SelfDecode™

Oxidative Stress Pathway

Sample Client

Report date: 07 September 2026

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Personal information

NAME

Sample Client

SEX AT BIRTH

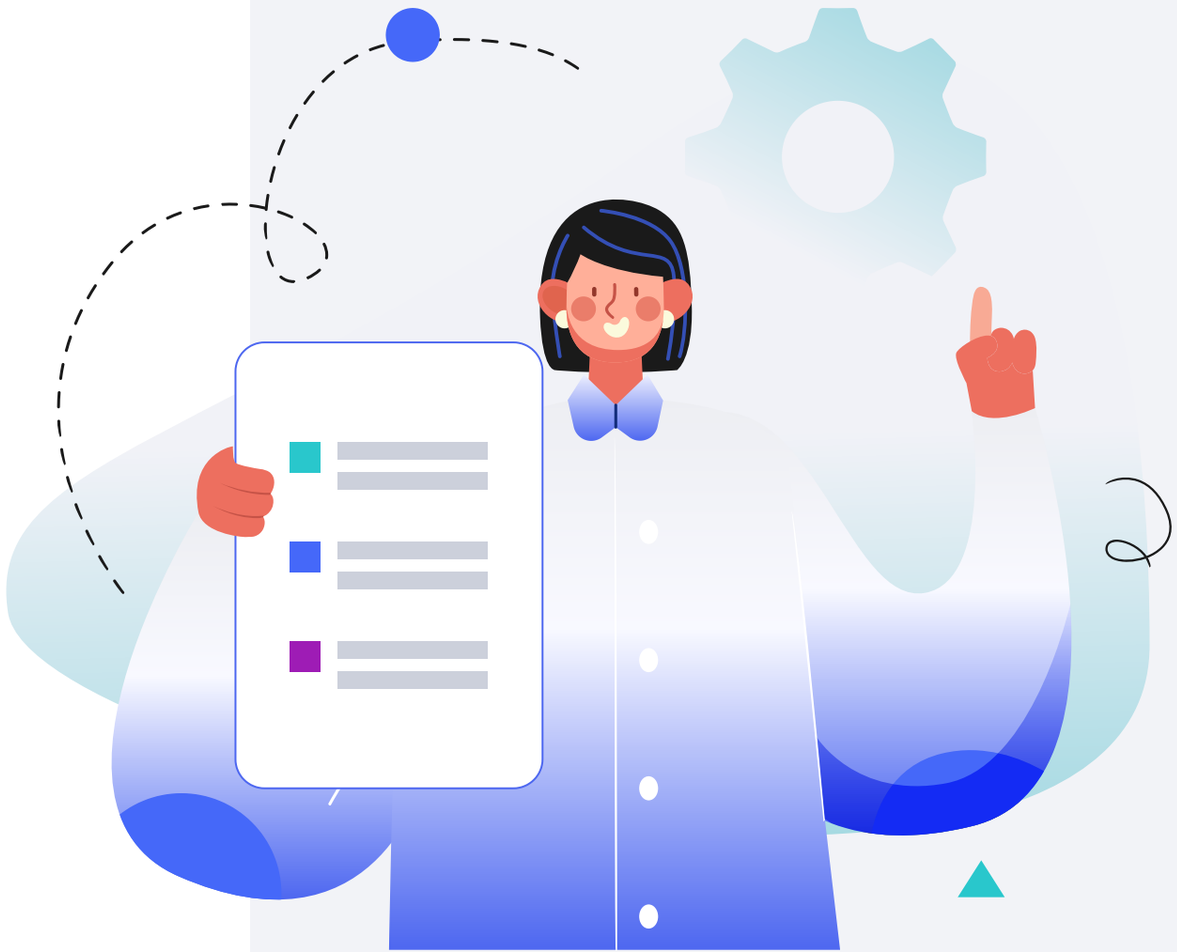
Male

HEIGHT

5ft 7" 170cm

WEIGHT

143lb 65kg



DISCLAIMER

This report does not diagnose this or any other health conditions. Please talk to a healthcare professional if this condition runs in your family, you think you might have this condition, or you have any concerns about your results.

How this works

Oxidative stress occurs when the production of reactive oxygen species (ROS) — natural byproducts of energy metabolism and immune activity — outpaces the body's ability to neutralize them. Left unchecked, these reactive molecules can damage DNA, proteins, and cell membranes, contributing to accelerated aging, chronic inflammation, and increased risk for cardiovascular disease, neurodegeneration, and cancer.

Because antioxidant defense involves many interacting genes and micronutrients, how well your body generates, buffers, and clears oxidative stress depends on the combined output of an entire genetic network.

Oxidative stress often begins with superoxide, a reactive byproduct generated during normal energy production and immune defense. **NOS3 and MPO** both influence how much oxidative burden is generated in the first place — NOS3 by producing nitric oxide (which can shift toward pro-oxidant byproducts under certain conditions), and MPO by generating reactive oxidants as part of the immune response.

The first line of defense against superoxide is **SOD2 and SOD3**, enzymes that convert superoxide into hydrogen peroxide — a necessary step, but one that creates a new reactive molecule that must itself be cleared. That responsibility falls to **CAT, GPX1, and GPX4**, which break hydrogen peroxide down into water and oxygen before it can cause further damage.

These peroxide-clearing enzymes depend heavily on **glutathione,** the body's primary antioxidant molecule. **GCLC** controls the rate-limiting step in glutathione production, while **GSR** recycles used glutathione back into its active form so it can be reused. Without adequate glutathione cycling, even well-functioning GPX1 and GPX4 enzymes run short of the raw material they need.

Much of this antioxidant machinery is coordinated by **NFE2L2 (NRF2)**, a master regulator that switches on genes involved in glutathione production, detoxification, and cellular repair in response to oxidative stress. **FOXO3** works alongside this system, supporting stress resistance and cellular longevity pathways that help offset long-term oxidative damage.

Beyond neutralizing ROS directly, the body also relies on detoxification enzymes to clear reactive byproducts and environmental toxins before they contribute to oxidative stress. **NQO1** supports antioxidant recycling and safe processing of reactive quinones, while **GSTP1, GSTA1, and PON1** help neutralize toxins, pollutants, and oxidized lipids.

HMOX1 plays a related role, breaking down heme in a way that limits its pro-oxidant potential, while **MTHFR and COMT** support methylation reactions that assist in hormone clearance and homocysteine regulation — both of which influence overall oxidative burden. **APOE**, meanwhile, affects how efficiently the body clears cholesterol and lipid byproducts, shaping long-term vulnerability to oxidative and inflammatory damage. **UCP2** adds another layer by dialing mitochondrial efficiency up or down, trading ATP output for reduced ROS generation.

This report analyzes your genetic variants across each stage of the oxidative stress pathway — from ROS generation and primary antioxidant defense to glutathione regeneration, detoxification, and long-term cellular protection — so you can better understand your unique oxidative stress tendencies and take a more personalized approach to supporting antioxidant capacity, detoxification, and long-term resilience.

How to Interpret Your Oxidative Stress Pathway Report

The two visual diagrams on the following pages are the core of the report. They show the step-by-step biochemical flow of oxidative stress generation and antioxidant defense, with each gene (and its associated SNPs) placed at the exact step where it acts. Reading these diagrams helps you understand not just how individual genes behave, but how they interact as a system.

Gene result labels tell you whether your variant is associated with:

- **Typical activity (blue)** — your gene functions within the normal expected range
- **Altered activity (orange or red)** — your variant differs from the most common form; this may mean higher or lower enzyme activity depending on the specific gene
- **Better activity (green)** — your variant is associated with a more favorable functional outcome for that step

Directional arrows on genes indicate whether a variant tends to push that step up (upregulating) or down (downregulating) relative to typical activity.

Biological compounds shown in the diagrams (e.g., homocysteine, 8-OH-dG, oxLDL) represent crucial lab markers and metabolites produced at each stage. Where lab result slots appear alongside these compounds, you can enter your own bloodwork values to see how your measured levels align with what your genetics might predict.

If a lab result is available for a specific compound, the badge color next to its name will indicate whether the result falls within the optimal range.

The **legend** in the bottom left corner explains the meaning of each element on the pathway, so please read it carefully.

Understanding the Pathway as a System

Because these genes operate as an interconnected network, your results should be read holistically rather than gene by gene. A few principles that help with interpretation:

- **Antioxidant defense happens in sequence, and every step needs to keep pace with the one before it.** SOD2 and SOD3 convert superoxide into hydrogen peroxide, but CAT, GPX1, and GPX4 are needed to finish the job. Strong superoxide dismutase activity paired with weak peroxide-clearing activity can actually increase oxidative stress rather than reduce it, since hydrogen peroxide builds up faster than it's cleared. This is the most important imbalance to look for across the core antioxidant genes.
- **Glutathione is the shared resource behind much of this system, so its supply matters as much as any single enzyme.** GPX1 and GPX4 both depend on glutathione to neutralize peroxides, GCLC controls how much new glutathione gets made, and GSR determines how efficiently used glutathione gets recycled. A variant that reduces glutathione production or recycling can blunt the benefits of otherwise strong GPX activity.
- **NFE2L2 (NRF2) sets the tone for the body's overall antioxidant response.** Because NRF2 helps activate many downstream genes — including those involved in glutathione production and detoxification — reduced NFE2L2 activity can have ripple effects across the pathway even when individual antioxidant genes are functioning normally.
- **Detoxification and antioxidant defense overlap more than they might appear to.** NQO1, GSTP1, GSTA1, PON1, and HMOX1 don't just clear toxins — they also prevent those toxins and their byproducts from generating additional oxidative stress. Weaker activity here doesn't just affect detox capacity; it can compound oxidative burden that the rest of the pathway then has to manage.
- **Methylation genes influence oxidative stress indirectly but meaningfully.** MTHFR and COMT support the clearance of hormones, homocysteine, and other reactive byproducts. Reduced activity in these genes doesn't generate ROS directly, but it can raise the baseline oxidative and inflammatory load the rest of the pathway needs to offset.
- **UCP2 represents a genuine trade-off, not a simple better-or-worse outcome.** Higher UCP2 activity reduces ROS production by making energy metabolism less efficient, while lower activity favors ATP output at the cost of higher oxidative byproducts. Which direction is more favorable depends on whether energy efficiency or oxidative load is the bigger concern for a given person.
- **APOE shapes long-term vulnerability rather than day-to-day oxidative stress.** Its role in clearing cholesterol and lipid byproducts means its effects tend to accumulate gradually, influencing long-term cardiovascular and neurological resilience rather than immediate antioxidant capacity — so it's worth reading as a longer-horizon risk factor alongside the more acute genes in this pathway.

Other Result Details

The **Results Overview** provides a structured table of every gene and SNP analyzed in the report, grouped by sub-pathway. Use this section to quickly locate your genotype for any specific gene. Each gene appears with its relevant SNP identifiers and your genotype. Alongside the genetic results, suggested lab markers are listed for each sub-pathway — these are the blood, urine, or saliva tests most relevant to validating or complementing your genetic findings.

The detailed **Gene–SNP Breakdown** section explains each gene and variant in depth. For every gene you will find:

- **What the gene does** — its biological role in fuel metabolism, mitochondrial function, antioxidant defense, etc.
- **What your specific genotype means** — whether your variant is associated with higher, lower, or typical activity at that step, and what health outcomes have been linked to it in research
- **Enhancers** — nutrients or compounds that support the function of that enzyme, listed where relevant

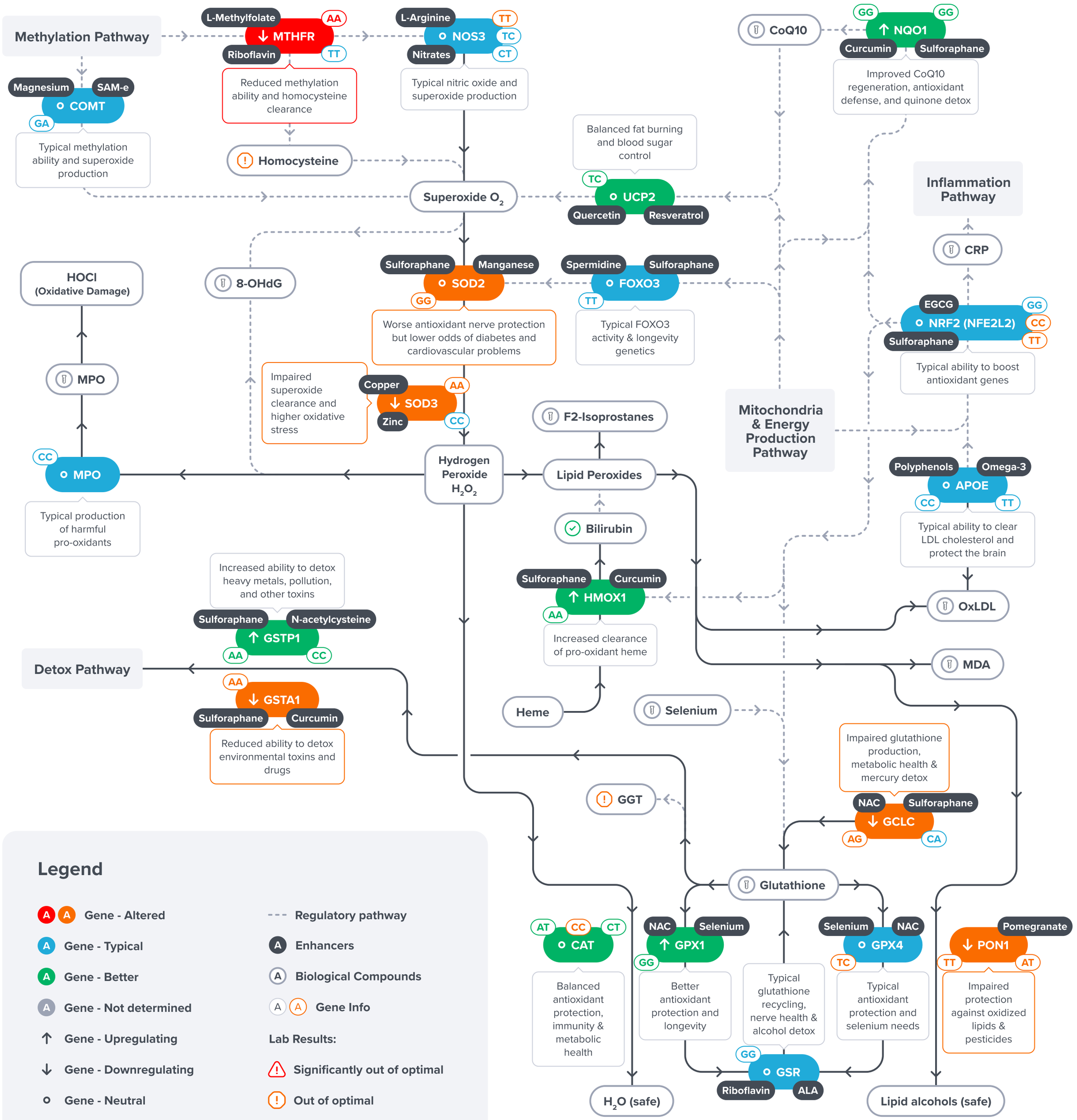
The **Recommendations** section lists personalized dietary, lifestyle, and supplement strategies, ranked by likely impact based on your specific gene variants. Each recommendation includes the genes it is relevant to and explains the biological mechanism behind it.

The recommendations are intended as a starting point for discussion with a healthcare provider, not as a prescriptive protocol. Some suggestions (such as limiting alcohol or eating fiber-rich foods) are broadly beneficial. Others (such as specific supplements at particular doses) are more targeted and should be considered in the context of your full health picture, current medications, and lab results.

Lab Markers to Check list the laboratory tests most relevant to your genetic profile, organized by the gene(s) that make them informative for you. Running these tests allows you to see whether the functional tendencies suggested by your genetics are actually showing up in your measured physiology. Genetics describes predisposition; labs confirm whether that predisposition is being expressed under your current diet, lifestyle, and environment.

Oxidative Stress Pathway

Oxidative Stress Pathway



Legend

- A A Gene - Altered
- A Gene - Typical
- A Gene - Better
- A Gene - Not determined
- ↑ Gene - Upregulating
- ↓ Gene - Downregulating
- o Gene - Neutral
- A A A Genotype
- ? Genotype not determined
- — Main pathway
- --- Regulatory pathway
- A Enhancers
- A Biological Compounds
- A A Gene Info

Lab Results:

- Significantly out of optimal
- Out of optimal
- Normal
- Optimal
- Missing lab result

Results Overview

Likely typical oxidative stress

Gene - SNP Summary

MTHFR	rs1801133	↓ AA
	rs1801131	○ TT
SOD2	rs4880	○ GG
APOE	rs7412	○ CC
	rs429358	○ TT
GSR	rs1002149	○ GG
NOS3	rs1799983	↓ TT
	rs2070744	○ CT
	rs1549758	○ TC
GSTP1	rs1695	↑ AA
	rs1138272	↑ CC

GCLC	rs17883901	↓ AG
	rs761142	○ CA
SOD3	rs2536512	↓ AA
	rs1799895	○ CC
FOXO3	rs12212067	○ TT
MPO	rs2333227	○ CC
CAT	rs7943316	○ AT
	rs1001179	○ CT
	rs769217	↓ CC
HMOX1	rs2071746	↑ AA
UCP2	rs659366	○ TC

GSTA1	rs3957357	↓ AA
PON1	rs662	↓ TT
	rs854560	↓ AT
COMT	rs4680	○ GA
GPX4	rs713041	○ TC
NFE2L2	rs6721961	○ GG
	rs35652124	↓ TT
	rs1806649	↓ CC
GPX1	rs1050450	↑ GG
NQO1	rs1800566	↑ GG
	rs1131341	↑ GG

Labs Summary

- ALT
- Blood Pressure (Diastolic)
- Blood Pressure (Systolic)
- Folate
- LDL Cholesterol
- GGT
- Homocysteine
- hs-CRP
- Iron
- Triglycerides
- Copper, Serum
- Ferritin
- HDL Cholesterol
- TIBC
- Vitamin B12
- Zinc
- 8-Hydroxy-2-deoxyguanosine, Urine
- ADMA (Asymmetric dimethylarginine)
- C-Reactive Protein (CRP)
- F2-Isoprostanes
- Malondialdehyde
- Manganese, Blood
- Mercury, Blood
- OxLDL
- Total Glutathione

Your recommendations

Your recommendations are prioritized according to the likelihood of it having an impact for you based on your lab results, along with the amount of scientific evidence supporting the recommendation.

You'll likely find common healthy recommendations at the top of the list because they are often the most impactful and most researched.

		DOSAGE
1	Sulforaphane	30 mg
2	Aerobic Exercise (Cardio)	1 hour
3	Dietary Polyphenols	
4	N-acetylcysteine (NAC)	1200 mg
5	Avoid Mycotoxin	
6	Dietary Iron	
7	Dietary Nitrates	
8	Selenium Supplements	50 mcg
9	Antioxidant Supplements	
10	Avoid Pesticide Exposure	
11	Choline Supplements	425 mg
12	Cocoa Flavanols	500 mg
13	Coenzyme Q10 (CoQ10)	100 mg
14	Cruciferous Vegetables	
15	Curcumin	500 mg
16	Dietary Copper	
17	Dietary Folate	
18	Dietary Glycine	
19	Extra Virgin Olive Oil (EVOO)	
20	Garlic	
21	Green Tea Extract	250 mg
22	Hydroxytyrosol	5 mg
23	L-Citrulline	3 g
24	Manganese supplements	1 mg
25	Omega-3 (Fish Oil)	2000 mg
26	Polyunsaturated Fatty Acids (PUFAs)	
27	Pomegranate	
28	Riboflavin (Vitamin B2)	25 mg
29	Vitamin B12	100 mcg
30	Vitamin E	200 iu

Sulforaphane

How to implement

Take a sulforaphane supplement, typically available in capsule form, with a dosage ranging from 30 to 60 milligrams per day. It is generally taken once daily, with or without food, according to the product's label instructions or a healthcare provider's advice. Continue this regimen daily for as long as you seek its benefits, but consult a healthcare provider for long-term use guidance.

TYPICAL STARTING DOSE

30 mg

How it helps

Sulforaphane is the single most potent known natural activator of NRF2, the master switch that turns on dozens of antioxidant and detoxification genes at once. Few interventions offer this broad a lift to endogenous defense in one step.

Personalized to Your Genes

↓ GCLC	NRF2 directly upregulates GCLC transcription, one of the few ways to raise expression of the rate-limiting synthesis enzyme itself.
↓ GSTA1	Sulforaphane induces GSTA1 along with the wider phase II detox network, directly upregulating the underactive enzyme.
○ SOD2	NRF2 activation upregulates SOD2 transcription and the wider mitochondrial antioxidant network, partially compensating for low baseline SOD2.
↓ SOD3	NRF2 activation upregulates the superoxide dismutase family and the wider antioxidant network, partially compensating for low SOD3.
○ NFE2L2	Sulforaphane (~10-30mg/day) is the most potent known natural NRF2 activator, directly driving the antioxidant gene program this variant underperforms.
○ CAT	NRF2 activation upregulates catalase along with the broader antioxidant network, partially compensating for the genetic downregulation.

Aerobic Exercise (Cardio)

How to implement

Engage in at least 150 minutes of moderate-intensity aerobic exercise or 75 minutes of vigorous-intensity activity each week. Distribute this time over at least 3 days per week, avoiding consecutive days of vigorous exercise to allow for recovery.

TYPICAL STARTING DOSE

1 hour

How it helps

Regular aerobic training hormetically upregulates the body's own antioxidant enzymes, including SOD2, SOD3, NRF2-driven defenses, and eNOS coupling. This 'good stress' primes cells to handle oxidative load more efficiently over time, strengthening endogenous defense rather than just neutralizing existing damage.

Personalized to Your Genes

SOD2	Regular moderate aerobic training hormetically upregulates SOD2 expression over time, strengthening the mitochondrial antioxidant defense this variant weakens.
↓ SOD3	Exercise upregulates extracellular SOD3 expression in the vasculature, one of the most effective ways to boost this specific enzyme.
NFE2L2	Exercise transiently activates NRF2, keeping the antioxidant response primed despite reduced baseline capacity.
NOS3	Shear stress from aerobic activity is a primary physiological stimulus for eNOS expression and coupling, directly countering the reduced-NO state.

Dietary Polyphenols

How to implement

Incorporate foods rich in dietary polyphenols into your daily diet. This can include consuming a variety of fruits like berries, apples, and grapes; vegetables such as onions and broccoli; nuts and seeds; as well as beverages like green tea and coffee. Aim for at least five servings of fruits and vegetables per day to achieve a beneficial intake of polyphenols.

How it helps

Polyphenols from berries, green tea, and cocoa act as direct antioxidants while also activating the body's own NRF2-driven defense genes. This dual action makes them broadly useful across nearly every oxidative-stress pathway.

Personalized to Your Genes

SOD2	Polyphenol- and carotenoid-rich produce (berries, dark leafy greens, colorful vegetables) provide layered antioxidant support to protect neural and mitochondrial membranes.
↓ SOD3	Dark cocoa, quercetin, and berries provide extracellular antioxidant capacity and protect nitric oxide from the superoxide that accumulates when SOD3 is low.
NFE2L2	EGCG, curcumin, and resveratrol activate NRF2 through complementary mechanisms, layering additional antioxidant gene induction.
CAT	Green tea, berries, and curcumin provide complementary peroxide-scavenging antioxidant capacity to offset low catalase activity.

N-acetylcysteine (NAC)

How to implement

Take 600 mg of N-Acetylcysteine (NAC) supplement daily with water. It can be taken at any time of the day, but try to take it at the same time each day for best results.

TYPICAL STARTING DOSE

1200 mg

How it helps

NAC supplies cysteine, the rate-limiting building block for the body's master antioxidant, glutathione. This directly replenishes the antioxidant reserve regardless of which specific enzyme in the glutathione pathway is underperforming.

Personalized to Your Genes

↓ GCLC	NAC (600-1200mg/day) supplies cysteine, the rate-limiting amino acid for glutathione synthesis, feeding the pathway downstream of the impaired GCLC step.
↓ GSTA1	GSTA1 requires glutathione as its conjugation substrate; NAC or liposomal glutathione maximizes the function of the impaired enzyme.
GPX4	GPX4 uses glutathione as its reducing substrate; NAC (600-1200mg/day) keeps the altered enzyme working at full capacity.
NFE2L2	NRF2 normally drives glutathione synthesis; NAC (600-1200mg/day) or liposomal glutathione directly compensates for the weakened endogenous response.

Avoid Mycotoxin

How to implement

To avoid mycotoxins, store grains and nuts in a dry, cool place, check for mold on foods before eating, and avoid consuming products with visible mold. Prefer buying whole foods over processed ones, and if possible, opt for foods tested for mycotoxin contamination. Discard any food that smells musty or shows signs of spoilage.

How it helps

Lowering exposure to environmental chemicals, heavy metals, and unnecessary xenobiotics reduces the workload on already-impaired detoxification enzymes, preventing a backlog of reactive intermediates that would otherwise cause oxidative damage.

Personalized to Your Genes

↓ GCLC	Glutathione is central to mercury detox; with impaired synthesis, reducing mercury/heavy-metal load (limit high-mercury fish, check amalgam/environmental sources) prevents depleting the limited supply.
↓ GSTA1	With impaired conjugation, lowering the incoming environmental toxin and unnecessary drug/xenobiotic load prevents overwhelming the weakened GSTA1 capacity.

Dietary Iron

How to implement

Incorporate iron-rich foods into your daily meals, such as red meat, chicken, turkey, fish, beans, lentils, tofu, cooked spinach, and fortified cereals. Aim for at least 18 mg of iron per day for adult women and 8 mg per day for adult men. It's also beneficial to pair these foods with vitamin C-rich foods like oranges, strawberries, or bell peppers to enhance iron absorption.

How it helps

Free iron is a potent catalyst for the Fenton reaction, which generates highly damaging hydroxyl radicals from otherwise mild oxidants like hydrogen peroxide. Avoiding iron excess prevents this reaction from amplifying oxidative damage.

Personalized to Your Genes

GPX4

GPX4 specifically protects against iron-driven lipid peroxidation, so avoiding iron overload reduces demand on the altered enzyme.

CAT

Catalase is a heme enzyme, so balanced iron status (neither deficient nor overloaded) supports maximal activity without adding pro-oxidant free iron.

Dietary Nitrates

How to implement

Incorporate foods high in dietary nitrates, such as beets, spinach, arugula, and celery, into your daily meals. Aim for about 300-400 mg of dietary nitrates daily, which equates to roughly 2 cups of nitrate-rich vegetables.

How it helps

Nitrates from beetroot and leafy greens convert into nitric oxide through a pathway that bypasses enzymatic NO production entirely. This restores protective vascular NO even when nitric oxide synthase itself is impaired or uncoupled.

Personalized to Your Genes

↓ SOD3

Since excess extracellular superoxide destroys NO, boosting NO availability via beetroot and leafy greens offsets the vascular consequence of impaired SOD3.

NOS3

Beetroot juice, arugula, and spinach feed the nitrate-nitrite-NO pathway, restoring NO availability independently of the impaired/uncoupled NOS3 enzyme.

Selenium Supplements

How to implement

Take 50 mcg of selenium supplements once daily, preferably with a meal to enhance absorption.

TYPICAL STARTING DOSE

50 mcg

How it helps

Selenium is the essential cofactor incorporated into the active site of glutathione peroxidase enzymes. Without adequate selenium, these enzymes cannot be properly synthesized or function, regardless of their genetic expression level.

Personalized to Your Genes

GPX4	GPX4 is a selenoprotein with elevated selenium needs in this variant; 1-2 Brazil nuts/day or 100-200mcg selenomethionine is the most direct way to sustain its activity.
CAT	The glutathione peroxidase system clears the same H2O2 catalase handles; selenium (100-200mcg) plus NAC (600-1200mg/day) compensates for reduced catalase capacity.

Antioxidant Supplements

How to implement

Take an antioxidant supplement daily with a meal to enhance absorption. The specific dose may vary depending on the type of antioxidant (e.g., vitamin C, vitamin E, selenium), but a common multivitamin or antioxidant blend can be used as directed on the product label. Consult with a healthcare provider for personalized advice and dosage.

How it helps

Vitamin C and mixed tocopherol vitamin E directly neutralize reactive oxygen species and reduce the oxidative triggers that drive compensatory overexpression of stress-response genes. Useful as general-purpose support, though effects are broad rather than targeted to a single pathway.

Personalized to Your Genes

NOS3	Tetrahydrobiopterin (BH4) deficiency causes NOS3 uncoupling; folate (5-MTHF) and vitamin C stabilize and recycle BH4, recoupling the enzyme toward NO.

Avoid Pesticide Exposure

How to implement

Purchase organic produce when possible, wash fruits and vegetables thoroughly under running water, and peel them if not organic. Use natural pest control methods instead of chemical pesticides at home and garden. Limit the use of non-organic lawn and garden chemicals.

How it helps

Organophosphate pesticides are specifically detoxified by PON1; lowering exposure through washing produce and choosing organic where relevant reduces the burden placed on an already underperforming enzyme.

Personalized to Your Genes

↓ PON1

PON1 detoxifies organophosphates; with reduced capacity, choosing organic for high-residue produce, washing produce, and avoiding organophosphate use prevents overwhelming the impaired detox system.

Choline Supplements

How to implement

Take choline supplements at a dosage of 425 mg to 550 mg daily, depending on age and gender, with a glass of water. It is best to consume choline supplements with a meal for optimal absorption. Continue this regimen daily as part of your dietary supplement routine.

TYPICAL STARTING DOSE

425 mg

How it helps

Betaine provides an alternate route to clear homocysteine, a compound that itself promotes oxidative and endothelial stress when elevated. This works independently of impaired methylation enzymes, offering a parallel pathway to reduce redox burden.

Personalized to Your Genes

↓ MTHFR

Betaine provides an alternative (BHMT) route to clear homocysteine independent of MTHFR, reducing its oxidative-endothelial burden.

Cocoa Flavanols

How to implement

Take a cocoa flavanol supplement daily, typically ranging from 500 to 1000 mg. It is best consumed with meals to enhance absorption. Continue this regimen for at least 2 to 4 weeks to start noticing potential health benefits.

TYPICAL STARTING DOSE

500 mg

How it helps

Flavanols like epicatechin activate nitric oxide pathways and have direct antioxidant and anti-inflammatory effects on the vascular endothelium. This helps counter the oxidative damage that occurs when nitric oxide signaling is impaired.

Personalized to Your Genes

NOS3

Cocoa/epicatechin and quercetin upregulate eNOS activity and improve endothelial NO output while adding antioxidant capacity against the superoxide surplus.

Coenzyme Q10 (CoQ10)

How to implement

Take a 100 mg Coenzyme Q10 (CoQ10) supplement once daily with a meal that contains fat for better absorption.

TYPICAL STARTING DOSE

100 mg

How it helps

Ubiquinol is both a direct antioxidant and an essential electron carrier in mitochondria, where most cellular ROS originates. Supplementing bypasses impaired endogenous production or recycling, restoring antioxidant capacity at the mitochondrial source of oxidative stress.

Personalized to Your Genes

SOD2

CoQ10/ubiquinol and mitochondria-targeted antioxidants like MitoQ neutralize superoxide directly within mitochondria, clearing the burden reduced SOD2 leaves.

Cruciferous Vegetables

How to implement

Incorporate a serving of cruciferous vegetables, such as broccoli, cauliflower, Brussels sprouts, kale, or cabbage, into at least one meal each day. A serving size is about a half cup cooked or one cup raw. Try to maintain this habit consistently over time for the best health outcomes.

How it helps

Beyond sulforaphane alone, whole crucifers deliver a range of glucosinolates and indoles that broadly induce phase II detoxification enzymes. This reduces the buildup of reactive, oxidative-damage-causing metabolic byproducts.

Personalized to Your Genes

↓ GSTA1

Daily cruciferous and allium vegetables (broccoli, Brussels sprouts, garlic, onions) deliver glucosinolates and organosulfur compounds that induce hepatic and intestinal GSTs.

Curcumin

How to implement

Take a 500 mg curcumin supplement daily with food. To enhance absorption, take it with a meal that contains fats or oils since curcumin is fat-soluble.

TYPICAL STARTING DOSE

500 mg

How it helps

Curcumin activates NRF2, inducing a wide range of downstream antioxidant and detoxification genes at once. This makes it a broadly effective, multi-pathway lever for boosting cellular defense against oxidative stress.

Personalized to Your Genes

↓ GSTA1

Curcumin upregulates GSTA1 expression via NRF2 activation, directly targeting the underactive enzyme alongside sulforaphane.

Dietary Copper

How to implement

Incorporate foods rich in copper into your daily diet. Include servings of shellfish, whole grains, nuts, seeds, potatoes, and dark leafy greens. Aim for an intake of 900 micrograms of copper per day for adults, adjusting slightly based on age and gender.

How it helps

Copper and zinc are the catalytic cofactors at the active site of SOD3, the primary extracellular antioxidant enzyme. Without adequate levels, the enzyme simply cannot function at full capacity regardless of gene expression.

Personalized to Your Genes

↓ SOD3

SOD3 is a copper/zinc-dependent enzyme; supplying its catalytic cofactors in balanced ratio maximizes the activity of the reduced enzyme.

Dietary Folate

How to implement

Increase your intake of folate-rich foods such as leafy green vegetables, fruits, nuts, and legumes. Aim to consume these foods daily, incorporating them into various meals throughout the day to meet the recommended dietary allowance of 400 micrograms for adults.

How it helps

Whole-food folate from leafy greens supports the broader methylation cycle, which feeds into glutathione production via the transsulfuration pathway. This provides foundational support for the body's master antioxidant system.

Personalized to Your Genes

↓ MTHFR

Whole-food folate from leafy greens (spinach, arugula, legumes) broadly supports the methylation cycle and downstream glutathione production the MTHFR bottleneck constrains.

Dietary Glycine

How to implement

To follow a glycine-rich diet, include foods that are high in glycine in your daily meals. Such foods include skin-on chicken, gelatin, bone broth, and various types of fish. Aim to consume these glycine-rich foods at least once a day to ensure you're getting a sufficient amount of glycine in your diet.

How it helps

Glutathione is a tripeptide requiring cysteine, glycine, and glutamate; a shortfall in any one limits total production regardless of enzyme activity. Ensuring all three amino acids removes a hidden bottleneck in antioxidant synthesis.

Personalized to Your Genes

↓ GCLC

Glutathione is a tripeptide of cysteine, glycine, and glutamate; ensuring glycine and glutamate/glutamine (dietary protein, collagen) prevents a second bottleneck downstream of GCLC.

Extra Virgin Olive Oil (EVOO)

How to implement

Incorporate 1-2 tablespoons of extra virgin olive oil into your daily diet. Use it as a dressing for salads, vegetables, or incorporate it into cooking, but avoid using it at high temperatures to preserve its health benefits.

How it helps

EVOO delivers oleocanthal and other polyphenols that raise PON1 activity and improve HDL's antioxidant function. This directly strengthens the body's defense against lipid oxidation, a key driver of vascular oxidative damage.

Personalized to Your Genes

↓ PON1

Olive oil polyphenols and MUFAs raise PON1 activity and improve the HDL environment the enzyme depends on to function.

Garlic

How to implement

Incorporate 1-2 cloves of raw garlic into your meals daily. This can be achieved by finely chopping or crushing the garlic and letting it sit for a few minutes before adding it to your dishes to maximize its health benefits.

How it helps

Garlic, onions, and other sulfur-containing foods support phase II detoxification enzymes and add their own antioxidant compounds, offering broad dietary reinforcement for the body's toxin-clearing and oxidative-defense systems.

Personalized to Your Genes

↓ GCLC

Dietary sulfur (e.g., from garlic) provides cysteine-rich substrate to support glutathione production despite reduced synthesis capacity.

Green Tea Extract

How to implement

Take a green tea extract supplement containing 250-500 mg of EGCG (the active compound in green tea) daily, preferably with a meal to enhance absorption. This dosage is typically split into two separate doses, taken in the morning and later in the day. Continue this regimen for at least three months to observe potential health benefits.

TYPICAL STARTING DOSE

250 mg

How it helps

EGCG is a specific, well-studied NRF2 activator that also has direct free-radical-scavenging activity. It provides a concentrated, targeted boost to the same antioxidant gene program that broader polyphenol intake supports more diffusely.

Personalized to Your Genes

NFE2L2

EGCG is a well-documented NRF2 activator, providing a specific, potent addition alongside sulforaphane to drive the antioxidant gene program this variant underperforms.

Hydroxytyrosol

How to implement

Take a hydroxytyrosol supplement at a dose of 5 to 10 mg per day, with or without food. This supplement should be taken daily for at least 8 to 12 weeks to observe potential health benefits.

TYPICAL STARTING DOSE

5 mg

How it helps

This specific olive-derived polyphenol has been shown to directly increase PON1 gene expression and enzymatic activity, offering a more concentrated and targeted boost than general olive oil intake alone.

Personalized to Your Genes

↓ PON1

Hydroxytyrosol, the primary olive polyphenol, is well-documented to directly increase PON1 activity, complementing broader olive oil intake with a specific, potent PON1 inducer.

L-Citrulline

How to implement

Take 1.5 to 5 grams of L-citrulline supplements per day, orally, with or without food. This dosage can be taken all at once or divided into two to three smaller doses throughout the day. It's generally recommended to start at the lower end of the dosage range and adjust based on your body's response.

TYPICAL STARTING DOSE

3 g

How it helps

Citrulline raises arginine levels more reliably than arginine supplementation itself due to better absorption and less first-pass metabolism, supporting nitric oxide synthase's ability to stay 'coupled' and produce NO instead of superoxide.

Personalized to Your Genes

NOS3

Citrulline (3-6g/day) raises L-arginine substrate for NOS3; adequate substrate keeps the enzyme coupled to producing NO rather than superoxide.

Manganese supplements

How to implement

Take 1-5 mg of manganese supplement daily, preferably with a meal to enhance absorption.

TYPICAL STARTING DOSE

1 mg

How it helps

Manganese sits at the catalytic core of SOD2, the primary antioxidant enzyme inside mitochondria. Without sufficient manganese, the enzyme cannot neutralize superoxide effectively no matter how much is genetically expressed.

Personalized to Your Genes

SOD2

Manganese is the catalytic cofactor at the SOD2 active site; optimizing it (whole grains, nuts, leafy greens, without excess) maximizes the reduced enzyme's activity.

Omega-3 (Fish Oil)

How to implement

Take 1-2 g of omega-3 (fish oil) supplement daily, preferably with a meal to enhance absorption.

TYPICAL STARTING DOSE

2000 mg

How it helps

EPA and DHA reduce inflammatory and oxidative signaling throughout the body while also supporting the lipid environment that protects cell membranes from peroxidation. Broadly protective across cardiovascular and neurological oxidative-stress pathways.

Personalized to Your Genes

↓ PON1

Omega-3s (2-3g/day EPA+DHA) and a favorable lipid environment support HDL function and PON1 activity, while limiting heavy alcohol (which suppresses PON1) protects it further.

Polyunsaturated Fatty Acids (PUFAs)

How to implement

Incorporate foods rich in polyunsaturated fatty acids (PUFAs) into your daily diet. Examples include eating fatty fish like salmon, mackerel, or sardines two to three times a week, using plant-based oils like flaxseed, soybean, and canola oil in cooking and salads, and adding nuts and seeds such as walnuts and flaxseeds to your meals or snacks daily.

How it helps

Polyunsaturated fats are the direct chemical substrate for lipid peroxidation. Pairing higher PUFA intake with vitamin E and other lipid-phase antioxidants protects cell membranes from the oxidative chain reactions these fats are otherwise prone to.

Personalized to Your Genes

GPX4

Polyunsaturated fats are the substrate for lipid peroxidation; moderating omega-6 and pairing omega-3s with vitamin E protects the membranes GPX4 guards.

Pomegranate

How to implement

Incorporate pomegranate into your diet by consuming fresh pomegranate seeds, drinking pure pomegranate juice, or adding pomegranate extract to smoothies or yogurts. Aim for at least one serving per day, which could be about half a cup of seeds or a glass (8 ounces) of juice, to gain its health benefits.

How it helps

Both are among the best-documented natural compounds shown to directly raise PON1 enzyme activity, strengthening the body's primary defense against oxidized lipids in the bloodstream.

Personalized to Your Genes

↓ PON1

Pomegranate polyphenols and quercetin are among the best-documented dietary upregulators of PON1 activity, directly targeting the reduced enzyme.

Riboflavin (Vitamin B2)

How to implement

Take a riboflavin (vitamin B2) supplement daily, with a dose ranging from 5mg to 400mg, depending on the specific health concern or advice from a healthcare provider. Swallow the supplement with water, preferably with a meal to enhance absorption. This regimen can be continued long-term or as directed by a healthcare professional.

TYPICAL STARTING DOSE

25 mg

How it helps

Riboflavin is the direct precursor to FAD, an essential cofactor for enzymes including glutathione reductase, MTHFR, and Complex I. Correcting even mild riboflavin insufficiency can meaningfully restore the activity of several FAD-dependent antioxidant and methylation enzymes at once.

Personalized to Your Genes

↓ MTHFR

Riboflavin is the FAD cofactor for the MTHFR enzyme itself; adequacy meaningfully improves activity of the reduced enzyme, especially in this variant.

Vitamin B12

How to implement

Take a 50 mcg vitamin B12 supplement daily, preferably with a meal to enhance absorption.

TYPICAL STARTING DOSE

100 mcg

How it helps

B12 and B6 are essential cofactors in the pathways that clear homocysteine, a compound that itself drives oxidative and endothelial stress when it accumulates. Optimizing them lowers this pro-oxidant burden at its source.

Personalized to Your Genes

↓ MTHFR

Methyl-B12 and B6 (P5P) are essential cofactors in homocysteine-clearing pathways; optimizing them lowers the pro-oxidant homocysteine this variant elevates.

Vitamin E

How to implement

Vitamin E works best when the form matches your goal. For general antioxidant support, look for a **mixed tocopherol** supplement (alpha, gamma, delta), not just alpha-tocopherol alone. If you're targeting **brain health, cholesterol balance, or deeper cellular protection**, consider a formula that also includes **tocotrienols**.

Because vitamin E is fat-soluble, take it **with a meal that contains healthy fats** to improve absorption. Many people do well with a **moderate daily dose**, rather than very high amounts of a single form.

Avoid long-term use of high-dose **alpha-tocopherol alone**, as it can crowd out other beneficial forms like gamma-tocopherol. A balanced approach is usually more effective. If you're on **blood thinners or have a bleeding disorder**, check with a healthcare professional before supplementing, as vitamin E can affect clotting.

TYPICAL STARTING DOSE

200 iu

How it helps

This lipid-soluble antioxidant works directly within cell membranes to neutralize lipid peroxides before they can propagate chain-reaction damage, complementing enzymatic defenses that operate at the membrane level.

Personalized to Your Genes

GPX4

Mixed tocopherols and CoQ10/ubiquinol are lipid-phase antioxidants that work alongside GPX4 to quench membrane lipid peroxides, sharing the protective load.

MTHFR

MTHFR Report

The MTHFR gene helps make an enzyme called methylenetetrahydrofolate reductase (MTHFR) [R].

MTHFR helps process folate (vitamin B9). Folate plays a role in [R, R, R, R]:

- DNA production
- Red blood cell production
- Normal fetal development
- Brain and heart health
- Clearing homocysteine, a protein breakdown product

Variants in the MTHFR gene can change how the enzyme functions. Two of the most widely studied variants reduce MTHFR enzyme activity [R, R, R, R].

Enhancers:

Folate | Riboflavin

SNP	Your Genotype
rs1801133	↓AA
Alleles **A:** Reduced MTHFR activity **G:** Typical MTHFR activity	**Your genotype is linked to reduced MTHFR activity, reduced methylation ability and homocysteine buildup**

Intro and Health Effects

The main *MTHFR* SNP is **rs1801133** (C677T). The **'A' variant** of this SNP decreases the activity of the MTHFR enzyme. People with two 'A' variants may have about 16% lower blood folate levels ('A' equals 'T' on the opposite DNA strand) [R].

SNP	Your Genotype
rs1801131 Alleles **G:** Reduced MTHFR activity **T:** Typical MTHFR activity	TT **Your genotype is linked to typical MTHFR activity, typical methylation ability and homocysteine buildup**

Intro and Health Effects

The **'G' variant*** of another SNP, **rs1801131** (A1298C), also decreases MTHFR enzyme activity, but less so than rs1801133. The effects of this variant may only be meaningful in people who also have the other low-activity variant, rs1801133-AA ('G' equals 'C' on the opposite DNA strand) [R, R, R, R, R].

Read this blog post for more details about MTHFR variants and potential ways to reduce their impact.

If you carry a lower-activity variant, make sure your diet is healthy, well-balanced, and contains plenty of folate-rich food sources. These include [R, R, R]:

- Spinach
- Black-eyed and green peas
- Asparagus
- Lettuce
- Avocado
- Broccoli
- Citrus fruits
- Fortified rice, bread, and pasta

Some sources recommend methylfolate supplements instead of folic acid. Methylfolate supplements would in theory bypass the MTHFR enzyme, which converts folic acid to methylfolate [R].

In addition to folate, there is some evidence that people with *MTHFR* variants may do better if they get more riboflavin (vitamin B2). This vitamin helps MTHFR work properly [R, R, R, R, R, R, R, R].

Good sources of riboflavin include [R, R]:

- Eggs
- Dairy (milk, cheese, yogurt)
- Lean and organ meats
- Green vegetables
- Fortified cereals
- Mushrooms
- Almonds

GCLC

GCLC Report

The GCLC gene helps make glutathione, often called the body's master antioxidant. Low GCLC activity can lead to:

- Reduced detoxification capacity
- Increased inflammation
- Greater susceptibility to environmental toxins
- Weakened immune response

During stress, illness, or toxin exposure, GCLC works harder to produce protective glutathione. Maintaining healthy GCLC function is key for overall wellness and longevity.

Enhancers:

N-acetylcysteine (NAC) Sulforaphane

SNP

rs17883901

Alleles

A: Reduced GCLC activity

G: Typical GCLC activity

Your Genotype

↓ AG

Your genotype is linked to reduced GCLC activity, impaired glutathione production, metabolic health & mercury detox

Intro and Health Effects

One GCLC variant, rs17883901-A, showed a link with higher levels of mercury in hair and blood in several studies. The effects were particularly big for people with two copies (AA). However, one small study found the opposite effect [R, R, R, R].

This variant may also be linked to:

- Diabetic eye disease (retinopathy) [R]
- Reduced kidney function in people with diabetes [R]
- Fatty liver (NASH) [R]
- Reduced lung function [R]
- High blood pressure in pregnancy (preeclampsia) [R]

SNP	Your Genotype
rs761142 Alleles **A:** Reduced GCLC activity **C:** Typical GCLC activity	CA **Your genotype is linked to typical GCLC activity, typical glutathione production, metabolic health & mercury detox**

Intro and Health Effects

Another less-studied GCLC gene variant, rs761142-A, may affect mercury levels and toxicity. In one study, mothers with two copies of this variant (AA) had higher hair mercury levels. Also, increased blood mercury levels during pregnancy were linked to developmental issues in their children [R].

GSTA1

GSTA1 Report

The GSTA1 gene encodes Glutathione S-transferase alpha 1, a crucial enzyme in the body's detoxification system. This protein belongs to the alpha class of GSTs, primarily expressed in the liver, kidney, and small intestine.

GSTA1 plays a vital role in Phase II detoxification by catalyzing the conjugation of reduced glutathione to various xenobiotics and endogenous compounds, making them more water-soluble and easier to excrete. The enzyme is particularly important for metabolizing environmental toxins, carcinogens, and certain medications.

GSTA1 also demonstrates peroxidase activity, helping protect cells from oxidative stress by neutralizing reactive oxygen species. Additionally, it participates in the metabolism of steroid hormones and prostaglandins, suggesting a broader role in maintaining cellular homeostasis beyond pure detoxification functions.

Enhancers:

Sulforaphane Curcumin

SNP	Your Genotype
rs3957357	↓ AA
Alleles **A:** Reduced GSTA1 activity **G:** Increased GSTA1 activity	**Your genotype is linked to reduced GSTA1 activity, reduced ability to detox environmental toxins and drugs**

Intro and Health Effects

The most studied GSTA1 variant is **rs3957357**. The **A allele** is associated with reduced enzyme activity compared to the G allele. Individuals carrying the A allele may have decreased detoxification capacity, particularly for specific environmental toxins and medications [R].

One study investigated the link between this SNP and Balkan Endemic Nephropathy (BEN) - a mysterious kidney disease that occurs almost exclusively in certain rural areas along the Danube River in southeastern Europe. People with the A allele were 60% more likely to have BEN. The study also suggested that GSTA1 is involved in **fungal toxin** (ochratoxin) metabolism [R].

This variant may also be linked to [R, R, R, R, R]:

- Higher odds of liver cancer
- Stronger adverse effects of chemo
- Higher odds of asthma and allergies

- Lower hemoglobin levels
- Lower free testosterone levels

PON1

PON1 Report

The PON1 gene, short for paraoxonase 1, encodes an enzyme that breaks apart several different types of chemical bonds. PON1 can break apart oxidized fats that could otherwise lead to heart disease, and destroy certain parts of harmful bacteria [R].

Perhaps most importantly, PON1 proteins can hydrolyze and therefore detoxify organophosphates, toxic compounds found in pesticides and biological weapons [R].

SNP

rs662

Alleles

C: Typical PON1 activity

T: Reduced PON1 activity

Your Genotype

↓TT

Your genotype is linked to reduced PON1 activity, impaired protection against oxidized lipids & pesticides

Intro and Health Effects

The rs662 variant influences how active the PON1 enzyme is. The risk allele “T” reduces the activity of the protein, resulting in less breakdown (and more potential buildup) of toxins [R].

SNP

rs854560

Alleles

A: Reduced PON1 activity

T: Typical PON1 activity

Your Genotype

↓AT

Your genotype is linked to reduced PON1 activity, impaired protection against oxidized lipids & pesticides

Intro and Health Effects

Another PON1 variant, rs854560, controls the levels of this protein in the blood. Carriers of the risk allele “A” have lower blood levels of PON1. This means they have a reduced ability to detox [R].

In line with this, a large analysis of 9 studies associated both variants with an increased risk of organophosphate toxicity. While carriers of the rs662 variant had 74% higher risk of organophosphate toxicity, the rs854560 increased the risk by 82%. In both cases, the risk was even higher in Caucasians [R].

Possibly due to their decreased breakdown of oxidized lipids, harmful bacteria, and organophosphate pesticides, these variants have also been associated with:

- Increased risk of heart disease
- Decreased longevity

SOD2

SOD2 Report

Superoxide dismutases are enzymes that transform the superoxide (O2-) radical into either ordinary oxygen (O2) or hydrogen peroxide (H2O2).

Superoxide is produced as a by-product of oxygen metabolism. If left unattended, it can cause widespread cell damage. Thus, SOD is an important part of antioxidant defense in all cells exposed to oxygen [R].

SOD2 (also called MnSOD) is one of the superoxide dismutase enzymes, alongside SOD1 and SOD3. SOD2 is unique in that it requires manganese (Mn) to work, whereas the other two need copper and zinc.

SOD2 transforms the superoxide produced by the mitochondria into the less toxic hydrogen peroxide and oxygen. This allows SOD2 to clear mitochondrial reactive oxygen species (ROS) and confer some protection against cell death [R, R].

Enhancers:

Sulforaphane

SNP

rs4880

Alleles

A: Higher SOD2 levels but lower mitochondrial activity

G: Lower SOD2 levels but higher mitochondrial activity

Your Genotype

GG

Your genotype is linked to worse antioxidant nerve protection but lower odds of diabetes and cardiovascular problems

Intro and Health Effects

The SOD2 gene has many described polymorphisms. Among them, rs4880 has got most of the spotlight in SOD2 research. Its minor **allele 'G'** is associated with **decreased SOD2 levels**. However, some cell research suggests this variant is linked to **higher SOD2 mitochondrial activity** [R].

Owing to its decreased SOD2 levels, this variant has been associated with diseases such as [R, R, R, R]:

- Breast, prostate, and colorectal cancer
- Hypertension
- Sporadic motor neuron disease
- Alzheimer's disease
- Parkinson's disease
- Noise-induced hearing loss

- Cisplatin-induced ear toxicity
- Infertility
- Phthalate-induced lung damage

In contrast, the **major 'A' variant** is more common among people with [R, R, R]:

- Diabetes and its complications
- Worse cardiovascular health (with some mixed findings)

The association of this variant with longevity isn't straightforward either. While a study found the 'G' variant was more common among very elderly Danish people, the 'A' variant was prevalent among very elderly Ashkenazi Jewish men in another study [R, R].

SOD3

SOD3 Report

The SOD3 gene codes for an enzyme called extracellular superoxide dismutase. It is a member of the superoxide dismutase (SOD) family, whose job is to take molecules of superoxide (a strong oxidant) and transform them into hydrogen peroxide and oxygen (weaker oxidants). This process is part of the body's natural antioxidant defenses [R].

On the whole, the SOD family of enzymes differ primarily in their location. SOD1 is located in the cytoplasm, or the cellular fluid, SOD2 is located in the mitochondria, and SOD3 is located outside of the cell — hence the name "extracellular superoxide dismutase." Another major difference is that SOD2 requires manganese to function, while SOD1 and SOD3 depend on copper and zinc [R, R].

Enhancers:

Zinc

SNP

rs2536512

Alleles

A: Lower SOD3 activity

G: Higher SOD3 activity

Your Genotype

↓AA

Your genotype is linked to lower SOD3 activity, impaired superoxide clearance and higher oxidative stress

Intro and Health Effects

The only association study to find a link between *SOD3* and human lifespan discovered that rs2536512 predicted age at death in a group of just over 1000 participants. In this study, people with the 'GG' genotype lived slightly longer than those with the 'AG' genotype, and those with the 'AG' genotype lived slightly longer than those with the 'AA' genotype. The total difference in age at death between 'GG' and 'AA' was about a year and a half (88.9 vs. 87.4) [R].

The risk 'A' variant has been associated with the following conditions [R, R]:

- High triglyceride levels
- Type 2 diabetes and insulin resistance

However, 'G' is the allele associated with high blood pressure [R].

The connection between SOD3 and longevity is broadly speculative at this point. Researchers have found an association between variants at this gene and longevity in mice, but the search for a human connection hasn't borne much fruit so far. In fact, at least one study of known SOD3 variants found no correlation at all with longevity in German volunteers [R].

That said, there's a reason why researchers keep looking. While studies on variants in the human SOD3 gene haven't yet turned up a direct correlation with lifespan, such variants do appear to influence:

- Risk of dying from COPD [R]
- Lung injury and mortality [R]
- Stroke in women [R]

SNP

rs1799895

Alleles

G: Reduced SOD3 activity

C: Typical SOD3 activity

Your Genotype

CC

Your genotype is linked to typical SOD3 activity and typical superoxide clearance in the arterial walls

Intro and Health Effects

rs1799895 is another well-researched SOD3 variant. Its rare 'G' allele increases SOD levels in the blood by approximately 10 times, but **reduces them in the arterial walls**. In line with this, this variant has been associated with an increased risk of several cardiovascular conditions [R, R, R, R].

APOE

APOE Report

The APOE gene makes Apolipoprotein E (ApoE). ApoE is a protein critical for the transport and metabolism of fats throughout the body [R, R, R, R, R].

ApoE also plays a role in [R, R, R, R, R, R]:

- Nerve cell function
- Heart and blood vessel function
- Clearance of proteins from the brain
- The immune response

The APOE gene can make different forms of the ApoE protein based on an individual's genetics. It may be harder for certain ApoE proteins to clear fats from the blood. This can raise levels of fat in the blood and contribute to artery hardening [R, R].

Despite the important role of ApoE in heart health, most of the research has been focused on the brain. APOE is the major gene affecting the risk of late-onset (after the age of 65) Alzheimer's disease [R, R].

There are three major forms (variants) of the APOE gene. These are called ε2, ε3, and ε4. You can have two copies of the same variant or two different variants [R, R].

Enhancers:

Omega-3

SNP

rs7412

Alleles

C: Typical APOE activity

T: Increased APOE activity

Your Genotype

CC

Your genotype is linked to typical APOE activity, brain protection, and cholesterol clearance.

Intro and Health Effects

ε3 is the most common APOE variant. It makes a protein that is good at clearing plaque from the brain and fats from the blood. Most people have two ε3 variants and a typical risk of Alzheimer's disease [R].

rs7412-T creates **ε2**, a less common APOE variant. It makes a protein that is better than ε3 at removing plaque from the brain, but not as good at removing fats from the blood. ε2 has been linked to a **lower risk of Alzheimer's disease** [R, R, R].

However, it has also been linked to a **higher risk of artery hardening in people with two ε2 variants** and an underlying chronic health condition, such as obesity or diabetes [R, R, R].

SNP

rs429358

Alleles

C: Reduced APOE activity

T: Typical APOE activity

Your Genotype

TT

Your genotype is linked to typical APOE activity, brain protection, and cholesterol clearance.

Intro and Health Effects

ε3 is the most common variant. It makes a protein that is good at clearing plaque from the brain and fats from the blood. Most people have two ε3 variants and a typical risk of Alzheimer's disease [R].

rs429358-C creates **ε4**, a less common APOE form. It makes a protein that is not as good at clearing plaque from the brain and fats from the blood. ε4 has been linked to a **higher risk of Alzheimer's disease and artery hardening** [R, R].

COMT

COMT Report

The COMT gene helps make an enzyme called catechol-O-methyltransferase (COMT). The COMT enzyme helps break down chemical messengers in the body. These include [R, R, R]:

- Dopamine
- Norepinephrine (noradrenaline)
- Epinephrine (adrenaline)

Dopamine triggers feelings of pleasure and reward. It is also important for many cognitive functions, such as memory and attention. Norepinephrine and epinephrine support the “fight or flight” stress response [R, R, R, R, R].

In addition, COMT helps break down other compounds such as estrogen byproducts, bioflavonoids (e.g., quercetin and fisetin), tea catechins, and certain drugs (e.g., L-DOPA and dobutamine) [R, R].

The activity of the COMT enzyme may influence [R, R, R, R, R]:

- Stress response
- Mental health
- Cognitive function
- Pain sensitivity

Enhancers:

Magnesium SAMe

SNP

rs4680

Alleles

A: Reduced COMT activity

G: Increased COMT activity

Your Genotype

GA

Your genotype is linked to typical COMT activity, typical methylation ability and clearance of hormones & pro-oxidants

Intro and Health Effects

One common variant of the *COMT* gene, rs4680, may affect COMT enzyme activity. Some people call rs4680 the “worrier or warrior” variant [R, R].

The “G” allele of this variant is linked to a higher COMT enzyme activity. People with two copies of this allele (GG) have been nicknamed the “warriors.” They break down stress-related chemical messengers more quickly. This may help improve their performance under stress [R].

On the negative side, “warriors” may have lower cognitive performance under relaxed conditions [R, R, R].

People with two copies of the “A” allele (AA) may have lower COMT enzyme activity. They have been nicknamed the “worriers.” They break down stress-related chemical messengers more slowly in the brain. For this reason, they may be more vulnerable to stress. This includes an increased susceptibility to heart disease, possibly due to the effects of these chemical messengers on blood pressure and heart rate [R, R, R, R].

The good news is that "worriers" may become more emotionally resilient with age. They also tend to have enhanced cognitive performance under relaxed conditions. Interestingly, "worriers" seem to have a more pronounced placebo response due to higher dopamine levels [R, R, R, R, R].

People carrying both alleles (AG) tend to be in between the described extremes [R, R].

Did you know? People with “warrior” genetics may be more likely to engage in combat sports, justifying the nickname of this variant [R].

However, keep in mind that your cognitive function and response to stress are also influenced by other factors, such as:

- Other variants in the *COMT* gene
- Many other genes
- Environmental factors

FOXO3

FOXO3 Report

The FOXO3 gene encodes a transcription factor, a protein that binds to DNA and regulates the activity of other genes. For instance, FOXO3 regulates insulin signalling and protein metabolism in muscles [R].

FOXO3 is involved in tumor suppression, immune function, DNA repair, and resistance to oxidative stress, which may explain its link with increased longevity [R, R, R].

FOXO3 also plays a role in managing oxidative stress. Mice without FOXO3 are highly susceptible to reactive oxygen species in their bones, resulting in decreased bone mass and increased risk of fracture (osteoporosis). The antioxidant effect of FOXO3 has not yet been investigated in aged mice [R, R].

Enhancers:

Sulforaphane

SNP	Your Genotype
rs12212067	TT
Alleles **G:** Increased FOXO3 activity **T:** Typical FOXO3 activity	**Your genotype is linked to typical FOXO3 activity & longevity genetics**

Intro and Health Effects

In the last decade or so, a large body of research has linked certain variants in the FOXO3 gene with longer lifespan. They include [R, R, R, R, R, R, R]:

- ‘G’ at rs12212067

Interestingly, the effects of some of these variants may be strongest in people who spend more time in nature [R].

The ‘G’ allele of rs12212067 has also been associated with a milder course of Crohn’s disease [R, R].

GPX4

GPX4 Report

In the intricate world of cellular metabolism, the GPX4 gene stands out as a critical guardian against oxidative stress. This gene encodes glutathione peroxidase 4 (GPX4), an enzyme pivotal in protecting cells from damage caused by reactive oxygen species (ROS). It is deeply intertwined with selenium and glutathione metabolism, making it a cornerstone of cellular health.

GPX4 supports the reduction of lipid hydroperoxides and hydrogen peroxide, converting them into harmless water and alcohol. What makes GPX4 unique among antioxidant enzymes is its reliance on selenium. Selenium is incorporated into GPX4 as the amino acid selenocysteine, critical for the enzyme's catalytic activity.

Without sufficient selenium, GPX4's function is compromised, leaving cells vulnerable to oxidative damage. GPX4's activity is also tightly linked to glutathione, the cell's master antioxidant. When glutathione levels are low, GPX4's ability to combat oxidative stress diminishes.

Enhancers:

Selenium | N-acetylcysteine (NAC)

SNP

rs713041

Alleles

C: Typical GPX4 activity

T: Altered GPX4 activity

Your Genotype

TC

Your genotype is linked to altered GPX4 activity, altered antioxidant protection and higher selenium needs

Intro and Health Effects

The main GPX4 variant is rs713041. Its "T" allele may be linked to:

- Colorectal cancer [R]
- Stroke [R]
- Poor prognosis of breast cancer [R]
- Pancreas inflammation [R]
- Vitamin B deficiencies [R]

However, some studies did not confirm the above findings [R, R].

Some studies even found protective associations between this variant and:

- High blood pressure in pregnancy [R]
- DNA damage [R]
- Heart problems in people with diabetes [R]
- Endometriosis [R]
- Alzheimer's disease [R]

Scientists are unsure about the reason behind these conflicting findings. This variant seems to be **beneficial when enough dietary selenium is available**, so people carrying it should pay special attention to selenium intake [R].

GSR

GSR Report

The GSR gene encodes a member of the class-I pyridine nucleotide-disulfide oxidoreductase family called ‘glutathione-disulfide reductase’.

This is a central enzyme of cellular antioxidant defense by reducing oxidized glutathione disulfide (GSSG) to the sulfhydryl form GSH, commonly referred to as glutathione. Maintaining a low GSSG/GSH ratio in the cell is key to maintaining oxidative balance [R].

GSH binds directly to reactive oxygen species and neutralizes them, thereby preventing them from causing damage to cells [R, R, R].

Enhancers:

Riboflavin | Alpha-lipoic acid

SNP	Your Genotype
rs1002149	GG
Alleles **G:** Typical GSR activity **T:** Altered GSR activity	**Your genotype is linked to typical GSR activity, typical glutathione recycling, nerve health & alcohol detox**

Intro and Health Effects

The main GSR gene variant is rs1002149. Its minor ‘T’ allele has been associated with an increased risk of noise-induced hearing loss and COPD [R, R].

Moreover, carriers of this variant may further increase their risk of breast cancer if they drink alcohol [R].

MPO

MPO Report

The MPO gene encodes myeloperoxidase, a peroxidase enzyme most abundant in the azurophilic granules of the neutrophils. The enzyme produces hypohalous acids from hydrogen peroxide and chloride anions during the respiratory burst [R, R].

Excessive MPO activity may increase the production of pro-oxidant components and contribute to oxidative stress [R, R].

SNP

rs2333227

Alleles

C: Typical MPO activity

T: Reduced MPO activity

Your Genotype

CC

Your genotype is linked to typical MPO activity and typical production of harmful pro-oxidants

Intro and Health Effects

The main *MPO* variant is **rs2333227**. Its minor 'T' allele disrupts the binding site of a protein that stimulates gene expression (the SP1 transcription factor), resulting in decreased *MPO* expression and reduced oxidative stress [R].

This allele has been associated with a decreased risk of:

- Bladder cancer [R]
- Cervical cancer [R, R]
- Aggressive prostate cancer. However, carriers may have an increased risk if they are smokers with low serum α-tocopherol or omega-3 fatty acid levels [R, R, R]
- Hypertension (in women only) [R]
- Coronary artery disease [R, R]
- Multiple sclerosis [R]
- Chromosome damage from benzene exposure [R]

However, this allele has been associated with an increased risk and worse prognosis of colorectal cancer, as well as with an increased risk of gestational diabetes [R, R].

NFE2L2

NFE2L2 Report

The NFE2L2 gene is responsible for encoding a protein called NRF2, which plays a major role in your body's detoxification process. More specifically, NRF2 is responsible for activating many of your other genes that produce detox proteins [R].

It does this by binding to an area of other genes called the "antioxidant response element" (ARE), which increases gene expression [R, R].

According to some researchers, impaired NRF2 could potentially lead to several conditions, such as liver disease, Parkinson's, and cancer [R].

Enhancers:

Sulforaphane EGCG (green tea)

SNP

rs6721961

Alleles

G: Typical NFE2L2 activity and detox ability

T: Reduced NFE2L2 activity and detox ability

Your Genotype

GG

Your genotype is linked to typical NFE2L2 activity and antioxidant & detox ability.

Intro and Health Effects

The "T" allele at rs6721961 is one of the NFE2L2 variants that can reduce the expression and activity of NRF2. Reduced NRF2 impairs the body's ability to detox and defend itself from oxidative stress [R, R].

NFE2L2 gene variants may be linked to:

- Liver damage due to alcohol and other toxins [R, R]
- Drug and arsenic toxicity [R, R]
- Parkinson's disease (mixed evidence!) [R, R, R]
- Cancer [R, R, R, R, R, R]

SNP

rs35652124

Alleles

C: Typical NFE2L2 activity

T: Reduced NFE2L2 activity

Your Genotype

↓TT

Your genotype is linked to reduced NFE2L2 activity and reduced ability to boost antioxidant genes

Intro and Health Effects

Research has identified several variations in the NFE2L2 gene that can reduce the expression and activity of NRF2. This may impair the body's ability to detox and defend itself from oxidative stress [R, R].

NFE2L2 gene variants like **rs35652124-T** may be linked to:

- Liver damage due to alcohol and other toxins [R, R]
- Drug and arsenic toxicity [R, R]
- Parkinson's disease (mixed evidence!) [R, R, R]
- Cancer [R, R, R, R, R, R]

SNP

rs1806649

Alleles

G: Typical NFE2L2 activity

T: Reduced NFE2L2 activity

Your Genotype

↓CC

Your genotype is linked to reduced NFE2L2 activity and reduced ability to boost antioxidant genes

Intro and Health Effects

Research has identified several variations in the NFE2L2 gene that can reduce the expression and activity of NRF2. This may impair the body's ability to detox and defend itself from oxidative stress [R, R].

NFE2L2 gene variants like **rs1806649-C** may be linked to:

- Liver damage due to alcohol and other toxins [R, R]
- Drug and arsenic toxicity [R, R]
- Parkinson's disease (mixed evidence!) [R, R, R]
- Cancer [R, R, R, R, R, R]

NOS3

NOS3 Report

The NOS3 gene codes for a protein also called NOS3, short for nitric oxide synthase 3. NOS3 is most abundant in the inner lining of the blood vessels, where it produces nitric oxide (NO) from the amino acid arginine [R, R].

NO is a messenger molecule with multiple functions in the immune and nervous systems. Additionally, NOS3-derived NO increases blood flow and lowers the risk of heart disease [R, R].

Enhancers:

Folate

SNP	Your Genotype
rs1799983	↓TT
Alleles **G:** Increased NOS3 activity **T:** Reduced NOS3 activity	**Your genotype is linked to reduced NOS3 activity, reduced nitric oxide production and worse heart health**

Intro and Health Effects

Among the different *NOS3* polymorphisms, rs1799983 has been most widely studied. The 'T' variant produces a protein that can't reach its activation sites in cell membranes, ultimately decreasing NO production [R].

In line with the beneficial cardiovascular effects of NO, the 'T' variant has been associated with an increased risk of coronary heart disease and heart attack in several studies. It's also more frequent in children with congenital heart disease and predicts a faster progression of heart damage in people with diabetes [R, R, R, R].

However, overproducing the 'GG' genotype may also have negative effects on the heart. It's associated with reduced heart function in people with kidney disease, increased risk of death in those with high blood pressure, and heart failure in African-Brazilians [R, R, R].

SNP

rs2070744

Alleles

C: Reduced NOS3 activity

T: Increased NOS3 activity

Your Genotype

CT

Your genotype is linked to typical NOS3 activity, typical nitric oxide production and heart health

Intro and Health Effects

Another SNP, rs2070744, is also linked to an increased risk of coronary heart disease. The ‘C’ allele can be bound by a protein that blocks *NOS3* production. However, the ‘T’ variant at this polymorphism is the one associated with myocardial infarction [R, R, R].

These variants may exert their harmful effects through their associations with:

- Higher blood pressure [R, R, R, R, R, R, R, R, R, R, R]
- Higher vessel stiffness and blood cholesterol [R, R, R, R]
- Increased risk of complications after heart surgery [R, R, R]
- Reduced effectiveness of conventional and alternative therapies [R, R]

SNP

rs1549758

Alleles

C: Increased NOS3 activity

T: Reduced NOS3 activity

Your Genotype

TC

Your genotype is linked to typical NOS3 activity, typical nitric oxide production and heart health

Intro and Health Effects

The ‘T’ allele of rs1549758 has also been associated with an increased risk of coronary heart disease and hypertension, but is usually inherited with the ‘T’ allele of rs1799983, meaning you will most likely have both or neither of them [R].

CAT

CAT Report

The CAT gene encodes subunits of catalase, a key antioxidant enzyme in the body's defense against oxidative stress. Four identical subunits, each attached to an iron-containing molecule called a heme group, form the functional catalase enzyme [R].

Catalase is active in cells and tissues throughout the body, where it breaks down hydrogen peroxide (H2O2) molecules into oxygen (O2) and water (H2O). Hydrogen peroxide acts as a signaling molecule, but high levels can be toxic because they lead to the formation of reactive oxygen species [R].

SNP	Your Genotype
rs7943316 Alleles **A:** Increased CAT activity **T:** Reduced CAT activity	AT **Your genotype is linked to neutral CAT activity and typical antioxidant protection**

Intro and Health Effects

Finally, the minor 'T' allele of rs7943316 (-21A>T) may lower CAT expression and has been associated with [R, R]:

- Increased risk of vitiligo [R]
- Increased risk of keratoconus [R]
- Increased risk of depression [R]
- Increased risk of breast cancer [R]
- Increased risk of male infertility [R]
- Increased risk of noise-induced hearing loss [R, R]
- Higher systolic blood pressure [R]

SNP	Your Genotype
rs1001179 Alleles **C:** Reduced CAT activity **T:** Increased CAT activity	CT **Your genotype is linked to neutral CAT activity, balanced antioxidant protection, immunity & metabolic health**

Intro and Health Effects

The best-characterized CAT polymorphism is rs1001179, commonly referred to as -262G>A. Its minor ‘T’ allele may increase CAT expression but decrease it in people with conditions such as type 2 diabetes or chronic hepatitis C. This variant has been associated with an increased risk of [R, R, R, R, R]:

- Type 2 diabetes [R]
- Prostate cancer [R, R]
- Breast cancer, especially in women using HRT or eating low amounts of fish [R, R, R]
- Aggressive chronic lymphocytic leukemia [R]
- Cerebral palsy [R]
- NAFLD and NASH [R, R]
- COVID-19 hospitalization [R]
- Liver scarring in hepatitis C patients [R]
- Malaria [R]

However, this variant has also been associated with:

- Lower oxidative stress [R]
- Higher folate and lower homocysteine levels [R]
- Reduced risk of glaucoma [R]
- Reduced risk of male infertility [R]
- Reduced risk of Crohn’s disease [R]
- Reduced risk of acoustic neuroma [R]
- Reduced risk of bronchiolitis [R]
- Later onset of Wilson’s disease [R]

SNP

rs769217

Alleles

C: Reduced CAT activity

T: Increased CAT activity

Your Genotype

↓ CC

Your genotype is linked to reduced CAT activity, reduced antioxidant protection, but lower risk of liver problems

Intro and Health Effects

Another well-researched polymorphism is rs769217, commonly referred to as -1167C>T. Its minor ‘T’ allele may increase CAT expression. This variant has been associated with [R]:

- Lower oxidative stress [R]
- Reduced risk of steroid-dependent nephrotic syndrome [R]

However, the variant has also been associated with an increased risk of

- Chronic hepatitis B, liver cirrhosis, and hepatocellular carcinoma [R]
- Noise-induced hearing loss [R]

GPX1

GPX1 Report

The GPX1 gene helps make glutathione peroxidase (GPx), one of the body's key antioxidant enzymes. This enzyme converts hydrogen peroxide and glutathione into glutathione disulfide and water. By doing so, GPx helps reduce oxidative stress [R].

Increased oxidative stress opens the door for DNA damage and cell death, which in turn can lead to tissue damage and a range of health conditions [R, R, R].

Glutathione is sometimes called the "mother of all antioxidants" because of how essential it is for the body's antioxidant defenses. It plays a central role in the function of GPx [R, R].

Enhancers:

Selenium | N-acetylcysteine (NAC)

SNP

rs1050450

Alleles

A: Lower GPX1 activity

G: Higher GPX1 activity

Your Genotype

↑GG

Your genotype is linked to higher GPX1 activity, better antioxidant protection, and longevity

Intro and Health Effects

One study found a direct link between a common GPX1 variant and human longevity. According to a cohort of elderly Danish people born in 1905, the heterozygous **genotype 'AG' at rs1050450 was significantly more common in the very elderly** than in the general population [R].

Other studies have strongly suggested that the **'G' allele at rs1050450 confers higher GPx activity**, which is linked to better health outcomes [R, R].

Along with other variants (like rs1800668 and rs3811699), this variant has also been linked to [R, R, R, R, R, R, R]:

- Rheumatoid arthritis
- Kashin-Beck disease
- Heart disease
- Some types of cancer

Kashin-Beck disease (KBD) is a bone disease that causes arthritis-like joint pain. People with KBD tend to have significantly higher oxidative stress and significantly lower selenium, suggesting that the disease could be caused (at least in part) by poor GPx activity [R, R].

GSTP1

GSTP1 Report

The GSTP1 gene codes for glutathione S-transferase pi 1 (GSTP1). GSTP1 is a phase II detoxification enzyme. It helps eliminate foreign chemicals (xenobiotics) from our bodies using the “master antioxidant” glutathione [R, R].

GSTP1 neutralizes harmful substances and makes them more water-soluble, facilitating their excretion from the body. The effectiveness of this detoxification process is influenced by various factors, including genetic polymorphisms in the GSTP1 gene.

GSTP1 is predominantly expressed in the liver but is also found in other tissues like the lungs, kidneys, and intestines. It is the most abundant GST subtype in the lungs, where it helps metabolize many carcinogenic compounds. GSTP1 also helps combat oxidative stress and inflammation [R, R, R].

Enhancers:

N-acetylcysteine (NAC) Sulforaphane

SNP	Your Genotype
rs1695	↑AA
Alleles **A:** Increased GSTP1 activity **G:** Reduced GSTP1 activity	**Your genotype is linked to increased GSTP1 activity, Increased ability to detox heavy metals, pollution, and other toxins**

Intro and Health Effects

The main GSTP1 gene variant is **rs1695 or Ile105Val**. The “**G**” allele of this variant changes the GSTP1 structure and reduces its activity. As a result, it may impact the body's ability to detoxify various substrates, including carcinogens, drugs, and products of oxidative stress.

Studies have linked it to:

- Increased drug toxicity (chemotherapy) [R]
- Increased mercury toxicity [R]
- Higher odds of asthma due to smoke exposure (“GG” genotype) [R]
- Higher odds of breast cancer [R]
- Allergic reactions in people exposed to air pollution [R]

However, some studies **failed to confirm** the link between this variant and asthma, mercury toxicity, or cancer [R, R, R, R].

The effects of rs1695-G on breast cancer may be more pronounced in women who eat less **cruciferous vegetables**. This finding makes sense given that cruciferous vegetables are rich in glutathione and other antioxidants [R].

SNP	Your Genotype
rs1138272	↑ CC
Alleles **C:** Increased GSTP1 activity **T:** Reduced GSTP1 activity	**Your genotype is linked to increased GSTP1 activity, increased ability to detox heavy metals, pollution, and other toxins**

Intro and Health Effects

Another important GSTP1 variant is **rs1138272 or Ala114Val**. Its minor "**T**" allele may be linked to:

- Stronger effects of smoking on Parkinson's disease [R]
- Increased mercury toxicity [R]
- Nerve problems [R]

However, many studies **didn't find the negative effects** of this variant on detox ability and cancer [R, R, R, R, R].

HMOX1

HMOX1 Report

The HMOX1 gene encodes an enzyme called ‘heme oxygenase’ that plays an essential role in heme metabolism. Specifically, this enzyme helps degrade heme into biliverdin, iron, and carbon monoxide. Biliverdin is then converted to bilirubin by biliverdin reductase [R].

Heme oxygenase occurs as 2 isozymes: an inducible isozyme (HO-1) encoded by the HMOX1 gene and a constitutive form (HO-2) encoded by HMOX2. HO-1 is induced by its substrate heme and by other substances such as reactive oxygen species [R].

Enhancers:

Sulforaphane Curcumin

SNP	Your Genotype
rs2071746 Alleles **A:** Increased HMOX1 activity **T:** Reduced HMOX1 activity	↑AA **Your genotype is linked to increased HMOX1 activity and increased clearance of pro-oxidant heme**

Intro and Health Effects

The main *HMOX1* polymorphism is **rs2071746**. Its minor ‘A’ allele may increase HO-1 production. This variant has been associated with a decreased risk of [R]:

- Atherosclerotic stroke [R]
- Alzheimer’s disease [R, R]
- Essential tremor [R]
- Restless legs syndrome [R]
- Sickle cell disease [R]
- Kidney complications [R]

However, this variant has been associated with an increased risk of Parkinson’s disease. This may be because increased HO-1 activity may contribute to iron buildup [R].

NQO1

NQO1 Report

The NQO1 gene encodes an enzyme also named NQO1, which is short for NADPH dehydrogenase quinone 1. The main responsibility of this enzyme is to detoxify quinones [R, R].

Quinones are a family of chemical compounds that can cause oxidative stress, cellular injury, and DNA damage. These negative effects can play a role in the development of cancer and other health conditions [R, R].

NQO1 helps detox quinones by converting them into hydroquinone, a much safer compound that generally does not cause oxidative stress. Other functions of NQO1 include converting ubiquinone (CoQ10) and vitamin E into their antioxidant forms [R].

Enhancers:

Sulforaphane Curcumin

SNP

rs1800566

Alleles

A: Reduced NQO1 activity

G: Increased NQO1 activity

Your Genotype

↑GG

Your genotype is linked to increased NQO1 activity, improved CoQ10 regeneration, antioxidant defense, and quinone detox

Intro and Health Effects

Certain NQO1 gene variants may impair the body's ability to detox by reducing the activity of this enzyme [R, R].

NQO1 gene variants may play a role in DNA damage. Research has found their associations with different types of cancer [R, R, R, R, R].

One of the main culprits is a variant called **NQO1*2**, which corresponds to rs1800566-A. People with this variant have reduced NQO1 activity [R, R].

Research shows that people with the NQO1*2 variant (rs1800566-A) are up to **7.6 times more likely to experience benzene toxicity**, even from low-level exposure [R, R, R].

SNP

rs1131341

Alleles

A: Reduced NQO1 activity

G: Increased NQO1 activity

Your Genotype

↑GG

Your genotype is linked to increased NQO1 activity, improved CoQ10 regeneration, antioxidant defense, and quinone detox

Intro and Health Effects

Another important variant is **NQO1*3**, which corresponds to rs1131341-A. It leads to a slower conversion of quinone into its safer metabolites [R].

UCP2

UCP2 Report

The UCP2 gene encodes a protein called ‘uncoupling protein 2’. In the mitochondria (the main powerhouse of cells), the breakdown of nutrient fuels is coupled to the production of the energy molecule ATP. Members of the uncoupling protein family prevent the production of this molecule and instead cause the energy to be dissipated as heat [R, R].

UCP2 is found in a wide variety of tissues, such as brown and white fat, muscles, pancreatic insulin-producing cells, the immune system, the heart, and the liver. Its expression is increased when blood fatty acid levels are high, such as during fat burning or when eating a high-fat diet. Conversely, obese and diabetic people have lower UCP2 levels [R, R, R, R, R, R].

UCP2 may also protect the brain and other tissues from oxidative stress [R, R, R, R, R].

Enhancers:

Quercetin

Resveratrol

SNP

rs659366

Alleles

C: Reduced UCP2 activity

T: Increased UCP2 activity

Your Genotype

TC

Your genotype is linked to balanced UCP2 activity, balanced fat burning and blood sugar control

Intro and Health Effects

The minor ‘**T’ variant of the rs659366 increases UCP2 expression** in the fatty tissue [R, R, R].

This allele showed a protective effect from obesity in multiple studies [R, R, R, R, R, R, R, R].

However, many studies failed to confirm these results or even found the opposite ones [R, R, R, R, R, R, R, R, R, R, R, R, R].

Despite these discrepancies, **all meta-analyses agree that the ‘T’ variant is associated with lower obesity rates and BMI in European** but not in Asian populations [R, R, R, R].

On the other hand, this variant may have **negative effects on blood lipids, glucose control, and diabetes outcomes** [R, R, R, R, R, R, R, R, R, R].

Lab markers to check

ALT — 63 U/L — 4 May 2026

Personalized to Your Genes

↓ GSTA1

GSTA1 is liver-concentrated; GGT in particular doubles as an oxidative-stress and glutathione-turnover indicator.

ApoB — 132 mg/dL — 13 Jan 2026

Blood Pressure (Diastolic) — 94 mmHg — 25 Mar 2026

Personalized to Your Genes

↓ SOD3

Extracellular superoxide scavenges vascular NO; blood pressure serves as a functional readout of the resulting endothelial impact.

○ NOS3

Reduced NO impairs vasodilation; blood pressure is the most accessible functional readout of NOS3-driven endothelial function.

Blood Pressure (Systolic) — 148 mmHg — 25 Mar 2026

Personalized to Your Genes

↓ SOD3

Extracellular superoxide scavenges vascular NO; blood pressure serves as a functional readout of the resulting endothelial impact.

NOS3

Reduced NO impairs vasodilation; blood pressure is the most accessible functional readout of NOS3-driven endothelial function.

Folate 120 nmol/L

31 Jan 2026

Personalized to Your Genes

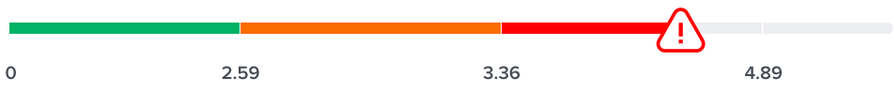
↓ MTHFR

Confirms whether substrate and cofactor status is adequate to support the impaired methylation pathway.

Hemoglobin A1c 40 mmol/mol

25 Mar 2026

LDL Cholesterol 4.4 mmol/L



25 Mar 2026

Personalized to Your Genes

↓ PON1

PON1 is HDL-associated; assessing HDL quantity and the broader lipid profile reveals the atherogenic risk from impaired oxidized-lipid protection.

LDL Particle Number 2064 nmol/L

13 Jan 2026

GGT 46 IU/L

25 Mar 2026

Personalized to Your Genes

↓ GSTA1

GSTA1 is liver-concentrated; GGT in particular doubles as an oxidative-stress and glutathione-turnover indicator.

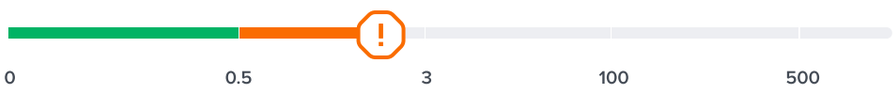
Homocysteine 12.84 umol/L

9 Nov 2025

Personalized to Your Genes

↓ MTHFR — The direct, defining marker of MTHFR dysfunction and a pro-oxidant/endothelial stressor to track and lower.

hs-CRP 2.41 mg/L



13 Jan 2026

Personalized to Your Genes

NFE2L2 — Reduced NRF2 raises oxidative-inflammatory tone; CRP reflects the systemic inflammatory burden that results.

NOS3 — Reflects the vascular oxidative-inflammatory tone that accompanies low NO and elevated endothelial superoxide.

Insulin, Fasting 8.7 uIU/mL

13 Jan 2026

Iron 69 ug/dL

13 Jan 2026

Personalized to Your Genes

GPX4 — GPX4 guards against iron-driven lipid peroxidation; monitoring iron avoids the overload that stresses the enzyme.

CAT — Confirms iron status is adequate (heme cofactor) but not in excess, which would add pro-oxidant burden to a weak catalase system.

Triglycerides 1.53 mmol/L

25 Mar 2026

Personalized to Your Genes

↓ MTHFR	Methylation feeds the transsulfuration route to glutathione; a low ratio reveals how much antioxidant capacity the MTHFR bottleneck is costing.
↓ GCLC	Directly measures the glutathione deficit that results from impaired synthesis, the central consequence of low GCLC.
↓ GSTA1	GSTA1 conjugation depletes glutathione; the ratio reveals whether detox demand is exhausting the reserve.
↓ PON1	PON1 is HDL-associated; assessing HDL quantity and the broader lipid profile reveals the atherogenic risk from impaired oxidized-lipid protection.
SOD2	Downstream antioxidant systems must compensate for weak SOD2; a falling ratio signals the reserve is being exhausted.
NFE2L2	NRF2 governs glutathione synthesis; a low reduced:oxidized ratio reveals depletion of the body's master antioxidant.
CAT	Reveals whether the compensating glutathione peroxidase system is keeping up or being depleted.

Bilirubin, Total	0.5 mg/dL	0.2 – 0.8 – 1.3 – 15	12 Jun 2026
Copper, Serum	94 ug/dL	70 – 105 – 140	13 Jan 2026

Personalized to Your Genes

↓ SOD3	Confirms adequate cofactor status for the SOD3 enzyme, the most correctable input to its activity.

Ferritin	130 ng/mL	30 – 50 – 150 – 380	13 Jan 2026

Personalized to Your Genes

GPX4

GPX4 guards against iron-driven lipid peroxidation; monitoring iron avoids the overload that stresses the enzyme.

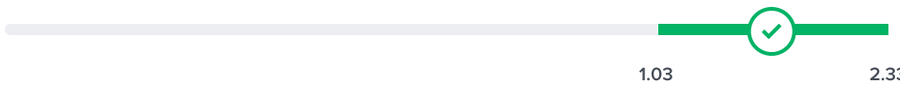
CAT

Confirms iron status is adequate (heme cofactor) but not in excess, which would add pro-oxidant burden to a weak catalase system.

Glucose, Fasting 81 mg/dL

12 Jun 2026

HDL Cholesterol 1.65 mmol/L



25 Mar 2026

Personalized to Your Genes

↓ PON1

PON1 is HDL-associated; assessing HDL quantity and the broader lipid profile reveals the atherogenic risk from impaired oxidized-lipid protection.

Magnesium 2.17 mg/dL

9 Nov 2025

Magnesium, RBC 5.7 mg/dL

13 Jan 2026

TIBC 379 mcg/dL

9 Nov 2025

Personalized to Your Genes

GPX4

GPX4 guards against iron-driven lipid peroxidation; monitoring iron avoids the overload that stresses the enzyme.

CAT

Confirms iron status is adequate (heme cofactor) but not in excess, which would add pro-oxidant burden to a weak catalase system.

Vitamin B12 414 pg/mL

13 Jan 2026

Personalized to Your Genes

↓ MTHFR — Confirms whether substrate and cofactor status is adequate to support the impaired methylation pathway.

Zinc 17.12 umol/L

9 Nov 2025

Personalized to Your Genes

↓ SOD3 — Confirms adequate cofactor status for the SOD3 enzyme, the most correctable input to its activity.

8-Hydroxy-2-deoxyguanosine, Urine

Personalized to Your Genes

↓ GCLC — Quantifies the oxidative damage that accumulates when glutathione production can't keep pace with demand.

↓ GSTA1 — GSTA1 clears lipid-peroxidation products; elevated markers reflect the oxidative burden left by impaired conjugation.

SOD2 — Directly quantifies the mitochondrial superoxide-driven oxidative damage that reduced SOD2 fails to neutralize.

↓ SOD3 — Directly quantifies the systemic and vascular oxidative burden left by impaired extracellular superoxide clearance.

NFE2L2 — Quantifies the oxidative damage that accumulates when the NRF2-driven antioxidant response is blunted.

NOS3 — An uncoupled NOS3 generates superoxide; these markers capture the resulting oxidative burden.

CAT — Directly quantifies the oxidative damage accumulating from reduced hydrogen-peroxide clearance.

ADMA (Asymmetric dimethylarginine)

Personalized to Your Genes

NOS3

ADMA competitively inhibits NOS3's use of L-arginine; elevated levels directly explain reduced NO output and guide whether arginine/citrulline support is warranted.

Arsenic, Blood

Arsenic, Random Urine

C-Peptide

C-Reactive Protein (CRP)

Personalized to Your Genes

NFE2L2

Reduced NRF2 raises oxidative-inflammatory tone; CRP reflects the systemic inflammatory burden that results.

NOS3

Reflects the vascular oxidative-inflammatory tone that accompanies low NO and elevated endothelial superoxide.

Coenzyme Q10

Epinephrine, Random Urine

F2-Isoprostanes

Personalized to Your Genes

↓ GCLC	Quantifies the oxidative damage that accumulates when glutathione production can't keep pace with demand.
↓ GSTA1	GSTA1 clears lipid-peroxidation products; elevated markers reflect the oxidative burden left by impaired conjugation.
○ SOD2	Directly quantifies the mitochondrial superoxide-driven oxidative damage that reduced SOD2 fails to neutralize.
↓ SOD3	Directly quantifies the systemic and vascular oxidative burden left by impaired extracellular superoxide clearance.
○ GPX4	Directly reflects the membrane lipid oxidative damage that GPX4 is uniquely responsible for controlling.
○ NFE2L2	Quantifies the oxidative damage that accumulates when the NRF2-driven antioxidant response is blunted.
○ NOS3	An uncoupled NOS3 generates superoxide; these markers capture the resulting oxidative burden.
○ CAT	Directly quantifies the oxidative damage accumulating from reduced hydrogen-peroxide clearance.

Free Fatty Acids

HOMA-IR

Lead, Blood

Lead, Urine

Malondialdehyde

Personalized to Your Genes

GPX4

Directly reflects the membrane lipid oxidative damage that GPX4 is uniquely responsible for controlling.

Manganese, Blood

Personalized to Your Genes

SOD2

Confirms adequate cofactor status for the SOD2 enzyme, the single most correctable input to its activity.

Mercury, Blood

Personalized to Your Genes

↓ GCLC

Given the impaired mercury-detox capacity, checking body burden identifies whether accumulation is occurring.

Mercury, Random Urine

Norepinephrine, Random Urine

OxLDL

Personalized to Your Genes

↓ PON1

PON1's core job is preventing lipid oxidation; elevated oxLDL directly reflects the consequence of reduced PON1 activity.

SAH, Serum

SAM-e, Serum

Selenium

Total Glutathione

Personalized to Your Genes

GPX4

Given the elevated selenium requirement, this is the most important and directly correctable marker for this variant.

Vanillylmandelic Acid (VMA), Random Urine

Vitamin B2 (Riboflavin), Plasma

Glossary

8-OHdG (8-Hydroxy-2'-Deoxyguanosine)
A marker of oxidative damage to DNA, often used to assess oxidative stress.

Bilirubin
A breakdown product of heme that also acts as an antioxidant, helping protect cells from oxidative damage.

CoQ10 (Coenzyme Q10)
A compound that helps mitochondria produce energy and protects cells from oxidative damage.

CRP (C-Reactive Protein)
A marker of inflammation that can indicate increased inflammatory activity in the body.

F2-Isoprostanes
Compounds formed when oxidative stress damages fats. They are commonly used as a marker of lipid peroxidation.

GGT (Gamma-Glutamyl Transferase)
An enzyme involved in glutathione metabolism. Elevated levels can indicate increased oxidative stress or liver stress.

Glutathione
A major antioxidant that helps neutralize reactive compounds and protect cells from oxidative damage.

H2O2 (Hydrogen Peroxide)
A reactive oxygen species produced during normal metabolism that can cause oxidative damage if it accumulates.

Heme
An iron-containing molecule found in hemoglobin and other proteins. Its breakdown produces bilirubin and can contribute to oxidative reactions.

HOCl (Hypochlorous Acid)
A highly reactive compound produced by immune cells to destroy pathogens. Excessive amounts can contribute to oxidative damage.

Homocysteine
An amino acid produced during methionine metabolism. High levels can promote oxidative stress and inflammation.

Lipid Alcohols
Less reactive products formed when lipid peroxides are reduced, helping limit further oxidative damage.

Lipid Peroxides
Damaged fats formed when reactive oxygen species attack cell membranes and other lipids.

MDA (Malondialdehyde)
A byproduct of lipid peroxidation and a commonly used marker of oxidative damage.

Methylation
A chemical process that helps regulate gene activity and supports the metabolism of compounds such as homocysteine.

Mitochondria
Structures inside cells that produce energy and are also a major source of reactive oxygen species.

MPO (Myeloperoxidase)
An enzyme in immune cells that produces HOCl to help fight infections, but can also contribute to oxidative damage.

OxLDL (Oxidized LDL)
LDL cholesterol damaged by oxidation, which can promote inflammation and contribute to cardiovascular disease.

Superoxide O_2
A reactive oxygen species produced during normal metabolism that can contribute to oxidative stress and form other reactive compounds.