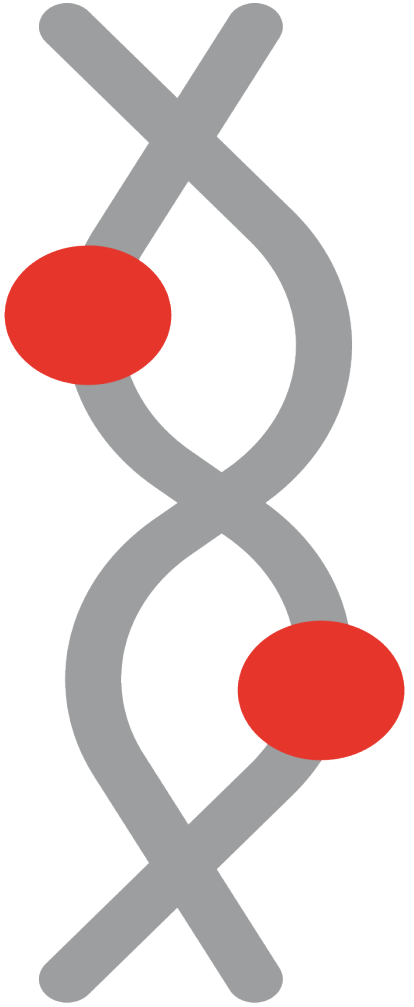
Fagron genomics



Gene Comprehensive Nutrigenomic Report

Accession Number: ##########
Specimen Collected: ##/##/####
Specimen Received: ##/##/####
Report Generated: January 11, 2023
Specimen Type: Buccal Swab
Provider: ######## ########
Patient Name: ###### ######
Patient DOB: ##/##/####
Patient Gender: Female

Do not make any decisions about your health solely based on the information contained in this report. Always consult with a licensed and experienced health practitioner when you receive this report.

GX Sciences | 844-258-5564 | www.GXSciences.com
Lab | 4150 Freidrich Lane, Ste H | Austin, TX 78744
Laboratory Director: James W. Jacobson, Ph.D

– 9 – Female

(-/-) No clinical abnormality | (+/-) Heterozygous result | (+/+) Homozygous result

rsID	Gene	Genetic Result	Therapeutics Associated With Positive Result	Highly Recommended Therapeutics	Provider Discretion: As Needed Formula Recommendations	Lifestyle Recommendations	Laboratory Recommendations
Early Childhood							
Neurotrophic Factors							
rs1142636	SYN1	-/-	RG3, Nicotinamide Riboside, Ginseng				
rs6265	BDNF	-/-	Curcumin, Lithium Orotate, D-Chiro-Inositol, Catechins, Resveratrol, Exercise				
rs6330	NGF	-/-					
Neuro-Inflammation							
rs10402876	C3	+/+	Anti-Inflammatory Therapy: Curcumin, Omega 3s, Resveratrol, Quercetin, Low Dose Naltrexone (LDN), CBD Oil	**CBD Oil** **PEA Soothe Support™** **Prescription Low Dose Naltrexone (LDN)**		Consider Low Inflammatory Diet	Consider Pregnenolone, Cortisol, T cell profile, Routine Thyroid Panel, Candida Titer, Food Allergy Panel, Environmental Allergy Testing
rs2569191	CD14	+/-					
rs2069812	IL5	+/-					
rs1800795	IL6	+/-					
rs1800925	IL13	-/-					
rs10181656	STAT4	+/-					
rs1800629	TNF	-/-					
rs231775	CTLA4	+/+					
rs1076560	DRD2	-/-	Increased Efficacy of Naltrexone				
External Inflammatory							
rs10156191	AOC1	-/-	Poor Ability To Break Down Histamine in Foods				
rs11558538	HMNT	-/-					
rs12995000	HMNT	-/-					
rs492602	FUT2	+/+	Pre-biotics and Probiotics Needed		Biotic Boost Chews or Powder For Kids™		Consider Microbiome Testing If GI Inflammation Present

rs2187668	HLA DQA1	-/-	High Risk of Severe Gluten Sensitivity				
rs2858331	HLA DQA2	-/-					
rs660895	HLA DRB1	-/-	High Reactivity To Mold / Fungi			Highly Recommend Avoiding Mold / Fungal / Yeast Exposure	Consider Candida Titers and MSH (Melanocyte Stimulating Hormone)
rs9275224	HLA DRB2	+/+					

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rsID	Gene	Genetic Result	Therapeutics Associated With Positive Result	Highly Recommended Therapeutics	Provider Discretion: As Needed Formula Recommendations	Lifestyle Recommendations	Laboratory Recommendations
Early Childhood							
Autophagy							
rs510432	ATG5	+/-	Curcumin, Lithium Orotate, D-Chiro-Inositol, Catechins, Resveratrol, Caffeine, 12 Hour Fasting	**DCI 500 or Metabolic Stimulator™** **N.A.S. Enhancer™**		12-15 Hour Fasting if appropriate for age	Routine Blood Sugar, Insulin and Hb A1c
rs26538	ATG12	+/-					
rs10210302	ATG16L1	-/-					
Detoxification							
rs1021737	CTH	-/-	N-Acetyl Cysteine (NAC), Glutathione				
rs7483	GSTM3	-/-	Glutathione				
rs1695	GSTP1 I105V	-/-					

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rsID	Gene	Genetic Result	Therapeutics Associated With Positive Result	Highly Recommended Therapeutics	Provider Discretion: As Needed Formula Recommendations	Lifestyle Recommendations	Laboratory Recommendations
Early Childhood							
Methylation and Folate Metabolism							
rs2071010	FOLR1	-/-	Methyltetrahydrofolate (5-MTHF)	**Methyl Folate Plus™ Twice Daily**			
rs651933	FOLR2	+/-					
rs1643649	DHFR	-/-					
rs6495446	MTHFS	+/-					
rs1076991	MTHFD1	-/-					
rs1801131	MTHFR A1298C	+/-					
rs1801133	MTHFR C677T	+/-					
rs1051266	SLC19A1	+/+					
Methlyation and B12 Metabolism							
rs1805087	MTR	-/-	L-5-Methyl THF, Methyl Cobalamin, Nicainamide (B3), Methionine				
rs1802059	MTRR A664A	-/-	Methyl B12	**Methylation Pro Topical™ OR Methylation Complete Fast Dissolves™ twice daily**			Consider Plasma B12
rs1801394	MTRR A66G	+/+					
rs526934	TCN1	+/+					
rs1801198	TCN2	-/-					

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(-/-) No clinical abnormality | (+/-) Heterozygous result | (+/+) Homozygous result

rsID	Gene	Genetic Result	Therapeutics Associated With Positive Result	Highly Recommended Therapeutics	Provider Discretion: As Needed Formula Recommendations	Lifestyle Recommendations	Laboratory Recommendations
Early Childhood							
Mitochondrial							
rs4147730	NDUFS3	-/-	CoQ 10, PQQ, L-Carnitine, Ornithine, Magnesium, NADH, Calcium	**Mito Cell PQQ**™			
rs809359	NDUFS7	-/-					
rs1051806	NDUFS8	+/-					
rs4850	UQCRC2	+/-					
rs11648723	UQCRC2	-/-					
rs8042694	COX5A	+/-					
rs4626565	COX6C	-/-					
rs1244414	ATP5C1	-/-					
rs6535454	CoQ2	+/+	CoQ 10	**Mito Cell PQQ**™			
Vitamin D Transport							
rs731236	VDR Taq	-/-	Vitamin D Vitamin K				

Summary for Early Childhood

Highly Recommended Therapeutics

- CBD Oil
- PEA Soothe Support™
- Prescription Low Dose Naltrexone (LDN)
- DCI 500 or Metabolic Stimulator™
- N.A.S. Enhancer™
- Methyl Folate Plus™ Twice Daily
- Methylation Pro Topical™ OR Methylation Complete Fast Dissolves™ twice daily
- Mito Cell PQQ™

Provider Discretion: As Needed Formula Recommendations

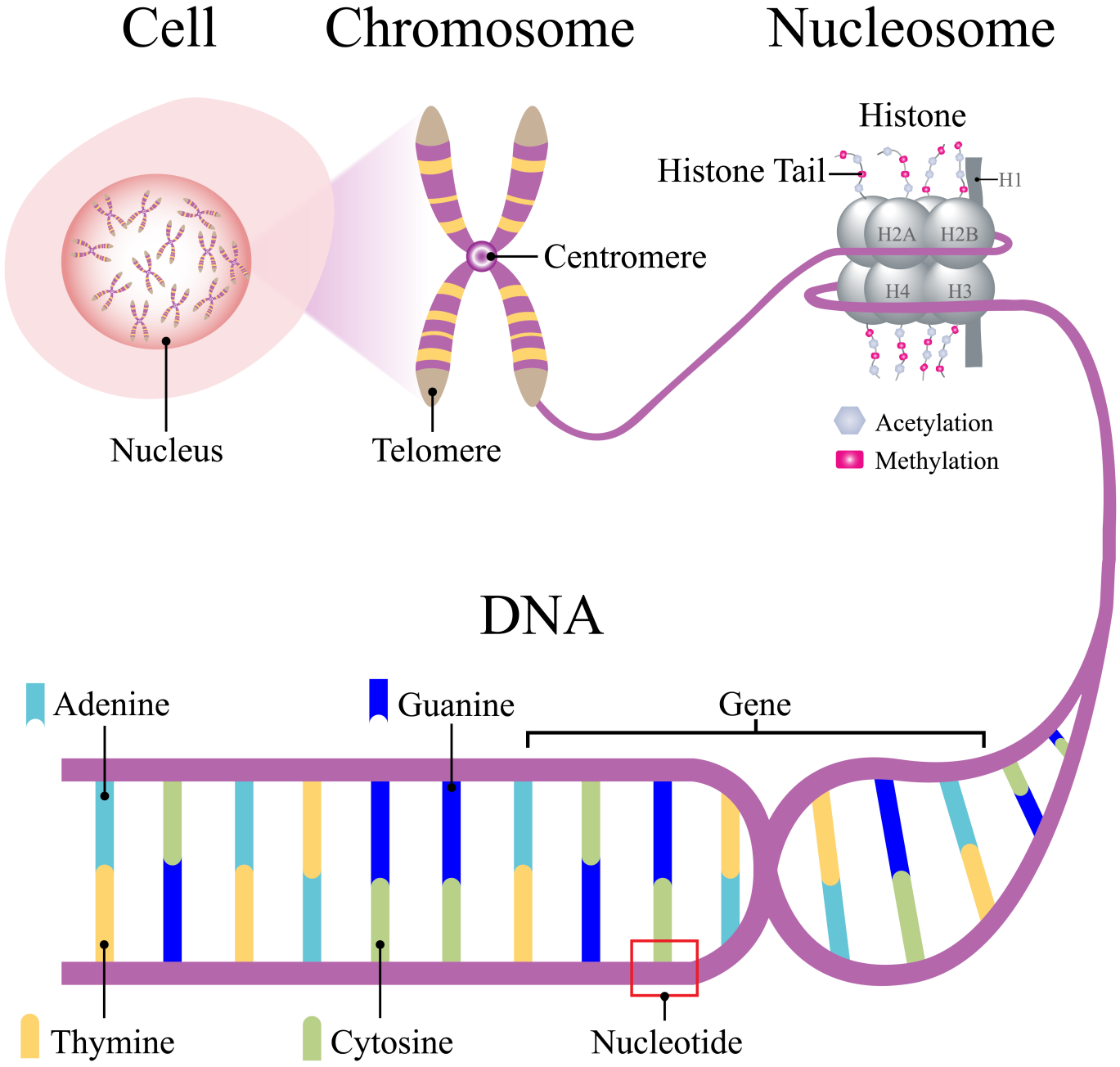
- Biotic Boost Chews or Powder For Kids ™

Lifestyle Recommendations

- Consider Low Inflammatory Diet
- Highly Recommend Avoiding Mold / Fungal / Yeast Exposure
- 12-15 Hour Fasting if appropriate for age

Laboratory Recommendations

- Consider Pregnenolone
- Cortisol
- T cell profile
- Routine Thyroid Panel
- Candida Titer
- Food Allergy Panel
- Environmental Allergy Testing
- Consider Microbiome Testing If GI Inflammation Present
- Consider Candida Titers and MSH (Melanocyte Stimulating Hormone)
- Routine Blood Sugar
- Insulin and Hb A1c
- Consider Plasma B12


Cell
Chromosome
Nucleosome
Histone
Histone Tail
H1
H2A
H2B
H4
H3
Centromere
Nucleus
Telomere
Acetylation
Methylation
DNA
Adenine
Guanine
Gene
Thymine
Cytosine
Nucleotide

Gene Information Key

rsID	Gene	"-" variant	"+" variant
rs10156191	AOC1	C	T
rs26538	ATG12	T	C
rs10210302	ATG16L1	C	T
rs510432	ATG5	C	T
rs1244414	ATP5C1	C	T
rs6265	BDNF	C	T
rs10402876	C3	G	C
rs2569191	CD14	T	C
rs6535454	CoQ2	A	G
rs8042694	COX5A	A	G
rs4626565	COX6C	T	C
rs1021737	CTH	G	T
rs231775	CTLA4	A	G
rs1643649	DHFR	T	C
rs1076560	DRD2	C	A
rs2071010	FOLR1	G	A
rs651933	FOLR2	A	G
rs492602	FUT2	A	G
rs7483	GSTM3	C	T
rs1695	GSTP1:I105V	A	G
rs2187668	HLA-DQA1	C	T
rs2858331	HLA-DQA2	A	G
rs660895	HLA-DRB1	A	G
rs9275224	HLA-DRB2	G	A
rs11558538	HMNT	C	T
rs12995000	HMNT	C	T

rsID	Gene	"-" variant	"+" variant
rs1800925	IL13	C	T
rs2069812	IL5	A	G
rs1800795	IL6	G	C
rs1076991	MTHFD1	C	T
rs1801131	MTHFR:A1298C	T	G
rs1801133	MTHFR:C677T	G	A
rs6495446	MTHFS	C	T
rs1805087	MTR	A	G
rs1802059	MTRR:A664A	G	A
rs1801394	MTRR:A66G	A	G
rs4147730	NDUFS3	G	A
rs809359	NDUFS7	A	G
rs1051806	NDUFS8	C	T
rs6330	NGF	G	A
rs1051266	SLC19A1	T	C
rs10181656	STAT4	C	G
rs1142636	SYN1	A	G
rs526934	TCN1	A	G
rs1801198	TCN2	C	G
rs1800629	TNF	G	A
rs11648723	UQCRC2	G	T
rs4850	UQCRC2	G	A
rs731236	VDR Taq	A	G

Definitions

DETOXIFICATION	Detoxification enzymes are responsible for clearing environmental chemicals and metabolites from our body. Accumulation of these chemicals and by-products can damage intracellular biochemical functions. Alterations in these systems can have a significant negative effect on the nervous system and immune systems functions. These polymorphisms can result in decreased "quality of life" and even decreased "life-span".
CTH	Glutathione production is dependent on the function of the enzyme cystathionine gamma-lyase (CTH). CTH converts cystathionine to cysteine. Individuals with mutations in the CTH gene are predicted to have decreased glutathione-mediated detoxification.
GSTM3	Glutathione S-transferase mu 3 is an enzyme that detoxifies drugs, environmental toxins, and carcinogens by conjugating toxins to glutathione and subsequent excretion by the kidneys. Mutations in GSTM3 are associated with decreased clearance of toxins, anesthetics and drugs from the nervous system.
GSTP1	Glutathione S-transferases (GSTs) are a family of enzymes that play an important role in detoxification. The glutathione S-transferase pi gene (GSTP1) functions in chemical clearance and anti-inflammatory properities. High concentration of GST-p are found in the skin, lungs, sinuses, bladder and the intestinal tract. Polymorphisms of this enzyme allow for increased inflammatory activity in these areas that include eczema, asthma, chronic sinusitis, IBS, "leaky" gut and interstitial cystitis.
DEVELOPMENTAL	The SNPs in this category have been identified as potential areas of weakness in the recovery of developmental disorders.
ATG12	Autophagy-related 12 protein is part of the core autophagy machinery inside the cell. Autophagy, a form of cellular "recycling" is necessary for many cell functions. ATG12 is specifically involved in turning off the innate immune response. Mutations in the ATG12 gene are predicted to lead to increased activity of the innate immune response, and overall inflammation.
BDNF	The BDNF (Brain Derived Neurotrophic Factor) gene encodes for a member of the nerve growth factor family of proteins. BDNF acts on both the central nervous system and the peripheral nervous system helping to support the survival of existing neurons and encourage the growth and differentiation of new neurons and synapses. It is highly expressed in the brain, as well as, the retina, cochlear-vestibular system and motor neurons. Although the vast majority of neurons in the brain are formed prenatally, parts of the adult brain retain the ability to grow new neurons from neural stem cells in a process known as neurogenesis. BDNF helps to stimulate and control neurogenesis, as well as playing an important role in normal neural development. Binding of this protein to its cognate receptor promotes neuronal survival in the adult brain. Expression of this gene is reduced in Alzheimer's, Parkinson's and Huntington's disease. This gene may play a role in the regulation of the stress response and the biology of mood disorders. Several mechanisms to increase BDNF have been discovered. These mechanisms revolve around autophagy stimulation. These include Intermittent Fasting with a single meal of 600 calories on the fast day can increase BDNF production by 50-400%. Cognitive Stimulation, Calorie Restriction, Exercise, Hormone therapy and supplements including Quercitin, Caffeine, Curcumin, Niacinamide, Lithium Oratate, Magnesium Threonate, Resveratrol, Ginseng, Theanine, Olive Leaf Extract and NAC.
NGF	This gene encodes a member of the nerve growth factor family of proteins. Alternative splicing results in multiple transcript variants, at least one of which encodes a preproprotein that is proteolytically processed to generate the mature protein. Binding of this protein to its cognate receptor promotes neuronal survival in the adult brain. Expression of this gene is reduced in Alzheimer's, Parkinson's, and Huntington's disease patients. This gene may play a role in the regulation of the stress response and in the biology of mood disorders
SYN1	SYN1 (Synapsin) codes for Synapsins that are responsible for synaptogenesis and the modulation of neurotransmitter release, suggesting a potential role in several neuropsychiatric diseases. This member of the synapsin family plays a role in regulation of axonogenesis and synaptogenesis. Mutations in this gene may be associated with X-linked disorders with primary neuronal degeneration such as Rett syndrome. Additionally, polymorphisms in this gene are associated with numerous neurological conditions, as well as, decreased recovery potential for neurological insults.
ESSENTIAL VITAMINS	The polymorphisms in this panel will identify any potential weakness of absorption, conversion or delivery or your essential vitamins.
COQ2	CoQ2 (Para-hydroxybenzoate—polyprenyltransferase, mitochondrial) codes for an enzyme that functions in the final steps in the biosynthesis of CoQ10 (ubiquinone).. This enzyme, which is part of the coenzyme Q10 pathway, catalyzes the prenylation of parahydroxybenzoate with an all-trans polyprenyl group. Mutations in this gene cause coenzyme Q10 deficiency. Polymorphisms in this gene can lead to severe fatigue, muscle weakness, exercise intolerance and general mitochondrial weakness.
INFLAMMATORY	This Enzyme category has significant effects on the inflammatory state of a person's body. Polymorphisms in these specific enzymes will significantly increase the levels of inflammation in the body. By supplementing these enzyme deficiencies, the patient will effectively reduce inflammatory damage to the body.
AOC1	The SNP rs10156191 encodes a weaker form of the histamine degradation enzyme Amine Oxidase, Copper Containing 1 (AOC1). This mutation, Thr16Met, is predicted to produce an enzyme with less catalytic activity and associated higher levels of pro-inflammatory amines like histamine and putrescine.
ATG16L1 rs10210302	The ATG16L1 gene encodes a protein that is a vital component of a protein complex necessary for the cellular phenomena known as autophagy. Autophagy is the process of degrading and cleaning of inert debris of the cell. Weakness in autophagy leads to abnormal accumulation of cellular “garbage” that will eventually affect the cellular function and lead to autophagy-related disease states in including many neurological and immunological diseases, DM Type 2 and fatty liver disease.

ATG5	Autophagy-related 5 protein (ATG5) is an important intracellular mediator of the autophagy response. ATG5 is involved in a wide range of "quality control" features inside the cell: autophagy vesicle formation, innate immune system signaling, consumption of damaged mitochondria, and apoptosis. Mutations in the ATG5 gene are associated with numerous neurological, immunological and endocrine syndromes.
C3	Essential for the immune response, C3 is a protein involved in initiation of the complement system. C3 polymorphisms are associated with susceptibility to asthma and other inflammatory disorders.
CD14	The CD14 protein is a macrophage cell surface receptor that binds bacterial cell wall components. As one of the initiators of the innate immune response, fully functional CD14 is necessary for normal response to potential pathogens. Mutations in the CD14 gene are associated with susceptibility to asthma and other allergen-mediated inflammatory processes.
CTLA4	Cytotoxic T-lymphocyte Associated protein 4 (CTLA4) is an important inhibitor of T-cell activity: CTLA4 is part of the signaling cascade that turns off overactive T cells. Mutations in the gene that encodes CTLA4 are associated with a host of diseases characterized by a heightened immune state.
DRD2	Dopamine receptor D2 is an important component of the neuroinflammation process. Activation of DRD2 signaling is thought to decrease TNFalpha release from inflammatory mast cells. Polymorphisms associated with decreased DRD2 signaling activity are predicted to lead to pro-inflammatory phenotypes.
FUT2	Fucosyltransferase 2 (FUT2) is responsible for producing specific sugar groups that are secreted by the intestinal cells into the bowel to attract "good bacteria" . Polymorphisms in this gene produce "poor secreter" status. Lack of these sugars allows for gut dysbiosis and a higher risk of inflammatory bowel disease.
HLA-DQA1	Major histocompatibility complex, DQ alpha 1 (HLA-DQA1) is a human gene responsible for a cell surface receptor essential to the function of the immune system. Patients with a polymorphism in this gene are at higher risk for auto-immune based inflammatory disease including Celiac disease, Crohn's, Ulcerative Colitis, and gluten sensitivity.
HLA-DQA2	Major histocompatibility complex, DQ alpha 2 (HLA-DQA2) is a human gene responsible for a cell surface receptor essential to the function of the immune system. Patients with a polymorphism in this gene are at higher risk for auto-immune based inflammatory disease including Celiac disease, Crohn's, Ulcerative Colitis, and gluten sensitivity.
HLA-DRB1	Human leukocyte antigen DRB1 (HLA-DRB1) is an important mediator of the adaptive immune system. HLA-DRB1 protein "presents" antigens from invading pathogens to other cells in the immune system. Mutations in this gene are associated with higher risk of auto-immunity and other chronic inflammatory diseases.
HLA-DRB2	Human leukocyte antigen DRB2 (HLA-DRB2) is a cell surface receptor involved in mediating the adaptive immune response. Mutations in HLA-DRB2 are associated with susceptibility to chronic inflammation and decreased ability to recover from toxic mold exposure.
HNMT rs12995000	The HNMT gene encodes the histamine degradative enzyme, histamine N-methyltransferase. HNMT, in contrast to AOC1, requires the methyl donor S-adenosylmethionine and a complete methylation pathway for normal function. Polymorphisms in HNMT gene expression or protein-coding are predicted to prolong the pro-inflammatory effects of histamine signaling.
HNMT Thr105Ile	The HNMT gene encodes the histamine degradative enzyme, histamine N-methyltransferase. HNMT, in contrast to AOC1, requires the methyl donor S-adenosylmethionine and a complete methylation pathway for normal function. Polymorphisms in HNMT gene expression or protein coding are predicted to prolong the pro-inflammatory effects of histamine signaling.
IL13	IL13 (Interleukin 13) is a member of the interleukin family of chemical messengers of the immune system. Polymorphisms in this gene are associated with changes in IL13 gene expression and increase the risk of more severe inflammatory responses to allergens.
IL5	The protein product of the Interleukin 5 gene (IL5) is important for normal development of B lymphocytes and eosinophils (a pro-inflammatory white blood cell). Inactivating mutations in the IL5 gene are associated with susceptibility to certain viral infections and increased aggression of inflammatory response. These polymorphisms are also associated with increased aggression of allergies, asthma and eosinophilia.
IL6	Interleukin 6, IL6, is an important pro-inflammatory cytokine. Polymorphisms in this gene leads to a more aggressive inflammatory response. Patients with IL-6 mutations require assistance with inflammatory control.
STAT4	The Signal Transducer and Activator of Transcription 4 (STAT4) gene encodes a transcription factor that responds to extracellular growth factors and cytokines. Mutations in the STAT4 gene are associated with inflammatory disorders like lupus and rheumatoid arthritis.
TNF	Tumor necrosis factor, TNF, is an important pro-inflammatory signaling molecule. Polymorphisms in the protein coding part of this gene are associated with more severe pro-inflammatory responses and require supplementation for inflammatory control.
VDR Taq1	The Vitamin D (calcitriol) Receptor is a member of the nuclear receptor family. Upon activation by vitamin D (a secosteroid), the VDR causes the activation or deactivation of protein production by the cell. Impaired vitamin D function can result in signigicant immune weakness and increased cancer risk, as well as, early bone loss, an increased risk of cognitive decline and mood disorders.
METHYLATION	Methylation is a primary biochemical process in the body that involves the addition of a "methyl" chemical group to a vitamin or neurotransmitter. The addition of the "methyl" group allows for very specific biochemical interactions. Poor "methylation" function alters the effectiveness, delivery and function of many vitamins and important chemicals in the cell.

DHFR	Dihydrofolate reductase, or DHFR, is an enzyme that reduces dihydrofolic acid to tetrahydrofolic acid. This enzyme is the second enzyme in the folic acid conversion chain. Having a mutation in this enzyme can create a methylaiton deficiency with a MTHFR mutation.
FOLR1	Folate Receptor 1 (FOLR1) is a member of the folate receptor (FOLR) family. Members of this gene family have a high affinity for folate. Polymorphisms in this gene allow for poor delivery of folate to the interior of cells. This can create a high plasma folic acid. This polymorphism does create a methylation deficiency. This polymorphism is associated with many disorders of pregnancy.
FOLR2	Folate Receptor 2 (FOLR2) is a member of the folate receptor (FOLR) family. Members of this gene family have a high affinity for folic acid. Polymorphisms in this gene allow for poor delivery of folic acid to the interior of cells. This can create a high plasma folic acid. This polymorphism does create a methylation deficiency. This polymorphism is associated with many disorders of pregnancy. This receptor is found in high quantities on the placenta, thymus and bone marrow. Can be affiliated with immune disorders.
MTHFD1	Methylenetetrahydrofolate Dehydrogenase 1 enzyme handles 2 significant enzymes conversions in the production of L-MTHF. This common polymorphism causes a significant methylation deficiency due to the fact that it is utilized in two steps in methyl-folate production.
MTHFR A1298C	Methylenetetrahydrofolate reductase (MTHFR) catalyzes the conversion of 5,10-methylenetetrahydrofolate to 5-methyltetrahydrofolate, the bioactive form of folic acid. Two significant polymorphism variants exist in this gene, the A1298C and the C677T. The 1298 confers a conversion weakness of 10% for one copy and approximately 20% for two copies. In contrast, the 677 variant is much more severe and conveys a 40% conversion weakness for one copy and 70% for two copies. A reduced level of MTHFolate produces significant biochemical effects including poor production of dopamine and serotonin, pregnancy complications, poor healing of the nervous system, weak mitochondrial function, reduced production of glutathione, poor cell turnover and poor function of T cell lymphocytes.
MTHFR C677T	Methylene tetrahydrofolate reductase (MTHFR) catalyzes the conversion of 5,10-methylenetetrahydrofolate to 5-methyltetrahydrofolate, the bioactive form of folic acid. Two significant polymorphism variants exist in this gene, the A1298C and the C677T. The 1298 confers a conversion weakness of 10% for one copy and approximately 20% for two copies. In contrast, the 677 variant is much more severe and conveys a 40% conversion weakness for one copy and 70% for two copies. A reduced level of MTHFolate produces significant biochemical effects including poor production of dopamine and serotonin, pregnancy complications, poor healing of the nervous system, weak mitochondrial function, reduced production of glutathione, poor cell turnover and poor function of T cell lymphocytes.
MTHFS	MTHFS (methenyletetrahydrofolate synthase) is an enzyme that catalyzes the conversion of 5-formyltetrahydrofolate to 5,10-methenyltetrahydrofolate, a precursor of reduced folates. This polymorphism codes for a decreased function of the enzyme and results in poor utilization of Leucovorin (5-formyltetrahydrofolate)..
MTR	MTR (Methionine Synthase) codes for the enzyme that catalyzes the final step in methionine biosynthesis. Polymorphisms in this gene lead to poor recycling of methionine from homocysteine. This enzyme work in coordination with MTRR and requires both MTHF and B12 for proper functioning. Deficiencies in Methionine leads to poor methylation that is associated with numerous neurological, cardiovascular and immunological disease states, as well as, infertility and birth defects.
MTRR Ala637=	Methionine Synthase Reductase is an enzyme responsible for the production of methionine, a very important amino acid. Polymorphisms in this enzyme require an increased amount of Methyl B12 to help this reaction.
SLC19A1	The SLC19A1 gene encodes the reduced folate carrier (RFC) protein. Mutations in the RFC are associated with reduced plasma folate.
TCN1	The protein product of the transcobalamin 1 (TCN1) gene binds Vitamin B12 and protects it from the low pH environment of the human stomach. Individuals homozygous for the G allele of the TCN1 SNP, rs526934, are predicted to have lower serum B12.
MITOCHONDRIA	The mitochondrial enzymes are responsible for energy production from the mitochondria. The mitochondria is known as the "powerhouse" of the cell and produces over 90% of the energy for a cell. The mitochondrial respiratory chain (also known as the electron transport chain) is where these 4 protein complexes are found. Polymorphic alterations in these enzymes reduce the energy output of the mitochondria and leads to symptoms of chronic fatigue, cognitive deficiency, exercise intolerance, low metabolic rate, muscle weakness, poor healing and higher rates of sleep disorders and mood abnormalities.
ATP5C1	ATPase 5c1 (ATP5C1) is an enzyme responsible for producing ATP (the energy component) in the mitochondria. This protein is known as Complex V (the 5th protein) in the mitochondrial respiratory chain. Polymorphisms in the gene confer a weakened energy production by the mitochondria.
COX5A	Cytochrome c oxidase subunit 5a (COX5A) is a protein in a subunit of the cytochrome c oxidase complex, also known as Complex IV of the mitochondrial electron transport chain. Polymorphisms in this enzyme produce a weakened energy production by the mitochondria.
COX6C	Cytochrome c oxidase subunit 6c (COX6C) is a protein in a subunit of the cytochrome c oxidase complex, also known as Complex IV of the mitochondrial electron transport chain. Polymorphisms in this enzyme produce a weakened energy production by the mitochondria.
NDUFS3	The NDUFS3 genes encodes a mitochondrial enzyme, NADH Dehydrogenase (Ubiquinone) Fe-S Protein 3. Like other NDUFS proteins, NDUFS3 is thought to require ubiquinone for full activity.
NDUFS7	NADH Dehydrogenase [ubiquinone] iron-sulfur protein 7 (NDUFS7) is a mitochondrial protein also know as Complex I of the mitochondrial respiratory chain. It is located in the mitochondrial inner membrane and is the largest of the five complexes of the electron transport chain. Polymorphisms in this enzyme produce a weakened energy production in the mitochondria.

NDUFS8	NADH Dehydrogenase (Ubiquinone) Fe-S Protein 8 (NDUFS8) encodes an enzyme in the mitochondrial respiratory chain. Mutations in the NDUFS8 gene are associated with Leigh Syndrome, osteoporosis, and mitochondrial complex I deficiency.
UQCRC2 Arg183Gln	Ubiquinol Cytochrome c Reductase (UQCR, Complex II) is a mitochondrial enzyme protein also known as Complex III of the electron transport chain. Polymorphisms in this enzyme produce a weakened energy production by the mitochondria.

Disclaimers

METHODOLOGY AND LIMITATIONS:

Testing for genetic variation/mutation on listed genes was performed using ProFlex PCR and Real-Time PCR with TaqMan® allele-specific probes on the QuantStudio 12K Flex. All genetic testing is performed by GX Sciences, 4150 Freidrich Lane, Ste H, Austin, TX. 78744. This test will not detect all the known alleles that result in altered or inactive tested genes. This test does not account for all individual variations in the individual tested. Test results do not rule out the possibility that this individual could be a carrier of other mutations/variations not detected by this gene mutation/variation panel. Rare mutations surrounding these alleles may also affect our detection of genetic variations. Thus, the interpretation is given as a probability. Therefore, this genetic information shall be interpreted in conjunction with other clinical findings and familial history for the administration of specific nutrients. Patients should receive appropriate genetic counseling to explain the implications of these test results. Details of assay performance and algorithms leading to clinical recommendations are available upon request. The analytical and performance characteristics of this laboratory developed test (LDT) were determined by GX Sciences' laboratory pursuant to Clinical Laboratory Improvement Amendments (CLIA) requirements.
CLIA #: 45D2144988 Laboratory Director: James Jacobson, PhD

DISCLAIMER:

This test was developed and its performance characteristics determined by GX Sciences. It has not been cleared or approved by the FDA. The laboratory is regulated under CLIA and qualified to perform high-complexity testing. This test is used for clinical purposes. It should not be regarded as investigational or for research. rsIDs for the alleles being tested were obtained from the dbSNP database (Build 142).

DISCLAIMER:

UND Result: If you have received the result Variant undetermined (UND) this indicates that we were not able to determine your carrier status based on your raw data. Please refer to the GX Sciences genetic knowledge database for more information: https://www.gxsciences.com/kb_results.asp

DISCLAIMER:

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