FUNCTIONAL MEDICINE
INSTITUTE OF THE MIDWEST
PATHWAY TO RESTORING VITALITY

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Date of Analysis: XX/XX/XXXX
GVA Version: 240

Patient: First/Last Name

Variants that Impact Gut Health & Digestion

For those with health challenges, gut inflammation, dysbiosis and digestive disturbances are often common. If there are issues here, this may be step number one of many steps in supporting the patient/client. Proper function of the intestinal tract is critical for the absorption of nutrients. Unfortunately, there are many genetic variants that can impact digestive function.

Inflammation from peroxynitrite can damage the delicate intestinal lining. SNPs that reduce Glutathione, SOD, NOS and BH4, as well as the CBS and BHMT SNPs that will increase ammonia, will increase the peroxynitrite and cause damage to the gut.

When there are low methyl groups as a result of MTHFR, along with SNPs in HNMT and ABP1 (that both degrade histamine) the high histamine will create higher levels of zonulin, which irritates the intestinal tract and potentially contributes to candida and leaky gut.

Variants in the HLA genes may contribute to gluten intolerance, and further gut inflammation.

And finally, variants in the FUT2 gene may impact the production of prebiotics, to support probiotics. Variants here may cause disruption in the good bacteria of the gut and impair B12 assimilation. Impaired B12, among other things, may reduce the production of methyl groups, thus resulting in less than optimal histamine clearing.

All of these factors should be considered when assessing gut health. Supporting gut health, if an issue, is likely the first step needed in supporting the patient/client. This would include, but are not limited to, reducing histamine, eliminating gluten if an issue, reducing peroxynitrite by supporting variants in SOD, GSH, NOS, CBS, BHMT, and creating BH4.

Gene Name	Variants	Metrics
HLA		
HLA-DQA2 (rs2858331)		AA 35.1%
HLA-DQA1 (rs2187668)		CC 79.0%
HLA-DQA2 (rs7454108)	1	TC 19.3%
HLA-DQB1 (rs7775228)		TT 74.4%
HLA-DRA (rs2395182)	1	GT 32.5%
HLA Enzymes	80%	
KIAA1109		
KIAA1109 (rs6822844)		GG 72.7%
KIAA1109 (rs13119723)		AA 72.3%
KIAA1109	100%	

Product Name	SNP Total	Lab Total	Symptoms
Histamine Scavenger	**4.17**	**#N/A**	**#N/A**

Variants in these genes may increase the chances of Celiac Disease or gluten intolerance. Other factors that may impact gut health are FUT variants that impact probiotics, HNMT and ABP1 variants that lessen histamine degradation and consequently cause zonulin production, low folate or high peroxynitrite.

With two HLA variants, there is a possibility for gluten intolerance, especially if it's the HLA DQA1. Additionally, if the gut is damaged from peroxynitrite and zonulin, gluten sensitivity can be worse.

The KIAA1109 gene is associated with susceptibility to celiac disease. Celiac disease is a common small intestinal inflammatory condition induced by dietary wheat, rye, and barley. Variants in this gene may increase the chances of Celiac Disease.

MCM6		
MCM6 (rs182549)	2	TT 35.3%
MCM6 (rs4988235)	2	AA 34.7%
Lactose Intolerance	0%	

The MCM6 gene is a protein coding gene. Single nucleotide polymorphisms in this gene can impact the neighboring LCT gene. The LCT gene provides instructions for making an enzyme called lactase.

Lactase breaks down lactose found in milk and dairy products into smaller sugars called glucose and galactose for absorption. The body then absorbs these simpler sugars into the bloodstream. Lactose intolerance in adulthood is caused by gradually decreasing activity of the LCT gene after infancy.

Variations in the MCM6 genes cause the LCT gene to remain active during adulthood. Because of this, individuals with increased variants are more likely able to digest the lactose found in milk and dairy products.

There are 4 variants in the MCM6 gene. With 4 variants, there is a high likelihood of being lactose tolerant.

Peanut Allergy		
HLA-DQA2 (rs9275596)	1	TC 43.9%
HLA-DRA (rs7192)	1	GT 45.4%
Peanut Allergy	50%	

Studies have shown that peanut allergies are one of the most common food allergies.

Peanuts are not the same as tree nuts such as almonds, cashews, and walnuts. Peanuts grow underground and are part the legume family. Other examples of legumes include beans, peas, lentils and soybeans.

Variants in rs9275596 and rs7192 are associated with the increased susceptibility of developing a peanut allergy is individuals with European ancestry.

With two variants, this individual has an increased risk of developing a peanut allergy.

Caffeine Consumption		
AHR (rs4410790)	1	TC 46.6%
CYP1A2 (rs2472297)	1	CT 32.2%
Caffeine Consumption	50%	

The AHR gene contains the instructions for a protein that helps regulate the amount of certain proteins. One of these proteins includes an enzyme, called CYP1A2.

The CYP1A2 gene contains the instructions for an enzyme that breaks down many substances, including caffeine. This enzyme is one of the many cytochrome P450 enzymes.

Studies have shown that variants in these SNPs related to a higher consumption of caffeine.

With 2 variants it is possible that this individual consumes an increased amount of caffeine

Caffeine Metabolization		
CYP1A2 C164A (rs762551)	2	AA 50.0%
Caffeine Metabolization	0%	

The CYP1A2 gene encodes a member of the cytochrome p450 family of proteins. These proteins metabolize nutrients and drugs. One well known substrate of CYP1A2 is caffeine

Caffeine is a bitter substance that can be found in coffee, tea, soft drinks, chocolate, kola nuts, and certain medicines. It has many effects on the body's metabolism, including stimulating the central nervous system.

Studies have shown that individuals with variants in this gene are faster metabolizers of caffeine and therefore will feel less of a stimulating effect from caffeine.

With 2 variants this individual is likely a fast caffeine metabolizer.

BCMO1		
BCMO1 A379V (rs7501331)		CC 60.3%
BCMO1 R267S (rs12934922)	1	AT 48.9%
BCMO1 (rs4889294)	2	CC 20.1%
BCMO1 (rs11643312)		GG 47.9%
BCMO1 (rs6564862)		CC 44.8%
BCMO1 (rs7192178)		AA 35.6%
BCMO1 (rs8046134)		GG 60.4%
BCMO1 (rs6564863)	1	TC 43.9%
BCMO1 (rs117523015)		AA 97.5%
BCMO1 (rs7202895)		AA 91.6%
BCMO1 (rs117887860)		CC 99.6%
BCMO1 (rs4889298)		CC 26.2%
BCMO1 (rs11865869)		AA 60.4%
BCMO1 (rs3803651)	2	GG 5.4%
BCMO1 (rs11647597)	2	GG 5.5%
BCMO1	78%	

BCMO1 or Beta-Carotene Oxygenase 1 is a protein coding gene. The protein encoded by this gene is a crucial enzyme in beta-carotene metabolism to vitamin A. It catalyzes the oxidative cleavage of beta-carotene into two retinal molecules. Vitamin A metabolism is important for vital processes such as vision, embryonic development, and skin protection. Polymorphisms in this gene can affect serum retinol concentration.

The most significant SNPs are BCMO1 A379V rs7501331, BCMO1 R267S rs12934922, and BCMO1 rs4889294

Research has found that double mutations in both BCMO1 A379V rs7501331 and BCMO1 R267S rs12934922 can cause a substantial reduction in the conversion of beta-carotene into retinol l in Females.

Our Phase II Lyme study also determined that variants in BCMO1 R267S rs12934922, and BCMO1 rs4889294 were much higher in patients with Lyme Disease.

FUT2		
FUT2 (rs492602)	2	GG 22.1%
FUT2 (rs601338)	2	AA 21.5%
FUT2 (rs602662)	2	AA 24.4%
FUT2 (rs16982241)		GG 73.4%
FUT2 (rs281377)		CC 29.9%
FUT2 (rs1800022)		CC 98.1%
FUT2 (rs1047781)		AA 98.6%
FUT2 (rs1800027)		CC 87.0%
FUT2 (rs1800028)		CC 99.9%
FUT2 (rs485186)	2	GG 24.5%
FUT2 (rs603985)	2	CC 24.6%
FUT2 (rs504963)	2	AA 24.1%
FUT2	54%	

Product Name	SNP Total	Lab Total	Symptoms
Pro Flora Max Plus	4.62	#N/A	#N/A

Variants in the FUT2 enzyme may lead to disruptions in the good intestinal bacteria. This enzyme variant may cause a predisposition to Crohn's disease. Monitor gut health with this variant. FUT2 may also be related to lowered immune function.

ABP1 (Histamine Breakdown)		
ABP1 (rs10156191)	1	CT 38.1%
ABP1 (rs1049742)		CC 85.8%
ABP1 (rs1049793)	1	CG 42.0%
ABP1 (rs35070995)		AA 99.8%
ABP1	80%	

Product Name	SNP Total	Lab Total	Symptoms
Histamine Scavenger	4.17	#N/A	#N/A

This is the gene that makes the DAO enzyme that helps degrade histamine. SNPs with this gene, combined with HNMT genes, and low methyl groups, may result in high histamine and high zonulin.

Eating less histamine containing and histamine reducing foods may be needed.

With only two variants in the APB1 gene, it would seem likely the production of the DAO enzyme is good. However, at this time, we do not know which SNP is most relevant, and homozygosity of the most important one could be clinically relevant.

HNMT (Histamine Transferase)		
HNMT (rs1020678)	1	TC 47.5%
HNMT (rs1050891)	1	AG 33.8%
HNMT (rs1349992)	1	GA 47.3%
HNMT (rs1378321)	1	AG 33.9%
HNMT (rs1455157)	1	TC 34.4%
HNMT (rs1455158)	1	CT 34.4%
HNMT (rs1455162)	1	AG 34.4%
HNMT (rs1455164)	1	GA 34.6%
HNMT (rs1455167)	1	TG 34.4%
HNMT (rs1580111)	1	CT 47.4%
HNMT (rs16840064)		GG 98.8%
HNMT (rs2198652)	1	CT 34.4%
HNMT (rs2737385)	1	TG 33.9%
HNMT (rs3100701)	1	GA 48.1%
HNMT (rs3100725)	1	GA 34.5%
HNMT (rs3791235)	1	CA 33.3%
HNMT (rs3828168)	1	CT 33.7%
HNMT (rs4245861)	1	CT 33.8%
HNMT (rs4646322)		CC 69.0%
HNMT (rs4646333)	1	GA 33.8%
HNMT (rs4954941)	1	GA 33.9%
HNMT (rs60444277)		GG 99.9%
HNMT (rs993891)	1	TG 34.5%
Histamine Clearing (HNMT)	57%	

Product Name	SNP Total	Lab Total	Symptoms
Histamine Scavenger	4.17	#N/A	#N/A

HNMT produces the enzyme that uses a methyl group to degrade histamine in the body. The ABP1 gene also clears histamine with the DAO enzyme.

If there are many SNPs, and low methyl groups, there is the potential for high histamine. This can result in high levels of zonulin, which can cause gut inflammation and the potential for leaky gut. Over time, this may contribute to autoimmune disorders.

Avoiding high histamine foods (alcoholic beverages and fermented foods) may be helpful as well as taking high amounts of Histamine Scavenger. Sometimes dosages need to be 9 to 12 per day in the beginning, and then can reduce over time.

Histamine Scavenger may need Pro SAMe if there are low methyl groups.

If there is a lot of histamine and zonulin, and if they also have an HLA gene, gluten sensitivity may be a problem as well.

GRHPR		
GRHPR (rs2768659)	2	GG 43.9%
GRHPR (rs309455)		CC 59.0%
GRHPR (rs309453)		TT 34.4%
GRHPR	67%	

GRHPR provides instructions for making the enzymes glyoxylate, and hydroxypyruvate reductase. This enzyme plays a role in preventing the buildup glyoxylate by converting it into glycolate. Glycolate can be easily eliminated from the body. This enzyme can also convert hydroxypyruvate to D-glycerate. D-glycerate is eventually converted into glucose, by other enzymes, and can be used for energy.

Variations in this gene can cause a reduction in the conversion of glyoxylate to glycolate. Glyoxylate builds up and is converted to a compound called oxalate. The oxalate is then filtered through the kidneys and is either excreted in the urine as a waste product or combines with calcium to form calcium oxalate. Calcium oxalate is a hard compound that is the main component of kidney and bladder stones.

A diet of low oxalate is suggested if there are variants present.

We are not aware of these SNPs being clinically relevant for Oxylate issues, but put them here as a consideration for someone has health challenges that cannot be found. If there are SNPs here, verification though OAT testing or other methods would be in order, rather than just relying on these SNPs.

Krebs Cycle - Genes that support the production of Acetyl-CoA and ATP

For the body to function properly, fats, carbs and proteins need to be carried into the cell and to be converted into Acetyl-CoA, the first step of the Citric Acid Cycle, for the production of ATP. These genes play a role in this process. If inadequate Acetyl-CoA is made, the individual may present with fatigue. Since ATP is needed for many functions, other parts of the body may suffer as a result with low ATP.

If there are a lot of SNPs here, this very well may be one of the first things that need to be addressed with the gut. If gut issues exist as well, you can work on both the gut and cellular energy at the same time.

Gene Name	Variants	Metrics
Carnitine Transportation		
SLC22A5 (rs13180043)		CC 91.9%
SLC22A5 (rs2631367)	1	CG 50.1%
SLC22A5 (rs13180186)		GG 83.6%
SLC22A5 (rs2631361)		CC 37.9%
SLC22A5 (rs2631362)		AA 48.4%
SLC22A5 (rs2631363)		AA 38.1%
SLC22A5 (rs17622208)	1	GA 48.1%
SLC22A5 (rs17689550)		CC 79.2%
SLC22A5 (rs2073642)		CC 84.9%
SLC22A5 (rs2073643)	1	TC 49.1%
SLC22A5 (rs2074610)		TT 99.7%
SLC22A5 (rs2631359)		CC 48.5%
SLC22A5 (rs274549)	2	CC 70.5%
SLC22A5 (rs274550)	2	TT 69.1%
SLC22A5 (rs274551)	2	CC 70.5%
SLC22A5 (rs274557)		TT 37.1%
SLC22A5 (rs274558)		AA 36.9%
SLC22A5 (rs274567)		CC 37.9%
SLC22A5 (rs274570)		CC 48.5%
SLC22A5 (rs274571)		AA 48.5%
SLC22A5 (rs4646301)		GG 85.0%
SLC22A5 (rs635619)		GG 48.5%
SLC22A5 (rs671473)		CC 48.4%
SLC22A5 (rs1045020)		CC 79.0%
SLC22A5 (rs2631366)		CC 99.8%
SLC22A5 (rs72552726)		GG 99.7%
SLC22A5 (rs274548)	2	CC 66.0%
Carnitine Transportation	84%	

Product Name	SNP Total	Lab Total	Symptoms
Fatty Acid Assist	**1.57**	**#N/A**	**#N/A**
Mitochondrial & Energy Assist	**8.17**	**#N/A**	**#N/A**
CBS / BHMT Assist	**3.19**	**#N/A**	**#N/A**

The SLC22A5 gene provides instructions for making a protein called OCTN2. This protein is positioned within the cell membrane, where it transports carnitine into the cell.

Carnitine is an amino acid derivative that is synthesized in the human body. Carnitine is primarily synthesized in the liver and is stored in the tissues that use fatty acids as their primary fuel (Such as skeletal and cardiac muscle). Carnitine is required for mitochondrial β-oxidation of long-chain fatty acids for energy production.

Variations in the SLC22A5 gene can result in a dysfunctional OCTN2 protein. This can cause a shortage of carnitine within cells. Without carnitine, fatty acids cannot enter mitochondria. This may cause muscle weakness and hypoglycemia. Fatty acids may also build up in cells and damage the heart, liver, and muscles.

Under certain conditions, the demand for Carnitine may exceed an individual's capacity to synthesize it, making it a conditionally essential.

High levels of Adipate, Suberate or Ethylmalonate in urine organic acid testing may also confirm lack of carnitine.

Consequently, if there are too many variants, supplementation with Acetyl-L Carnitine and other nutrients to support fat transportation/utilization may be needed to consumed with meals.

PANK		
PANK1 (rs12412483)		GG 94.6%
PANK1 (rs2038921)	2	GG 31.8%
PANK1 (rs10509577)		AA 88.0%
PANK1 (rs10160034)		CC 79.5%
PANK1 (rs10881606)		TT 44.2%
PANK1 (rs6586201)	1	CT 39.7%
PANK1 (rs17482070)		AA 82.4%
PANK1 (rs7921294)		GG 16.0%
PANK1 (rs7091402)		TT 16.0%
PANK1 (rs997456)	1	GA 34.2%
PANK2 (rs6107373)	1	GA 5.6%
PANK2 (rs6084513)	2	AA 27.6%
PANK2 (rs6084506)	1	CT 46.9%
PANK2 (rs4815628)	2	CC 28.8%
PANK2 (rs4815621)	2	GG 50.8%
PANK3 (rs11952767)		CC 99.0%
PANK4 (rs7535528)		GG 41.2%
PANK4 (rs2246732)	1	AG 36.7%
PANK4 (rs2236395)	1	AG 36.7%
PANK4 (rs1980789)		AA 91.6%
PANK1	71%	
PANK2	43%	
PANK3	100%	
PANK4	75%	
Total PANK	61%	

Product Name	SNP Total	Lab Total	Symptoms
Fatty Acid Assist	1.57	#N/A	#N/A
A-L-O Formula	0	#N/A	#N/A

This gene encodes members of the pantothenate kinase family. Pantothenate kinase catalyzes the ATP-dependent phosphorylation of pantothenate (vitamin B5) to give 4′-phosphopantothenate. This reaction is the first and rate limiting step in the synthesis of coenzyme A (CoA).

Coenzyme A (CoA) is a pantothenic acid derived metabolite that is essential for many crucial cellular processes including energy, lipid and amino acid metabolism. About 4% of all known enzymes utilize CoA as a cofactor and CoA thioesters are essential for over 100 different reactions of the intermediary metabolism, such as the Krebs Cycle. In humans, CoA synthesis requires cysteine, pantothenate, and ATP.

PANK1 encodes a member of the pantothenate kinase family.

PANK2 is the only member of the pantothenate kinase family to be expressed in mitochondria.

PANK3 is expressed most abundantly in the liver

PANK4 is most abundant in muscle but is expressed in all tissues.

ACAT

Product Name	SNP Total	Lab Total	Symptoms
Fatty Acid Assist	1.57	#N/A	#N/A
Fatty Acid Liquescense	1.57	#N/A	#N/A
Mitochondrial & Energy Assist	8.17	#N/A	#N/A
Gastrogest	5.71	#N/A	#N/A
ACAT Assist	5.71	#N/A	#N/A
Adenosyl-Cobalamin B12 Assist	5.71	#N/A	#N/A

ACAT1-02 (rs3741049)		GG 78.5%
ACAT-1 (rs10890819)	2	TT 10.2%
ACAT-2 (rs3798211)	2	CC 31.1%
ACAT-1 (rs2280332)		AA 61.8%
ACAT-2 (rs9347340)	2	CC 55.4%
ACAT-2 (rs25683)	2	GG 31.4%
ACAT-2 (rs3465)		GG 38.9%
ACAT (All Genes)	43%	
ACAT - (Single SNP Most Relevent)	100%	

The ACAT Gene may be one of the most important, as it supports the conversion of fats and proteins into Acetyl-CoA. This may be one of the first variants to support, as it slows the production of ATP from the Citric Acid Cycle. Checking the Fatty Acid Metabolism Markers in a Urine Organic Acid test may verify how serious of a problem ACAT is creating.

There are eight variants in the ACAT enzyme. If the variant is the first one listed, the ACAT1-02, then support for ACAT is needed. This is the most significant SNP for ACAT. If there are no variants in ACAT1-02 but eight other variants, support may be needed.

If you want to find out if these SNPs are impacting the utilization of fats and proteins, run the Genova Urine Organic Acids, and see if the markers for fat usage and protein usage indicate a problem.

There are no variants in the most clinically significantly ACAT gene. However, many SNPs in the others may have an impact. Urine Organic Acid testing may indicate a problem if there are numerous other SNPs.

SLC16A1

Product Name	SNP Total	Lab Total	Symptoms
ACAT Assist	5.71	#N/A	#N/A
Carb Assist	0	#N/A	#N/A

SLC16A1 (rs7169)	2	AA 33.5%
SLC16A1 (rs76612089)		CC 96.3%
SLC16A1 (rs11585690)		AA 95.1%
SLC16A1 (rs71659381)		GG 86.3%
SLC16A1 (rs3849174)	1	TG 34.4%
SLC16A1 (rs12028967)	1	TG 44.0%
SLC16A1 (rs4301628)	1	CT 43.8%
SLC16A1	64%	

The protein encoded by this gene modulates the cellular levels of lactate and pyruvate.

Pyruvate is the end product of glycolysis, which is then converted into acetyl coA that enters the Krebs cycle when there is sufficient oxygen present. When the oxygen is insufficient, pyruvate is broken down anaerobically, creating lactate.

Lactate is produced by almost all tissues in the body, with the highest level of production found in muscle tissues. Under normal conditions, lactate is rapidly cleared by the liver.

Variants in this gene may lead to metabolic myopathy and exercise-induced hyperinsulinemic hypoglycemia.

ACSL1

Product Name	SNP Total	Lab Total	Symptoms
Fatty Acid Assist	1.57	#N/A	#N/A
A-L-O Formula	0	#N/A	#N/A

ACSL1 (rs9997745)	1	GA 24.5%
ACSL1 (rs4862417)		AA 55.3%
ACSL1 (rs13120078)	1	GA 44.6%
ACSL1 (rs12503643)	2	TT 17.1%
ACSL1 (rs41278587)		GG 94.7%
ACSL1 (rs6552828)	2	GG 35.0%
ACSL1 (rs72695682)		CC 90.2%
ACSL1 (rs72695685)		TT 86.4%
ACSL1	63%	

The protein encoded by this gene is an isozyme of the long-chain fatty-acid-coenzyme A ligase family. Although differing in substrate specificity, subcellular localization, and tissue distribution, all isozymes of this family convert free long-chain fatty acids into fatty acyl-CoA esters, and thereby play a key role in lipid biosynthesis and fatty acid degradation.

At this time, no particular SNPs have been specifically identified as having the greatest impact on lipids, so these genes are added for general information and may mean that more weight should be given to ACAT or carnitine issues when these genes have high amounts of variants.

If there are many variants, be aware of fat utilization impairments.

TALDO1		
TALDO1 C749776T (rs11246300)		CC 62.7%
TALDO1	100%	

Product Name	SNP Total	Lab Total	Symptoms
A-L-O Formula	0	#N/A	#N/A

TALDO1 is a key enzyme in the synthesis of NADPH for lipid biosynthesis. This enzyme also helps to maintain glutathione in a reduced state to prevent cellular damage from oxygen radicals.

In our Phase II Lyme study, we found that variants in TALDO1 were much higher in patients with Lyme Disease

NDUFS		
NDUFS1 (rs1044120)		CC 37.5%
NDUFS1 (rs4147727)		AA 32.8%
NDUFS1 (rs4147720)		AA 33.3%
NDUFS1 (rs186057450)		GG 99.8%
NDUFS1 (rs1801318)		TT 46.9%
NDUFS1 (rs11548670)		AA 93.2%
NDUFS1 (rs4147713)		AA 28.3%
NDUFS1 (rs4147712)		TT 31.5%
NDUFS1 (rs4147709)		CC 31.3%
NDUFS2 (rs10908826)	1	CT 25.1%
NDUFS2 (rs4656994)	1	GA 35.2%
NDUFS2 (rs10797094)	2	AA 35.7%
NDUFS2 (rs1136224)		AA 72.4%
NDUFS3 (rs4147730)		GG 74.5%
NDUFS4 (rs1532163)	2	GG 59.0%
NDUFS4 (rs1994648)		AA 64.5%
NDUFS4 (rs2637002)	1	CA 26.0%
NDUFS4 (rs3103600)	1	GA 38.4%
NDUFS4 (rs4147735)	1	TG 27.6%
NDUFS4 (rs432020)	1	CT 26.1%
NDUFS4 (rs381575)	1	AC 45.5%
NDUFS4 (rs11743262)	1	TG 26.9%
NDUFS4 (rs2124948)	1	CT 49.3%
NDUFS4 (rs2636993)	1	GA 40.6%
NDUFS4 (rs365578)	1	GT 40.7%
NDUFS4 (rs42565)	1	AG 26.2%
NDUFS4 (rs31304)	2	CC 93.1%
NDUFS4 (rs31303)	2	GG 59.0%
NDUFS4 (rs31302)	2	TT 41.1%
NDUFS4 (rs12517465)	1	GA 49.1%
NDUFS4 (rs10513019)	1	TC 27.7%
NDUFS4 (rs1388111)		TT 47.5%
NDUFS4 (rs372215)	1	GA 26.1%
NDUFS4 (rs4147736)	1	GA 49.3%
NDUFS4 (rs2607508)	1	TC 26.4%
NDUFS4 (rs256116)	2	TT 58.9%
NDUFS4 (rs4147737)		AA 78.9%
NDUFS4 (rs12515547)		GG 64.4%
NDUFS4 (rs12522533)		GG 64.4%
NDUFS4 (rs4147739)	1	GA 27.6%
NDUFS4 (rs31308)	1	AG 26.1%
NDUFS4 (rs256094)		GG 51.9%
NDUFS4 (rs4147740)		TT 68.0%
NDUFS4 (rs4147742)	1	GA 27.5%
NDUFS4 (rs445347)	1	AG 26.1%
NDUFS4 (rs2607506)		TT 71.3%

Product Name	SNP Total	Lab Total	Symptoms
Mitochondrial & Energy Assist	8.17	#N/A	#N/A
Pro NADH	1.5	#N/A	#N/A

NDUFS1, NDUFS2, NDUFS3, NDUFS4, NDUFS5, NDUFS6, NDUFS7, and NDUFS8 encode a protein that is part of a subunit for Complex I.

Complex I is the first enzyme of the mitochondrial electron transport chain. There are over 40 subunits found in Complex I.

The electron transport chain consists of a series of redox reactions in which electrons are transferred from a donor molecule to an acceptor molecule.

Each electron donor passes electrons to a more electronegative acceptor. The electronegative acceptor then donates these electrons to another acceptor until the electrons are passed to oxygen, the most electronegative and terminal electron acceptor in the electron transport chain. Passage of electrons between donor and acceptor releases energy, which is used to generate a proton gradient across the mitochondrial membrane by “pumping” protons into the intermembrane space. The resulting proton gradient is used to make ATP via ATP synthase.

NADH → Complex I → Q → Complex III → cytochrome c → Complex IV → O2

Complexes I, III and IV are the proton pumps, while Q and cytochrome c are mobile electron carriers.

Variations in these genes may cause Complex I to be deficient.

NDUFS7	50%	
NDUFS1	100%	
NDUFS2	50%	
NDUFS3	100%	
NDUFS4	54%	
NDUFS5	25%	
NDUFS6	56%	
NDUFS8	63%	
NDUFS Total	61%	
NDUFS8 (rs2075626)	1	TC 36.0%
OGDH		
OGDH (rs142839706)		GG 96.7%
OGDH (rs17133537)		TT 30.9%
OGDH (rs740094)		GG 31.4%
OGDH (rs2268308)		CC 92.2%
OGDH (rs757702)		GG 92.2%
OGDH (rs10951768)	1	GT 14.9%
OGDH (rs799434)	2	CC 81.2%
OGDH (rs12155014)	1	TC 14.8%
OGDH (rs10247064)		CC 84.6%
OGDH (rs710887)	2	CC 48.0%
OGDH (rs3735474)	1	AG 15.0%
OGDH (rs3801401)		CC 94.2%
OGDH (rs7805156)		CC 92.9%
OGDH (Alpha-Ketoglutarate to Succinyl-CoA and CO2)	73%	

Product Name	SNP Total	Lab Total	Symptoms
Pro Alpha Ketoglutarate Plus	**0**	**#N/A**	**#N/A**

OGDH or Oxoglutarate (Alpha-Ketoglutarate) Dehydrogenase (Lipoamide) encodes one subunit of the 2-oxoglutarate dehydrogenase complex. This complex catalyzes the overall conversion of alpha-ketoglutarate to succinyl-CoA and CO2.

Studies have shown that variants in OGDH lead to hypotonia, metabolic acidosis, and hyperlactatemia

Detoxification Capacity - SOD, Glutathione (Phase 2 Liver Detox) and CYP (Phase 1 Liver Detox)

SOD (Superoxide Dismutase), Glutathione, CYP (cytochrome P-450) and PON1 represent the body's ability to neutralize free radicals and to detox properly. The more variants here, the less ability to deal with free radicals, which may increase inflammation, aid in the eventual breaking down of the body, and lower the ability to clear toxins. After supporting the gut and ATP production, supporting the neutralizing of free radicals and detoxifying may be the next most important part of the body to support.

Superoxide Dismutase turns the free radical Superoxide into H2O2, which is then turned into water and oxygen by glutathione and catalase. If SOD does not neutralize the free radical, it combines with nitric oxide and creates the very strong oxidizing agent peroxynitrite.

If there is low glutathione, and folate is given, the folate can stimulate Phase 1 detox but overwhelm Phase 2. This can cause inflammation. This is why many people have negative reactions to folate, even when they have MTHFR and folate deficiency. Always make sure you have adequately supported Phase 2 Liver Detox before proceeding with folate.

Variants in PON1 will reduce the ability to clear herbicides and pesticides, further straining glutathione levels.

Gene Name	Variants	Metrics
Detox Ability - SOD		
SOD2 (rs2758331)	2	AA 22.9%
SOD2 A16V (rs4880)	2	GG 24.4%
SOD3 (rs2855262)	1	TC 45.7%
SOD Production	17%	

Product Name	SNP Total	Lab Total	Symptoms
Pro SOD/Catalase Support	**7.83**	**#N/A**	**#N/A**
Nutrition & Anti-Oxidant Accelerator	**8.33**	**#N/A**	**#N/A**

The free radical Super Oxide, may be created 5% of the time when the cells create ATP, and is made in large quantities with NOS uncoupling, and thus making the very dangerous oxidizing agent Peroxynitrite. Since inflammation may be one of the major causes of premature aging and disease, controlling the superoxide free radical is critical. Superoxide Dismutase turns the superoxide free radical into H2O2, so glutathione and catalase can turn them into water and oxygen. The SOD2 genes make superoxide dismutase (SOD) inside the cells, while SOD3 make the SOD outside the cells. Nutrition & Anti-Oxidant Accelerator supports the production of SOD, while Pro SOD/Catalase contains the actual enzymes in a capsule that only opens in the intestinal tract.

With 5 SOD variants, Nutrition & Anti-Oxidant Accelerator will likely not be enough superoxide dismutase protection. Consequently, use 3 Nutrition & Anti-Oxidant Accelerator capsules and at least 2-3 Pro SOD/Catalase. This can be increased to 4 if there are other factors that increase the peroxynitrite. Pro SOD will be a permanent supplement for this individual. Nrf2 Accelerator would also likely be needed.

Gene Name	Variants	Metrics
Detox Ability - Glutathione		
GSTM1 (rs1056806)		CC 87.9%
GSTP1 A114V (rs1138272)		CC 84.3%
GSTP1 I105V (rs1695)	1	AG 44.4%
SHMT2 (rs34095989)		GG 38.1%
CTH (rs1021737)	1	GT 40.8%
Glutathione Enzymes	83%	
Glycine / SHMT	100%	
Cysteine Production	50%	

Product Name	SNP Total	Lab Total	Symptoms
Glutathione Accelerator	**4.63**	**#N/A**	**#N/A**
Peroxynitrite Scavenger	**4.5**	**#N/A**	**#N/A**
S Acetyl Glutathione	**4.63**	**#N/A**	**#N/A**
Peroxynitrite Scavenger P.M.	**4.5**	**#N/A**	**#N/A**
GSH Assist	**4.63**	**#N/A**	**#N/A**
Nrf2 Accelerator	**4.5**	**#N/A**	**#N/A**
SHMT Assist	**0**	**#N/A**	**#N/A**

Glutathione is a critical antioxidant that is very important in Phase II Liver Detox. Studies have shown those with the highest glutathione live the longest. If there is insufficient glutathione, you will age prematurely, and be prone to lowered ability to detox. Individuals with low glutathione often report an inability to tolerate strong smells and are very prone to inflammatory conditions, especially when this is combined with other variants that cause inflammation.

Glutathione Accelerator has NAC and enzyme support, while GSH Assist has glycine when this is needed, or NAC is contraindicated. Nrf2 Accelerator supports the production of Glutathione. Always make sure you have supported Glutathione before giving folate.

With 1 glutathione enzyme variant, there may be a slight need for Glutathione Accelerator, unless there is a lot of peroxynitrite production, or the SHMT or CTH variants are present.

There are no variants in the SHMT gene, so there may be adequate glycine, unless other variants use it up in excess.

With one variant in the CTH gene, it is possible that Cysteine may be lower than necessary to create adequate glutathione. Performing Urine Organic Testing can help determine if there is a glutathione need. Variants in glutathione enzymes and SHMT can further aggravate glutathione levels.

Catalase		
CAT (rs1049982)	2	CC 43.1%
CAT (rs11032703)		CC 84.0%
CAT (rs2300181)	1	CT 38.1%
CAT (rs480575)		AA 49.1%
CAT (rs494024)	2	CC 39.0%
CAT (rs484214)		AA 49.5%
CAT (rs17881734)		GG 97.3%
CAT (rs2284369)		AA 58.5%
CAT (rs1408036)		AA 80.7%
CAT (rs769218)		GG 58.4%
CAT (rs17881288)		AA 97.3%
CAT (rs7933285)	1	CT 38.0%
CAT (rs2073058)	1	AG 38.1%
CAT (rs12273124)	1	AG 10.0%
CAT (rs769217)		CC 58.5%
CAT (rs17881586)		GG 96.6%
CAT (rs511895)	2	CC 38.9%
CAT (rs10488736)	2	TT 9.4%
CAT	67%	

Product Name	SNP Total	Lab Total	Symptoms
Pro SOD/Catalase Support	7.83	#N/A	#N/A

The CAT gene provides instructions for making an enzyme called catalase. Catalase is a key antioxidant enzyme in the body's defense against oxidative stress. Oxidative stress is when there is an imbalance between the production of free radicals and the body's defense against the free radicals harmful effects.

Studies have hypothesized that oxidative stress plays a role in the development of many chronic or late-onset conditions such as diabetes, asthma, Alzheimer's disease, and rheumatoid arthritis.

Catalase will convert the reactive oxygen species hydrogen peroxide to water and oxygen.

2 H2O2 → 2 H2O + O

This alleviates the toxic effects of hydrogen peroxide.

Variants in this gene have been associated with decreases in catalase activity.

Detox Ability - CYP		
CYP1A1 (rs4986883)		TT 99.8%
CYP1A1*2C A4889G (rs1048943)		TT 91.1%
CYP1A1*4 C2453A (rs1799814)		GG 91.4%
CYP1A2 C164A (rs762551)	2	AA 50.0%
CYP1B1 L432V (rs1056836)		CC 19.3%
CYP1B1 R48G (rs10012)		GG 15.9%
CYP1B1 N453S (rs1800440)		TT 67.9%
CYP2A6*2 A1799T (rs1801272)		AA 95.2%
CYP2C19 (rs12248560)		CC 62.5%
CYP2C9*3 A1075C (rs1057910)		AA 87.9%
CYP2C9*2 A C430T (rs1799853)		CC 77.0%
CYP2D6 T100C (rs1065852)	1	GA 34.9%
CYP2D6 (rs1135840)		GG 32.7%
CYP2D6 T2850C (rs16947)	1	GA 53.1%
CYP2E1*1B A10023G (rs55897648)		GG 99.6%
CYP2E1*1B G9896C (rs2070676)	2	CC 75.5%
CYP2E1*4 A4768G (rs6413419)		GG 94.2%
CYP3A4*1B (rs2740574)	2	TT 91.2%
CYP3A4*3 M445T (rs4986910)		AA 98.5%
CYP - Phase 1 Liver Detox	81%	

Product Name	SNP Total	Lab Total	Symptoms
Nrf2 Accelerator	4.5	#N/A	#N/A
Detox Liquescense	5	#N/A	#N/A

Inside the liver cells there are sophisticated mechanisms that break down toxic substances. Every drug, artificial chemical, pesticide, and hormone is broken down by enzyme pathways inside the liver cells.

Many of the toxic chemicals that enter the body are fat-soluble, which means they dissolve only in fatty or oily solutions and not in water. This makes them difficult for the body to excrete. The body's primary defense against metabolic poisoning is carried out by the liver. The liver has two mechanisms designed to convert fat-soluble chemicals into water soluble chemicals so that they may then be easily excreted from the body via watery fluids such as bile and urine.

There are two detoxification pathways inside the liver cells, which are called the Phase 1 and Phase 2 detoxification pathways. Phase one detoxification consists of oxidation reduction and hydrolysis. Phase one detoxification is catalyzed by enzymes referred to as the cytochrome P450 enzyme. These enzymes reside on the membrane system of the liver cells (called Hepatocytes). Human liver cells possess the genetic code for many isoenzymes of P-450 whose synthesis can be induced upon exposure to specific chemicals. This provides a mechanism of protection from a wide variety of toxic chemicals.

This pathway converts a toxic chemical into a more harmful chemical. This is achieved by various chemical reactions (such as oxidation, reduction and hydrolysis), and during this process free radicals are produced which, if excessive, can damage the liver cells. Antioxidants (such as vitamin C and E and natural carotenoids) reduce the damage caused by these free radicals. If antioxidants are lacking and toxin exposure is high, toxic chemicals become far more dangerous.

The more CYP variants, the more difficulty there may be in detoxification of toxins and drugs.

We may develop a supplement to support CYP, but in the meantime if there are many variants, support Nrf2 and glutathione.

ABP1		
ABP1 (rs10156191)	1	CT 38.1%
NSAID Sensitivity	50%	

Non-steroidal anti-inflammatory drugs (NSAIDs) are the drugs most frequently involved in hypersensitivity drug reactions. Histamine is released in the allergic response to NSAIDs and is responsible for some of the clinical symptoms.

Studies have shown that individuals with variants in SNP rs10156191 have a hypersensitivity to NSAIDS.

With 1 variant this individual may be hypersensitive to NSAIDS.

PON1 - Peroxynase		
PON1 Q192R (rs662)		TT 48.1%
PON1 (rs854555)	2	CC 40.4%
PON1 (rs3917550)		GG 76.4%
PON1 (rs3917548)		AA 88.4%
PON1 (rs3917542)		CC 60.4%
PON1 (rs2074354)		GG 79.6%
PON1 (rs854561)	2	TT 12.9%
PON1 (rs3917498)		GG 42.8%
PON1 (rs2272365)		AA 71.9%
PON1 (rs2049649)		AA 45.3%
PON1 (rs2299260)		TT 67.0%
PON1 (rs2299262)		CC 38.4%
PON1 (rs854569)	2	GG 58.7%
PON1 (rs2237584)		CC 86.2%
PON1 (rs3917478)		TT 79.3%
PON1 (rs854566)	2	GG 66.5%
PON1 (Single Most Relevant)	100%	
PON1 (All Genes)	81%	

Product Name	SNP Total	Lab Total	Symptoms
PON1 Assist	0	#N/A	#N/A
Addex	0	#N/A	#N/A

Pesticide use has been increasing over the years, and has become quite controversial.

Our body needs the ability to detox from them and the PON1 (Peroxynase) gene, along with Glutathione, plays an important role in helping the body clear them.

PON1 (Paraoxonase) plays a large role in removing pesticides. It is also involved with supporting HDL function, crucial for healthy circulation.

The most important gene so far is the first one listed, the PON1 Q192R, however, the rest may play an important role as well.

Consider using Addex Homeopathic Spray and PON1 Assist for those with this genetic variant.

Although there are no PON1 rs662 variants, if there are many others, support may be needed. It is unknown how the other PON1 variants may impact detoxification.

NAT Genes		
NAT1 (rs4986782)		GG 96.2%
NAT1 (rs7017402)	1	AG 21.6%
NAT1 (rs11203943)		GG 81.3%
NAT1 (rs4921581)		AA 11.6%
NAT1 (rs13253389)		AA 12.8%
NAT1 (rs17693103)		GG 71.5%
NAT1 (rs9325827)		TT 71.4%
NAT1 (rs6586714)	2	GG 75.6%
NAT1 (rs17126350)		AA 86.5%
NAT1 (rs8190837)		AA 83.0%
NAT1 (rs8190844)		CC 97.7%
NAT1 (rs8190845)		GG 72.5%
NAT1 (rs8190847)		GG 94.3%
NAT1 (rs4987076)		GG 94.3%
NAT1 (rs4986990)		GG 94.4%
NAT1 (rs4986783)		TT 94.3%
NAT1 (rs56172717)		AA 99.5%
NAT1 (rs15561)	2	CC 51.8%
NAT1 (rs4986993)	2	GG 51.9%
NAT2 (rs11780272)	1	TC 47.5%
NAT2 (rs2087852)		AA 50.8%
NAT2 (rs1390358)	1	TC 46.8%
NAT2 (rs2410556)		TT 76.0%
NAT2 (rs1961456)	1	AG 41.5%
NAT2 (rs973874)	2	CC 98.7%
NAT2 (rs7832071)	1	CT 47.6%
NAT2 (rs56011192)		CC 99.2%
NAT2 (rs2552)		TT 91.3%
NAT2 C282T (rs1041983)		CC 45.3%
NAT2 C481T (rs1799929)	1	CT 47.5%
NAT2 G286E (rs1799931)		GG 94.0%
NAT2 I114T (rs1801280)	1	TC 64.3%
NAT2 K268R (rs1208)	1	GA 47.6%
NAT2 R197Q (rs1799930)		GG 50.1%
NAT2 R64Q (rs1801279)		GG 99.6%
NAT1	82%	
NAT2	72%	

Product Name	SNP Total	Lab Total	Symptoms
Nrf2 Accelerator	4.5	#N/A	#N/A

The NAT1 and NAT2 genes encode an enzyme that catalyzes the transfer of an acetyl group from acetyl-CoA to various arylamine and hydrazine substrates. This enzyme helps in the metabolization of drugs.

Variations in these genes are associated with higher incidences of drug toxicity

Folate Creation & Pathways

For the methylation cycle to work, there needs to be adequate amounts of folate. Variants along the pathway will reduce the folate in the body. In addition to many other roles in the body, folate is needed to work with B12 to convert homocysteine into methionine, so more SAMe can be made.

Before supporting folate, always make sure there is adequate B12, that the transsulfuration pathway is not going too fast and thus creating glutamate, and glutathione levels are adequate. If there is not enough B12, you can get folate trapping. If CBS is variated, you can create anxiety by making more glutamate, and if phase II (glutathione) is not adequate, the folate can stimulate Phase I and cause inflammation. It is usually best to add folate LAST, after inflammation, CBS, and B12

is properly addressed.

FOLR -> DHFR -> DHF (dihydrofolic acid) -> DHFR -> THF (tetrahydrofolic acid) -> MTHFD1 -> 10-FORMYL THF - > MTHFD1 -> 5-10 Methenyl THF (a form of tetrahydrofolate) MTHFD1 -> 5-10 Methenyl THF (a form of tetrahydrofolate) -> MTHFD1 -> 5-10 Methenylene THF (the substrate used by the enzyme methylenetetrahydrofolate reductase to generate 5-methyltetrahydrofolate) - MTHFR -> 5 -MTHF

Gene Name	Variants	Metrics
Folate Receptor Sites		
FOLR1 (adult) (rs2071010)		GG 88.5%
FOLR2 (fetal) (rs651933)	1	GA 48.9%
FOLR3 (gamma) (rs7925545)		AA 90.7%
Folate Assimilation	83%	

Product Name	SNP Total	Lab Total	Symptoms
Detox Accelerator	1.67	#N/A	#N/A
Methylation Assist Liquescense	2.11	#N/A	#N/A

Folate plays many critical roles in the body, and the first step of folates is the folate receptor sites. Variants in the folate receptor sites will likely reduce the amount of folate absorbed for usage, and increase the need for supplementation. Methylation Assist Liquescence may support the absorption of Folate.

With 1 variant, folate supplementation may be appropriate.

Gene Name	Variants	Metrics
DHFR		
DHFR (rs1643649)		TT 54.6%
DHFR (rs1650697)	1	AG 37.7%
DHFR (rs865646)		GG 47.4%
DHFR A20965G (rs1643659)		TT 54.6%
DHFR C19483A (rs1677693)		GG 54.6%
DHFR	90%	

Product Name	SNP Total	Lab Total	Symptoms
BH4 Assist	3.67	#N/A	#N/A
Pro NADH	1.5	#N/A	#N/A

The DHFR gene is a protein coding gene. DHFR converts dihydrofolate into tetrahydrofolate. DHFR has a key role in cell growth.

Variants here will indicate the need for methyl folate and PRO NADH. BH4 Support and Glutathione Support may also be needed.

With one DHFR variant, folate production may be impaired. FOLR and MTHFR variants along with this can impact folate production. Since DHFR also supports BH2 to BH4 conversion, also check QDPR. This would compound the BH4 recycling.

Gene Name	Variants	Metrics
MTHFR		
MTHFD1 C105T (rs1076991)	1	TC 48.6%
SHMT2 (rs34095989)		GG 38.1%
MTHFS (rs6495446)		CC 54.5%
MTHFR A1298C (rs1801131)		TT 47.7%
MTHFR C677T (rs1801133)	1	GA 45.0%
MTHFR Production (C677T & A1298C)	75%	
Overall Folate Production	83%	

Product Name	SNP Total	Lab Total	Symptoms
BH4 Assist	3.67	#N/A	#N/A
Pro Bioactive Folate	1.67	#N/A	#N/A
MTHFR/BHMT Assist	3.63	#N/A	#N/A
MTHFR/MTR/MTRR/BHMT Assist	0	#N/A	#N/A

It is currently theorized that the C677T impacts the methionine cycle more significantly while the A1298 impacts the BH4 cycle. If this theory is correct, those with C677 would need more support in reducing Homocysteine while A1298C may need more support with creating BH4 and preventing NOS uncoupling. Nonetheless, there is usually a need for folate with these variants. However, to be sure, checking the urine organic acids can confirm, and can also be used as a measure when adequate levels are reached when supplementing.

With 1 heterozygous MTHFR variant, there is a possible need for folate, methylation, and possibly circulation support. Folate production may be about 70%. However, doing the Genova Urine Organic Acid test will verify if there is a need for folate. In some instances, they are compensating OK.

With 3 folate production variants, folate supplementation may be very important.

Methionine Cycle

The Methionine Cycle takes the amino acid methionine, uses the MAT gene to make SAMe. SAMe is the methyl donor that gives a methyl group where it is needed for well over 100 functions. The GAMT gene takes SAMe to make creatine.

After donating a methyl group and making creatine, SAMe turns into SAH and then the AHCY gene turns it into homocysteine.

Variants in MTRR, MTR, BHMT, PEMT will slow the conversion of homocystine into methionine.

Gene Name	Variants	Metrics
MTR (upregulation)		
MTR A2756G (rs1805087)		AA 65.8%
MTR	100%	

Product Name	SNP Total	Lab Total	Symptoms
Pro Hydroxocobalamin	6.25	#N/A	#N/A
Methylation Assist	12.5	#N/A	#N/A

MTR combines folate, Methyl B12 and Homocysteine into Methionine. Variants in MTR are upregulations, so it tries to go faster. MTRR attaches a methyl group to B12, and variants here will slow the process. When both MTR and MTRR exist, dysfunction can occur.

With no MTR variants, the gene is acting normally, in trying to convert Homocysteine into Methionine using methyl B12 and methylfolate. Variants in B12 or folate will impede the process.

Gene Name	Variants	Metrics
MTRR		
MTRR A66G (b12) (rs1801394)		AA 23.0%
MTRR	100%	

Product Name	SNP Total	Lab Total	Symptoms
Pro Hydroxocobalamin	6.25	#N/A	#N/A
Methylation Assist	12.5	#N/A	#N/A
Methylation Assist Liquescense	2.11	#N/A	#N/A

THE MTRR enzyme places a methyl group on B12 so it can be used by MTR to convert Homocysteine into Methionine. Variants here may hamper this function. When combined with other variants that impact the absorption and transport of B12, and if the MTR variant exists (which is trying to make it go faster), this function may be impaired. Supplementation with Methyl B12 is needed (Methylation Assist), unless the individual has excess methyl groups due to other variants such as GAMT and COMT.

There are no variants in MTRR, but B12 levels could still be low if there are other B12 variants, and there may be a need for B12 if the MTR is upregulated. Genova Urine Organic Acids can measure cellular B12.

Gene Name	Variants	Metrics
All B12 Factors		
MTR A2756G (rs1805087)		AA 65.8%
MTRR A66G (b12) (rs1801394)		AA 23.0%
FUT2 (rs492602)	2	GG 22.1%
FUT2 (rs601338)	2	AA 21.5%
FUT2 (rs602662)	2	AA 24.4%
GIF (TCN3) (rs558660)	1	AG 28.7%
TCN1 (rs526934)	2	AA 52.8%
TCN2 C766G (rs1801198)	1	GC 49.9%
B12 Production / Need / Utilization	38%	

Product Name	SNP Total	Lab Total	Symptoms
Pro Hydroxocobalamin	6.25	#N/A	#N/A
Methylation Assist	12.5	#N/A	#N/A

Variants in the FUT genes may decrease probiotics, and hence decrease the absorption of B12. The MTR variant is an upregulation, creating a higher demand for B12, while the MTRR variants reduces the ability to put methyl groups on B12, thus reducing availability of methyl B12.

GIF (gastric intrinsic factor) reduces the absorption of B12, while TCN1 and TCN2 limit the transport of B12. Any combination of higher demand, less absorption and transport will impair the functions dependent upon B12.

FUT variants may create a need for immune support as well.

With ten variants of the SNPs related to B12, supplementation is most likely needed, especially if both the MTR and MTRR have variants.

Gene Name	Variants	Metrics
Choline Usage		
PEMT (rs4244593)		TT 17.0%
PEMT (rs4646406)		TT 25.7%
PEMT (rs7946)	1	CT 40.9%
Choline Production	83%	

Product Name	SNP Total	Lab Total	Symptoms
Glutamate Scavenger/Calming Formula	0	#N/A	#N/A
CBS / BHMT Assist	3.19	#N/A	#N/A
A-L-O Formula	0	#N/A	#N/A

Variants in PEMT can impede choline production. Choline is needed by the liver and brain. PEMT variants have been tied to fatty liver. When there are PEMT variants, use CBS/BHMT with meals. The CBS variants, especially the CBS C699T, will cause homocysteine to rush downstream too quickly, potentially causing high glutamate and ammonia.

With just one variant in PEMT, choline supplementation may not be needed, but may be needed if there are numerous BHMT variants.

Gene Name	Variants	Metrics
PNPLA3		
PNPLA3 (rs738409)		CC 58.3%
PNPLA3	100%	

Product Name	SNP Total	Lab Total	Symptoms
Fatty Acid Assist	1.57	#N/A	#N/A
Nrf2 Accelerator	4.5	#N/A	#N/A

Variants in this gene may cause a predisposition for fatty liver.

BHMT		
BHMT (rs6875201)	1	AG 18.1%
BHMT R239Q (rs3733890)		GG 48.8%
BHMT-02 (rs567754)	2	TT 11.1%
BHMT-08 (rs651852)		CC 27.3%
BHMT	63%	

Variants in the BHMT gene will slow the conversion of homocysteine into methionine. Be aware of BHMT-08 as this may push the homocysteine down faster through the transsulfuration pathway, potentially causing excess glutamate, anxiety attacks, high levels of stress, high cortisol and adrenal fatigue.

With three variants in BHMT support may be needed, especially if there are variants in BHMT-08. This variant will push the homocysteine down through transsulfuration, potentially causing high ammonia, glutamate and anxiety. Also, look at this variant in conjunction with PEMT, which makes the choline needed to be used by the BHMT enzyme to turn homocysteine into methionine.

AHCY		
AHCY-01 (rs819147)		TT 54.4%
AHCY-19 (rs819171)		TT 54.6%
AHCY	100%	

AHCY variants slow the conversion of SAH into homocysteine with no predictable results. It may lower homocysteine and consequently glutathione, but not always. The Doctor's Data serum methylation pathway blood test is quite helpful when these variants are present. SAMe and amino acids with methionine might be contraindicated and N-Acetylcysteine in Glutathione Accelerator may be helpful if cysteine blood levels are low.

With no AHCY variants, it would be likely that S-adenosylhomocysteine would convert properly to homocysteine.

GAMT (Creatine)		
GAMT (rs17851582)		GG 82.7%
GAMT (rs55776826)		CC 74.0%
GAMT (rs80338734)		CC 98.8%
GAMT (Creatine)	100%	

Product Name	SNP Total	Lab Total	Symptoms
GAMT Assist	0	#N/A	#N/A

GAMT is the gene that converts SAMe and other cofactors into creatine, needed for muscle strength. Variants here could lead to muscle weakness. GAMT Assist contains Creatine in a capsule that only opens in the intestinal tract for better absorption. However, use this with caution in kidney disease and hypertension.

No GAMT variants present.

MAT Gene		
MAT1 (rs11595587)		GG 93.3%
MAT1 (rs12242871)	1	GA 48.5%
MAT1 (rs1819684)		GG 83.1%
MAT1 (rs1985908)		AA 46.3%
MAT1 (rs2993763)	1	GA 50.0%
MAT1 (rs4934028)	1	GA 49.5%
MAT1 (rs7081756)	2	TT 41.8%
MAT1 (rs756208)	1	AG 42.0%
MAT	63%	

The MAT Gene turns methionine into SAMe. Variants here may decrease the production of SAMe and create high methionine. This can cause some serious health problems, including neurological issues. If there are a lot of variants, consider doing the Doctor's Data Methylation Plasma test. This is especially true when there are AHCY and GAMT variants as well. If there is high methionine, supporting BHMT may be contraindicated. Glycine (GSH Assist) and reducing foods high in methionine may be helpful.

There are six variants in the MAT gene, so as long as there is adequate methionine and ATP, SAMe should be produced adequately, but there may be some issues. To find out for sure, you may want to do the Doctors Data Blood Plasma test to measure Methionine, SAMe, SAH, and Homocysteine. Glycine, supporting ATP production may support the production of SAMe.

Transsulfuration Pathway

The transsulfuration pathway takes homocosysteine, and pulls it down into glutathione, ammonia, cortisol, sulfites and sulfates.

If there are variants that cause less than optimal conversion of homocysteine back into methionine, and then if there are variants in the CBS genes, especially the CBS699, then homocystine can travel too fast down the transsulfuration pathway and create glutamate, which can cause stress and anxiety. Variants in the GAD genes can worsen the problem.

CBS variants can also create excess ammonia, that can be worse if the urea cycle is less than optimal. The excess ammonia can cause mental stress, sleeping problems, and deplete the much needed BH4, needed for neurotransmitter production.

The excess glutamate can also raise cortisol levels, and eventually lead to adrenal fatigue.

To support this function, you need to support both pathways that convert homocysteine into methionine. One can use Calming Formula/Glutamate Scavenger, reducing the glutamate.

Check sulfites and sulfates to see if SUOX is overwhelmed.

Gene Name	Variants	Metrics
Transsulfuration		
CBS A13637G (rs2851391)	1	TC 47.1%
CBS C19150T (rs4920037)		GG 61.5%
CBS C699T (rs234706)		GG 45.5%
CBSA360A (rs1801181)	1	GA 45.3%
CTH (rs1021737)	1	GT 40.8%
CBS	75%	

Product Name	SNP Total	Lab Total	Symptoms
Glutamate Scavenger/Calming Formula	0	#N/A	#N/A
CBS / BHMT Assist	3.19	#N/A	#N/A
Ammonia Scavenger	5.62	#N/A	#N/A

The CBS gene is similar to a brake, governor, or a dam in the river. It allows homocysteine to move down the transsulfuration pathway at an appropriate pace. Although still being researched, it is believed that some variants in CBS, and especially the CBS 699, cause the homocysteine to move down too quickly, potentially to stress SUOX, and to create excess glutamate that could create anxiety, ammonia and adrenal stress. Checking the urine sulfite and sulfate levels may give clues to what is occurring.

With two variants in the CBS gene, it is likely that homocysteine is coming down the Transsulfuration Pathway at an appropriate pace, unless it is the CBS 699T. This variant may substantially speed up this pathway, causing excess ammonia, sulfites, sulfates, glutamate and stimulating the adrenals with excess cortisol. Other contributing factors could be that if the conversion of homocysteine into methionine is impaired by variants in PEMT, BHMT, MTR, MTRR and MTHFR, CBS may be overwhelmed.

Gene Name	Variants	Metrics
Ammonia & Glutamate Production Estimates		
BHMT-08 (rs651852)		CC 27.3%
CBS C699T (rs234706)		GG 45.5%
Potential Ammonia & Glutamate Clearing Ability	100%	

Product Name	SNP Total	Lab Total	Symptoms
Glutamate Scavenger/Calming Formula	0	#N/A	#N/A
CBS / BHMT Assist	3.19	#N/A	#N/A
Ammonia Scavenger	5.62	#N/A	#N/A

Estimated production of ammonia and glutamate in this calculation is based on variants in BHMT-08 and CBS 699, thus potentially creating glutamate and ammonia. Measuring sulfites and sulfates can give some clues (higher levels would go along with higher glutamate and ammonia levels). The Genova urine organic acids test can give estimates of ammonia and blood tests can measure ammonia as well. Excitability, anxiety and high cortisol levels would go along with high glutamate. Checking cortisol levels may be helpful in the assessment.

With no variants in CBS699 and BHMT-08, there is less chance for excess glutamate and ammonia from excessive Homocysteine being pulled down transsulfuration pathway. However, ammonia can also be high from a weakened Urea Cycle and digestive issues. If they present with a lot of ammonia symptoms, consider checking the Genova Urine Organic Acids.

Neurotransmitters - Serotonion, Dopamine, Glutamate, GABA

Neurotransmitters impact many functions beside emotions. Serotonin and GABA are generally considered relaxing, while dopamine, norepinephrine and epinephrine are considered excitatory.

Variants in MAO may be sparing to serotonin, and may be helpful if there is low production due to low BH4. Variants in COMT can cause a myriad of issues, as the COMT enzyme uses methyl groups to break down dopamine, and is also involved in other detox functions. Variants in COMT has the potential to cause excess methyl groups in the body, thus negative reactions to methyl folate and methyl B12.

The GAD genes convert glutamate to GABA. If there is an overproduction of glutamate, as well as variants in GAD (especially homozygous), the patient/client may experience severe anxiety.

DAO variants may also create overexcitement in the brain, and may contribute to ammonia production as well.

BH4 is needed to create neurotransmitters. See the next page for BH4 production estimates, as low BH4 may impact neurotransmitter production.

Gene Name	Variants	Metrics
Serotonin		
MAO A (R297R) (rs6323)	1	GT 50.2%
MAO A	50%	

The MAO enzyme breaks down serotonin. Variants will actually preserve serotonin. This can be helpful when there is low BH4, poor availability of amino acids that are the precursors to high peroxynitrite.

Note: Males only have the potential for 1 MAO SNP. Currently, the software prints a 2 when there is one for males. Females will print 0, 1 or 2. This will be modified soon, and only reflect 0 or 1 for males.

There is one variant in MAO. This may impact the breakdown of serotonin.

Dopamine		
COMT (MIR4761) (rs6269)	1	AG 46.8%
COMT H62H (MIR4761) (rs4633)	1	CT 49.0%
COMT V158M (MIR4761) (rs4680)	1	GA 48.8%
COMT-61 P199P (mood swings) (rs769224)		GG 95.7%
COMT	63%	

COMT is involved in breaking down excitatory neurotransmitters and detox reactions. Click on the enzyme rating for more information.

Glutamate Production Factors		
BHMT-08 (rs651852)		CC 27.3%
CBS C699T (rs234706)		GG 45.5%
Potential Glutamate Reduction Ability	100%	

Product Name	SNP Total	Lab Total	Symptoms
Glutamate Scavenger/Calming Formula	**0**	**#N/A**	**#N/A**

If the theory is correct that BHMT-08 and CBS699T causes Homocysteine to be converted into Glutamate, this estimate may give some clues if this needs support. Checking sulfite and sulfate urine levels, and cortisol will give additional information. Glutamate Scavenger/Calming Formula may help with the excess glutamate, SUOX Assist may be needed for supporting the sulfite to sulfate process, and Ammonia Scavenger may be needed if the urea cycle is overwhelmed and cannot clear all the ammonia. CBS/BHMT Assist will support the conversion of Homocysteine into Methionine.

There are no BHMT-08 or CBS699T variants. This lessens the chance of excess glutamate, but does not completely rule it out.

GABA (Glutamate to GABA)		
GAD1 (rs3749034)		GG 59.3%
GAD1 (rs2241165)	2	TT 54.2%
GAD1 (rs769407)	2	CC 6.8%
GAD1 (rs2058725)		TT 56.8%
GAD1 (rs3791851)	2	CC 6.8%
GAD1 (rs3791850)		GG 57.9%
GAD1 (rs12185692)		CC 35.7%
GAD1 (rs3791878)	2	TT 9.7%
GAD1 (rs10432420)		GG 49.7%
GAD1 (rs3828275)		CC 32.3%
GAD1 (rs701492)	2	TT 7.9%
Glutamate to GABA Conversion	55%	

The GAD enzyme converts glutamate to GABA. When someone has high glutamate and a lot of variants in GAD, it creates conditions that may have high glutamate and low GABA that could increase stress and conditions related to high glutamate. It has been observed, that Homozygous variants in GAD have more impact that many Heterozygous. SER-GAB Assist and GABA Assist may be helpful if there is low GABA.

GLS (Glutamine to Glutamate Conversion)		
GLS (rs1517354)	2	TT 84.5%
GLS (rs1921915)	2	AA 87.0%
GLS (rs3088307)	2	GG 18.4%
GLS (rs3771311)		TT 77.7%
GLS (rs3771316)		AA 81.8%
GLS (rs6758866)	2	GG 33.5%
GLS (rs62179862)		AA 95.3%
GLS (Glutamine to Glutamate Conversion)	43%	

GLS or Glutaminase encodes a protein that catalyzes the hydrolysis of glutamine to glutamate and ammonia.

GLS2 (Glutamine to Glutamate Conversion)		
GLS2 (rs2638315)	2	CC 3.8%
GLS2 (rs6581096)		GG 48.1%
GLS2 (Glutamine to Glutamate Conversion)	50%	

GLS2 or Glutaminase 2 encodes a protein that catalyzes the hydrolysis of glutamine to stoichiometric amounts of glutamate and ammonia.

GLUL (Glutamate to Glutamine Conversion)		
GLUL (rs12403634)	1	CT 29.1%
GLUL (rs12735664)	1	AC 17.3%
GLUL (Glutamate to Glutamine Conversion)	50%	

GLUL or Glutamate-ammonia Ligase encodes a protein that catalyzes the synthesis of glutamine from glutamate and ammonia in an ATP-dependent reaction.

Glutamate to Alpha-Ketoglutarate Conversion		
GLUD1 (rs9421574)	1	CT 16.6%
GLUD1 (rs1923939)	1	AG 34.2%
GLUD1 (rs9421580)	1	CT 33.2%
GOT1 (rs12768505)		CC 88.8%
GOT1 (rs2234971)	1	CT 21.6%
GOT1 (rs9971274)		GG 84.4%
GOT1 (rs9971275)		GG 84.3%
GOT1 (rs11190083)		AA 84.3%
GOT1 (rs4328160)		TT 81.3%
GOT1 (rs3793935)	2	TT 69.5%
GOT2 (rs30842)	1	AC 43.4%
GOT2 (rs30838)	1	TC 43.3%
GOT2 (rs863944)	1	AC 48.0%
GPT (rs1063739)	2	AA 22.0%
Glutamate to Alpha-Ketoglutarate Conversion	63%	

Product Name	SNP Total	Lab Total	Symptoms
Pro Alpha Ketoglutarate Plus	0	#N/A	#N/A

GLUD1 or Glutamate Dehydrogenase 1 encodes glutamate dehydrogenase which catalyzes the oxidative deamination of glutamate to alpha-ketoglutarate and ammonia. This enzyme has an important role in regulating amino acid-induced insulin secretion.

Glutamic-oxaloacetic transaminase is a pyridoxal phosphate-dependent enzyme which exists in cytoplasmic and inner-membrane mitochondrial forms, GOT1 and GOT2, respectively. GOT plays a role in the conversion of glutamate to alpha-ketoglutarate.

Glutamic-Pyruvate Transaminase also plays a role in the conversion of glutamate to alpha-ketoglutarate.

DAO		
DAO (rs2070586)	1	GA 28.1%
DAO (rs3741775)	1	AC 49.3%
DAOA (rs2391191)		GG 38.3%
DAO	67%	

The name of this gene is D-amino-acid oxidase and DAO is the gene's official symbol. Health conditions observed with this variant are: Schizophrenia, Bipolar Disorder, Primary Hyperoxaluria, ALS (Type 18) , Autism and Crohn's Disease.

Studies have found that the A allele in rs2391191 is a possible genetic feature of certain health conditions such as Schizophrenia, and Bipolar Disorder.

With two variants, there may be some stress/anxiety. When combined with high glutamate and GAD variants, even more potential for stress/anxiety.

Oxytocin Receptor		
OXTR (rs2139184)		CC 96.0%
OXTR (rs11706648)	2	CC 10.8%
OXTR (rs237888)		TT 87.8%
OXTR (rs2268492)	1	CT 39.7%
OXTR (rs2268494)		TT 83.6%
OXTR (rs35498753)		TT 78.1%
OXTR (rs237893)	2	GG 19.4%
OXTR (rs11711703)		AA 67.5%
OXTR (rs237901)		GG 100.0%
OXTR (rs237902)		GG 48.3%
OXTR (rs189386)		CC 83.8%
OXTR (rs237906)		CC 100.0%
OXTR (rs237907)		CC 100.0%
OXTR (rs237908)		CC 99.9%
OXTR (rs237915)	2	CC 8.4%
OXTR (rs35413809)		GG 81.9%
OXTR (rs2301261)		CC 82.3%
OXTR (rs9860869)		TT 78.5%
OXTR (rs237897)	2	GG 27.4%
OXTR (rs237887)	2	AA 33.4%
OXTR (rs53576)	2	GG 46.3%
OXTR (rs7632287)		GG 58.8%
OXTR (rs2268491)		CC 75.9%
OXTR (rs2254298)		GG 75.6%
OXTR (rs1042778)	2	TT 14.8%
OXTR (rs13316193)	1	TC 45.6%
OXTR (rs4686302)		CC 77.8%
OXTR	70%	

OXTR or Oxytocin Receptor encodes a protein that belongs to the G-protein coupled receptor family and acts as a receptor for oxytocin. Oxytocin receptors regulate a variety of different behaviors such as stress, anxiety, social recognition, bonding, and maternal behavior.

Variants in this gene can lead to a higher sensitivity to stress, and conduct disorders.

OXTR Empathy		
OXTR (rs53576)	2	GG 46.3%
OXTR Empathy	100%	

For rs53576, studies have shown that individuals with the GG genotypes are more empathetic, can become more attached, feel less lonely, have a decreased level of sociality, employ more sensitive parenting techniques, and have lower rates of autism.

There are two variants in the OXTR rs53576. Variants in this gene have been shown to be associated with people who are more empathetic, feeling less lonely, employ more sensitive parenting techniques, and have lower rates of autism.

BH4 Cycle, Nitric Oxide & Peroxynitrite (Inflammation) Estimates

The NOS enzyme uses BH4 and L-Arginine to create Nitric Oxide, the critical molecule needed for vasodilation and many other factors. If there is inadequate BH4 or variants in NOS, the arginine may instead create the free radical superoxide. Superoxide then combines with nitric oxide to create the very strong oxidizing agent peroxynitrite. This is called NOS uncoupling. NOS uncoupling causes inflammation and may weaken the immune system.

The more factors that lessen BH4, (A1298C, DHFR, QDPR) and the more NOS variants, and the more SOD variants, the higher the likelihood of peroxynitrite production.

Urea cycle dysfunction will contribute to lowering BH4, because BH4 is needed to clear ammonia not removed by the Urea Cycle.

Gene Name	Variants	Metrics
Nitric Oxide & NOS		
NOS2 (rs2297518)		GG 64.1%
NOS2 (rs2274894)	2	GG 38.9%
NOS2 (rs2248814)	2	GG 38.6%
NOS3 (rs1800779)	2	AA 39.3%
NOS3 (rs3918188)	1	CA 46.2%
NOS3 D298E (rs1800783)	2	TT 37.7%
NOS Production	25%	
BH4 Production Factors		
CBS C699T (rs234706)		GG 45.5%
BHMT-08 (rs651852)		CC 27.3%
DHFR (rs1643649)		TT 54.6%
SHMT2 (rs34095989)		GG 38.1%
MTHFR A1298C (rs1801131)		TT 47.7%
BH4	100%	
BH2 to BH4 Conversion		
QDPR (rs1031326)	1	TC 46.1%
QDPR (rs11722315)		CC 67.1%
QDPR (rs12645938)		GG 91.5%
QDPR (rs3796809)	1	GA 37.9%
QPDR	75%	

Product Name	SNP Total	Lab Total	Symptoms
NOS Assist	7.5	#N/A	#N/A
Pro SOD/Catalase Support	7.83	#N/A	#N/A

The NOS enzymes convert L-Arginine and BH4 into Nitric Oxide. Variants in the NOS enzymes and if along with low BH4 will result in the free radical Superoxide being created instead. If there is not enough Superoxide Dismutase to neutralize the superoxide, the superoxide molecules combines with Nitric Oxide to create the very dangerous and damaging Peroxynitrite, in a process called NOS uncoupling.

NOS Assist supports the NOS Enzyme, while Nitric Oxide Accelerator does as well, but has L Arginine. L Arginine may be contraindicated with NOS variants and low BH4. NOS3 D298 may be the most important NOS variant that impacts Nitric Oxide Production.

If there a lot of NOS variants, supporting SOD and Glutathione while scavenging Peroxynitrite may be needed as well. Checking inflammation markers in Urine Organic Acids may be beneficial, to determine if there is excess inflammation.

There are 12 NOS variants. NOS support is highly recommended. BH4 and Peroxynitrite Scavenger may be needed as well.

Product Name	SNP Total	Lab Total	Symptoms
BH4 Assist	3.67	#N/A	#N/A
Pro NADH	1.5	#N/A	#N/A

BH4 is critical for neurotransmitter production and making nitric oxide. Low BH4 can lead to impaired neurotransmitter production and NOS uncoupling, thus resulting in the creation of the very dangerous, peroxynitrite. These variants may lower the production or recycling of BH4.

With no variants that support the production of BH4, production may be adequate. However, check QDPR genes for recycling of BH2 to BH4.

Product Name	SNP Total	Lab Total	Symptoms
BH4 Assist	3.67	#N/A	#N/A
Pro NADH	1.5	#N/A	#N/A

QDPR produces the enzyme quinoid dihydropteridine reductase that recycles BH2 to BH4. Variants here, along with other variants that impact BH4 production, may contribute to NOS uncoupling, where the dangerous Peroxynitrite oxidizing agent is created.

With two variants in QDPR, BH2 to BH4 conversion may be slightly compromised. However, DHFR also impacts BH2 to BH4 and MTHFR1298C supports the folinic acid needed for BH4. All of these need to be taken into consideration.

Peroxynitrite Factors		
MTHFR A1298C (rs1801131)		TT 47.7%
SHMT2 (rs34095989)		GG 38.1%
DHFR (rs1643649)		TT 54.6%
QDPR (rs1031326)	1	TC 46.1%
QDPR (rs11722315)		CC 67.1%
QDPR (rs12645938)		GG 91.5%
QDPR (rs3796809)	1	GA 37.9%
BHMT-08 (rs651852)		CC 27.3%
CBS C699T (rs234706)		GG 45.5%
CTH (rs1021737)	1	GT 40.8%
GSTM1 (rs1056806)		CC 87.9%
GSTP1 A114V (rs1138272)		CC 84.3%
GSTP1 I105V (rs1695)	1	AG 44.4%
SOD2 (rs2758331)	2	AA 22.9%
SOD2 A16V (rs4880)	2	GG 24.4%
SOD3 (rs1799895)		CC 97.7%
SOD3 (rs2855262)	1	TC 45.7%
NOS2 (rs2274894)	2	GG 38.9%
NOS2 (rs2248814)	2	GG 38.6%
NOS3 (rs3918188)	1	CA 46.2%
NOS2 (rs2297518)		GG 64.1%
NOS3 (rs1800779)	2	AA 39.3%
NOS3 D298E (rs1800783)	2	TT 37.7%
Peroxynitrite Reduction Efficiency	55%	

Product Name	SNP Total	Lab Total	Symptoms
NOS Assist	7.5	#N/A	#N/A
Glutathione Accelerator	4.63	#N/A	#N/A
Peroxynitrite Scavenger	4.5	#N/A	#N/A
S Acetyl Glutathione	4.63	#N/A	#N/A
Ammonia Scavenger	5.62	#N/A	#N/A
Peroxynitrite Scavenger P.M.	4.5	#N/A	#N/A
Pro SOD/Catalase Support	7.83	#N/A	#N/A
GSH Assist	4.63	#N/A	#N/A
Nrf2 Accelerator	4.5	#N/A	#N/A
Pro NADH	1.5	#N/A	#N/A

The Peroxynitrite Support Estimate is a summation of all the variants that could contribute to the creation of peroxynitrite. Reducing peroxynitrite may be the most important step you take nutritionally.

The variants listed here are related to those that reduce the creation of BH4 (MTHFR A1298C, SHMT), those that would slow the recycling of BH2 to BH4 (DHFR and QDPR), those that would reduce BH4 by creating excess ammonia (BHMT 08 and CB 699) and the variants that would reduce SOD and glutathione. Also, look at the NOS variants that would make superoxide rather than nitric oxide.

Reviewing this list may give you clues as to how severe peroxynitrite production is, and how suited they are to reduce it with glutathione and SOD and the most appropriate strategies to reduce the peroxynitrite.

Another component to consider, is to view the Urea Cycle function as well, as lowered urea function will cause more BH4 to be used for ammonia reduction.

Vitamin D, Cell Membrane, Intestinal Bacteria, SHBG & Cardiovascular, Iron

These SNPs may be reflective of need for Vitamin D, prebiotics, hormonal support and cardiovascular support.

Patient: First/Last Name

Gene Name	Variants	Metrics
Vitamin D Receptor		
VDR BSM (rs1544410)	2	TT 15.2%
VDR Fok (blood sugar) (rs2228570)		AA 14.1%
VDR Taq (methy group) (rs731236)	2	GG 14.9%
Vitamin D Production (TAQ Only)	0%	

Product Name	SNP Total	Lab Total	Symptoms
Vitamin D3 5000	**10**	**#N/A**	**#N/A**

Variants in the VDR (Vitamin D receptor) Taq may lower the Vitamin D in the body and supplementation is needed. More information will be coming on the VDR FoI and VDR BSM. The Vitamin D rating, is only calculated based upon the TAQ, and not the other Vitamin D genes. More information will be added here on the others in the future.

There are 2 variants. Measure Vitamin D, and consider 10,000 IU/day supplementation.

Gene Name	Variants	Metrics
Cell Membrane Protection		
G6PD (rs1050828)		CC 99.7%
G6PD (rs1050829)		TT 98.9%
G6PD	100%	

Information coming soon.

Gene Name	Variants	Metrics
SHBG		
SHBG (rs1799941)		GG 58.0%
SHBG	100%	

Variants in the SHBG gene may cause dysregulation in testosterone and estrogen levels and lowered progesterone. Hormone testing may be in order if hormonal symptoms exist. For Men (especially older men), SHBG variants may indicate more circulating SHBG resulting in lowered testosterone levels. For women, SHGB variants may indicate less SHBG resulting in higher androgen levels overall.

There are no variants in the SHBG genes. This lessens the changes of hormonal issues from SHBG.

Gene Name	Variants	Metrics
Cardiovascular Genes		
ACE Del 16 (rs4343)	1	GA 49.5%
ADD1 G460W (rs4961)		GG 65.0%
AGT M235T/C4072T (rs699)	2	GG 20.4%
MTHFR C677T (rs1801133)	1	GA 45.0%
Cardio Protection	50%	

Product Name	SNP Total	Lab Total	Symptoms
Circulation Accelerator	**8.66**	**#N/A**	**#N/A**

Variants in these genes may contribute to circulatory issues. More information coming soon.

The more SNPs in these genes, the higher the risk for cardiovascular issues, especially when combined with carnitine and ACAT variants.

Gene Name	Variants	Metrics
HFE		
HFE C282Y (rs1800562)		GG 89.2%
HFE H63D (rs1799945)		CC 74.4%
HFE S65C (rs1800730)		AA 97.2%
HFE (rs1572982)	1	GA 49.1%
HFE 6382T>G (rs2794719)		TT 36.6%
HFE 8828T>C (rs2071303)	1	TC 44.0%
HFE	83%	

Product Name	SNP Total	Lab Total	Symptoms
HFE Assist	**8.35**	**#N/A**	**#N/A**

H63D represents a SNP that accounts for a mild form of hereditary hemochromatosis (HH), an iron overload condition in which mutations of certain genes involved in iron metabolism disrupt the body's ability to regulate uptake of iron, causing increased intestinal iron absorption. The most common form is caused by mutations in the HFE gene, which are inherited recessively.

A mutation at amino acid 282 (C282Y) was found to be homozygous in 83 percent of patients with HH. This is a point mutation from guanine to adenine, resulting in a missense mutation from cysteine to tyrosine. Such mutations are commonly found in people with European ancestry.

The three most common HH-causing mutations in the HFE gene are C282Y and S65C At least 17 other mutations in the HFE gene have been linked to HH. 60-90% of people with HH have two copies of the C282Y mutation. The H63D mutation is also quite common, about 20% of people carry a copy of the mutation, and about 3% have two copies. This mutation is not as severe as the C282Y mutation, and only causes symptoms when someone has both the H63D and the C282Y mutations. Even then, only a small fraction of people with one copy of each mutation actually exhibit evidence of iron overload. Additionally, those who have two copies of H63D do not exhibit any symptoms and are not at risk for iron overload. The S65C mutation is less common, and will also only cause symptoms if in combination with C282Y. For both H63D/C282Y and S65C/C282Y single mutation individuals, symptoms are usually mild if they develop at all.

Iron oxidation Potential		
BHMT-08 (rs651852)		CC 27.3%
CBS C699T (rs234706)		GG 45.5%
CTH (rs1021737)	1	GT 40.8%
GSTM1 (rs1056806)		CC 87.9%
GSTP1 A114V (rs1138272)		CC 84.3%
GSTP1 I105V (rs1695)	1	AG 44.4%
HFE C282Y (rs1800562)		GG 89.2%
HFE H63D (rs1799945)		CC 74.4%
HFE S65C (rs1800730)		AA 97.2%
SOD2 (rs2758331)	2	AA 22.9%
SOD2 A16V (rs4880)	2	GG 24.4%
SOD3 (rs1799895)		CC 97.7%
SOD3 (rs2855262)	1	TC 45.7%
Iron Oxidation Potential	73%	

Product Name	SNP Total	Lab Total	Symptoms
HFE Assist	8.35	#N/A	#N/A
Pro SOD/Catalase Support	7.83	#N/A	#N/A
GSH Assist	4.63	#N/A	#N/A
Pro NADH	1.5	#N/A	#N/A

HFE SNPS, in combination with these others, may increase the potential for Oxidized Iron. More information coming soon.

DNA Repair

DNA repair genes code the proteins whose normal function is to correct errors that arise when cells duplicate their DNA prior to cell division. These errors in the DNA can occur from things such as ultraviolet light, inhaled cigarette smoke, or endogenous weak mutagens.

Mutations in the DNA repair genes can lead to a failure in correcting the DNA, which in turn allows subsequent mutations to accumulate.

If the rate of DNA damage exceeds the capacity of the cell to repair itself, the buildup of errors can overwhelm the cell.

Gene Name	Variants	Metrics
mutL homolog 1		
MLH1 (rs1800734)	2	AA 5.6%
MLH1 (rs35045067)		AA 99.9%
MLH1	50%	

Product Name	SNP Total	Lab Total	Symptoms
Cellular Health Assist	7.74	#N/A	#N/A

The MLH1 gene provides the instructions for making a protein that plays an essential role in DNA repair. This protein helps fix mistakes that are made when DNA is copied in DNA replication in preparation for cell division.

Ataxia telangiectasia mutated		
ATM (rs1801516)		GG 73.8%
ATM (rs664143)	2	GG 34.9%
ATM (rs664677)	2	TT 35.1%
ATM (rs1801673)		AA 98.4%
ATM (rs1800058)		CC 96.4%
ATM (rs1800056)		TT 97.5%
ATM (rs1800054)		CC 97.8%
ATM (rs3218707)		GG 99.7%
ATM (rs3092856)		CC 99.5%
ATM (rs623860)	2	TT 35.1%
ATM (rs2235006)		TT 99.8%
ATM (rs3092857)		AA 99.8%
ATM (rs227060)	2	TT 11.2%
ATM (rs227062)	2	AA 33.3%
ATM (rs17412803)		AA 92.4%
ATM (rs227092)	2	TT 31.8%
ATM (rs600931)	2	TT 33.2%
ATM (Most Relevant)	78%	
ATM (All Genes)	59%	

The main role of ATM is to repair double-stranded DNA breaks.

The first 9 are shown to be the most relevant, and the last 9 are included for informational research purposes.

Studies have shown that variants in the most relevant genes listed can increase chances of cells being damaged from oxidative stress and not repairing as quickly. It would be advantageous for individuals with these variants to reduce exposure to free radical producing agents and support anti-oxidant protection.

For now, we have no suggested protocols, other than adequately controlling oxidative stress. This information is just being presented now for research purposes.

Urea Cycle

Ammonia is the product of oxidative deamination reactions and is a toxin even in small amounts and must be removed from the body. The urea cycle facilitates the removal of ammonia as urea. The ammonia is first converted into urea in the liver. After conversion, the urea is then transported to the kidneys where it is excreted.

A urea cycle disorder can occur if there is a mutation that results in a deficiency of CPS1, OTC, ASS1, ASL, or ARG1 which could result in higher ammonia concentration in the blood.

Gene Name	Variants	Metrics
Carbamoyl-Phosphate Synthase 1		
CPS1 (rs918233)	2	TT 42.7%
CPS1 (rs1509821)		CC 81.6%
CPS1 (rs981024)		GG 36.5%
CPS1 (rs2012564)		AA 35.6%
CPS1 (rs17773128)		CC 85.4%
CPS1 (rs6749597)		GG 74.3%
CPS1 (rs2887913)		AA 36.5%
CPS1 (rs9789405)		CC 74.3%
CPS1 (rs2287603)		AA 64.4%
CPS1 (rs2287602)		AA 74.6%
CPS1 (rs10515951)		GG 85.7%
CPS1 (rs6714124)		CC 25.9%
CPS1 (rs7573258)		GG 18.1%
CPS1 (rs2371000)		TT 18.0%
CPS1 (rs2371001)		AA 25.7%
CPS1 (rs3821135)		TT 76.9%
CPS1 (rs7607205)		TT 35.7%
CPS1 (rs12468557)		CC 40.6%
CPS1 (rs2302909)		GG 84.5%
CPS1 (rs2371011)	2	GG 8.7%
CPS1 (rs13010236)		TT 82.6%
CPS1 (rs2287598)	2	GG 67.8%
CPS1 (rs6435580)		CC 45.6%
CPS1 (rs2270476)		GG 88.2%
CPS1 (rs12997383)		CC 75.6%
CPS1 (rs4672587)	2	GG 10.3%
CPS1 (rs4567871)		CC 75.8%
CPS1	85%	

Product Name	SNP Total	Lab Total	Symptoms
Ammonia Scavenger	**5.62**	**#N/A**	**#N/A**
A-L-O Formula	**0**	**#N/A**	**#N/A**

The enzyme encoded by this gene catalyzes the synthesis of carbamoyl phosphate from ammonia and bicarbonate. This synthesis is the first step in the Urea Cycle.

Carbamoyl phosphate is an intermediary metabolite in nitrogen disposal.

A mutated CPS1 gene may result in a carbamoyl phosphate synthetase I enzyme that is smaller than normal, not correct in shape, or the enzyme may not be produced at all.

Studies have shown that polymorphisms in the CPS1 gene have been associated with pulmonary hypertension. Polymorphisms in CPS1 may also reduce the production of nitric oxide (NO). A reduced amount of nitric oxide can also lead to circulatory problems.

Gene Name	Variants	Metrics
Ornithine Transcarbamylase		
OTC (rs72554348)		GG 99.9%
OTC (rs7056866)	1	GA 49.1%
OTC (rs5917584)	1	CT 31.2%
OTC (rs5963418)		GG 78.7%
OTC (rs5963419)		TT 47.3%
OTC (rs12557315)	1	CT 30.3%
OTC	75%	

Product Name	SNP Total	Lab Total	Symptoms
Ammonia Scavenger	**5.62**	**#N/A**	**#N/A**
A-L-O Formula	**0**	**#N/A**	**#N/A**

The OTC gene is responsible for providing the instructions for making the enzyme ornithine transcarbamylase Ornithine transcarbamylase controls the reaction between carbamoyl phosphate (From the first step of the urea cycle) with ornithine to form citrulline.

A mutated OTC gene will not be able to control the reaction between carbamoyl phosphate and ornithine correctly. This in turn can cause a buildup of ammonia in the body.

Argininosuccinate synthase 1		
ASS1 (rs12554609)		TT 84.4%
ASS1 (rs11243372)		AA 32.3%
ASS1 (rs4740158)	2	CC 55.5%
ASS1 (rs914983)	2	GG 45.1%
ASS1 (rs1615006)		GG 11.4%
ASS1 (rs1653332)		GG 16.2%
ASS1 (rs1215988)		GG 39.0%
ASS1 (rs1215985)	2	TT 56.1%
ASS1 (rs590086)	2	TT 81.7%
ASS1 (rs12551145)		GG 86.1%
ASS1 (rs10901072)		CC 77.6%
ASS1 (rs652313)	2	GG 74.8%
ASS1 (rs1215972)	2	AA 76.0%
ASS1 (rs41302903)	1	GA 11.3%
ASS1 (rs75912463)		TT 97.8%
ASS1 (rs540140)		GG 51.8%
ASS1 (rs480313)		GG 51.6%
ASS1 (rs11243474)		GG 75.2%
ASS1 (rs474330)		GG 47.8%
ASS1 (rs17147023)		TT 59.0%
ASS1 (rs553696)		AA 45.5%
ASS1 (rs12375699)	2	TT 19.2%
ASS1 (rs634432)	2	TT 71.7%
ASS1 (rs544701)		AA 76.1%
ASS1	65%	

Product Name	SNP Total	Lab Total	Symptoms
Ammonia Scavenger	5.62	#N/A	#N/A
A-L-O Formula	0	#N/A	#N/A

The ASS1 gene is responsible for providing the instructions for making the enzyme argininosuccinate synthase 1. Argininosuccinate synthase 1 controls the reaction between the two amino acids citrulline (From the second step of the urea cycle) and aspartate to form argininosuccinic acid.

A mutated ASS1 gene can prevent the liver from processing excess nitrogen into urea. This in turn can cause a buildup of ammonia and other byproducts of the urea cycle (for example citrulline) in the bloodstream.

Argininosuccinate Lyase		
ASL (rs12530898)		GG 91.7%
ASL (rs313830)	1	TC 37.6%
ASL (rs313829)	1	AG 40.8%
ASL	67%	

Product Name	SNP Total	Lab Total	Symptoms
Ammonia Scavenger	5.62	#N/A	#N/A
A-L-O Formula	0	#N/A	#N/A

The ASL gene is responsible for providing the instructions for making the protein argininosuccinate lyase. Argininosuccinate lyase creates arginine and fumarate from argininosuccinate acid (From the third step of the urea cycle). The arginine is later broken down into urea and is excreted from the body.

A mutated ASL gene may not be able to form arginine and fumarate properly. This could lead to a buildup of ammonia in the blood.

Arginase 1		
ARG1 (rs2246012)		TT 70.7%
ARG1	100%	

Product Name	SNP Total	Lab Total	Symptoms
Ammonia Scavenger	5.62	#N/A	#N/A
A-L-O Formula	0	#N/A	#N/A

The ARG1 gene is responsible for providing the instructions for making the enzyme arginase. Arginase controls the last step of the urea cycle. In this step, arginase removes nitrogen from arginine (From the fourth step in the urea cycle) and converts this nitrogen into urea to be excreted from the body. Ornithine is also produced in this reaction which is then used to repeat the cycle.

A mutated ARG1 gene may not be able to from a stable arginase enzyme. This can cause a build of ammonia and arginine in the body.

Variants that Impact Exercise and Fitness Potential

Gene Name	Variants	Metrics
Muscle Fiber Composition		
ACTN3 (rs1815739)		CC 31.8%
Muscle Fiber Composition	100%	

Muscles are made up of two main types of muscle fibers, "fast-twitch" and "slow-twitch." Endurance athletes tend to have more slow-twitch muscle, while sprinters tend to have more fast-twitch muscle. Some of the variation in muscle fibers is dependent on a protein called alpha-actinin-3.

The ACTN3 gene contains instructions for making alpha-actinin-3. The alpha-actinin-3 protein can be found in certain types of fast-twitch muscle fibers. People who make this protein tend to have a greater proportion of fast-twitch muscle and are better sprinters than people who do not make this protein.

With 0 variants in the ACTN3 gene, this person has fast-twitch muscle fiber and is likely a sprinter.

Aerobic Exercise Potential		
ADRB2 (rs1042713)	2	GG 38.6%
ADRB2 (rs1042714)	2	GG 16.9%
PPARGC1A (rs8192678)	2	TT 11.0%
VEGF (rs833069)	1	CT 43.6%
Aerobic Exercise Potential	13%	

Our bodies need oxygen while exercising. VO2 max is a test that can be used by scientist to measure the optimum rate at which someone’s body can effectively use oxygen when exercising. There are certain genes that can help at better understanding someone’s natural VO2 max capacity.

Studies have shown that individuals with a higher number of variants in these genes are less responsive to endurance training.

Exercise Recovery Speed		
CRP (rs1205)	2	CC 43.9%
IL6 (rs1800795)		GG 38.7%
IL6R (rs4129267)	1	TC 48.1%
SOD2 A16V (rs4880)	2	GG 24.4%
TNFA (rs1800629)		GG 72.0%
Exercise Recovery Speed	50%	

Research has shown that certain genetic factors can determine whether or not someone can quickly recover after workouts.

Studies have shown that individuals with variations in these genes require longer recovery times due to higher levels of inflammation during strenuous exercise.

Exercise Injury Risk		
COL1A1 (rs1800012)	2	CC 67.9%
COL5A1 (rs12722)	1	CT 46.9%
GDF (rs224329)		CC 36.9%
Exercise Injury Risk	50%	

Research has shown that certain genetic factors can determine an individuals exercise injury risk.

Studies have shown that individual's with variations in these genes are at higher risk for tendon and ligament injuries.

FTO		
FTO	52%	

FTO or Fat Mass and Obesity Associated, is a Protein Coding gene. Variations in this gene may cause growth delay, developmental delay, facial dysmorphism and overnutrition.

All 63 of the FTO SNPs can be viewed in the Gene Report.

FADS		
FADS1 (rs174546)		CC 45.1%
FADS1 (rs174547)		TT 45.1%
FADS1 (rs174548)		CC 49.2%
FADS1 (rs174549)		GG 50.6%
FADS1 (rs174550)		TT 45.1%
FADS1 (rs174556)		CC 50.8%
FADS2 (rs174570)		CC 73.2%
FADS2 (rs1535)		AA 44.6%
FADS2 (rs174575)		CC 55.9%
FADS2 (rs174576)		CC 43.4%
FADS2 (rs2072114)		AA 77.5%
FADS2 (rs174579)		CC 63.3%
FADS2 (rs2851682)		AA 81.9%
FADS2 (rs174592)		AA 39.7%
FADS2 (rs174602)		TT 60.6%
FADS2 (rs498793)		TT 16.7%
FADS2 (rs174611)		TT 51.9%
FADS2 (rs482548)		CC 82.2%
FADS3 (rs174450)	2	TT 27.4%
FADS3 (rs1000778)	2	GG 53.7%
FADS1	100%	
FADS2	100%	
FADS3	0%	
FADS Total	90%	

The proteins encoded by the FADS1, FAD2, and FADS3 genes are members of the fatty acid desaturase (FADS) gene family. Desaturase enzymes regulate the unsaturation of fatty acids through the introduction of a double bond between the carbons of the fatty acyl chain.

A fatty acid is a carboxylic acid with a long aliphatic chain. This aliphatic chain can either be saturated or unsaturated. Fatty acids that have carbon-carbon double bonds are known as unsaturated. Fatty acids without double bonds are known as saturated.

Fatty acids are usually derived from triglycerides or phospholipids. Fatty acids are important sources of fuel because, when they are metabolized, they yield large quantities of ATP. Fatty acid composition in membranes plays an important role in cellular processes. Many cell types can use either glucose or fatty acids for this purpose.

Variations in these genes may affect long-chain polyunsaturated fatty acids metabolism.

Electrical Sensitivity Potential

Gene Name	Variants	Metrics
CACNA1C		
CACNA1C (rs216013)		AA 70.5%
CACNA1C (rs2159100)		CC 45.5%
CACNA1C (rs1006737)		GG 45.4%
CACNA1C (rs2302729)	1	CT 29.2%
CACNA1C	88%	

This gene encodes an alpha-1 subunit of a voltage-dependent calcium channel. Calcium channels mediate the influx of calcium ions into the cell upon membrane polarization.

Variants in these genes may impact the potential to have negative effects from high levels of electrical field exposure.

Lyme Study Phase I SNPs

These are the SNPS that were found to be most prevalent for individuals with chronic Lyme, and is not a test for Lyme. Inherited genetic mutations when expressed may reduce enzyme production. This can lead to, nutrient deficiencies, an increased production of free radicals or other toxic substances, or a slow clearing of toxic substances. Any one of these or a combination of may have the potential to allow Lyme to be resistant to traditional treatment by suppressing the immune system or susceptible to creating toxic conditions.

Gene Name	Variants	Metrics
HFE and Potential Hydroxyl Radical Production SNPs		
HFE C282Y (rs1800562)		GG 89.2%
HFE H63D (rs1799945)		CC 74.4%
CBS C699T (rs234706)		GG 45.5%
BHMT-08 (rs651852)		CC 27.3%
SOD2 (rs2758331)	2	AA 22.9%
SOD2 A16V (rs4880)	2	GG 24.4%
GSTP1 A114V (rs1138272)		CC 84.3%
GSTP1 I105V (rs1695)	1	AG 44.4%
CTH (rs1021737)	1	GT 40.8%
PEMT (rs4244593)		TT 17.0%
PEMT (rs7946)	1	CT 40.9%
PEMT (rs4646406)		TT 25.7%
HFE and Potential Hydroxyl Radical Production SNPs	71%	

The following SNPs may increase the potential of the Fenton Reaction and those with Lyme had a higher number of SNPs in each of the genes. CBS699 and BHMT-08 may increase the cysteine, while the glutathione variants may slow the conversion of cysteine into glutathione. Variants in SOD genes may slow the ability to neutralize the hydroxyl radicals.

Further research is needed to determine if iron oxidation from the Fenton Reaction is a contributing factor to those with chronic Lyme, and if nutritional interventions with nutrients that may slow iron absorption, regulate iron, support cysteine to glutathione conversion and NADH to recycle glutathione and superoxide dismutase may be an appropriate holistic support.

Gene Name	Variants	Metrics
Mitochondrial Function SNPs		
SLC22A5 (rs17622208)	1	GA 48.1%
SLC22A5 (rs2073643)	1	TC 49.1%
SLC22A5 (rs1045020)		CC 79.0%
ACAT-2 (rs3465)		GG 38.9%
ACAT-2 (rs3798211)	2	CC 31.1%
ACAT-2 (rs25683)	2	GG 31.4%
NDUFS7 (rs1142530)	1	CT 46.0%
Mitochondrial Function SNPs	50%	

The following SNPs relate to mitochondrial function.

These findings may suggest that lowered energy production in the Krebs Cycle, may be a contributing factor to Chronic Lyme. Further studies of these findings are needed to confirm if these observations are clinically relevant, and if nutritional intervention with carnitine, choline, NADG, CoQ10 and pantethene may be a useful therapy when these variants are present.

Methylation Cycle SNPs			These findings may suggest that an increased amount of SNPs in the MTHFR gene, in particular, and the entire Methylation pathway, may be a contributing factor in chronic Lyme. Further studies of these findings are needed to confirm if these observations are clinically relevant. As a result of these observations, further analysis on a larger scale, and other lab testing may be warranted to see if these observed variants play a role in Chronic Lyme Disease and if supplementation of methyl folate, methyl B12, choline, B6, TMG and SAMe may be helpful holistic therapies.
MTHFR C677T (rs1801133)	1	GA 45.0%	
MTHFR A1298C (rs1801131)		TT 47.7%	
Methylation Cycle SNPs	75%		
Urea Cycle SNPs			The following SNPs relate to the Urea Cycle. As a result of these observations, larger scale testing and associated lab work may be needed to see if these variants create increased ammonia burden and are clinically significant in those with Chronic Lyme Disease, and if supporting the Urea Cycle and ammonia clearance would be an appropriate nutritional therapy. Digestive support therapies that reduce ammonia may be appropriate as well.
CPS1 (rs1509821)		CC 81.6%	
CPS1 (rs6435580)		CC 45.6%	
CPS1 (rs12468557)		CC 40.6%	
CPS1 (rs7607205)		TT 35.7%	
ASS1 (rs12375699)	2	TT 19.2%	
ARG2 (rs3742879)	1	AG 40.2%	
ARG2 (rs742869)	1	GA 47.8%	
Urea Cycle SNPs	71%		
Detoxification SNPs			The following SNPs relate to detoxification. As a result of these observations, larger scale testing and associated lab work may be needed to see if these variants are clinically significant in those with Chronic Lyme Disease, and if supporting the detox mechanisms controlled by CYP, PON1, SOD, and glutathione would be an appropriate nutritional therapy. Additional nutritional therapies that scavenge peroxynitrite should be investigated as well.
CYP1A1*4 C2453A (rs1799814)		GG 91.4%	
CYP1B1 N453S (rs1800440)		TT 67.9%	
PON1 (rs854561)	2	TT 12.9%	
SOD2 (rs2758331)	2	AA 22.9%	
GSTP1 A114V (rs1138272)		CC 84.3%	
Detoxification SNPs	60%		
Glutamate SNPs			The following SNPs relate to glutamate. As a result of these findings, future research may be needed to see if higher glutamate levels and peroxynitrite are associated with symptoms related to Lyme Disease, or if supporting the conversion into GABA may be a part of a holistic treatment plan.
GAD1 (rs3791850)		GG 57.9%	
GAD1 (rs3828275)		CC 32.3%	
GAD1 (rs12185692)		CC 35.7%	
GAD1 (rs3791878)	2	TT 9.7%	
Glutamate SNPs	75%		
DNA Repair SNPs			If those with chronic Lyme disease have higher rates of oxidative stress due to mitochondrial dysfunction, lowered ability to detox, iron oxidation, etc., higher rates of variants in the ATM genes may also play a contributing role. Further research may be warranted.
ATM (rs1801516)		GG 73.8%	
DNA Repair SNPs	100%		
Total Lyme Study SNPs			
Total Lyme Study Phase I SNPs	67%		