direct antioxidant effects. Mutation impairs the gene and the protein activity. This leads to reduced production of coenzyme Q10 resulting in its deficiency. Coenzyme Q10 deficiency can thus affect the above-mentioned functions.Homozygous (abnormal) individuals who have reduced production of coenzyme Q10 resulting in COQ10 deficiency.Susceptible individuals are advised to consume a diet rich in COQ10. High amounts of COQ10 are found organ meat and whole grains. The diet can also include coenzyme Q10 dietary supplements.

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs121909307	GSS	Impaired production of glutathione	⊕⊕A/A	Elevated	G/G

Associated nutrient: **Glutathione Oxidized**. The GSS gene provides instructions for making an enzyme called glutathione synthetase. Glutathione synthetase participates in a process called the gamma-glutamyl cycle. These reactions are necessary for the production of glutathione, a small molecule made of three protein building blocks (amino acids). Glutathione is called an antioxidant because of its role in protecting cells from the damaging effects of these unstable molecules. Glutathione also helps build DNA, proteins, and other important cellular components. Mutation in the gene reduces the activity of the enzyme and glutathione synthase deficiency. This disrupts the gamma-glutamyl cycle, preventing adequate production of glutathione. Thus mutations lead to glutathione deficiency.Homozygous (abnormal) individuals who have low glutathione synthase levels are associated with a glutathione deficiency.Foods rich in glutathione (mushrooms, avocados, spinach, okra) is recommended to affected individuals. The diet can also include dietary N-acetylcysteine (NAC) supplements.

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs121918252	MUT	Affected activity of the enzyme that metabolizes fat and protein leading to the accumulation of methylmalonic acid.	⊕⊕T/T	Elevated	G/G

Associated nutrient: **MMA (Methymalonic Acid)**. The MUT gene is responsible for making an enzyme, methylmalonyl-CoA mutase which is active in components of the cells that serve as energy-producing sites. The enzyme metabolizes proteins and fats. Mutation results in enzyme deficiency and abolish its activity disrupting normal amino acid and fat metabolism. This allows accumulation of substances (including methylmalonic acid (MMA)) which are harmful to the body. This damages the nervous system, kidneys and other organs. Affected individuals have excess of MMA in the blood and urine.Homozygous mutants (abnormal) individuals have altered gene function leading to elevated MMA levels associated with the risk of methylmalonic aciduria.Affected individuals are recommended to include a low-protein diet, containing the amino acids isoleucine, methionine, threonine, and valine and certain fats but high in calories. As increased MMA levels are indicative of a vitamin B12 deficiency, patients can be supplemented with vitamin B12-rich foods and supplements.

Nutrient Genetics - Summary

Amino Acids

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs291466	HICBH	Variation in methylmalonic acid (MMA) levels which can be associated with vitamin B12 deficiency.	⊕⊕C/C	Elevated	T/T

Associated nutrient: **MMA (Methymalonic Acid)**. Methylmalonic acid (MMA) is a substance produced in the body during metabolism (conversion of food to energy). It is an essential biomarker for vitamin B12 deficiency. Vitamin B12 plays an important role in metabolism. It reacts with MMA to form coenzyme A (CoA). During a vitamin B12 deficiency, the level of MMA is seen to increase. A polymorphisms in the HICBH gene accounts for a variation in the MMA levels which reacts with vitamin B12 to produce a coenzyme essential for normal cellular function. Vitamin B12 (cobalamin) deficiency leads to increase in MMA .Individuals with the CC genotype have altered gene function and higher levels of MMA.Affected individuals may be recommended to restrict intact protein and supplement with amino acid-based formula. As increased MMA levels are indicative of a vitamin B12 deficiency, patients can be supplemented with vitamin B12-rich foods and supplements.

rs5030853	PAH	Failure of phenylalanine metabolism.	⊕⊕T/T	Elevated	G/G

Associated nutrient: **Phenylalanine**. The PAH gene provides instructions for making an enzyme called phenylalanine hydroxylase (PAH). The PAH is involved in the first step of metabolizing phenylalanine to tyrosine. Phenylalanine is involved in the production of the neurotransmitters norepinephrine and dopamine, which are essential for the proper functioning of the brain and nervous system. Phenylalanine also helps with the production of the pigment melanin, which gives color to the skin, hair, and eyes. The mutation decreases gene expression resulting in tetrahydrobiopterin deficiency resulting in the failure of phenylalanine metabolism. Accumulation of phenylalanine in the blood or other tissues disrupts brain functioning and the condition is called phenylketonuria or hyperphenylalaninemia.Homozygous mutant (abnormal) individuals who have tetrahydrobiopterin deficiency, have failure of phenylalanine metabolism. They have elevated phenylalanine levels associated with the risk of phenylketonuria.Patients with decreased metabolism of the amino acid phenylalanine are advised to avoid protein-rich food such as meat, dairy, and fish. Patients may benefit from a phenylalanine-restricted diet. Several nutrients such as carnitine, selenium, zinc, vitamin B6, vitamin B12, calcium, folate, iron, DHA, and EPA are also supplemented to avoid nutrient inadequacy that might occur due to the protein-restricted diet.

rs3733890	BHMT	Impaired NOS3 gene function	⊕⊖A/G	Partially elevated	A/A

Associated nutrient: **Choline**. (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.Heterozygous (partially abnormal) individuals have reduced levels of folic acid and choline.Folic acid and choline supplementation are recommended to individuals with susceptible genotypes.

rs786204770	COQ4	Impaired production of coenzyme Q10	⊕⊖A/G	Partially elevated	A/A

Associated nutrient: **Coenzyme Q10 (Ubiquinone + Ubiquinol), Total**. Coenzyme Q4 is a protein that in humans is encoded by the COQ4 gene. This protein is involved in the production of a molecule called coenzyme Q10. Coenzyme Q10 functions as one of the most significant lipid antioxidants in the cell that prevents the generation of free radicals and modifications of proteins, lipids, and DNA. It is also required for cellular growth and immune function. Coenzyme Q10 (CoQ10) is an essential cofactor in oxidative phosphorylation in mitochondria and is fundamentally important to cellular energy (ATP) production. Additionally, CoQ10 has direct antioxidant effects. Mutation impairs the gene and the protein activity. This leads to reduced production of coenzyme Q10 resulting in its deficiency. Coenzyme Q10 deficiency can thus affect the above-mentioned functions.Heterozygous (partially abnormal) individuals who have reduced production of coenzyme Q10 resulting in COQ10 deficiency.Susceptible individuals are advised to consume a diet rich in COQ10. High amounts of COQ10 are found organ meat and whole grains. The diet can also include coenzyme Q10 dietary supplements.

Nutrient Genetics - Summary

Amino Acids

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs1695	GSTP1	Affected modification of toxic compounds to glutathione	⊕⊖A/G	Partially elevated	A/A

Associated nutrient: **Glutathione Oxidized**. Glutathione S-transferase P is an enzyme that in humans is encoded by the GSTP1 gene. This enzyme catalyzes the conjugation of electrophilic substrates to glutathione. Glutathione is an amino acid that is an antioxidant because of its role in protecting cells from the damaging effects of these unstable molecules. Glutathione also helps build DNA, proteins, and other important cellular components. Glutathione is essential for the immune system's proper functioning and is vital in building and repairing tissue. Mutation in the gene reduces the activity of the enzyme and disrupts the conjugation of electrophilic substrates to glutathione. Thus mutations lead to glutathione deficiency.Individuals with the AG genotype have disrupted the conjugation of electrophilic substrates to glutathione, have a glutathione deficiency.Susceptible individuals are advised to consume a diet rich in glutathione. High amounts of glutathione are found in Spinach, avocados, asparagus, okra, beef, fish, broccoli, brussels sprouts, cauliflower, kale, watercress, garlic, shallots, and onions. The diet can also include dietary N-acetylcysteine (NAC) supplements.

Nutrient Genetics

Vitamins

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs11645428	BCMO1	Impaired beta carotene conversion	⊕⊕G/G	Elevated	A/A
rs12934922	BCO1	Impaired beta carotene conversion	⊕⊖A/T	Partially elevated	A/A
rs1667255	TTR	Impaired transportation of Vitamin A	⊕⊕A/A	Elevated	C/C
rs6564851	BCMO1	Impaired beta carotene conversion	⊕⊖G/T	Elevated	T/T
rs7501331	BCO1	Impaired beta carotene conversion	⊕⊕T/T	Elevated	C/C
rs11645428	BCMO1	Impaired beta carotene conversion	⊕⊖A/G	Partially elevated	A/A
rs17514104	SLC35F3	Impaired thiamine cell-uptake protein function	⊕⊖C/T	Partially elevated	C/C
rs492602	FUT2	Impaired absorption of vitamin B12	A/A	Unavailable	T/T
rs526934	TCN1	Impaired cellular uptake of vitamin B12	⊕⊕G/G	Elevated	A/A
rs602662	FUT2	Impaired cellular uptake of vitamin B12	⊕⊕G/G	Elevated	A/A
rs1799983	NOS3	Impaired NOS3 gene function	⊕⊕T/T	Elevated	G/G
rs13078881	BTD	Affected activity of the enzyme that is responsible for the reuse and recycle of biotin.	⊕⊕C/C	Elevated	G/G
rs1801131	MTHFR	Impaired methylation which is associated with the conversion of inactive to active folate	⊕⊕C/C	Elevated	A/A
rs1801133	MTHFR	Impaired methylation which is associated with the conversion of inactive to active folate	⊕⊕T/T	Elevated	C/C
rs33972313	SLC23A1	Impaired cellular uptake of vitamin C	⊕⊕A/A	Elevated	G/G
rs4257763	SLC23A1	Impaired cellular uptake of vitamin C	⊕⊕G/G	Elevated	A/G, A/A
rs6139591	SLC23A2	Impaired cellular uptake of vitamin C	G/G	Unavailable	C/T, C/C
rs6596473	SLC23A1	Impaired cellular uptake of vitamin C	⊕⊕C/C	Elevated	G/G
rs4588	VDR	Affected activity of the vitamin D receptor gene that acts as a regulatory factor.	⊕⊖A/C	Partially elevated	C/C
rs10741657	CYP2R1	Impaired vitamin D conversion to its active form	⊕⊕G/G	Elevated	A/A
rs12785878	NADSYN1	Affected activity of the gene which is linked to serum vitamin D concentrations.	⊕⊖G/T	Partially elevated	G/G

Nutrient Genetics

Vitamins

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs2282679	GC	Impaired vitamin D3 transport	G/G	Unavailable	A/A
rs10766197	CYP2R1	Impaired conversion of vitamin D to vitamin D [25(OH)D].	⊖⊖A/A	Normal	A/A
rs10877012	CYP27B1	Impaired conversion of vitamin D to its active form	⊕⊕G/G	Elevated	T/T
rs12272004	APOA5	Affected vitamin E transport	⊖⊖C/C	Normal	C/C
rs2108622	CYP4F2	Affected gene activity associated with vitamin K function	⊕⊕T/T	Elevated	C/C

Minerals

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs4516035	VDR	Affected intestinal calcium absorption and bone mineralization	G/G	Unavailable	T/T
rs76151636	ATP7B	Impaired transport of copper to the cells (cellular absorption) and elimination of copper	A/A	Unavailable	G/G
rs4284505	ESR1	Affected enamel mineralization in the presence of fluoride leading to fluorosis	⊕⊕G/G	Elevated	unavailable
rs225014	DIO2	Affected iodine-associated gene which is responsible for maintaining T3	⊕⊕C/C	Elevated	T/T
rs1799945	HFE	Impaired absorption of iron	⊕⊕G/G	Elevated	C/C
rs1800562	HFE	Impaired absorption of iron	⊕⊕A/A	Elevated	G/G
rs3811647	TF	Impaired iron transportation	⊕⊕A/A	Elevated	G/G
rs4820268	TMPRSS6	Impaired iron content regulation	⊕⊖A/G	Normal	A/G
rs855791	TMPRSS6	Impaired iron content regulation	A/A	Unavailable	C/C
rs4680	COMT	Lowered COMT gene activity resulting in increased dopamine levels in the brain. Magnesium can enhance COMT activity.	⊕⊕A/A	Elevated	G/G
rs13107325	SLC39A8	Impaired absorption of manganese	⊕⊕T/T	Elevated	C/C
rs594445	MOCOS	Affected molybdenum cofactor enzyme	⊕⊕A/A	Elevated	C/C
rs4074995	RGS14	Impaired phosphorus re-absorption	A/G	Unavailable	A/A
rs4343	ACE	Impaired regulation of ACE activity by potassium	⊕⊕G/G	Elevated	A/A
rs1050450	GPX1	Affected gene associated with selenium concentration.	⊕⊕T/T	Elevated	C/C

Nutrient Genetics

Minerals

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs3877899	SEPP1	Affected transport of selenium	⊕⊕G/G	Elevated	A/A
rs2304478	SLC12A3	Impaired sodium reabsorption	⊕⊖A/G	Partially elevated	A/A
rs7204044	SLC12A3	Impaired sodium reabsorption	⊕⊖A/G	Partially elevated	A/A
rs5030853	PAH	Affected production of tetrahydrobiopterin	⊕⊕T/T	Elevated	G/G
rs8007267	GCH1	Affected synthesis of tetrahydrobiopterin	A/A	Unavailable	C/C
rs11126936	SLC30A3	Affected absorption of zinc influencing its serum concentrations.	A/C	Unavailable	A/A

Amino Acids

Test Name	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs3733890	BHMT	Impaired NOS3 gene function	⊕⊖A/G	Partially elevated	A/A
rs7946	PEMT	Impaired conversion of phosphatidylethanolamine (PE) into phosphotidyl choline (PC)	⊕⊕T/T	Elevated	C/T, C/C
rs775607037	COQ4	Impaired production of coenzyme Q10	⊕⊕T/T	Elevated	C/T, C/C
rs786204770	COQ4	Impaired production of coenzyme Q10	⊕⊖A/G	Partially elevated	A/A
rs121909307	GSS	Impaired production of glutathione	⊕⊕A/A	Elevated	G/G
rs1695	GSTP1	Affected modification of toxic compounds to glutathione	⊕⊖A/G	Partially elevated	A/A
rs121918252	MUT	Affected activity of the enzyme that metabolizes fat and protein leading to the accumulation of methylmalonic acid.	⊕⊕T/T	Elevated	G/G
rs291466	HICBH	Variation in methylmalonic acid (MMA) levels which can be associated with vitamin B12 deficiency.	⊕⊕C/C	Elevated	T/T
rs5030853	PAH	Failure of phenylalanine metabolism.	⊕⊕T/T	Elevated	G/G

Risk and Limitations

Genetic testing is helpful in analyzing risks to various diseases. However, it is essential 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. 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 the analysis.

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.

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 collecting 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.

Variant risk classification may not align with associated disease risk or may change ex: a benign variant may be reported as pathogenic. Misclassification may be due to updated research studies, allele dropouts or interpretation pitfalls. Variant risk classification may also not be relevant to the tested individual of different or mixed ethnicities in comparison to the study group(s) from literature. Vibrant conducts internal audits, post market surveillance and feedback from providers and customers on an ongoing basis to keep our reports updated with the most current findings. 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 prior to any interventions and diet/supplement/lifestyle changes.

Genetic SNP testing is performed using real time PCR systems. It is important to note that allele calling for a particular SNP is performed using the Autocall methodology of the instrument manufacturer. Failure or error in autocalling could occur and is usually associated with outlier wells or software issues relevant to making an allele call. As with all genetic SNP testing, there is a small chance that the laboratory could report these incorrect results.

Genetic testing is not intended to diagnose a disease, tell you anything about your current state of health, or be used to make medical decisions, including whether you should take a medication/supplement or how much of a medication/supplement you should take. It is intended to provide users with their genetic information and suggestions to inform lifestyle decisions and conversations with their doctor or other health care professionals.