UGENOME

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Toxins & Detox

Summary Report

REPORT CATEGORY

DETOX

Sample Client

Report date: 30 April 2026

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Personal information

NAME

Sample Client

SEX AT BIRTH

Male

HEIGHT

5ft 10" 178cm

WEIGHT

215lb 97.5kg

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.

Viewing this medical test requires a medical doctor or use one of our contracted genetic counselors. By accessing these results, you acknowledge and agree that you will consult with a licensed physician or one of our contracted genetic counselors to review and interpret the results, and you agree not to rely on this information as a substitute for professional medical advice, diagnosis, or treatment.

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Summary

The impact of environmental toxins on health is an increasingly important topic in personalized health and functional medicine. Our genetic makeup plays a significant role in how our bodies process, detoxify, and respond to various toxins.

This report delves into the genetic predispositions that influence individual responses to a range of harmful substances, including **mold, heavy metals, pesticides, plastics, air pollution**, and more. Understanding these genetic factors can help tailor strategies for **minimizing exposure and supporting detoxification processes**.

By examining how genetic variations affect toxin metabolism and sensitivity, this report aims to provide actionable insights for optimizing health and reducing the risk of **toxin-related adverse effects**. This knowledge empowers individuals to make informed decisions about their environment and lifestyle, promoting a proactive approach to health and well-being.

This summary report contains:

58 Genetic Results

15 Recommendations

Overview of Your Results

Pesticides

TYPICAL

Pesticide Sensitivity

Likely typical pesticide sensitivity

TYPICAL

Organophosphate Sensitivity

Likely typical organophosphate sensitivity

TYPICAL

Glyphosate Sensitivity

Likely typical glyphosate sensitivity

Heavy Metals

HIGHER LEVELS

Aluminum

Predisposed to higher aluminum levels

TYPICAL LEVELS

Lead

Predisposed to typical levels of lead

TYPICAL LEVELS

Mercury

Predisposed to typical mercury levels

TYPICAL LEVELS

Arsenic

Predisposed to typical levels of arsenic

TYPICAL LEVELS

Cadmium

Predisposed to typical levels of cadmium

Air Pollution

TYPICAL

Air Pollution Sensitivity

Likely typical air pollution sensitivity

Plastics

TYPICAL

BPA Sensitivity

Likely typical BPA sensitivity

TYPICAL

Phthalate Sensitivity

Likely typical phthalate sensitivity

Mold

WORSE GENETICS

ITGB3 (Mold Sensitivity)

Likely worse ITGB3 genetics

LOWER ACTIVITY

CHIT1 (Mold Sensitivity)

Likely lower CHT1 activity

LOWER ACTIVITY

GSTA1 (Detox/ Glutathione)

Predisposed to lower GSTA1 activity

TYPICAL

Sensitivity to Airborne Mold

Likely typical sensitivity to airborne mold

TYPICAL

Sensitivity to Foodborne Mold

Likely typical sensitivity to foodborne mold

TYPICAL ACTIVITY

GSDMB (Mold Sensitivity)

Likely typical GSDMB activity

TYPICAL ACTIVITY

XPC (DNA Damage)

Based on the genetic variants we looked at, you are predisposed to typical XPC activity. In people with these variants, toxins from mold and cigarette smoke may have a typical impact on DNA damage. However, keep in mind that other genetic and environmental factors influence DNA damage and repair.

TYPICAL ACTIVITY

XRCC (DNA Damage)

Likely typical XRCC activity

Other Pollutants

HIGHER

PBDE Sensitivity (CYP2B6)

Likely higher PBDE sensitivity

TYPICAL

Benzene Sensitivity

Predisposed to typical benzene sensitivity

TYPICAL LIKELIHOOD

Multiple Chemical Sensitivity

Typical likelihood of multiple chemical sensitivity

LOWER

PAH Sensitivity

Likely lower PAH sensitivity

Detox

LOWER ACTIVITY

PON1 (Detox)

Likely lower PON1 activity

LOWER ACTIVITY

UGT2A1 (Cognition)

Likely lower UGT2A1 activity

LOWER ABILITY

Phase I Detox

Predisposed to lower phase I detox ability

LOWER ACTIVITY

GCLC (Glutathione & Detox)

Predisposed to lower GCLC activity

LOWER ACTIVITY

SLCO1B1 (Detox)

Predisposed to lower SLCO1B1 activity

LOWER ACTIVITY

GSTA1 (Detox/ Glutathione)

Predisposed to lower GSTA1 activity

HIGHER ACTIVITY

CYP1B1 (Detox/ Skin Health)

Predisposed to higher CYP1B1 activity

TYPICAL ABILITY

Detox

Predisposed to typical detox ability

TYPICAL FUNCTION

Glutathione

Predisposed to typical glutathione function

HIGHER ACTIVITY

SULT1A1 (Detox)

Likely higher SULT1A1 activity

TYPICAL ACTIVITY

UGT1A1 (Detox)

Likely typical UGT1A1 activity

TYPICAL ACTIVITY

AHR (Detox)

Likely typical AHR activity

TYPICAL ACTIVITY

NFE2L2/NRF2 (Detox)

Likely typical NFE2L2 activity

TYPICAL ABILITY

Phase II Detox

Predisposed to typical phase II detox ability

TYPICAL ACTIVITY

CYP1A1 (Detox)

Likely typical CYP1A1 activity

TYPICAL METABOLIZER

CYP2C9 (Detox)

Likely a typical metabolizer

TYPICAL ACTIVITY

CYP3A4 (Detox)

Likely typical CYP3A4 activity

INTERMEDIATE

NAT2 (Detox)

Likely an intermediate acetylator

HIGHER ACTIVITY

NQO1 (Detox)

Likely higher NQO1 activity

HIGHER ACTIVITY

GSTP1 (Detox)

Likely higher GSTP1 activity

HIGHER ACTIVITY

GPX1 (Glutathione/Detox)

Likely higher GPX1 activity

HIGHER ACTIVITY

Sulfation (Detox)

Predisposed to higher sulfation activity

HIGHER ACTIVITY

UGT (Detox)

Likely higher UGT activity

HIGHER ACTIVITY

CYP1A2 (Detox)

Likely higher CYP1A2 activity

EXTENSIVE METABOLIZER

CYP2C19 (Detox)

Likely an extensive metabolizer

Oxidative Stress

LOWER ACTIVITY

COQ2 (Oxidative Stress)

Likely lower COQ2 activity

LOWER ACTIVITY

SOD2 (Oxidative Stress)

Likely lower SOD2 activity

WORSE

SOD3 (Oxidative Stress)

Likely worse SOD3 genetics

LOWER ACTIVITY

GCLC (Glutathione & Detox)

Predisposed to lower GCLC activity

LOWER ACTIVITY

GSTA1 (Detox/ Glutathione)

Predisposed to lower GSTA1 activity

TYPICAL

Oxidative Stress

Likely typical oxidative stress

TYPICAL

Lipid Oxidation

Likely typical lipid oxidation

TYPICAL LEVELS

DNA Damage

Predisposed to typical DNA damage

TYPICAL ACTIVITY

GPX4 (Selenium & Glutathione)

Predisposed to typical GPX4 activity

HIGHER ACTIVITY

GPX1 (Glutathione/Detox)

Likely higher GPX1 activity

Recommendations Overview

Your recommendations are prioritized according to the likelihood of it having an impact for you based on your genetics, 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	N-acetylcysteine (NAC)	1200 mg
2	Dietary Antioxidants	
3	Selenium Supplements	50 mcg
4	Dietary Polyphenols	
5	Zeolite	1 tsp
6	Vitamin C	2000 mg
7	Zinc	15 mg
8	Vitamin E	200 iu
9	Magnesium	350 mg
10	Green Tea	400 mg
11	Avoid Aluminum Exposure	
12	Glutathione supplements	
13	Sulforaphane	30 mg
14	Avoid Endocrine Disruptors	
15	Avoid PBDE	

Your Results in Details

Pesticides

Pesticides are widely used in agriculture and can be found in varying levels in our food and environment. Genetic differences can influence how individuals metabolize and respond to these chemicals. This section explores genetic predispositions to pesticide sensitivity, focusing on key compounds like organophosphates and glyphosate.

TYPICAL

Pesticide Sensitivity

Likely typical pesticide sensitivity

TYPICAL

Organophosphate Sensitivity

Likely typical organophosphate sensitivity

TYPICAL

Glyphosate Sensitivity

Likely typical glyphosate sensitivity

Pesticide Sensitivity

Key Takeaways:

- Genetic variants impacting pesticide sensitivity may involve oxidative stress, DNA repair, brain health and cognition, immune response, and vitamin D activity.
- Risk factors involve exposure to pesticides like in certain occupations or consuming produce on which pesticides get used.
- Acute pesticide poisoning is responsible for up to **300,000** deaths per year worldwide. Chronic low exposure has been linked to various health conditions.
- If you have high genetic risk, you may lower overall risk by taking steps to avoid exposure to pesticides.

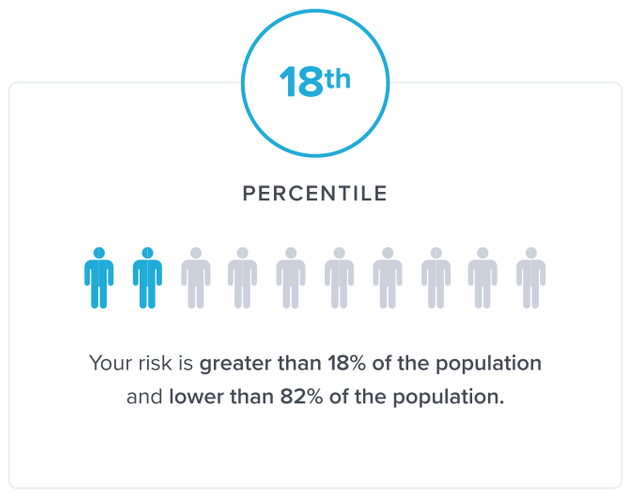
Should we all be equally worried about pesticides? Although adverse effects of chronic pesticide exposure are inevitable, some people seem to be at a much higher risk. The reason for this lies partly in genetics!
In people carrying certain gene variants, pesticides have shown a stronger link with DNA damage, Parkinson's disease, cancer, and more. These variants belong to genes that play a role in [R, R, R, R]:

- Pesticide detox (PON1, ABCB1, CYP1A1)
- Oxidative stress (SOD2, NOS1)
- DNA repair (ERCC6L2, XRCC1, XPC)
- Brain health and cognition (BCHE)
- Immune response (HLA-DQA2)
- Vitamin D activity (GC, VDR)

However, keep in mind that your other gene variants, lifestyle, and environment may also influence your pesticide sensitivity.

TYPICAL

Likely typical pesticide sensitivity based on 38 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PON1	rs662	TT
MTRNR2L13	rs6851004	AA
/	rs2279343	AG
BACH2	rs6919587	TA
NOS1	rs3741480	TC
GC	rs7041	AC
CCDC24	rs539096	GA
ABCB1	rs2032582	CT
BCHE	rs1803274	CT
OTX1	rs2710647	CT
PON1	rs854560	TA
ALOXE3	rs3027208	TC
ALOX5	rs7099684	TT
ACSL5	rs12098576	TG
ABCG8	rs4953028	GA
HLA-DQA2	rs3129882	GA
GC	rs12512631	TC
ALOX15	rs916055	GA
STAT6	rs1800159	AG
TMEM106C	rs4328262	GG
SOD2	rs4880	GG
XPC	rs2228001	GT

GENE	SNP	GENOTYPE
CASTOR1	rs2072159	AG
LDLR	rs8110695	AT
A4GALT	rs8136914	GA
UGT2B15	rs1902023	AC
XRCC1	rs1799782	GG
CAT	rs1001179	CT
CYP1A1	rs1048943	TT
ABCB1	rs1045642	GG
NAT2	rs1799931	GG
GSTP1	rs1695	AA
RPS18	rs1547387	CC
POU5F1B	rs4242382	GG
FBXO21	rs2682826	GG
PPA2	rs7679673	CC
CYP2C19	rs4244285	GG
USPL1	rs9579645	AA
ERCC2	rs1799793	CC
ADIPOR1	rs12733285	TT
MTG1	rs2031920	CC
CYP2D6	rs3892097	CC
OGG1	rs1052133	CC

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Organophosphate Sensitivity

Although adverse effects of chronic OP exposure are inevitable, some people seem to be at a much higher risk. The reason for this lies partly in genetics!

In people carrying certain gene variants, OP pesticides have shown a stronger link with DNA damage, Parkinson's disease, cancer, and more. These variants belong to genes that play a role in [R, R, R, R, R]:

- Pesticide detox (PON1, ABCB1, CYP1A1)
- Oxidative stress (NOS1)
- Brain health and cognition (BCHE)
- Vitamin D activity (GC, VDR)

However, keep in mind that your other gene variants, lifestyle, and environment may also influence your OP sensitivity.

TYPICAL

Likely typical organophosphate sensitivity based on 26 genetic variants we looked at

21st

PERCENTILE

Your risk is **greater than 21% of the population** and **lower than 79% of the population.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ABCB1	rs1045642	GG
RPS18	rs1547387	CC
POU5F1B	rs4242382	GG
FBXO21	rs2682826	GG
NOS1	rs3741480	TC
USPL1	rs9579645	AA
BCHE	rs1803274	CT
ADIPOR1	rs12733285	TT
PON1	rs854560	TA
ALOXE3	rs3027208	TC
GC	rs12512631	TC
CYP2D6	rs3892097	CC
CASTOR1	rs2072159	AG
LDLR	rs8110695	AT
A4GALT	rs8136914	GA
PON1	rs662	TT
ABCB1	rs2032582	CT
TMEM106C	rs4328262	GG

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Glyphosate Sensitivity

Key Takeaways:

- Glyphosate (RoundUp) is the most widely used weed killer in the world.
- Most people are exposed to this chemical to a certain degree.
- Many countries have banned it due to potential links with cancer and other adverse effects.
- People with certain gene variants may be more sensitive to the adverse effects of glyphosate.

Genetics might be one of the reasons for conflicting data on glyphosate side effects. People may be more or less sensitive to this chemical depending on certain gene variants they carry.

Glyphosate and other organophosphates tend to reduce the levels of an enzyme called cholinesterase. This effect may be more pronounced in people carrying the **'C' allele at rs1048943**. The authors suggest that reduced enzyme levels may be a sign of **liver damage** [R].

This variant belongs to the *CYP1A1* gene, which plays a role in pesticide detox [R].

In another study, glyphosate exposure showed a stronger link with **prostate cancer** in people carrying the **'C' allele at rs9579645**. This variant belongs to the *ALOX5AP* gene, which plays a role in fat metabolism and inflammation [R, R].

Please note: Your other gene variants, diet, and environment may also influence your glyphosate sensitivity. The available genetic research for this report was limited, so please take your results with a grain of salt.

Ways to reduce your pesticide exposure include [R, R]:

- Eating a variety of fruits and vegetables to avoid high exposure to one pesticide
- Buying organic food when possible
- Thoroughly washing and drying your food, even if it's labeled organic
- Scrubbing or peeling fruits and vegetables when possible
- Discarding the outer layer of leafy vegetables

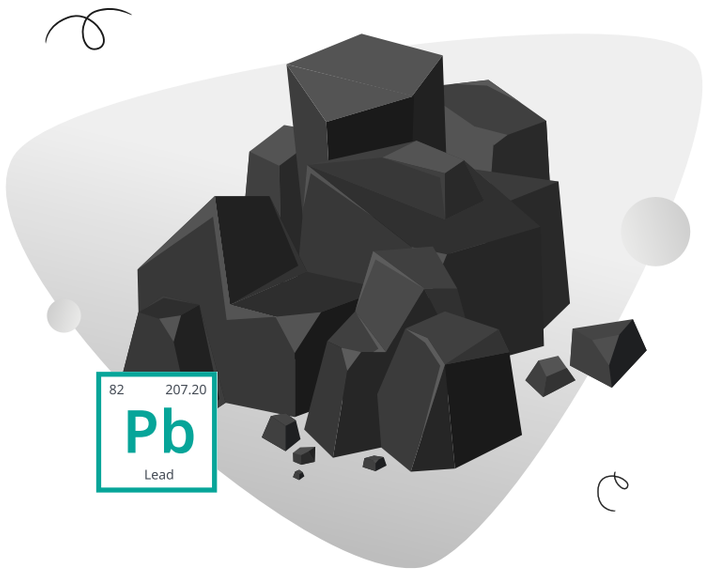
TYPICAL

Likely typical glyphosate sensitivity based on 2 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CYP1A1	rs1048943	TT
USPL1	rs9579645	AA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.



Heavy Metals

Heavy metals like aluminum, lead, mercury, arsenic, and cadmium can accumulate in the body and cause various health issues. Genetic variations can affect the body's ability to detoxify and eliminate these toxic elements.

This section analyzes genetic predispositions to heavy metal sensitivity and retention, offering insights into personalized strategies for minimizing exposure and supporting detoxification processes.

HIGHER LEVELS

Aluminum

Predisposed to higher aluminum levels

TYPICAL LEVELS

Lead

Predisposed to typical levels of lead

TYPICAL LEVELS

Mercury

Predisposed to typical mercury levels

TYPICAL LEVELS

Arsenic

Predisposed to typical levels of arsenic

TYPICAL LEVELS

Cadmium

Predisposed to typical levels of cadmium

Aluminum

A study of over 2000 Chinese individuals identified several variants associated with blood levels of aluminum and other minerals [R].

Some strategies to reduce exposure to aluminum include:

- Opting for stainless steel or glass cookware and avoiding cooking acidic foods in aluminum pans, as they can cause more aluminum to leach.
- Limiting the intake of foods and additives that contain aluminum.
- Choosing aluminum-free deodorants and cosmetics if concerned about exposure.
- Consulting with a healthcare provider about alternatives to aluminum-containing medications, especially antacids.

HIGHER LEVELS

Predisposed to higher aluminum levels based on 12,017 genetic variants we looked at

80th

PERCENTILE

Your risk is **greater than 80% of the population** and **lower than 20% of the population.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
EFR3A	rs72728663	CC
/	rs369179362	CC
TTF2	rs1289661	TT
GK2	rs62297754	TT
IL11RA	rs10814173	TG
DAPK1	rs4878080	GA
AP2B1	rs149814693	GG
EPHA4	rs146559885	TT
OSBPL11	rs4574233	CC
KCNJ6	rs2835950	TT

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Lead

Some people may be predisposed to higher levels of lead than others. This may put them at a higher risk of lead poisoning, especially when they are exposed to lead in their environment [R, R].

This may be due to genetics. In fact, about **40%** of differences in lead levels may be attributed to genetic factors [R].

Genes involved may influence [R]:

- Lead absorption and buildup
- Lead distribution in the body

Keep in mind that the environment may also influence your lead levels. The only way to determine your blood lead levels is to take a blood test.

If your levels are high, your doctor may recommend chelation therapy. This is a form of treatment that binds heavy metals and helps remove them from the body [R, R].

TYPICAL LEVELS

Predisposed to typical levels of lead based on 14 genetic variants we looked at

28th

PERCENTILE

Your risk is **greater than 28% of the population** and **lower than 72% of the population.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MTHFR	rs1801133	AA
AGTR1	rs79019069	AA
MTHFR	rs1476413	CC
SLC11A2	rs224589	GG
SRGAP3	rs76153987	CC
/	rs9863067	CC
CAPZB	rs12136530	AA
ALAD	rs1805313	AA
PEPD	rs16968074	AA
/	rs366631	AG
MAGI2	rs798338	AC
RGS5	rs2662776	GA
ALAD	rs1800435	CC
GSTP1	rs1695	AA
THSD7A	rs116864947	TT
GSTP1	rs1138272	CC
TTC26	rs60580184	GG
PTPN2	rs144653651	GG
HFE	rs1800562	GG
HFE	rs1799945	CC
BSPRY	rs10121150	AA
HDHD3	rs8177812	GG

GENE	SNP	GENOTYPE
ABO	rs550057	TT

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Mercury

The most important compound for mercury detox is the “master antioxidant” glutathione. It binds mercury, turning it into less toxic forms that can be removed from the body [R].

In line with this, genes that play a role in glutathione production and activity may affect mercury levels.

The *GCLC* gene helps make an enzyme that enables glutathione production.

One *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].

The *GSTP1* gene helps make an enzyme that enables glutathione to bind toxins. One variant in this gene, rs1138272-T, may be linked to higher mercury levels in the blood and hair [R, R, R].

BONUS CONTENT

A so-called **“null” *GSTM1* variant** may be linked to **higher mercury levels** in the blood and hair. *GSTM1* is another gene that supports glutathione function [R, R, R, R].

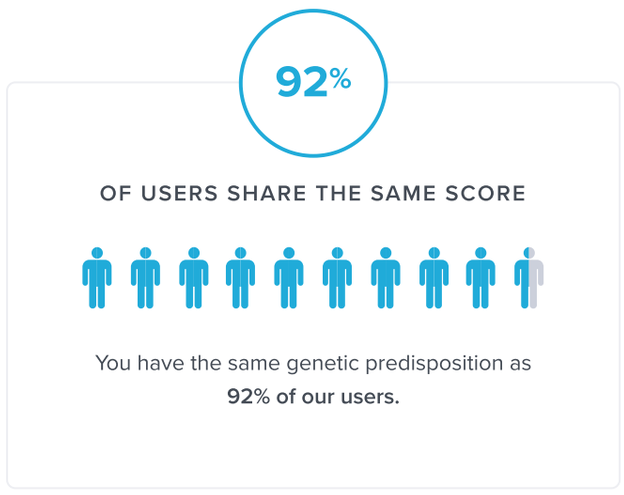
If your genotype for rs366631 is “**AA**”, you most likely carry this variant. In that case, you should pay more attention to mercury exposure [R, R, R, R].

Some people’s genetic files don’t contain this variant, so we couldn’t include it in the report. If you don’t see your genotype for this variant, **consider ordering a SelfDecode kit** to get a more complete picture about your mercury sensitivity.

Please note: *The links between gene variants and mercury levels are not fully clear, and there have been some conflicting results. Also, keep in mind that your diet and the environment may influence your mercury levels* [R, R, R, R].

TYPICAL LEVELS

Predisposed to typical mercury levels based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GCLM	rs41303970	GG
GCLC	rs761142	CA
GSTP1	rs1138272	CC

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Arsenic

Some people may be better than others at processing and removing arsenic from their bodies. Up to **60%** of these differences may be due to genetics [R].

For example, a gene called *AS3MT* helps make an enzyme that turns arsenic into its less toxic form. Certain variants in this gene are linked to enhanced arsenic metabolism. As a result, people with these variants may have lower odds of developing skin lesions due to arsenic poisoning [R, R, R].

On the other hand, some variants are linked to slower arsenic metabolism. Arsenic exposure from drinking water may have a stronger impact on skin lesions in the presence of these variants [R].

Keep in mind that the environment may also influence your arsenic levels. The only way to determine your arsenic levels is to take a blood or urine test.

If your levels are high, your doctor may recommend chelation therapy. This is a form of treatment that binds heavy metals and helps remove them from the body [R].

TYPICAL LEVELS

Predisposed to typical levels of arsenic based on 13 genetic variants we looked at

Your risk is **greater than 32% of the population** and **lower than 68% of the population.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MTHFR	rs1801133	AA
GSTO1	rs4925	AC
BORCS7	rs11191527	CC
MTHFR	rs1476413	CC
GSTO1	rs11191979	CT
GSTO1	rs2164624	AG
BORCS7	rs3740393	GC
AS3MT	rs3740390	CT
ATP5MK	rs7911488	AG
AS3MT	rs11191439	TT
GSTP1	rs1695	AA
GSTP1	rs1138272	CC

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Cadmium

Some people may be predisposed to higher levels of cadmium than others, in part due to genetic factors. This may put them at a higher risk of toxicity from cadmium exposure [R].

The genes involved may influence cadmium absorption and metabolism [R].

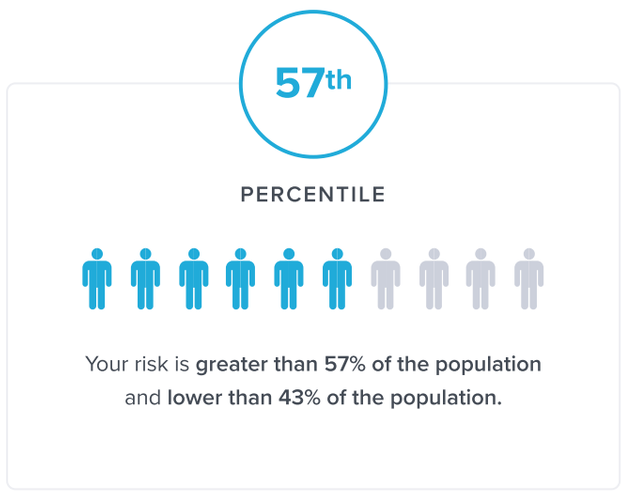
Interestingly, the effect of genetics on cadmium levels may be stronger in people who don't smoke. Cigarettes are a major source of cadmium, so they may mask subtle genetic differences [R].

Other factors such as diet and the environment may also influence your cadmium levels. The only reliable way to determine your cadmium levels is to get tested. Blood tests, urine tests, and more can be used to determine your cadmium levels. [R].

If your cadmium levels are high, your doctor may recommend chelation therapy. This is a form of treatment that binds heavy metals and helps remove them from the body [R, R].

TYPICAL LEVELS

Predisposed to typical levels of cadmium based on 9 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
/	rs12228069	GG
MLLT3	rs76878079	GG
THBS1	rs166722	AA
PUM3	rs10813093	TC
USP24	rs396511	CC
WDR72	rs79052248	AA
HRH4	rs17204045	GG
HRH4	rs880423	TT

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Air Pollution

Air pollution is a growing concern, particularly in urban areas, and can have significant health impacts. Sensitivity to air pollutants can vary based on genetic makeup, influencing the body's response to substances like particulate matter and chemical pollutants.

This section examines your genetic predisposition to air pollution sensitivity, providing a deeper understanding of how genetics can affect respiratory health and overall well-being.

TYPICAL

Air Pollution Sensitivity

Likely typical air pollution sensitivity

Air Pollution Sensitivity

Not everyone reacts the same to air pollution. The reason for this may partly be in genetics. In people with certain gene variants, air pollution may have stronger effects on [R, R, R, R, R, R, R]:

- Asthma (*TGFB1*, *TLR1*, *TLR2*)
- Lung function (*SERPINA1*, *XPC*)
- Heart health (*MTHFR*)
- Cognitive function (*APOE*)
- Blood sugar (*GSTP1*)

Keep in mind that your other gene variants and environment may also influence your air pollution sensitivity.

TYPICAL

Likely typical air pollution sensitivity based on 27 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MAFB	rs7361259	TT
ARSA	rs6151412	GG
CTLA4	rs11571319	GA
CDC14C	rs13309098	GG
HSP90B1	rs2070908	GG
STAT4	rs1031509	TG
IL1F10	rs6743376	CC
CXCL12	rs1619661	CT
MTHFR	rs1801133	AA
STK10	rs9313579	CC
CYP2E1	rs2070673	AT
FERD3L	rs6950598	GG
COBL	rs11761214	AC
SERPINA1	rs17580	TT
CTLA4	rs11571316	GG
TLR4	rs10759931	AA
APOE	rs429358	TT
FGD4	rs12312730	GG
TLR2	rs4696480	TT
TLR4	rs2770150	AA
SNX16	rs10504754	GG
ERBB4	rs6725041	CC
GSTP1	rs1695	AA
OR4A5	rs10902298	CC
/	rs4971775	TT

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Plastics

Plastics are ubiquitous in modern life, and compounds like BPA and phthalates, commonly found in plastics, can disrupt endocrine function and contribute to health issues. Genetic factors play a role in determining how individuals metabolize and react to these compounds.

This section focuses on genetic predispositions to BPA and phthalate sensitivity, offering insights into how these chemicals can affect hormone balance and overall health.

TYPICAL

BPA Sensitivity

Likely typical BPA sensitivity

TYPICAL

Phthalate Sensitivity

Likely typical phthalate sensitivity

BPA Sensitivity

Key Takeaways:

- We are all exposed to BPA from plastic bottles, food packages, and more.
- BPA is a well-known hormone disruptor.
- Research has linked BPA exposure to heart disease, diabetes, altered behavior, and more.
- People with certain gene variants may be more sensitive to the adverse effects of BPA.

Although we are all exposed to BPA, it won't affect everyone the same. The reason for this is partly in our genes, which can make us more or less susceptible to harmful effects of BPA.

In people with certain gene variants, BPA has shown a stronger potential link with:

- Liver damage [R, R]
- Asthma [R]
- Cancer [R, R, R]
- High blood pressure [R]
- PCOS [R]

These variants affect the genes involved in:

- BPA detox (*UGT2B7*)
- Antioxidant protection (*CAT*, *SOD2*, *GSTP1*)
- DNA repair (*PARP4*, *XRCC3*, *RAD51*, *ERCC5*)
- Blood vessel function (*NOS1*)
- Hormone metabolism (*CYP17A1*, *ESR1*, *ESR2*)

Genes such as UGT1A1 and *UGT2B7* help produce crucial enzymes for BPA detox. Read this post for more information about UGT gene variants and plastic detox.

Please note: Your other gene variants, diet, lifestyle, and environment can also affect BPA sensitivity.

TYPICAL

Likely typical BPA sensitivity based on 22 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PTGS2	rs5277	CC
GSTP1	rs1695	AA
XRCC3	rs12432907	CC
ERCC5	rs2296147	CC
CAT	rs10488736	CC
PARP4	rs3783073	GG
ESR2	rs1256049	CC
RMDN3	rs2412547	AA
UGT2B7	rs7439366	CT
GCHFR	rs2304579	AA
XRCC3	rs861537	TT
NOS3	rs1549758	TC
XRCC3	rs861531	CC
PARP4	rs2275660	CT
CCDC170	rs2046210	GA
BORCS7	rs743572	AG
CAT	rs769217	CC
SOD2	rs4880	GG
ERCC5	rs17655	GG
CAT	rs4755374	AA
TMEM176A	rs11771443	CC
ESR2	rs4986938	CC

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Phthalate Sensitivity

In the body, phthalates are mainly broken down in the liver and partly in the gut. Phthalate breakdown takes place in several steps. The enzyme encoded by the *CYP2C9* gene allows their oxidation into secondary metabolites. Certain variants of this gene, such as ‘T’ at rs1799853, ‘C’ at rs1057910, and ‘C’ at rs12248560, have been associated with lower urinary levels of secondary metabolites, suggesting increased phthalate sensitivity [R, R].

Most of these secondary metabolites undergo a reaction called conjugation to allow their elimination with urine. A family of enzymes called UDP-glucuronyl transferases is responsible for this reaction. For instance, a variant of the *UGT1A7* gene (‘T’ at rs11692021) has been associated with decreased phthalate breakdown [R].

Some strategies to reduce exposure to phthalates include:

- Choosing phthalate-free products: Look for phthalate-free labels on toys, personal care products, and food packaging.
- Avoiding plastic containers for food and drink: Use glass, stainless steel, or BPA- and phthalate-free plastics, especially for hot food and liquids.
- Ventilation: Ensure good ventilation in the home to reduce indoor air exposure.
- Read labels: Be cautious with products that list "fragrance" or "perfume" as these can contain phthalates.

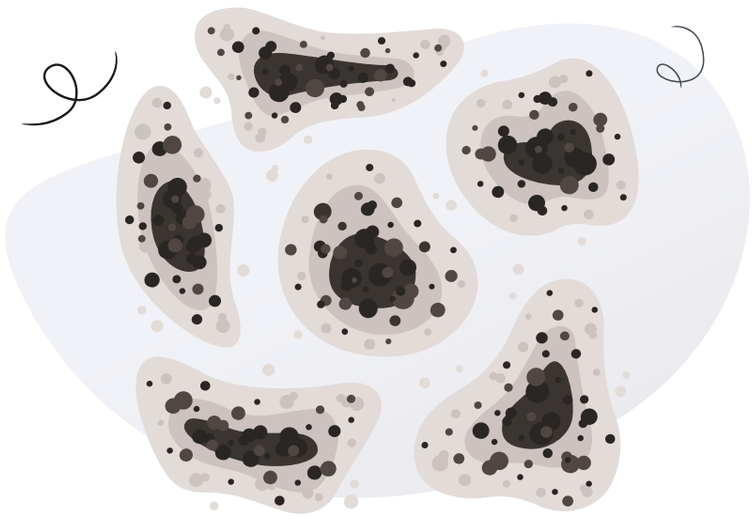
TYPICAL

Likely typical phthalate sensitivity based on 4 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NOC3L	rs12248560	CC
UGT1A7	rs11692021	TT
CYP2C9	rs1799853	CC
CYP2C9	rs1057910	AA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.



Mold

Mold exposure can occur through airborne spores or contaminated food and can lead to various health problems, particularly for those with genetic predispositions. Sensitivity to mold toxins, or mycotoxins, can vary widely among individuals.

This section examines genetic markers associated with mold sensitivity, both airborne and foodborne.

WORSE GENETICS

ITGB3 (Mold Sensitivity)

Likely worse ITGB3 genetics

LOWER ACTIVITY

CHIT1 (Mold Sensitivity)

Likely lower CHT1 activity

LOWER ACTIVITY

GSTA1 (Detox/ Glutathione)

Predisposed to lower GSTA1 activity

TYPICAL

Sensitivity to Airborne Mold

Likely typical sensitivity to airborne mold

TYPICAL

Sensitivity to Foodborne Mold

Likely typical sensitivity to foodborne mold

TYPICAL ACTIVITY

GSDMB (Mold Sensitivity)

Likely typical GSDMB activity

TYPICAL ACTIVITY

XPC (DNA Damage)

Based on the genetic variants we looked at, you are predisposed to typical XPC activity. In people with these variants, toxins from mold and cigarette smoke may have a typical impact on DNA damage. However, keep in mind that other genetic and environmental factors influence DNA damage and repair.

TYPICAL ACTIVITY

XRCC (DNA Damage)

Likely typical XRCC activity

ITGB3 (Mold Sensitivity)

A study of 1243 participants associated the ‘A’ allele of rs2056131 with a decreased sensitization to molds in people with asthma. Moreover, the following alleles were associated with mold sensitization in people with asthma and the ‘TT’ genotype at *TLR2*/+596 [R]:

- ‘T’ of rs4525555
- ‘T’ of rs10514919
- ‘C’ of rs8074094
- ‘A’ of rs2015729
- ‘T’ of rs2292699
- ‘C’ of rs15908
- ‘G’ of rs2292863
- ‘C’ of rs3809863
- ‘T’ of rs11650072
- ‘C’ of rs11079772

WORSE GENETICS

Likely worse ITGB3 genetics based on 11 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ITGB3	rs2056131	GG
ITGB3	rs2015729	AA
EFCAB13	rs2292699	TT
ITGB3	rs15908	CC
TBKBP1	rs8074094	CC
EFCAB13	rs4525555	TT
TBKBP1	rs10514919	TT
TBKBP1	rs2292863	GG
EFCAB13	rs3809863	TC
EFCAB13	rs11650072	CT
EFCAB13	rs11079772	AC

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

CHIT1 (Mold Sensitivity)

A study including 395 children and their parents associated the following *CHIT1* variants with severe asthma exacerbations upon exposure to high levels of airborne molds [R]:

- 'C' of rs2486953
- 'G' of rs4950936
- 'C' of rs1417149

These alleles may decrease *CHIT1* activity. Because these alleles are usually inherited together, you will most likely carry either all or none of them. Interestingly, these variants have also been linked to reduced lung infection [R].

LOWER ACTIVITY

Likely lower CHT1 activity based on the genetic variants we looked at

72%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **72% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CHIT1	rs2486953	CT
CHIT1	rs4950936	GA
CHIT1	rs1417149	CT

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

GSTA1 (Detox/ Glutathione)

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

Another GSTA1 variant, rs3957356, has shown similar associations. These two variants are almost always inherited together, meaning that you likely have either none or both of them.

LOWER ACTIVITY

Predisposed to lower GSTA1 activity based on the genetic variants we looked at

20%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **20% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GSTA1	rs3957357	AA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Sensitivity To Airborne Mold

In people with mold sensitivity, the immune system overreacts to inhaled mold. A reaction can cause unpleasant symptoms such as **coughing and itchy eyes** [R].

People with asthma are more prone to mold sensitivity. In some of them, mold exposure can disrupt breathing and trigger asthma attacks [R].

Other risk factors for mold sensitivity include [R]:

- Having a family history of allergies
- Occupational exposure to mold (e.g., in farming, baking, millwork, winemaking)
- Living in a house with high humidity (>50%) or poor ventilation
- Working or living in a building exposed to excess moisture

Genetics may also influence sensitivity to airborne mold. Involved genes may play a role in [R, R, R, R]:

- Immune response to mold (*ITGB3*)
- Breakdown of fungal cells (*CHIT1*)
- Inflammation (*GSDMB*)
- Skin barrier function (*CLDN1*)

In individuals with asthma, variants in some of these genes may be linked to higher mold sensitivity and worse asthma symptoms [R, R, R]:.

Please keep in mind two **important limitations**:

- It's not sure whether these gene variants affect people who don't have asthma
- The number of available gene variants is low, which may affect the accuracy of this report

If you are sensitive to mold, do your best to reduce your exposure to the types of mold that cause your reaction. Consult your doctor about potential treatment options if your symptoms are persistent or severe.

TYPICAL

Likely typical sensitivity to airborne mold based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ITGB3	rs2056131	GG
GSDMB	rs7216389	TC
CHIT1	rs2486953	CT
CLDN16	rs9290929	AG
/	rs366631	AG
CYP1A2	rs762551	AA
SCAMP5	rs2069526	TT
LMAN1L	rs2069514	GG

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Sensitivity To Foodborne Mold

Mycotoxins are the most dangerous aspect of foodborne mold. Exposure to higher amounts of these toxins can damage the organs and promote cancer growth [R, R, R].

The ability of our bodies to deal with mycotoxins partly depends on genetics. Involved genes play a role in [R, R, R, R, R]:

- Mycotoxin metabolism and detox (*CYP1A2*)
- DNA repair (*XPC*, *XRCC4*, *ATXN3*)
- Cancer protection (ADAMTS5)
- Mycotoxin transport (*SLCO1B1*)
- Glutathione function (*GSTA1*)

People with certain variants in these genes may be more prone to negative effects of mycotoxins, including [R, R, R, R, R]:

- Liver cancer
- Liver damage
- Kidney damage

Remember that your other gene variants, diet, and environment also influence your sensitivity to foodborne mold.

TYPICAL

Likely typical sensitivity to foodborne mold based on 14 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
XPC	rs2228001	GT
PRKDC	rs7003908	CA
ADAMTS5	rs2830581	AG
GSTA1	rs3957357	AA
SLCO1B1	rs4149056	TC
XRCC3	rs861539	GG
XRCC4	rs28383151	GG
CYP1A2	rs762551	AA
XRCC4	rs3734091	GG
XRCC1	rs25487	CC
ERCC2	rs13181	TT
ATXN3	rs8021276	AA
SCAMP5	rs2069526	TT
LMAN1L	rs2069514	GG

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

GSDMB (Mold Sensitivity)

A study of 645 asthmatic and 910 non-asthmatic children associated the ‘T’ allele of rs7216389 with increased risk of asthma, especially in those exposed to airborne molds. This polymorphism is believed to increase *ORMDL3* expression, leading to increased production of inflammatory cytokines. Other studies have confirmed the link of this polymorphism with asthma and allergic rhinitis [R, R, R, R, R].

In line with this, engineered mice lacking *ORMDL* were protected against asthma and showed a decrease in lung and airway changes induced by molds (*Alternaria*) [R].

TYPICAL ACTIVITY

Likely typical GSDMB activity based on the genetic variants we looked at

51%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **51% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GSDMB	rs7216389	TC

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

XPC (DNA Damage)

The main *XPC* variant is **rs2228001**. The **G allele** carriers may experience [R, R, R, R]:

- Increased susceptibility to **DNA damage** from mold toxins
- Higher rates of **liver cancer** due to mold (aflatoxin) exposure

Many studies have found a link between this variant and cancer, especially liver and lung cancer [R, R].

The link between this variant and cancer may be stronger in people who **smoke or use tobacco** products [R].

It may reduce the ability of XPC to **repair DNA damage** caused by mold, cigarette smoke, and other stressors.

However, some studies have found the opposite association between this variant and cancer [R, R].

Another well-researched *XPC* variant is **rs2228000**. People with the **A allele** may have slightly higher odds of cancer, especially **bladder cancer**. This link tends to be stronger in **smokers** [R, R].

TYPICAL ACTIVITY

Based on the genetic variants we looked at, you are predisposed to typical XPC activity. In people with these variants, toxins from mold and cigarette smoke may have a typical impact on DNA damage. However, keep in mind that other genetic and environmental factors influence DNA damage and repair. based on 2 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
XPC	rs2228001	GT
XPC	rs2228000	GG

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

XRCC (DNA Damage)

Certain genetic variations in *XRCC* genes reduce its ability to repair DNA damage caused by mold toxins, potentially increasing the risk of liver cancer in people exposed to high levels.

For instance, a study of 1486 patients with hepatocellular carcinoma and 1996 controls exposed to the foodborne mold toxin aflatoxin B1 identified the following variants as increasing the risk [R]:

- 'T' at *XRCC1* rs25487
- 'A' at *XRCC3* rs861539
- 'A' at *XRCC4* rs28383151
- 'C' at *XRCC7* rs7003908

The association of the rs25487 polymorphism with increased risk of hepatocellular carcinoma and DNA damage in response to aflatoxins was confirmed in other studies. This variant has also been associated with [R, R]:

- Brain cancer [R, R]
- Cervical cancer [R, R]
- Pancreatic cancer [R]
- Breast cancer [R]
- Bladder cancer [R]
- Prostate cancer [R]
- Thyroid cancer [R]
- Age-related cataracts [R]
- Worse outcome in lung cancer patients [R]
- Worse response to platinum chemotherapy [R, R, R]

The association of rs861539 with aflatoxin-related hepatocellular carcinoma was confirmed in two more studies. This variant has also been associated with [R, R]:

- Ovarian cancer [R, R]
- Brain cancer [R, R]
- Oral cancer [R]
- Nasopharyngeal cancer [R]
- Stomach cancer [R]
- Cervical cancer [R]
- Bladder cancer [R]
- Osteosarcoma [R]
- Leukemia [R]
- Lupus [R]
- Radiation-induced adverse effects [R]

Another study confirmed the relationship between the rs7003908 polymorphism and aflatoxin-related hepatocellular carcinoma. This variant has also been associated with [R]:

- Prostate cancer [R]
- Brain cancer [R]
- Esophageal cancer [R]
- Bladder cancer [R]

Finally, a study of 1499 liver cancer cases and 2045 controls associated the 'G' allele of *XRCC4* rs3734091 with a slightly increased risk of hepatocellular carcinoma due to aflatoxin B1 exposure and confirmed the association of rs28383151 with this disease. The rs3734091 variant has also been associated with [R]:

- Nasopharyngeal cancer [R]
- Breast cancer [R]
- Acute lymphocytic leukemia [R]
- Oral cancer [R]

TYPICAL ACTIVITY

Likely typical XRCC activity based on 5 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PRKDC	rs7003908	CA
XRCC4	rs3734091	GG
XRCC3	rs861539	GG
XRCC4	rs28383151	GG
XRCC1	rs25487	CC

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Other Pollutants

Environmental pollutants like polycyclic aromatic hydrocarbons (PAHs) and polybrominated diphenyl ethers (PBDEs) can accumulate in the body, potentially leading to health issues. Sensitivity to these compounds can be influenced by genetic factors.

This section explores genetic predispositions related to pollutants like PAH and PBDE sensitivity, highlighting the importance of understanding how your body responds to them.

HIGHER

PBDE Sensitivity (CYP2B6)

Likely higher PBDE sensitivity

TYPICAL

Benzene Sensitivity

Predisposed to typical benzene sensitivity

TYPICAL LIKELIHOOD

Multiple Chemical Sensitivity

Typical likelihood of multiple chemical sensitivity

LOWER

PAH Sensitivity

Likely lower PAH sensitivity

PBDE Sensitivity (CYP2B6)

The CYP2B6 enzyme is the main one responsible for breaking down several PBDEs such as PBDE-47, PBDE-99, and PBDE-100 in the human body [R].

The change from a 'G' to a 'T' at **rs3745274** encodes the *CYP2B6*6* variant, which has **markedly reduced enzyme activity**. This variant is very common, especially in Africans, Asians, and Hispanics. A study has linked it to a higher persistence of PBDEs in the blood and breast milk [R].

Some strategies to reduce exposure to PBDEs include:

- Regularly cleaning and vacuuming homes to reduce dust that may contain PBDEs.
- Properly disposing of electronics and furniture that may contain PBDEs.
- Limiting the consumption of high-fat dairy products and certain fish known to accumulate PBDEs.
- Choosing products labeled as free from PBDEs or opting for naturally flame-resistant materials.

This variant is also linked to a slower metabolism of multiple drugs such as methadone, efavirenz, bupropion, cyclophosphamide, sertraline, and ketamine [R, R, R, R, R, R, R].

Likely higher PBDE sensitivity based on the genetic variants we looked at

41%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **41% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CYP2B6	rs3745274	GT

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Benzene Sensitivity

Some gene variants may affect benzene sensitivity by influencing benzene detoxification and DNA damage repair.

The *NQO1* gene encodes an enzyme called NAD(P)H quinone oxidoreductase 1. NQO1 is an important antioxidant enzyme involved in fighting oxidative stress, DNA damage, and benzene toxicity. The 'A' allele of rs1800566 (NQO1*2) reduces enzyme activity and has been associated with **up to 7.6 times higher risk of benzene toxicity**, even from low-level exposure [R, R, R, R, R, R].

The *XRCC1* gene encodes a protein called X-ray repair cross-complementing protein 1 and involved in DNA repair. The 'T' allele of rs25487 and the 'A' allele of rs1799782 have been associated with chronic benzene poisoning, as well as with chromosome aberrations in people exposed to this chemical [R, R, R, R].

The *ERCC1* gene encodes another enzyme involved in DNA repair. The 'A' allele of rs11615 has been associated with chronic benzene poisoning, especially in non-smokers, women, and those exposed for over 12 years [R].

The *EPHX1* gene encodes an enzyme that converts epoxides from the degradation of aromatic compounds to trans-dihydrodiols, which can be conjugated and excreted from the body. The major 'T' allele of rs1051740 has been associated with chronic benzene poisoning, as well as with decreased telomere length and increased DNA damage in people exposed to this chemical [R, R, R].

The *GSTP1* gene codes for a phase II detox enzyme that helps eliminate toxins using the "master antioxidant" glutathione. The 'A' allele of rs1695 has been associated with chronic benzene poisoning, especially in people who drink alcohol [R].

The *MPO* gene encodes myeloperoxidase, an enzyme that produces toxic molecules that harm bacteria. However, these molecules also damage the body's tissues. The minor 'T' allele of rs2333227 has been associated with decreased odds of chromosome aberrations and bladder cancer in response to benzene exposure [R, R].

The *CYP2D6* gene encodes one of the cytochrome P450 monooxygenases (CYPs). This enzyme accounts for only 2-5 % of the liver CYPs but metabolizes 25% of all drugs. The 'G' allele of rs1065852 has been linked to chronic benzene poisoning [R, R, R, R].

Another cytochrome P450 monooxygenase, CYP2E1, is involved in the detoxification of drugs, toxic chemicals, and environmental toxins. The 'T' allele of rs2031920 and the 'C' allele of rs3813867 have been associated with DNA damage and low white blood cell counts in people chronically exposed to benzene [R, R, R].

CYP1A1 encodes another cytochrome enzyme. The 'T' allele of rs4646903 has been associated with chronic benzene poisoning [R].

The low-activity *UGT1A6* variants 'G' at rs6759892 and 'C' at rs1105879 have been associated with chronic benzene poisoning [R].

The *CDKN2A* gene encodes several proteins, which includes p16(INK4a) and the p14(ARF) proteins. Both function as tumor suppressors and help regulate the cell cycle. The 'CC' genotype of rs3731245 has been associated with chronic benzene poisoning [R, R].

The *TNF* gene encodes a cytokine (TNF-alpha or cachexin) that plays a central role in the immune response and inflammation. The 'A' allele of rs1800629 is associated with 6-7 times higher

TYPICAL

Predisposed to typical benzene sensitivity based on 14 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GSTP1	rs1695	AA
EPHX1	rs1051740	TT
MPO	rs2333227	CC
ERCC1	rs11615	AA
CDKN2A	rs3731245	CC
CYP2D6	rs1065852	GG
NQO1	rs1800566	GG
XRCC1	rs25487	CC
XRCC1	rs1799782	GG
UGT1A6	rs6759892	TT
SYCE1	rs3813867	GG
MTG1	rs2031920	CC
TNF	rs1800629	GG
UGT1A6	rs1105879	AA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

levels of TNF-alpha and has been linked to chronic benzene poisoning [R].

Multiple Chemical Sensitivity

While the exact causes of MCS remain unclear, researchers believe that a combination of environmental and genetic factors can contribute to its development.

Emerging research suggests that genetics may influence an individual's susceptibility to MCS. Though the genetics of MCS are not fully understood, several factors may contribute to the condition:

Variants in the genes encoding different **CYP enzymes** can greatly influence phase I detox ability. Examples include *CYP1A1*, *CYP2E1*, *CYP1B1*, *CYP2A6*, CYP2B6 *and* *CYP2D6*. Variants in these genes are linked to:

- Harmful effects of cigarette smoke [R, R, R, R, R]
- Pesticide sensitivity [R, R, R]
- Air pollution sensitivity [R]

A variant in the *CYP1A2* gene may impair **caffeine detox** and contribute to its adverse effects [R].

The *GSTP1* gene codes for a phase II detox enzyme that helps eliminate toxins using the "master antioxidant" glutathione. Studies have linked its variants to harmful effects of **air pollution, cigarette smoke, mercury**, and more [R, R, R, R, R].

The UGT enzymes encoded by genes like *UGT1A1* and *UGT2A1* are also vital for phase II detox. They help produce glutathione and remove toxins found in **plastics, cigarette smoke**, and more [R, R].

Other genes that help make glutathione and support its detox function include *GCLC*, *GSTA1* and *GPX1*. Variants in these genes may influence the detox of **mercury, mold**, and more [R, R, R, R].

The *NAT2* codes for another major enzyme in phase II detox. Due to the variants in this gene, people can be **"slow acetylators"**, which means they may have a harder time detoxing **cigarette smoke and some chemicals and drugs** [R, R, R, R].

While the exact genetic and environmental mechanisms of MCS remain an area of ongoing research, it is clear that a complex interplay between genetic, immune, and environmental factors contributes to the development and severity of the condition.

TYPICAL LIKELIHOOD

Typical likelihood of multiple chemical sensitivity based on 63 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PON1	rs662	TT
SOD2	rs4880	GG
XPC	rs2228001	GT
UGT2A1	rs10518065	GA
ADH1B	rs1229984	CT
NFE2L2	rs35652124	TT
MTHFR	rs1801133	AA
CYP1B1	rs1056836	GG
NAT2	rs1495741	AG
PON1	rs854560	TA
GCLC	rs761142	CA
CYP1B1	rs1800440	CT
GSTO2	rs156697	GA
SLCO1B1	rs4149056	TC
BCHE	rs1803274	CT
GSTA1	rs3957357	AA
COMT	rs4680	GA
BORCS7	rs743572	AG
UGT2B15	rs1902023	AC
CAT	rs1001179	CT
XRCC1	rs1799782	GG
PTGS2	rs5277	CC
/	rs12228069	GG
MLLT3	rs76878079	GG
MTG1	rs2031920	CC
EGLN2	rs28399433	AA
/	rs2279343	AG
CYP2E1	rs2070673	AT
GSDMB	rs7216389	TC
CSK	rs2606345	AC

GENE	SNP	GENOTYPE
UGT2B7	rs7439366	CT
GSTP1	rs1695	AA
ALDH2	rs671	GG
GSTP1	rs1138272	CC
CYP1A1	rs1048943	TT
COX15	rs717620	CC
NFE2L2	rs6721961	GG
NAT1	rs4986782	GG
SLC6A1	rs41293373	GG
CYP2D6	rs3892097	CC
/	rs369692318	TT
TRIM4	rs2740574	TT
SCAMP5	rs2069526	TT
NQO1	rs1800566	GG
NQO1	rs1131341	GG
EPHX1	rs1051740	TT
GPX1	rs1050450	GG
CYP1A2	rs762551	AA
UGT1A6	rs6742078	GG
CYP1A1	rs4646903	AA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

PAH Sensitivity

The *NAT2* gene encodes a key enzyme in the breakdown of PAHs. A variant of this gene ('G' at rs1208) has been associated with decreased elimination of secondary metabolites with urine, suggesting increased PAH sensitivity [R].

Some strategies to reduce exposure to PAHs include:

- Avoiding cigarette smoke, both active and secondhand.
- Limiting consumption of charred or burnt food. Instead, opt for gentler cooking methods like boiling, baking, or steaming.
- Being aware of air quality in industrial areas and using air purifiers to reduce indoor air pollution.
- Using appropriate protective equipment and following safety guidelines to reduce occupational exposure to PAHs.

LOWER

Likely lower PAH sensitivity based on the genetic variants we looked at

79%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **79% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NAT2	rs1208	AA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Detox

The body's ability to detoxify and eliminate harmful substances is crucial for maintaining health, particularly when dealing with environmental toxins. Genetic variations can significantly influence detoxification pathways, affecting how efficiently the body processes and removes toxins like heavy metals, pesticides, and pollutants.

This section provides an overview of detoxification mechanisms and focuses on specific genes involved in these processes.

LOWER ACTIVITY

PON1 (Detox)

Likely lower PON1 activity

LOWER ACTIVITY

UGT2A1 (Cognition)

Likely lower UGT2A1 activity

LOWER ABILITY

Phase I Detox

Predisposed to lower phase I detox ability

LOWER ACTIVITY

GCLC (Glutathione & Detox)

Predisposed to lower GCLC activity

LOWER ACTIVITY

SLCO1B1 (Detox)

Predisposed to lower SLCO1B1 activity

LOWER ACTIVITY

GSTA1 (Detox/ Glutathione)

Predisposed to lower GSTA1 activity

HIGHER ACTIVITY

CYP1B1 (Detox/ Skin Health)

Predisposed to higher CYP1B1 activity

TYPICAL ABILITY

Detox

Predisposed to typical detox ability

TYPICAL FUNCTION

Glutathione

Predisposed to typical glutathione function

HIGHER ACTIVITY

SULT1A1 (Detox)

Likely higher SULT1A1 activity

TYPICAL ACTIVITY

UGT1A1 (Detox)

Likely typical UGT1A1 activity

TYPICAL ACTIVITY

AHR (Detox)

Likely typical AHR activity

TYPICAL ACTIVITY

NFE2L2/NRF2 (Detox)

Likely typical NFE2L2 activity

TYPICAL ABILITY

Phase II Detox

Predisposed to typical phase II detox ability

TYPICAL ACTIVITY

CYP1A1 (Detox)

Likely typical CYP1A1 activity

TYPICAL METABOLIZER

CYP2C9 (Detox)

Likely a typical metabolizer

TYPICAL ACTIVITY

CYP3A4 (Detox)

Likely typical CYP3A4 activity

INTERMEDIATE

NAT2 (Detox)

Likely an intermediate acetylator

HIGHER ACTIVITY

NQO1 (Detox)

Likely higher NQO1 activity

HIGHER ACTIVITY

GSTP1 (Detox)

Likely higher GSTP1 activity

HIGHER ACTIVITY

GPX1 (Glutathione/Detox)

Likely higher GPX1 activity

HIGHER ACTIVITY

Sulfation (Detox)

Predisposed to higher sulfation activity

HIGHER ACTIVITY

UGT (Detox)

Likely higher UGT activity

HIGHER ACTIVITY

CYP1A2 (Detox)

Likely higher CYP1A2 activity

EXTENSIVE METABOLIZER

CYP2C19 (Detox)

Likely an extensive metabolizer

PON1 (Detox)

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].

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

LOWER ACTIVITY

Likely lower PON1 activity based on the genetic variants we looked at

62%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **62% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PON1	rs662	TT
PON1	rs854560	TA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

UGT2A1 (Cognition)

A large-scale study with over 3,000 participants found that the minor 'G' allele of the rs10518065 polymorphism was significantly associated with overall intelligence levels, as measured by a large and diverse range of cognitive tests. People with the 'A' allele performed better across all of these tests, whereas people with the rarer 'G' allele tended to show relatively poorer cognitive performance [R].

Because increased oxidative stress has been associated with a wide variety of cognitive impairments in many different human populations, this strongly suggests that **the negative effects of the 'G' allele are likely due to impaired antioxidant protection** [R, R].

LOWER ACTIVITY

Likely lower UGT2A1 activity based on the genetic variants we looked at

27%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **27% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
UGT2A1	rs10518065	GA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Phase I Detox

Some people might have genetics that make crucial detox components work differently. Recognizing that each of us might detoxify differently because of our unique genetic makeup is an important step towards personalized healthcare and keeping ourselves healthy.

Variants in the genes encoding different **CYP enzymes** can greatly influence phase I detox ability. Examples include *CYP1A1*, *CYP2E1*, *CYP1B1*, *CYP2A6*, CYP2B6 *and* *CYP2D6*. Variants in these genes are linked to:

- Harmful effects of cigarette smoke [R, R, R, R, R]
- Pesticide sensitivity [R, R, R]
- Air pollution sensitivity [R]

A variant in the *CYP1A2* gene may impair **caffeine detox** and contribute to its adverse effects [R].

Alcohol detox is crucial to minimize its side effects, especially if consumed in higher amounts. Two genes, *ADH1B* and *ALDH2*, help make enzymes that process alcohol, and their variants can greatly affect detox potential [R].

Another phase I enzyme, *PON1*, helps by detoxing **pesticides (organophosphates)** and other toxins [R, R].

Finally, *NQO1* is involved in fighting oxidative stress, DNA damage, and **benzene** toxicity [R, R, R].

LOWER ABILITY

Predisposed to lower phase I detox ability based on 32 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
UGT2A1	rs10518065	GA
ADH1B	rs1229984	CT
CYP1B1	rs1056836	GG
BORCS7	rs743572	AG
CYP1B1	rs1800440	CT
CYP1B1	rs10012	GC
CYP2D6	rs16947	AG
EGLN2	rs28399433	AA
/	rs2279343	AG
CYP2E1	rs2070673	AT
CSK	rs2606345	AC
CSK	rs2472304	AG
CYP2E1	rs2480256	AG
CYP2E1	rs1329149	TC
CYP2E1	rs2070676	GC
ALDH2	rs671	GG
CYP1A1	rs1048943	TT
NAT1	rs4986782	GG
CYP2D6	rs3892097	CC
TRIM4	rs2740574	TT
EPHX1	rs1051740	TT
CYP1A2	rs762551	AA
CYP1A1	rs4646903	AA
CYP2C19	rs4244285	GG
CYP2A6	rs1801272	AA
CYP1A1	rs1799814	GG
CYP2D6	rs1065852	GG
SYCE1	rs8192772	TT
CYP2E1	rs6413419	GG
XRCC4	rs28383151	GG

GENE	SNP	GENOTYPE
CYP2C9	rs1799853	CC
NOC3L	rs12248560	CC
CYP2C9	rs1057910	AA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

GCLC (Glutathione & Detox)

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]

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].

LOWER ACTIVITY

Predisposed to lower GCLC activity based on the genetic variants we looked at

16%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **16% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GCLC	rs17883901	AG
GCLC	rs761142	CA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

SLCO1B1 (Detox)

The ‘C’ allele of rs4149056 (T521C, also called SLCO1B1*5) consists of a change from valine to alanine at residue 174 and results in a protein with **reduced transport activity**, leading to the buildup in the blood of drugs transported by this protein [R, R].

A study of 475 participants with liver damage and 475 healthy controls identified this variant as a risk factor for **liver damage** among those exposed to high **mold toxin** (aflatoxin B1) levels. The link between this variant and **higher bilirubin** levels may put additional strain on the liver [R, R].

This variant may also reduce the protein's ability to transport **statins** into the liver. As a result, more of the medication stays in the bloodstream, which can increase the risk of side effects like **statin-induced muscle pain** [R, R, R, R, R, R, R, R, R].

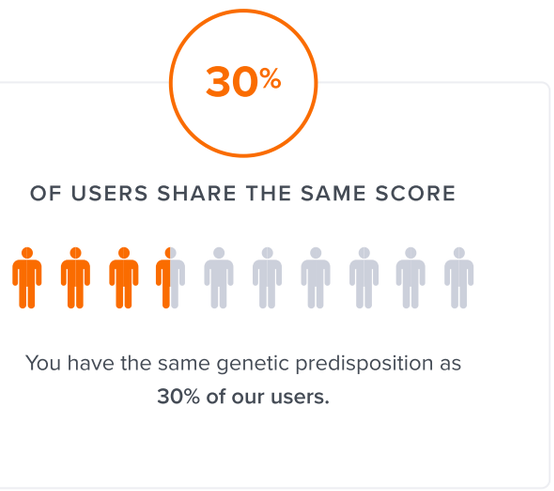
This variant has also been linked to slower clearance of the following drugs, as well as increased risk of adverse effects in people taking them:

- Methotrexate [R, R]
- Chemotherapeutic drugs [R, R]
- Rifampicin [R]

Another SLCO1B1 variant, rs4363657, showed a link with statin-induced muscle pain. However, that variant is almost always inherited together with rs4149056, so it doesn’t represent an independent genetic factor [R].

LOWER ACTIVITY

Predisposed to lower SLCO1B1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SLCO1B1	rs4149056	TC

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

GSTA1 (Detox/ Glutathione)

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

Another GSTA1 variant, rs3957356, has shown similar associations. These two variants are almost always inherited together, meaning that you likely have either none or both of them.

LOWER ACTIVITY

Predisposed to lower GSTA1 activity based on the genetic variants we looked at

20%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **20% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GSTA1	rs3957357	AA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

CYP1B1 (Detox/ Skin Health)

The main *CYP1B1* variant is **rs1056836** (Leu432Val). The **G (Val) allele** seems to **increase** the enzyme's activity. This may lead to increased production of toxic metabolites of hormones and toxins.

In line with this, studies have found a link between this allele and higher odds of multiple myeloma and lung cancer [R, R].

On the other hand, increased CYP1B1 activity may protect the skin against excessive UV radiation. The high-activity G allele may be linked to lower odds of skin cancer [R].

Other important *CYP1B1* variants include:

- **rs1800440**: The “C” allele may increase enzyme activity. It’s linked to lower odds of skin cancer but also lower vitamin D levels and higher odds of asthma [R]
- **rs10012**: The “C” allele may increase enzyme activity. It’s linked to lower odds of skin cancer but higher odds of bladder cancer [R, R]

Predisposed to higher CYP1B1 activity based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CYP1B1	rs1056836	GG
CYP1B1	rs1800440	CT
CYP1B1	rs10012	GC

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Detox

Some people might have genetics that make crucial detox components work differently. Recognizing that each of us might detoxify differently because of our unique genetic makeup is an important step towards personalized healthcare and keeping ourselves healthy.

Variants in the genes encoding different **CYP enzymes** can greatly influence phase I detox ability. Examples include *CYP1A1*, *CYP2E1*, *CYP1B1*, *CYP2A6*, CYP2B6 *and* *CYP2D6*. Variants in these genes are linked to:

- Harmful effects of cigarette smoke [R, R, R, R, R]
- Pesticide sensitivity [R, R, R]
- Air pollution sensitivity [R]

A variant in the *CYP1A2* gene may impair **caffeine metabolism** and contribute to its adverse effects [R].

The *GSTP1* gene codes for a phase II detox enzyme that helps eliminate toxins using the “master antioxidant” glutathione. Studies have linked its variants to harmful effects of **air pollution, cigarette smoke, mercury**, and more [R, R, R, R, R].

The UGT enzymes encoded by genes like *UGT1A1* and *UGT2A1* are also vital for phase II detox. They help produce glutathione and remove toxins found in **plastics, cigarette smoke**, and more [R, R].

Other genes that help make glutathione and support its detox function include *GCLC*, *GSTA1*, and *GPX1*. Variants in these genes may influence the detox of **mercury, mold**, and more [R, R, R, R].

The *NAT2* codes for another major enzyme in phase II detox. Due to the variants in this gene, people can be **“slow acetylators”**, which means they may have a harder time detoxing **cigarette smoke and some chemicals and drugs** [R, R, R, R].

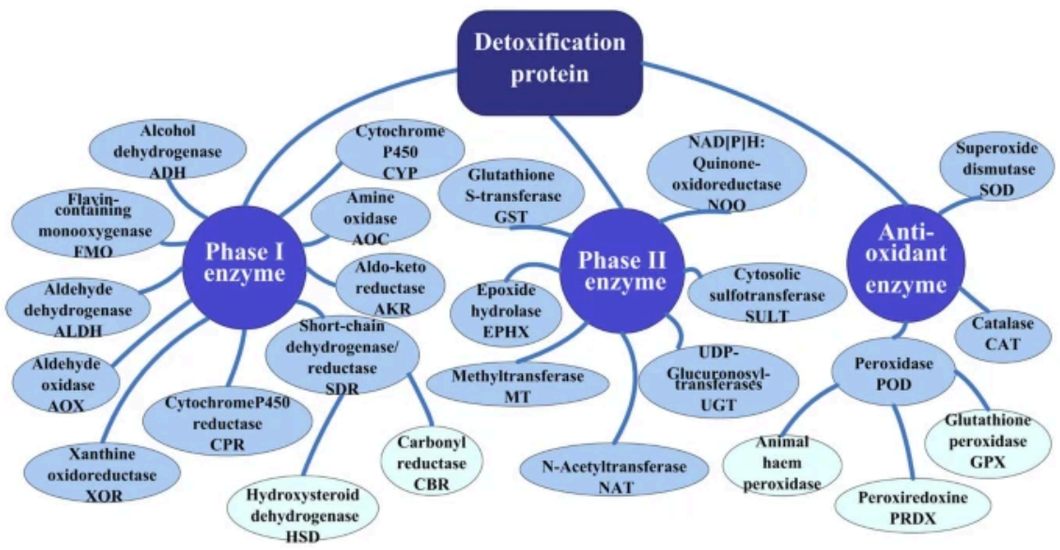


Image source: Yang et al. 2011

The *NFE2L2* gene helps make **NRF2**, a “master” protein that activates a range of antioxidant and detox genes. Its variants may affect the toxicity of alcohol, heavy metals, drugs, and more [R, R, R, R].

Alcohol detox is crucial to minimize its side effects, especially if consumed in higher amounts. Two genes, *ADH1B* and *ALDH2*, help make enzymes that process alcohol, and their variants can greatly affect detox potential [R].

Variants in the following genes also play a role in detox:

- *NQO1*: fighting oxidative stress, DNA damage, and **benzene** toxicity [R, R, R]
- *SULT1A1*: processing toxins found in **cigarette smoke and well-done meat** [R, R, R]
- *PON1*: detoxing **pesticides** and other toxins [R, R]

TYPICAL ABILITY

Predisposed to typical detox ability based on 61 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PON1	rs662	TT
SOD2	rs4880	GG
XPC	rs2228001	GT
UGT2A1	rs10518065	GA
ADH1B	rs1229984	CT
NFE2L2	rs35652124	TT
MTHFR	rs1801133	AA
CYP1B1	rs1056836	GG
NAT2	rs1495741	AG
PON1	rs854560	TA
GSTA1	rs3957357	AA
CYP1B1	rs1800440	CT
GSTO2	rs156697	GA
/	rs72547513	CC
COMT	rs4680	GA
BORCS7	rs743572	AG
NAT2	rs1041983	TC
NAT2	rs1799930	AG
ASAH1	rs4271002	GC
/	rs366631	AG
XRCC1	rs1799782	GG
PTGS2	rs5277	CC
/	rs12228069	GG
MLLT3	rs76878079	GG
MTG1	rs2031920	CC
EGLN2	rs28399433	AA
CYP2D6	rs16947	AG
/	rs2279343	AG
CYP2E1	rs2070673	AT
GSDMB	rs7216389	TC

- *SOD2* and *CAT* : reducing oxidative stress and detoxing **BPA and pesticides** [R, R]
- *COMT* : detoxing **endocrine disruptors** by methylation [R]
- *MTHFR* : supporting methylation and influencing **air pollution** sensitivity [R]
- *XPC*, *XRCC1*, *XRCC4* : repairing DNA damage caused by **pesticides, air pollution, and mold** [R, R, R, R]

GENE	SNP	GENOTYPE
CSK	rs2606345	AC
UGT2B7	rs7439366	CT
CYP1B1	rs1056827	CA
ITCH	rs819147	TT
GSTM1	rs1056806	CT
CTH	rs1021737	TG
GSTP1	rs1695	AA
ALDH2	rs671	GG
GSTP1	rs1138272	CC
CYP1A1	rs1048943	TT
COX15	rs717620	CC
NFE2L2	rs6721961	GG
NAT1	rs4986782	GG
CYP2D6	rs3892097	CC
TRIM4	rs2740574	TT
NQO1	rs1800566	GG
NQO1	rs1131341	GG
EPHX1	rs1051740	TT
GPX1	rs1050450	GG
CYP1A2	rs762551	AA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Glutathione

The following factors may affect glutathione levels [R, R, R]:

- Age: Natural glutathione production decreases with age.
- Diet: Consuming foods rich in sulfur-containing amino acids (like garlic, onions, and cruciferous vegetables) can boost glutathione levels.
- Lifestyle Factors: Smoking, alcohol consumption, and chronic stress can deplete glutathione levels.
- Health Conditions: Certain diseases, including liver and heart diseases, can lower glutathione levels.

Genetics can also play a role. Variants in genes that encode enzymes involved in glutathione synthesis (such as *GCLC* and *GCLM*) or activity (such as *GSTP1*, *GSTM1*, and *GSTT1*) may affect its levels [R].

In addition, the following strategies may help raise glutathione levels [R, R]:

- Dietary Changes: Eating foods high in sulfur-containing amino acids, vitamins C and E, selenium, and alpha-lipoic acid can support glutathione synthesis.
- Supplements: Glutathione supplements are available, although their effectiveness in increasing cellular glutathione levels varies. N-acetylcysteine (NAC) and whey protein are also known to boost glutathione.
- Lifestyle Modifications: Regular exercise, adequate sleep, and stress reduction can help maintain healthy glutathione levels.

TYPICAL FUNCTION

Predisposed to typical glutathione function based on 14 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GSR	rs2551715	CC
GCLC	rs761142	CA
TRPC4AP	rs6060124	CA
GSTA1	rs3957357	AA
CTH	rs1021737	TG
GSTM1	rs1056806	CT
/	rs366631	AG
UGT2A1	rs10518065	GA
UGT1A6	rs34983651	CC
GSTP1	rs1695	AA
GSTP1	rs1138272	CC
GPX1	rs1050450	GG
GSTM3	rs7483	CC
UGT1A6	rs6742078	GG
UGT1A1	rs4148323	GG
GSTM5	rs3754446	AA
CTH	rs12723350	TT

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

SULT1A1 (Detox)

A variant in this gene known as **SULT1A1*2** may reduce its activity. People with the **“T” allele at rs1042028** (previously named rs9282861) carry this variant [R].

As mentioned, **SULT1A1 is a double-edged sword when it comes to detox**.

In theory, **lower** SULT1A1 activity may increase the toxicity of some compounds present in smoke, like polycyclic aromatic hydrocarbons (PAHs). On the other hand, it should be protective against some other toxins, like heterocyclic amines (HAs) [R, R].

In line with this, some studies have linked the lower-activity variant, **SULT1A1*2 (rs1042028-T),** to:

- Higher odds of stomach, lung, and colon cancers in smokers [R, R, R]
- Higher odds of breast cancer in those who eat more smoked meat [R]

However, other studies have linked this variant to **lower odds** of prostate, bladder, colon, and oral cancers in smokers [R, R, R, R].

Some studies failed to confirm any link between this variant and detox [R, R].

Researchers have found other SULT1A1 variants that influence the activity of this gene [R, R, R]:

- rs6839
- rs1042157
- rs1801030

However, the impact of these variants is much lower compared with the main one, and their relevance for detox is unclear.

HIGHER ACTIVITY

Likely higher SULT1A1 activity based on the genetic variants we looked at

64%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **64% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SULT1A1	rs1042028	CC

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

UGT1A1 (Detox)

Certain genetic variations in *UGT1A1* reduce the enzymatic activity of UGT, which impairs the body's ability to detox and results in a buildup of toxic substances in the body. The normal version of *UGT1A1* (the one with normal detox activity) is called *UGT1A1*1*, or the "wild" type allele [R].

One of the most important and well-studied genetic variants is ***UGT1A1*28*** (**rs34983651**). This variant causes an "insertion" mutation, which means it adds an extra 'TA' into the gene where there should be none. According to some reports, ***UGT1A1*28*** **causes a 70% reduction in enzyme activity,** leading to 10 times slower removal of BPA from breast tissues, as well as an increased risk of adverse reactions from the following drugs [R, R, R, R, R]:

- Irinotecan, used to treat colon and lung cancer
- Raloxifene, used to treat osteoporosis and prevent breast cancer
- Raltegravir, used to treat HIV
- Indinavir, used to treat HIV
- Atazanavir, used to treat HIV
- Sorafenib, used to treat various cancers

Other variants include ***UGT1A1*6*** (**rs4148323**) and ***UGT1A1*27*** (**rs6742078**), which also lower enzyme activity, leading to a reduced ability to detox. All of these variations in *UGT1A1* can lead to toxicity disorders. The minor variants of UGT1A1*27 and UGT1A1*28 are usually inherited together [R].

The minor 'A' variant of UGT1A1*6 has been associated with:

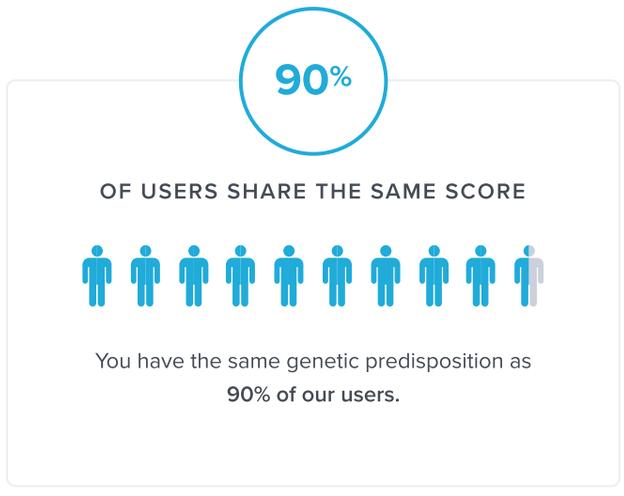
- High bilirubin in adults and bilirubin toxicity in infants [R, R, R, R, R]
- Adverse reactions of irinotecan chemotherapy [R]
- Risk of congenital heart disease in babies born to mothers exposed to polycyclic aromatic hydrocarbons [R]
- Reduced risk of anti-tuberculosis drug-induced hepatotoxicity [R]

In turn, the minor 'T' variant of UGT1A1*27 has been linked to:

- High bilirubin in adults [R, R]
- Increased risk of gallstones [R, R]
- Faster clearance of telmisartan [R]

TYPICAL ACTIVITY

Likely typical UGT1A1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
UGT1A6	rs6742078	GG
UGT1A1	rs4148323	GG

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

AHR (Detox)

The **“A” allele of rs2066853** decreases AhR levels. Lower levels of AhR would mean less protection from toxins, oxidative stress, and inflammation [R].

This variant may be linked to:

- Colorectal polyps in people exposed to toxins from well-done meat [R]
- Lung cancer in smokers [R]
- Acute lung failure (ARDS) [R]
- Reduced sperm quality [R, R]

Interestingly, people with this variant may also have slower caffeine metabolism and thus consume less coffee and tea [R].

TYPICAL ACTIVITY

Likely typical AHR activity based on the genetic variants we looked at

75%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **75% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
AHR	rs2066853	GG

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

NFE2L2/NRF2 (Detox)

Research has identified several variations in the NFE2L2 gene that can reduce the expression and activity of NRF2. Some researchers have claimed that reduced NRF2 impairs the body's ability to detox and defend itself from oxidative stress, which can ultimately lead to various health problems [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]

The exact links between NFE2L2 variants and these conditions have not been fully explained. Future research will clarify whether those variants have a causal role in health conditions.

TYPICAL ACTIVITY

Likely typical NFE2L2 activity based on 8 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NFE2L2	rs35652124	TT
NFE2L2	rs6726395	AA
NFE2L2	rs1806649	CC
NFE2L2	rs6721961	GG
AGPS	rs2364723	GG
AGPS	rs1962142	GG
HNRNPA3	rs13001694	GG
AGPS	rs10497511	AA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Phase II Detox

Some people might have genetics that make crucial detox components work differently. Recognizing that each of us might detoxify differently because of our unique genetic makeup is an important step towards personalized healthcare and keeping ourselves healthy.

The UGT enzymes encoded by genes like *UGT1A1* and *UGT2A1* are vital for phase II detox. They help produce glutathione and remove toxins found in **plastics, cigarette smoke**, and more [R, R].

The *GSTP1* gene codes for a phase II detox enzyme that helps eliminate toxins using the "master antioxidant" glutathione. Studies have linked its variants to harmful effects of **air pollution, cigarette smoke, mercury**, and more [R, R, R, R, R].

Other genes that help make glutathione and support its detox function include *GCLC*, *GSTA1* and *GPX1*. Variants in these genes may influence the detox of **mercury, mold**, and more [R, R, R, R].

The *NAT2* codes for another major enzyme in phase II detox. Due to the variants in this gene, people can be **"slow acetylators"**, which means they may have a harder time detoxing **cigarette smoke and some chemicals and drugs** [R, R, R, R].

The *NFE2L2* gene helps make **NRF2**, a "master" protein that activates a range of antioxidant and detox genes. Its variants may affect the toxicity of alcohol, heavy metals, drugs, and more [R, R, R, R].

The *COMT* gene plays a key role in detoxing certain **endocrine disruptors** by methylation [R].

MTHFR also supports methylation and influences **air pollution** sensitivity [R].

SULT1A1 processes toxins found in **cigarette smoke and well-done meat** [R, R, R].

Finally, XPC, XRCC1, and XRCC4 are involved in repairing DNA damage caused by **pesticides, air pollution, and mold** [R, R, R, R].

TYPICAL ABILITY

Predisposed to typical phase II detox ability based on 26 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
XPC	rs2228001	GT
NFE2L2	rs35652124	TT
MTHFR	rs1801133	AA
NAT2	rs1495741	AG
PON1	rs854560	TA
GSTA1	rs3957357	AA
CYP1B1	rs1800440	CT
GSTO2	rs156697	GA
COMT	rs4680	GA
THSD7A	rs116864947	TT
NAT2	rs1208	AA
UGT2B7	rs7439366	CT
GSTM1	rs1056806	CT
GSTP1	rs1695	AA
GSTP1	rs1138272	CC
COX15	rs717620	CC
NFE2L2	rs6721961	GG
NQO1	rs1800566	GG
NQO1	rs1131341	GG
GPX1	rs1050450	GG
UGT1A6	rs6742078	GG
UGT1A1	rs4148323	GG
NAT2	rs1799931	GG
GSTM3	rs7483	CC
MTG1	rs2031920	CC
ERCC5	rs17655	GG
SULT1A1	rs1042028	CC

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

CYP1A1 (Detox)

The ‘G’ allele of rs4646903, linked to higher CYP1A1 levels, has been associated with an increased risk of leukemia, cervical, hepatocellular, lung, prostate, colorectal, breast and head and neck cancers, possibly by turning PAHs into cancer-causing chemicals. This variant may also increase the risk of PCOS, recurrent pregnancy loss, and male infertility through its role in estrogen metabolism [R, R, R, R, R, R, R, R, R].

Similarly, the ‘C’ variant of rs1048943 increases CYP1A1 activity and has been associated with an increased risk of lung cancer, laryngeal cancer, oral cancer, colorectal cancer, cervical cancer, hepatocellular carcinoma, prostate cancer, and leukemia. In the case of lung cancer, the risk may be even higher in smokers carrying this variant [R, R, R, R, R, R, R, R, R, R, R].

The ‘A’ allele of rs2606345 may decrease CYP1A1 levels. This variant has been associated with an increased risk of lung conditions such as pneumonia or ARDS, possibly by increasing inflammation and oxidative stress in the lungs. In contrast, the ‘C’ allele has been associated with esophageal and testicular cancer [R, R, R, R, R, R, R, R].

The ‘T’ allele of rs2472297 may increase CYP1A1 activity. This variant has been associated with greater coffee consumption, as well as with a decreased increment in blood glucose after consuming caffeine [R, R, R, R].

Finally, the ‘A’ allele of rs4646421 has been associated with hepatocellular carcinoma, endometrial cancer (especially in women with abdominal obesity), esophageal cancer (in hot tea drinkers), laryngeal cancer, decreased lung function in elderly people exposed to PAHs, and chronic hepatitis B infection [R, R, R, R, R, R].

TYPICAL ACTIVITY

Likely typical CYP1A1 activity based on 4 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CSK	rs2606345	AC
ULK3	rs2472297	CC
CYP1A1	rs4646903	AA
CYP1A1	rs1048943	TT
ARID3B	rs4646421	GG

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

CYP2C9 (Detox)

The CYP2C9 gene is highly polymorphic, with more than 50 known alleles affecting its metabolic activity compared to the wild-type CYP2C9*1 allele [R].

The two most common CYP2C9 gene polymorphisms decreasing enzyme activity are known as CYP2C9*2 and CYP2C9*3. Individuals with one copy of any of these alleles or two copies of the CYP2C9*2 allele are considered intermediate metabolizers, while those with one copy of each one or two copies of the CYP2C9*3 allele are considered poor metabolizers.

CYP2C9*2 ('T' at rs1799853) consists of the change of the amino acid arginine with the amino acid cysteine at position 144 while CYP2C9*3 ('C' at rs1057910) consists of the change of the amino acid isoleucine with the amino acid leucine at position 359. **THC metabolism is especially impaired in people with two copies of the CYP2C9*3 variant** [R, R].

The CYP2C9*5 allele ('G' at rs28371686) consists of the change of the amino acid aspartic acid with the amino acid glutamic acid at position 360 and is believed to decrease enzyme activity [R, R].

The CYP2C9*8 allele ('A' at rs7900194) consists of the change from an arginine to a histidine at position 150 and also encodes an enzyme with decreased activity. This variant is especially common in African Americans [R, R, R].

Finally, the CYP2C9*11 allele ('T' at rs28371685) consists of the change from an arginine to a tryptophan at position 335 and also encodes a protein with decreased enzyme activity [R, R].

TYPICAL METABOLIZER

Likely a typical metabolizer based on 5 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CYP2C9	rs1057910	AA
CYP2C9	rs7900194	GG
CYP2C9	rs28371686	CC
CYP2C9	rs28371685	CC
CYP2C9	rs1799853	CC

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

CYP3A4 (Detox)

The ‘A’’ allele of **rs35599367**, also known as CYP3A4*22, reduces CYP3A4 levels and activity by approximately half, resulting in slower drug metabolism [R, R, R, R].

Similarly, the minor alleles ‘C’ of **rs12721627** (CYP3A4□16) and ‘G’ of **rs55785340** (CYP3A4*2) have been associated with lower enzyme activity [R, R].

Two other variants with decreased enzyme activity are ‘C’ at **rs2740574** (CYP3A4*1B) and ‘T’ at **rs2242480** (CYP3A4*1G) [R, R].

The minor ‘G’ allele of **rs680055** (CYP3A4*3) encodes a protein with an amino acid change believed to reduce enzyme activity [R].

In carriers of these variants, the breakdown of cannabinoids such as THC and CBD may be slower. Moreover, people with these variants shouldn’t use cannabis if they are being treated with ketoconazole, fluconazole, or diltiazem, which are CYP3A inhibitors [R, R].

In contrast, the ‘G’ allele of **rs28371759** (CYP3A4*18) increases enzyme activity [R, R].

TYPICAL ACTIVITY

Likely typical CYP3A4 activity based on 7 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CYP3A4	rs28371759	AA
CYP3A43	rs680055	CC
CYP3A4	rs55785340	AA
TRIM4	rs35599367	GG
TRIM4	rs2740574	TT
CYP3A5	rs2242480	CC
/	rs12721627	GG

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

NAT2 (Detox)

NAT2 activity or status varies widely among individuals due to **genetic differences**. This status influences how one's body processes a range of environmental toxins.

Research has linked a lot of *NAT2* gene variants to changes in enzyme activity, but most resources use 7 main variants to determine the acetylator status. The frequency of this status varies widely between different ethnicities. Roughly **45%** of the overall population and **60%** of European descendants are **slow acetylators** [R].

Being a slow NAT2 acetylator may be linked to higher odds of different types of cancer, especially bladder cancer. This link tends to be stronger in people exposed to **cigarette smoke and chemical dyes** [R, R, R, R, R].

The link between the slow NAT2 status and other types of cancer and other chronic conditions like asthma and diabetes is weaker [R, R, R].

Slow acetylators may also have a harder time detoxing certain drugs, which may put them at higher odds of side effects. For example, they tend to have higher rates of liver injury from the anti-tuberculosis drug isoniazid [R].

On the other hand, fast acetylators may be more prone to colon cancer, especially if they consume **well-done meat** frequently. This may be due to activation (O-acetylation) of certain toxins by NAT2 in the colon [R, R].

NAT2 also indirectly affects **histamine metabolism** by acetylating compounds that influence histamine levels. "Slow acetylators" with reduced NAT2 activity may experience more pronounced histamine intolerance symptoms due to difficulty processing dietary histamine or histamine-releasing substances.

INTERMEDIATE

Likely an intermediate acetylator based on 7 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NAT2	rs1041983	TC
NAT2	rs1799930	AG
ASAH1	rs4271002	GC
NAT2	rs1801280	TT
NAT2	rs1801279	GG
NAT2	rs1799931	GG
NAT2	rs1799929	CC
NAT2	rs1208	AA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

NQO1 (Detox)

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

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

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].

This variant may also be linked to Alzheimer's disease, but the evidence is mixed [R].

HIGHER ACTIVITY

Likely higher NQO1 activity based on the genetic variants we looked at

60%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **60% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NQO1	rs1800566	GG
NQO1	rs1131341	GG

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

GSTP1 (Detox)

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].

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].

HIGHER ACTIVITY

Likely higher GSTP1 activity based on the genetic variants we looked at

38%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **38% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GSTP1	rs1695	AA
GSTP1	rs1138272	CC

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

GPX1 (Glutathione/Detox)

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].

The authors of the study suggested there could be some kind of survival benefit for the 'AG' genotype, but they did not speculate as to why the heterozygote might have an advantage over 'AA' and 'GG' [R].

That said, 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, enlarged joints, and decreased range of motion. 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].

Please note: These three variants are closely linked, so if you have a "bad" allele at one, you will likely also have "bad" alleles at others.

HIGHER ACTIVITY

Likely higher GPX1 activity based on the genetic variants we looked at

43%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **43% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GPX1	rs1050450	GG

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Sulfation (Detox)

Sulfation is a key detoxification pathway that helps your body process and remove various compounds like hormones, neurotransmitters, drugs, and environmental toxins. Your sulfation genes determine how efficiently this process works.

SULT1A1 (rs1042157, rs1042028): The *SULT1A1* gene encodes an enzyme known as sulfotransferase 1A1. The workhorse of sulfation, SULT1A1 handles with a wide range of compounds including hormones and medications. This enzyme is most active in the liver and gut. A variant in this gene known as **SULT1A1*2** may reduce its activity. People with the **"T" allele at rs1042028** (previously named rs9282861) carry this variant. Another variant influencing the activity of this gene is rs1042157 [R].

SULT1C3 (rs13392744, rs2219078): SULT1C3 is particularly important for processing environmental toxins and certain medications. This enzyme is more active during fetal development.

SULT2A1 (rs2547231): The SULT2A1 enzyme specializes in processing steroid hormones and bile acids. By doing so, it helps maintain hormone balance and supports liver detoxification. The 'A' allele of rs2547231 is involved in cholesterol metabolism and has been linked to gallstones [R, R].

Variations in these genes can affect your body's ability to process different compounds, potentially influencing how well you handle certain medications, hormones, or environmental exposures.

HIGHER ACTIVITY

Predisposed to higher sulfation activity based on 5 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SULT2A1	rs2547231	AA
SULT1A1	rs1042028	CC
SULT1A1	rs1042157	GG
SULT1C3	rs2219078	GG
SULT1C3	rs13392744	CC

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

UGT (Detox)

Certain genetic variations in *UGT1A1* reduce the enzymatic activity of UGT, which impairs the body's ability to detox and results in a buildup of toxic substances in the body. The normal version of *UGT1A1* (the one with normal detox activity) is called *UGT1A1*1*, or the "wild" type allele [R].

One of the most important and well-studied genetic variants is ***UGT1A1*28*** (rs34983651). This variant causes an "insertion" mutation, which means it adds an extra 'TA' into the gene where there should be none. According to some reports, ***UGT1A1*28*** **causes a 70% reduction in enzyme activity** [R].

Other variants include ***UGT1A1*6*** (rs4148323) and ***UGT1A1*27*** (rs6742078), which also lower enzyme activity, leading to a reduced ability to detox. All of these variations in *UGT1A1* can lead to toxicity disorders. The minor variants of UGT1A1*27 and UGT1A1*28 are usually inherited together [R].

Other, less well-researched *UGT1A1* variants with reduced enzyme activity include:

- 'A' at rs10929302 (*UGT1A1*93*) [R, R, R, R]
- 'T' at rs887829 (*UGT1A1*80*) [R, R, R]
- 'G' at rs4124874 (*UGT1A1 *60*) [R, R, R]
- 'T' at rs4148325 [R, R]
- 'T' at rs199539868 [R]
- 'T' at rs114982090 [R]

A *UGT2A1* variant, 'G' at rs10518065, has been associated with poorer cognitive performance. The negative effects of this allele are likely due to decreased production of glutathione, leading to increased oxidative stress in the brain [R, R, R].

The low-activity *UGT1A6* variants 'G' at rs2070959 and 'C' at rs1105879 have been associated with higher bilirubin levels and increased risk of toxicity from aspirin, benzene, and vinyl chloride [R, R, R, R].

Finally, two *UGT2B7* variants, 'G' at rs7438135 and 'T' at rs7439366, have been associated with increased enzyme activity and decreased effectiveness of opioids [R, R, R, R, R].

HIGHER ACTIVITY

Likely higher UGT activity based on 11 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
UGT2A1	rs10518065	GA
UGT2B7	rs7439366	CT
USP40	rs4124874	GT
UGT1A6	rs34983651	CC
UGT1A6	rs887829	CC
UGT1A6	rs6742078	GG
UGT1A6	rs4148325	CC
UGT1A1	rs4148323	GG
UGT2B7	rs7438135	GG
UGT1A6	rs2070959	AA
UGT1A6	rs1105879	AA
UGT1A6	rs10929302	GG
UGT1A1	rs199539868	CC
UGT1A1	rs114982090	CC

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

CYP1A2 (Detox)

CYP1A2 is an enzyme that helps break down caffeine, drugs, and certain toxins like mold. Variants in the *CYP1A2* gene affect how fast people break down those substances [R, R, R].

The "slow metabolizer" variants make a less efficient enzyme. People who carry these variants may be more **sensitive to caffeine**. Accordingly, they may be more likely to experience negative effects when drinking coffee [R, R, R].

In terms of detox, they may be more susceptible to the adverse effects of certain drugs and toxins. However, the link between CYP1A2 variants and environmental toxins is more complex and requires further investigation [R, R].

The "fast metabolizer" variant makes a protein that breaks down caffeine. People with these variants may be less sensitive to its effects [R, R, R, R].

Nevertheless, "fast metabolizers" may experience the benefits of caffeine supplementation on athletic performance after a short time while "slow metabolizers" may need a longer ingestion period [R, R].

The following factors and substances may **increase** CYP1A2 activity:

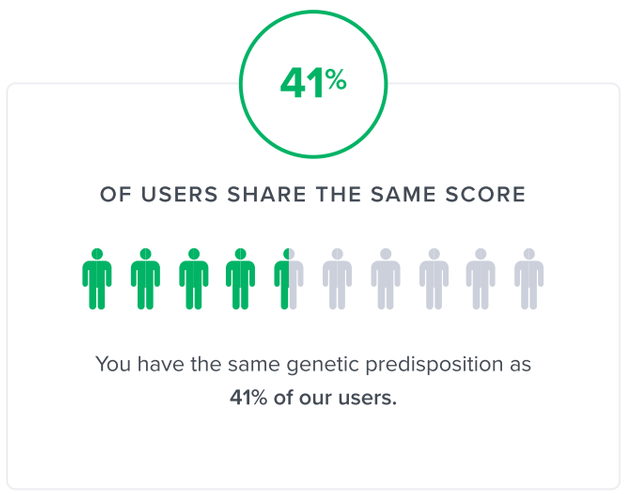
- Cigarette smoke: 1.72-fold for >20 cigarettes per day [R, R]
- Coffee consumption: 1.45-fold per liter of coffee drunk daily [R, R]
- Meat pan-fried at high temperatures: 1.4-fold [R]
- Chargrilled meat: 1.89-fold [R]
- Cruciferous vegetables [R, R, R]
- Green and black tea [R]
- Insulin [R]
- Being female: 0.90-fold [R]
- Heavy exercise [R]
- Omeprazole [R]
- Evodioa
- Reishi
- Andrographis,
- Modafinil
- Glycyrrhizin (liquorice)

The following factors and substances may **decrease** CYP1A2 activity:

- Apiaceous vegetables (carrots, parsnips, celery, and parsley) [R]
- Curcumin [R]
- Grapefruit juice and its component naringenin [R]
- Echinacea [R]
- Quercetin [R]
- Antibiotic fluoroquinolones [R]
- Fluvoxamine, an antidepressant [R]
- Peppermint, chamomile, and dandelion tea [R]
- Garlic
- Berberine
- Chamomile
- Lactoferrin
- Hops
- Galangin (galangal root)
- Scutellaria baicalensis,
- Tangeritin
- Trans-resveratrol

HIGHER ACTIVITY

Likely higher CYP1A2 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CYP1A2	rs762551	AA
LMAN1L	rs2069514	GG

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

CYP2C19 (Detox)

There are more than 30 known *CYP2C19* variants. Based on the variants they carry, individuals can be categorized as [R, R]:

- Ultrarapid metabolizers (*1 /*17 or *17 /*17)
- Extensive metabolizers (*1/*1)
- Intermediate metabolizers (*1 /*2, *1 /*3, or *2 /*17)
- Poor metabolizers (*2 /*2 or *2 /*3)

The normal version of the gene is called CYP2C19*1.

The two most common polymorphisms associated with reduced CYP2C19 activity and clopidogrel resistance are CYP2C19*2 (**rs4244285**) and CYP2C19*3 (**rs4986893**). Carriers of one copy of the minor 'A' alleles have a reduced ability to break down drugs, including CBD. Those with two copies can metabolize very little or none of the drug and are classified as poor metabolizers [R, R, R].

The **rs12248560** polymorphism, also known as CYP2C19*17, is located in the region that controls gene expression (the *promoter*). Its minor allele 'T' is associated with increased gene expression and protein activity. People with this variant are classified as ultrarapid metabolizers [R, R].

The following supplements and chemicals may decrease CYP2C19 activity [R, R, R, R, R, R, R, R, R, R, R]:

- Propolis
- Caffeic acid
- Quercetin
- Ginger
- Kale
- African lettuce
- Saint John's wort
- Astaxanthin
- Canthaxanthin
- Hops
- Licorice
- Berberine
- Capsaicin
- Bulbocapnine, canadine, and protopine
- Fluconazole

EXTENSIVE METABOLIZER

Likely an extensive metabolizer based on the genetic variants we looked at

62%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **62% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NOC3L	rs12248560	CC
CYP2C19	rs4986893	GG
CYP2C19	rs4244285	GG

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Oxidative Stress

Oxidative stress occurs when there is an imbalance between free radicals and the body's ability to neutralize them, potentially leading to cellular damage and chronic health issues. Genetic factors influence how well the body manages oxidative stress and repairs damage caused by reactive oxygen species (ROS).

This section examines key genetic markers, including **COQ2**, **SOD2**, and **SOD3**, which play a role in antioxidant defense, lipid oxidation, and DNA protection. Understanding your genetic predisposition can provide insights into how your body handles oxidative stress and guide personalized strategies for supporting cellular health and longevity.

LOWER ACTIVITY

COQ2 (Oxidative Stress)

Likely lower COQ2 activity

LOWER ACTIVITY

SOD2 (Oxidative Stress)

Likely lower SOD2 activity

WORSE

SOD3 (Oxidative Stress)

Likely worse SOD3 genetics

LOWER ACTIVITY

GCLC (Glutathione & Detox)

Predisposed to lower GCLC activity

LOWER ACTIVITY

GSTA1 (Detox/ Glutathione)

Predisposed to lower GSTA1 activity

TYPICAL

Oxidative Stress

Likely typical oxidative stress

TYPICAL

Lipid Oxidation

Likely typical lipid oxidation

TYPICAL LEVELS

DNA Damage

Predisposed to typical DNA damage

TYPICAL ACTIVITY

GPX4 (Selenium & Glutathione)

Predisposed to typical GPX4 activity

HIGHER ACTIVITY

GPX1 (Glutathione/Detox)

Likely higher GPX1 activity

COQ2 (Oxidative Stress)

The most well-researched *COQ2* polymorphism is rs148156462 (V393A). Its minor 'G' allele reduces gene expression and thus coenzyme Q10 production. This allele has been associated with an increased risk of:

- Multiple system atrophy (cerebellar type) [R, R, R]
- Parkinson's disease [R]

The major 'C' allele of another variant, rs4693075, has been associated with statin-induced muscle damage. However, not all studies found this effect [R, R, R, R, R].

LOWER ACTIVITY

Likely lower COQ2 activity based on 2 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
COQ2	rs4693075	CC
COQ2	rs148156462	AA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

SOD2 (Oxidative Stress)

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 activity and worse protection against oxidative stress. However, some cell research suggests that this variant can cross the mitochondrial membrane more easily [R].

Owing to its decreased antioxidant activity, 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]:

- Cardiomyopathy
- Atherosclerosis
- Lung cancer

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. Nevertheless, **the growing consensus is that 'G' is the risk allele of rs4880** [R, R].

A second SNP, rs2758331, has also been associated with lifespan. In one study of exceptionally long-lived people in New England, the 'C' allele of rs2758331 was significantly more common in the oldest old than in the general population [R].

This SNP is nowhere near as well-studied as rs4880, and only a single study has investigated its effect on lifespan so far. Furthermore, while the 'A' allele of rs2758331 has been associated with prostate cancer (thereby supporting the idea of a beneficial 'C' allele), the 'C' allele has been associated with liver damage after bisphenol A (BPA) exposure [R, R].

LOWER ACTIVITY

Likely lower SOD2 activity based on the genetic variants we looked at

23%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **23% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SOD2	rs4880	GG
TCP1	rs2758331	AA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

SOD3 (Oxidative Stress)

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]

Among them, rs1799895 is the most well-researched one. 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].

Furthermore, low SOD3 levels have been associated with polyneuropathy in people with diabetes [R].

These are all potentially life-threatening conditions. Thus, the search goes on for a more direct link between SOD3 and lifespan.

Likely worse SOD3 genetics based on the genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SOD3	rs2536512	AA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

GCLC (Glutathione & Detox)

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]

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].

LOWER ACTIVITY

Predisposed to lower GCLC activity based on the genetic variants we looked at

16%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **16% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GCLC	rs17883901	AG
GCLC	rs761142	CA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

GSTA1 (Detox/ Glutathione)

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

Another GSTA1 variant, rs3957356, has shown similar associations. These two variants are almost always inherited together, meaning that you likely have either none or both of them.

LOWER ACTIVITY

Predisposed to lower GSTA1 activity based on the genetic variants we looked at

20%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **20% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GSTA1	rs3957357	AA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Oxidative Stress

The causes of oxidative stress can be multifaceted, including environmental factors such as pollution, radiation, and toxins, as well as lifestyle factors like dietary choices, smoking, alcohol consumption, and chronic stress. The body's metabolism also naturally produces free radicals as byproducts. Oxidative stress is implicated in the pathogenesis of numerous diseases, including neurodegenerative diseases like Alzheimer's and Parkinson's, cardiovascular diseases, diabetes, and inflammatory conditions.

It is also involved in the aging process itself. Therefore, maintaining a balance between oxidative stress and antioxidants is critical for health, and enhancing antioxidant defenses through diet and lifestyle changes is often suggested as a preventive strategy.

TYPICAL

Likely typical oxidative stress based on 60 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SOD2	rs4880	GG
SOD3	rs2536512	AA
CAT	rs769217	CC
FOXO3	rs12212067	TT
FOXO3	rs4946936	CC
PON1	rs662	TT
FOXO3	rs12202234	CC
FOXO3	rs17069665	AA
FOXO3	rs9398171	TT
FOXO3	rs3800230	TT
FOXO3	rs9400239	CC
FOXO3	rs479744	GG
SIRT1	rs7895833	AA
CAT	rs7943316	AT
GPX4	rs713041	CT
CAT	rs1001179	CT
APEX1	rs1130409	GG
NOS3	rs2070744	CT
GCLC	rs1555903	CT
UGT1A6	rs1105879	AA
NOS1	rs1879417	TC
SIRT1	rs12778366	TC
GSTO2	rs156697	GA
UCP2	rs659366	TC
TFAM	rs1937	GC
PPARGC1A	rs8192678	CT
CDKN2A	rs10811661	TT
UCP1	rs1800592	TC
HNRNPA3	rs13001694	GG
GPX1	rs1050450	GG

GENE	SNP	GENOTYPE
MVD	rs9932581	CT
SOD1	rs2234694	AA
NQO1	rs1800566	GG
GSTP1	rs1695	AA
PON1	rs854560	TA
ARMC2	rs6911407	AA
MRPS31	rs4581585	CC
GCLM	rs41303970	GG
FOXO3	rs2802292	GG
MPO	rs2333227	CC
TOM1	rs2071746	AA
OGG1	rs1052133	CC
ARMC2	rs768023	GG
ALDH2	rs671	GG
APOE	rs429358	TT
SLC23A1	rs33972313	CC
FOXO3	rs2802288	AA
NOS2	rs2297518	GG
FOXO3	rs2253310	CC
FOXO3	rs1935952	CC

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Lipid Oxidation

Genetic variants can influence lipid oxidation by affecting processes such as fatty acid metabolism, lipoprotein function, and the body's ability to manage oxidative stress. Understanding these genetic factors offers valuable insights into how your body processes fats and manages oxidative stress, enabling tailored interventions to optimize lipid metabolism and promote cardiovascular health.

PON1 (rs662): The *PON1* gene encodes paraoxonase-1, an enzyme that protects lipids from oxidation. The 'C' allele of the rs662 polymorphism is associated with lower PON1 activity, resulting in increased susceptibility to lipid peroxidation and oxidative stress.

PON1 (rs854560): Another variant in the *PON1* gene, rs854560, is also linked to reduced antioxidant activity. The 'T' allele results in a higher risk of oxidized lipids and a compromised ability to neutralize oxidative damage to fatty acids.

LPA (rs10455872): The *LPA* gene affects lipoprotein(a) levels, which are linked to lipid transport and metabolism. The 'A' allele of rs10455872 is associated with reduced lipid clearance and increased oxidative stress, potentially impairing lipid oxidation and contributing to cardiovascular disease risk.

APOB (rs11751605): The *APOB* gene impacts the structure and function of apolipoprotein B, a key component of LDL cholesterol. The 'T' allele of rs11751605 may influence lipid oxidation processes, leading to increased LDL oxidation and a greater risk of atherosclerosis.

FADS1 (rs9457933): The *FADS1* gene regulates fatty acid desaturation and can affect the balance of omega-3 and omega-6 fatty acids. The 'C' allele of rs9457933 is associated with impaired fatty acid metabolism, reducing the efficiency of lipid oxidation and promoting lipid accumulation.

HNF1A (rs3124785): The *HNF1A* gene is involved in cholesterol and triglyceride metabolism. The 'A' allele of the rs3124785 variant can influence lipid oxidation by altering the hepatic clearance of lipids, potentially increasing oxidative stress and the risk of dyslipidemia.

CETP (rs9355816): The *CETP* gene is involved in HDL-mediated lipid transport, affecting lipid oxidation indirectly. The 'C' allele of the rs9355816 polymorphism may impair HDL function, reducing its ability to prevent lipid peroxidation and leading to oxidative damage.

PPARGC1A (rs10755578): The *PPARGC1A* gene impacts mitochondrial function and energy metabolism. The 'G' allele of the rs10755578 polymorphism may reduce the efficiency of lipid oxidation, leading to increased fat storage and metabolic imbalance.

TYPICAL

Likely typical lipid oxidation based on 8 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
LPA	rs10455872	AA
LPA	rs9355816	CC
PON1	rs854560	TA
PLG	rs11751605	TT
LPA	rs9457933	CC
PLG	rs3124785	AG
SLC22A3	rs10755578	GC
PON1	rs662	TT

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

DNA Damage

DNA damage can occur due to a variety of internal and external factors:

- **Environmental factors:** exposure to UV radiation from the sun is a major cause of DNA damage, particularly in the form of thymine dimers. Other environmental factors that contribute to DNA damage include ionizing radiation (e.g., X-rays and gamma rays), chemical agents (e.g., tobacco smoke and environmental pollutants), and certain chemotherapy drugs.
- **Metabolic byproducts:** cells produce reactive oxygen species (ROS) such as superoxide anions and hydrogen peroxide as byproducts of normal cellular metabolism. These species can attack DNA, causing base modifications, strand breaks, and other forms of damage. Chronic oxidative stress can lead to the accumulation of DNA damage and mutations.
- **Replication stress:** during DNA replication, the DNA polymerase may encounter obstacles, such as DNA secondary structures or tightly bound proteins. This can lead to stalled replication forks, which increase the likelihood of DNA breaks and mutations.
- **Inflammation:** chronic inflammation, often associated with diseases like arthritis, infections, or cancer, can lead to increased production of ROS and reactive nitrogen species (RNS), which damage DNA. Inflammatory cytokines can also indirectly promote DNA damage by inducing oxidative stress.

The consequences of DNA damage depend on the type of damage and the cell's ability to repair it. If DNA damage is not repaired or if it accumulates over time, it can lead to several adverse outcomes such as:

- **Mutagenesis:** DNA damage can cause mutations—changes in the DNA sequence that may affect gene expression or protein function. If these mutations occur in critical genes (e.g., tumor suppressor genes or oncogenes), they can lead to cancer development.
- **Genomic Instability:** when DNA damage is not properly repaired, it can result in genomic instability, characterized by an increased rate of mutations, chromosomal rearrangements, or aneuploidy (abnormal number of chromosomes). Genomic instability is a hallmark of many cancers.
- **Cell death:** in cases where DNA damage is too severe to be repaired, cells may undergo programmed cell death (apoptosis) to prevent the damage from passing on to daughter cells. While apoptosis protects the organism from potentially harmful mutations, excessive cell death can contribute to tissue degeneration and aging.
- **Aging:** the accumulation of DNA damage over time is a key factor in the aging process. Reduced DNA repair efficiency and increased oxidative stress contribute to age-related diseases, such as neurodegeneration, cardiovascular diseases, and sarcopenia (muscle wasting).
- **Cancer:** one of the most significant consequences of DNA damage is cancer. If DNA repair mechanisms fail, mutations may accumulate in genes that regulate the cell cycle, apoptosis, or DNA repair itself. This can lead to uncontrolled cell proliferation, a hallmark of cancer.

TYPICAL LEVELS

Predisposed to typical DNA damage based on 69 genetic variants we looked at

61st

PERCENTILE

Your risk is **greater than 61% of the population** and **lower than 39% of the population.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ATM	rs1801516	GG
SLC6A3	rs2736108	CC
SOD2	rs4880	GG
FOXE1	rs1867277	AA
PARP1	rs3219090	TT
FOXO3	rs479744	GG
SIRT1	rs7895833	AA
FOXO3	rs12202234	CC
FOXO3	rs9398171	TT
FOXO3	rs3800230	TT
FOXO3	rs4946936	CC
TERT	rs2075786	GG
SLK	rs9419958	CC
GSTO1	rs9420907	AA
ORMDL1	rs2289226	TT
CAT	rs769217	CC
FCGR2A	rs1801274	AA
MRPS31	rs2755209	AA
FOXO3	rs12212067	TT
FOXO3	rs17069665	AA
FOXO3	rs9400239	CC
STN1	rs10786775	CC

GENE	SNP	GENOTYPE
GSTO1	rs4387287	CC
CFHR3	rs10801555	GG
TFAM	rs1937	GC
TERT	rs7705526	AC
TERT	rs2736100	CA
SLC6A3	rs2735940	AG
TERT	rs2853677	GA
COMT	rs4680	GA
TERT	rs2853672	CA
TERT	rs2853676	TC
ADA	rs73598374	TC
NQO1	rs689453	TC
SIRT1	rs12778366	TC
TERT	rs10069690	TC
TERT	rs13167280	GA
TERT	rs2242652	AG
BCHE	rs17713088	GT
BCHE	rs9881048	CA
CAT	rs480575	GA
TRMO	rs10984009	GA
UGT2A1	rs10518065	GA
SIRT1	rs7896005	GA
TERT	rs4449583	TC
STN1	rs11191865	AG
ARHGEF3	rs1354034	TC
SLC22A5	rs2240032	CC
TERT	rs2736098	CC
SLC22A5	rs2040704	AA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

GPX4 (Selenium & Glutathione)

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].

TYPICAL ACTIVITY

Predisposed to typical GPX4 activity based on the genetic variants we looked at

80%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **80% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GPX4	rs713041	CT

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

GPX1 (Glutathione/Detox)

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].

The authors of the study suggested there could be some kind of survival benefit for the 'AG' genotype, but they did not speculate as to why the heterozygote might have an advantage over 'AA' and 'GG' [R].

That said, 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, enlarged joints, and decreased range of motion. 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].

Please note: These three variants are closely linked, so if you have a "bad" allele at one, you will likely also have "bad" alleles at others.

HIGHER ACTIVITY

Likely higher GPX1 activity based on the genetic variants we looked at

43%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **43% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GPX1	rs1050450	GG

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Recommendations Details

1 N-acetylcysteine (NAC)

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

Helps with these Symptoms & Conditions:

- Anxiety

Helps with these Goals:

- Energy
- Immunity
- Mood

Helps with these DNA Risks:

- Aluminum
- GCLC (Glutathione & Detox)
- Phase I Detox
- PON1 (Detox)
- SOD2 (Oxidative Stress)
- UGT2A1 (Cognition)

2 Dietary Antioxidants

Incorporate foods rich in antioxidants, such as fruits (berries, oranges, plums), vegetables (spinach, kale, bell peppers), nuts (walnuts, almonds), and seeds (flaxseeds, chia seeds) into your daily meals. Aim for at least 5 servings of fruits and vegetables per day, ensuring a variety of colors to cover different antioxidants.

Helps with these Goals:

- Mood

Helps with these DNA Risks:

- Aluminum
- COQ2 (Oxidative Stress)
- Phase I Detox
- PON1 (Detox)
- SOD2 (Oxidative Stress)

SOD3 (Oxidative Stress)

UGT2A1 (Cognition)

3 Selenium Supplements

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

TYPICAL STARTING DOSE

50 mcg

Helps with these Goals:

Immunity

Mood

Helps with these DNA Risks:

Aluminum

GCLC (Glutathione & Detox)

PON1 (Detox)

SOD2 (Oxidative Stress)

4 Dietary Polyphenols

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.

Helps with these Goals:

Exercise Recovery

Helps with these DNA Risks:

Aluminum

PON1 (Detox)

SOD3 (Oxidative Stress)

5 Zeolite

Take clinoptilolite zeolite (the common “zeolite supplement”) by mixing ½ to 1 level teaspoon (about 1–3 g, depending on how fine the powder is) into a glass of water (200–250 ml) once daily to start, ideally 30 minutes

TYPICAL STARTING DOSE

before or after meals; if you tolerate it well, some products allow 1–3 doses per day, but do not exceed your product's stated daily maximum. Keep zeolite at least 2 hours away from other medications or supplements, drink plenty of water, and continue this regimen for up to 8–12 weeks (or do a 2-week trial), then take a break or consult a healthcare professional for longer use.

1 tsp

Helps with these DNA Risks:

Aluminum

6 Vitamin C

Take 500-2000 mg of vitamin C supplement daily. It can be taken at any time of the day, with or without food, according to personal preference or tolerance.

TYPICAL STARTING DOSE

2000 mg

Helps with these Symptoms & Conditions:

Anxiety

High Blood Pressure

Helps with these Goals:

Immunity

Helps with these DNA Risks:

Aluminum

7 Zinc

Take a 15 mg zinc supplement daily, ideally with a meal to enhance absorption.

TYPICAL STARTING DOSE

15 mg

Helps with these Symptoms & Conditions:

High Blood Pressure

Migraines

Helps with these Goals:

Immunity

Mood

Helps with these DNA Risks:

Aluminum

8 Vitamin E

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**.

TYPICAL STARTING DOSE

200 iu

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.

Helps with these Symptoms & Conditions:

Anxiety

High Blood Pressure

Migraines

Helps with these Goals:

Exercise Recovery

Mood

Helps with these DNA Risks:

Aluminum

9 Magnesium

Take up to 350 mg of magnesium daily as a supplement, preferably with a meal to enhance absorption.

TYPICAL STARTING DOSE

350 mg

Helps with these Symptoms & Conditions:

Anxiety | High Blood Pressure | Migraines

Helps with these Goals:

Energy | Exercise Recovery | Immunity | Mood

Helps with these DNA Risks:

Aluminum

10 Green Tea

Consume 400 mg of green tea extract daily. This can be taken in the form of capsules or tablets available that specify the amount of green tea extract. Ensure the supplement is taken according to the product's specific instructions, usually once a day with water.

TYPICAL STARTING DOSE

400 mg

Helps with these Symptoms & Conditions:

Anxiety | High Blood Pressure

Helps with these Goals:

Energy | Immunity | Mood

Helps with these DNA Risks:

Aluminum

11 Avoid Aluminum Exposure

Use aluminum-free deodorants and avoid cooking or storing food in aluminum foil or aluminum cookware. Instead, opt for glass, stainless steel, or ceramic options for cooking and storing food. Check labels on over-the-counter medications, such as antacids, for aluminum compounds and choose aluminum-free alternatives. Aim to do this consistently for the long term to effectively minimize your aluminum exposure.

Helps with these DNA Risks:

Aluminum

12 Glutathione supplements

Take glutathione supplements orally, usually in pill or powder form, with a recommended dose ranging from 500mg to 1000mg daily, divided into two doses. It's best taken on an empty stomach or as directed by a healthcare professional. Continuous use is advised for sustained benefits, but consulting with a healthcare provider for personalized advice and duration is important.

Helps with these DNA Risks:

- GCLC (Glutathione & Detox)
- Phase I Detox
- SOD2 (Oxidative Stress)
- SOD3 (Oxidative Stress)
- UGT2A1 (Cognition)

13 Sulforaphane

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

Helps with these Symptoms & Conditions:

- High Blood Pressure

Helps with these Goals:

- Immunity
- Mood

Helps with these DNA Risks:

- GCLC (Glutathione & Detox)
- PON1 (Detox)
- SOD2 (Oxidative Stress)
- SOD3 (Oxidative Stress)

14 Avoid Endocrine Disruptors

Minimize exposure to endocrine disruptors by opting for organic foods to reduce pesticide intake, using glass or stainless steel instead of plastic containers for food and beverages, avoiding cosmetics and personal care products with parabens or phthalates, and regularly vacuuming and dusting your home to reduce contact with flame retardants found in household dust.

Helps with these DNA Risks:

- PBDE Sensitivity (CYP2B6)
- Phase I Detox

15 Avoid PBDE

To avoid PBDEs, replace old furniture that might contain these chemicals, vacuum and dust your home regularly to reduce dust particles that might contain PBDEs, and use products labeled as PBDE-free. Ensure you check labels on electronics, furnishings, and textiles for mention of being PBDE-free.

Helps with these DNA Risks:

- PBDE Sensitivity (CYP2B6)