ULTAGENOMICS

DECODE YOUR DNA. GUIDE YOUR HEALTH.

Detoxification & Inflammation Response

GENETICS REPORT

1	7	130
Report	Report Panels	Gene Variants

Example Client
Prepared for

September 19, 2026
Report Date

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

THESE GENE RESULTS MIGHT TELL YOU

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

THESE GENE RESULTS WILL NOT TELL YOU

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

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

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

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

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

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

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

OVERVIEW

Results at a Glance

This report covers 7 panels and 21 individual results. The summary below shows where the genetic profile indicates areas worth focusing on, areas to monitor, and areas where results are favorable. The Moderate results are where targeted support may have the most impact.

2

NEEDS ATTENTION

- NAT2 Acetylation (Standard)
- Choline Need

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MODERATE

- General Medication Metabolism (CYP2D6)
- SOD2 Activity
- MTHFR Activity
- Glucuronidation
- Glutathione Level Propensity
- Methylation Propensity
- MTR
- AHCY
- NAT2 Acetylation
- Oxidative Stress Support

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OPTIMAL

- Acetaminophen Metabolism (CYP2E1)
- Mold Metabolism (CYP1A2)
- NSAIDs Metabolism (CYP2C9)
- Phthalate Metabolism (CYP2B6)
- Catalase Activity
- NRF2 Activity
- MTRR
- COMT
- Alcohol Inflammation Risk

1 Phase 1 Detoxification Systems

17 variants analyzed

In plain terms: The Phase 1 detoxification system in our bodies plays a crucial role in metabolizing and eliminating various substances, including toxins, medications, and environmental chemicals. This section explores genetic predispositions for specific aspects of Phase 1 detoxification, providing insights into how the body may process substances.

Once exposed to toxicants, the first phase of detoxification involves chemically transforming the compounds to make them easier to remove from the body. The effect of this transformation is that the molecules often become more toxic. Yet it is typically more desirable to have more effective phase 1 metabolism as long as the phase 2 system is working effectively.

Two aspects covered in this section are mold and phthalate metabolism. Genetics play a role is determining predispositions toward the breakdown of these compounds. Understanding these predispositions can assist in providing more precise recommendations.

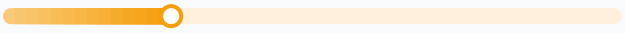
Additionally, the metabolism of medications is also influenced by a number of genetic factors. While many enzymes are involved in medication metabolism, typically the most impactful is the CYP2D6 enzyme. This enzyme is responsible for metabolizing a wide range of medications.

In addition to general CYP2D6 medications, genetic results for over-the-counter medications such as NSAIDs and acetaminophen are also included. Metabolism genetics can vary greatly and may influence outcomes.

Understanding genetic tendencies related to Phase 1 detoxification can assist in informed decisions about environmental exposures, medication choices, and potential areas of support for detoxification pathways. It's important to note that genetics is just one factor in detoxification, and lifestyle choices, environmental exposures, and overall health are also factors.

YOUR RESULTS

General Medication Metabolism (CYP2D6)



BELOW AVERAGE METABOLISM

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with below average general medication metabolism genetic results process medications that rely on the CYP2D6 enzyme more slowly than average. CYP2D6 handles many antidepressants, antipsychotics, ADHD medications, the cough suppressant dextromethorphan and the pain relievers codeine and tramadol. Codeine and tramadol need CYP2D6 to become active, so reduced activity can mean weaker pain relief. A pharmacogenomic report is recommended, especially for individuals taking psychiatric or pain medications.

YOUR RESULTS

Acetaminophen Metabolism (CYP2E1)



NORMAL METABOLISM

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with normal acetaminophen metabolism genetic results process and clear acetaminophen within the average range. Following recommended dosing and checking with a pharmacist about combination products helps keep acetaminophen use safe.

YOUR RESULTS

Mold Metabolism (CYP1A2)



RAPID METABOLISM

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with rapid metabolism mold metabolism (cyp1a2) genetic results have a rapid metabolism of mold-related substances. Their genetic profile suggests their body may have an enhanced ability to metabolize and eliminate molds efficiently. This may provide a relatively better ability to handle mold exposure and reduce susceptibility to adverse health effects. However, it is still important for individuals with rapid metabolism mold metabolism (cyp1a2) genetics to be cautious of potential mold-related risks and take appropriate preventive measures to minimize exposure.

YOUR RESULTS

NSAIDs Metabolism (CYP2C9)



NORMAL METABOLISM

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with normal NSAIDs metabolism genetic results have typical metabolism and are likely to have standard effects from over-the-counter NSAIDs.

YOUR RESULTS

Phthalate Metabolism (CYP2B6)



RAPID METABOLISM

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with rapid phthalate metabolism genetic results may break down and clear phthalates more efficiently than average, which may offer somewhat better protection from phthalate exposure. Limiting exposure, such as by avoiding heating food in plastic and choosing fragrance-free products, is still worthwhile.

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
GSTP1 A313G	rs1695	**AA**	**CYP1A2 C-163A**	rs762551	**AC**
COMT G472A	rs4680	**GA**	**CYP2B6 T983C**	rs28399499	**TT**
CYP3A4 T-392C	rs2740574	**TT**	**CYP2B6 T-82C**	rs34223104	**TT**
CYP2C9 C430T	rs1799853	**CC**	**CYP2B6 G516T**	rs3745274	**GG**
CYP2C19 G681A	rs4244285	**GG**	**CYP2B6 A785G**	rs2279343	**AG**
CYP1A2 C1291T	rs28399424	**CC**	**CYP1A1 C-27+606A**	rs2606345	**AA**
CYP1A2 C-729T	rs12720461	**CC**	**CYP1A1 T1384C**	rs1048943	**TT**
CYP1A2 T739G	rs2069526	**TT**	**CYP2E1 C1263T**	rs2515641	**CC**
CYP1A2 G-3860A	rs2069514	**GG**			

2 Phase 2 Detoxification Overview

41 variants analyzed

In plain terms: Phase 2 detoxification is a crucial process in the body's efforts to eliminate harmful substances and chemicals. It involves the transformation of fat-soluble toxins into water-soluble compounds, allowing them to be easily excreted through urine or bile. This section provides an overview of Phase 2 detoxification systems, highlighting their importance and genetic influences.

There are several pathways within Phase 2 detoxification, each responsible for processing different types of toxins. These pathways include glucuronidation, acetylation, sulfation, methylation, glutathione conjugation, and amino acid conjugation. Genetic variations can influence the efficiency of these pathways, affecting how effectively the body processes and eliminates various toxins.

Understanding genetic predispositions for Phase 2 detoxification pathways can provide insights into how efficiently the body processes and eliminates toxins. By knowing which pathways may be more or less efficient, informed lifestyle choices can be made to support the body's natural detoxification processes. However, it's important to remember that while genetics play a role, other factors such as diet, environment, and overall health also contribute to the body's detoxification capabilities. Consulting with healthcare professionals can help tailor a holistic approach to detoxification that aligns with genetic insights and supports well-being.

YOUR RESULTS

SOD2 Activity



TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with typical sod2 activity genetic results have typical SOD2 activity. Their genetic profile suggests their body can produce and maintain SOD2 levels within the average range. It is still important for individuals with typical sod2 activity genetics to continue supporting their antioxidant defense system by maintaining a balanced diet, engaging in regular physical activity, and avoiding excessive exposure to environmental toxins.

RECOMMENDATIONS

SUPPLEMENT

Manganese (as manganese bisglycinate)

5 to 10 mg/day

Manganese is the essential metal cofactor of SOD2 (mitochondrial superoxide dismutase). SOD2 cannot function without sufficient manganese; most supplement formulas omit it.

SUPPLEMENT

R-Alpha Lipoic Acid

100 to 200 mg/day

Mitochondria-targeted antioxidant that independently reduces superoxide at the mitochondrial membrane; complements reduced SOD2 activity through a parallel detoxification pathway.

DIET

Manganese-Rich Foods (whole grains, nuts, legumes, tea)

Dietary manganese is the primary cofactor supply for SOD2. Brown rice, pumpkin seeds, hazelnuts, and black tea are the richest sources.

DIET

Mitochondria-Supportive Diet (MCT oil, riboflavin, CoQ10 foods)

SOD2 acts in the mitochondrial matrix; supporting overall mitochondrial health reduces the superoxide burden on SOD2. Organ meats (liver, heart) provide the highest CoQ10 and riboflavin density.

LIFESTYLE

Moderate Exercise (not overtraining)

30 to 45 min, 4 to 5x/week

Moderate aerobic exercise upregulates SOD2 expression as an adaptive response to exercise-generated mitochondrial ROS. Overtraining produces oxidative stress faster than SOD2 can adapt; moderate intensity is the effective therapeutic window.

LIFESTYLE

Avoid Smoking and Secondhand Smoke

Smoke adds to the oxidative stress SOD2 helps clear.

FURTHER TESTING

Serum Manganese

Manganese deficiency is the most modifiable cause of SOD2 functional impairment. Low serum manganese confirms cofactor deficiency; repleting it can meaningfully restore SOD2 activity regardless of genotype.

FURTHER TESTING

Organic Acids Test (OAT), Mitochondrial Markers

Elevated succinate, fumarate, or aconitate on OAT indicates mitochondrial superoxide burden from insufficient SOD2 activity, confirming functional SOD2 impairment at the metabolite level.

YOUR RESULTS

Catalase Activity



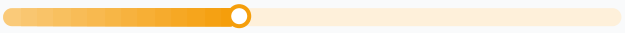
IDEAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with ideal catalase activity genetic results are likely to have standard catalase activity. This provides robust antioxidant defense, helping to protect cells from oxidative damage caused by ROS. While this genetic makeup may be favorable, it is still essential for individuals with ideal catalase activity genetics to maintain a balanced lifestyle and adopt healthy practices that support overall well-being.

YOUR RESULTS

MTHFR Activity



60-70% ENZYME ACTIVITY

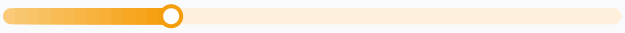
UNDERSTANDING WHAT THIS MAY MEAN

Individuals with 60-70% MTHFR enzyme activity genetic results have a moderate reduction in enzyme function and a modestly elevated risk for increased homocysteine levels. A healthcare provider can help ensure adequate methylated folate and B vitamin intake. Suggested dosing for folate: 400-800 mcg depending on needs.

Recommendations for this result are in Section 3, Methylation Expanded.

YOUR RESULTS

Glucuronidation



DECREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with decreased glucuronidation genetic results may have reduced glucuronidation function. Genetic variations associated with this result suggest that their bodies may have a decreased capacity to perform glucuronidation efficiently. It is important for individuals with this result to be mindful of their toxicant exposure and consider lifestyle practices that support overall detoxification and elimination processes. This may include consuming a nutrient-rich diet, staying hydrated, engaging in regular physical activity, and avoiding excessive exposure to environmental toxins.

Recommendations for this result are in Section 5, Phase 2 - Acetylation and Glucuronidation.

YOUR RESULTS

NAT2 Acetylation (Standard)

SLOW ACETYLATION

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with slow NAT2 acetylation activity genetic results may be at an increased risk for adverse reactions to certain medications. This is because slow acetylation can lead to slower clearance of certain drugs from the body, resulting in higher drug levels and potentially harmful side effects. It is important for individuals with slow acetylation activity to work with their medical providers to carefully monitor medication use and adjust doses as needed.

RECOMMENDATIONS

SUPPLEMENT

Pantothenic Acid (Vitamin B5)

500 to 1,000 mg/day

B5 is the precursor to coenzyme A (CoA), the acetyl group donor in NAT2-mediated acetylation reactions. Adequate CoA supply supports NAT2 function even when enzyme efficiency is reduced by genetic variants.

SUPPLEMENT

Acetyl-L-Carnitine (ALCAR)

500 to 1,000 mg/day

Provides acetyl groups that feed into the acetyl-CoA pool, supporting NAT2 acetylation capacity. Secondary benefit as a mitochondrial membrane transporter.

DIET

Avoid Processed and Cured Meats (priority for slow acetylators)

Heterocyclic amines in cooked red meat and nitrosamines in cured meats are the primary substrates for NAT2. Slow acetylators accumulate these carcinogenic intermediates longer before neutralization.

DIET

Adequate Protein for CoA Synthesis

0.7 to 1 g per lb bodyweight

CoA synthesis requires pantothenate plus several amino acid precursors. Adequate protein ensures the full complement of CoA building blocks for acetylation reactions.

LIFESTYLE

Minimize Occupational Chemical Exposure (dyes, aromatic amines)

NAT2 primary substrates include industrial chemicals, aromatic amines in hair dyes, tobacco smoke, and manufacturing. Slow acetylators face significantly higher cancer risk with occupational or cosmetic exposure.

LIFESTYLE

Inform Prescribing Physicians of NAT2 Status

Multiple medications are NAT2 substrates: isoniazid, hydralazine, procainamide, dapsone, sulfonamides. Slow acetylators experience drug toxicity at standard doses; physicians should adjust dosing and monitor more closely.

FURTHER TESTING

Urine Aromatic Amine Metabolites

Measures products of NAT2-mediated acetylation to assess functional throughput. Elevated unacetylated amines confirm slow acetylator phenotype and quantify the degree of incomplete detoxification.

FURTHER TESTING

Pharmacogenomic Report

NAT2 affects how some medications are processed.

YOUR RESULTS

NRF2 Activity

INCREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with increased nrf2 activity genetic results may have an increased NRF2 activity. Their genetic profile suggests their body may have an enhanced ability to activate the NRF2 pathway, resulting in a potentially stronger antioxidant and detoxification response. While this may confer certain benefits, it is important for individuals with improved nrf2 activity genetics to maintain a balanced approach and not rely solely on NRF2 activation. A well-rounded healthy lifestyle, including a balanced diet, regular physical activity, and other lifestyle practices that support overall well-being, is still important.

Recommendations for this result are in Section 4, Glutathione.

YOUR RESULTS

Glutathione Level Propensity

DECREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with decreased glutathione level propensity genetic results 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.

Recommendations for this result are in Section 4, Glutathione.

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
CYP3A4 T-392C	rs2740574	**TT**	**NRF2 C45+11108T**	rs1806649	**CC**
CYP2C9 C430T	rs1799853	**CC**	**NRF2 A45+10270G**	rs13001694	**AA**
CYP2C19 G681A	rs4244285	**GG**	**NRF2 A46-4230G**	rs6726395	**GG**
CYP1A1 C-27+606A	rs2606345	**AA**	**SULT1A1 C628T**	rs1042028	**TC**
COMT C186T	rs4633	**CT**	**UGT1A9 T-275A**	rs6714486	**TT**
COMT G597A	rs769224	**GG**	**UGT1A6 G627T**	rs17863783	**GG**
GSTO2 A424G	rs156697	**GG**	**UGT1A6 A541G**	rs2070959	**AG**
GSTO1 g.G4494A	rs2164624	**AA**	**UGT2B15 A253C**	rs1902023	**CC**
GSTA1 A-135G	rs3957357	**GG**	**UGT1A1 C-364T**	rs887829	**TC**
GSTO1 C419A	rs4925	**AA**	**NAT2 G191A**	rs1801279	**GG**
GSTM1 (A>G)	rs366631	**AG**	**NAT2 G857A**	rs1799931	**GG**
GSTP1 C341T	rs1138272	**CC**	**NAT2 G590A**	rs1799930	**AA**
GSTP1 A313G	rs1695	**AA**	**NAT2 T341C**	rs1801280	**TT**
GPX1 G599A	rs1050450	**AA**	**BHMT G716A**	rs3733890	**AG**
SOD2 C512+816A	rs2758331	**AC**	**MTHFR G677A**	rs1801133	**GA**

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
SOD2 T*1993G	rs10370	**TG**	**MTHFR T1298G**	rs1801131	**TT**
SOD2 A47G	rs4880	**AG**	**MTRR A66G**	rs1801394	**AG**
SELENOP C700T	rs3877899	**CC**	**MTR G2775+157T**	rs2275565	**GG**
CAT C1167T	rs769217	**CC**	**MTR A2756G**	rs1805087	**AA**
CAT (C>T)	rs1001179	**CC**	**COMT G472A**	rs4680	**GA**
NRF2 G45+2714C	rs2364723	**GG**			

3 Methylation Expanded

10 variants analyzed

In plain terms: Methylation is a vital biochemical process that occurs in every cell of our body. It plays a crucial role in various biological functions, including DNA synthesis, gene expression, detoxification, neurotransmitter synthesis, and more. This section explores genetics related to methylation and provides insights into methylation capacity and potential risks.

Genetic variations in key genes involved in the methylation pathway, such as MTHFR, COMT, and others, can influence methylation capacity. These variations can impact the efficiency of methylation reactions, potentially leading to imbalances in various metabolic processes.

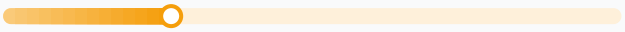
Understanding genetic predispositions for methylation can help shed light on how the body may process and utilize essential nutrients like folate, vitamin B12, and other cofactors involved in the methylation cycle. It can also provide insights into potential risks associated with impaired methylation, such as elevated homocysteine levels or reduced detoxification capacity.

This section also includes related micronutrients which are relevant to methylation activity.

Optimal methylation is important for overall health and well-being. Lifestyle factors, diet, and targeted supplementation can assist in supporting methylation capacity. This can include ensuring adequate intake of methyl-donating nutrients, adopting a healthy diet rich in whole foods, managing stress, and optimizing lifestyle choices that promote overall methylation balance.

YOUR RESULTS

Methylation Propensity



UNDERMETHYLATION POTENTIAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with undermethylation potential genetic results may have a reduced capacity for methylation, which can affect many biochemical pathways. This does not always lead to undermethylation. A balanced diet with enough B vitamins, magnesium, choline and zinc, along with guidance from a healthcare provider, can help support healthy methylation.

RECOMMENDATIONS

SUPPLEMENT

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

400 to 800 mcg methylfolate + 500 to 1,000 mcg methylcobalamin + 25 to 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 to 3,000 mg/day

Provides three methyl groups via the BHMT pathway, a methylation shortcut that bypasses the folate cycle entirely.

DIET

Choline-Rich Foods (eggs, liver, beef)

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

Choline is the primary dietary methyl donor alongside folate. Each egg provides ~150 mg choline; liver provides ~400 mg per 3 oz.

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

Manage Stress

10 to 20 minutes daily

Stress hormones use methyl groups.

FURTHER TESTING

Whole Blood Folate (not serum)

RBC folate reflects tissue folate stores over the previous 3 months; serum folate reflects only recent intake. Whole blood folate is the appropriate methylation-cycle assessment tool.

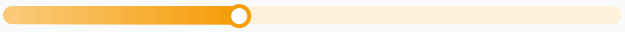
FURTHER TESTING

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.

YOUR RESULTS

MTHFR Activity



60-70% ENZYME ACTIVITY

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with 60-70% MTHFR enzyme activity genetic results have a moderate reduction in enzyme function and a modestly elevated risk for increased homocysteine levels. A healthcare provider can help ensure adequate methylated folate and B vitamin intake. Suggested dosing for folate: 400-800 mcg depending on needs.

RECOMMENDATIONS

SUPPLEMENT

Riboflavin (B2)

25 to 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.

SUPPLEMENT

5-MTHF (Methylfolate)

400 to 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.

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.

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.

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 to 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.

YOUR RESULTS

MTR



TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with typical MTR genetic results carry the most common form of this gene, found in about 56% of people. MTR helps convert homocysteine back to methionine using vitamin B12. Although common, this result is linked with the greatest potential for undermethylation and higher homocysteine of the three possible results, so adequate vitamin B12 and folate are especially helpful, and checking homocysteine may be useful alongside other methylation results.

RECOMMENDATIONS

SUPPLEMENT

Methylcobalamin (B12)

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

MTR uses methylcobalamin to transfer a methyl group from methylfolate to homocysteine, regenerating methionine.

SUPPLEMENT

Methylfolate (5-MTHF)

400 to 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.

FURTHER TESTING

Active B12 (Holotranscobalamin)

Holotranscobalamin (holoTC)

Measures the fraction of B12 actually available for cellular uptake which is more sensitive than total serum B12. Low holoTC with normal serum B12 indicates transport or absorption problems limiting B12 availability to MTR.

FURTHER TESTING

Vitamin B12 and Folate Levels

Confirms that the key methylation nutrients are adequate.

YOUR RESULTS

MTRR



TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with typical MTRR genetic results carry the most common form of this gene, which recycles vitamin B12 so the methylation cycle keeps running. Enzyme activity is not meaningfully reduced. Adequate vitamin B12 and folate still support the methylation cycle, and periodic B12 and homocysteine checks are a reasonable precaution.

YOUR RESULTS

COMT



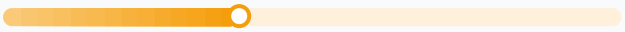
TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with typical COMT activity genetic results 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.

YOUR RESULTS

AHCY



ONE VARIANT

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with one AHCY variant genetic results carry a common variant in the enzyme that clears S-adenosylhomocysteine, a byproduct of methylation. Peer-reviewed studies have not found a meaningful effect from this variant in otherwise healthy individuals, although some other sources suggest a possible effect. This result is best read together with the Methylation Propensity result.

YOUR RESULTS

Choline Need



INCREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with increased choline need genetic results have a higher than average need for choline. Choline supports brain function, methylation, nerve function and metabolism, and good food sources include eggs and liver. Supplementation may also be helpful; 500 to 1,000 mg of alpha-GPC or at least 1,000 mg of phosphatidylcholine are likely to be most effective.

RECOMMENDATIONS

SUPPLEMENT

Alpha-GPC

300 to 600 mg daily

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

SUPPLEMENT

Citicoline (CDP-Choline)

250 to 500 mg daily

Increases choline availability and enhances phosphatidylcholine production supporting brain health and memory.

DIET

Eggs Daily

1 to 2 eggs

One egg provides about 150 mg of choline, a large share of daily needs.

DIET

Soy and Legumes

Soybeans, tofu, kidney beans

Plant sources of choline.

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.

FURTHER TESTING

Plasma Choline Level

Identifies choline insufficiency before symptoms become apparent; guides supplement form and dose selection.

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
MTHFR T1298G	rs1801131	**TT**	**MTRR A66G**	rs1801394	**AG**
MTHFR G677A	rs1801133	**GA**	**BHMT G716A**	rs3733890	**AG**
COMT G472A	rs4680	**GA**	**CBS T317-917C**	rs2851391	**TT**
MTR A2756G	rs1805087	**AA**	**CBS G*565A**	rs706209	**GG**
MTR G2775+157T	rs2275565	**GG**	**MTHFD1 G1958A**	rs2236225	**AG**

4 Glutathione

17 variants analyzed

In plain terms: Glutathione, often referred to as the 'master antioxidant,' plays a crucial role in protecting our cells from oxidative stress and maintaining overall health. This section explores genetic predispositions for glutathione levels, providing insights into how effectively the body may produce and utilize this essential antioxidant.

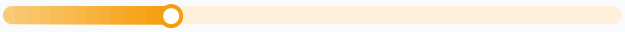
Glutathione levels refer to the amount of glutathione present in the body. Glutathione is involved in various detoxification processes and is known for its ability to neutralize harmful free radicals. Genetic variations can influence the production and regulation of glutathione, which can impact the body's overall antioxidant capacity.

NRF2 is a transcription factor that plays a key role in activating the body's antioxidant response and detoxification pathways. It is considered the ‘master regulator’ of antioxidant and inflammation-related genes including those involved in glutathione production. Genetic variations in NRF2 activity can affect its ability to regulate the expression of antioxidant enzymes, potentially influencing the body's response to oxidative stress and its ability to combat damaging free radicals.

Understanding genetic tendencies for glutathione levels and NRF2 activity can provide valuable insights into the body's antioxidant status and its ability to manage oxidative stress.

YOUR RESULTS

Glutathione Level Propensity



DECREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with decreased glutathione level propensity genetic results 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.

RECOMMENDATIONS

SUPPLEMENT

N-Acetylcysteine (NAC)

600 to 1,200 mg daily

The most evidence-backed way to raise intracellular glutathione; precursor to cysteine, the rate-limiting amino acid.

SUPPLEMENT

Glutathione or liposomal glutathione

100 to 300 mg daily

Directly replenishes intracellular stores.

DIET

Sulfur-Rich Foods

Garlic, onions, eggs, and cruciferous vegetables provide cysteine and methionine needed for glutathione synthesis.

DIET

Cruciferous Vegetables

2 to 5 cups daily

Sulforaphane activates Nrf2, the master regulator of glutathione synthesis and antioxidant gene expression.

LIFESTYLE

Prioritize Sleep

7 to 9 hours nightly

Short sleep is linked with lower antioxidant capacity.

LIFESTYLE

Reduce Stress

Chronic stress depletes glutathione through oxidative burden; stress management preserves glutathione pools.

FURTHER TESTING

Glutathione (whole blood or RBC)

Directly measures intracellular glutathione status; guides NAC and glutathione supplementation decisions.

FURTHER TESTING

GGT (Gamma-Glutamyl Transferase)

A routine liver test that can reflect glutathione demand.

YOUR RESULTS

NRF2 Activity



INCREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with increased nrf2 activity genetic results may have an increased NRF2 activity. Their genetic profile suggests their body may have an enhanced ability to activate the NRF2 pathway, resulting in a potentially stronger antioxidant and detoxification response. While this may confer certain benefits, it is important for individuals with improved nrf2 activity genetics to maintain a balanced approach and not rely solely on NRF2 activation. A well-rounded healthy lifestyle, including a balanced diet, regular physical activity, and other lifestyle practices that support overall well-being, is still important.

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
GPX1 G599A	rs1050450	**AA**	**NRF2 G45+2714C**	rs2364723	**GG**
GSTM1 (A>G)	rs366631	**AG**	**NRF2 C45+11108T**	rs1806649	**CC**
GSTP1 A313G	rs1695	**AA**	**NRF2 A45+10270G**	rs13001694	**AA**
GSTA1 A-135G	rs3957357	**GG**	**NRF2 A46-4230G**	rs6726395	**GG**
GSTO2 A424G	rs156697	**GG**	**NQO1 G559A**	rs1800566	**AG**
CBS G699A	rs234706	**AG**	**NQO1 G415A**	rs1131341	**GG**
GSTO1 g.G4494A	rs2164624	**AA**	**SELENOP C700T**	rs3877899	**CC**
GSTO1 C419A	rs4925	**AA**	**TXN A-793G**	rs2301241	**AA**
GSTP1 C341T	rs1138272	**CC**			

5 Phase 2 - Acetylation and Glucuronidation

9 variants analyzed

In plain terms: Acetylation and Glucuronidation are important processes that play roles in the metabolism and elimination of various substances, including medications and environmental toxins. This section explores genetic predispositions for acetylation and glucuronidation, providing insights into how efficiently the body may perform these processes.

Genetic variations can impact the activity of these enzymes, affecting how quickly or slowly certain substances are metabolized. Understanding genetic tendencies for acetylation and glucuronidation can help guide medication choices and dosages, as well as provide insights into potential sensitivities or susceptibilities to certain environmental toxins.

YOUR RESULTS

NAT2 Acetylation

INTERMEDIATE ACETYLATION

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with intermediate NAT2 acetylation genetic results carry one faster and one slower copy of the NAT2 gene, which places their acetylation speed between rapid and slow acetylators. This is one of the most common results in the general population. Medications cleared by NAT2, such as isoniazid, hydralazine and some sulfonamides, may build up slightly more than average, although the risk of side effects is generally lower than for slow acetylators. Individuals with this result may benefit from sharing it with their healthcare providers when new medications are started.

RECOMMENDATIONS

SUPPLEMENT

Folate (Methylfolate)

400 to 800 mcg/day

Supports the one-carbon metabolism that produces acetyl-CoA via the citrate shuttle pathway; methylation and acetylation are metabolically interlinked through shared cofactor pools.

DIET

Limit Charred and Processed Meats

Choose baking or stewing over grilling

Cooking meat at high heat forms compounds that NAT2 helps clear.

DIET

Brassica Vegetables Most Days

Cabbage, kale, broccoli

Support the body's other detox pathways.

LIFESTYLE

Limit Hair Dye and Solvent Exposure

Ventilate and wear gloves

Some dyes and solvents are cleared by NAT2.

LIFESTYLE

Ventilate When Using Solvents or Dyes

Gloves and open windows

Reduces exposure to compounds NAT2 clears.

YOUR RESULTS

Glucuronidation

DECREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with decreased glucuronidation genetic results may have reduced glucuronidation function. Genetic variations associated with this result suggest that their bodies may have a decreased capacity to perform glucuronidation efficiently. It is important for individuals with this result to be mindful of their toxicant exposure and consider lifestyle practices that support overall detoxification and elimination processes. This may include consuming a nutrient-rich diet, staying hydrated, engaging in regular physical activity, and avoiding excessive exposure to environmental toxins.

RECOMMENDATIONS

SUPPLEMENT

Calcium D-Glucarate

500 to 1,000 mg/day

Inhibits beta-glucuronidase, the gut enzyme that reverses glucuronidation and allows toxins and hormones to re-enter circulation. Directly supports Phase II detox capacity.

SUPPLEMENT

Milk Thistle

150 to 300 mg daily

May support liver detox pathways.

DIET

Fiber-Rich Foods

25 g fiber daily

Fiber helps carry cleared compounds out of the body.

DIET

Quercetin-Rich Foods (apples, onions, berries)

Daily in diet

Quercetin and polyphenols support glucuronidation enzyme activity and reduce gut beta-glucuronidase. Note COMT genotype before high-dose quercetin supplementation; dietary quercetin at food levels is appropriate for all genotypes.

LIFESTYLE

Reduce Gut Dysbiosis (Probiotic Support and Low Sugar Diet)

Ongoing

Dysbiotic gut bacteria are the primary source of beta-glucuronidase. A high-sugar diet fuels these bacteria.

LIFESTYLE

Regular Physical Activity

150 minutes weekly

Supports healthy liver function.

FURTHER TESTING

OAT Detox Markers (Pyroglutamic Acid, 2-Hydroxyhippuric Acid)

These OAT markers reflect Phase II conjugation capacity. Useful as a baseline and for monitoring progress after implementing glucuronidation support.

FURTHER TESTING

Bilirubin (on a routine panel)

Mildly higher bilirubin can reflect slower glucuronidation (as in Gilbert syndrome).

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
NAT2 G857A	rs1799931	**GG**	**UGT2B15 A253C**	rs1902023	**CC**
NAT2 G191A	rs1801279	**GG**	**UGT1A9 T-275A**	rs6714486	**TT**

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
NAT2 G590A	rs1799930	**AA**	**UGT1A6 A541G**	rs2070959	**AG**
NAT2 T341C	rs1801280	**TT**	**UGT1A6 G627T**	rs17863783	**GG**
UGT1A1 C-364T	rs887829	**TC**			

6 Oxidative Stress

15 variants analyzed

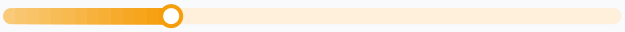
In plain terms: Oxidative stress occurs when there is an imbalance between free radicals and antioxidants in the body. Free radicals are molecules that can damage cells, proteins, and DNA, while antioxidants are substances that neutralize these harmful effects. Oxidative stress is linked to aging and various health conditions, including heart disease, diabetes, and neurodegenerative disorders. Understanding the body's ability to manage oxidative stress can provide valuable insights into one's health and potential risk factors for disease.

Genes play a crucial role in how the body handles oxidative stress. Specific genes, such as SOD2, GPX1, and CAT, are involved in producing enzymes that combat oxidative damage. Variations in these genes can affect how well these protective enzymes function. For example, some genetic variations might lead to lower enzyme activity, resulting in less effective neutralization of free radicals and higher levels of oxidative stress. By examining these genetic variations, it is possible to understand an individual's susceptibility to oxidative stress and related health issues.

Knowing one's genetic predisposition to oxidative stress can help in creating a personalized health plan. For those with genetic variations that reduce the effectiveness of antioxidant enzymes, there may be a higher risk of oxidative damage and related health problems. Recommendations to manage oxidative stress can include increasing the intake of antioxidant-rich foods, such as fruits and vegetables, and possibly using supplements. Additionally, lifestyle changes such as regular exercise, avoiding smoking, and reducing exposure to environmental pollutants can help mitigate oxidative stress. This personalized approach aims to enhance overall health and reduce the risk of diseases associated with oxidative stress.

YOUR RESULTS

Oxidative Stress Support



DECREASED

UNDERSTANDING WHAT THIS MAY MEAN

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

RECOMMENDATIONS

SUPPLEMENT

Coenzyme Q10 (CoQ10)

100 to 200 mg daily

CoQ10 acts as an electron carrier in the mitochondrial respiratory chain and as an independent lipid-phase antioxidant, protecting against the superoxide generated at Complexes I and III where the greatest electron leak occurs.

SUPPLEMENT

Natural Vitamin E

200-400 IU daily

Fat-soluble antioxidant that protects cell membranes from oxidative damage.

DIET

Antioxidant-Rich Diet

Berries, dark chocolate, green tea, and colorful vegetables provide the broadest antioxidant defense.

DIET

Limit Refined Sugars

Excess sugar is a primary driver of oxidative stress through advanced glycation end products.

LIFESTYLE

Reduce Sedentary Behavior

Regular exercise upregulates endogenous antioxidant enzymes including SOD, catalase, and glutathione peroxidase.

LIFESTYLE

Adequate Sleep

7-8 hours/night

Oxidative stress accumulates during sleep deprivation; consistent sleep is the most powerful antioxidant reset.

FURTHER TESTING

8-OHdG (urine)

Gold standard marker of DNA oxidative damage; tracks the effectiveness of antioxidant interventions.

FURTHER TESTING

Oxidized LDL

Measures lipid oxidation burden; elevated levels indicate insufficient antioxidant defense.

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
SIRT1 g.T3660C	rs12778366	**TT**	**GPX1 G599A**	rs1050450	**AA**
NFE2L2 (G>T)	rs6721961	**GG**	**NRF2 C45+11108T**	rs1806649	**CC**
UCP2 G164A	rs660339	**AG**	**NRF2 A45+10270G**	rs13001694	**AA**
UCP1 (T>C)	rs1800592	**TT**	**NRF2 A46-4230G**	rs6726395	**GG**
PPARGC1A C1444T	rs8192678	**TC**	**GSTA1 A-135G**	rs3957357	**GG**
NQO1 G559A	rs1800566	**AG**	**NRF2 G45+2714C**	rs2364723	**GG**
SOD2 C512+816A	rs2758331	**AC**	**CAT (C>T)**	rs1001179	**CC**
SOD2 A47G	rs4880	**AG**			

7 Ethanol Inflammation

21 variants analyzed

In plain terms: Alcohol is processed mainly in the liver, and breaking it down produces byproducts such as acetaldehyde that can drive inflammation and oxidative stress. Genetic variation in the body's inflammatory response can influence how strongly tissues react to alcohol exposure. This section looks at genetic tendencies for alcohol-related inflammation, which may help guide decisions about how much and how often to drink and ways to support the liver and the body's antioxidant defenses.

YOUR RESULTS

Alcohol Inflammation Risk



TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with typical alcohol inflammation risk genetic results have a typical risk associated with alcohol consumption. This genetic profile suggests a response to alcohol that is similar to the general population. It is important for individuals with typical alcohol inflammation risk genetics to follow recommended guidelines for moderate alcohol consumption. It is essential to be mindful of individual tolerance, personal health conditions, and any medication interactions.

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
ADH1C T1048C	rs698	**TT**	**CNR1 C1359T**	rs1049353	**CC**
ADH1B G1108A	rs2066702	**GG**	**CNR1 T-64+2592G**	rs6454674	**TG**
GCKR T1337C	rs1260326	**TC**	**GABRA2 A255+423G**	rs279826	**AG**
DRD3 C723+146T	rs2134655	**TC**	**GABRA2 T396C**	rs279858	**TC**
DRD2 C811-83A	rs1076560	**CC**	**APOE C526T**	rs7412	**CC**
SLC6A4 A*463C	rs1042173	**AC**	**APOE C388T**	rs429358	**TT**
FAAH C385A	rs324420	**AC**	**OPRM1 A118G**	rs1799971	**AG**
CYP2E1 C-1055T	rs2031920	**CC**	**ALDH2 G1510A**	rs671	**GG**
CYP2E1 g.G3739C	rs3813867	**GG**	**ADH1B T143C**	rs1229984	**CC**
ANKK1 G2137A	rs1800497	**GG**	**GABRA4 G76T**	rs2229940	**TG**
CNR1 T*3475C	rs806368	**TT**			

APPENDIX A

Supplement Recommendations

Based on your results

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

SUPPLEMENT	RATIONALE	PANEL
Manganese (as manganese bisglycinate) 5 to 10 mg/day	Manganese is the essential metal cofactor of SOD2 (mitochondrial superoxide dismutase). SOD2 cannot function without sufficient manganese; most supplement formulas omit it.	Phase 2 Detoxification Overview
R-Alpha Lipoic Acid 100 to 200 mg/day	Mitochondria-targeted antioxidant that independently reduces superoxide at the mitochondrial membrane; complements reduced SOD2 activity through a parallel detoxification pathway.	Phase 2 Detoxification Overview
Pantothenic Acid (Vitamin B5) 500 to 1,000 mg/day	B5 is the precursor to coenzyme A (CoA), the acetyl group donor in NAT2-mediated acetylation reactions. Adequate CoA supply supports NAT2 function even when enzyme efficiency is reduced by genetic variants.	Phase 2 Detoxification Overview
Acetyl-L-Carnitine (ALCAR) 500 to 1,000 mg/day	Provides acetyl groups that feed into the acetyl-CoA pool, supporting NAT2 acetylation capacity. Secondary benefit as a mitochondrial membrane transporter.	Phase 2 Detoxification Overview
Methylated B-Vitamin Complex (Methylfolate + Methylcobalamin + P5P) 400 to 800 mcg methylfolate + 500 to 1,000 mcg methylcobalamin + 25 to 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.	Methylation Expanded
TMG (Trimethylglycine / Betaine) 500 to 3,000 mg/day	Provides three methyl groups via the BHMT pathway, a methylation shortcut that bypasses the folate cycle entirely.	Methylation Expanded
Riboflavin (B2) 25 to 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.	Methylation Expanded
5-MTHF (Methylfolate) 400 to 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.	Methylation Expanded
Methylcobalamin (B12) 1,000 to 2,000 mcg/day sublingual or intramuscular	MTR uses methylcobalamin to transfer a methyl group from methylfolate to homocysteine, regenerating methionine.	Methylation Expanded
Methylfolate (5-MTHF) 400 to 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.	Methylation Expanded
Alpha-GPC 300 to 600 mg daily	Highly bioavailable form of choline that crosses the blood-brain barrier; increases acetylcholine for memory and learning.	Methylation Expanded
Citicoline (CDP-Choline) 250 to 500 mg daily	Increases choline availability and enhances phosphatidylcholine production supporting brain health and memory.	Methylation Expanded

SUPPLEMENT	RATIONALE	PANEL
N-Acetylcysteine (NAC) 600 to 1,200 mg daily	The most evidence-backed way to raise intracellular glutathione; precursor to cysteine, the rate-limiting amino acid.	Glutathione
Glutathione or liposomal glutathione 100 to 300 mg daily	Directly replenishes intracellular stores.	Glutathione
Folate (Methylfolate) 400 to 800 mcg/day	Supports the one-carbon metabolism that produces acetyl-CoA via the citrate shuttle pathway; methylation and acetylation are metabolically interlinked through shared cofactor pools.	Phase 2 - Acetylation and Glucuronidation
Calcium D-Glucarate 500 to 1,000 mg/day	Inhibits beta-glucuronidase, the gut enzyme that reverses glucuronidation and allows toxins and hormones to re-enter circulation. Directly supports Phase II detox capacity.	Phase 2 - Acetylation and Glucuronidation
Milk Thistle 150 to 300 mg daily	May support liver detox pathways.	Phase 2 - Acetylation and Glucuronidation
Coenzyme Q10 (CoQ10) 100 to 200 mg daily	CoQ10 acts as an electron carrier in the mitochondrial respiratory chain and as an independent lipid-phase antioxidant, protecting against the superoxide generated at Complexes I and III where the greatest electron leak occurs.	Oxidative Stress
Natural Vitamin E 200-400 IU daily	Fat-soluble antioxidant that protects cell membranes from oxidative damage.	Oxidative Stress

APPENDIX B

Further Testing Recommendations

Based on your results

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

TEST / ASSESSMENT	CLINICAL RATIONALE	PANEL
Serum Manganese	Manganese deficiency is the most modifiable cause of SOD2 functional impairment. Low serum manganese confirms cofactor deficiency; repleting it can meaningfully restore SOD2 activity regardless of genotype.	Phase 2 Detoxification Overview
Organic Acids Test (OAT), Mitochondrial Markers	Elevated succinate, fumarate, or aconitate on OAT indicates mitochondrial superoxide burden from insufficient SOD2 activity, confirming functional SOD2 impairment at the metabolite level.	Phase 2 Detoxification Overview
Urine Aromatic Amine Metabolites	Measures products of NAT2-mediated acetylation to assess functional throughput. Elevated unacetylated amines confirm slow acetylator phenotype and quantify the degree of incomplete detoxification.	Phase 2 Detoxification Overview
Pharmacogenomic Report	NAT2 affects how some medications are processed.	Phase 2 Detoxification Overview
Whole Blood Folate (not serum)	RBC folate reflects tissue folate stores over the previous 3 months; serum folate reflects only recent intake. Whole blood folate is the appropriate methylation-cycle assessment tool.	Methylation Expanded
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.	Methylation Expanded
Homocysteine	Primary clinical outcome of MTHFR activity. Elevated homocysteine confirms functional MTHFR impairment regardless of genotype; target <7 umol/L. Retesting 8 to 12 weeks after starting methylfolate confirms treatment response.	Methylation Expanded
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.	Methylation Expanded
Active B12 (Holotranscobalamin) Holotranscobalamin (holoTC)	Measures the fraction of B12 actually available for cellular uptake which is more sensitive than total serum B12. Low holoTC with normal serum B12 indicates transport or absorption problems limiting B12 availability to MTR.	Methylation Expanded
Vitamin B12 and Folate Levels	Confirms that the key methylation nutrients are adequate.	Methylation Expanded
Liver Function Tests	Choline deficiency presents as NAFLD; testing tracks liver health.	Methylation Expanded
Plasma Choline Level	Identifies choline insufficiency before symptoms become apparent; guides supplement form and dose selection.	Methylation Expanded
Glutathione (whole blood or RBC)	Directly measures intracellular glutathione status; guides NAC and glutathione supplementation decisions.	Glutathione
GGT (Gamma-Glutamyl Transferase)	A routine liver test that can reflect glutathione demand.	Glutathione

TEST / ASSESSMENT	CLINICAL RATIONALE	PANEL
OAT Detox Markers (Pyroglutamic Acid, 2-Hydroxyhippuric Acid)	These OAT markers reflect Phase II conjugation capacity. Useful as a baseline and for monitoring progress after implementing glucuronidation support.	Phase 2 - Acetylation and Glucuronidation
Bilirubin (on a routine panel)	Mildly higher bilirubin can reflect slower glucuronidation (as in Gilbert syndrome).	Phase 2 - Acetylation and Glucuronidation
8-OHdG (urine)	Gold standard marker of DNA oxidative damage; tracks the effectiveness of antioxidant interventions.	Oxidative Stress
Oxidized LDL	Measures lipid oxidation burden; elevated levels indicate insufficient antioxidant defense.	Oxidative Stress