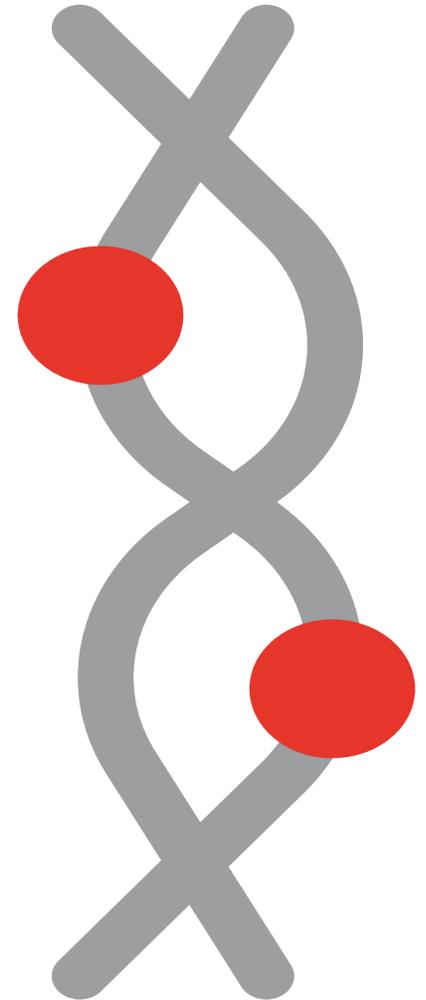




Fagron

genomics



Gene Comprehensive Nutrigenomic Report

Accession Number: #####

Specimen Collected: ##/##/####

Specimen Received: ##/##/####

Report Generated: November 17, 2022

Specimen Type: Buccal Swab

Provider: #####

Patient Name: #####

Patient DOB: ##/##/####

Patient Gender: Male

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.

– 36 – Male

(-/-) 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
Neurotransmitters							
rs4680	COMT	+/-	Taurine, Choline, Trimethylglycine (TMG), Dimethylglycine (DMG), Methionine, SAME, Inositol		Full Focus+		Consider Neurotransmitter Metabolite Testing
rs6323	MAO-A	-/NA	B2 (Riboflavin), Methyl Donors (Taurine, Choline, Trimethylglycine (TMG), Dimethylglycine (DMG), Inositol, Methionine		Full Focus+ Mood Plus	Higher Risk of Depression / Anxiety Around Stressful Events	Consider Neurotransmitter Metabolite Testing
rs1799836	MAO-B	+/NA					
rs3828275	GAD1	+/+	Prescription Amantadine Glycine, N-Acetyl Cysteine (NAC), Zinc, Magnesium, Elderberry, L-Theanine, Melatonin	May Benefit from Prescription Amantadine		Consider avoiding MSG (monosodium glutamate) and glutamine supplements	Consider Neurotransmitter Metabolite Testing
rs769407	GAD1	-/-					

– 36 – Male

(-/-) 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
Inflammation Cellular							
rs10402876	C3	+/-	Anti-Inflammatory Therapy: Curcumin, Omega 3s, Resveratrol, Quercetin, Low Dose Naltrexone (LDN), CBD Oil		Pregnenolone Plus Adrenal Enhance™ Ultra Omega 550™	Consider Low Inflammatory Diet	Consider Pregnenolone, Cortisol, Progesterone, DHEA and Testosterone
rs2069812	IL5	-/-					
rs1800795	IL6	-/-					
rs1800925	IL13	-/-					
rs10181656	STAT4	-/-					
rs1800629	TNF	-/-					
rs231775	CTLA4	+/+					
rs1076560	DRD2	+/-	Higher Response Rate To Low Dose Naltrexone			Expect High Response Rate to Low Dose Naltrexone (LDN)	
Detox							
rs1021737	CTH	-/-	N-Acetyl Cysteine (NAC)				
rs819147	AHCY	-/-					
rs1695	GSTP1	+/-	Glutathione				
rs7483	GSTM3	-/-					

– 36 – Male

(-/-) 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
Autophagy							
rs510432	ATG5	+/+	Curcumin, Lithium Orotate, D-Chiro-Inositol, Catechins, Resveratrol, Caffeine, 12 Hour Fasting	DCI 500mg NAS Enhancer™		12-15 Hour Fasting	Routine Blood Sugar, Insulin and Hba1c
rs26538	ATG12	+/-					
rs10210302	ATG16L1	+/-					
Methylation							
rs2071010	FOLR1	-/-	Methyltetrahydrofolate (5-MTHF)	Methyl Folate Plus™ Twice Daily			Consider Plasma Homocysteine
rs651933	FOLR2	-/-					
rs1643649	DHFR	+/-					
rs1076991	MTHFD1	-/-					
rs1801131	MTHFR A1298C	-/-					
rs1801133	MTHFR C677T	+/-					
rs1051266	SLC19A1	+/-					

Summary for TBI / Post Concussion

Highly Recommended Therapeutics

- May Benefit from Prescription Amantadine
- DCI 500mg
- NAS Enhancer™
- Methyl Folate Plus™ Twice Daily

Provider Discretion: As Needed Formula Recommendations

- Full Focus+
- Mood Plus
- Pregnenolone Plus
- Adrenal Enhance™
- Ultra Omega 550™

Lifestyle Recommendations

- Higher Risk of Depression / Anxiety Around Stressful Events
- Consider avoiding MSG (monosodium glutamate) and glutamine supplements
- Consider Low Inflammatory Diet
- Expect High Response Rate to Low Dose Naltrexone (LDN)
- 12-15 Hour Fasting

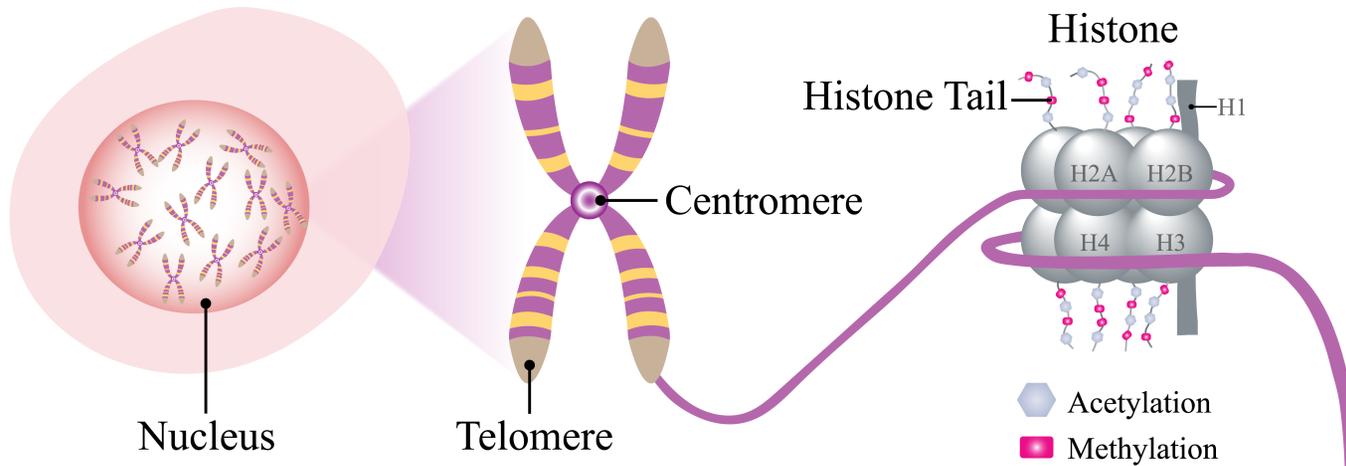
Laboratory Recommendations

- Consider Neurotransmitter Metabolite Testing
- Consider Pregnenolone
- Cortisol
- Progesterone
- DHEA and Testosterone
- Routine Blood Sugar
- Insulin and Hba1c
- Consider Plasma Homocysteine

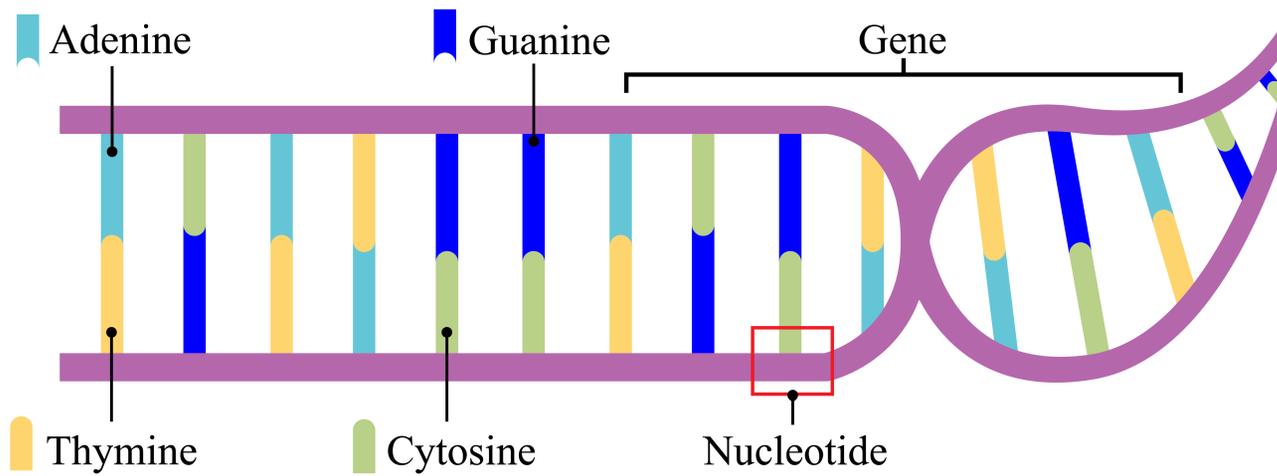
Cell

Chromosome

Nucleosome



DNA



NEUROTROPHIC FACTORS

VARIANTS IN THE SYN1, NGF & BDNF GENES CAUSE DECREASED NEURON SYNTHESIS



Promote growth, development, survival, synaptic plasticity (strengthening) and repair of neurons



Regulate the development of the peripheral and central nervous systems



Regulate the formation of long-term memories

LOW LEVELS ARE CORRELATED WITH

- Neurodegenerative disorders
- Aging
- Chronic stress
- Mood disorders

WAYS TO INCREASE LEVELS



Exercise (physical or cognitive)



Social interactions



Reduce stress via breathing exercises and/or meditation

LOW-INFLAMMATORY

FOODS TO EAT



Fruits: strawberries, blueberries, cherries, oranges



Fatty fish: salmon, mackerel, tuna, sardines



Spices - turmeric, ginger



Green leafy vegetables & tomatoes



Dark chocolate



Olive oil



LOW-INFLAMMATORY DIET

FOODS TO AVOID



Soda & other sugar-sweetened drinks



Dairy products



Fried foods



Red & Processed meats (hotdogs, sausage)



Refined carbohydrates: white bread, pastries



Margarine, shortening, lard

BENEFITS



Reduces inflammation



Reduces risk for cardiovascular disease & Type II diabetes

DETOXIFICATION

GLUTATHIONE IN DETOXIFICATION

Relevant genes for production are AHCY, CTH, GSTP1, GSTM1, GSTM3, GSR, MTRR & MTR

WHY IS IT IMPORTANT?



Maintains health by protecting the body from toxins



Regulates cell production and programmed cell death



Critical role in chemical detoxification



Vital for proper mitochondrial function



WAYS TO INCREASE GLUTATHIONE

- Limit alcohol intake
- N-acetyl-cysteine (NAC)
- Glutathione therapies
- (ie. IV Glutathione, Glutathione suppository, Liposomal Glutathione)
- Include whey in diet, unless allergic or intolerant
- Methylation Support - if necessary

SUPEROXIDES & ANTIOXIDANTS

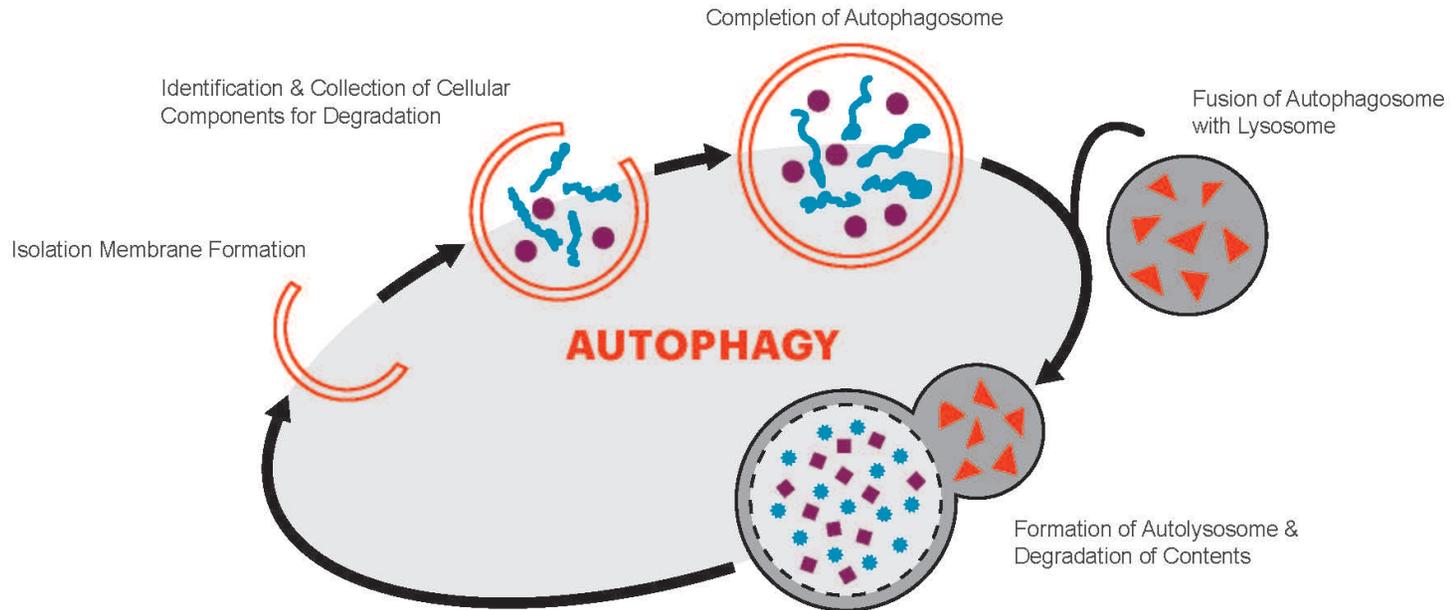
- SOD1, SOD2, SOD3 genes are important to transform superoxides to protect against mitochondrial damage
- Reactive Oxygen Species (ROS) can damage mitochondria and cause cell death.
- Antioxidants such as Vitamin A, Vitamin C and Vitamin E act as a defense against ROS

DEFICIENCY CAUSES

- Auto-immune diseases
- Cardiovascular diseases
- Neurodegenerative diseases
- Cell death
- Poor mitochondrial function

AUTOPHAGY

VARIANTS IN THE ATG GENES HAVE BEEN ASSOCIATED WITH CELLULAR BLOCKAGE



DEFECTS LEAD TO:

- Neurodegenerative Diseases
- Aging
- Heart Disease
- Developmental Disorders
- Type II Diabetes
- Insulin Resistance
- Fatty Liver
- Cancers



Intermittent fasting
or low-calorie diet



Routine Exercise



Ketogenic diets
(high fat, low carbs)



Medications &
Supplements
D-Chiro Inositol (B8)
Metformin

WAYS TO INCREASE

Gene Information Key

rsID	Gene	"-" variant	"+" variant
rs819147	AHCY	T	C
rs26538	ATG12	T	C
rs10210302	ATG16L1	C	T
rs510432	ATG5	C	T
rs10402876	C3	G	C
rs4680	COMT	G	A
rs1021737	CTH	G	T
rs231775	CTLA4	A	G
rs1643649	DHFR	T	C
rs1076560	DRD2	C	A
rs2071010	FOLR1	G	A
rs651933	FOLR2	A	G
rs3828275	GAD1	C	T
rs769407	GAD1	G	C
rs7483	GSTM3	C	T
rs1695	GSTP1	A	G
rs1800925	IL13	C	T
rs2069812	IL5	A	G
rs1800795	IL6	G	C
rs6323	MAO-A	T	G
rs1799836	MAO-B	T	C
rs1076991	MTHFD1	C	T
rs1801131	MTHFR:A1298C	T	G
rs1801133	MTHFR:C677T	G	A
rs1051266	SLC19A1	T	C
rs10181656	STAT4	C	G
rs1800629	TNF	G	A

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".
AHCY	Adenosylhomocysteinase (AHCY) is an enzyme that breaks down S-adenosylhomocysteine (SAH) to homocysteine and adenosine. Polymorphisms in this gene will lead to lower levels of homocysteine and glutathione.
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 properties. 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.
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.
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 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.
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.
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.
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.
SLC19A1	The SLC19A1 gene encodes the reduced folate carrier (RFC) protein. Mutations in the RFC are associated with reduced plasma folate.
NEUROTRANSMITTER	Neurotransmitters are chemicals that are used to produce specific effects in the nervous system. These specific neurotransmitter genomics assess a person's risk for anxiety, depression and dysphoria.
COMT V158M	Catechol-O-methyltransferase (COMT) is one of several enzymes that degrade catecholamine neurotransmitters such as dopamine, epinephrine, and norepinephrine. COMT's main function is to inactivate neurotransmitters (dopamine, epinephrine, and norepinephrine) by the addition of a methyl group to the catecholamine. Normal COMT function allows people to rapidly reverse feelings of anxiety or depression. COMT (+/-) patients have sluggish ability to alter anxiety or depression episodes. COMT (+/+) patients are more prone to prolonged episodes of anxiety, depression and OCD.
GAD1 rs3828275	Glutamic Acid Decarboxylase (GAD 1) is the enzyme responsible for conversion of glutamic acid (a stimulant neurotransmitter) to GABA (a calming neurotransmitter). Deficiency of GABA from polymorphisms in this enzyme are associated with sleep disorders, "half glass empty" syndrome, dysphoria, and spasticity.
MAOA	Monoamine oxidase A (MAOA) is one of the classic neurotransmitter degradation enzymes. By degrading serotonin, dopamine, epinephrine, and norepinephrine, MAO-A ends neuronal signaling induced by those neurotransmitters. Mutations in the MAO-A gene leads to decreased degradation of these neurotransmitters and can be associated with increased aggression, mood disorders and drug addiction.
MAOB	Monoamine Oxidase B (MAO B) catalyzes the neuroactive amines, such as dopamine, epinephrine, norepinephrine, and plays a role in the stability of mood in the central nervous system,. MAO B's primary purpose is to degrade dopamine. Patients who possess polymorphisms of MAO B have a higher risk of clinical depression and mood disorders.

Disclaimers

TESTING:

Testing Performed By: TY

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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DETOXIFICATION SNP References

AHCY

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ATG16L1

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