MOSAIC DIAGNOSTICS

DNA Signature Mind & Mood

GENETICS REPORT

1	6	120
Report	Report Panels	Gene Variants

Example Client
Prepared for

Order ID: _____
DOB: _____

August 26, 2026
Report Date
Practitioner: Erika Gray

Table of Contents

Result Scores Summary

Overview of all 20 engine results across this section.

1	9	10
NEEDS ATTENTION	MODERATE	OPTIMAL

Needs Attention (1)

Mood-Induced Sleeplessness Risk	Increased	Sleep

Moderate (9)

Depression Propensity	Increased	Mood & Behavior
Stress Adaptation	Below average	Mood & Behavior
Rumination	Increased	Mood & Behavior
Anxiety Propensity	Increased	Mood & Behavior
GAD1 Activity	Low activity	Mood & Behavior
Parkinson's Propensity	Increased	Neurodegeneration Risk
Reported Sleep Quality	Below average	Sleep
MTHFR Activity	60-70% enzyme activity	Neuro-Nutrients
Choline Need	Typical	Neuro-Nutrients

Optimal (10)

OCD Likelihood	Typical	Mood & Behavior
Stress Phenotype	Mediator	Mood & Behavior
General Cognition	Above average	Cognitive Function
White Matter Damage Propensity	Typical	Cognitive Function

Memory	Typical	Cognitive Function
Attention	Typical	Cognitive Function
MCI	Decreased	Cognitive Function
Alzheimer's Propensity	Typical	Neurodegeneration Risk
CoQ10 Supplementation Benefit	Typical	Neuro-Nutrients
Vitamin B9 Need	Typical	Neuro-Nutrients

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 risk assessments
- ✕ False positives
- ✕ Predictions about future health conditions
- ✕ How lifestyle factors outside of genetics impact health outcomes

This commentary is designed solely to support the practitioner's clinical evaluation, as all diagnostic interpretations, treatment plans, and medical management decisions remain entirely under the practitioner's independent authority. The findings in this report outline genetic predispositions for informational purposes only and do not constitute diagnostic or treatment recommendations, guarantee health outcomes, or replace direct clinical consultation. These statements have not been evaluated by the Food and Drug Administration, and our tests are not intended to diagnose, treat, cure, or prevent any disease. Patients must be advised not to initiate or modify any diet, exercise, medication, or lifestyle regimen without consultation with a licensed healthcare provider.

1 Mood & Behavior

34 variants analyzed

DEPRESSION PROPENSITY

YOUR RESULTS

INCREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with increased depression propensity genetics have a heightened genetic susceptibility to depression above the general population baseline. Recognizing this risk supports earlier and more intentional mental health self-monitoring, including building a strong support network and exploring therapeutic strategies proactively. Consult a mental health professional to establish a management plan before symptoms become severe.

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

Vitamin D3 + K2

D3 5,000 IU + K2 100 mcg/day

Vitamin D receptor is present throughout the limbic system; vitamin D deficiency impairs serotonin synthesis and increases neuroinflammatory cytokines that drive depression. Meta-analysis (n=73,000+) confirms inverse relationship between 25-OH vitamin D and depression; supplementation reduces depression risk by 23%.

SUPPLEMENT

Saffron Extract

28-30 mg/day

At this dose, saffron extract has demonstrated antidepressant efficacy comparable to fluoxetine and sertraline in RCTs, with superior sexual side effect profile. Mechanism: inhibits serotonin, dopamine, and norepinephrine reuptake simultaneously plus direct NMDA receptor modulation. One of the few non-pharmaceutical supplements with head-to-head SSRI comparison data.

DIET

Fermented Foods + Prebiotic Fiber (gut-brain axis support)

Daily: kefir, yogurt, kimchi + 25-30g fiber

The gut-brain axis is the strongest emerging target in depression biology. A 4-week dietary intervention (SMILES trial) using Mediterranean-style eating showed larger depression effect size than pharmacotherapy in some subgroups. Fermented foods directly increase tryptophan availability via reduced kynurenine pathway competition.

DIET

Reduce Refined Sugar and Ultra-Processed Foods

Sugar and UPF intake independently predict depression incidence in prospective studies. Mechanism: promotes neuroinflammation via advanced glycation end-products and dysbiosis-mediated LPS translocation; disrupts insulin signaling in the prefrontal cortex; promotes tryptophan shunting toward kynurenine (neuroinflammatory) over serotonin pathway.

LIFESTYLE

Behavioral Activation (structured activity scheduling)

Daily minimum 2 absorbing activities

Behavioral activation is equally or more effective than CBT for depression in multiple RCTs. The key mechanism is re-engagement with positive reinforcement schedules that depression neurologically suppresses by restoring dopaminergic reward signaling through real-world behavioral reconnection, not insight or cognition.

LIFESTYLE

Social Connection (scheduled, not incidental)

Weekly minimum 2 meaningful interactions

Loneliness and social isolation produce the same inflammatory and HPA changes as major depression; they are not just consequences but neurobiological causes of depression. Intentional scheduling of meaningful social contact (not social media) is a primary intervention, not a nice-to-have.

FURTHER TESTING

hsCRP + IL-6 (Inflammatory Subtype Screen)

The most clinically useful triage tool for depression subtypes. CRP >1 mg/L or IL-6 >2 pg/mL identifies the 'inflammatory depression' subtype, these individuals respond poorly to SSRIs but well to anti-inflammatory interventions (EPA omega-3, exercise, curcumin). Results guide treatment selection, not just lifestyle advice.

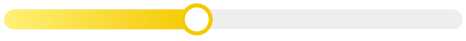
FURTHER TESTING

Genetic Pharmacogenomics Panel (CYP2D6, CYP2C19, SLC6A4, COMT)

Identifies which antidepressants are likely to be effective vs. problematic based on metabolizer phenotype. SLC6A4 (serotonin transporter) and COMT variants predict SSRI response; CYP2D6 predicts metabolism of TCAs and many SSRIs. Reduces antidepressant trial-and-error time from years to weeks.

STRESS ADAPTATION

YOUR RESULTS



BELOW AVERAGE

UNDERSTANDING WHAT THIS MAY MEAN

Those with below average Stress Adaptation genetic markers suggest a somewhat reduced capacity to manage stress effectively compared to the general population. While not as pronounced as the low adaptation group, individuals here might find stress more challenging to navigate and could benefit from proactive stress management practices. Engaging in stress reduction activities, cultivating resilience-building habits, and possibly working with a counselor or therapist can help enhance their stress coping mechanisms

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

Eleuthero (Siberian Ginseng - Eleutherococcus senticosus)

300-600 mg/day (standardized to eleutherosides)

The original adaptogen with 40+ years of Russian military research. Specifically increases stress resistance and cognitive performance under sustained load which is distinct from acute anxiety relief. Most relevant for stress adaptation genotypes characterized by reduced capacity to maintain performance under prolonged stress rather than acute anxiety.

SUPPLEMENT

Panax Ginseng

200-400 mg/day (standardized to >4% ginsenosides)

Improves HPA axis recovery speed, specifically reducing the time to cortisol normalization after stress exposure. In individuals with stress adaptation SNPs, this recovery latency is prolonged; Panax ginseng addresses the recovery phase that Rhodiola and Ashwagandha (which reduce acute cortisol) do not.

DIET

Time-Restricted Eating (TRE) with morning breakfast anchor

8-10 hour eating window, first meal within 1 hour of waking

TRE aligns cortisol rhythms with food timing, improving HPA axis diurnal regularity. Morning breakfast specifically stabilizes the cortisol awakening response, the critical physiological signal that stress adaptation variants often dysregulate. Skipping breakfast worsens cortisol chaos in stress-susceptible genetics.

DIET

Adaptogenic Herb Culinary Use (ashwagandha, holy basil, ginger in cooking)

Daily integration

Culinary use of adaptogenic herbs provides consistent low-dose exposure that may produce tonic HPA modulation without the dose-concentration peaks of capsule supplementation. Holy basil (tulsi) tea daily is the most accessible format.

LIFESTYLE

Heart Rate Variability Training (device-guided)

20 min/day: HeartMath Inner Balance, Garmin HRV4Training, or Elite HRV

HRV is the most quantifiable biomarker of stress adaptation capacity, directly reflects autonomic nervous system flexibility. Device-guided biofeedback at resonance frequency (0.1 Hz / 6 breaths/minute) reliably increases HRV 30-50% over 8 weeks. The most evidence-based lifestyle intervention for stress adaptation genotypes specifically.

LIFESTYLE

Cold Water Immersion (2-3 min cold shower or plunge)

3-5x/week, morning

Regular cold exposure habituates the sympathoadrenal response, over time, cold-adapted individuals show smaller cortisol spikes and faster normalization in response to psychological stressors. The physiological stress of cold water is a hormetic training stimulus for the same HPA axis pathways that stress adaptation SNPs impair.

FURTHER TESTING

Cortisol metabolites + DHEA

Maps the full cortisol production, clearance, and metabolite patterns, not just a single cortisol reading. The DHEA-S:cortisol ratio and the total cortisol metabolite load reveal whether stress adaptation impairment is due to overproduction, under-production, or excessive clearance. Guides whether supporting production or reducing clearance is the therapeutic target.

FURTHER TESTING

Heart Rate Variability Baseline (wearable - 7-day average)

Oura, WHOOP, Garmin, or Apple Watch

7-day average HRV baseline provides an objective quantification of current stress adaptation capacity without requiring lab testing. Allows tracking of intervention response in real time. Most cost-effective first-line assessment; low HRV with stress adaptation SNPs confirms genotype-phenotype expression.

RUMINATION

YOUR RESULTS

INCREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with increased propensity for rumination have genetic markers that may predispose them to more frequently engage in rumination. This heightened tendency can impact mental health, leading to increased stress, anxiety, and depression risk. Awareness of this predisposition allows individuals to take proactive steps in managing their mental health. Implementing techniques such as cognitive-behavioral strategies, seeking professional counseling, and practicing stress-reduction activities can be particularly effective in reducing rumination and its negative effects on well-being.

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

Saffron Extract

28-30 mg/day

Saffron directly reduces ruminative cognition through serotonin reuptake inhibition and NMDA modulation which are the two pathways most implicated in the default mode network (DMN) hyperactivity that sustains rumination. Two RCTs specifically measuring rumination (not just overall mood) show significant improvement.

SUPPLEMENT

Lion's Mane Mushroom (Hericium erinaceus)

500-1,000 mg/day (standardized extract)

Stimulates NGF (nerve growth factor) production in the prefrontal cortex, the regulatory region that dampens DMN rumination. A Japanese RCT showed significant reduction in depression and anxiety over 4 weeks; mechanism involves prefrontal neuroplasticity that improves top-down control of ruminative circuits.

DIET

High-Tryptophan + Moderate-Carbohydrate Meals (especially evening)

Turkey, eggs, pumpkin seeds, oats at dinner

Serotonin is the primary neuromodulator of DMN regulation; insufficient serotonin sustains ruminative activity. Evening tryptophan + moderate carbohydrate maximizes serotonin synthesis and melatonin conversion, reducing overnight rumination and improving next-day emotional baseline.

DIET

Polyphenol-Rich Diet (berries, dark chocolate, olive oil, green tea)

Polyphenols upregulate BDNF, which supports hippocampal neuroplasticity needed for contextualizing ruminative memories and placing them in perspective. BDNF deficiency is a confirmed correlate of ruminative depression; dietary polyphenols are the most accessible BDNF-elevating nutritional strategy.

LIFESTYLE

Physical Exercise (aerobic, especially outdoor)

30-45 min, 4x/week; nature setting preferred

Exercise acutely disrupts rumination by engaging the task-positive network (TPN) which is mutually inhibitory with the DMN. Nature-based exercise (green exercise) additionally reduces prefrontal cortex activity associated with self-referential rumination. The mutual inhibition of TPN/DMN is the most mechanistically grounded lifestyle intervention for rumination.

LIFESTYLE

Mindfulness-Based Cognitive Therapy (MBCT)

8-week group program, 2 hrs/week + daily 20-45 min practice

MBCT is the most evidence-based psychological intervention for rumination specifically. Teaches decentering (observing thoughts as mental events rather than facts), which directly disrupts the self-referential DMN loops that constitute rumination. 50% relapse prevention in recurrent depression.

FURTHER TESTING

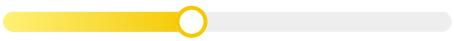
Salivary Cortisol Awakening Response (CAR)

Home collection, 3 samples at 0, 15, 30 min post-waking

Blunted CAR is the most specific biological marker of ruminative depression, it indicates the stress system cannot mount an appropriate morning regulatory response, sustaining ruminative overnight processing into the day. A simple, affordable home test that distinguishes ruminative depression from other subtypes.

ANXIETY PROPENSITY

YOUR RESULTS



INCREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with increased Depression propensity genetics have a heightened genetic susceptibility to depression above the general population baseline. Recognizing this risk supports earlier and more intentional mental health self-monitoring, including building a strong support network and exploring therapeutic strategies proactively. Consult a mental health professional to establish a management plan before symptoms become severe.

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

Ashwagandha (KSM-66 or Sensoril standardized extract)

300-600 mg/day

Reduces cortisol 15-30% and anxiety scores in multiple RCTs. Mechanism distinct from GABA modulation; ashwagandha downregulates HPA axis reactivity and reduces cortisol-driven amygdala hyper-reactivity. Particularly effective for anxiety with accompanying fatigue or sleep disruption; effects build over 4-8 weeks.

SUPPLEMENT

Kava (standardized to kavalactones)

120-250 mg kavalactones/day

The strongest herbal anxiolytic with clinical trial data; multiple meta-analyses confirm efficacy for generalized anxiety comparable to low-dose benzodiazepines, without dependency risk at normal doses. Mechanism: positive allosteric modulation of GABA-A receptors via kavain and dihydrokavain. Use water-soluble noble kava extract; avoid ethanol/acetone extracts associated with hepatotoxicity.

DIET

Low-Sugar, High-Fiber Diet (blood glucose stability = anxiety reduction)

Blood glucose fluctuations directly drive anxiety symptoms through adrenaline-mediated glycemic correction. A low-glycemic, fiber-rich diet stabilizes glucose throughout the day, eliminating a major non-psychological driver of anxiety symptoms. Practical: always pair carbohydrates with fat or protein.

DIET

Omega-3 (EPA-dominant, >60% EPA)

2-3 g EPA/day from food or supplement

EPA-dominant omega-3 specifically reduces amygdala hyperreactivity and HPA axis over-activation, the neurobiological drivers of anxiety propensity. High EPA fish (mackerel, sardines, herring) or EPA-dominant supplement (not standard fish oil with balanced DHA:EPA).

LIFESTYLE

Reduce Caffeine (critical for anxiety propensity genetics)

<100 mg/day total; <1 coffee

Caffeine directly antagonizes adenosine receptors and increases norepinephrine release, the catecholamine most responsible for anxious arousal. Genetic variants in CYP1A2 and ADORA2A determine caffeine's anxiogenic impact; anxiety propensity genetics typically indicate heightened caffeine sensitivity.

LIFESTYLE

Vagal Nerve Stimulation (humming, gargling, cold face immersion)

5-10 min daily

Directly activates the vagus nerve, the primary neural pathway of the parasympathetic 'brake.' Humming, singing, gargling, and facial cold water immersion are the most accessible forms. Increases vagal tone persistently with daily practice; more targeted than general mindfulness for acute anxiety.

FURTHER TESTING

Organic Acids Test (OAT) - Neurotransmitter Metabolites

Mosaic Diagnostics

Measures kynurenic acid, quinolinic acid, HVA (dopamine), VMA (norepinephrine), 5-HIAA (serotonin), and GABA markers which are comprehensive neurochemical guides to different anxiety subtypes. Suggests whether serotonin, GABA, catecholamine, or inflammatory pathways are driving the anxiety phenotype.

FURTHER TESTING

Thyroid Panel (TSH, Free T3, Free T4, TPO antibodies)

Subclinical hyperthyroidism and Hashimoto's thyroiditis are among the most commonly missed organic causes of anxiety. TSH below 1.0 or TPO antibodies above normal with anxiety symptoms warrant full thyroid investigation before attributing anxiety to genetic propensity alone.

OCD LIKELIHOOD

YOUR RESULTS

TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with typical OCD propensity genetics 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.

GAD1 ACTIVITY

YOUR RESULTS

LOW ACTIVITY

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with Low Activity GAD1 genetics are likely to have reduced conversion of glutamate to GABA in the brain. Some anecdotal reports link low GAD1 activity to sensitivities to MSG, grains, and glutamic acid; however, no scientific studies currently support this association.

LITERATURE DERIVED RECOMMENDATION

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.

SUPPLEMENT

GABA + L-Theanine (combined)

GABA 250 mg + Theanine 200 mg

Emerging evidence suggests that GABA combined with L-theanine crosses the BBB more effectively than GABA alone through complementary transport mechanisms. Theanine's alpha-wave induction supports the inhibitory tone that GAD1 impairment fails to supply, while combined GABA provides direct receptor substrate.

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. Clinical studies in epilepsy (which shares GAD1 insufficiency biology with anxiety) confirm dramatic GABA elevation on ketogenic diet.

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. In individuals with GAD1 impairment, the already-elevated glutamate is further unopposed.

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. The brain-state achieved (between sleep and waking) is uniquely associated with GABAergic upregulation. More effective than standard meditation for GAD1 impairment specifically.

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. High glutamate + low GABA with clinical anxiety confirms functional GAD1 insufficiency; high glutamine (storage form) with low conversion suggests cofactor (P5P) deficiency is the limiting factor.

STRESS PHENOTYPE

YOUR RESULTS

MEDIATOR

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with Mediator type genetics have a balanced approach to stress, with an average response to stressful situations. Maintaining a healthy lifestyle, including regular physical activity, balanced nutrition, and relaxation techniques, can support optimal stress management.

2 Cognitive Function

18 variants analyzed

GENERAL COGNITION

YOUR RESULTS

ABOVE AVERAGE

UNDERSTANDING WHAT THIS MAY MEAN

Individuals identified with an above-average predisposition for general cognition tend to perform better than the average population in cognitive tasks. This advantage spans memory, attention, problem-solving abilities, and processing speed. To maximize their cognitive potential, it is advisable for these individuals to continually challenge their cognitive abilities, pursue lifelong learning, and maintain a lifestyle that supports mental health and cognitive wellness.

WHITE MATTER DAMAGE PROPENSITY

YOUR RESULTS

TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individual with a typical risk have an average likelihood of developing leukoaraiosis, aligning with the general population. This indicates a standard susceptibility to the brain white matter changes that leukoaraiosis entails, making general preventive measures and monitoring for vascular health still advisable.

MEMORY

YOUR RESULTS

TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Those with a typical predisposition for memory are expected to have recall abilities that align with the average population. Their capacity to remember information, learn new concepts, and recall past experiences should match general expectations. To support and possibly enhance memory function, consider engage in regular cognitive challenges, healthy lifestyle practices, and continuous learning opportunities.

ATTENTION

YOUR RESULTS

TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Those with a typical predisposition for attention are likely to exhibit focus and concentration abilities that align with the average population. Their capability to stay engaged with tasks, avoid distractions, and manage multiple tasks simultaneously should match general expectations. Engaging in activities that require sustained focus, along with maintaining a healthy lifestyle and practicing mindfulness, can support and potentially improve attentional skills

MCI

YOUR RESULTS

DECREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with a decreased genetic risk are less likely to develop mild cognitive impairment, a condition characterized by slight but noticeable declines in cognitive abilities, including memory and thinking skills. This reduced risk suggests a lower probability of experiencing these early cognitive changes compared to the average population.

3 Neurodegeneration Risk

46 variants analyzed

PARKINSON'S PROPENSITY

YOUR RESULTS

INCREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with increased genetics show a higher than average likelihood of developing Parkinson's. Monitoring neurological health and adopting preventive measures, such as regular exercise and a healthy diet, can be beneficial.

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

NAC (N-Acetylcysteine) - IV or oral high-dose

IV: 50 mg/kg biweekly; Oral: 600 mg 3x/day

Substantia nigra neurons are selectively vulnerable to oxidative stress because they produce neuromelanin (requiring high dopamine metabolism) with relatively low endogenous glutathione. An RCT of IV NAC in early Parkinson's showed significant DaT-SPECT improvement, the first supplement to show dopaminergic neuron protection by objective imaging.

SUPPLEMENT

Creatine Monohydrate

5-10 g/day

Creatine supplementation showed dopaminergic neuroprotection in multiple animal models and epidemiological data shows inverse relationship between creatine intake and Parkinson's risk. While the NINDS NEAT-PD trial found no disease modification after diagnosis, preventive use in genetic risk contexts (before symptoms) remains biologically supported.

DIET

Reduce Dairy Intake (association with PD risk)

<1 serving/day

Prospective studies consistently show dairy consumption is the strongest dietary risk factor for Parkinson's disease, particularly low-fat dairy (skim milk). Proposed mechanisms include urate-lowering effects of dairy (urate is neuroprotective for dopaminergic neurons) and potential organochlorine pesticide contamination in commercial dairy. Replace with plant-based calcium sources.

DIET

Mediterranean Diet with Emphasis on Urate-Preserving Foods (legumes, coffee)

Mediterranean base; 2-4 cups coffee/day

Uric acid is neuroprotective for substantia nigra neurons, GWAS studies show urate-raising genetic variants reduce Parkinson's risk. Coffee (urate-raising via xanthine oxidase inhibition) has the strongest inverse dietary association with PD risk in prospective data. Legumes similarly raise urate while providing dietary magnesium and B vitamins.

LIFESTYLE

Vigorous Aerobic Exercise (SPARX trial protocol)

High-intensity aerobic: 80-85% max HR, 3x/week, 45 min

The SPARX trial showed that high-intensity (not moderate) treadmill exercise slowed Parkinson's motor progression at 6 months, the first prospective evidence of disease modification through exercise. For genetic risk contexts, vigorous aerobic exercise is the most evidence-supported neuroprotective intervention available.

LIFESTYLE

Boxing / Martial Arts / Dance Training

Rock Steady Boxing or similar adaptive program 2x/ week

Non-contact boxing programs designed for Parkinson's risk show improvements in balance, gait, and motor control that conventional exercise does not match. The combinatorial demand on basal ganglia circuitry, cerebellum, and motor cortex produces neuroplastic gains in circuits vulnerable to Parkinson's pathology.

FURTHER TESTING

Serum Uric Acid

Target 5.5-7.0 mg/dL

Uric acid is the most well-documented natural neuroprotective compound for dopaminergic neurons. Prospective studies show each 1 mg/dL increase in uric acid reduces Parkinson's risk by 8-11% in men. Target the upper-normal range (6-7 mg/dL) rather than minimizing urate; interventions that lower uric acid should be carefully weighed against neurological risk.

FURTHER TESTING

Skin Biopsy for Alpha-Synuclein Detection

Alpha-synuclein aggregates deposit in cutaneous autonomic nerve fibers years before motor symptoms in Parkinson's disease. Skin biopsy (3mm punch, 3 sites) is now a validated biomarker for prodromal Parkinson's pathology. Available through Amprion laboratory; provides the earliest available biological confirmation of Parkinson's pathology in genetic risk contexts.

ALZHEIMER'S PROPENSITY

YOUR RESULTS

TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Those at a typical risk level possess an average likelihood of developing Alzheimer's disease, reflecting the general population's predisposition. This group is advised to follow general recommendations for cognitive health, such as engaging in regular physical and mental activities, maintaining a healthy diet, and monitoring and managing cardiovascular health.

4 Sleep

10 variants analyzed

REPORTED SLEEP QUALITY

YOUR RESULTS

BELOW AVERAGE

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with below average subjective sleep quality genetics may experience lower-than-average self-reported sleep satisfaction. Subjective and objective sleep quality do not always align. A biometric tracker can help determine whether interventions are working. Consultation with a provider or coach may be beneficial if poor sleep quality is persistent.

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

Magnesium L-Threonate

1.5-2 g/day (evening dose 30-60 min before bed)

Increases brain magnesium specifically in the structures regulating sleep architecture, the pineal gland and hypothalamic sleep centers. RCTs in older adults with poor sleep show magnesium supplementation increases slow-wave sleep (deep sleep) duration and reduces sleep onset latency. L-threonate's brain penetrance is specific and superior to glycinate for sleep quality (though glycinate also has sedative properties).

SUPPLEMENT

Apigenin

50 mg/day (from chamomile extract or pure apigenin)

GABA-A receptor partial agonist, a flavonoid that binds the benzodiazepine allosteric site without causing dependency or tolerance. The recommendation (50 mg pure apigenin) is based on its GABA-A binding data and sleep quality evidence from chamomile RCTs. Reduces sleep onset latency and cortisol in the evening transition.

DIET

Tart Cherry Juice (Montmorency)

8-16 oz concentrate/day or 1,200 mg tart cherry extract

The highest food source of melatonin and tryptophan simultaneously. Two RCTs showed tart cherry juice increased total sleep time by ~90 minutes and reduced insomnia severity. Contains the melatonin precursor tryptophan AND proanthocyanidins that inhibit tryptophan breakdown, increasing its availability for melatonin synthesis.

DIET

High-Carbohydrate Evening Meal (tryptophan transport optimization)

Moderate carbohydrates at dinner, low fat

Dietary carbohydrates at dinner increase the tryptophan:LNAA ratio in plasma, maximizing brain tryptophan uptake and melatonin production. This is the opposite of a protein-heavy dinner, which floods plasma with competing amino acids that reduce tryptophan transport efficiency. Note: incompatible with ketogenic diet protocols recommended for GAD1 Activity or MCI traits.

LIFESTYLE

Strict Sleep-Wake Timing (circadian anchor)

Same wake time every day +/- 20 min (including weekends)

Circadian rhythm consistency is the single highest-impact sleep quality intervention. The sleep pressure system (adenosine) and circadian timing system must be synchronized; social jet lag (varying wake time by 2+ hours on weekends) desynchronizes these systems and produces the equivalent of chronic jet lag.

LIFESTYLE

Blue Light Elimination After Sunset (blue-light-blocking glasses)

Amber-lens glasses 2-3 hours before bed; all screens filtered

Melanopsin cells in the retina are maximally sensitive to short-wave (blue, 480nm) light; even brief evening blue light exposure delays melatonin onset by 90 minutes. Blue-light-blocking glasses with amber lenses (not clear 'blue light glasses') completely block melanopsin-activating wavelengths while preserving vision.

FURTHER TESTING

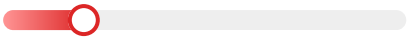
Wrist Actigraphy (7-14 nights)

Oura Ring, WHOOP, or medical-grade actigraph

Provides objective sleep data (total sleep time, efficiency, sleep architecture proxies, restless nights) that subjective reporting dramatically misestimates. A 2-week actigraphy baseline reveals patterns (sleep phase, consistency, efficiency) invisible to self-report and guides whether sleep timing, sleep pressure, or sleep quality is the primary problem.

MOOD-INDUCED SLEEPLESSNESS RISK

YOUR RESULTS



INCREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with increased mood-induced sleeplessness risk are more susceptible to sleep disruption from low mood or emotional fatigue. In more persistent cases, interventions such as psychotherapy, mindfulness, or behavioral sleep interventions may be beneficial.

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

Ashwagandha (evening dose)

300-600 mg KSM-66 at bedtime

Mood-induced sleeplessness reflects HPA axis dysregulation carrying emotional arousal into sleep onset. Ashwagandha's cortisol-reducing effect is particularly relevant at bedtime, it reduces the ACTH-driven cortisol reactivation that mood disruption triggers in the nighttime window and is directly documented in RCTs to improve sleep quality in anxious individuals.

SUPPLEMENT

5-HTP

50-150 mg (with carbidopa-free formulation) 30-60 min before bed

5-HTP directly provides the immediate serotonin and melatonin precursor bypassing the tryptophan-to-5HTP conversion step that is the rate-limiting reaction. Mood-induced sleeplessness is driven by emotional arousal maintaining serotonin insufficiency into the nighttime; 5-HTP addresses both the mood and sleep components simultaneously. Do not combine with SSRIs/SNRIs without physician supervision.

DIET

Sleep Ritual Tea (chamomile + passionflower + lemon balm + valerian)

Standardized herb tea blend 60-90 min before bed

Synergistic herbal GABA modulators that collectively address mood-induced arousal through multiple pathways. Chamomile (apigenin), passionflower (chrysin), lemon balm (GABA transaminase inhibition), and valerian (valerenic acid) each have distinct GABA or benzodiazepine receptor mechanisms, combined, they provide broader inhibitory tone without any single herb's side effects.

DIET

Avoid Alcohol as Sleep Aid (completely contraindicated for mood-induced sleeplessness)

Zero alcohol for sleep induction

Alcohol's sedative effect is mediated by GABA-A agonism; however, alcohol is rapidly metabolized overnight, producing a rebound sympathetic activation (glutamatergic rebound) that disrupts sleep architecture in the second half of the night. For mood-induced sleeplessness, this rebound worsens nighttime anxiety, increases cortisol, and fragments deep sleep, the opposite of therapeutic.

LIFESTYLE

Evening Mood Processing Ritual (journaling or NSDR before bed)

20-30 min, 60-90 min before intended sleep time

Mood-induced sleeplessness is specifically driven by unprocessed emotional material activating the amygdala during the sleep-wake transition. A structured evening processing ritual (writing without resolution-seeking, body scan, NSDR) allows cognitive and emotional material to be externalized before the sleep window, reducing the amygdala arousal that delays sleep onset.

LIFESTYLE

Reduce Screen Emotional Content After 7pm (no news, social media, conflict content)

Hard stop on emotionally activating content 2-3 hours before bed

Mood-induced sleeplessness is maintained by late-day emotional activation from media content. News, social media conflict, and emotionally charged video content activate the amygdala-HPA axis cycle precisely in the window when melatonin should be rising. Replacing screen content with neutral activities (music, reading, nature) reduces the emotional arousal load that feeds sleeplessness.

FURTHER TESTING

4-Point Salivary Cortisol with Evening Sample

Maps the cortisol pattern underlying mood-induced sleeplessness. Elevated late-day or midnight cortisol confirms HPA-driven mood arousal as the primary mechanism, guiding whether phosphatidylserine (cortisol reduction), ashwagandha (HPA normalization), or GABA support (direct inhibitory tone) should be prioritized.

FURTHER TESTING

Salivary Melatonin Timing (DLMO)

Measures the actual time melatonin secretion begins, the precise circadian timing signal for sleep onset. Delayed DLMO (after 10pm) indicates delayed circadian phase that mood disruption amplifies; early DLMO with sleeplessness indicates circadian timing is adequate and the disruption is emotional arousal, not phase delay.

5 Neuro-Nutrients

6 variants analyzed

MTHFR ACTIVITY

YOUR RESULTS

60-70% ENZYME ACTIVITY

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with 60-70% MTHFR enzyme activity have a moderate reduction in enzyme activity and may be at an increased risk for elevated homocysteine levels and associated health problems. However, the risk is generally not as high as for individuals with further reduced enzyme activity. Individuals with this enzyme activity level may still benefit from folate and other b vitamins to support overall health.

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

L-Methylfolate (5-MTHF) at Neurological Dosing

1-15 mg/day (start at 1 mg, titrate to symptom response)

In the neuro context, methylfolate is required for monoamine neurotransmitter synthesis, it is the methyl donor for the BH4 (tetrahydrobiopterin) regeneration that is rate-limiting for dopamine, serotonin, and norepinephrine production. Neurological dosing (up to 15 mg) is distinct from nutritional dosing (400-800 mcg); in depression, OCD, and cognitive impairment associated with MTHFR, clinical response requires therapeutic-range methylfolate.

SUPPLEMENT

Methylcobalamin B12 (neuro-specific pairing)

1,000-5,000 mcg/day sublingual

In the neuro context, the methylfolate-B12 pair supports myelin synthesis (B12 is essential for myelin basic protein methylation) and homocysteine-to-methionine conversion in neurons. Myelin integrity (white matter health) is directly dependent on this methylation cycle; neurological MTHFR impact is distinct from cardiovascular homocysteine impact.

DIET

Liver (chicken or beef) 1-2x/week

3 oz chicken liver = 536 mcg natural folate

Liver provides the most concentrated natural folate of any food, in polyglutamate form that is better retained in tissues than synthetic folic acid. In the neuro context, consistently meeting folate requirements through food builds tissue folate reserves that buffer against the episodic stress-driven depletion that worsens neurological MTHFR symptoms.

DIET

Eliminate All Synthetic Folic Acid Sources (cereals, bread, supplements with 'folic acid')

Read all supplement and food labels

Synthetic folic acid competes with methylfolate at the folate receptor but cannot be converted to active methylfolate by impaired MTHFR. In neurological contexts, unmetabolized folic acid may interfere with neurological methylfolate receptor availability, a specific mechanism for worsening cognitive and mood outcomes that is especially important in this neuro panel context.

LIFESTYLE

Reduce Nitrous Oxide Exposure (absolute priority for MTHFR + neuro risk)

Alert all dental and medical providers

Nitrous oxide irreversibly inactivates methylcobalamin, the methyl-accepting partner of methylfolate in the methylation cycle. In the context of MTHFR-impaired neuro function, even a single nitrous oxide exposure can trigger acute functional folate/B12 failure with neurological consequences. MTHFR status must be disclosed to all providers before any procedure involving nitrous oxide.

FURTHER TESTING

Plasma Homocysteine (neuro target <7 µmol/L)

Test every 6 months while optimizing

In the neuro context, the target homocysteine is <7 µmol/L, substantially below the commonly cited 'normal' of <15 µmol/L. The OPTIMA trial showed that homocysteine >9 µmol/L predicted accelerated brain atrophy; below 7 µmol/L is the level at which brain atrophy rate normalizes. This tighter target is neurologically motivated.

FURTHER TESTING

Folate Receptor Alpha Antibodies Test (FRAAT)

Tests for antibodies blocking the folate receptor that transports methylfolate across the blood-brain barrier. Positive FRAAT (Folate Receptor Alpha Antibodies Test) indicates cerebral folate deficiency despite adequate blood folate, requiring folinic acid (bypasses the receptor) rather than methylfolate. Clinically important when neuropsychiatric or cognitive symptoms persist despite high-dose methylfolate.

COQ10 SUPPLEMENTATION BENEFIT

YOUR RESULTS

TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Genetic results show a typical response to CoQ10 supplementation. This indicates an average likelihood of experiencing benefits from CoQ10 supplementation including cognitive and mental/emotional support. Maintaining a balanced diet that includes sources of CoQ10, such as fatty fish and organ meats, can support overall health.

VITAMIN B9 NEED

YOUR RESULTS

TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with typical folate (B9) genetics have average requirements for this nutrient. Consume folate-rich foods, leafy green vegetables, legumes, and fortified cereals, to meet daily needs.

CHOLINE NEED

YOUR RESULTS

TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with this result have a neutral genetic propensity for choline need. Since it is one of the most common deficiencies, it is important to consume sufficient amounts of choline through dietary sources such as eggs or liver as choline plays a critical role in various physiological processes such as brain function, nerve function, and metabolism. Additionally, supplementation with choline may be beneficial for individuals who have difficulty reaching sufficient choline levels.

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

Alpha-GPC

300-600 mg daily

Highly bioavailable choline that crosses the blood-brain barrier; increases acetylcholine for memory and learning.

SUPPLEMENT

Citicoline (CDP-Choline)

250-500 mg daily

Increases choline availability and enhances phosphatidylcholine production supporting brain health.

DIET

Choline-Rich Foods

Eggs (especially yolks), beef liver, salmon, and chicken are excellent sources; aim for 425-550 mg daily.

DIET

Cruciferous Vegetables

Broccoli, Brussels sprouts, and quinoa provide plant-based choline for those limiting animal products.

LIFESTYLE

Adequate Sleep

7-8 hours/night

Choline supports acetylcholine synthesis critical for memory consolidation during sleep.

LIFESTYLE

Brain Exercise

30+ min daily

Cognitive activity increases acetylcholine demand; adequate choline intake supports sustained mental performance.

FURTHER TESTING

Liver Function Tests

Choline deficiency presents as NAFLD; testing tracks liver health in deficient individuals.

This report uses a color-coded system to indicate the clinical significance of each genetic result and the level of recommended action. Each result is assigned one of three status levels based on your genetic variants.

No Action Required

Your genetic result in this area falls within a typical range. No specific intervention is indicated at this time. General wellness practices apply.

Practitioner Review / Action

Your genetic result suggests a moderate predisposition that warrants attention. Review with your practitioner and consider targeted lifestyle or supplementation support.

Practitioner Action

Your genetic result indicates a significant predisposition. Practitioner-guided intervention including targeted supplementation, dietary modification, and follow-up testing is recommended.

HOW TO READ YOUR RESULTS

- Each module page shows one or more gauges representing specific genetic results within that category.
- The gauge position and color indicate where your result falls relative to the general population.
- Personalized recommendations appear only for Moderate and Needs Attention results — Optimal results require no specific action.

DNA Signature - Mind & Mood Genetic Variants Summary

All 115 genotyped variants across this section, grouped by report panel.

- Homozygous Variant (+/+) — two copies of the variant
- Heterozygous (-/+) — one copy of the variant
- Homozygous Normal (-/-) — no copies of the variant

Mood & Behavior

34

ANKK1 G2137A rs1800497	GG	**FKBP5 C*1136A** rs3800373	AC
COMT G472A rs4680	AG	**SOD2 A47G** rs4880	AG
GAD1 C-64+894A rs12185692	AC	**ACE A-239T** rs4291	TT
GAD1 C146-3245T rs3828275	TC	**MTHFR A359+160G** rs17367504	AA
GAD1 G-151A rs3749034	AG	**ESR1 A453-351G** rs9340799	GG
HTR1A (C>G) rs6295	CC	**DIO2 C-143T** rs12885300	CC
HTR1B1 C861G rs6296	CG	**DIO2 T274C** rs225014	CC
HTR2A C-998T rs6311	TT	**CRHR1 C241+1631T** rs242939	TT
MGLL G263-1443A rs604300	GG	**BDNF C196T** rs6265	CC
MTHFD1L G643+208A rs11754661	AG	**ARNTL T671-305G** rs11022778	TT
OXTR G922+4581A rs53576	GG	**ABCC9 G3474-1222A** rs11046205	GG
PER3 C2566G rs228697	CC	**CRHR1 A122-1309C** rs242941	AC
SLC1A1 T1110C rs301430	TT	**TPH2 g.G4298T** rs4570625	GG
TCF7L2 C450+33966T rs7903146	TC	**CNR1 C1359T** rs1049353	CC
TNF G-238A rs361525	AG	**FAAH C385A** rs324420	CC
TNF G-308A rs1800629	GG	**ADORA2A T1083C** rs5751876	TC
MAO-A T891G rs6323	GG	**ADRA1A A883+323G** rs573514	AG

Cognitive Function

18

AGT A803G rs699	AA	**COMT G597A** rs769224	GG
COX-2 T1195C rs689466	TT	**COMT G472A** rs4680	AG
ESR1 A453-351G rs9340799	GG	**KL T1054G** rs9536314	TT
FBF1 T493C rs2305913	CC	**SNAP-25 G-63-21820A** rs363050	AG

ICAM1 A1405G rs5498	AG	**BDNF C196T** rs6265	CC
LDLR (A>G) rs11668477	AG	**CHRM2 T-46-5906A** rs324650	AT
PLAU T422C rs2227564	TC	**APOE C526T** rs7412	TC
PTGS2 C872G rs20417	CC	**APOE C388T** rs429358	TT
SLC25A27 rs10807344	CC	**ADA C22T** rs73598374	CC

Neurodegeneration Risk

46

MCCC1 G39-8882A rs11711441	GG	**APOE C388T** rs429358	TT
MIR469 (T>C) rs329648	TT	**APOE C526T** rs7412	TC
NSF G2106A rs199533	GG	**BCHE C-32T** rs1126680	CC
NUCKS1 g.C801T rs823118	TT	**BCKDK G615A** rs14235	AA
NUCKS1 g.G4841A rs823114	AA	**BDNF A-21-4385G** rs11030104	AA
NUCKS1 G17+5707A rs823128	AA	**BDNF C196T** rs6265	CC
RAB7L1 (A>G) rs947211	GG	**BDNF G-21-14109C** rs2049045	GG
RIT2 T103+17147C rs4130047	TC	**BST1 A*12+183G** rs4538475	AA
SIPA1L2 C-269-13257T rs10797576	CC	**BST1 C852-575A** rs11724635	AC
SLC2A13 G717-6250A rs1994090	TG	**CCDC62 A2002-4327G** rs11060180	GG
SLC50A1 G12+93A rs12726330	GG	**CRHR1 n.A1935G** rs393152	AA
SNCA (G>T) rs11931074	GG	**DGKQ C351+422** rs11248060	CC
SNCA A1290-15425G rs17577094	AA	**FAM47E C871-236T** rs6812193	TC
SNCA A1774-6612G rs8070723	AA	**FERMT2 T158-14555C** rs17125944	TT
SNCA C*1783T rs11012	CC	**GAK C3166+578T** rs11248051	CC
SNCA G307-28113A rs2736990	GG	**GBA (C>T)** rs34372695	CC
SORL1 T3050-2062C rs11218343	TT	**GBA C1093T** rs2230288	CC
SPPL2C A1411G rs12185268	AA	**GCH1 C344-16715T** rs11158026	CC
SREBF1 G*835A rs11868035	AG	**GPNMB A224-42G** rs199347	AG
TMEM175 (C>T) rs6532194	TC	**HLA-DQB1 (C>T)** rs9275326	CC
TMEM175 (T>C) rs356220	TC	**TC TMEM175 G-31-2384A** rs6599389	AG
LRRK2 C237+1366G rs1491942	GC	**TMEM175 T1178C** rs34311866	TC
MCCC1 G1083+764A rs12637471	GG	**VPS13C (G>A)** rs2414739	AG

Sleep

10

MEIS1 A889-10767G rs12469063	AG	**COMT G472A** rs4680	AG
PTPRD G103-113009A rs1975197	AG	**BTBD9** rs3923809	AA
BDNF C196T rs6265	CC	**GSK3B A-1001G** rs334558	AG

FABP7 C182T rs2279381	**CC**	**TPH2 A1125T** rs4290270	**TA**
CLOCK A793-1130G rs11932595	**AG**	**MEIS1 T965+6302G** rs2300478	**TG**

Neuro-Nutrients 7

NQO1 G559A rs1800566	**AG**	**CYP7A1 g.G2020T** rs3808607	**TT**
MTHFR G677A rs1801133	**AG**	**CD36 G-180+14244A** rs1761667	**GG**
MTHFR T1298G rs1801131	**TT**	**APOE C388T** rs429358	**TT**
BHMT G716A rs3733890	**AA**		

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