MaxGen LABS

MaxFunction Professional Series

Client Name:	Sample	Sample Received:	00/00/0000
Client DOB:	00/00/0000	Report Date:	00/00/0000
Vial Number:	MGP00000	MaxGen PTID#:	0000
Client Sex:	Male	CLIA Certification:	0000000000
Referring Account:	Sample		
Admin Notes:	Sample		
Lab Notes:	Sample		

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Basic Genetics & Information

Nutrigenomics: The study of how enetic expression is influenced by nutrition. Small variations in genetic structure may require specific nutritional support that is unique to each individual. Genetic testing provides insight to this need.

Genes: Transferred from parent to offspring, genes are the basic unit of heredity. Genes are found on chromosomes and are made up of DNA. Each person has two copies of a gene, one from each parent. Genes are named for the protein they create or the function they have, often being simplified into abbreviations (example: MTHFR – short for methylenetetrahydrofolate reductase).

DNA: Deoxyribonucleic Acid, or DNA, is a molecule within a gene that contains the instructions an organism needs to grow, function, and reproduce. It is the carrier of all genetic information and is made up of chemical base pairs: adenine (A), thymine (T), cytosine (C), guanine (G). The order of sequence determines the information needed to maintain life.

Single Nucleotide Polymorphism (SNPs): A variation in base pair sequencing that may alter the function of a gene. Nutrigenomic testing looks at these variations to determine how a gene may function. Each combination of base pairs may alter the function of a gene in different ways. The variations are described as:

Wild Type – most commonly found pairing in nature; no variation
Heterozygous – one variant copy from a parent; one non-variant copy from a parent
Homozygous – two variant copies, one from each parent

Vitamin D & Your Genetics

Vitamin D is a fat-soluble vitamin that must be converted in the liver and kidneys. Limited foods supply Vitamin D, so substantial exposure to sunlight or specific supplementation can be used when a deficiency is present. Vitamin D is crucial for calcium concentrations, bone growth, immune function, and the reduction of inflammation.

For daily use, both D2 and D3 forms of Vitamin D are beneficial, but D3 (cholecalciferol) should be used for therapeutic dosing during a deficiency. Supplements between 5,000 IU and 10,000 IU are ideal for daily therapeutic dosing. Daily intake should be between 1,000 – 2,000 IU of Vitamin D or cod liver oil for general wellness.

Ideally, Vitamin D should be absorbed from natural sunlight exposure. Between the hours of 10am – 3pm, UV rays should hit the face, neck, arms, and shoulders for 10-30 minutes at least twice a week (avoid skin burns).

The three VDR SNPs in this test are from a physician poll of the most common SNPs needed in clinical practice. For blood work, practitioners tend to look at 25(OH) D by itself, while other practitioners also look at 1,25(OH)2D. The 1,25-dihydroxyvitamin D is formed from 25(OH)D in the kidneys under the influence of Parathyroid Hormone and specific enzymes; whereas, 25(OH)D is converted in the liver. It is also recommended to measure HbA1c for blood sugar control.

Vitamin D Foods

Cod Liver Oil
Swordfish
Salmon
Beef Liver
Egg Yolks
Cheese

Vitamin D Testing

1,25 OH Vitamin D may be helpful in some complicated cases. Your Doctor may order the following tests.:
25-hydroxy (OH) vitamin D
1,25 dihydroxyvitamin D

Health Conditions

Rickets
Osteoporosis
Cancer
Inflammatory Bowel Disease
Multiple Sclerosis
Type I and II Diabetes

VDR-BSM

No variant detected for Vitamin D deficiency.

VDR-TAQ

No variant detected for Vitamin D deficiency.

VDR-FOK

No variant detected for Vitamin D deficiency.

B12 & Your Genetics Report

Do you get enough Cobalamin, or vitamin B12? Do you take the right form of B12? Since your body does not produce B12, it is important to make sure you get adequate amounts of it in the correct form. B12 is important for a number of processes in the body, especially the production of neurotransmitters, energy, and blood cells.
People often feel better switching to the correct form of B12 based on genetics and/or increasing their consumption. Consider yearly micronutrient testing. Always avoid cyanocobalamin.

B12 Blood Levels

Many genes are associated with decreased serum B12 levels. Increasing supplementation or using dermal or injectable B12 can help bypass a possible genetic issue.

Genes: FUT2 & TCN

You have a risk for low serum Vitamin B12 levels. Use organic acid or homocysteine testing to verify your need for B12.

Methy-B12 Need

Produced by the enzyme MTRR, Methylcobalamin is the main form of B12 used fordetoxification and neurotransmitter production. It is bio-active and can be found in good quality supplements.

Genes: MTRR

You have one heterozygous variation on the MTRR enzyme. This could create a need for B12 supplementation. Use organic acid or homocysteine testing to verify your need for B12.

Adenosyl-B12 Need

Adenosylcobalamin is mainly used to produce energy within the mitochondria. Many people report increased energy with Adeno-B12 supplementation.

Genes: MUT & MMAB

There are no genetic indications that you need Adenosylcobalamin supplementation. Adenosylcobalamin could be used in cases of fatigue.

Methyl-B12-Sensitivity

Some people report sensitivities to methylated B12, including increased aggression and hyperactivity. We can occasionally predict these sensitivities by looking at other variations.

Genes: COMT & VDR

There are no genetic indications for Methylcobalamin sensitivity.

Low B12 Symptoms

- Anxiety
- Pale Skin
- Smooth Tongue
- Constipation
- Diarrhea
- Heart Palpitations
- Fatigue
- Poor Balance
- Memory loss
- Neuropathy
- Tingling feet
- Depression

Folate & Your Genetics

Folate, or B9, is a vitamin required for numerous processes in the body. DNA replication, neurotransmitter production and degradation, detoxification, and prevention of cardiovascular disease are just a few. It is found naturally in uncooked leafy green vegetables.

Folate - MTHFR

The MTHFR enzyme processes dietary folates into methyl-folate, crucial for methylation and over 200 processes in the body. Low levels of methylfolate have been associated with numerous symptoms and diseases. There are two main variants: C667T and A1298C.

Since MTHFR creates methylfolate, you can supplement with oral methylfolate. This can speed up the methylation cycle, returning detoxification and neurotransmitter production back to normal. This testing and approach has become common in fertility and psychiatric practices.

It is important to start slow and titrate up when using methyl folate. 400mcg is a common starting point for adults. Some research points to benefits from 400mcg to 15mg; however, many people do very well on doses under 2mg. Please see a practitioner for help with dosing.

MTHFR Symptoms

Depression
Anxiety
ADD/ADHD
Miscarriage
Cardiovascular Disease
Blood Clots
Bipolar
Schizophrenia
Cancer
Midline defects
And More

Your MTHFR Results

Genes: MTHFR

You have one copy of the A1298C MTHFR variation. This is the least influential MTHFR variation and has little effect on the activity of the MTHFR enzyme. It Is still recommended to avoid synthetic folic acid.

Avoiding synthetic folic acid and consuming a diet full of green leafy vegetables is important. MethylFolate supplementation may be appropriate if you have symptoms associated with Folate deficiency.

Methylfolate Sensitivity

Some people can be sensitive to methylfolate. In this case, different forms of vitamin B9 may be used. Consider folinic acid and working with a practitioner.

Genes: COMT & VDR

There are no genetic indications for MethylFolate sensitivity.

Follow Up Testing

Genes: MTHFR & SCL19A1

Your genetics indicate near normal levels of MethylFolate. RBC Folate can still be low. homocysteineand SAM/SAH ratio tests may be ordered by your doctor.

You have a homozygous variation on one of the Folate receptors. This can lead to low levels of Folate inside the cell.

A RBC Folate test can verify your need for supplementation.

Vitamin A & Your Genetics Expanded

Vitamin A is essential for proper vision, growth, immune function, and gut health. There are two types of vitamin A: retinoids and carotenoids. Carotenoids are found in orange plants, such as carrots, and are precursors to retinoids (the bioavailable form). Retinol is the active form that is required for health.

Vitamin A - BCMO1

When most people think about increasing their vitamin A levels, they typically reach for a carrot or orange-colored vegetable. However, this is a carotenoid, not a retinoid or retinol. Our bodies have to convert carotenoids into retinoids by an enzyme called BCMO1. Some people have issues in BCMO1 that slow down their ability to form retinol from beta carotene. Your test checked for five different variations that might slow down retinal formation within your body. Consider working with a provider to monitor your vitamin levels.

Low Vitamin A Symptoms

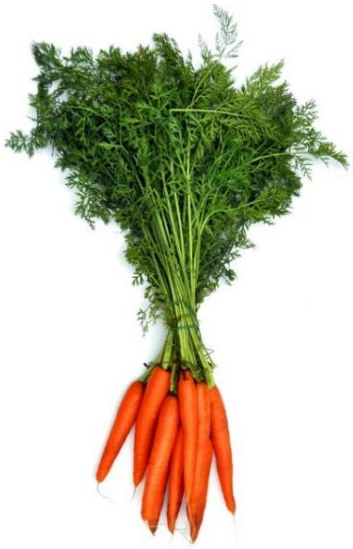
Vision issues
Infertility
Mood disorders
Skin problems
Thyroid dysfunction
Growth delays
Infections
Chronic Infections

Dietary Sources Of Retinoids

Free range eggs
Organic Heavy Cream
Shrimp
Cod-liver oil
Grass fed butter
Grass fed beef liver
Grass fed beef
Wild caught fatty fish

High Vitamin A Symptoms

Hair loss
Liver damage
Mental confusion



Your Results

Genes: BCMO1

You have multiple heterozygous variants on BCMO1. This alone should not lead to Vitamin A deficiency. Test micronutrients yearly to determine your need for supplementation.

Vitamin A Caution

Vitamin A is a fat soluble vitamin and there are studies that show excessive intake can lead to toxic levels. High levels of retinol might contribute to increased levels of heart disease and cancer. Please discuss supplementation with a trained provider and monitor blood retinol levels.

Inflammation & Your Genetics

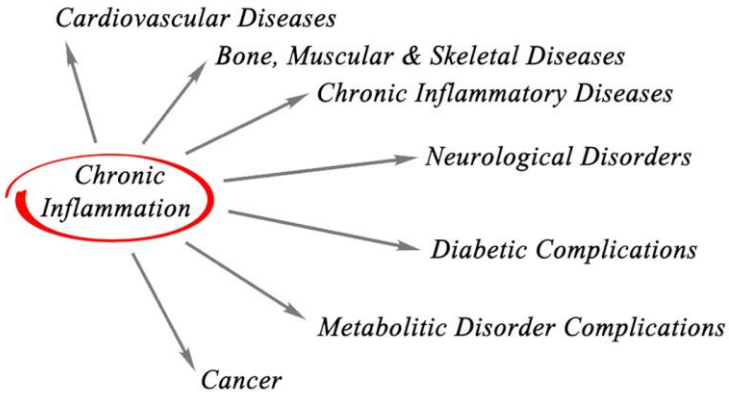
Inflammation is a natural part of our immune system that is used to protect us; however, it can become overactive. This increase in inflammation can cause many problems, such as cardiovascular, neurological, and autoimmune diseases. The Standard American Diet (SAD) is full of inflammatory foods and chemicals that add to this disease process. Your genes make you more susceptible to inflammation. Maintaining low levels of inflammation is the key to health.

Anti-Inflammatory Foods

- Blueberries
- Ginger/Turmeric
- Dark Chocolate
- Good fats
- Grass fed butter
- Free-range eggs
- Grass fed beef
- Wild caught fatty fish

Pro-Inflammatory Foods

- Sugar
- Vegetable oils
- Fried foods
- Wheat flour
- Dairy
- Bad fats
- Processed meats
- Trans fats
- Fast foods
- Conventional meats



Other Causes of Inflammation

- Lack of sleep
- Lack of exercise
- Lack of rest
- Over training
- Poor gut health
- Infection
- Toxic exposures
- Food Sensitivities

Labs Your Physician May Order

HS-CRP: High Sensitive C-Reactive Protein
ESR: Erythrocyte Sedimentation Rate
Omega 3/6 Ratios or Fatty Acid Tests
LPS: lipopolysaccharide

Generalized Inflammation

Genes: TNF

You do not have the genetic marker for increased levels of inflammation. There may still be inflammation present.

Arachidonic Acid

Genes: FADS1

You are at risk for higher levels of the pro-inflammatory fatty acid, arachidonic acid. Consider Omega 3 supplementation and avoid high Omega 6 containing foods. Omega 3:6 ratio testing might be benefitial.

Detoxification & Your Genetics

Every day, we are exposed to hundreds of toxic chemicals in our environment. Our bodies also make toxic metabolic waste that has to be filtered hourly. Many of these pathways can be slowed down by different genetic variations. This section will break down some of your variations.

Insecticide Sensitivity

Organophosphate insecticides are one of the most toxic substances on the planet. They can cause diarrhea, PDD, autism, depression, aggression, and other emotional conditions. Children exposed to these have twice the risk of autism and PDD. Children tend to be more susceptible to insecticides.

Genes: PON1

You are not genetically sensitive to pesticides. They should still be avoided. Consume organic foods and use a water filter.

Acetaminophen

Due to the prevalence of acetaminophen use, knowing your genetic potential for toxic side effects is crucial. It has been associated with liver conditions, asthma, autism, GI issues, acidosis, blood cancers, and immune system depression. These are due to lowered glutathione levels and liver involvement.

Genes: CYP2E1

You are not genetically predisposed for a toxic response to acetaminophen use. You should still consider natural alternatives, as it reduces Glutathione when used.

Glutathione

Glutathione is our master antioxidant and detoxifying molecule. Oxidative stress and toxic exposures can cause low levels of glutathione. Those with genetic predisposition to low levels may be more susceptible to the effects of environmental toxins. MTHFR and methylation SNPs can also affect glutathione levels.

Genes: GPX & GSTP1

You are genetically predisposed to reduced Glutathione production. Consider organic acid testing.

Women's Health

In women, excessive levels of estrogen can lead to many conditions, including anxiety, fertility issues, and cancer. There are certain genetic situations that might limit someone's ability to remove estrogen from the body, which will increase estrogen levels.

Estrogen Levels

Genes: COMT & CYP1B1

You have one heterozygous marker associated with conditions in estrogen metabolism. Monitor hormones with your doctor.

4-OH Estradiol

Genes: CYP 1b1

You are not genetically predisposed to metabolizing estrogen down the highly reactive 4-OH pathway. It is still recommended to monitor hormones with your doctor.

APoE & Your Genetics

Apolipoprotein E (APOE) is a gene that codes for a transport lipoprotein that carries fats and cholesterol throughout the body. There are several E types, namely E2, E3, and E4. Both E1 and E5 exist; however, they are extremely rare. Most of the population carries the E3 status, and it is considered neutral for disease risk. Everyone has two E types (example: E2/E2, E3/E4, E4/E4, E3/E4 etc.), where one type is inherited from each parent. APOE status plays a role in cardiovascular disease and Alzheimer's risk. Exercise, especially lifting, climbing, and movement-based exercises are beneficial for all types.

E4 Risk Factors

Alzheimer's Disease
Faster progression of MS
Traumatic Brain Injury
Cardiovascular disease
Unable to detoxify heavy metals

E2 Risk Factors & Benefits

Hyperlipoproteinemia Type III
Elevated Triglycerides & LDL
Insulin & Glucose Concerns
Less risk for Alzheimer's
Vertebral Fractures
Neuro-protective
Vascular disease
Psoriasis

E4 Diet Recommendations

Intermittent Fasting
Mediterranean, Low fat, or Paleo Diets
Avoid alcohol & saturated fat
Limit Seafood that is high in mercury
Limit cholesterol intake
Consider monitoring iodine levels
Consider regular sauna visits

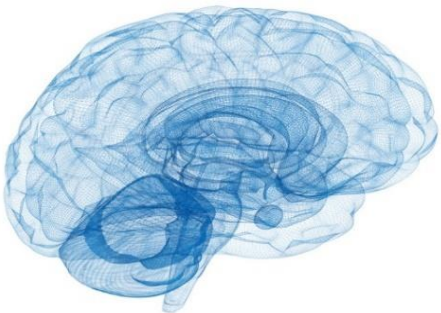
E2 Diet Recommendations

Mediterranean Diet
Low Glycemic Diet
Intermittent Fasting
Avoid Refined Carbs
Paleo Diet
Low Fat

Your APOE Status

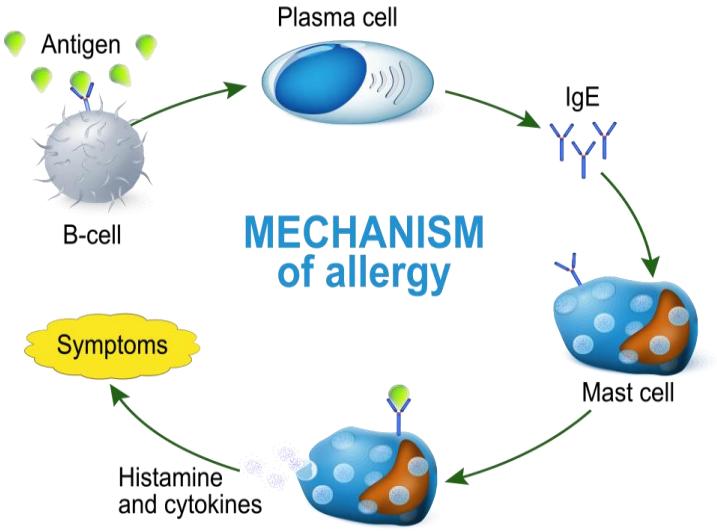
Genes: APOE

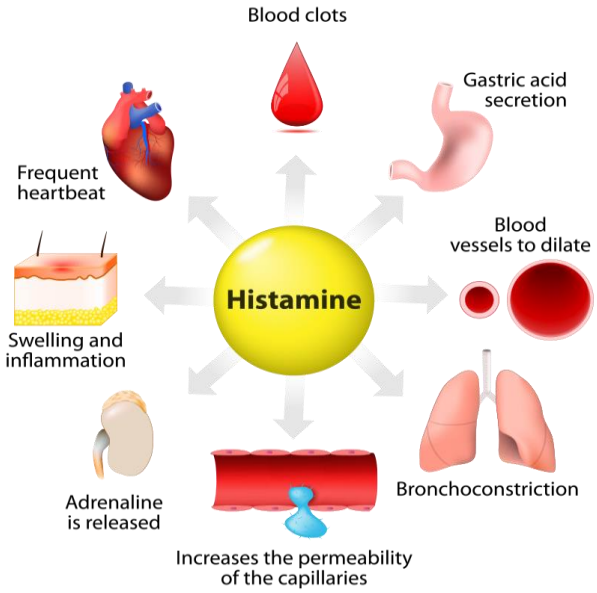
Your results: E3/E4 Please speak with your Physician.



Histamine & Your Genetics

Histamine is commonly known as an immune system chemical that is released during mast cell degranulation or when exposed to allergens. However, it is also a neurotransmitter in the brain and plays a role in digesting food in the stomach. In humans, histamine is broken down by two main pathways, Histamine N-Methyltransferase (HNMT) and Diamine Oxidase (DAO/AOC1). Excessive histamine can cause numerous issues in the body, and there are some genetic predispositions that enhance these issues.





High Histamine Foods

- Alcohol/Ferments
- Citrus Fruits
- Dried Fruits
- Soured Foods
- Smoked Meats
- Aged Cheese
- Tomatoes
- Walnuts
- Cashews
- Peanuts
- Spinach
- Eggplant
- Shellfish
- Bananas
- Wheat
- Strawberries
- Beans
- Chocolate
- Food Dyes
- Food Additives

High Histamine Symptoms

- Headaches
- Migraines
- Digestive Issues
- Anxiety
- Eczema
- Other Skin conditions
- Nasal Congestion
- Fatigue/Adrenal Fatigue
- Irregular Menstrual Cycles
- Blood Pressure Issues
- Nasal Congestion

DAO (AOC1)

The DAO Enzymes is responsible for breaking down dietary histamine and histamine outside of your cells. It requires adequate levels of copper and can be inactivated by certain drugs and curcumin.

Genes: DAO

You are not genetically predisposed to reduced DAO enzyme activity.

HNMT

HNMT is responsible for breaking down histamine inside of your cells and is common in asthma. This enzyme requires adequate levels of SAMe from the methylation cycle.

Genes: HNMT

You are not genetically predisposed to increased cellular histamine.

MAO & Your Genetics

*Please see your physician before making nutritional changes.

Monoamine oxidase (MAO) has two types, A and B, and plays a role in the oxidation of neurotransmitters. MAOA is chiefly responsible for the oxidation of serotonin and norepinephrine, while MAOB oxidizes phenylethylamine. Both oxidize dopamine. Each genetic variation of MAO creates different outcomes of the enzyme. MAO is also found on the X chromosome, so males who inherit the variation are technically hemizygous. Our algorithm, however, reports it as homozygous since we do not know the sex of each person performing this test. If your results suggest you have decreased MAO activity, it is suggested that you avoid cheese and other fermented/aged foods that are high in tyramine.

MAO-A (RS6323)	MAO-A (RS72554632)	MAO-B
You have the slower form of the MAO-A Enzyme. If you have symptoms associated with Slow MAO, consider the nutritional support listed below.	No variants detected. This should not cause symptoms.	Possible decrease in MAO activity. Follow SLOW MAO suggestions below. Possible elevation in histamine. Use Low Tyramine Diet if symptoms occur.

Fast MAO

A fast MAO enzyme will significantly decrease neurotransmitter levels and create symptoms of deficiency. Depression, anxiety, and low mood are common symptoms. Your practitioner may want to try nutraceuticals like St. Johns Wort, 5-HTP, tyrosine, resveratrol, B vitamins, sun and light exposure to help support a healthy mood.

Low Serotonin

Anxiety / Depression
Insomnia / Paranoia
Loss of pleasure
Weight Issues
Inner rage
Support:
5-HTP & St. John's Wort

Low Dopamine

Depression / Hopelessness
Lack of Motivation
Brain Fog/ Fatigue
Weight Issues
Low Libido
GI Issues
Support: Tyrosine, Bacopa

Low Norepinephrine

Brain Fog
Depression
Low Blood Pressure
Adrenal Fatigue
Support: Vit. C,
Copper Balancing, Tyrosine

Low PEA

Brain Fog
Depression
Difficulty Paying
Attention
Incomplete Thoughts
Support: B6
DL-phenylalanine

Slow MAO

A slow MAO enzyme will allow for greater levels of neurotransmitters and cause symptoms of excess. Increased aggression and lack of empathy are common. In general, it is recommended to avoid caffeine, smoking, and stress when possible. Utilizing meditation techniques, trying a low tyramine diet, and insuring proper B2, lithium orotate, zinc and hormone levels are all possible options to support a healthy mood.

High Serotonin

Headaches
Diarrhea
Muscle Twitching
Confusion
Seizures
High Blood Pressure
Support: B2, B5

High Dopamine

Excessive Energy
ADD/ADHD
Anxiety
Agitation
Insomnia
Addiction
Support: B2, Methylation &

High Norepinephrine

Anxiety
Heart Palpitations
Sweating
Constipation
Support:
Methylation & B2

High PEA

Mind Racing
Insomnia
Anxiety
Schizophrenia
Support: Methylation &
L-Threonine

COMT & Your Genetics

*Please see your physician before making nutritional changes.

Catechol-O-methyltransferase (COMT) is a gene that creates an enzyme that breaks down dopamine, norepinephrine, epinephrine, and estrogen. These chemicals play a major role in mood, stress response, and productivity. Estrogen needs to be balanced and reduced appropriately to avoid issues. COMT does require the methylation cycle, with SAMe and magnesium being required in adequate amounts. It has been observed that individuals with slower COMT tend to be sensitive to methyl donors. In these cases, non-methylated vitamins like Folinic Acid and Hydroxocobalamin might be better options. People who are sensitive to these tend to have mood swings and anger issues. It has also been observed that carriers of the VDR-TAQ variation have additional risks.

COMT V158M	COMT H62H	VDR-TAQ
You have a fast COMT. Warrior tendencies. If you have symptoms associated with Fast COMT, consider the nutritional support listed below.	No variants detected. This should not cause symptoms.	Possible increased Dopamine sensitivity, which can worsen slow COMT and help fast COMT symptoms.

Fast COMT

Fast versions of the COMT enzyme are associated with decreased levels of neurotransmitters like dopamine. People with this have been shown to have higher pain thresholds, are capable of operating under adverse stress (The Warrior Gene), and have lower levels of anxiety.

Low Dopamine

Depression	Constipation	**Support**:
Lack of Motivation	GERD	Tyrosine
Fatigue	Muscle Cramps	Bacopa
Focus Issues		

Low Epinephrine

Depression	Migraines	**Support**:
Restless Leg	Sleep Disorders	Methionine
		Tyrosine

Low Norepinephrine

Focus Issues	Brain Fog	**Support**:
Depression	Low Blood Pressure	Tyrosine
		Vit. C
		Copper Balancing

Slow COMT

Slower versions of the COMT enzyme are associated with increased levels of neurotransmitters like dopamine. This has been shown to lower pain thresholds, increase a person's sensitivity to stress, and increase anxiety (The Worrier Gene). However, these individuals typically have an advantage at memory and attention based tasks.

High Dopamine

ADD/ADHD	Insomnia	**Support**:
Anxiety	Addiction	Riboflavin
Excessive Energy	Mania	Vit. C
		Methylation

High Epinephrine

Anxiety	Weight Loss	**Support**:
Sweating	Constipation	Adaptogens
Heart Palpitations		Phosphatidylserine

High Norepinephrine

Anxiety	Sweating	**Support:**
Heart Palpitations	Constipation	Methylation, Riboflavin

Your Genetic Summary

B12 Levels	• You have a risk for low serum Vitamin B12 levels. Use organic acid or homocysteine testing to verify your need for B12.
Methyl-B12	• You have one heterozygous variation on the MTRR enzyme. This could create a need for B12 supplementation. Use organic acid or homocysteine testing to verify your need for B12.
B12 Sensitivity	• There are no genetic indications for Methylcobalamin sensitivity.
Adeno-B12	• There are no genetic indications that you need Adenosylcobalamin supplementation. Adenosylcobalamin could be used in cases of fatigue.
Vitamin A	• You have multiple heterozygous variants on BCMO1. This alone should not lead to Vitamin A deficiency. Test micronutrients yearly to determine your need for supplementation.
Vitamin D	• There are no indications of genetic Vitamin D metabolism issues.
Folate/MTHFR	• You have one copy of the A1298C MTHFR variation. This is the least influential MTHFR variation and has little effect on the activity of the MTHFR enzyme. It Is still recommended to avoid synthetic folic acid.
Folate Sensitivity	• There are no genetic indications for MethylFolate sensitivity.
Dietary Histamine	• You are not genetically predisposed to reduced DAO enzyme activity.
Cellular Histamine	• No variant detected that increases cellular histamine.
DHA Fish Oil	• You are not genetically predisposed to a deficiency in Omega 3 Fatty Acids. Regular intake of fish or omega-3 oils should be consumed.
Phos-Choline	• You have two of the four genetic markers associated with Phosphatidylcholine deficiency. Consider supplementing if support is needed for brain or liver health and pregnancy.
Arachidonic Acid	• You are at risk for higher levels of the pro-inflammatory fatty acid, arachidonic acid. Consider Omega 3 supplementation and avoid high Omega 6 containing foods. Omega 3:6 ratio testing might be benefitial.
Inflammation	• You do not have the genetic marker for increased levels of inflammation. There may still be inflammation present.
Estrogen levels	• You have one heterozygous marker associated with conditions in estrogen metabolism. Monitor hormones with your doctor.
Bad Estrogen	• You are not genetically predisposed to metabolizing estrogen down the highly reactive 4-OH pathway. It is still recommended to monitor hormones with your doctor.
Pesticides	• You are not genetically sensitive to pesticides. They should still be avoided. Consume organic foods and use a water filter.
Glutathione	• You are genetically predisposed to reduced Glutathione production. Consider organic acid testing.
Probiotic	• There are no probiotic recommendations based on your results. See the box below if there are additional recommendations.
Secretor Status	• FUT2 Secretor. There are no probiotic recommendations associated with this variant.

SNP Report

Gene	RS#	Result	Client	Minor	Short Description
AHCY-01	rs819147	-- Wild Type	TT	C - 31%	No genetic cause for low homocysteine or glutathione.
APOE	rs429358	-+ Heterozygous	CT	C - 15%	See APOE page for details. If rs7412 is T = E1 (Rare) If RS7412 is C = E4
APOE	rs7412	-- Wild Type	CC	T - 8%	See APOE page for details. If rs429358 is C = E4 If rs 429358 is T = E3 (Normal)
BCMO1	rs11645428	-+ Heterozygous	GA	A - 15%	Genetic cause for Vitamin A deficiency. See Vitamin A page for details.
BCMO1	rs12934922	-- Wild Type	AA	T - 22%	No genetic cause for Vitamin A deficiency.
BCMO1	rs6564851	-+ Heterozygous	GT	G - 47%	Genetic cause for Vitamin A deficiency. See Vitamin A page for details.
BCMO1	rs7501331	-+ Heterozygous	CT	T - 21%	Genetic cause for Vitamin A deficiency. See Vitamin A page for details.
BCMO1	rs6420424	-- Wild Type	GG	A - 43%	No genetic cause for Vitamin A deficiency.
CBS	rs28934891	-- Wild Type	CC	T - .00%	No genetic cause for reduced CBS enzyme activity.
CBS	rs4920037	-- Wild Type	GG	A - 13%	No genetic cause for reduced CBS enzyme activity.
CBS	rs2851391	-- Wild Type	CC	T - 38%	No genetic cause for reduced CBS enzyme activity.
CBS 360	rs1801181	++ Homozygous	AA	A - 29%	Genetic cause for upregulated CBS enzyme activity. Test homocysteine.
CBS 699	rs234706	-- Wild Type	GG	A - 19%	No genetic cause for upregulated CBS enzyme activity.
COMT 61 P199P	rs769224	-- Wild Type	GG	A - 2%	No genetic cause for down regulation of COMT.
COMT H62H	rs4633	-- Wild Type	CC	T - 237%	No genetic cause for down regulation of COMT.
COMT L136L	rs4818	-+ Heterozygous	GC	G - 29%	No genetic cause for down regulation of COMT.
COMT V158M	rs4680	-- Wild Type	GG	A - 36%	Fast COMT (Warrior) gene. See COMT page for details.
CYP1B1 L432V	rs1056836	-+ Heterozygous	CG	G - 42%	No genetic cause for elevated 4-OH estradiol. Test hormones with your doctor.
CYP2E1 *6	rs6413432	-- Wild Type	TT	A - 16%	No genetic cause for NAPQI toxicity from Acetaminophen.
DAOA/DAAO	rs3741775	-+ Heterozygous	CA	C - 31%	Genetic risk for Schizophrenia. Test Vitamin B2 levels. Consider SAMe.
DAO (AOC1)	rs10156191	-- Wild Type	CC	T - 31%	No genetic cause for reduced DAO enzyme activity.
DHFR	rs1643649	-+ Heterozygous	CT	C - 22%	Genetic cause for low tetrahydrofolate. Avoid Bactrim, EGCG,and grape seed.
Factor 5	rs6025	-+ Heterozygous	CT	T - .00%	Genetic cause for 4x risk of thrombosis. Consult with your doctor.
FADS1	rs174548	-- Wild Type	CC	G - .00%	No genetic cause for phosphatidylcholine deficiency.
FADS1(MYRF)	rs174537	-- Wild Type	GG	T - 30%	Genetic cause for high Arachidonic Acid levels. Limit Omega 6 foods.
FADS2	rs1535	-- Wild Type	AA	G - 32%	No genetic cause for decreased DHA production. Associated with High IQ.
FOLR2	rs651933	-- Wild Type	GG	A - 45%	No genetic cause for intracellular folate deficiency.
FUT2	rs602662	-- Wild Type	GG	A - 32%	Genetic cause for low serum B12 levels. See B12 page for details.
FUT2	rs492602	-- Wild Type	AA	G - 32%	No genetic cause for B12 deficiency.
FUT2 W143X	rs601338	-- Wild Type	GG	A - 32%	Norovirus susceptibility. Secretor status.
G6PD	rs1050828	-- Wild Type	CC	T - 3%	No genetic need to avoid IV Vitamin C & H202.
G6PD	rs1050829	-- Wild Type	TT	C - 9%	No genetic need to avoid IV Vitamin C & H202.
G6PD	rs5030868	-- Wild Type	GG	A .00%	No genetic need to avoid IV Vitamin C & H202.
GPX1	rs1050450	-- Wild Type	GG	A - 2%	No genetic cause for glutathione deficiency and heavy metal toxicity.
GSTP1	rs1138272	-- Wild Type	CC	T - 3%	No genetic cause for inability to detoxify.
GSTP1	rs1695	-+ Heterozygous	GA	G - 35%	Genetic cause for inability to detoxify. Consider glutathione supplementation.
HFE	rs1799945	-- Wild Type	CC	G - 7%	No hemochromatosis risk
HFE	rs1800562	-- Wild Type	GG	A - 1%	No hemochromatosis risk
HFE	rs1800730	-- Wild Type	AA	T - .00%	No hemochromatosis risk
HNMT	rs1050891	-- Wild Type	AA	G - 20%	No genetic cause for elevated serum levels of histamine.
LRRK2	rs34637584	-- Wild Type	GG	A - .00%	No genetic risk of Parkinson's Disease.

Gene	RS#	Result	Client	Minor	Short Description
MAOA T1410C	rs1137070	-- Wild Type	CC	T - 44%	Genetic cause for reduced MAO activity & elevated serotonin levels.
MAOA	rs6323	-- Wild Type	TT	G - 37%	Genetic cause for SLOW MAO-a status. See MAO page for details.
MAOA	rs72554632	-- Wild Type	CC	T - .00%	No genetic cause for MAO deficiency.
MAOB	rs1799836	++ Homozygous	CC	C - 45%	Genetic cause for decreased MAO-b activity. See MAO page for details.
**Notice: MAO is a X linked gene and is only passed down from the maternal line. Male Children are technically "hemizygous."					
MAT1A R264H	rs72558181	-- Wild Type	CC	T - .00%	No genetic cause for hypermethioniemia.
MMAB	rs2287182	-- Wild Type	CC	T - 13%	No genetic cause for methylmalonic acidemia.
MTHFS	rs6495446	-+ Heterozygous	CT	T - 29%	Genetic cause for folinic acid or Leucovorin avoidance. See Folate page.
MTHFD1	rs2236225	++ Homozygous	AA	A - 34%	Potential cause for 5,10 methylenetetrahydrofolate deficiency.
MTHFR A1298C	rs1801131	-+ Heterozygous	GT	G - 25%	Genetic cause for Folate deficiency. See Folate page for details.
MTHFR C677T	rs1801133	-- Wild Type	GG	A - 24%	No genetic cause for Folate deficiency.
MTR	rs1805087	-- Wild Type	AA	G - 21%	Decreased activity of MTR. Methyl B12 may be useful if Homocysteine is high.
MTRR	rs1801394	-+ Heterozygous	GA	G - 36%	Genetic cause for B12 deficiency. See B12 page for details. Test homocysteine.
MTRR	rs1532268	-- Wild Type	CC	T - 27%	No genetic cause for B12 deficiency.
MUT	rs1141321	-+ Heterozygous	CT	T - 26%	Genetic cause for methylmalonic acidemia. Consider adenosylcobalamin.
MUT	rs9369898	-+ Heterozygous	GA	G - 40%	Genetic cause for methylmalonic acidemia. Consider adenosylcobalamin.
NOS3	rs1799983	-+ Heterozygous	GT	T - 17%	Genetic risk for small artery disease due to low Nitric Oxide. Use l-arginine.
NOS3	rs2070744	-+ Heterozygous	CT	C - 23%	No genetic cause for cardiovascular disease.
NQO1	rs1800566	-- Wild Type	GG	A - 28%	No genetic cause for increased oxidative stress.
PEMT	rs4244593	-- Wild Type	GG	T - 42%	No genetic cause for phosphatidylcholine deficiency.
PEMT	rs4646406	++ Homozygous	AA	A - 28%	Genetic cause for phosphatidylcholine deficiency. Consider supplementation.
PEMT	rs7946	++ Homozygous	TT	T - 30%	Genetic cause for phosphatidylcholine deficiency. Consider supplementation.
PON1 Q192R	rs662	-- Wild Type	TT	C - 45%	No tendency for insecticide sensitivity.
Prothrombin (F2)	rs1799963	-- Wild Type	GG	A - .00%	No genetic cause for thrombosis or cerebral stroke.
SHMT1	rs1979277	-- Wild Type	GG	A - 23%	No genetic cause for inadequate methylation.
SLC19A1	rs1051266	++ Homozygous	TT	T - 48%	Genetic cause for Folate deficiency. Test RBC Folate.
SOD1	rs2070424	-- Wild Type	AA	G - 24%	No genetic cause for high levels of SOD1.
SOD1	rs4998557	-- Wild Type	GG	A - 33%	No genetic cause for oxidative stress.
SOD2	rs2758331	-+ Heterozygous	CA	A - 33%	Genetic cause of oxidative stress. Consider SOD supplementation.
SOD2	rs4880	-+ Heterozygous	AG	G - 41%	Genetic cause of oxidative stress. Consider SOD supplementation.
SOD3	rs1799895	-- Wild Type	CC	G - 2%	No genetic cause for oxidative stress.
SUOX(A628C)	rs7297662	-+ Heterozygous	GA	A - 47%	Possible sulfite oxidase deficiency. Use molybdenum supplementation.
SUOX(S370S)	rs773115	-- Wild Type	CC	G - .00%	No genetic cause for sulfite oxidase deficiency.
TCN1	rs526934	-- Wild Type	AA	G - 19%	No genetic cause for B12 deficiency.
TCN2	rs1801198	-- Wild Type	CC	G - 42%	No genetic cause for B12 deficiency.
TNF	rs1800629	-- Wild Type	GG	A - 9%	No genetic cause for high inflammation.
VDR TAQ	rs731236	-- Wild Type	AA	G - 38%	No genetic cause for Vitamin D deficiency.
VDR-BSM	rs1544410	-- Wild Type	CC	T - 29%	No genetic cause for Vitamin D deficiency.
VDR-FOK	rs2228570	-- Wild Type	GG	A - 32%	No genetic cause for Vitamin D deficiency.

Client: Your genotype. Minor: The genotype that is found least in nature.
Wild Type: The genotype that is found most often in nature, this is reported as green. This isn't always ideal.
Homozygous: This means you tested for both copies of the minor type allele. This typically has more severe issues.
Heterozygous: : This means you tested for one copy of the minor allele and one copy of the wild type allele.

SNP Information

AHCY-01

rs819147	-- Wild Type

Adenosyl homocysteinase
This enzyme is responsible for the breakdown of the amino acid, methionine. Health risk with this gene mutation is hypermethioninemia, which is associated with a short stature, low homocysteine, and low glutathione. Consider testing.

APoE

rs429358	-+ Heterozygous
rs7412	-- Wild Type

Your results: E3/E4
Please speak with your Physician.

The APoE gene codes for a protein responsible for moving cholesterol and fat around the body. Issues here can result in cardiovascular conditions and is a risk factor for Alzheimer's and dementia. APoE status is technically defined by these two SNPs, **rs429358** and rs7412.

e1 = rs429358(C or +) & rs7412 (T or +)
e2 = rs429358(T or -) & rs7412(T or +)
e3 = rs429358(T or -) & rs7412(C or -)
e4 = rs429358(C or +) & rs7412(C or -)

E1 is extremely rare.
E2's have a greater risk for vascular disease and hyperlipoproteinemia, cholesterol and triglyceride levels can be 2x to 3x higher, and is implicated in Parkinson's disease. E2's tend to respond well to high carb, low fat diets.
E3's is the neural type and is found in humans around 78% of the time.
E4's is implicated in Alzheimer's disease, impaired cognitive function, reduced hippocampal volume within the brain, faster progression of MS and associated with higher levels of Vit. D. E4's tend to respond well to hormone replacement therapy and high carb, low fat diets. Discuss this mutation with your PCP or cardiologist.

BCMO

rs11645428	-+ Heterozygous
rs12934922	-- Wild Type
rs6564851	-+ Heterozygous
rs7501331	-+ Heterozygous
rs6420424	-- Wild Type

beta-carotene oxygenase 1

The BCMO gene codes for the enzyme responsible for creating Vitamin A from dietary carotenoids. SNP's here can slow down the conversion of beta carotene from the diet into Vitamin A. These individuals can have low vitamin A levels while eating a diet rich in carotenoids. Interesting enough, this SNP can cause someone's skin to turn orange if they eat a large amount of beta carotene.

These SNPs may lead someone to needing Retinyl Palmitate to bypass this conversion problem. This becomes increasingly useful during times of sickness because of the necessity for vitamin A for the immune system. Discuss Vitamin A supplementation with your physician or nutritional consultant.

CBS

rs28934891	-- Wild Type
rs4920037	-- Wild Type
rs2851391	-- Wild Type
rs1801181 (360)	++ Homozygous
rs234706 (699)	-- Wild Type

cystathionine beta-synthase
CBS is a gene that encodes the enzyme cystathionine beta-synthase, which is responsible for using vitamin B6 to convert the amino acids homocysteine and serine to cystathionine. Health conditions associated with this gene include homocystinuria. When homocysteine levels are affected, we see skeletal abnormalities, cognitive issues, eye problems, and abnormal blood clotting. It is important to get your homocysteine levels checked yearly, especially if this mutation is present. It is not a common practice for physicians to order homocysteine levels, so one must ask for it. Also, consider getting your B6 levels checked yearly.
CBS SNPs are typically considered to slow down the CBS enzyme, potentially causing elevated homocysteine and low levels of the master antioxidant glutathione. However, many consider RS1801181 (CBS 360) and RS234706 (CBS 699) to be up regulated SNPs. If CBS is truly upregulated it can cause excess ammonia levels and sulfite levels. Gut health becomes paramount in ammonia removal. Many products on the market are designed to increase butyrate within the colon. Butyrate helps remove ammonia from the body and is commonly assessed on extensive stool testing. As well in the case of a CBS upregulation, the neurotoxic chemical, sulfite can increase. Sulfite is broken down by the enzyme SUOX. Maximized Genetics is one of the only labs who look at SNPs within the SUOX gene. These SNPs could potential increased sulfite levels even further.
CBS upregulations is also clinically seen with toxic compounds and when the body's glutathione levels become low. Focusing on lowering toxin exposure and gut health should be highly considered.

COMT

rs769224	-- Wild Type
rs4633	-- Wild Type
rs4818	-+ Heterozygous
rs4680	-- Wild Type

COMT is a gene (with multiple forms) that codes for the enzyme catechol-O-methyltransferase, which is specifically used to break down neurotransmitters in the brain, kidneys, liver, and blood. These neurotransmitters play an important role in the pre-frontal cortex of the brain, where impulsivity, planning, short term memory, and emotions are controlled. Dopamine and norepinephrine levels are particularly affected by mutations in this gene.
Health concerns related to this gene mutation generally revolve around mental health disorders. Particularly, schizophrenia has been related to a mutation in the COMT V158M snp. Other disorders that may be related are bipolar disorder, eating disorders, OCD, panic disorders, and anxiety.
Research suggests that this gene can be used to choose various medications related to ADHD.
Pain response may also be related to this mutation.
If this gene mutation is present, note that stress is a driver for inflammation and disease. Work with your functional medicine practitioner to discuss lowering inflammation throughout the body by creating a healthy diet and lifestyle. Also, consider an organic acids test to look for neurotransmitter levels.

CYP1b1

rs1056836	-+ Heterozygous

Cytochrome P450 family 1 subfamily B member 1
This gene encodes for an enzyme that is responsible for detoxing drugs and fats. Mutations in this gene may lead to early onset glaucoma.
Estrogen is also broken down by this enzyme. RS1056836, when ++ is an upregulation of it's ability to produce the estrogen metabolite 4-oh-E2. This metabolite is associated with breast cancer. Discuses having a 4-oh-E2 level checked.

CYP2E1

rs6413432 (*6)	-- Wild Type

Cytochrome P450 family 2 subfamily E member 1.
This gene encodes for an enzyme that is responsible for detoxifying drugs like acetaminophen (Tylenol), ethanol, chlorzoxazone, and sevoflurane. These two SNPs, *5b and *6, increase the activity of the CYP2E1 enzyme. This enzyme speeds up conversion of acetaminophen into a toxic metabolite, NAPQI. Numerous studies show the connection between acetaminophen and liver damage, mitochondrial conditions, depletion of glutathione, neuronal death, ADHD, asthma, autism, kidney failure, gastroschisis, blood cancers, and numerous other conditions. Based upon current research, acetaminophen should be used with caution. Persons with these two mutations, could be at increased risk for oxidative/toxic damage from acetaminophen.

DAOA/DAAO

rs3741775	-+ Heterozygous

DAO/DAAO (rs3741775) should not be confused the DAO(AOC1) gene. It is common amongst "genetic experts" and websites to get these confused. DAAO is D-Amino-Acid Oxidase and breaks down D-Amino acids, especially targeting D-Serine. The DAO(AOC1) enzyme targets extracellular histamine and is a completely different gene.

The DAAO SNP is assumed to be an upregulation, meaning the enzyme is faster than normal. This creates an issue with lack of D-Serine. D-Serine is a NMDA receptor agonist and this SNP can result in a less NMDA activity which has been associated with schizophrenia. There can also be disruptions in glutamate receptor stimulation as well. And lastly, it's believed that SNPs here can potentially increase oxalate production. If symptoms of high oxalates are present you may want to consider a low oxalate diet.

People with DAAO sometimes respond favorably to Piracetam (500mg 2x a day), Vitamin C, and SAMe. And once again, do not get this SNP confused with AOC1.

DAO(AOC1)

rs10156191	-- Wild Type

DAO/AOC1 is an enzyme that degrades extracellular histamine. The SNP tested is assumed to downregulate of the activity of DAO, per current research. This can result in increased levels of histamine and excess histamine symptoms. The DAO enzyme is commercially available for supplementation and should be considered if laboratory histamine ranges are elevated along with this SNP.
DAO supplementation has the potential to created ammonia and hydrogen peroxide. Consider catalase and butyrate supplementation if this becomes a problem. DAO is a copper and B6 dependent enzyme so these should be evaluated as well.
Finally, a low histamine diet should be discussed with a physician or nutritional expert.

DHFR

rs1643649	-+ Heterozygous

Dihydrofolate reductase
This gene mutation is associated with megaloblastic anemia, which can cause seizures and learning difficulties. Avoid the use of folic acid and the antibiotic Bactrim. Folinic acid can help bypass this enzyme SNP.

Factor 5

rs6025	-+ Heterozygous

Factor 5
Mutation in this gene leads to possible venous thromboembolism. Warning: tamoxifen used for breast cancer treatment in a female with this gene mutation may lead to thromboembolism.

FADS1

rs174548	-- Wild Type
rs174537	-- Wild Type
rs1535	-- Wild Type

FADS1 (rs174548, rs174537)
This gene is used to create fatty acid unsaturation. If present, low levels of phosphatidylcholine may be present. Phosphatidylcholine is needed as a precursor to acetyl-choline, which is extremely important for neurological function. Choline, in general, is considered beneficial for memory, motivation, and muscle function. As an essential part of every cell membrane, people with this gene mutation may want to consider supplementation of phosphatidylcholine.
A second FADS1 gene (rs174537) is considered crucial in heart disease. If this gene is present, avoid consumption of Omega 6 fatty acids, as they will increase inflammation. Examples of Omega 6 fatty acids include vegetable oils (canola, sunflower, soy, corn), and grains

FADS2

rs1535	-- Wild Type

FADS2 (RS1535)
This is a gene to create fatty acid unsaturation. It is associated with lower DHA (Omega 3) levels. It is also connected to higher IQ in breastfed babies and hyperactivity in children. If this gene mutation is present, consider taking a high quality fish oil, specifically DHA.

FOLR2

rs651933	-- Wild Type

FOLR2
Folate receptor beta
This gene codes folate receptors on the cell membrane. It has a high affinity for folic acid, which can block the reduction of bioavailable folate. Research is currently limited about this SNP, but some cite its association to neural tube defects, rheumatoid arthritis, and cerebral folate transport deficiency. FOL2 receptors are found in high quantity within the placenta.

FUT2

rs602662	-- Wild Type
rs492602	-- Wild Type
rs601338	-- Wild Type

FUT2
fucosyltransferase 2
This gene is associated with vitamin B12 levels. When this mutation is present, a person may have a deficiency or an increase in B12. If a person specifically has a mutation in rs601338, he/she may be immune from Norovirus, but it creates potential gut dysbiosis. Consider B12 deficiency testing and supplementation with methylcobalamin.

G6PD

rs1050828	-- Wild Type
rs1050829	-- Wild Type
rs5030868	-- Wild Type

glucose-6-phosphate dehydrogenase
The enzyme that is encoded from this gene helps protect the red blood cell from oxidative stress. Mutations in this gene create glucose-6-phosphate dehydrogenase deficiency, which can lead to hemolytic anemia and/or neonatal jaundice. Fava beans and IV vitamin C and Hydrogen Peroxide (H202) need to be avoided with this mutation. Discuss this with your physician to see if certain medications will make this worse.

GPX1

rs1050450	-- Wild Type

glutathione peroxidase 1
With a mutation in this SNP, one has the potential for glutathione deficiency. Glutathione is protective against oxidative cellular damage, but when deficient, multiple diseases can occur. Research states that the following diseases can be linked to this gene mutation: brain tumors, breast cancer, osteoporosis, selenium deficiency induced osteoporosis, and cardiovascular risk associated with diabetes. Consider glutathione supplementation.

GSTP1

rs1138272	-- Wild Type
rs1695	-+ Heterozygous

Glutathione S-Transferase Pi 1 is a gene responsible for the pi class of enzymes responsible for detoxification of xenobiotics in the body. With a mutation in this gene, a person may be more susceptible to cancers. Consider glutathione testing and supplementation.

HFE

rs1799945	-- Wild Type
rs1800562	-- Wild Type
rs1800730	-- Wild Type

This class of genes is responsible for hereditary hemochromatosis, which causes difficulty with iron metabolism. This gene creates hepcidin, which regulates iron. Symptoms of hemochromatosis include issues with joints, skin, liver, heart, thyroid, and reproductive organs. People with this gene mutation may not notice issues until their 40's or later. Check iron levels, liver enzymes, and other standard lab work on a regular basis.

HNMT

rs1050891	-- Wild Type

Histamine N-Methyltransferase
This gene encodes for the enzyme histamine n-methyltransferase, which is found in cytosol and uses a major methyl donor. In the brain, histamine is a major neurotransmitter, and in the gut is controlled by DAO. Health conditions associated with a mutation in this gene include asthma and mental retardation. In ADHD children, certain food additives can be troublesome, including all food dyes and sodium benzoate.

LRRK2

rs34637584	-- Wild Type

This gene is associated with the development of Parkinson's. If this gene is present, research suggests a 15% chance of developing Parkinson's by age 60, 21% by age 70, and 32% chance by age 80. A high fat, low carb diet may provide some protection. We suggest getting nutrient levels tested on a yearly basis, and pay close attention to B-vitamin levels. Work with a provider who can come up with a specific diet plan, and order the Nutrigenomic Panel to understand other genetic risks involved.

MAO-A

rs1137070	-- Wild Type
rs6323	-- Wild Type
rs72554632	-- Wild Type

MAO-B

rs1799836	++ Homozygous

MAOA & MAOB
Monoamine Oxidase A and B are enzymes involved in the breakdown of neurotransmitters (serotonin, dopamine, norepinephrine, etc.). When the rs72554632 variant is present, a person may have monoamine oxidase deficiency (aka Brunner Syndrome), which causes a build of neurotransmitters in the brain. This can lead to symptoms such as impulsivity, aggression, depression, and other psychiatric issues. Considered a male issue, boys tend to be diagnosed as autistic or ADHD. Other concerns related to this gene mutation include weak muscles, repetitive hand movements, behavioral and/or developmental delays, and panic disorders (especially in females). Cheese appears to make symptoms worse. Research suggests that these genes may be connected to Parkinson's as well. If this gene mutation is present, consider organic acid testing, neurotransmitter testing, and nutrient deficiency testing on a yearly basis. The gut-brain axis needs to be monitored closely.
We report the G allele for rs 6323 as the minor allele per dbsnp and the research we reviewed indicated the G allele as being the minor and more problematic allele (increases the MAO activity). This is a highly researched SNP and some other reporting and testing companies for some reason report the opposite for this SNP.

MAT1A

rs72558181	-- Wild Type

Methionine Adenosyl transferase 1A gene mutations may create hypermethioninemia, a condition that can have significant neurological delays. Symptoms include muscle weakness, liver problems, delay in motor skills, and a cabbage smell from breath and sweat. With a mutation in this gene, diets high in protein cause a build up in the amino acid, methionine. Many people may not even realize they have this condition. Consider a lower protein diet as it relates to the rest of your genetic profile.

MMAB

rs2287182	-- Wild Type

A gene mutation here causes methylmalonic academia, which is a condition that creates difficulty in breaking down proteins and lipids. Adenosylcobalamin is the active mitochondrial form of B12 needed to create the enzyme methylmalonyl CoA mutase. Motor and other developmental delays may be of immediate concern with this genetic mutation, and long term mitochondrial issues may occur. Consider taking Adenosylcobalamin for your B12 needs.

MTHFS

rs6495446	-+ Heterozygous

Methenyltetrahydrofolate Synthetase
People with this gene mutation should avoid folinic acid.

MTHFD1

RS2236225	++ Homozygous

methylenetetrahydrofolate dehydrogenase, cyclohydrolase and formyltetrahydrofolate synthetase 1
If a mutation in this gene is present, a person may have low serum levels of folate (vitamin B9). Folic acid should be avoided. Health risks include neural tube defects and colorectal cancer. Levels are made worse with additional MTHFR mutations.

MTHFR

rs1801131 A1298C	-+ Heterozygous
rs1801133 C677T	-- Wild Type

methylenetetrahydrofolate reductase
The MTHFR gene is responsible for coding the enzyme that processes amino acids, namely homocysteine and methionine, through the conversion of various forms of folate (vitamin B9).
Several conditions have been associated with mutations in the MTHFR gene: homocystinuria, anencephaly, spina bifida, glaucoma, high blood pressure, heart disease, psychiatric disorders, and various cancers. Research in varied and conflicting as it relates to MTHFR mutations and their impact on disease. While one can find many associations between the gene and certain conditions, reproducibility of such research is virtually non-existent.
When homocysteine levels are affected, we see skeletal abnormalities, cognitive issues, eye problems, and abnormal blood clotting. This alone may be attributed to a link between MTHFR and health conditions. It is important to get your homocysteine levels checked yearly, especially if this mutation is present. It is not a common practice for physicians to order homocysteine levels, so one must ask for it.
As a part of the methylation cycle, MTHFR mutations can affect hundreds of chemical conversions throughout the body. Multiple nutrients are involved in this cycle, but close attention is given to Folate (B9), Cobalamin (B12), P5P (B6), and Riboflavin (B2). It is important to check nutrient status yearly as well. Work with a practitioner who is proficient in both serum and intracellular lab work.
Currently, attention is given to the two main forms of MTHFR, 677 and 1298, with significantly more importance being placed on 677. There are many more forms of MTHFR, but they have no clinical significance yet. The type of mutation, both by rsid and whether it is homozygous or heterozygous, determines the effects one has. However, keep in mind that a genetic mutation does not have to be present to have difficulties with methylation or any named health condition. Attention must be given to diet and lifestyle, as well as environmental factors.
If a MTHFR mutation is present in your report, there are several things you should do:
1. Receive yearly blood work that checks homocysteine, methionine, folate, B12, B6, B2, and glutathione levels.
2. Avoid folic acid, which is the synthetic form of folate. This is common in supplements and packaged foods.
3. Talk with your physician or functional medicine doctor about supplementation.
4. Consider how this mutation works as a whole with the rest of your genetic data. Rarely does it act alone.
5. Monitor your diet and lifestyle, as this plays a larger role in your overall health than your genetic makeup.

MTR

rs1805087	-- Wild Type

5-methyltetrahydrofolate-homocysteine methyltransferase
This gene encodes for the enzyme, methionine synthase, which is needed for the metabolism of methionine, and amino acid. It requires the use of methylcobalamin (an active form of B12). This gene mutation can lead to homocystinuria. When homocysteine levels are affected, we see skeletal abnormalities, cognitive issues, eye problems, and abnormal blood clotting. It is important to get your homocysteine levels checked yearly, especially if this mutation is present. It is not a common practice for physicians to order homocysteine levels, so one must ask for it. This genetic mutation has also been suggested in Down Syndrome formation.

MTRR

rs1801394	-+ Heterozygous
rs1532268	-- Wild Type

5-methyltetrahydrofolate-homocysteine methyltransferase reductase
This gene encodes for the enzyme, methionine synthase reductase, which is needed for the metabolism of methionine synthase. This gene mutation can lead to homocystinuria. When homocysteine levels are affected, we see skeletal abnormalities, cognitive issues, eye problems, and abnormal blood clotting. It is important to get your homocysteine levels checked yearly, especially if this mutation is present. It is not a common practice for physicians to order homocysteine levels, so one must ask for it. This genetic mutation has also been suggested in Down Syndrome formation.

MUT

rs1141321	-+ Heterozygous
rs9369898	-+ Heterozygous

methylmalonyl CoA mutase
This gene encodes for an enzyme that is responsible for breaking down lipids and proteins for energy use in the mitochondria. With a mutation here, methylmalonic acidemia is a concern. Symptoms occur in early infancy and include failure to thrive, lethargy, vomiting, weak muscle tone, and fatigue. If severe, survival expectation is low. Long term effects may be pancreatitis, kidney disease, and intellectual disabilities. Consult an expert geneticist for official diagnosis. MUT mutations can benefit from additional Adeno-B12.

NOS3

rs1799983	-+ Heterozygous
rs2070744	-+ Heterozygous

Nitric Oxide Synthase 3 is an enzyme that allows for the production of nitric oxide from L-arginine. Nitric oxide is needed for vasodilation of arteria vessels and plays a major role in heart health. With this gene mutation, deficiencies in nitric oxide may be a concern. This can lead to ischemic stroke, myocardial infarction, essential hypertension, pre-eclampsia, and Alzheimer's. Consider getting your NO levels checked and/or supplement with L-arginine. Talk with your functional medicine provider before using supplementation. Be sure to have full cardiometabolic lab work done twice a year.

NQO1

rs1800566	-- Wild Type

NAD(P)H Quinone Dehydrogenase 1
Mutations in this gene have been associated with breast cancer, lung cancer, tardive dyskinesia, and Alzheimer's.

PEMT

rs4244593	-- Wild Type
rs4646406	++ Homozygous
rs7946	++ Homozygous

Phosphatidylethanolamine N-Methyltransferase
Mutations in this gene may lead to deficiencies in phosphatidylcholine, a phospholipid needed for cell membrane integrity. Health concerns related to this class of gene mutations include: endometriosis, orofacial clefts, and non-alcoholic fatty liver disease. Phosphatidylcholine is the precursor to Acetylcholine, a neurotransmitter responsible for memory formation. Clinically we have observed increased memory retention and function with Phosphatidylcholine supplementation with these SNPs.
Discuss supplementation with your practitioner.

PON1

rs662	-- Wild Type

paraoxonase 1 gene
This gene allows for the breakdown of toxic chemicals, especially pesticides, medications, and heavy metals. Mutations in this gene allow for susceptibility to heart disease, diabetes, atherosclerosis, and pesticide poisoning. This is especially critical for microvascular issues related to eyesight in diabetes. Make sure you are choosing organic food sources and avoiding pesticide use. Some advanced laboratories offer pesticide and environmental toxin

Prothrombin

rs1799963	-- Wild Type

Coagulation factor II, thrombin
This gene is needed for proper blood coagulation. Mutations in this gene lead to an increase of thrombosis, loss of pregnancy, and cerebral stroke. Follow an anti-inflammatory, Mediterranean Diet. Discuss testing options with your physician.

SHMT

rs1979277	-- Wild Type

Serine Hydroxy methyltransferase 1
RS1979277 SNPs can reduce the function by up to 50%. Research suggests this gene is associated with Adult Acute Lymphocytic Leukemia and cardiovascular disease. SNPs here can lower glycine levels in the body resulting in decreased glutathione and cartridge production. Consider increasing glycine containing foods. As well, this can SNP can increase Uracil levels and is potentially implicated in some cancers. B6 in the P-5-P form may be beneficial to helping SHMT function.

SLC19A1

rs1051266	++ Homozygous

This gene regulates the transport and levels of intracellular folate. Consider RBC folate testing and supplementation. This gene is associated with methotrexate metabolism difficulty and colorectal cancer. Synthetic Folic acid should be avoided.

SOD1

rs2070424	-- Wild Type
rs4998557	-- Wild Type

superoxide dismutase 1
This gene encodes for superoxide dismutase, and enzyme that binds with copper and zinc to break down free radicles. Mutations in this gene are associated with amyotrophic lateral sclerosis (ALS), which is a disease characterized by muscle weakness and wasting. It is thought that this mutation increases the chance of oxidative stress on the motor

SOD2

rs2758331	-+ Heterozygous
rs4880	-+ Heterozygous

Superoxide dismutase 2
Mutations of this gene have been linked to idiopathic cardiomyopathy, premature aging, cancer, and sporadic motor neuron disease. Consider SOD supplementation.

SOD3

rs1799895	-- Wild Type

Superoxide dismutase 3
This gene is associated with riding the body of free radicals and oxidative stress; however, this mutation is linked to an increase of oxidative stress. This gene mutation is associated with copper and folate pathways. Consider SOD supplementation.

SUOX	
rs7297662	-+ Heterozygous
rs773115	-- Wild Type

Sulfite oxidase
This gene encodes for an enzyme that is needed in the final stages of degradations of sulfur containing amino acids, cysteine and methionine. Specifically the degradation of Sulfite into sulfate. Mutations in this gene are linked to early childhood neurological conditions, especially seizures and sulfite sensitivities. Consider molybdenum supplementation to help improve SUOX activity.

TCN1/2	
rs526934	-- Wild Type
rs1801198	-- Wild Type

Transcobalamin 1
This gene is necessary for the transportation of vitamin B12. With a mutation here, consider testing and supplementation with methylcobalamin. Research suggests difficulty with digestion and stomach acid. With a mutation in this gene, B12 levels may be low. Peripheral neuropathy is common with this deficiency. Consider intracellular nutrient testing and B12 supplementation.

TNF	
rs1800629	-- Wild Type

Tumor necrosis factor
This gene is responsible for creating a pro-inflammatory cytokine that has a number of duties, including cell proliferation and differentiation, apoptosis, and lipid metabolism. A mutation here can lead to cancer, autoimmune disease, and insulin resistance. It has been specifically connected to rheumatoid arthritis, juvenile idiopathic arthritis, migraines, asthma, and narcolepsy. Follow a low-inflammatory diet and work with a functional medicine provider to reduce autoimmune chances.

VDR	
rs731236	-- Wild Type
rs1544410	-- Wild Type
rs2228570	-- Wild Type

VDR
This gene encodes for the receptor site of vitamin D, which is responsible for regulating calcium and phosphate. Health concerns that are directly connected to a mutation in this

Client: Your genotype.
Minor: The genotype that is found least in nature.
Wild Type: The genotype that is found most often in nature, this is reported as green. This isn't always ideal.
Homozygous: This means you tested for both copies of the minor type allele. This typically has more severe issues.
Heterozygous: : This means you tested for one copy of the minor allele and one copy of the wild type allele.
Gene: This is the specific gene we are looking at for variations.
RS#: This is the specific variation within the gene. There are multiple locations within a gene for potential variations, all of which can indicate a different issue or severity.

Disclaimer: This test was developed by MaxGen Labs, LLC and has not been approved by the FDA. It is not intended to diagnose, treat, cure or prevent disease. This test should be considered for educational purposes only. Do not make decisions about your health without discussing it with a licensed practitioner. The information contained within the report does not consider other genetic variations or environmental factors that might contribute to someone's phenotype or symptoms. This test does not analyze all variations within a gene that someone might carry. The rs#'s contained within the report were picked from scientific literature, multiple physician collaborations, and clinical observation by MaxGen Labs and are subject to change at any time.