



DNA Signature Metabolic Path

GENETICS REPORT

1

Report

6

Report
Panels

207

Gene
Variants

Example Client

Prepared for

Order ID: _____

DOB: _____

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Result Scores Summary

Overview of all 26 engine results across this section.

5

NEEDS ATTENTION

11

MODERATE

10

OPTIMAL

● Needs Attention (5)

● Magnesium Deficiency Risk	Elevated risk	Nutrients
● Anxiety Propensity	Elevated risk	Mental Health
● Stress Adaptation	Below average	Mental Health
● Depression Propensity	Elevated risk	Mental Health
● Gut Permeability	Elevated risk	GI & Detox

● Moderate (11)

● Methylation Ability	Undermethylation potential	Methylation
● MTHFR Activity	48% enzyme activity	Methylation
● MTR	High activity	Methylation
● Phase II Detox Efficiency	Reduced	Phase II Detox Efficiency
● Glutathione Level Propensity	Reduced	Nutrients
● Vitamin D3 Deficiency Risk	Increased	Nutrients
● Vitamin B12 Level	Reduced	Nutrients
● Rumination	Increased	Mental Health
● GAD1 Activity	Decreased activity	Mental Health
● Mold Sensitivity	Increased	GI & Detox
● Oxidative Stress Support	Decreased	GI & Detox

● Optimal (10)

● MTRR	Increased activity	Methylation
● COMT	Typical activity	Methylation
● CoQ10 Supplementation Benefit	Typical	Nutrients
● Iron Deficiency Risk	Typical	Nutrients
● Vitamin C Need	Typical level	Nutrients
● Vitamin B9 Need	Typical	Nutrients
● Vitamin B6 Level Propensity	Typical	Nutrients
● Stress Phenotype	Mediator	Mental Health
● OCD Likelihood	Typical	Mental Health
● Mitochondrial Function	Typical	GI & Detox

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.

METHYLATION ABILITY

YOUR RESULTS



UNDERMETHYLATION POTENTIAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with undermethylation potential methylation propensity genetics have a potential for undermethylation. Their genetic profile suggests their body may have a tendency towards reduced methylation capacity, but it may not necessarily lead to undermethylation in all cases. It is still important for Individuals with undermethylation potential methylation propensity genetics to be mindful of factors that can influence methylation and take proactive steps to support optimal methylation through a well-balanced diet, adequate nutrient intake, and lifestyle practices that promote overall well-being. Foundational micronutrients which benefit from support include B vitamins, magnesium, choline and zinc.

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

Methylated B-Vitamin Complex (Methylfolate + Methylcobalamin + P5P)

400–800 mcg methylfolate + 500–1,000 mcg methylcobalamin + 25–50 mg P5P daily

The three active-form B vitamins that directly fuel the methylation cycle without requiring genetic conversion steps. The methylated forms bypass MTHFR, MTR, and MTRR enzyme bottlenecks identified in this panel.

SUPPLEMENT

TMG (Trimethylglycine / Betaine)

500–3,000 mg/day

Provides three methyl groups via the BHMT pathway, a methylation shortcut that bypasses the folate cycle entirely. Particularly valuable when multiple SNPs reduce folate-cycle throughput; TMG lowers homocysteine independently of B12 and folate.

DIET

Choline-Rich Foods (eggs, liver, beef)

2–3 whole eggs daily + liver 1–2x/week

Choline is the primary dietary methyl donor alongside folate. Each egg provides ~150 mg choline; liver provides ~400 mg per 3 oz. Meeting the AI of 425–550 mg/day through food significantly reduces methyl group demand on the folate cycle.

DIET

Betaine-Rich Foods (beets, quinoa, spinach, wheat germ)

Betaine provides methyl groups directly through the BHMT pathway and significantly lowers homocysteine. Regular consumption of betaine-rich foods acts as a buffer when the folate-cycle methylation pathway is genetically impaired.

LIFESTYLE

Minimize Xenobiotic Exposure (pesticides, plastics, solvents)

Environmental toxins consume methylation capacity for their detoxification via Phase II pathways. Reducing toxic load preserves methyl groups for neurological, cardiovascular, and immune methylation functions.

LIFESTYLE

Limit Alcohol (major methylation depleter)

Less than 1 drink/week ideally

Alcohol is the most potent single dietary disruptor of methylation; it depletes folate, B12, B6, and zinc simultaneously while upregulating SAH (the methylation inhibitor). Even moderate alcohol measurably impairs methylation capacity.

FURTHER TESTING

Plasma Homocysteine

The single most clinically useful marker of methylation status. Homocysteine accumulates when methylation cycle throughput is reduced. Target <7 umol/L for optimal cardiovascular and neurological health (vs the commonly cited <15 umol/L normal).

FURTHER TESTING

Methylmalonic Acid (urine or plasma)

Functional marker of B12 sufficiency at the cellular level. Elevated MMA indicates B12-dependent methylation is impaired even when serum B12 appears normal; more sensitive to functional deficiency than serum B12 alone.

MTHFR ACTIVITY

YOUR RESULTS



UNDERSTANDING WHAT THIS MAY MEAN

Individuals with 48% MTHFR enzyme activity may have a mild reduction in enzyme activity and may be at a somewhat 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. It is still important for individuals with this enzyme activity level to work with their medical provider to ensure adequate intake of folate and other B vitamins and to avoid certain medications that may further decrease MTHFR activity.

 SUPPLEMENT**Riboflavin (B2)***25–100 mg/day*

The most overlooked MTHFR cofactor. Clinical trials show that in C677T homozygotes, riboflavin supplementation alone reduces homocysteine by 22% and can restore meaningful MTHFR function. Essential for thermolabile MTHFR variants.

 SUPPLEMENT**5-MTHF (Methylfolate)***400–1,000 mcg/day; up to 5 mg/day under practitioner supervision*

Provides the active end-product of the MTHFR enzyme reaction, bypassing the conversion bottleneck entirely. Start low and increase gradually; some individuals with COMT variants experience irritability or anxiety if methylfolate is introduced too fast.

 DIET**Natural Food Folates (leafy greens, lentils, asparagus)***400+ mcg daily from food*

Natural folates from food are better tolerated than folic acid for MTHFR variants. Dark leafy greens, lentils, avocado, and asparagus provide polyglutamate folates that follow different absorption kinetics than synthetic folic acid.

 DIET**Avoid Folic Acid in Fortified Foods and Supplements**

Synthetic folic acid requires MTHFR to be converted to usable methylfolate. With reduced MTHFR activity, unmetabolized folic acid accumulates in plasma. Avoid cereals, breads, and supplements with folic acid listed as the folate source.

 LIFESTYLE**Avoid Nitrous Oxide (dental sedation)***Inform dentist of MTHFR status*

Nitrous oxide irreversibly inactivates methylcobalamin and can trigger acute functional folate/B12 deficiency in MTHFR variants. Highly recommend sharing MTHFR status to dental providers and requesting alternative sedation.

 LIFESTYLE**Limit Alcohol**

Alcohol specifically depletes folate and B12, the two nutrients most impaired by MTHFR variants. Even social drinking measurably worsens functional methylation status in MTHFR carriers.

 FURTHER TESTING**Homocysteine**

Primary clinical outcome of MTHFR activity. Elevated homocysteine confirms functional MTHFR impairment regardless of genotype; target <7 umol/L. Retesting 8–12 weeks after starting methylfolate confirms treatment response.

 FURTHER TESTING**Plasma Riboflavin (B2)**

B2 deficiency worsens thermolabile MTHFR variants; testing reveals whether riboflavin supplementation will meaningfully improve MTHFR function. Low B2 is the most modifiable cofactor for MTHFR impairment.

MTR

YOUR RESULTS



UNDERSTANDING WHAT THIS MAY MEAN

MTR encodes methionine synthase, an enzyme that converts homocysteine back to methionine using vitamin B12 as a cofactor. This reaction is central to the methylation cycle and plays a key role in maintaining healthy homocysteine levels and supporting DNA synthesis, neurotransmitter production, and overall methylation capacity. This result reflects multiple MTR variants associated with elevated enzyme activity, which typically corresponds with lower homocysteine levels, a favorable outcome for cardiovascular and metabolic health. However, some research has associated high MTR activity with a modest increase in certain cognitive and emotional health considerations, possibly related to shifts in methionine and SAMe availability. Supporting overall methylation balance through B12, folate, and a nutrient-dense diet is still recommended. Discussing this result with a healthcare provider can help contextualize any relevant risks.

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

Methylcobalamin (B12)

1,000–2,000 mcg/day sublingual or intramuscular

MTR uses methylcobalamin to transfer a methyl group from methylfolate to homocysteine, regenerating methionine. MTR variants reduce this transfer efficiency; higher methylcobalamin ensures adequate substrate despite reduced enzyme function.

SUPPLEMENT

Methylfolate (5-MTHF)

400–800 mcg/day

Provides the methyl group that MTR transfers to homocysteine. MTR and methylfolate are interdependent; supplementing B12 alone without methylfolate creates a substrate imbalance.

DIET

B12-Rich Animal Foods (meat, shellfish, eggs, dairy)

Daily sources

MTR function depends on dietary B12 supply. Shellfish (clams, oysters) have the highest B12 density of any food (~84 mcg per 3 oz clams).

DIET

Glycine-Rich Foods (bone broth, collagen, gelatin)

Glycine supports the folate cycle by providing one-carbon units via the glycine cleavage system; reduces methyl group demand when MTR throughput is reduced.

LIFESTYLE

Avoid Nitrous Oxide

Nitrous oxide irreversibly oxidizes the cobalt center of MTR, permanently inactivating the enzyme molecule. MTR variants already have reduced baseline activity; nitrous oxide can cause acute methylation failure. Inform all providers.

FURTHER TESTING

Homocysteine

MTR converts homocysteine to methionine; reduced MTR activity causes homocysteine accumulation. Homocysteine >7 umol/L in the presence of adequate B12 and folate suggests MTR functional impairment.

FURTHER TESTING

Methylmalonic Acid (MMA)

Elevated MMA specifically indicates B12 insufficiency at the cellular/mitochondrial level, independent of MTR genotype. If both homocysteine and MMA are elevated, functional B12 deficiency is driving the MTR impairment.

MTRR

YOUR RESULTS



INCREASED ACTIVITY

UNDERSTANDING WHAT THIS MAY MEAN

MTRR encodes methionine synthase reductase, an enzyme responsible for reactivating methionine synthase (MTR) when it becomes inactive. MTR requires vitamin B12 in its active form to function; MTRR recycles this cofactor so the methylation cycle can continue running. Reduced MTRR activity can impair MTR function even when B12 intake is sufficient, making micronutrient status particularly important in individuals with MTRR variants. This result reflects a homozygous variant associated with increased MTRR activity, providing greater-than-average support for MTR reactivation and methylation cycle function. This is generally a favorable finding, suggesting efficient recycling of the B12 cofactor needed to sustain healthy homocysteine metabolism. Maintaining a nutrient-dense diet with adequate B12 and folate continues to support optimal methylation outcomes.

COMT

YOUR RESULTS



UNDERSTANDING WHAT THIS MAY MEAN

Those with typical or average COMT activity exhibit average methylation and neurotransmitter breakdown, mirroring the general population. Maintaining a balanced diet rich in nutrients that support methylation, such as folate, vitamin B12, and betaine, is recommended. Regular physical and mental health check-ups can help ensure that methylation processes are functioning optimally

PHASE II DETOX EFFICIENCY

YOUR RESULTS



UNDERSTANDING WHAT THIS MAY MEAN

Individuals with this genetic result may have reduced levels of glutathione. Genetic variations associated with this result suggest that their bodies may have a decreased capacity to produce or maintain optimal glutathione levels. It is important for individuals with this result to prioritize lifestyle practices that support glutathione production and overall antioxidant defense. This may include consuming sulfur rich foods as well as considering glutathione precursors such as NAC under the guidance of a healthcare practitioner.

LITERATURE DERIVED RECOMMENDATION

 SUPPLEMENT**Liposomal Glutathione***500–1,000 mg/day*

Provides pre-formed glutathione with dramatically improved bioavailability over standard oral glutathione (which is largely degraded in the GI tract). S-acetyl glutathione is an alternative cell-permeable form.

 SUPPLEMENT**Milk Thistle (Silymarin)***200–400 mg/day (standardized to 70–80% silymarin)*

Upregulates glutathione synthesis in hepatocytes through direct transcriptional activation of glutamate-cysteine ligase, the rate-limiting enzyme in glutathione production. Protects existing glutathione from oxidative depletion.

 DIET**Sulfur-Rich Foods (garlic, onion, cruciferous, eggs)***Daily*

Provide cysteine, the rate-limiting amino acid in glutathione synthesis. Garlic's organosulfur compounds (allicin, diallyl sulfide) also directly induce glutathione S-transferase enzymes used in Phase II conjugation.

 DIET**Glycine-Rich Foods (bone broth, gelatin, collagen, skin-on poultry)***1–2 cups bone broth/day or collagen supplement*

Glycine is co-equally rate-limiting with cysteine in glutathione synthesis; most people consume far more cysteine than glycine. Glycine deficiency is an underappreciated bottleneck in glutathione production.

LIFESTYLE

Limit Acetaminophen (Tylenol)

Use minimum effective dose, space doses widely

Acetaminophen is metabolized via glutathione conjugation, each dose depletes hepatic glutathione. Chronic low-dose use can significantly impair glutathione reserves, particularly when SNPs already compromise synthesis.

FURTHER TESTING

Whole Blood Glutathione (reduced GSH + oxidized GSSG + ratio)

Measures the primary and rate-limiting antioxidant system. The GSH:GSSG ratio is more informative than total glutathione, a low ratio indicates oxidative stress is actively consuming glutathione faster than it is being regenerated.

FURTHER TESTING

Pyroglutamic Acid (OAT)

MosaicDX

Pyroglutamate elevated on OAT indicates high glutathione demand (the gamma-glutamyl cycle is running in overdrive). Pyroglutamate is the most sensitive early marker of glutathione depletion, often elevated before total glutathione falls.

GLUTATHIONE LEVEL PROPENSITY

YOUR RESULTS

REDUCED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with this genetic result may have reduced levels of glutathione. Genetic variations associated with this result suggest that their bodies may have a decreased capacity to produce or maintain optimal glutathione levels. It is important for individuals with this result to prioritize lifestyle practices that support glutathione production and overall antioxidant defense. This may include consuming sulfur rich foods as well as considering glutathione precursors such as NAC under the guidance of a healthcare practitioner.

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

GlyNAC (Glycine + N-Acetylcysteine)

0.1 g/kg each (glycine + NAC) per day

The most evidence-based approach to restoring glutathione. Clinical trials demonstrate that glycine and cysteine are co-equally rate-limiting; providing both simultaneously doubles glutathione restoration compared to either alone. A landmark RCT in older adults showed 100% glutathione increase.

SUPPLEMENT

Liposomal or S-Acetyl Glutathione

500–1,000 mg/day

Provides reduced glutathione with improved cellular delivery compared to standard oral forms (which are >80% degraded in the GI tract). S-acetyl glutathione has good cell membrane permeability; liposomal encapsulation also significantly improves bioavailability.

DIET

Sulfur-Rich Foods (garlic, onion, leeks, cruciferous vegetables)

Daily

Provide dietary cysteine and organosulfur compounds that both serve as glutathione precursors and independently upregulate glutathione synthesis enzymes. Garlic's allicin increases glutathione-S-transferase activity by 30–40%.

LIFESTYLE

Minimize Acetaminophen and Alcohol (primary glutathione depletors)

Use minimum effective doses

Both alcohol and acetaminophen are metabolized through glutathione conjugation, directly depleting the hepatic glutathione pool. Combined use is particularly depleting and raises acetaminophen hepatotoxicity risk substantially.

FURTHER TESTING

Whole Blood Glutathione (GSH + GSSG + ratio)

Whole blood (not serum) measurement captures both intracellular and plasma glutathione. The GSH:GSSG ratio below 10:1 indicates oxidative stress is outpacing glutathione regeneration; guides whether synthesis support (NAC, glycine) or reduction of glutathione consumption is the primary issue.

FURTHER TESTING

Pyroglutamic Acid (OAT)

MosaicDX

The most sensitive early OAT marker of glutathione demand exceeding synthesis capacity. Pyroglutamate is elevated when the gamma-glutamyl cycle is running in overdrive to recycle glutathione components, often elevated before whole blood glutathione falls below the reference range.

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.

MAGNESIUM DEFICIENCY RISK

YOUR RESULTS



ELEVATED RISK

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with elevated Risk magnesium deficiency risk genetics may have a elevated risk of experiencing magnesium deficiency. It is crucial for these individuals to consume a balanced diet that includes magnesium-rich foods and/or consider magnesium supplementation to prevent deficiency and associated health risks. Consider evaluating medications that may interfere with magnesium absorption such as proton pump inhibitors with your health care provider.

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

Magnesium Glycinate

300–400 mg elemental magnesium/day

The best-tolerated form for long-term supplementation with minimal laxative effect. Glycinate chelation improves absorption and bioavailability over oxide, sulfate, or citrate. Evening dosing supports sleep quality as a secondary benefit.

SUPPLEMENT

Magnesium L-Threonate (for cognitive applications)

1.5–2 g/day (providing ~144 mg elemental Mg)

The only form shown to cross the blood-brain barrier and increase brain magnesium levels. If magnesium deficiency is manifesting as cognitive symptoms, anxiety, or sleep disruption, threonate addresses the neurological compartment that glycinate cannot reach.

DIET

Pumpkin Seeds, Dark Chocolate, Almonds, Spinach, Avocado

Daily rotation

Pumpkin seeds (156 mg per oz) and dark chocolate (64 mg per oz) are the most magnesium-dense convenient foods. A daily handful of pumpkin seeds plus 1 oz dark chocolate provides ~220 mg.

LIFESTYLE

Reduce Factors That Deplete Magnesium (alcohol, caffeine, stress, diuretics)

Alcohol and caffeine both increase renal magnesium excretion significantly. Chronic stress activates the HPA axis which increases magnesium demand. Thiazide and loop diuretics cause substantial magnesium wasting, patients on these require active monitoring and supplementation.

FURTHER TESTING

RBC Magnesium (not serum)

Serum magnesium is maintained in a narrow range at the expense of intracellular stores. Serum can be normal when cells are profoundly depleted. RBC magnesium reflects intracellular status and is the appropriate test for deficiency assessment. Target RBC magnesium >5.5 mg/dL.

FURTHER TESTING

24-Hour Urine Magnesium (if deficiency mechanism unclear)

Distinguishes between inadequate intake (low urine Mg) and excessive renal wasting (elevated urine Mg despite deficiency). High urine magnesium with low RBC magnesium confirms renal wasting as the primary driver.

IRON DEFICIENCY RISK

YOUR RESULTS



TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

This standard risk level suggests that dietary intake and lifestyle choices play a crucial role in supporting iron levels and hair health. To manage this typical risk, individuals should ensure their diet includes iron-rich foods, consider pairing iron sources with vitamin C-rich foods to enhance absorption, and avoid excessive consumption of inhibitors like calcium and caffeine during meals.

VITAMIN D3 DEFICIENCY RISK

YOUR RESULTS



UNDERSTANDING WHAT THIS MAY MEAN

Individuals with increased vitamin D deficiency risk genetics are more likely to develop deficiency even with adequate sun exposure and dietary intake. Monitor vitamin D levels closely and consider vitamin D3 supplementation under healthcare guidance.

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

Vitamin D3 + K2 (MK-7)

D3 2,000–5,000 IU + K2 100–200 mcg/day

D3 is the form made by skin from sunlight and is significantly more effective at raising 25-OH vitamin D than D2. K2 (MK-7 form) ensures calcium mobilized by D3 is directed to bone, not arteries.

SUPPLEMENT

Magnesium (glycinate or malate)

300–400 mg/day

Vitamin D conversion requires magnesium at both the 25-hydroxylation (liver) and 1-alpha-hydroxylation (kidney) steps. Supplementing D3 without magnesium can paradoxically worsen magnesium deficiency and impair D3 activation.

DIET

Fatty Fish 3x/Week, Egg Yolks, UV-Exposed Mushrooms

Dietary vitamin D3 from fatty fish (600–1,000 IU per serving wild salmon) supplements sun exposure meaningfully. Egg yolks provide D3 plus vitamin A (needed for vitamin D receptor expression).

LIFESTYLE

Midday Sun Exposure

10–30 min, arms/legs uncovered, 10am–2pm

The most efficient D3 production occurs when solar angle is optimal. 20 min of midday sun on arms and legs produces approximately 10,000 IU equivalent.

FURTHER TESTING

25-OH Vitamin D (target 60–80 ng/mL)

Test every 6 months when supplementing

The clinical target for optimal immune, endocrine, and mental health function is 60–80 ng/mL, substantially above the sufficient threshold of 30 ng/mL. Re-test 3 months after changing dose.

FURTHER TESTING

Parathyroid Hormone (PTH)

PTH inversely tracks vitamin D status; PTH above 40 pg/mL indicates functional vitamin D deficiency even when 25-OH D appears borderline adequate. PTH normalization (<35 pg/mL) is the functional endpoint of vitamin D repletion.

VITAMIN C NEED

YOUR RESULTS



UNDERSTANDING WHAT THIS MAY MEAN

Individuals with a typical vitamin C propensity exhibit average abilities to utilize and maintain vitamin C levels, aligning with the general population's capacity. This means their dietary needs for vitamin C, which supports immune health, collagen synthesis, and antioxidant activity, can typically be met through a balanced diet that includes a variety of fruits and vegetables rich in vitamin C. Regular consumption of these foods is recommended to support general well-being and health maintenance.

VITAMIN B9 NEED

YOUR RESULTS



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.

VITAMIN B6 LEVEL PROPENSITY

YOUR RESULTS



UNDERSTANDING WHAT THIS MAY MEAN

Individuals with Typical B6 genetics are likely to maintain sufficient vitamin B6 levels with a balanced diet. B6 supports mood regulation and sleep quality.

VITAMIN B12 LEVEL

YOUR RESULTS



UNDERSTANDING WHAT THIS MAY MEAN

Individuals with Reduced B12 genetics typically have a lower genetic propensity for maintaining sufficient vitamin B12 levels. Low B12 can cause fatigue, weakness, and anemia. Prioritize B12-rich foods, fish, meat, and dairy, and consider B12 supplementation as appropriate.

 SUPPLEMENT

Methylcobalamin or Hydroxocobalamin

1,000–2,000 mcg/day sublingual

Active B12 forms that bypass the intrinsic factor and transcobalamin transport system when taken sublingually. Methylcobalamin directly supports MTR and methylation; hydroxocobalamin is preferred for those with COMT variants.

 SUPPLEMENT

Adenosylcobalamin (for mitochondrial B12 function)

500–1,000 mcg/day

The mitochondrial form of B12 used in the methylmalonyl-CoA mutase reaction. Those with elevated methylmalonic acid on OAT specifically need adenosylcobalamin, not just methylcobalamin.

 DIET

Animal Protein Daily (shellfish, meat, eggs, dairy)

3 oz shellfish or 2 eggs or 1 serving meat

B12 is found exclusively in animal products; vegans and vegetarians are at high risk for functional deficiency. Clams are the highest food source (~84 mcg/3 oz); beef liver provides ~70 mcg/3 oz.

 LIFESTYLE

Review Medications That Deplete B12 (metformin, PPIs, oral contraceptives)

Metformin reduces ileal B12 absorption by up to 30%; PPIs reduce gastric acid needed for B12 release from food; oral contraceptives deplete multiple B vitamins. Anyone on these medications needs active B12 monitoring and supplementation.

 LIFESTYLE

Optimize Stomach Acid for B12 Absorption

B12 requires pepsin (activated by stomach acid) to be released from food proteins. Age-related hypochlorhydria and PPI use are the most common causes of B12 deficiency in people eating adequate animal protein.

 FURTHER TESTING

Methylmalonic Acid (urine or plasma)

Gold standard functional B12 marker. MMA rises before serum B12 falls and before neurological symptoms appear; identifies functional deficiency even when serum B12 is normal. Target MMA <0.27 umol/L.

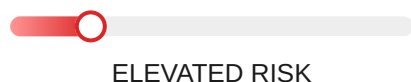
 FURTHER TESTING

Active B12 (Holotranscobalamin, holoTC)

Measures only the fraction of B12 in transit and available for cellular use. The earliest and most sensitive marker of B12 depletion; falls before total serum B12 declines.

ANXIETY PROPENSITY

YOUR RESULTS



UNDERSTANDING WHAT THIS MAY MEAN

Individuals with elevated Risk Depression propensity genetics carry genetic markers that significantly amplify the likelihood of experiencing depression, underscoring the importance of proactive and sustained mental health management. Early intervention strategies, including therapy, consistent engagement with mental health professionals, and potentially medication, are critical components of a personalized care plan. Build a strong support network and discuss a long-term mental health strategy with a qualified provider.

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

Passionflower Extract (*Passiflora incarnata*)

250–500 mg/day

MAO-inhibiting and GABA-A modulating flavonoids. A double-blind RCT showed passionflower was equivalent to oxazepam (a benzodiazepine) for generalized anxiety, with significantly less cognitive impairment and next-day sedation.

SUPPLEMENT

Inositol (Myo-inositol)

12–18 g/day in divided doses

At therapeutic doses, myo-inositol is the most underused anxiety supplement with RCT evidence. It desensitizes serotonin 5-HT_{2A} receptors and modulates GABA-A signaling, comparable to SSRIs in some panic disorder trials and better tolerated. Works over 4–6 weeks.

DIET

Omega-3 Rich Diet (EPA 2–3 g/day specifically)

Fatty fish 3x/week or EPA-dominant fish oil

EPA (not DHA) is the omega-3 specifically associated with anxiety reduction in clinical trials. EPA reduces neuroinflammation that drives HPA hyper-reactivity; pure EPA or EPA-dominant (>60% EPA) fish oil is more effective for anxiety than balanced EPA/DHA.

DIET

Fermented Foods Daily (kefir, yogurt, kimchi, sauerkraut)

1–2 servings/day

The gut-brain axis directly modulates anxiety through the vagus nerve and GABA production by gut bacteria. *Lactobacillus rhamnosus* specifically produces GABA and reduces anxiety-like behavior. Kefir contains the highest density of GABA-producing bacteria.

LIFESTYLE

Diaphragmatic Breathing (physiological sigh)

2 long inhales through nose + extended exhale, 5 min daily

The physiological sigh (double inhale through the nose followed by an extended oral exhale) is the fastest way to reduce physiological anxiety; it maximally deflates the alveoli and triggers the parasympathetic brake. More effective than single-breath box breathing for acute anxiety reduction.

LIFESTYLE

Cold Exposure (cold shower, 2 min)

3–5x/week, morning

Regular cold exposure builds stress resilience by habituating the sympathoadrenal response. Cold-acclimated individuals show blunted cortisol and lower baseline anxiety.

FURTHER TESTING

4-Point Salivary Cortisol

MosaicDX

Maps the HPA axis pattern underlying anxiety; elevated evening cortisol and blunted morning peak are the most common patterns. Guides whether the anxiety is driven by HPA hyperactivation (needs adaptogens) or circadian dysregulation (needs light therapy and sleep timing).

FURTHER TESTING

Plasma or Urine GABA and Glutamate

The glutamate:GABA ratio is the neurochemical basis of anxiety propensity. Elevated glutamate or reduced GABA on urine or plasma amino acid panel guides whether GABA-supportive supplements (theanine, inositol, magnesium) or glutamate-reducing dietary changes are the primary target.

STRESS PHENOTYPE

YOUR RESULTS

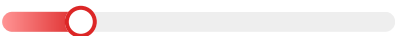


UNDERSTANDING WHAT THIS MAY MEAN

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.

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

Ashwagandha

300–600 mg/day

The most clinically validated adaptogen for HPA axis normalization. Reduces cortisol 15–30% in RCTs and improves stress resilience. In the OAT context, chronic stress drives methylation depletion, glutathione consumption, and mitochondrial dysfunction simultaneously.

SUPPLEMENT

Phosphatidylserine

300–400 mg/day

Directly blunts the ACTH-cortisol response to psychological and exercise stress. In the OAT context, cortisol-driven methylation demand is a key interaction; phosphatidylserine reduces the systemic methylation tax that chronic stress imposes.

DIET

Magnesium-Rich Foods Daily (pumpkin seeds, dark chocolate, spinach)

Chronic stress rapidly depletes magnesium, which in turn amplifies NMDA excitotoxicity, impairs GAD1 (GABA synthesis), and worsens COMT function. Food magnesium replenishment acts at multiple OAT pathway intersections simultaneously.

LIFESTYLE

HRV Biofeedback Training

20 min/day with device (HeartMath, Elite HRV)

Trains vagal tone and improves HRV by 30–50% over 8–12 weeks. Device-guided biofeedback provides real-time feedback unavailable in traditional mindfulness and accelerates autonomic nervous system retraining.

FURTHER TESTING

4-Point Salivary Cortisol + DHEA-S

MosaicDX

Maps the full HPA pattern and adrenal reserve. The DHEA-S:cortisol ratio is the most informative single marker of stress adaptation capacity. A low ratio indicates adrenal depletion requiring more aggressive support than high-cortisol-alone patterns.

RUMINATION

YOUR RESULTS



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 (Safranal + Crocin)

28–30 mg/day

Two RCTs show saffron significantly reduces rumination scores independently of its antidepressant effects. Crocin and safranal modulate serotonin reuptake and NMDA receptor activity, the two pathways most implicated in ruminative cognition.

SUPPLEMENT

EPA-Dominant Omega-3

2–3 g EPA/day

EPA specifically reduces the neuroinflammation and default mode network (DMN) hyperactivity associated with ruminative thinking. The DMN is the brain network most overactive in rumination; EPA's anti-inflammatory effects dampen its excessive engagement.

DIET

Mediterranean Diet (anti-inflammatory, DMN-stabilizing)

The dietary pattern most associated with reduced ruminative cognition through inflammatory and microbiome-mediated gut-brain mechanisms. High olive oil, fish, and polyphenol intake reduces the pro-inflammatory milieu that sustains DMN hyperactivity.

LIFESTYLE

Mindfulness-Based Cognitive Therapy (MBCT)

8-week structured program, 2 hrs/week

The most evidence-based intervention for rumination specifically. MBCT was designed for rumination prevention in depression and has the strongest evidence base; teaches metacognitive awareness of thought patterns without engagement, directly reducing ruminative loops.

LIFESTYLE

Behavioral Activation (scheduled absorbing activity)

30+ min daily of an absorbing task

Cognitive displacement through absorbing activities (music, craft, exercise requiring coordination) interrupts rumination by engaging working memory. Schedule specific activities rather than relying on motivation.

FURTHER TESTING

4-Point Salivary Cortisol

MosaicDX

Chronic HPA dysregulation is a primary neurobiological driver of rumination; the stress system maintains the memory consolidation that feeds ruminative loops. Evening cortisol elevation predicts next-day rumination severity.

FURTHER TESTING

hsCRP

Neuroinflammation sustains the default mode network hyperactivity underlying rumination. Elevated CRP confirms an inflammatory subtype where EPA omega-3, curcumin, and anti-inflammatory diet should be the primary interventions.

OCD LIKELIHOOD

YOUR RESULTS



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



DECREASED ACTIVITY

UNDERSTANDING WHAT THIS MAY MEAN

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

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

P5P (Pyridoxal-5-Phosphate)

25–50 mg/day

P5P is the essential cofactor for GAD1 (glutamic acid decarboxylase), the enzyme that converts glutamate to GABA. Low P5P is the most modifiable bottleneck in GAD1 function; replenishing P5P can restore meaningful enzyme activity independent of genetic variant severity.

SUPPLEMENT

Taurine

500–2,000 mg/day

Taurine acts as a GABA-mimetic by binding glycine and GABA receptors. Provides inhibitory neurotransmission compensation when GAD1-driven GABA synthesis is genetically reduced; particularly useful at night for sleep quality when GABA-driven inhibitory tone is critical.

DIET

Reduce Glutamate-Rich Foods (MSG, aged cheese, soy sauce, mushroom extract, nutritional yeast)

Eliminate or significantly limit

Free glutamate in food directly loads the same pool that GAD1 is tasked with converting. Low GAD1 activity means this dietary glutamate accumulates rather than being converted to GABA, worsening the excitatory/inhibitory imbalance.

DIET

Fermented Foods in Moderation (contain GABA directly) — kimchi, kefir, yogurt

Kimchi, kefir, and yogurt — 1 serving/day

Lacto-fermented foods contain GABA produced by *Lactobacillus* during fermentation. Provides direct GABA supplementation through diet. Avoid high-glutamate fermented foods (aged cheese, miso, soy sauce); stick to kimchi, kefir, and plain yogurt.

LIFESTYLE

Mindfulness Meditation (GABA-upregulating effect)

20+ min daily

MRI spectroscopy studies show that a single 60-minute yoga/meditation session increases GABA levels by 27%. Regular practice upregulates GABA synthesis capacity, a neuroplastic compensation for genetic GAD1 impairment.

FURTHER TESTING

Organic Acids Test (OAT) — GABA, Glutamate, and Quinolate Markers

MosaicDX

OAT measures GABA metabolites (beta-alanine pathway) and kynurenic acid vs. quinolinic acid balance, reflecting the downstream output of GAD1 activity and glutamate disposition. Elevated quinolinate with low GABA markers confirms GAD1-driven excitatory/inhibitory imbalance.

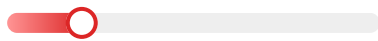
FURTHER TESTING

Plasma Amino Acids (glutamine, glutamate, GABA)

Elevated plasma glutamate with low GABA confirms functional GAD1 insufficiency at the metabolite level. Guides whether dietary glutamate restriction, GAD1 cofactor support (P5P), or GABA-supportive supplements should be prioritized.

DEPRESSION PROPENSITY

YOUR RESULTS



ELEVATED RISK

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with elevated Risk depression propensity genetics carry genetic markers that significantly amplify the likelihood of experiencing depression, underscoring the importance of proactive and sustained mental health management. Early intervention strategies, including therapy, consistent engagement with mental health professionals, and potentially medication, are critical components of a personalized care plan. Build a strong support network and discuss a long-term mental health strategy with a qualified provider.

 SUPPLEMENT**EPA-Dominant Omega-3 (>60% EPA)***2–4 g EPA/day*

EPA is the most evidence-based supplement for depression. A meta-analysis of 26 RCTs shows consistent antidepressant effects at 2g+ EPA/day. EPA reduces neuroinflammation via resolvin and protectin pathways; the anti-inflammatory mechanism is distinct from neurotransmitter modulation.

 SUPPLEMENT**SAMe (S-Adenosylmethionine)***400–800 mg/day on empty stomach*

Methyl donor that directly elevates brain monoamine levels (serotonin, dopamine, norepinephrine) and supports phospholipid methylation. Multiple RCTs show antidepressant effects comparable to tricyclic antidepressants. Works synergistically with SSRIs; start low as SAMe can cause irritability in slow COMT individuals.

 DIET**Mediterranean Diet Pattern**

The strongest dietary evidence base for depression prevention and treatment. The SMILES trial (RCT) showed a Mediterranean-style diet reduced depression scores comparably to psychotherapy. Emphasis on olive oil, fish, legumes, and vegetables addresses inflammatory, gut-brain, and nutrient pathways simultaneously.

 DIET**Tryptophan-Rich Protein Sources***Turkey, eggs, pumpkin seeds, salmon daily*

Tryptophan is the sole precursor to serotonin. Consuming tryptophan-rich foods with moderate carbohydrates maximizes brain tryptophan uptake, as carbohydrates reduce competing large neutral amino acids via insulin.

 LIFESTYLE**Aerobic Exercise***30–45 min, 3–5x/week (target 150+ min/week)*

Multiple meta-analyses show effect sizes comparable to antidepressant medication. Exercise increases BDNF, serotonin, norepinephrine, and dopamine, and reduces inflammatory cytokines that drive neuroinflammatory depression subtypes.

 LIFESTYLE**Bright Light Therapy***10,000 lux for 30 min within 1 hour of waking*

Clinical guidelines recommend bright light therapy not only for seasonal depression but year-round for non-seasonal depression, where effect sizes match antidepressants. Timing is critical, within 1 hour of waking to suppress excess melatonin and anchor the circadian cortisol awakening response.

FURTHER TESTING

hsCRP (High-Sensitivity CRP)

30–40% of people with depression have elevated inflammatory markers (hsCRP >1 mg/L); this neuroinflammatory subtype responds poorly to SSRIs but well to anti-inflammatory interventions (EPA omega-3, curcumin, exercise). Testing hsCRP identifies which treatment strategy is appropriate.

FURTHER TESTING

Thyroid Panel (TSH, Free T3, Free T4)

Hypothyroidism is among the most commonly missed causes of depression, particularly in women. Subclinical hypothyroidism (TSH 2.5–10 with symptoms) can cause treatment-resistant depression; Free T3 is the most functionally relevant thyroid marker for mood.

MOLD SENSITIVITY

YOUR RESULTS



UNDERSTANDING WHAT THIS MAY MEAN

Individuals with this genetic result have an increased severity risk of mold-related asthma and allergies. Genetic variations associated with this result suggest that their bodies may have an enhanced response to mold exposure, potentially leading to more severe asthma symptoms or allergic reactions. It is important for individuals with this result to be extra cautious, take proactive steps to minimize mold exposure, and work closely with healthcare professionals to develop a comprehensive management plan.

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

Activated Charcoal (or Cholestyramine if Rx available)

1–2 g activated charcoal away from meals and medications

Mycotoxin binder in the intestinal lumen. Activated charcoal adsorbs a broad range of mycotoxins before systemic absorption. Take at least 2 hours away from medications and meals to avoid binding nutrients or drugs. Cholestyramine (Rx) is more effective for ochratoxin and aflatoxin specifically.

SUPPLEMENT

Liposomal Glutathione + NAC

500 mg glutathione + 600 mg NAC/day

Mycotoxins are detoxified via glutathione conjugation; mold-sensitive individuals with impaired Phase II detox rapidly deplete glutathione when exposed. This combination directly replenishes the primary mycotoxin detox reservoir.

DIET

Low-Mycotoxin Diet (eliminate corn, peanuts, dried fruits, coffee from non-tested sources)

Eliminate highest-risk foods

Corn, peanuts, and dried fruits have the highest mycotoxin contamination rates of common foods, frequently testing positive for aflatoxin and ochratoxin. Switching to low-risk alternatives dramatically reduces ongoing mycotoxin intake.

DIET

Cruciferous Vegetables + Garlic Daily

Broccoli, Brussels sprouts, garlic, onion

NRF2-activating foods upregulate Phase II detox pathways (glutathione S-transferase, glucuronidation) that clear mycotoxins. This is the dietary backbone of mold sensitivity management when environmental control is underway.

LIFESTYLE

Home/Workplace ERMI Testing

Environmental Relative Moldiness Index test

The single most important intervention for mold-sensitive individuals is identifying and remediating active mold sources. ERMI dust sampling is the most sensitive method for identifying hidden mold in buildings. Without environmental remediation, supplementation is inadequate.

LIFESTYLE

HEPA Air Filtration (IQ Air, Austin Air, or equivalent)

Run 24/7 in bedroom and primary living spaces

High-efficiency HEPA filters remove airborne mold spores and mycotoxin-carrying particles. Bedroom filtration during sleep (8 hours/night) provides the most meaningful reduction in daily mycotoxin inhalation exposure.

FURTHER TESTING

OAT (Organic Acids) — Fungal Markers (arabinose, citramalic)

Elevated arabinose and citramalic acid on OAT indicate active fungal/yeast overgrowth, a common secondary consequence of mold exposure that perpetuates detox demand. Guides whether antifungal interventions are needed alongside mycotoxin binders.

FURTHER TESTING

Urine Mycotoxins

MosaicDX

Identifies specific mycotoxin species (ochratoxin A, trichothecenes, aflatoxins, gliotoxin) currently being excreted, confirming active exposure and guiding targeted binder selection. Not all binders work for all mycotoxins; species identification matters.

MITOCHONDRIAL FUNCTION

YOUR RESULTS



TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Those with typical mitochondrial function exhibit genetic markers that reflect the average population's mitochondrial efficiency in energy production. This indicates a standard level of mitochondrial function, where lifestyle and environmental factors can still play a significant role in optimizing mitochondrial health. Maintaining a balanced lifestyle with regular exercise, a diet rich in fruits, vegetables, and omega-3 fatty acids.

OXIDATIVE STRESS SUPPORT

YOUR RESULTS



DECREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with decreased genetics show a slightly higher than average vulnerability to oxidative stress. Increasing the intake of antioxidant-rich foods and considering supplements may be beneficial.

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

Sulforaphane (Broccoli Sprout Extract)

10–30 mg/day

Primary NRF2 activator. Upregulates the body's entire antioxidant enzyme network (SOD, catalase, glutathione peroxidase, glutathione-S-transferase). In the GI context, NRF2 activation also protects intestinal epithelial cells from oxidative damage driving permeability.

SUPPLEMENT

Liposomal Glutathione

500–1,000 mg/day

In the GI and detox context, glutathione is the primary conjugation substrate for toxins, heavy metals, and reactive oxygen species generated in the gut. Liposomal delivery is required for meaningful systemic and intestinal glutathione restoration.

DIET

30+ Diverse Plant Types Per Week (for antioxidant enzyme induction)

Polyphenol diversity is the strongest dietary predictor of systemic antioxidant enzyme activity. Different polyphenol classes activate different NRF2 target genes; diversity of plant intake produces complementary antioxidant enzyme induction that no single supplement can replicate.

LIFESTYLE

Moderate Exercise (not overtraining)

30–45 min, 4–5x/week

Moderate aerobic exercise upregulates SOD, catalase, and glutathione peroxidase through adaptive NRF2 signaling. Overtraining generates oxidative stress markers visible on OAT, more is not better.

FURTHER TESTING

Urinary Oxidative Stress Markers (8-OHdG, hydroxymethylglutarate, methylsuccinate)

These provide direct metabolite evidence of mitochondrial and systemic oxidative stress, more specific and nuanced than serum antioxidant panels. Elevated hydroxymethylglutarate indicates HMG-CoA reductase-driven oxidative stress; methylsuccinate indicates Complex II dysfunction.

FURTHER TESTING

Whole Blood Glutathione (GSH:GSSG ratio)

Measures the real-time antioxidant reserve in the context of ongoing oxidative challenge from gut-derived toxins and dietary antigens. Low ratio confirms active glutathione consumption outpacing synthesis, the primary clinical target in GI-driven oxidative stress.

GUT PERMEABILITY

YOUR RESULTS



ELEVATED RISK

UNDERSTANDING WHAT THIS MAY MEAN

Your genetic profile suggests an elevated susceptibility to increased intestinal permeability, sometimes referred to as "leaky gut." Variants in genes involved in tight junction regulation, mucosal barrier integrity, and inflammatory signaling may make your gut lining more vulnerable to disruption from dietary triggers, stress, dysbiosis, or toxin exposure. When the gut barrier is compromised, partially digested food particles, microbial byproducts, and environmental toxins can enter the bloodstream, activating immune responses that contribute to systemic inflammation, food sensitivities, and downstream effects on mood, metabolism, and detoxification capacity. Targeted nutritional support, stress management, and identifying personal food triggers are especially important given this genetic predisposition.

LITERATURE DERIVED RECOMMENDATION

SUPPLEMENT

Zinc-Carnosine

75 mg/day (37.5 mg twice daily)

The most targeted supplement for tight junction stabilization. Zinc-carnosine adheres to the intestinal mucosa, providing sustained local zinc release that upregulates occludin and claudin tight junction proteins. Multiple RCTs confirm intestinal permeability reduction.

SUPPLEMENT

Colostrum (bovine)

500 mg–2 g/day

Contains secretory IgA, lactoferrin, and growth factors (IGF-1, TGF-beta) that repair intestinal tight junctions and restore mucosal immune function. A marathon runner RCT showed colostrum prevented exercise-induced gut permeability better than glutamine alone.

DIET

Bone Broth (glycine and gelatin-rich)

1–2 cups/day

Provides glycine, glutamine, and collagen peptides that directly fuel enterocyte repair and support tight junction protein synthesis. Glycine is the most abundant amino acid in the intestinal extracellular matrix.

DIET

Elimination of Identified Food Triggers (gluten, dairy, eggs if reactive)

4–6 week elimination, then reintroduce

Intestinal permeability is both caused by and worsened by food antigens entering the bloodstream. Identifying and eliminating personal triggers through an elimination protocol removes the ongoing antigenic stimulus driving tight junction disruption.

LIFESTYLE

Stress Reduction (HPA axis drives intestinal permeability directly)

Cortisol directly increases intestinal permeability through CRH receptor activation in the gut wall. This is bidirectional: stress increases permeability, and increased permeability drives systemic inflammation that worsens stress reactivity.

FURTHER TESTING

GI360 (Stool Test)

MosaicDX

Identifies pathogens, parasites, bacterial dysbiosis, and inflammatory markers that are primary causes of intestinal permeability. Calprotectin and secretory IgA quantify intestinal inflammation and immune status, the two most clinically actionable permeability drivers.

FURTHER TESTING

Lactulose/Mannitol Urine Test

2-hour and 6-hour collection post-ingestion

Gold standard for intestinal permeability assessment. Elevated lactulose:mannitol ratio confirms paracellular (tight junction) leakage; the 6-hour collection window captures both small intestinal and colonic permeability.

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 - Metabolic Path

Genetic Variants Summary

All 201 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

Methylation

21

BHMT C477+52T rs567754	CC ●	MTHFR G677A rs1801133	AG ●
AHCY C112T rs819147	TC ●	COMT G472A rs4680	AG ●
AHCY G973-179A rs819134	AG ●	MTR A2756G rs1805087	AA ●
AHCY C*7+849T rs819171	TC ●	MTR G2775+157T rs2275565	GG ●
MTRR C1783T rs10380	TC ●	MTRR A66G rs1801394	AA ●
MTRR C1243T rs2287780	CC ●	BHMT G716A rs3733890	AA ●
MTRR G1911A rs1802059	GG ●	CBS T317-917C rs2851391	TC ●
BHMT (A>C) rs617219	AA ●	CBS G*565A rs706209	AG ●
CBS G699A rs234706	AG ●	MTHFD1 G1958A rs2236225	GG ●
CBS G1080A rs1801181	AG ●	BHMT C33+1319T rs651852	CC ●
CBS G636A rs2298758	GG ●		

Phase II Detox Efficiency

39

NRF2 G45+2714C rs2364723	GG ●	CYP2C9 C430T rs1799853	CC ●
NRF2 C45+11108T rs1806649	TC ●	CYP2C19 G681A rs4244285	AG ●
NRF2 A45+10270G rs13001694	GG ●	CYP1A1 C-27+606A rs2606345	AC ●
NRF2 A46-4230G rs6726395	AA ●	COMT C186T rs4633	TC ●
UGT1A9 T-275A rs6714486	TT ●	COMT G597A rs769224	GG ●
UGT1A6 G627T rs17863783	GG ●	GSTO2 A424G rs156697	AA ●
UGT1A6 A541G rs2070959	AG ●	GSTO1 g.G4494A rs2164624	AG ●
UGT2B15 A253C rs1902023	AC ●	GSTA1 A-135G rs3957357	AA ●
UGT1A1 C-364T rs887829	TC ●	GSTO1 C419A rs4925	CC ●
NAT2 G191A rs1801279	GG ●	GSTM1 (A>G) rs366631	AA ●

NAT2 G857A rs1799931	GG 	GSTP1 C341T rs1138272	CC 
NAT2 G590A rs1799930	GG 	GSTP1 A313G rs1695	AA 
NAT2 T341C rs1801280	TC 	GPX1 G599A rs1050450	AG 
BHMT G716A rs3733890	AA 	SOD2 C512+816A rs2758331	AC 
MTHFR G677A rs1801133	AG 	SOD2 T*1993G rs10370	TG 
MTHFR T1298G rs1801131	TT 	SOD2 A47G rs4880	AG 
MTRR A66G rs1801394	AA 	CAT C1167T rs769217	TC 
MTR G2775+157T rs2275565	GG 	CAT (C>T) rs1001179	TC 
MTR A2756G rs1805087	AA 	CAT g.G4246A rs769214	AG 
COMT G472A rs4680	AG 		

Nutrients

52

GSTO2 A424G rs156697	AA 	APOE C388T rs429358	TT 
GSTP1 A313G rs1695	AA 	ATP2B1 (A>G) rs7965584	AG 
GSTP1 C341T rs1138272	CC 	BTBD9 rs3923809	AA 
HBS1L-MYB (A>G) rs4895441	AA 	HFE C187G (H63D) rs1799945	CC 
CACNA2D3 rs1868505	CC 	HFE G845A (C282Y) rs1800562	GG 
CD36 G-180+14244A rs1761667	GG 	HIST1H2B (C>T) rs13194491	CC 
CUBN A758G rs1801222	GG 	MTHFR G677A rs1801133	AG 
CYP27B1 G-1260T rs10877012	TG 	MTR A2756G rs1805087	AA 
CYP2R1 (G>A) rs10766197	AG 	MUC1 C66T rs4072037	TC 
CYP2R1 g.C536T rs1562902	TC 	NADSYN1 rs3829251	AG 
CYP2R1 g.G3442A rs2060793	AG 	NADSYN1 rs12785878	TG 
CYP2R1 G177A rs12794714	AG 	CYP7A1 g.G2020T rs3808607	TT 
NBPF3 C134-911T rs4654748	TC 	DCDC1 (T>C) rs3925584	TT 
NPC1L1 G6735C rs2072183	GC 	DHCR7 g.T22128C rs1790349	TC 
NQO1 G559A rs1800566	AG 	FUT2 A204G rs492602	AG 
SHROOM3 rs13146355	AG 	FUT2 G461A rs601338	AG 
SLC19A1 C80T rs1051266	TC 	FUT2 G772A rs602662	AG 
SLC23A1 A207+1767G rs6133175	AG 	GC A1296C rs7041	AC 
SLC23A1 C790T rs33972313	CC 	GC A56+827G rs2298849	AA 
SLC23A1 T281-6135C rs6053005	CC 	GC T*26-796G rs2282679	TG 
SLC23A1 T379+521C rs10063949	TC 	GC T58+6190C rs1155563	TC 
TCN1 G79+372A rs526934	AG 	GPX1 G599A rs1050450	AG 
TMPRSS6 A*1241G rs855791	AG 	GSTA1 A-135G rs3957357	AA 

TCN2 G776C rs1801198	CC 	GSTM1 (A>G) rs366631	AA 
SLC1A1 C1194-956T rs301434	TC 	GSTO1 C419A rs4925	CC 
TRPM6 T4750C rs2274924	TT 	GSTO1 g.G4494A rs2164624	AG 

Mental Health

35

ESR1 A453-351G rs9340799	GG 	MAO-A T891G rs6323	GG 
FKBP5 C*1136A rs3800373	AC 	ADRA1A A883+323G rs573514	AG 
FKBP5 T106-2636C rs1360780	TC 	GAD1 G-151A rs3749034	AG 
GAD1 C-64+894A rs12185692	AC 	FAAH C385A rs324420	CC 
GAD1 C146-3245T rs3828275	TC 	BDNF C196T rs6265	CC 
HTR1A (C>G) rs6295	CC 	ADRB2 G46A rs1042713	AG 
HTR1B1 C861G rs6296	CG 	ADORA2A T1083C rs5751876	TC 
HTR2A C-998T rs6311	TT 	PER3 C2566G rs228697	CC 
MGLL G263-1443A rs604300	GG 	SLC1A1 T1110C rs301430	TT 
MTHFD1L G643+208A rs11754661	AG 	ABCC9 G3474-1222A rs11046205	GG 
MTHFR A359+160G rs17367504	AA 	ACE A-239T rs4291	TT 
OXTR G922+4581A rs53576	GG 	ANKK1 G2137A rs1800497	GG 
SOD2 A47G rs4880	AG 	ARNTL T671-305G rs11022778	TT 
TCF7L2 C450+33966T rs7903146	TC 	TNF G-238A rs361525	AG 
COMT G472A rs4680	AG 	TNF G-308A rs1800629	GG 
CRHR1 A122-1309C rs242941	AC 	TPH2 g.G4298T rs4570625	GG 
DIO2 C-143T rs12885300	CC 	SLC1A1 C1194-956T rs301434	TC 
DIO2 T274C rs225014	CC 		

GI & Detox

54

UGT1A1 C-364T rs887829	TC 	NQO1 G415A rs1131341	GG 
UGT2B15 A253C rs1902023	AC 	NQO1 G559A rs1800566	AG 
UGT1A6 G627T rs17863783	GG 	MTHFD1 G1958A rs2236225	GG 
UGT1A6 A541G rs2070959	AG 	CBS T317-917C rs2851391	TC 
NRF2 G45+2714C rs2364723	GG 	CBS G*565A rs706209	AG 
NRF2 C45+11108T rs1806649	TC 	MTHFR G677A rs1801133	AG 
NRF2 A46-4230G rs6726395	AA 	MTHFR T1298G rs1801131	TT 
NRF2 A45+10270G rs13001694	GG 	CYP2E1 C1263T rs2515641	CC 
CAT C1167T rs769217	TC 	CYP1A1 C-27+606A rs2606345	AC 
CAT (C>T) rs1001179	TC 	CYP1A1 T1384C rs1048943	TT 

SOD2 C512+816A rs2758331	AC 	CYP2B6 G516T rs3745274	TG 
SOD2 A47G rs4880	AG 	CYP2B6 T-82C rs34223104	TT 
GSTP1 A313G rs1695	AA 	CYP2B6 T983C rs28399499	TT 
GSTP1 C341T rs1138272	CC 	CYP1A2 C1291T rs28399424	CC 
GSTM1 (A>G) rs366631	AA 	CYP1A2 C-729T rs12720461	CC 
GSTA1 A-135G rs3957357	AA 	CYP1A2 T739G rs2069526	TT 
GSTO1 C419A rs4925	CC 	CYP1A2 G-3860A rs2069514	GG 
GSTO1 g.G4494A rs2164624	AG 	CYP1A2 C-163A rs762551	AC 
GSTO2 A424G rs156697	AA 	COMT G472A rs4680	AG 
FUT2 A204G rs492602	AG 	MTR A2756G rs1805087	AA 
FUT2 G461A rs601338	AG 	MTR G2775+157T rs2275565	GG 
FUT2 G772A rs602662	AG 	MTRR A66G rs1801394	AA 
GPX1 G599A rs1050450	AG 	BHMT G716A rs3733890	AA 
SIRT1 T1718C rs3758391	CC 	NAT2 G191A rs1801279	GG 
SIRT6 C137T rs352493	TT 	NAT2 G857A rs1799931	GG 
SLCO1B1 T521C rs4149056	TC 	NAT2 G590A rs1799930	GG 
XPC G2815T rs2228001	GG 	NAT2 T341C rs1801280	TC 

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