ULTAGENOMICS

DECODE YOUR DNA. GUIDE YOUR HEALTH.

DNA Foundations

GENETICS REPORT

1	10	134
Report	Report Panels	Gene Variants

Example Client
Prepared for

November 9, 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 10 panels and 22 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.

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- Benzene Risk
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- Caffeine Effects on Sleep

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- MTHFR Activity
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- Glutathione Level Propensity
- NRF2 Activity
- CHD Benefit from Lower LDL
- Lipid Impairment Propensity
- LP(a) Risk
- Glucose Metabolism

1 Ideal Sleep Window

9 variants analyzed

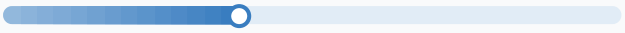
In plain terms: The Ideal Sleep Window is the optimal period of time to get the most restful and rejuvenating sleep based on circadian rhythm. Circadian rhythm refers to the internal clock that regulates various biological processes in the body, including sleep-wake cycles. Every person has a unique circadian propensity, and it determines levels of alertness and restfulness throughout the day.

There are three categories of individuals associated with varying sleep windows: morning larks, intermediate, and night owls. Morning larks are early risers who feel most alert during the first half of the day, with the earliest sleep window. On the other hand, night owls tend to feel more alert in the evening and night hours and have a later sleep window. Intermediate types fall somewhere in between morning larks and night owls.

When individuals regularly sleep outside of their ideal sleep window, performance can diminish significantly. Understanding one's ideal sleep window can provide valuable insights into how the body responds to different sleep patterns, and provide the opportunity to create the optimal sleep schedule.

YOUR RESULTS

Ideal Sleep Window Overview



INTERMEDIATE

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with an intermediate sleep window genetic result do not have a strong propensity toward being a night owl or an early morning lark. This is a relatively normal genetic result. In general, studies have found increased benefit from shifting to earlier sleep and wake times, but in general, people with this result tend to do best with bedtimes between 9 pm and 12 am and wake times between 5 and 8 am.

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
CSNK1D T130C	rs104894561	**TT**	**CRY1 G636A**	rs8192440	**GG**
PER3 T1916G	rs10462020	**TT**	**PER3 C2566G**	rs228697	**CC**
PER3 A3473G	rs10462021	**AA**	**PER3 A1247G**	rs139315125	**AA**
PER2 T1984C	rs121908635	**TT**	**PER3 C1240G**	rs150812083	**CC**
CLOCK A*213G	rs1801260	**AG**			

2 Gluten

12 variants analyzed

In plain terms: Gluten sensitivity refers to an immune or non-immune reaction to gluten, a protein found in wheat, barley, and rye. Unlike celiac disease, gluten sensitivity does not cause intestinal damage, but it can lead to digestive discomfort, fatigue, or other systemic symptoms. Genetic factors play a role in determining susceptibility to gluten sensitivity and how the body reacts to gluten. Understanding these predispositions can guide dietary choices and improve overall well-being.

Genes related to immune function and inflammation influence the body's response to gluten. Variations in the HLA-DQ2 and HLA-DQ8 genes are strongly associated with celiac disease but are also present in many individuals with non-celiac gluten sensitivity. These genes regulate immune system interactions with gluten proteins, and certain variants may trigger heightened immune responses. Additionally, genetic variations in inflammatory pathways, such as IL6, can amplify the body's reaction to gluten, contributing to symptoms even in the absence of celiac disease.

For individuals with genetic predispositions to gluten sensitivity, adopting a gluten-free or gluten-reduced diet may alleviate symptoms and improve overall health. Monitoring digestive and systemic responses to gluten-containing foods can help identify triggers. Including anti-inflammatory foods, such as fatty fish, leafy greens, and berries, can support gut health and reduce inflammation. Regular follow-ups with a healthcare provider can help assess dietary adjustments and monitor for potential nutrient deficiencies. By understanding genetic factors related to gluten sensitivity, healthcare providers can create tailored strategies for managing symptoms and promoting better health outcomes.

YOUR RESULTS

Gluten Risk



TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with typical gluten sensitivity genetic results have an average likelihood of experiencing gluten-related symptoms, consistent with the general population. Paying attention to the body's response to gluten-containing foods, and seeking medical advice if symptoms suggest sensitivity, may be helpful.

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
HLA (A>G)	rs4713586	**AA**	**IL-21 (A>G)**	rs13119723	**AA**
IL-18RAP (T>C)	rs917997	**TC**	**IL-21 (G>T)**	rs6822844	**GG**
RGS1 (C>A)	rs2816316	**AA**	**HLA-DRA (G>T)**	rs2395182	**TT**
IL-12A-AS1 (A>G)	rs17810546	**AG**	**HLA-DQB1 (T>C)**	rs7775228	**TC**
CCR3 g.T1184C	rs6441961	**TC**	**HLA C82+567T**	rs2187668	**CC**
ATXN2 C732-14033T	rs653178	**TC**	**HLA (T>C)**	rs7454108	**TT**

3 Vitamin D

14 variants analyzed

In plain terms: Vitamin D is an essential nutrient that plays a vital role in bone health, immune function, and overall well-being. This section provides insight into genetic predisposition for vitamin D levels, which may help guide steps to optimize vitamin D status and support overall health.

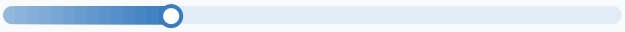
Vitamin D is often referred to as the "sunshine vitamin" because it is produced by the body in response to sunlight exposure. However, many people do not get enough Vitamin D through sunlight exposure alone and may require supplementation or dietary sources.

Vitamin D deficiency has been linked to a range of health issues, including bone weakness, weakened immune function, and an increased risk of chronic diseases. Understanding genetic predisposition for vitamin D levels may help guide targeted actions to optimize vitamin D status and reduce the risk of associated health issues.

This section describes how genetics may affect vitamin D levels and offers personalized recommendations for maintaining optimal vitamin D levels, such as getting adequate sunlight exposure, consuming Vitamin D-rich foods, and supplementing when necessary.

YOUR RESULTS

Vitamin D Deficiency Risk



DECREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with decreased vitamin D deficiency risk genetic results have a decreased likelihood of deficiency with adequate sun exposure and a balanced diet. Periodic monitoring of vitamin D levels supports early identification of any shortfalls.

YOUR RESULTS

Vitamin D Dietary Absorption



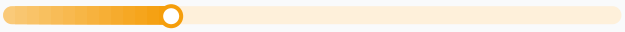
TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with typical dietary vitamin D absorption genetic results absorb vitamin D from food in a manner consistent with the general population. Maintaining adequate intake through food and/or supplementation is still important for optimal levels.

YOUR RESULTS

Inactive vs Active Vitamin D Correlation



DECREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with decreased correlation between inactive and active vitamin D genetic results may have a lower predictive relationship between 25-OH-D (inactive) and 1,25-OH-D (active) levels. This may reduce the accuracy of standard lab markers in estimating true active vitamin D status. Testing both forms of vitamin D3 (25-OH-D and 1,25-OH-D) may be worth discussing with a healthcare provider.

RECOMMENDATIONS

SUPPLEMENT

Vitamin D3 (with fat)

2,000 to 4,000 IU daily (taken with meals)

Vitamin D3 is fat-soluble; taking with a fat-containing meal increases absorption by up to 50%.

SUPPLEMENT

Magnesium (glycinate or citrate)

200 to 400 mg daily

Essential cofactor for vitamin D activation enzymes in the kidney; deficiency impairs conversion to active form.

DIET

Healthy Fats with Vitamin D

Taking vitamin D with olive oil, avocado, or nuts maximizes absorption of this fat-soluble vitamin.

DIET

Vitamin D-Rich Foods

Fatty fish, egg yolks, and fortified dairy provide dietary vitamin D alongside supplementation.

LIFESTYLE

Sun Exposure

~20 min daily without SPF

Direct UVB exposure is the most efficient way to produce active vitamin D; 20 minutes for most individuals.

LIFESTYLE

Adequate Sleep

7 to 8 hours/night

Vitamin D regulates circadian rhythm; optimizing levels supports sleep quality and sleep-dependent processes.

FURTHER TESTING

Vitamin D3 Level (Active and Inactive)

25-OH-D and 1,25-OH-D

Measuring both the inactive (25-OH-D) and active (1,25-OH-D) forms may give a clearer picture of vitamin D status when the two do not track closely; a common 25-OH-D target is 50 to 80 ng/mL.

FURTHER TESTING

PTH (Parathyroid Hormone)

Elevated PTH in the setting of low vitamin D confirms functional deficiency; guides treatment urgency.

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
NADSYN1 G146+1233T	rs12785878	**TG**	**GC T58+6190C**	rs1155563	**TT**
CYP2R1 G177A	rs12794714	**GG**	**CYP2R1 g.C536T**	rs1562902	**CC**
CYP2R1 (G>A)	rs10766197	**GG**	**NADSYN1 G1319+496A**	rs3829251	**AG**
CYP2R1 g.G3442A	rs2060793	**AA**	**VDR G2A**	rs2228570	**AA**
CYP24A1 T641-66G	rs4809958	**TT**	**VDR C1025-49A**	rs7975232	**AC**
GC T*26-796G	rs2282679	**TT**	**VDR C1024+283T**	rs1544410	**TC**
GC A1296C	rs7041	**CC**	**KL T1054G**	rs9536314	**TG**

4 Environmental Sensitivity

24 variants analyzed

In plain terms: Environmental sensitivity refers to an individual's genetic predisposition to react or respond differently to various environmental factors and chemicals. This section explores genetic risks associated with general chemical sensitivity, pesticides, and bisphenol A (BPA).

General chemical sensitivity encompasses an individual's susceptibility to adverse effects from exposure to a wide range of environmental chemicals, such as cleaning products, fragrances, and pollutants. Genetic variations can influence the body's ability to metabolize and eliminate these chemicals, potentially leading to heightened sensitivity or increased risk of adverse reactions.

Pesticide risk refers to an individual's genetic susceptibility to the potential harmful effects of pesticide exposure. Being aware of genetic predispositions for pesticide risk can guide decisions about pesticide exposure and encourage the use of protective measures when necessary.

Bisphenol A (BPA) risk focuses on an individual's genetic susceptibility to the potential health effects of BPA, a chemical commonly found in plastics and other consumer products. Genetic variations can influence how efficiently the body metabolizes and eliminates BPA, which can affect risk of BPA-related health concerns.

Understanding genetic predispositions for these enviro-toxins may help guide choices that minimize exposure and promote overall well-being.

It's important to note that while genetics play a role in environmental sensitivity, other factors such as lifestyle choices, overall health, and individual circumstances also contribute to how an individual responds to environmental factors.

YOUR RESULTS

General Chemical Sensitivity



SIGNIFICANTLY INCREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with significantly increased general chemical sensitivity genetic results may have a high risk of chemical sensitivity. Genetic variations associated with this result indicate a significant vulnerability to adverse reactions or sensitivities when exposed to certain environmental toxins. Individuals with this result may benefit from taking comprehensive measures to minimize exposure to potentially triggering substances. This may include using alternative products, maintaining a clean and well-ventilated environment, and seeking guidance to manage and reduce potential risks.

RECOMMENDATIONS

SUPPLEMENT

Glutathione

300 mg daily

Converts fat-soluble toxins to water-soluble compounds for excretion; primary detoxification antioxidant.

SUPPLEMENT

N-Acetylcysteine (NAC)

600 mg daily

Precursor to glutathione; makes toxins more water-soluble and supports liver detoxification.

DIET

Cruciferous Vegetables

2 to 5 cups daily

Contain sulforaphane, a potent Nrf2 activator that boosts phase II detox enzymes.

DIET

Sulfur-Rich Foods

Garlic, onions, and eggs support glutathione synthesis, the body's master detoxification antioxidant.

LIFESTYLE

Reduce Toxic Exposures

Use fragrance-free personal care products, avoid synthetic cleaning agents, and prioritize air filtration.

FURTHER TESTING

Organic Acids Test (OAT)

Identifies impaired detox pathways, mitochondrial dysfunction, and oxidative stress markers.

YOUR RESULTS

Benzene Risk

SIGNIFICANTLY INCREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with significantly increased benzene risk genetic results carry genetic predispositions that significantly amplify their likelihood of experiencing adverse health effects from benzene exposure. Given this pronounced risk, these individuals may benefit from comprehensive strategies to minimize exposure to benzene. This includes avoiding cigarette smoke, ensuring good ventilation when using products that may release benzene, and following safety protocols in industrial or laboratory settings where benzene use is necessary. Regular health screenings may also help with early detection and management of any benzene-related health conditions.

RECOMMENDATIONS

SUPPLEMENT

Sulforaphane

20 to 40 mg daily

Sulforaphane activates Nrf2, upregulating Phase II glutathione-S-transferase and UDP-glucuronosyltransferase enzymes that conjugate benzene oxide for urinary excretion.

SUPPLEMENT

Alpha-Lipoic Acid (ALA)

300 mg daily

Potent antioxidant that binds to heavy metals and supports liver function and phase II detox.

DIET

Avoid Charred and Smoked Foods

Charring and smoking food at high temperatures generates benzene and polycyclic aromatic hydrocarbons at concentrations that contribute meaningfully to dietary benzene load on top of environmental exposure.

LIFESTYLE

Avoid Benzene Sources

Gasoline vapor, tobacco smoke, industrial solvents, and adhesives are the largest benzene exposure sources, with petroleum products and cigarette smoke the most common household exposures.

LIFESTYLE

Adequate Hydration

64+ oz daily

Water-soluble benzene metabolites including trans-trans-muconic acid and phenylmercapturic acid are eliminated through renal excretion following Phase II conjugation, and high fluid intake ensures continuous renal flushing of these conjugates.

FURTHER TESTING

Urine trans-trans-Muconic Acid (TTMA)

TTMA is the specific urinary metabolite of benzene ring-opening that confirms active benzene exposure and reflects current metabolic processing through the primary detoxification pathway.

FURTHER TESTING

Complete Blood Count (CBC) with Differential

Benzene is a myelotoxin that selectively damages bone marrow progenitor cells, manifesting as progressive reductions in neutrophil, platelet, and red blood cell counts before clinical pancytopenia develops.

YOUR RESULTS

Pesticide Risk

TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with typical pesticide risk genetic results have a typical risk of adverse effects related to pesticide exposure, including organophosphates. Their genetic profile suggests their body responds to pesticide exposure in a manner similar to the general population. It is still worthwhile for individuals with typical pesticide risk genetics to be mindful of potential pesticide exposures and take appropriate precautions to minimize exposure. This may include using protective clothing, practicing safe handling and application of pesticides, and considering organic or pesticide-free alternatives whenever possible.

YOUR RESULTS

BPA Risk

TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with typical BPA risk genetic results have a typical risk of adverse effects associated with exposure to bisphenol A (BPA). BPA is a chemical commonly found in plastics, food containers, and other consumer products. Their genetic profile suggests their body may respond to BPA exposure in a manner similar to the general population. It is important for individuals with typical BPA risk genetics to be aware of potential sources of BPA exposure and take necessary steps to minimize exposure, such as using BPA-free products, avoiding prolonged contact with plastics, and choosing alternative packaging options whenever possible.

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
CYP2C9 A1075C	rs1057910	**AA**	**XPC G2815T**	rs2228001	**TG**
CYP2C9 C430T	rs1799853	**TC**	**SLCO1B1 T521C**	rs4149056	**TT**
GSTM1 (A>G)	rs366631	**AA**	**ANKK1 G2137A**	rs1800497	**GG**
GSTA1 A-135G	rs3957357	**AG**	**CYP1A1 T1384C**	rs1048943	**TT**
GSTP1 A313G	rs1695	**AA**	**CYP2D6 C506-1T**	rs3892097	**CC**
SOD2 C512+816A	rs2758331	**CC**	**NAT2 G857A**	rs1799931	**GG**
SOD2 A47G	rs4880	**AA**	**SOD2 C*441T**	rs5746136	**TC**
CAT (C>T)	rs1001179	**CC**	**NAT2 G191A**	rs1801279	**GG**
CYP2E1 C-1055T	rs2031920	**CC**	**NAT2 G590A**	rs1799930	**AA**
CYP2E1 g.G3739C	rs3813867	**GG**	**NAT2 T341C**	rs1801280	**TT**
BCHE C1699T	rs1803274	**CC**	**CAT C1167T**	rs769217	**CC**

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
PON1 T575C	rs662	**TT**	**HSPA1L G1478A**	rs2227956	**AA**

5 Caffeine

11 variants analyzed

In plain terms: Many people rely on caffeine to wake up and stay alert during the day. However, the effects of caffeine on sleep, and on long-term health can vary significantly from person to person.

Caffeine Effects on Sleep refers to an individual's unique response to the stimulating effects of caffeine on sleep. Some individuals are more sensitive to caffeine and may experience difficulties falling asleep, staying asleep, or experience restless and disrupted sleep patterns even with relatively small amounts of caffeine intake. On the other hand, some individuals may be less sensitive to the sleep-disrupting effects of caffeine and can consume it without significant impact on their sleep quality.

Measured effects and self-reported effects on sleep quality can vary, so using a biometric tracker can assist in ensuring an accurate reading of effects.

Caffeine metabolism is the speed at which caffeine is broken down in the body. In general, caffeine has an average half-life of 5 hours. This means that for the average individual, half of the caffeine has been removed after 5 hours, and 75% of it after 10 hours. However, metabolism can vary between 1.5 and 9.5 hours depending on a number of factors including genetics. Slow metabolizers of caffeine can have lingering effects for longer periods of time.

This page provides information on caffeine metabolism and likely effects on sleep, suggesting how different individuals may respond to caffeine.

YOUR RESULTS

Caffeine Metabolism (CYP1A2)



SLOW METABOLISM

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with slow caffeine metabolism genetic results break down caffeine more slowly than average, which means caffeine remains active in the system longer and is more likely to interfere with sleep quality depending on dosage and timing. Research shows increased health risks associated with coffee consumption for this metabolizer type. Limiting caffeine to earlier in the day may help.

RECOMMENDATIONS

SUPPLEMENT

Ashwagandha

300 to 500 mg daily

Caffeine that lingers in circulation due to slow CYP1A2 metabolism maintains HPA axis activation and elevates cortisol for significantly longer than in fast metabolizers, creating a chronic low-grade stress state.

DIET

Limit Caffeine to Morning Only

1 cup before 10 AM max

With slow CYP1A2, caffeine consumed after mid-morning remains active into the evening, disrupting sleep.

DIET

Avoid Caffeine Entirely If Sleep Is Poor

Slow metabolizers gain the most benefit from eliminating caffeine completely; even green tea may affect sleep.

LIFESTYLE

Turn Off Electronics at Night

1 hour before bed

Blue light exposure in the hour before bed suppresses melatonin onset by approximately 90 minutes in the general population, and this effect is compounded in slow caffeine metabolizers whose adenosine signaling is already disrupted by circulating caffeine at the time of sleep onset.

LIFESTYLE

Cut Off Caffeine by Early Afternoon

No caffeine after noon to 2 PM

Slow caffeine clearance means afternoon caffeine can still be active at bedtime.

FURTHER TESTING

Cortisol (4-point salivary)

Slow caffeine metabolism elevates evening cortisol; monitoring guides timing and lifestyle interventions.

FURTHER TESTING

Blood Pressure Check

Home or clinic readings

Slow caffeine metabolizers may see larger blood pressure rises from coffee.

YOUR RESULTS

Caffeine Effects on Sleep



INCREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with increased caffeine sensitivity genetic results may experience significant sleep disruption even from moderate caffeine amounts, including difficulty falling and staying asleep. Limiting caffeine to morning hours, and choosing non-caffeinated alternatives for afternoon alertness such as a short walk or light exercise, may help.

RECOMMENDATIONS

SUPPLEMENT

Glycine

3 g about 1 hour before bed

May shorten the time to fall asleep and improve how rested sleep feels.

DIET

Switch Afternoon Coffee to Decaf or Herbal Tea

Keeps the habit of an afternoon drink without adding caffeine that may linger into the night.

DIET

Watch Hidden Caffeine

Tea, chocolate, soda, energy drinks, pre-workout

Caffeine from less obvious sources may add up and affect sleep in caffeine-sensitive individuals.

LIFESTYLE

Caffeine Cutoff 8+ Hours Before Bed

A firm cutoff may reduce trouble falling asleep and lighter, more broken sleep.

LIFESTYLE

Track Sleep With and Without Caffeine

2-week comparison with a sleep tracker

Measured sleep may show caffeine effects that are not always noticed.

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
ADORA2A C-274-2427T	rs3761422	**CC**	**CYP1A2 C1291T**	rs28399424	**CC**
ADORA2A C333-527T	rs2236624	**CC**	**CYP1A2 C-729T**	rs12720461	**CC**
ADORA2A G3088-4992A	rs5751862	**AA**	**CYP1A2 T739G**	rs2069526	**TT**
ADORA2A-AS1 (C>G)	rs4822492	**GG**	**CYP1A2 G-3860A**	rs2069514	**GG**
ADORA2A C-275+1797T	rs2298383	**TT**	**CYP1A2 C-163A**	rs762551	**AC**

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
ADORA2A T1083C	rs5751876	**CC**			

6 Methylation

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.

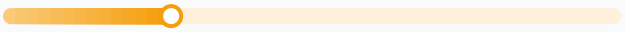
Understanding genetic predispositions for methylation can help shed light on how the body may process and utilize essential nutrients like folate, vitamin B12, and other cofactors involved in the methylation cycle. It can also provide insights into potential risks associated with impaired methylation,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

Choline Need



TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

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

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

Fish and Seafood

2 to 3 times weekly

Good sources of choline and omega-3s.

LIFESTYLE

Brain Exercise

30+ min daily

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

FURTHER TESTING

Liver Function Tests

ALT, AST, GGT, alkaline phosphatase (ALP), total bilirubin, albumin

Low choline may contribute to fatty liver (NAFLD); this panel tracks liver health, with ALT and AST as the main markers.

FURTHER TESTING

Plasma Choline Level

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

YOUR RESULTS

MTHFR Activity



48% ENZYME ACTIVITY

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with 48% MTHFR enzyme activity genetic results have a mild reduction in enzyme function and a slightly elevated risk for increased homocysteine levels and associated health concerns. The risk is lower than with further decreased enzyme activity. Work with a healthcare provider to ensure adequate methylated folate and B vitamin intake. Suggested dosing for folate: 400 to 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.

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
CBS G699A	rs234706	**GG**	**MTRR A66G**	rs1801394	**AG**
SHMT1 G1420A	rs1979277	**GG**	**MTR A2756G**	rs1805087	**AG**
MTHFR T1298G	rs1801131	**TG**	**BHMT G716A**	rs3733890	**AG**
MTHFR G677A	rs1801133	**AG**	**CBS T317-917C**	rs2851391	**TC**
COMT G472A	rs4680	**AG**	**MTHFD1 G1958A**	rs2236225	**GG**

7 Glutathione

12 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



TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with typical glutathione level propensity genetic results have typical levels of glutathione. Their genetic profile suggests their body can maintain glutathione levels within the average range. It is still important for individuals with typical glutathione level propensity genetics to support their body's natural antioxidant defense system by maintaining a balanced diet, engaging in regular physical activity, and avoiding excessive exposure to environmental toxins.

YOUR RESULTS

NRF2 Activity



INCREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with increased NRF2 activity genetic results may have 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 increased 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	**GG**	**GSTO1 C419A**	rs4925	**CC**
GSTM1 (A>G)	rs366631	**AA**	**GSTP1 C341T**	rs1138272	**CC**
GSTP1 A313G	rs1695	**AA**	**NRF2 A45+10270G**	rs13001694	**AG**
GSTA1 A-135G	rs3957357	**AG**	**NQO1 G559A**	rs1800566	**AG**
CBS G699A	rs234706	**GG**	**NQO1 G415A**	rs1131341	**GG**
GSTO1 g.G4494A	rs2164624	**GG**	**TXN A-793G**	rs2301241	**AG**

8 Oxidative Stress

11 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 to 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.

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**	**SOD2 A47G**	rs4880	**AA**
NFE2L2 (G>T)	rs6721961	**GG**	**GPX1 G599A**	rs1050450	**GG**
UCP2 G164A	rs660339	**GG**	**NRF2 A45+10270G**	rs13001694	**AG**
PPARGC1A C1444T	rs8192678	**TC**	**GSTA1 A-135G**	rs3957357	**AG**
NQO1 G559A	rs1800566	**AG**	**CAT (C>T)**	rs1001179	**CC**
SOD2 C512+816A	rs2758331	**CC**			

9 Lipid Impairment

17 variants analyzed

In plain terms: Lipid impairment, often referred to as dyslipidemia, involves abnormal levels of lipids (fats) in the blood, such as cholesterol and triglycerides. This condition is a significant risk factor for cardiovascular diseases, including heart attack and stroke. Lipid levels in the blood are influenced by both genetic and lifestyle factors. Understanding the genetic basis of lipid impairment can help identify individuals at higher risk and guide personalized approaches to prevention and treatment.

Several genes are involved in the regulation of lipid metabolism, including LDLR, APOB, and PCSK9. The LDLR gene encodes the low-density lipoprotein receptor, which plays a crucial role in removing LDL cholesterol (often called "bad" cholesterol) from the bloodstream. Variations in this gene can lead to reduced receptor function, resulting in higher LDL cholesterol levels. The APOB gene encodes apolipoprotein B, a primary component of LDL particles, and mutations can affect cholesterol transport and clearance. The PCSK9 gene encodes a protein that regulates the degradation of LDL receptors, and certain variants can lead to higher LDL cholesterol levels by reducing receptor availability. By analyzing these genetic variations, it is possible to assess an individual's risk for lipid impairment and cardiovascular disease.

Genetic insights into lipid impairment can inform personalized health strategies. For individuals with genetic variations that predispose them to higher cholesterol or triglyceride levels, lifestyle changes such as adopting a heart-healthy diet, increasing physical activity, and maintaining a healthy weight are essential. In some cases, medications like statins, which lower LDL cholesterol, or other lipid-lowering therapies may be recommended. For those with specific genetic mutations, newer treatments targeting the PCSK9 pathway might be particularly effective. Regular monitoring of lipid levels and personalized intervention plans can help manage lipid impairment and reduce the risk of cardiovascular complications. By leveraging genetic information, healthcare providers can develop targeted and effective strategies to improve lipid profiles and overall cardiovascular health.

YOUR RESULTS

CHD Benefit from Lower LDL



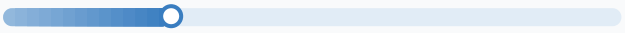
TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with typical CHD benefit from lower LDL genetic results show an average likelihood of experiencing cardiovascular benefits from decreased LDL levels. Maintaining a heart-healthy lifestyle, including a balanced diet, regular exercise, and appropriate medical care, can support overall cardiovascular health.

YOUR RESULTS

Lipid Impairment Propensity



DECREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with decreased lipid impairment propensity genetic results show a significantly lower likelihood of experiencing issues with lipid metabolism, such as high cholesterol or triglyceride levels. Maintaining a balanced diet and regular physical activity can help support overall lipid health.

YOUR RESULTS

LP(a) Risk



TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with typical Lp(a) risk genetic results have variants associated with normal function and normal to lower Lp(a) levels. Follow-up blood testing is still suggested to confirm genetic results.

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
HMGCR T*372C	rs12916	**TC**	**PCSK9 (T>C)**	rs11206510	**TT**
LDLR C81T	rs2228671	**CC**	**PCSK9 G137T**	rs11591147	**GG**

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
SORT1 (C>T)	rs646776	**TT**	**LDLR G67+2015T**	rs6511720	**GG**
SORT1 (G>A)	rs599839	**AA**	**APOC1 A*459G**	rs4420638	**AA**
ABCG8 G166-718T	rs4299376	**TG**	**LPL C1019-1582T**	rs285	**TT**
IL6/IL6-AS1 (G>C)	rs1800795	**GC**	**LPL C1421G**	rs328	**CG**
TNF G-308A	rs1800629	**AG**	**CETP g.C4402A**	rs1800775	**AC**
GCKR T1423-418C	rs780094	**CC**	**LIPC C-40-517T**	rs1800588	**TC**
ApoB A7545G	rs693	**AG**			

10 Insulin Resistance

14 variants analyzed

In plain terms: Insulin resistance is a condition characterized by the body's reduced response to the hormone insulin, leading to impaired glucose metabolism and an increased risk of developing type 2 diabetes. This section explores genetic predispositions for insulin resistance and provides insights into risk factors.

Insulin resistance can be influenced by a combination of genetic and lifestyle factors. Understanding the genetic risk for insulin resistance can help make informed decisions about diet, exercise, and overall lifestyle to reduce risk.

Genetic variations can affect how the body processes and responds to insulin. Some individuals may have a genetic predisposition for increased insulin resistance, meaning they may be more prone to developing insulin resistance even with normal or moderate levels of body fat. Others may have a lower genetic risk for insulin resistance, but lifestyle factors such as poor diet and lack of physical activity can still contribute to the development of this condition.

By knowing genetic predispositions for insulin resistance, proactive steps can be taken to reduce risk. Adopting a healthy lifestyle that includes a balanced diet, regular exercise, stress management, and adequate sleep can help improve insulin sensitivity and reduce the likelihood of developing insulin resistance.

It's important to note that genetics is just one aspect of insulin resistance, and lifestyle factors play a significant role in its development. By combining genetic insights with other clinical assessments and professional guidance, a comprehensive plan can be created to manage insulin resistance risk and promote overall health and well-being.

YOUR RESULTS

Insulin Resistance Propensity



INCREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with increased insulin resistance propensity genetic results have an increased risk of insulin resistance. Genetic variations associated with this result suggest a higher likelihood of developing insulin resistance compared to the average population. Individuals with this result may benefit from paying extra attention to lifestyle habits. Adopting a well-balanced diet with emphasis on whole foods, managing carbohydrate intake, engaging in regular physical activity, and maintaining a healthy weight can help mitigate the risk of insulin resistance and promote better metabolic health.

RECOMMENDATIONS

SUPPLEMENT

Berberine

500 mg 3x/day

Activates AMPK, improves hepatic insulin signaling, and decreases intestinal glucose absorption.

SUPPLEMENT

Myo-Inositol

2,000 mg + 50 mg D-chiro-inositol twice daily

Acts as a second messenger for insulin signaling, improving sensitivity at the cellular level.

DIET

Low Glycemic Index Foods

Prioritizing low-GI foods reduces blood sugar variability and improves insulin sensitivity over time.

DIET

Dietary Fiber

25 to 30 g daily

Soluble fiber slows glucose absorption and feeds beneficial gut bacteria that improve insulin signaling.

LIFESTYLE

Reduce Stress

Chronic cortisol elevation directly impairs insulin receptor function and drives glucose dysregulation.

LIFESTYLE

Intermittent Fasting

14+ hours fasting daily

Fasting periods reset insulin sensitivity and improve glucose metabolism significantly.

FURTHER TESTING

Fasting Insulin & HOMA-IR

Direct measure of insulin resistance; should be assessed before fasting glucose becomes abnormal.

FURTHER TESTING

HbA1c & Fasting Glucose

Long-term and snapshot glucose control markers; together provide a comprehensive picture.

YOUR RESULTS

Glucose Metabolism



TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with typical glucose metabolism genetic results exhibit genetic markers that reflect the average population's ability to metabolize glucose. This indicates a standard efficiency in processing blood sugar, with lifestyle and dietary habits playing a significant role in maintaining healthy glucose levels. Individuals with this predisposition may benefit from a healthy lifestyle, including regular exercise, a balanced diet, and regular medical check-ups, to support optimal glucose metabolism and prevent metabolic disorders.

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
ADIPOQ G3998A	rs17300539	**GG**	**NAT2 A803G**	rs1208	**AA**
IL6/IL6-AS1 (G>C)	rs1800795	**GC**	**IGF1 A26G**	rs35767	**AA**
ABCC8 C4105A	rs757110	**AC**	**DIO2 T274C**	rs225014	**TT**
GLP1R G502A	rs6923761	**AG**	**GCK C-30T**	rs1799884	**CC**
IRS1 (T>C)	rs2943641	**CC**	**GCKR T1423-418C**	rs780094	**CC**
IRS1 C2911T	rs1801278	**CC**	**PPARG C34G**	rs1801282	**CC**
MTNR1B C223+5596G	rs10830963	**CC**	**PPM1K (C>T)**	rs1440581	**TC**

APPENDIX A

Supplement Recommendations

Based on your results

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

SUPPLEMENT	RATIONALE	PANEL
Vitamin D3 (with fat) 2,000 to 4,000 IU daily (taken with meals)	Vitamin D3 is fat-soluble; taking with a fat-containing meal increases absorption by up to 50%.	Vitamin D
Magnesium (glycinate or citrate) 200 to 400 mg daily	Essential cofactor for vitamin D activation enzymes in the kidney; deficiency impairs conversion to active form.	Vitamin D
Glutathione 300 mg daily	Converts fat-soluble toxins to water-soluble compounds for excretion; primary detoxification antioxidant.	Environmental Sensitivity
N-Acetylcysteine (NAC) 600 mg daily	Precursor to glutathione; makes toxins more water-soluble and supports liver detoxification.	Environmental Sensitivity
Sulforaphane 20 to 40 mg daily	Sulforaphane activates Nrf2, upregulating Phase II glutathione-S-transferase and UDP-glucuronosyltransferase enzymes that conjugate benzene oxide for urinary excretion.	Environmental Sensitivity
Alpha-Lipoic Acid (ALA) 300 mg daily	Potent antioxidant that binds to heavy metals and supports liver function and phase II detox.	Environmental Sensitivity
Ashwagandha 300 to 500 mg daily	Caffeine that lingers in circulation due to slow CYP1A2 metabolism maintains HPA axis activation and elevates cortisol for significantly longer than in fast metabolizers, creating a chronic low-grade stress state.	Caffeine
Glycine 3 g about 1 hour before bed	May shorten the time to fall asleep and improve how rested sleep feels.	Caffeine
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
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
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
Citicoline (CDP-Choline) 250 to 500 mg daily	Increases choline availability and enhances phosphatidylcholine production supporting brain health and memory.	Methylation
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

SUPPLEMENT	RATIONALE	PANEL
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
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 to 400 IU daily	Fat-soluble antioxidant that protects cell membranes from oxidative damage.	Oxidative Stress
Berberine 500 mg 3x/day	Activates AMPK, improves hepatic insulin signaling, and decreases intestinal glucose absorption.	Insulin Resistance
Myo-Inositol 2,000 mg + 50 mg D-chiro-inositol twice daily	Acts as a second messenger for insulin signaling, improving sensitivity at the cellular level.	Insulin Resistance

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
Vitamin D3 Level (Active and Inactive) 25-OH-D and 1,25-OH-D	Measuring both the inactive (25-OH-D) and active (1,25-OH-D) forms may give a clearer picture of vitamin D status when the two do not track closely; a common 25-OH-D target is 50 to 80 ng/mL.	Vitamin D
PTH (Parathyroid Hormone)	Elevated PTH in the setting of low vitamin D confirms functional deficiency; guides treatment urgency.	Vitamin D
Organic Acids Test (OAT)	Identifies impaired detox pathways, mitochondrial dysfunction, and oxidative stress markers.	Environmental Sensitivity
Urine trans-trans-Muconic Acid (TTMA)	TTMA is the specific urinary metabolite of benzene ring-opening that confirms active benzene exposure and reflects current metabolic processing through the primary detoxification pathway.	Environmental Sensitivity
Complete Blood Count (CBC) with Differential	Benzene is a myelotoxin that selectively damages bone marrow progenitor cells, manifesting as progressive reductions in neutrophil, platelet, and red blood cell counts before clinical pancytopenia develops.	Environmental Sensitivity
Cortisol (4-point salivary)	Slow caffeine metabolism elevates evening cortisol; monitoring guides timing and lifestyle interventions.	Caffeine
Blood Pressure Check Home or clinic readings	Slow caffeine metabolizers may see larger blood pressure rises from coffee.	Caffeine
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
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
Liver Function Tests ALT, AST, GGT, alkaline phosphatase (ALP), total bilirubin, albumin	Low choline may contribute to fatty liver (NAFLD); this panel tracks liver health, with ALT and AST as the main markers.	Methylation
Plasma Choline Level	Identifies choline insufficiency before symptoms become apparent; guides supplement form and dose selection.	Methylation
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
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
8-OHdG (urine)	Gold standard marker of DNA oxidative damage; tracks the effectiveness of antioxidant interventions.	Oxidative Stress

TEST / ASSESSMENT	CLINICAL RATIONALE	PANEL
Oxidized LDL	Measures lipid oxidation burden; elevated levels indicate insufficient antioxidant defense.	Oxidative Stress
Fasting Insulin & HOMA-IR	Direct measure of insulin resistance; should be assessed before fasting glucose becomes abnormal.	Insulin Resistance
HbA1c & Fasting Glucose	Long-term and snapshot glucose control markers; together provide a comprehensive picture.	Insulin Resistance