ULTAGENOMICS

DECODE YOUR DNA. GUIDE YOUR HEALTH.

Mental Health and Behavior

GENETICS REPORT

1	5	52
Report	Report Panels	Gene Variants

Example Client
Prepared for

September 19, 2026
Report Date

Our genetic test provides insights into how your unique genetic profile influences health areas. By understanding these genetic tendencies, you and your healthcare provider can make more informed lifestyle and wellness choices to support long-term health goals.

THESE GENE RESULTS MIGHT TELL YOU

- ✓ How your body may respond to certain nutrients
- ✓ Dietary patterns and environmental factors
- ✓ Potential predispositions in detoxification, metabolism, and physical performance
- ✓ Why some wellness strategies may work differently for you

THESE GENE RESULTS WILL NOT TELL YOU

- ✕ Any medical diagnoses
- ✕ Cancer diagnoses or clinical cancer risk assessments
- ✕ Predictions about future health conditions
- ✕ How lifestyle factors outside of genetics impact health outcomes

This report is intended for informational purposes only and does not constitute medical advice. Results reflect genetic predispositions, not guarantees of health outcomes. Always consult a qualified healthcare practitioner before making changes to your health regimen.

A typical or decreased result does not mean there is no risk, and an increased result does not mean a condition will develop. Environment, lifestyle and family history also play a role. Any result that mentions cancer reflects a genetic tendency only. It is not a clinical cancer risk assessment and does not replace recommended cancer screening.

Talk with a healthcare provider before starting any supplement, especially if pregnant or breastfeeding, taking prescription medications, or managing a medical condition. Do not start, stop or change any medication based on these results without first talking with the prescribing provider.

Statements about supplements in this report have not been evaluated by the Food and Drug Administration. These products are not intended to diagnose, treat, cure, or prevent any disease.

Research on how genes relate to health is still developing. Some recommendations are based on limited evidence and may be updated as new research becomes available.

Testing is performed by a CLIA-certified, CAP-accredited laboratory. Genotyping is highly accurate, but no laboratory test is completely error-free. Results that will change clinical decisions may warrant confirmatory testing.

OVERVIEW

Results at a Glance

This report covers 5 panels and 8 individual results. The summary below shows where the genetic profile indicates areas worth focusing on, areas to monitor, and areas where results are favorable. The Moderate results are where targeted support may have the most impact.

1

NEEDS ATTENTION

- Mood-induced Sleeplessness Risk

2

MODERATE

- Rumination Propensity
- GAD1 Activity

5

OPTIMAL

- Depression Propensity
- Anxiety Propensity
- Stress Adaptation Ability
- Stress Phenotype
- OCD Propensity

1 Depression

17 variants analyzed

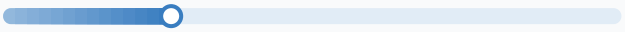
In plain terms: Depression, a complex and multifaceted mental health condition, is influenced by a myriad of factors, including genetic predispositions, environmental stressors, and lifestyle choices. This section delves into the genetic underpinnings of depression, aiming to shed light on how genetic variations can influence an individual's propensity for developing depression and guide personalized intervention strategies.

Research has identified several genetic markers associated with an increased risk of depression. Variations in genes related to neurotransmitter systems, such as serotonin and dopamine, play significant roles in mood regulation and can affect an individual's vulnerability to depression. Additionally, genes involved in the hypothalamic-pituitary-adrenal (HPA) axis, which regulates stress response, may contribute to the development and severity of depressive symptoms. The relationship between genetics and depression is further complicated by environmental influences, such as life stressors, trauma, and social support networks. Genetic predispositions can interact with these external factors, modulating the risk and expression of depression.

Identifying genetic predispositions to depression can inform personalized approaches to mental health care, including preventative measures, lifestyle modifications, and tailored treatment plans. For instance, individuals at higher genetic risk may benefit from early intervention strategies, such as stress management techniques, regular physical activity, and enhanced social support, to mitigate the impact of stressors and reduce the likelihood of developing depression. Genetic testing can also guide the selection of antidepressant medications and psychotherapy approaches, enhancing treatment efficacy and reducing trial-and-error processes. Pharmacogenomics, the study of how genes affect a person's response to drugs, is particularly promising in identifying which antidepressants are likely to be most effective or have fewer side effects for a given individual, based on their genetic makeup.

YOUR RESULTS

Depression Propensity



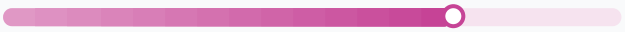
DECREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with decreased anxiety propensity genetic results may have a lower genetic likelihood of experiencing anxiety than average. Stress, life events and other health factors still play a role, so healthy sleep, regular movement and supportive relationships remain helpful, and a healthcare provider can help if symptoms develop.

YOUR RESULTS

Mood-induced Sleeplessness Risk



INCREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with increased mood-induced sleeplessness genetic results are more susceptible to sleep disruption from low mood or emotional fatigue. Low mood and poor sleep can reinforce each other, so addressing both early matters. Keep a consistent sleep schedule, limit evening screens and late caffeine, and use relaxation or mindfulness practices. If sleeplessness is persistent, psychotherapy or cognitive behavioral therapy for insomnia may help, and a healthcare provider can help rule out depression.

RECOMMENDATIONS

SUPPLEMENT

Ashwagandha

300 to 500 mg before bed

Reduces cortisol and HPA axis overactivation that keeps mood-dysregulated individuals awake.

SUPPLEMENT

Magnesium

200 to 400 mg before bed (glycinate)

Promotes sleep by regulating GABA and reducing cortisol; supports muscle relaxation.

DIET

Limit Caffeine After 2 PM

Caffeine's half-life is 5-7 hours; afternoon intake significantly delays sleep onset.

DIET

Butternut Squash Seeds

1 oz with last meal

High in L-tryptophan, a serotonin and melatonin precursor that supports sleep readiness.

LIFESTYLE

Consistent Sleep Schedule

7 to 8 hours/night

Establishing consistent sleep and wake times is the highest-leverage sleep intervention.

LIFESTYLE

Turn Off Electronics at Night

1 hour before bed

Blue light suppresses melatonin and prolongs sleep latency; screen-free wind-down is essential.

FURTHER TESTING

Cortisol (4-point salivary)

Elevated evening cortisol is the primary driver of mood-induced sleeplessness; guides treatment.

FURTHER TESTING

Depression and Anxiety Screening (PHQ-9, GAD-7)

With a healthcare provider

Mood and sleep affect each other, so a short screen can guide next steps.

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
FKBP5 T106-2636C	rs1360780	**CC**	**CRHR1 C241+1631T**	rs242939	**TT**
FKBP5 C*1136A	rs3800373	**AA**	**BDNF C196T**	rs6265	**CC**
SOD2 A47G	rs4880	**AG**	**ARNTL T671-305G**	rs11022778	**TT**
ACE A-239T	rs4291	**AA**	**ABCC9 G3474-1222A**	rs11046205	**AG**
MTHFR A359+160G	rs17367504	**AA**	**CRHR1 A122-1309C**	rs242941	**CC**
ESR1 A453-351G	rs9340799	**GG**	**TPH2 g.G4298T**	rs4570625	**GG**
DIO2 C-143T	rs12885300	**TT**	**CNR1 C1359T**	rs1049353	**CC**
DIO2 T274C	rs225014	**TT**	**FAAH C385A**	rs324420	**AC**
NR3C1 G1184+646C	rs41423247	**GG**			

2 Anxiety

11 variants analyzed

In plain terms: Anxiety disorders, encompassing a range of conditions characterized by excessive fear, worry, and related behavioral disturbances, are influenced by a complex interplay of genetic, environmental, and psychological factors. This section delves into the genetic aspects of anxiety, highlighting how genetic predispositions can shape the susceptibility to and manifestation of anxiety disorders, and how personalized interventions can be developed based on these insights.

Research has identified several genes associated with an increased risk of developing anxiety disorders. Variations in genes that regulate neurotransmitter systems, including serotonin, dopamine, and gamma-aminobutyric acid (GABA), play significant roles in emotional regulation and can influence an individual's predisposition to anxiety. Additionally, genes involved in the stress response system, particularly those affecting the hypothalamic-pituitary-adrenal (HPA) axis, can impact the way individuals respond to stress, potentially increasing vulnerability to anxiety disorders.

The manifestation of anxiety disorders is not solely determined by genetics; environmental factors such as life experiences, trauma, and stress levels significantly interact with genetic predispositions. Understanding the dynamics of gene-environment interactions is crucial for identifying individuals at higher risk and for developing effective prevention and intervention strategies.

Genetic insights offer the potential for personalized approaches to managing anxiety. This includes tailored psychological therapies, lifestyle interventions, and pharmacological treatments. For instance, individuals with specific genetic variations may respond better to certain types of antidepressants or anxiolytics, or may benefit more from specific psychotherapeutic approaches, such as cognitive-behavioral therapy (CBT). Beyond medical treatment, understanding one's genetic predispositions can guide the adoption of lifestyle interventions and supportive measures that reduce anxiety symptoms. These may include stress management techniques, regular physical activity, dietary adjustments, and mindfulness practices, all of which can be personalized to align with individual genetic profiles and lifestyle preferences.

YOUR RESULTS

Anxiety Propensity

DECREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with decreased depression propensity genetic results have a somewhat decreased genetic likelihood of depression compared to the average population. Environmental factors and life experiences still influence mental health regardless of genetic baseline. Maintain proactive mental health practices, including social connection, regular physical activity, and stress management, to support ongoing well-being.

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
MAO-B T1300-36C	rs1799836	**TT**	**BDNF C196T**	rs6265	**CC**
MAO-A T891G	rs6323	**TG**	**ADRB2 G46A**	rs1042713	**AG**
ADRA1A A883+323G	rs573514	**AA**	**ADORA2A T1083C**	rs5751876	**CC**
ADRA1A C883+3787T	rs17426222	**CC**	**PER3 C2566G**	rs228697	**CC**
GAD1 G-151A	rs3749034	**AG**	**SLC1A1 T1110C**	rs301430	**TT**
FAAH C385A	rs324420	**AC**			

3 Stress Response

12 variants analyzed

> **In plain terms:** Stress, a universal human experience, varies widely in its impact from one individual to another, influenced by a complex web of genetic, environmental, and psychological factors. This section delves into the genetic factors that underlie these individual differences in stress responses, exploring how genetic variability contributes to resilience, vulnerability, and the overall physiological and psychological impact of stress.

Key genes involved in the regulation of the hypothalamic-pituitary-adrenal (HPA) axis, which orchestrates the body's response to stress, have been identified as critical in determining individual stress resilience. Variations in these genes can influence cortisol levels, the primary stress hormone, affecting how individuals react to and recover from stress. Genes play significant roles in these processes, with certain variants linked to either heightened stress sensitivity or increased resilience.

Beyond the HPA axis, genetic variations in neurotransmitter systems, including those for serotonin, dopamine, and gamma-aminobutyric acid (GABA), also contribute to individual differences in stress response. These variations can affect mood regulation, anxiety levels, and the threshold for experiencing stress, thus influencing an individual's susceptibility to stress-related disorders.

Understanding an individual's genetic predispositions related to stress response can inform personalized interventions aimed at enhancing resilience and mitigating stress-related health impacts. This may include targeted psychological therapies, stress management strategies, lifestyle changes, and, where appropriate, pharmacological treatments designed to address specific genetic vulnerabilities. The interplay between genetic predispositions and lifestyle/environmental factors is crucial in shaping the stress response. Factors such as physical activity, diet, sleep quality, and social support can interact with genetic predispositions, either amplifying or mitigating stress impacts. Recognizing this interaction is key to developing comprehensive strategies for stress management that are tailored to an individual's genetic makeup and life circumstances.

YOUR RESULTS

Stress Adaptation Ability



TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with typical stress adaptation level genetic results exhibit an average ability to cope with stress, aligning with the general population's resilience. Regular exercise, balanced nutrition and adequate sleep can further support stress resilience.

YOUR RESULTS

Stress Phenotype



MEDIATOR

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with mediator genetic results show a balanced approach to stress, with an average response to stressful situations. Regular physical activity, balanced nutrition and relaxation techniques can support stress management.

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
COMT C186T	rs4633	**CT**	**OXTR G922+4581A**	rs53576	**GG**
COMT G597A	rs769224	**GG**	**FAAH C385A**	rs324420	**AC**
MAO-A T891G	rs6323	**TG**	**HTR1B1 C861G**	rs6296	**CC**
TCF7L2 C450+33966T	rs7903146	**CC**	**HTR1A (C>G)**	rs6295	**GG**
MGLL G263-1443A	rs604300	**GG**	**CNR1 C1359T**	rs1049353	**CC**
COMT G472A	rs4680	**GA**	**FKBP5 T106-2636C**	rs1360780	**CC**

4 OCD & Rumination

8 variants analyzed

In plain terms: Obsessive-Compulsive Disorder (OCD) and rumination represent complex psychological conditions characterized by repetitive thoughts and behaviors. These conditions are influenced by a tapestry of genetic, environmental, and neurobiological factors. This section aims to unpack the genetic components that contribute to the predisposition and manifestation of OCD and rumination, offering insights into personalized intervention strategies.

Research has identified several genetic markers associated with an increased risk of developing OCD. Variants in genes related to the serotonin system, such as SLC1A1, which encodes for the serotonin transporter, have been implicated in OCD's pathogenesis. Additionally, genes involved in the dopamine system and brain-derived neurotrophic factor (BDNF) have been linked to OCD, suggesting that multiple neurotransmitter systems contribute to the disorder.

While specific genetic studies on rumination are less prevalent than those on OCD, the overlap in cognitive and neural processes suggests that similar genetic factors may be involved. Genetic variations that affect mood regulation, stress response, and executive function may contribute to the tendency to engage in rumination, a maladaptive pattern of persistent negative thinking.

The expression of genetic predispositions to OCD and rumination is significantly influenced by environmental factors, such as stress, trauma, and early life experiences. Understanding the complex interactions between genetic makeup and environmental exposures is crucial for identifying individuals at risk and developing effective preventive measures and interventions. Insights into the genetic underpinnings of OCD and rumination can guide personalized treatment approaches, including pharmacological and psychotherapeutic strategies. For example, individuals with specific genetic variants may respond better to certain types of serotonin reuptake inhibitors (SSRIs) or benefit more from cognitive-behavioral therapy (CBT) techniques tailored to address rumination and compulsive behaviors.

YOUR RESULTS

OCD Propensity



TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with typical OCD propensity genetic results have a likelihood of developing OCD consistent with the general population, their genetic profile neither elevates nor reduces risk meaningfully. Environmental factors, stress, and personal history still influence mental health outcomes for everyone. Maintain general mental health awareness and seek professional support if OCD-type symptoms such as intrusive thoughts or compulsive behaviors emerge.

YOUR RESULTS

Rumination Propensity



INCREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with increased rumination propensity genetic results may experience more obsessive-compulsive and rumination type thoughts and behaviors. These are influenced by genetic variations in serotonin, dopamine, and stress-response pathways. Genetic predisposition interacts with environmental stressors and early life experiences to shape individual risk.

RECOMMENDATIONS

SUPPLEMENT

Omega-3 Fatty Acids (EPA/DHA)

1,000 to 2,000 mg daily (EPA-dominant)

Reduces neuroinflammation, regulates serotonin and dopamine, and supports cognitive flexibility.

SUPPLEMENT

L-Theanine

200 mg daily

Promotes relaxation via GABA and serotonin; increases alpha brain wave activity that quiets rumination.

DIET

Anti-Inflammatory Diet

Neuroinflammation drives ruminative thinking; antioxidant-rich foods directly address this.

DIET

Limit Refined Sugars

Blood sugar instability amplifies negative emotional states and worsens ruminative loops.

LIFESTYLE

Meditation

10 to 20 min daily

Mindfulness meditation is the most evidence-based intervention for breaking ruminative thought patterns.

LIFESTYLE

Adequate Sleep

7 to 8 hours/night

Sleep deprivation amplifies negative emotional processing and increases ruminative thinking.

FURTHER TESTING

Vitamin D (25-OH)

Low vitamin D is linked with low mood.

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
TNF G-238A	rs361525	**GG**	**BDNF C196T**	rs6265	**CC**
ANKK1 G2137A	rs1800497	**GG**	**HTR2A C-998T**	rs6311	**CT**
SLC1A1 T1110C	rs301430	**TT**	**MTHFD1L G643+208A**	rs11754661	**AG**
COMT G472A	rs4680	**GA**	**SLC1A1 C1194-956T**	rs301434	**TC**

5 GAD1 Activity

4 variants analyzed

In plain terms: GAD1 encodes glutamate decarboxylase, the enzyme that converts glutamate into GABA, the brain's main calming neurotransmitter. This section looks at genetic variation that may influence how efficiently this conversion takes place, which can play a role in mood, stress response and relaxation.

YOUR RESULTS

GAD1 Activity

DECREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with decreased gad1 activity genetic results are likely to have decreased conversion of glutamate to GABA in the brain. Some anecdotal reports have linked this to various food sensitivities including MSG, grains, and glutamic acid, yet there are currently no scientific studies to support this assertion.

RECOMMENDATIONS

SUPPLEMENT

Valerian Root (standardized to valerenic acid)

300-600 mg at bedtime or 150 mg 3x/day

Valerenic acid inhibits GABA-A receptor degradation and may weakly inhibit GABA-T (GABA transaminase), effectively increasing available GABA. Most relevant for GAD1 impairment because it works downstream of the conversion bottleneck by increasing GABA receptor sensitivity rather than requiring more GABA synthesis.

DIET

Ketogenic or Low-Carbohydrate Diet

Net carbs <50g/day for therapeutic effect

Ketogenic metabolism increases brain GABA:glutamate ratio through two mechanisms: beta-hydroxybutyrate inhibits vesicular glutamate transport (reduces excitatory neurotransmission) and upregulates GAD1 expression.

DIET

Restrict Aspartame and Artificial Sweeteners

Aspartame is metabolized to phenylalanine which competitively inhibits tyrosine hydroxylase, reducing catecholamine synthesis while simultaneously failing to buffer glutamate.

LIFESTYLE

Yoga Nidra (Non-Sleep Deep Rest, NSDR)

20-30 min daily

Yoga Nidra produces the most reliably measured GABA increase of any non-pharmacological intervention with a 27% increase in brain GABA on MRI spectroscopy after a single 60-minute session.

FURTHER TESTING

EEG Neurofeedback Assessment (beta/alpha/theta mapping)

Qeeg assessment with neurofeedback practitioner

GAD1 insufficiency produces characteristic EEG signatures: excess high-beta waves (anxious rumination) and insufficient alpha waves (GABAergic inhibitory tone). qEEG-guided neurofeedback directly trains GABAergic alpha wave production, compensating for GAD1 genetic impairment at the functional brain level.

FURTHER TESTING

Plasma Amino Acids Panel (glutamine, glutamate, taurine, GABA)

Directly measures the amino acid substrates and products of GAD1 activity.

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
GAD1 G-151A	rs3749034	**AG**	**GAD1 G-64+2259T**	rs3791878	**GG**
GAD1 C-64+894A	rs12185692	**AC**	**GAD1 C146-3245T**	rs3828275	**TC**

APPENDIX A

Supplement Recommendations

Based on your results

Supplements listed below are recommended for panels where at least one result was outside the typical range.

SUPPLEMENT	RATIONALE	PANEL
Ashwagandha 300 to 500 mg before bed	Reduces cortisol and HPA axis overactivation that keeps mood-dysregulated individuals awake.	Depression
Magnesium 200 to 400 mg before bed (glycinate)	Promotes sleep by regulating GABA and reducing cortisol; supports muscle relaxation.	Depression
Omega-3 Fatty Acids (EPA/DHA) 1,000 to 2,000 mg daily (EPA-dominant)	Reduces neuroinflammation, regulates serotonin and dopamine, and supports cognitive flexibility.	OCD & Rumination
L-Theanine 200 mg daily	Promotes relaxation via GABA and serotonin; increases alpha brain wave activity that quiets rumination.	OCD & Rumination
Valerian Root (standardized to valerenic acid) 300-600 mg at bedtime or 150 mg 3x/day	Valerenic acid inhibits GABA-A receptor degradation and may weakly inhibit GABA-T (GABA transaminase), effectively increasing available GABA. Most relevant for GAD1 impairment because it works downstream of the conversion bottleneck by increasing GABA receptor sensitivity rather than requiring more GABA synthesis.	GAD1 Activity

APPENDIX B

Further Testing Recommendations

Based on your results

Lab tests and assessments recommended for panels where at least one result was outside the typical range.

TEST / ASSESSMENT	CLINICAL RATIONALE	PANEL
Cortisol (4-point salivary)	Elevated evening cortisol is the primary driver of mood-induced sleeplessness; guides treatment.	Depression
Depression and Anxiety Screening (PHQ-9, GAD-7) With a healthcare provider	Mood and sleep affect each other, so a short screen can guide next steps.	Depression
Vitamin D (25-OH)	Low vitamin D is linked with low mood.	OCD & Rumination
EEG Neurofeedback Assessment (beta/alpha/theta mapping) Qeeg assessment with neurofeedback practitioner	GAD1 insufficiency produces characteristic EEG signatures: excess high-beta waves (anxious rumination) and insufficient alpha waves (GABAergic inhibitory tone). qEEG-guided neurofeedback directly trains GABAergic alpha wave production, compensating for GAD1 genetic impairment at the functional brain level.	GAD1 Activity
Plasma Amino Acids Panel (glutamine, glutamate, taurine, GABA)	Directly measures the amino acid substrates and products of GAD1 activity.	GAD1 Activity