









Bluegrass Precision Wellness Report

Health Report

REPORT CATEGORIES —

-  MENTAL HEALTH
-  NUTRITION
-  FOOD SENSITIVITIES
-  HEART & BLOOD VESSELS
-  SLEEP
-  DETOX
-  FITNESS
-  SKIN & BEAUTY

Sample Client

Report date: 30 April 2026

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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.

Personal information

NAME

Sample Client

SEX AT BIRTH

Male

HEIGHT

5ft 10" 178cm

WEIGHT

215lb 97.5kg

REPORT PROVIDED BY

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Summary

Welcome to your personalized genetic health analysis, a comprehensive exploration of how your unique DNA influences virtually every aspect of your health and well-being. This report examines the fundamental genetic variations that shape your body's responses to everything from the foods you eat to the way you sleep, exercise, and age.

Your genetic code serves as a blueprint that influences countless biological processes—from how quickly you metabolize caffeine and absorb vitamins to your susceptibility to certain health conditions and your body's ability to recover from exercise. Understanding these genetic predispositions empowers you to make informed decisions about your lifestyle, nutrition, fitness routines, and health monitoring strategies.

This analysis covers nine key areas of your genetic health profile. The Foundational Genomics section explores essential genetic variants, including **APOE and MTHFR**, that affect metabolism and disease risk. Sleep genetics reveal how your genes influence rest quality and caffeine sensitivity. Diet & Nutrition examines your body's unique responses to macronutrients, vitamins, and food sensitivities.

Your genetic profile also influences detoxification capacity (Phase 1 Detox), skin health and aging, fitness performance and injury risk, brain health and cognitive function, cardiovascular disease susceptibility, and more.

Rather than viewing genetics as destiny, this report presents your genetic information as valuable insights that can guide personalized health strategies. By understanding your genetic strengths and vulnerabilities, you can optimize your approach to nutrition, exercise, sleep, and preventive care, ultimately supporting your long-term health and vitality.

This summary report contains:

64 Genetic Results

15 Recommendations











4 Lifestyle Assessments

Overview of Your Results













Foundational Genomics

 LOWER ACTIVITY MTHFR Likely lower MTHFR activity	 E3/E3 APOE You carry two APOE ε3 variants	 HIGHER ACTIVITY CYP1A2 (Detox) Likely higher CYP1A2 activity
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Sleep

 LOWER LEVELS Melatonin Predisposed to lower melatonin levels	 TYPICAL Sleep Quality Predisposed to typical sleep quality	 TYPICAL Sleep Duration Predisposed to typical sleep duration
 TYPICAL LIKELIHOOD Restless Legs Typical likelihood of restless legs syndrome	 TYPICAL LIKELIHOOD Sleep Movement Typical likelihood of sleep movement	 TYPICAL LIKELIHOOD Caffeine-Related Sleep Problems Typical likelihood of caffeine-related sleep problems
 DEEP Deep Sleep (ADA) Likely a deep sleeper	 LOWER Sleep Latency Predisposed to lower sleep latency	 LOWER Caffeine Sensitivity Predisposed to lower caffeine sensitivity
 LESS LIKELY Daytime Sleepiness Less likely to experience daytime sleepiness		






Diet & Nutrition

<p> HIGHER Alcohol Sensitivity</p> <p>Likely higher sensitivity to alcohol</p>	<p> WORSE RESPONSE Protein Metabolism</p> <p>Predisposed to worse protein metabolism</p>	<p> INCREASED NEED Folate (Vitamin B9)</p> <p>Likely increased need for folate</p>
<p> INCREASED NEED Vitamin B6 (Pyridoxine)</p> <p>Likely increased need for vitamin B6</p>	<p> TYPICAL LIKELIHOOD Tendency to Overeat</p> <p>Typical likelihood of overeating</p>	<p> TYPICAL GENETICS HLA-DQ (Gluten)</p> <p>Likely typical HLA-DQ genetics</p>
<p> TYPICAL Carbohydrate Metabolism</p> <p>Predisposed to typical carbohydrate metabolism</p>	<p> TYPICAL RESPONSE Saturated Fat</p> <p>Predisposed to typical saturated fat response</p>	<p> TYPICAL NEED Vitamin B12</p> <p>Likely typical need for vitamin B12</p>
<p> TYPICAL NEED Biotin</p> <p>Likely typical need for biotin</p>	<p> TYPICAL Salt Sensitivity</p> <p>Likely typical sensitivity to salt</p>	<p> TYPICAL NEED Potassium</p> <p>Likely typical need for potassium</p>
<p> TYPICAL NEED Magnesium</p> <p>Likely typical need for magnesium</p>	<p> LIKELY TOLERANT Lactose Intolerance</p> <p>Likely lactose tolerant</p>	<p> LESS LIKELY Binge Eating</p> <p>Less likely to binge eat</p>
<p> BETTER Fat Metabolism</p> <p>Predisposed to better fat metabolism</p>		




Phase I Detox


<p> TYPICAL METABOLIZER CYP2C9 (Detox)</p> <p>Likely a typical metabolizer</p>	<p> TYPICAL Sensitivity to Foodborne Mold</p> <p>Likely typical sensitivity to foodborne mold</p>	<p> TYPICAL ACTIVITY CYP2D6 (Mental Health, Detox)</p> <p>Predisposed to a typical CYP2D6 activity</p>
<p> TYPICAL ACTIVITY CYP2B6 (Drug Metabolism)</p> <p>Predisposed to a typical CYP2B6 activity</p>		

Skin Health

<p> MORE LIKELY Melanoma</p> <p>More likely to get melanoma</p>	<p> TYPICAL Glycation</p> <p>Predisposed to typical glycation</p>	<p> TYPICAL LIKELIHOOD Basal Cell Carcinoma</p> <p>Typical likelihood of basal cell carcinoma</p>
<p> TYPICAL ACTIVITY MC1R (Pigmentation & Skin Damage)</p> <p>Likely typical MC1R activity</p>	<p> TYPICAL Facial Wrinkles</p> <p>Predisposed to a typical amount of facial wrinkles</p>	

Fitness


<p> LOWER Muscle Mass</p> <p>Predisposed to lower muscle mass</p>	<p> TYPICAL Muscle Recovery</p> <p>Predisposed to typical muscle recovery</p>	<p> TYPICAL Strength</p> <p>Predisposed to typical strength</p>
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 **TYPICAL ACTIVITY**
COL5A1 (Collagen)

Likely typical COL5A1 activity

 **TYPICAL LIKELIHOOD**
Achilles Tendon Injury


Typical likelihood of Achilles tendon injury

 **HIGHER**
Endurance

Predisposed to higher endurance


 **HIGHER ACTIVITY**
COL1A1 (Collagen)

Predisposed to higher COL1A1 activity


 **LESS LIKELY**
Tendon Injury

Less likely to have tendinopathy


Brain Health

 **WORSE**
GAD1 (Glutamate/GABA)


Likely worse GAD1 genetics

 **MORE LIKELY**
THC and Psychosis


More likely to experience psychotic symptoms from cannabis use

 **INCREASED**
Psychedelic Effects of THC (Functional)


Predisposed to increased psychedelic effects of THC

 **TYPICAL LIKELIHOOD**
Anxiety


Typical likelihood of anxiety

 **TYPICAL**
Cognitive Function


Predisposed to typical cognition

 **TYPICAL**
Attention

Typical likelihood of ADHD

 **TYPICAL**
Memory Performance


Predisposed to typical memory performance

 **TYPICAL LIKELIHOOD**
Alzheimer's Disease

Typical likelihood of Alzheimer's disease


 **E3/E3**
APOE

You carry two APOE ε3 variants


 **LESS LIKELY**
Cognitive Decline

Less likely to have cognitive decline


Heart & Blood Vessels

 **MORE LIKELY**
Coronary Artery Disease


More likely to have coronary artery disease

 **TYPICAL ACTIVITY**
NOS3 (Cardiovascular)

Likely typical NOS3 activity


 **TYPICAL LEVELS**
Lipoprotein(a)

Predisposed to typical Lipoprotein(a) levels


 **TYPICAL LIKELIHOOD**
Atrial Fibrillation

Typical likelihood of atrial fibrillation


Miscellaneous

 **MORE LIKELY**
Asthma

More likely to get asthma

 **TYPICAL**
Insulin Resistance

Predisposed to typical insulin resistance

 **TYPICAL LIKELIHOOD**
Hashimoto's Disease

Typical likelihood of Hashimoto's disease

 **LESS LIKELY**
Graves' Disease

Less likely to have Graves' disease

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		DOSAGE
1 Aerobic Exercise (Cardio)	1 hour	2 Strength Training	1 hour
3 Omega-3 (Fish Oil)	500 mg	4 Vitamin C	500 mg
5 Music Therapy	30 minutes	6 Mediterranean Diet	
7 Yoga	30 minutes	8 Ginkgo	120 mg
9 Relaxation Techniques	30 minutes	10 Magnesium	250 mg
11 Tai Chi	1 hour	12 Avoid Air Pollution	
13 Meditation	30 minutes	14 Methylfolate	400 mcg
15 Walking	30 minutes		

Your Results in Details




Foundational Genomics


This Foundational Genomics section examines the genetic factors that shape how our bodies respond to everyday compounds and biological processes, with particular attention to **caffeine and melatonin metabolism, APOE status, and MTHFR genetic variants**.

Understanding these genetic influences can reveal how individual variations affect our capacity to process caffeine, how effectively melatonin functions in our sleep regulation, APOE's contribution to cholesterol processing and Alzheimer's disease susceptibility, and the ways MTHFR genetic differences influence folate metabolism and general health outcomes.

This genetic information plays a crucial role in recognizing personal variations in nutritional requirements, sleep patterns, health risks, and how our bodies utilize nutrients. This foundational genomics exploration aims to be both informative and empowering, offering valuable insights into how our distinct genetic profiles influence our health and well-being.

 **LOWER ACTIVITY**
MTHFR

Likely lower MTHFR activity

 **E3/E3**
APOE

You carry two APOE ε3 variants

 **HIGHER ACTIVITY**
CYP1A2 (Detox)

Likely higher CYP1A2 activity

MTHFR

Key Takeaways:

- MTHFR is an enzyme that helps your body process folate, an important nutrient for many body functions and processes.
- If you have lower MTHFR activity due to genetics, make sure you include folate-rich foods in your diet, like fruits and vegetables or other fortified foods. This is even more important with pregnancy.

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

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

Read [this blog post](#) for more details about MTHFR variants and potential ways to reduce their impact.

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

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

Some sources recommend methylfolate supplements instead of folic acid. Methylfolate supplements would in theory bypass the MTHFR enzyme, which converts folic acid to methylfolate. However, even if you have lower-activity *MTHFR* variants, experts say you can still process folic acid without any issues [R].

Importantly, CDC notes that folic acid is the only folate supplement proven to reduce neural tube defects. Methylfolate supplements have not been properly studied [R].

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

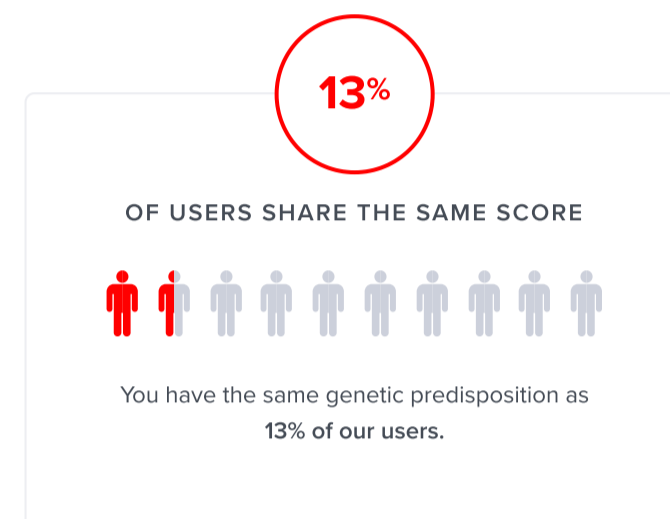
Good sources of riboflavin include [R, R]:

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



LOWER ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MTHFR	rs1801133	AA
MTHFR	rs1801131	TT

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

APOE

Key Takeaways:

- If you carry one or both **ε4** variants, your risk for Alzheimer's disease may be higher.
- The risk is greatest for late onset (after age 65) Alzheimer's disease.
- Even if your risk is higher due to the **ε4** variants, numerous other factors from your environment to lifestyle to other genetic variants impact overall risk.
- People with both variants may never get Alzheimer's, and some who have neither variant can get the disease.

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

ε2, ε3, and ε4 change the shape of the ApoE protein. This can impact how well ApoE functions [R, R].

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

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

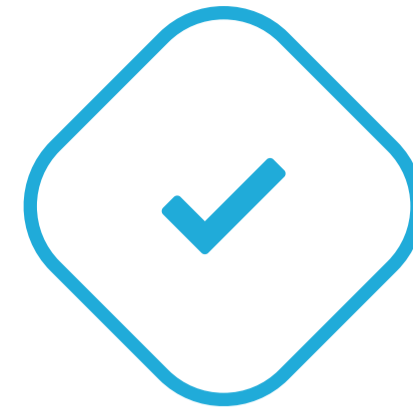
ε2 is another less common variant. It makes a protein that is better than ε3 at removing plaque from the brain, but not as good at removing fats from the blood. ε2 has been linked to a lower risk of Alzheimer's disease [R, R, R].

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

Did you know? The **ε4** variant was much more common among ancient hunter-gatherers. Scientists suggest this variant might have improved their [R]:

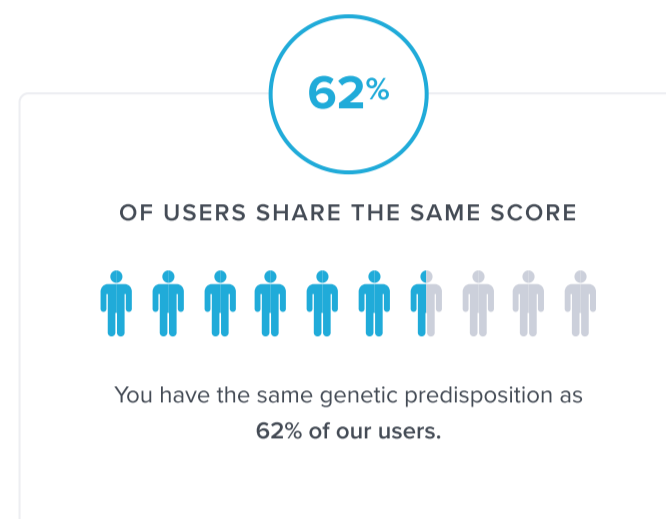
- Inflammatory response to germs in the wilderness
- Vitamin D status in less sunny European areas
- Aerobic endurance, crucial for a hunter-gatherer lifestyle

As humans largely switched to farming, some effects of this variant became useless or even harmful. For this reason, evolution strongly favored the **ε3** variant in ancient farmers and their modern descendants [R].



E3/E3

You carry two APOE ε3 variants based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
APOE	rs7412	CC
APOE	rs429358	TT

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]
- Evodia
- Reishi
- Andrographis,
- Modafinil
- Glycyrrhizin (liquorice)

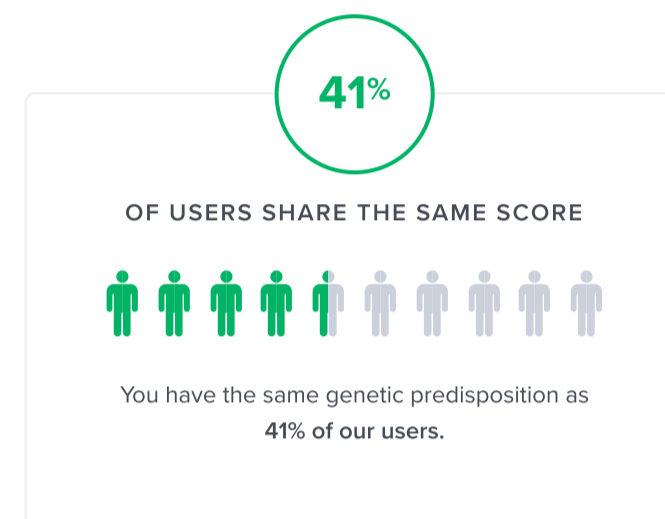
The following factors and substances may **decrease** CYP1A2 activity:

- 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.



Sleep

This section explores the complex genetic factors that influence your sleep patterns, quality, and overall sleep health. Understanding these genetic variations provides insights into individual differences in sleep architecture, caffeine sensitivity, and circadian rhythm regulation.

Your genetic makeup plays a significant role in determining how long you sleep, how well you sleep, and how external factors like caffeine affect your rest. This analysis examines various aspects of sleep biology, from basic sleep metrics to specific genetic variants that influence your body's response to stimulants and sleep-regulating hormones.

This comprehensive sleep assessment is designed to help you understand your unique sleep profile, empowering you to make informed decisions about sleep hygiene, caffeine consumption timing, and lifestyle choices that support optimal rest and recovery.

Topics include:


- Time in Bed
- Sleep Sensitivity to Caffeine
- Excessive Sleepiness
- Sleep Movement
- Melatonin



LOWER LEVELS

Melatonin


Predisposed to lower melatonin levels



TYPICAL

Sleep Quality


Predisposed to typical sleep quality



TYPICAL

Sleep Duration


Predisposed to typical sleep duration



TYPICAL LIKELIHOOD

Restless Legs


Typical likelihood of restless legs syndrome



TYPICAL LIKELIHOOD

Sleep Movement


Typical likelihood of sleep movement



TYPICAL LIKELIHOOD

Caffeine-Related Sleep Problems


Typical likelihood of caffeine-related sleep problems



DEEP

Deep Sleep (ADA)


Likely a deep sleeper



LOWER

Sleep Latency

Predisposed to lower sleep latency



LOWER

Caffeine Sensitivity

Predisposed to lower caffeine sensitivity



LESS LIKELY

Daytime Sleepiness

Less likely to experience daytime sleepiness

Melatonin

Key takeaways:

- Up to **80%** of differences in people's melatonin levels may be due to genetics.
- Low melatonin is a common issue. It may disrupt sleep and cause tiredness.
- Artificial light, stress, caffeine, alcohol, and tobacco may reduce melatonin levels.
- Lack of sunlight, liver issues, and some supplements and drugs may increase melatonin levels too much.

Up to 80% of differences in people's melatonin levels may be due to genetics [\[R\]](#).

Genes involved in melatonin levels may influence [\[R\]](#), [\[R\]](#):

- Light sensitivity
- Sleep-wake cycle
- Brain development and health
- Mental health

Factors linked to **low melatonin levels** include [\[R\]](#), [\[R\]](#), [\[R\]](#):

- Artificial light, including your phone and TV
- Long daylight in the summer
- Stress
- Caffeine, alcohol, tobacco
- Late-night physical activity
- Some drugs (e.g., beta-blockers)
- Aging

Excessive melatonin levels may result from [\[R\]](#), [\[R\]](#):

- Colder and darker winter months
- Liver issues
- Supplements (e.g., 5-HTP, tryptophan, vitamin B3 or B6)
- Some antidepressants



LOWER LEVELS

Predisposed to lower melatonin levels based on 5 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GALNT13	rs7571016	GG
GALNT15	rs142037747	GG
LDLRAD3	rs9645614	AA
ANXA2R	rs6451653	GG
ZFH3	rs17681554	AA

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

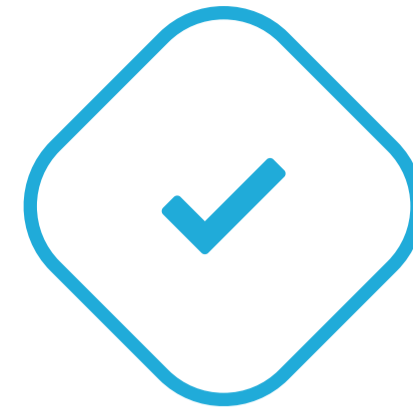
Sleep Quality

The quality of your sleep can have a big impact on how much energy you have during the day [R, R].

Many genetic variants influence sleep [R]. However, your environment and your habits also affect sleep quality.

Some strategies that may improve sleep quality include [R]:

- Reducing your bright light exposure (screen time) in the evenings
- Sticking to a regular sleep schedule
- Avoiding hunger or large meals before bed
- Avoiding nicotine, caffeine, and alcohol before bed
- Maintaining a sleep area that's cool, dark, and quiet



TYPICAL

Predisposed to typical sleep quality based on 576,285 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
/	rs71365296	AA
KANSL1	rs1107820	TT
VGLL2	rs4946246	TG
RALYL	rs118149821	CC
CHRM2	rs146885652	GG
FOXO6	rs2226263	TT
/	rs184060364	GG
TENM4	rs117191802	AA
TCF21	rs13201465	AA
RALYL	rs191939331	GG
FBN2	rs115375165	GG
TJP2	rs7030480	AA
COQ8A	rs113207574	CT
PRICKLE1	rs11829548	GT
ADCY1	rs79209880	CC
CHRM2	rs74757156	CC
/	rs111921861	AA
/	rs140707667	GG
FUT9	rs142123475	CC
UFL1	rs75842709	CC
COX7C	rs2964898	CC
ERCC4	rs74321030	TT
VRK1	rs78807545	GG
/	rs147738873	CC
MSX2	rs28450080	CC
RFX4	rs11610873	GG
CDH13	rs111702115	GG
CASP3	rs7695597	AA
TNF	rs1800629	GG

GENE	SNP	GENOTYPE
NREP	rs140529718	GG
EPB41L4A	rs146128029	TT
RBMS3	rs17023449	TT
SASH1	rs112390069	GG
FAM107B	rs74122981	AA
PLK2	rs76395602	GG
RGS6	rs36032616	AA
PIGZ	rs4916588	CC
PLK2	rs170741	TT
ENC1	rs76768179	TT
ZNF626	rs6511152	CC
CD36	rs4437584	TT
LAMA2	rs11962701	CC

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

Sleep Duration

The amount of sleep you need depends on many factors, such as your age. Adults typically need 7-9 hours of sleep per night [R, R, R].

How much sleep you actually get may depend both on your genes and the environment. Around 50% of the differences in sleep duration between people may be due to genetics [R].

Many of the genes that affect sleep duration are involved in the circadian clock. This is our internal clock that helps control and adjust the sleep-wake cycle to the 24-hour cycle of the Earth's day [R, R, R, R].

Strategies that may help you get the recommended amount of sleep include [R]:

- Sticking to a regular sleep schedule
- Maintaining a sleep area that's cool, dark, and quiet
- Reducing your bright light exposure (screen time) in the evenings
- Avoiding hunger or large meals before bed
- Avoiding nicotine, caffeine, and alcohol before bed



TYPICAL

Predisposed to typical sleep duration based on 7,192,262 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
IGKV10R2-108	rs62158206	TT
MBOAT2	rs79512144	AA
ZCCHC7	rs10973207	GG
RBFOX1	rs77684884	AA
TTC21B	rs12463754	CC
COG5	rs6979198	TT
LPCAT1	rs365663	GG
DAB1	rs540431	GG
/	rs12791153	AT
VRK2	rs116219610	TC
CLOCK	rs12649507	AG
SEMA6D	rs13329140	GA
TCF4	rs12607679	CT
PAM	rs6889592	GA
FOXP2	rs1668331	TG
ANKK1	rs17601612	GC
PAX8	rs2863244	AG
BUD13	rs1263056	GA
BANK1	rs13109404	TT
ERC2	rs112230981	AA
NRXN3	rs11621908	CC
ACTR1A	rs144625846	AA
CLOCK	rs2070062	AC
MRM2	rs3823624	CC
ZSCAN23	rs34388845	AA
VRK2	rs11682175	CC
H2BC12	rs12215241	GG
MAPT	rs62061734	TT
FTO	rs8047587	GG

GENE	SNP	GENOTYPE
ZFP57	rs1633063	CC
PM20D2	rs9451146	CC
PRKG2	rs41501452	AA
GPD2	rs11883686	AA
COG5	rs7778250	AA
/	rs9362971	TT
/	rs4688116	TT

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

Restless Legs

Key Takeaways:

- Around **60%** of the differences in people's risk of developing RLS may be due to genetics.
- Other risk factors include iron deficiency, pregnancy, toxicurea levels, and certain medical conditions.
- RLS is fairly common, occurring in about **1 in 10 people**.
- If you have a high genetic risk, your overall risk may be lowered by taking action on risk factors that you can change.
- Click the **Recommendations** tab for potential dietary and lifestyle changes and **next steps** for relevant labs.

Restless legs syndrome (RLS) is a condition that causes an uncontrollable urge to move the legs. It is relatively common - around **1 in 10 people** have it [R].

Experts aren't sure what causes RLS. There is evidence that it may have to do with iron or dopamine levels in the brain [R, R].

RLS occurs in all age groups. Factors that may raise the risk for RLS include [R, R]:

- Iron deficiency
- Pregnancy
- Toxic buildup of urea in the blood (often found in people with kidney disease)
- Other chronic medical conditions
- **Genetics**

The feelings associated with RLS are described as pulling, creeping, and tugging. Typically, RLS symptoms [R]:

- Begin with rest, such as driving or sitting at a movie theater
- Relieve with movement, such as stretching, jiggling the legs, or walking
- Worsen in the evening
- May be associated with limb twitching

If left untreated, RLS may disrupt sleep. This can lead to daytime sleepiness, irritability, or problems at work.

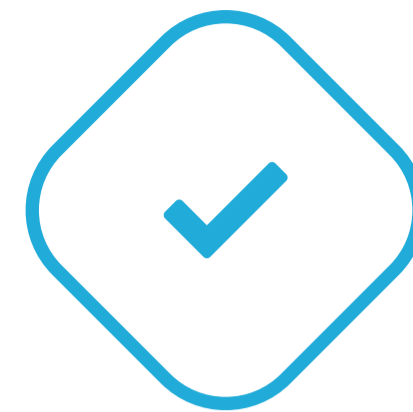
The following lifestyle changes can help improve symptoms [R, R]:

- Exercise
- Massage
- Applying heat using a heating pad or a warm bath
- Avoiding or limiting caffeine, alcohol, and tobacco

Some supplements, like iron, may help people with RLS who have low iron. For severe cases, a doctor may prescribe medications.

Around 60% of the differences in people's risk of developing RLS may be due to genetics. Genes involved with RLS may influence [R, R, R, R]:

- Iron metabolism
- Nerve function
- Dopamine activity



TYPICAL LIKELIHOOD

Typical likelihood of restless legs syndrome based on 34 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GLO1	rs9296249	TT
GLO1	rs9357271	TT
GLO1	rs3923809	GA
MEIS1	rs2300478	GT
SEMA6D	rs111652004	GG
TOX3	rs3104767	GT
CCDC167	rs17636328	AA
MEIS1	rs6747972	AG
HOXB2	rs12450895	AA
PTPRD	rs62535767	CC
PRMT6	rs12046503	CT
ZNF804B	rs10952927	AG
C1D	rs1820989	AC
PIK3R4	rs35987657	GA
PKP4	rs80319144	TC
OPRL1	rs365032	AG
RANBP17	rs10068599	CT
CRBN	rs1848460	AT
SLC40A1	rs10188680	TA
PMAIP1	rs58127855	CC
MEIS1	rs113851554	GG

GENE	SNP	GENOTYPE
MAP2K5	rs12593813	AA
PTPRD	rs4626664	GG
PTPRD	rs1975197	GG
MICALL2	rs112716420	CC
DPH6	rs996064	AA
DCDC2C	rs10208712	GG
DACH1	rs340561	GG
SETBP1	rs12962305	CC

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

Sleep Movement

Have you ever been told you move while you sleep? Sleep movements are common. They don't usually disturb the mover's sleep. However, when they do disrupt sleep, they can make a person sleepy during the day [R, R].

People may be more likely to experience periodic limb movements as they age. About 1 in 3 people over the age of 60 experience sleep movements [R, R]

Other factors that may influence sleep movement include [R, R]:

- **Sex** – men tend to move more in their sleep
- **Diet** – a lack of iron in the diet may make sleep movements worse
- **Genetics**

Scientists have linked several genes to increased sleep movement. For example:

- **MEIS1** gene, which influences the iron metabolism in the brain [R, R, R, R, R].
- **BTBD9** gene, which may influence the brain dopamine metabolism [R, R, R, R, R].
- **TOX3** gene, which may play an important role in nerve cell activity [R, R, R].



TYPICAL LIKELIHOOD

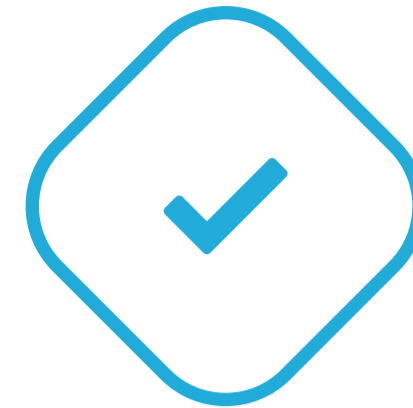
Typical likelihood of sleep movement based on 7,138,059 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MEIS1	rs2300478	GT
GLO1	rs3923809	GA
TOX3	rs3104788	TC
MEIS1	rs113851554	GG

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

Caffeine-Related Sleep Problems



TYPICAL LIKELIHOOD

Typical likelihood of caffeine-related sleep problems based on 18 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

Caffeine is a well-known stimulant. It can promote wakefulness and interrupt sleep by reducing the activity of [adenosine](#), a sleep-promoting compound that builds up while we are awake and decreases while we sleep. **Caffeine’s effects on sleep tend to be strongest in the evening, before bedtime.** Older people and those who sleep less may be more sensitive to caffeine [[R](#), [R](#), [R](#)].

Up to **40%** of differences in caffeine-related sleep problems may be due to genetics. Interestingly, the majority of these genetic factors are specific to caffeine, and they don’t affect sleep in general [[R](#)].

A gene called [ADORA2A](#) helps make a receptor for adenosine. Caffeine promotes wakefulness and interrupts sleep by blocking this receptor. Unsurprisingly, a variant belonging to this gene, [rs5751876](#), is linked to caffeine-related sleep problems [[R](#), [R](#), [R](#)].

What’s surprising is the opposite effect of this variant on caffeine-related anxiety. **People carrying the “T” allele may be less prone to sleep problems but more prone to anxiety due to caffeine intake** [[R](#), [R](#), [R](#)].

Other gene variants linked to caffeine-related sleep problems may affect:

- Melatonin ([MTNR1B](#))
- Compounds similar to adenosine ([GBP1](#), [GBP4](#))
- Brain function ([ADGRL2](#))
- Heart function ([NEBL](#))

Please note: Your other gene variants, lifestyle, and environment may also affect caffeine-related sleep problems.

GENE	SNP	GENOTYPE
ADORA2A	rs5751876	CT
NEBL	rs9665295	CC
ALDH7A1	rs13172305	GG
FCHSD2	rs1791933	TC
KCTD15	rs11878836	TT
MDFIC2	rs7628219	CC
GBP7	rs521704	CA
ADGRL2	rs12725617	CT
COMT	rs4680	GA
PRIMA1	rs6575353	AG
PRIMA1	rs12895096	GT
/	rs16905439	CC
TMEM51	rs2103117	GG
GBP3	rs12407812	CC
ADORA2A	rs2065779	GG
SLC7A1	rs2388082	CC
MTNR1B	rs10830964	CC
LRTM1	rs11706236	AA
NEDD4L	rs158856	CC

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

Deep Sleep (ADA)

Deep sleep is one of the stages of non-REM sleep. It is also called *slow-wave sleep* or *delta-wave sleep* because the brain emits slow (delta) waves during this stage [R, R, R].

A compound called [adenosine](#) builds up in the brain while we are awake and peaks at bedtime, causing us to feel sleepy. The longer we stay awake, the sleepier we get. Adenosine decreases while we sleep, so we feel rested when we wake up [R].

The [ADA](#) gene helps make an enzyme that breaks down adenosine. Scientists suggest that one variant in this gene reduces the enzyme's activity. This variant is linked to a faster buildup of adenosine [R].

People with at least one copy of this variant tend to [R, R, R]:

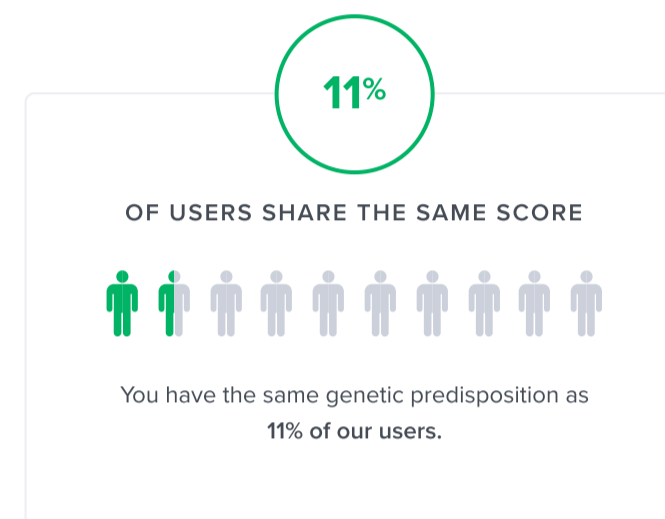
- Be sleepier
- Awaken less often during the night
- Have longer deep sleep
- Have stronger slow (delta) waves

[Caffeine](#) can promote wakefulness and interrupt sleep by reducing adenosine activity. However, it's still not clear if the effects of caffeine on sleep depend on this gene variant [R].



DEEP

Likely a deep sleeper based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ADA	rs73598374	TC

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

Sleep Latency

How long does it take for you to fall asleep? Do you pass out as soon as your head hits the pillow? Or do you toss and turn for what seems like hours, before you drift asleep? The time it takes for you to fall asleep is referred to as **sleep latency** [R].

Many factors can affect your sleep latency, including your genes. About **20-45% of differences in sleep latency are due to genetics**. Scientists identified several genes that play a key role in sleep latency by influencing the release of neurotransmitters such as GABA or by affecting the setting of the body's internal clock [R, R, R].

If you are struggling to fall asleep at night, these may improve your sleep latency [R, R, R, R, R, R]:

- Sticking to a regular sleep schedule and having a consistent sleep routine
- Maintaining a sleep area that's cool, dark, and quiet
- Avoiding hunger or large meals before bed
- Avoiding nicotine, caffeine, and alcohol before bed
- Getting enough daytime exposure to natural light
- Getting regular physical activity
- Addressing stress and anxiety, e.g. by mindfulness and relaxation techniques



LOWER

Predisposed to lower sleep latency based on 7,281,604 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MDFIC2	rs147786325	TT
SLC26A7	rs77942316	GA
SLC26A7	rs61134957	AC
NDUFA4	rs73678284	CC
IL21	rs62324204	TC
SP140L	rs17273810	AC
MCTP2	rs11074250	AT
PMP22	rs9907309	GA
CSNK2A1	rs112815007	AA
CSNK2A1	rs74448913	TT
CSMD1	rs62480124	GG
FRAS1	rs6842385	GG
/	rs62217115	TT
GRM7	rs185934952	GG
RANBP3L	rs114965806	CC
ERCC4	rs12934187	CC
CDC42EP3	rs144261420	AA
HLF	rs79692461	AA
KLHL29	rs62126824	AA
RANBP3L	rs74541420	CC
MALRD1	rs2686656	AA
NAT1	rs7845127	CC
VAV3	rs17556028	CC

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

Caffeine Sensitivity

Genetic variants may explain about **70%** of the difference in caffeine sensitivity [R, R]. Genes associated with caffeine sensitivity impact how quickly caffeine is broken down and the strength of its effects in different parts of the body.

The **CYP1A2** gene is a **key enzyme responsible for caffeine breakdown**. The **rs762551** variant (-163 A>C) determines whether someone is a “fast” or “slow” caffeine metabolizer. Carriers of the “C” allele are slow metabolisers. This means they caffeine stays in their body for a longer time, increasing their caffeine sensitivity [R, R, R, R].

Other notable genes and variants include:

- **AHR (rs4410790)**: The AHR gene regulates the expression of **CYP1A2**, mentioned above. Certain variants at AHR slow down caffeine metabolism, prolonging the effects caffeine has on the body [R].
- **ADORA2A (rs5751876)**: This ADORA2A gene encodes the adenosine A2A receptor, a primary target of caffeine in the brain. Caffeine may make those with the “T” variant more anxious, especially women. Interestingly, this variant seems to have the opposite effects on sleep problems (“C” carriers may be more affected) [R, R, R, R].
- **COMT (rs4680)**: The COMT gene is involved in the breakdown of the neurotransmitter dopamine. The rs4680 variant (Val158Met G>A) may increase sensitivity to both positive and negative effects of caffeine [R].
- **NAT2 (R/S, rs1495741)**: NAT2 helps break down various substances, including caffeine. Individuals with a “slow” acetylator status in the NAT2 gene may break down caffeine more slowly, leading to prolonged effects and increased sensitivity [R].

These genetic variations collectively influence individual sensitivity to caffeine, determining both the intensity and duration of the response to caffeine in the body. Understanding these genetic factors can help tailor caffeine intake to avoid adverse effects and optimize performance and well-being.



LOWER

Predisposed to lower caffeine sensitivity based on 5 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
AHR	rs4410790	CT
ADORA2A	rs5751876	CT
NAT2	rs1495741	AG
COMT	rs4680	GA
CYP1A2	rs762551	AA

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

Daytime Sleepiness

Daytime sleepiness is the feeling of excessive sleepiness during the day. People with excessive daytime sleepiness often complain of not feeling well-rested, having difficulty waking up, or accidentally falling asleep during the day, among others [R, R, R].

Daytime sleepiness is usually a consequence of poor sleep duration or quality. Other risk factors include [R]:

- Shift work [R]
- Sleep disorders (obstructive sleep apnea, narcolepsy) [R, R]
- Mood disorders (depression, bipolar disorder) [R]
- Allergies (asthma, hay fever) [R, R]
- GERD [R]
- Parkinson’s disease [R]
- Epilepsy [R]
- Obesity [R]
- **Genetics**

Up to 40% of differences in people’s chances of experiencing daytime sleepiness may be due to genetics. Genes involved may influence the sleep/wake cycle and sleep duration or quality [R, R, R, R].

Interventions aimed at improving sleep duration and quality may also reduce daytime sleepiness. These include [R, R, R, R]:

- Reducing your bright light exposure (screen time) in the evenings
- Maintaining a sleep area that’s cool, dark, and quiet
- Sticking to a regular sleep schedule
- Avoiding hunger or large meals before bed
- Avoiding nicotine, caffeine, and alcohol before bed



LESS LIKELY

Less likely to experience daytime sleepiness based on 993,458 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ASB3	rs76645968	GG
PATJ	rs12140153	GG
/	rs920065	CC
AP3B2	rs17507216	AG
TMEM144	rs115320831	AG
FGGY	rs371398972	AA
AR	rs73536079	G
/	rs573716401	GG
/	rs553962214	TT
SLC24A3	rs565444861	GG
PTPRM	rs558006880	GG
LBP	rs6099524	CC
SLC2A9	rs543431433	TT
TENT5A	rs189689339	CC
FGGY	rs192315283	TT
TMEM132B	rs142261172	GG
/	rs182765975	GG

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



Diet & Nutrition


This section examines the genetic factors that influence how your body processes foods, nutrients, and dietary compounds. Understanding these genetic variations provides insights into individual differences in hunger regulation, macronutrient metabolism, vitamin absorption, and food sensitivities.

Your genetic profile affects everything from how you experience hunger and satiety to how efficiently you metabolize carbohydrates, fats, and proteins. This analysis explores your body's unique responses to different foods and nutrients, including potential sensitivities and optimal dietary approaches based on your genetic makeup.


This comprehensive nutritional genetics assessment empowers you to make personalized dietary choices that align with your genetic predispositions, supporting optimal health, energy levels, and overall well-being through targeted nutrition strategies.

Topics include:

- Hunger and Fullness
- Response to Macros - Optimal Diet
- Food Sensitivity
- Alcohol Sensitivity
- B Vitamins
- Electrolytes

 **HIGHER**
Alcohol Sensitivity

Likely higher sensitivity to alcohol

 **WORSE RESPONSE**
Protein Metabolism


Predisposed to worse protein metabolism

 **INCREASED NEED**
Folate (Vitamin B9)


Likely increased need for folate

 **INCREASED NEED**
Vitamin B6 (Pyridoxine)


Likely increased need for vitamin B6

 **TYPICAL LIKELIHOOD**
Tendency to Overeat


Typical likelihood of overeating

 **TYPICAL GENETICS**
HLA-DQ (Gluten)


Likely typical HLA-DQ genetics

 **TYPICAL**
Carbohydrate Metabolism

Predisposed to typical carbohydrate metabolism

 **TYPICAL RESPONSE**
Saturated Fat


Predisposed to typical saturated fat response

 **TYPICAL NEED**
Vitamin B12


Likely typical need for vitamin B12

 **TYPICAL NEED**
Biotin


Likely typical need for biotin

 **TYPICAL**
Salt Sensitivity


Likely typical sensitivity to salt

 **TYPICAL NEED**
Potassium


Likely typical need for potassium

 **TYPICAL NEED**
Magnesium


Likely typical need for magnesium

 **LIKELY TOLERANT**
Lactose Intolerance

Likely lactose tolerant

 **LESS LIKELY**
Binge Eating

Less likely to binge eat

 **BETTER**
Fat Metabolism

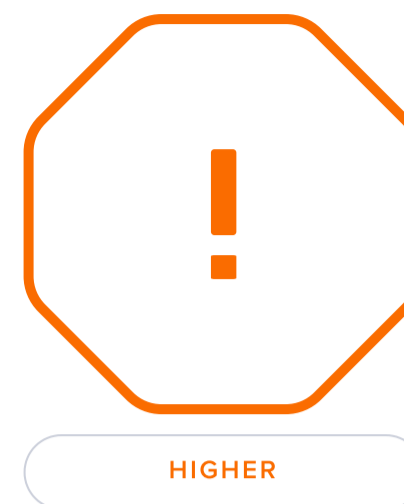
Predisposed to better fat metabolism

Alcohol Sensitivity

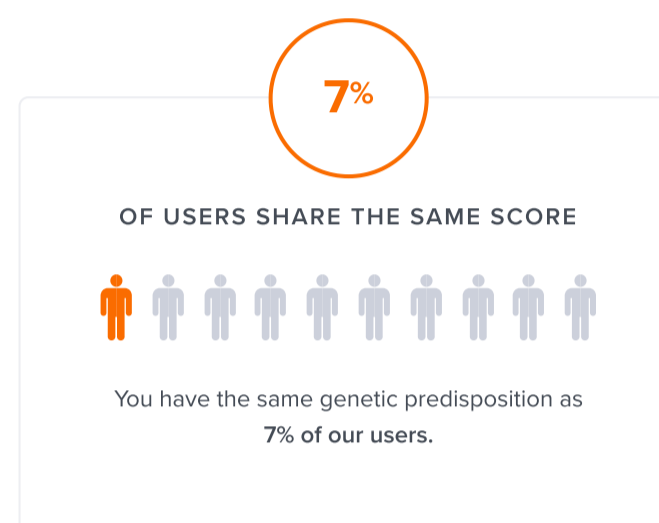
There are two enzymes involved in clearing alcohol from the body. They are coded by genes called [ADH1B](#) and [ALDH2](#). The first enzyme breaks down alcohol to toxic acetaldehyde, while the second one breaks down acetaldehyde into a less harmful substance [\[R\]](#).

Genetic variants that speed up the first enzyme or slow down the second enzyme contribute to the buildup of acetaldehyde and increase alcohol sensitivity [\[R\]](#).

Variants that increase the buildup of acetaldehyde are common in East Asian populations but rare in other parts of the world [\[R\]](#).



Likely higher sensitivity to alcohol based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ADH1B	rs1229984	CT
ALDH2	rs671	GG

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

Protein Metabolism

Some people do better on low- and others on high-protein diets. Your genes may affect your response to protein. Specifically, genes that affect your response to protein may also influence [\[R, R, R\]](#):

- Body weight
- Food preference
- Metabolism

In people with a better response to dietary protein, higher amounts of protein in a diet may improve weight control and metabolism. On the other hand, high-protein diets may have adverse metabolic effects in people with a worse response [\[R, R, R\]](#).

However, other variants and environmental factors may also influence your dietary protein response. Try to get most of your protein from healthy sources such as legumes, poultry, and fish.



WORSE RESPONSE

Predisposed to worse protein metabolism based on 17 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
FTO	rs9939609	TT
FTO	rs1558902	TT
GLP1R	rs6923761	GG
ST6GAL1	rs1501299	GG
CNDP2	rs4891558	TT
NADSYN1	rs12785878	GG
MTNR1B	rs10830963	CG
APOA1	rs670	CC
FUCA1	rs3123554	GA
NTN5	rs838147	GA
CLOCK	rs3749474	TC
UCP3	rs1800849	GG
ADRB3	rs4994	AA
FABP2	rs1799883	CC
TFAP2B	rs987237	AA
CNDP1	rs7244647	TT
TNF	rs1800629	GG

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

Folate (Vitamin B9)

Key Takeaways:

- Folate is an essential vitamin. It is particularly important for pregnant women.
- Folate is needed for brain and heart health, gene signaling, and to protect DNA. It is readily available in citrus fruits, green vegetables, and fortified foods.
- Your diet, smoking, heavy drinking, gut issues, and genes may impact folate levels.
- Click the **next steps** tab for relevant labs.

Vitamin B9, also known as folate or folic acid, is an essential nutrient. Most adults should get **400 micrograms (mcg)** of folate per day. Among other functions, folate helps the body make red blood cells [R, R, R, R].

Low levels of folate are not common. However, women may be at a higher risk than men [R, R].

Additional risk factors and causes of low folate levels include:

- **Low intake of fruits and vegetables** [R, R]
- Heavy drinking [R, R]
- Smoking [R, R]
- Gut issues such as inflammatory bowel disease (IBD) or celiac disease [R, R, R]
- Certain medications [R]

A variant in a gene called *MTHFR* is linked to slightly lower folate levels. People who carry two copies of this variant may have about 16% lower blood folate [R].

Genetically higher folate levels may be causally associated with [R, R, R, R, R, R, R, R]:

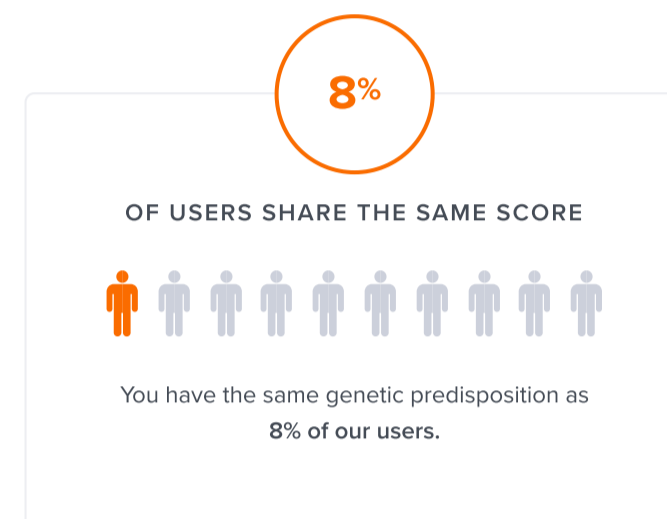
- IgE levels
- Fatty liver
- Vitiligo
- Joint pain
- HDL cholesterol
- Heart health (lower CHD)
- Stroke
- Gut inflammation (lower UC)

Read [this blog post](#) for more details about MTHFR variants and potential ways to reduce their impact.



INCREASED NEED

Likely increased need for folate based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MTHFR	rs1801133	AA

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

Vitamin B6 (Pyridoxine)

Some people may have genetically higher vitamin B6 levels than others. Genes involved may influence vitamin B6 metabolism [\[R\]](#), [\[R\]](#), [\[R\]](#).



INCREASED NEED

Likely increased need for vitamin B6 based on 5 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NBPF3	rs4654748	CC
ALPL	rs1256341	TT
NBPF3	rs1697421	CT
CBS	rs234706	GA
MTHFR	rs1801133	AA
PDXK	rs2010795	AG
ALPL	rs1772719	CA
PDXK	rs147242481	GG
ALPL	rs1256335	AA

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

Tendency To Overeat

Several genetic variants are associated with overeating behaviors, appetite control, and sensitivity to hunger and satiety signals. These variants influence hormonal regulation, reward pathways, and metabolic processes that can contribute to individual differences in eating behavior.

FTO (rs9939609, 87653 T>A): The *FTO* gene is strongly associated with appetite and obesity risk. The rs9939609 variant (T>A) can influence hunger and satiety, with the A allele linked to increased appetite and a higher likelihood of overeating and weight gain [R].

FTO (rs6713532, A>G): Another *FTO* variant, rs6713532 (A>G), has been linked to increased risk of obesity and a tendency toward overeating. Individuals with the G allele may have a higher susceptibility to weight gain due to increased appetite and reduced satiety signals.

LEPR (rs1805094, Lys656Asn G>C): *LEPR* encodes the leptin receptor, which is crucial for regulating hunger and satiety. The rs1805094 variant (Lys656Asn G>C) may impact leptin sensitivity, potentially affecting how full one feels after eating, influencing portion sizes and eating frequency.

LEPR (rs1137101, Gln223Arg A>G): Another variant in the *LEPR* gene, rs1137101 (Gln223Arg A>G), affects leptin signaling. The G allele is associated with a reduced feeling of fullness, which may lead to larger meal sizes and frequent hunger cues [R, R, R].

TAS2R38 (rs713598, Ala262Val C>T): *TAS2R38* is a taste receptor gene that affects taste sensitivity, particularly to bitter flavors. The rs713598 variant (Ala262Val C>T) can influence food preferences and eating behavior, potentially impacting overeating tendencies, especially in response to sweet or fatty foods [R, R, R].

TAS2R38 (rs1726866, Ala49Pro G>A): Another *TAS2R38* variant, rs1726866 (Ala49Pro G>A), affects bitter taste perception. Individuals with the A allele may have a preference for bitter-tasting vegetables like cruciferous greens. This variant can influence dietary choices and eating behaviors, indirectly impacting overall nutrition and weight management.

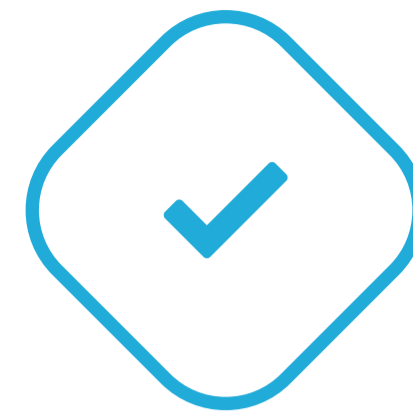
APOA2 (rs5082, -492 T>C): *APOA2* is involved in lipid metabolism and satiety regulation. The rs5082 variant (-492 T>C) has been associated with higher intake of saturated fats and a tendency to overeat, especially in individuals with the CC genotype.

CLOCK (rs1801260, 3111 T>C): The *CLOCK* gene influences circadian rhythms, which affect eating patterns and metabolism. The rs1801260 variant (3111 T>C) can disrupt regular eating schedules, potentially leading to increased food intake during irregular hours and contributing to overeating [R, R, R].

DRD2 (rs1800497, Taq1A C>T): *DRD2* encodes a dopamine receptor, which is involved in reward and pleasure pathways in the brain. The rs1800497 variant (Taq1A C>T) can affect dopamine sensitivity, potentially leading to increased cravings and reward-seeking behaviors related to food [R, R, R, R].

FAAH (rs324420, Pro129Thr C>A): *FAAH* is involved in endocannabinoid signaling, which influences appetite and pleasure from eating. The rs324420 variant (Pro129Thr C>A) can increase the tendency to snack and overeat, particularly high-fat foods, due to heightened pleasure responses to food.

MC4R (rs17782313, T>C): The *MC4R* gene is associated with energy balance and appetite control. The rs17782313 variant (T>C) has been linked to increased appetite and a higher risk of



TYPICAL LIKELIHOOD

Typical likelihood of overeating based on 20 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
POMC	rs6713532	TT
NPY	rs16147	TT
ANKK1	rs1800497	GA
CLOCK	rs1801260	AG
CNR1	rs1049353	TT
LEPR	rs1805096	GG
LEPR	rs1137101	AG
TAS2R38	rs713598	GC
LEP	rs7799039	GG
COMT	rs4680	GA
FAAH	rs324420	AC
TAS2R38	rs1726866	GA
LEPR	rs1137100	AG
TCF7L2	rs7903146	TC
FTO	rs9939609	TT
MC4R	rs17782313	TT
MC4R	rs12970134	GG
SLC2A2	rs5400	GG
FCER1G	rs5082	AA
NPY	rs16139	TT
POMC	rs1042571	GG

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

obesity, as individuals with the C allele may feel less satiated after eating [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

MC4R (rs12970134, G>A): The rs12970134 variant (G>A) near the *MC4R* gene has been linked to increased hunger and a reduced feeling of fullness. Individuals with the A allele may be more prone to overeating and weight gain due to a diminished satiety response, making appetite control more challenging [\[R\]](#).

SLC2A2 (rs5400, Thr110Ile C>T): SLC2A2 plays a role in glucose transport and blood sugar regulation. The rs5400 variant (Thr110Ile C>T) can influence carbohydrate cravings and eating patterns, with certain alleles associated with a preference for high-carb foods.

HADH (rs1042571, C>T): The *HADH* gene encodes hydroxyacyl-CoA dehydrogenase, an enzyme involved in fatty acid metabolism and energy regulation. The rs1042571 variant (C>T) has been associated with differences in metabolic efficiency and energy utilization.

NPY (rs16147, A>G): The *NPY* gene encodes the neuropeptide Y, which is involved in regulating stress response, appetite, and arousal. The rs16147 variant (A>G) has been associated with differences in appetite control and stress-induced eating behaviors. Individuals with the G allele may be more prone to overeating, especially in response to stress [\[R\]](#).

CNR1 (rs1049353, C>T): The *CNR1* gene encodes the cannabinoid receptor 1, which is involved in the endocannabinoid system that regulates appetite, mood, and reward pathways. Individuals with the T allele of rs1049353 may have an increased tendency to cravings and overeating, particularly for high-calorie or "comfort" foods, due to heightened activity in the brain's reward centers.

COMT (rs4680, Val158Met G>A): The *COMT* gene encodes an enzyme that breaks down dopamine in the brain, affecting mood, reward sensitivity, and impulse control. The rs4680 variant (Val158Met G>A) influences dopamine levels, with the A allele (Met) associated with slower dopamine breakdown. Individuals with the A allele may have heightened sensitivity to rewards, which can increase susceptibility to cravings and overeating.

ADRB3 (rs1805096, Trp64Arg T>C): The *ADRB3* gene encodes the beta-3 adrenergic receptor, which is involved in regulating fat breakdown and energy expenditure. The rs1805096 variant (Trp64Arg T>C) has been associated with reduced receptor function, leading to decreased fat metabolism and an increased risk of weight gain.

HLA-DQ (Gluten)

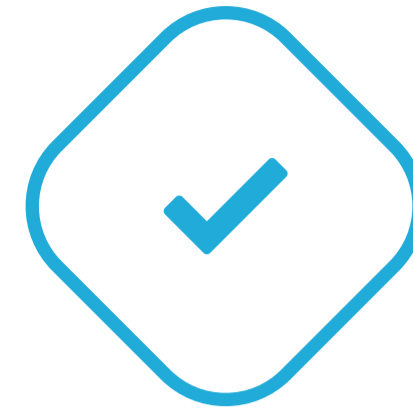
Different alleles in these genes can produce different types of HLA-DQ structures. The **DQ2** type (especially the DQ2.5 subtype) is present in up to 98% of celiac disease patients, depending on the population. That is among the strongest known links to autoimmunity in the entire HLA system [R, R].

Two alleles — DQA1*0501 and DQB1*0201 — form the DQ2.5 haplotype, which codes for the DQ2.5 receptor on white blood cells. The DQ2.5 receptor binds gluten and presents it to T-helper cells, initiating widespread gut inflammation [R, R].

The 'T' variant of [rs2187668](#) serves as a genetic marker — it tags the DQ2.5 haplotype with high precision. In other words, the vast majority of people with this allele will have this haplotype. A study of over 27,000 subjects identified this SNP as the primary genetic factor for celiac disease. People carrying the 'T' allele had over six times higher chances of being diagnosed with celiac disease. A smaller trial of 889 participants came to a similar conclusion [R, R].

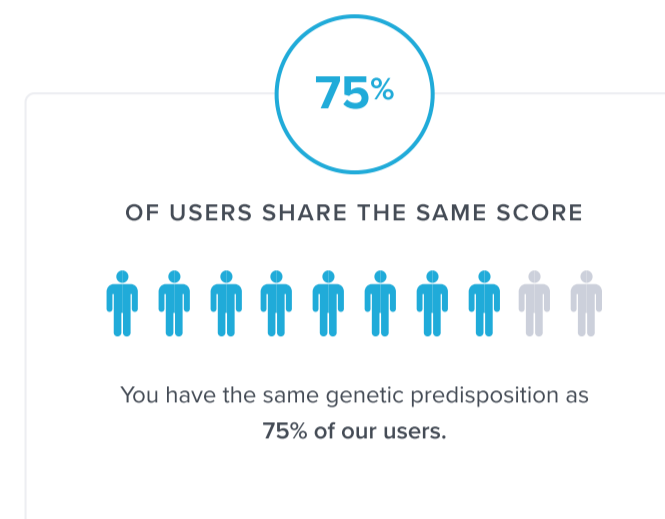
The 'C' variant of [rs74541084](#) tags the DQ8 haplotype, an additional marker for gluten sensitivity in people who don't carry DQ2.5.

Please note: this report only analyzes the HLA-DQ gene. Variants in many other genes have shown associations with gluten sensitivity.



TYPICAL GENETICS

Likely typical HLA-DQ genetics based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HLA-DQB1	rs2858331	AA
HLA-DQA1	rs2187668	CC
HLA-DQA2	rs7454108	TT

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

Carbohydrate Metabolism

For much of our history, we were all nomadic hunter-gatherers. We ate what we could find: roots, plants, berries, nuts, fish, and meat. This varied by location, climate, and season. At this point in time, the way the body processed and responded to complex carbs wasn't very relevant [R, R].

About 12,000 years ago, farming changed that. Suddenly, there were more starchy foods such as grains in our diets. More carbs meant more readily available energy. But this also meant more blood sugar spikes and a higher risk of metabolic disorders. Luckily, variants in genes like [TCF7L2](#) allowed us to process these new food sources in a more productive, less harmful way [R].

The [TCF7L2](#) gene affects insulin release after eating foods like grains. It is one of the genes most strongly associated with **diabetes**. Depending on which variant of this gene you carry, your body may respond differently to carbs [R].

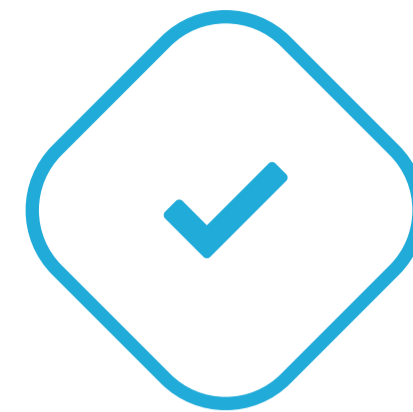
The "farmer" variant ([rs7903146-C](#)) is linked to a better response to carbs. **In people with this variant, carbs don't tend to spike blood sugar.** The "hunter-gatherer" variant ([rs7903146-T](#)) is linked to a worse response to carbs [R, R, R].

Other variants also shape our genetic predisposition to carbohydrate metabolism. They include:

- **PPARG [rs1801282-G](#):** linked to better metabolism and longevity markers on a low-carb diet [R, R, R]
- **FTO [rs9939609-A](#):** linked to obesity, especially on a high-carb diet [R, R]
- **IRS1 [rs2943641-C](#):** linked to better carb metabolism (mixed evidence) [R, R, R]
- **CETP [rs5883-T](#) and [rs3764261-C](#):** linked to lower obesity rates and better metabolic profiles on a low-carb/high-fat diet [R, R]

Additional variants that may have a smaller or indirect impact on carb metabolism include:

- **ADIPOQ -11391 G>A** (rs17300539): Associated with alterations in adiponectin levels, which can influence glucose and carbohydrate metabolism.
- **ADRB2 Gln27Glu C>G** (rs1042714): Linked to differences in β 2-adrenergic receptor function, which may affect glucose uptake and metabolism.
- **DRD2 C>T** (rs1800497): Linked to dopamine receptor function, which may influence eating behaviors (sugar and carb cravings) and glucose regulation.
- **TAS1R2 Ile191Val G>A** (rs35874116): Impacts the sweet taste receptor, potentially influencing sugar intake and carbohydrate metabolism.
- **SLC2A2 Thr110Ile C>T** (rs5400): Affects glucose transporter 2 (GLUT2), which is key in glucose sensing and carbohydrate metabolism (linked to higher sugar intake but better metabolism!)



TYPICAL

Predisposed to typical carbohydrate metabolism based on 16 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TCF7L2	rs7903146	TC
AMY2B	rs4244372	TA
IRS1	rs2943641	TC
TAS1R2	rs35874116	TT
SLC2A2	rs5400	GG
FTO	rs1121980	GG
ADRB2	rs1042714	CG
ANKK1	rs1800497	GA
LEPR	rs1137101	AG
FTO	rs9939609	TT
PPARG	rs1801282	CC
CETP	rs5883	CC
NLRC5	rs3764261	AA
FABP2	rs1799883	CC
ADRB3	rs4994	AA
PPARG	rs3856806	CC
RFC4	rs17300539	GG

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

Saturated Fat

Some people may tolerate more saturated fat than others. This difference may be genetic. If they eat a lot of saturated fats, people who are sensitive to saturated fat may have a higher risk of [\[R, R, R\]](#):

- Elevated cholesterol
- Weight gain
- Reduced bone strength



TYPICAL RESPONSE

Predisposed to typical saturated fat response based on 42 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TLR4	rs5030728	GG
PPARA	rs135549	TT
APOA1	rs670	CC
ABCA1	rs2230806	CC
TCF7L2	rs7903146	TC
SIDT2	rs5070	GG
FTO	rs1121980	GG
SIDT2	rs2854117	CC
APOE	rs429358	TT
ADAM10	rs2070895	GG
STAT6	rs1799986	CT
CETP	rs5882	GA
APOB	rs693	AG
FTO	rs1558902	TT
FTO	rs1421085	TT
FTO	rs17817449	TT
STAT3	rs2293152	CG
LPL	rs13702	CT
AHSG	rs4917	CT
CD36	rs1984112	GA
CLOCK	rs1801260	AG
CLOCK	rs4580704	CC
PKDREJ	rs4253778	CG
PEX11A	rs894160	CT
FCER1G	rs5082	AA
PPARG	rs1801282	CC
PCSK7	rs662799	AA
FTO	rs9939609	TT
AGT	rs699	GG

GENE	SNP	GENOTYPE
ACE	rs4343	AA
APOC1	rs405509	GG
ADAM10	rs1800588	CC
PPARA	rs1800206	CC
MED24	rs1568400	TT
PPARG	rs10865710	GG
SIDT2	rs964184	CC
STAT3	rs8069645	AA
STAT3	rs744166	AA
APOE	rs7412	CC
PPARG	rs3856806	CC
LPL	rs328	CC
MC4R	rs12970134	GG
LPL	rs1121923	GG
STAT3	rs1053005	TT

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

Vitamin B12

Key Takeaways:

- Vitamin B12 is important for making energy and red blood cells, building DNA, and nerve function.
- It is most easily obtained via animal products like meat, eggs, dairy, and fortified foods.
- If you have an increased need or you tested as deficient, you may want to examine your current diet. You should talk to your doctor before taking B12 supplements.
- Click the **next steps** tab for relevant labs.

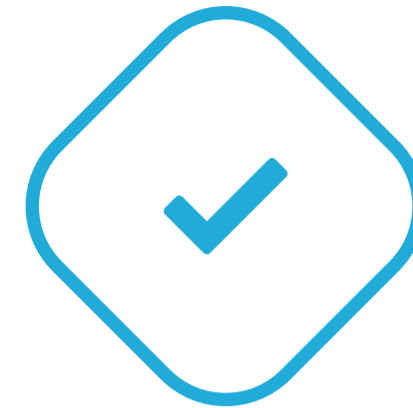
People more prone to low levels of vitamin B12 include [\[R\]](#):

- Vegetarians and vegans
- Older adults
- People with gut disorders (e.g., Crohn's disease, celiac disease)

A hallmark of vitamin B12 deficiency is a lack of healthy red blood cells (anemia). Anemia can cause symptoms like weakness and fatigue. A sign of long-term vitamin B12 deficiency is nerve damage [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

Vitamin B12 deficiency can be detected with a blood test. After it is diagnosed, you may need to work with your doctor to figure out the cause. Your doctor may recommend oral supplements or injections of vitamin B12 to help correct the deficiency [\[R\]](#), [\[R\]](#).

If you are not deficient, it is best to get vitamin B12 from food. Talk to your doctor before taking vitamin B12 supplements [\[R\]](#).



TYPICAL NEED

Likely typical need for vitamin B12 based on 1,018,082 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TCN2	rs1801198	GG
FUT2	rs1047781	AA
FUT2	rs602662	GA
CUBN	rs11254363	GG
ADGRE1	rs62123070	CC
FUT2	rs601338	GA
MTRR	rs1801394	GA
ABCD4	rs4148077	CC
TCN1	rs526934	AG
FUT5	rs3760775	GT
FUT3	rs708686	CT
FUT2	rs516246	CT
CBS	rs234706	GA
MMAB	rs7134594	CT
CUBN	rs1801222	GG
TCN1	rs34324219	CC
RGS7	rs7544372	TT
/	rs1990193	AA
/	rs1513859	AA
FAM240C	rs12478296	CC
SLC25A2	rs3749779	AA
FOXK1	rs314590	AA
CFAP299	rs1385890	AA
LAMA4	rs76190642	GG
CHODL	rs34988353	AA
ARAP2	rs142554771	TT
LAMA4	rs144505878	GG
C1QL3	rs79770840	GG
RGS18	rs114973754	CC

GENE	SNP	GENOTYPE
ADGRL3	rs545255284	TT
C16ORF82	rs139645308	CC
POU3F3	rs188141458	GG
KCNK2	rs72761546	TT
KCNK2	rs189754522	AA
PCSK2	rs141477158	GG
TMEM179	rs79885401	CC
LRRC6	rs117429467	AA
STT3B	rs188968123	AA
SPATA18	rs142766122	CC
SRRM4	rs73215576	CC
MICA	rs556990455	GG
CADM2	rs188586547	AA
CENPF	rs72759663	GG
SMYD3	rs148487271	TT
HSPB7	rs144839376	AA
AKAIN1	rs7239302	CC
ST8SIA6	rs188363440	AA
DACT1	rs118119041	GG
MMUT	rs9473555	GG
TCN2	rs9606756	AA

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

Biotin

A crucial gene affecting biotin levels is [BTB](#). It helps make an enzyme called *biotinidase*. This enzyme helps release biotin from dietary protein and ‘recycle’ it from proteins in our body [\[R\]](#).

One *BTB* variant, [rs13078881-C](#), may reduce enzyme activity by up to 50%. People with this variant might need more dietary biotin to compensate [\[R\]](#).

Important: this variant may be detrimental only when combined with other rare variants, such as [rs71627145-G](#) and [rs34885143-A](#). People carrying single copies of these variants may have higher biotin needs, but they are **not likely to have biotinidase deficiency** [\[R\]](#).

Please also note that other factors, especially your diet, can greatly affect your biotin needs.



TYPICAL NEED

Likely typical need for biotin based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
BTB	rs34885143	GG
BTB	rs13078881	GG
ANKRD28	rs71627145	AA

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

Salt Sensitivity

People who are salt sensitive will experience a bump in blood pressure when they eat salty foods. This happens because their kidneys function a bit differently [R, R, R].

Salt sensitivity is partly determined by the genes we carry. Genes involved in salt sensitivity may influence [R, R, R, R, R, R]:

- Sodium levels in the blood and kidney
- Blood vessel function
- Blood pressure

However, other genes and environmental factors may also influence your salt needs. It is important to get the right amount of salt for you.



TYPICAL

Likely typical sensitivity to salt based on 68 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
BCAT1	rs7961152	AA
SGK1	rs9389154	GG
POC1B	rs2681472	AG
NR2F2	rs2398162	GA
ACE2	rs184874220	T
PRKG1	rs7905063	TC
PRKG1	rs7897633	AC
SLC4A5	rs7571842	GA
SCNN1A	rs4764586	AA
SCNN1G	rs4299163	GG
GC	rs4254735	TT
CLGN	rs2567241	CC
RAD52	rs2301880	TC
FGF5	rs16998073	TA
WNK1	rs12828016	TG
CSTF2T	rs12414562	GA
HYAL1	rs10510755	TC
ADRB2	rs1042714	CG
SCNN1G	rs7404408	TC
SCNN1G	rs5735	CT
SCNN1G	rs4073930	CT
SCNN1G	rs4073291	CA
RENBP	rs78377269	G
ACE2	rs714205	C
AGT	rs699	GG
ACE	rs4343	AA
SLC24A3	rs3790261	AA
ACE2	rs2285666	C
GSKIP	rs11847625	GG

GENE	SNP	GENOTYPE
SGK1	rs9376026	CC
RAD52	rs880054	CT
CPA3	rs75367686	AA
SLC8A1	rs434082	CC
SLC8A1	rs11893826	GG
CSTF2T	rs10997916	GG
SLC4A5	rs10177833	AA
SLC4A4	rs10022637	TT
KL	rs9536314	TT
SCNN1G	rs4499238	CC
SCNN1A	rs3741914	CC
TNFRSF1A	rs11614164	AA

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

Potassium

Key Takeaways:

- Up to **60%** of differences in people's potassium levels may be due to genetics.
- Other risk factors for potassium deficiency include poor diet, dehydration, magnesium deficiency, and certain medications and health conditions.
- The rate of potassium deficiency in the U.S. has risen from **4% to 11%** in the past two decades.
- If you are at high genetic risk, you may lower overall risk by taking action on factors that you can change.
- Click the **next steps** tab for relevant labs.

Up to **60%** of differences in people's potassium levels may be due to genetics. Involved genes may play a role in [\[R, R, R\]](#):

- Potassium transport
- Electrolyte balance
- Kidney function

Keep in mind that your diet and the environment may also influence your potassium levels. A [blood test](#) is the only reliable way to determine your potassium levels.



TYPICAL NEED

Likely typical need for potassium based on 152,421 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
RGS7	rs183294212	CC
LURAP1	rs111512785	AA
PRG4	rs141261421	GG
NVL	rs78473436	AA
HMCN1	rs138057810	AA
CASQ2	rs117999962	GG
FMO2	rs184768578	AA
SSBP3	rs182561930	CC
PTPRC	rs141793725	TT
ETV3	rs75349367	GG
OLFM3	rs140864890	AA
GALNT2	rs80258856	CC
BEND5	rs139642127	GG
FYB2	rs143507390	GG
SSBP3	rs117932658	TT
RRAGC	rs144130357	CC
DMBX1	rs78451089	GG
/	rs80302144	CC
/	rs7548119	GG
RGS13	rs12071444	GG
HMCN1	rs61829629	GG
PLPP3	rs139123937	GG
PLPP3	rs12075340	AA
ATP1A1	rs10924092	GG
KCNT2	rs77824746	GG
SEC16B	rs75022918	TT
LY9	rs73017956	GG
KCNT2	rs76559586	GG
KCNT2	rs59729886	CC

GENE	SNP	GENOTYPE
PRKAA2	rs138876064	GG
PLPP3	rs146657061	GG
PRKAA2	rs184786990	GG
RNF186	rs7516526	GG
CACHD1	rs185466715	AA
GNAI3	rs76531483	GG
C1ORF174	rs74643023	GG
MTARC1	rs3849284	GG
LRRC52	rs17471444	TT
C1ORF21	rs139777178	AA
LYPLAL1	rs79278760	GG

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

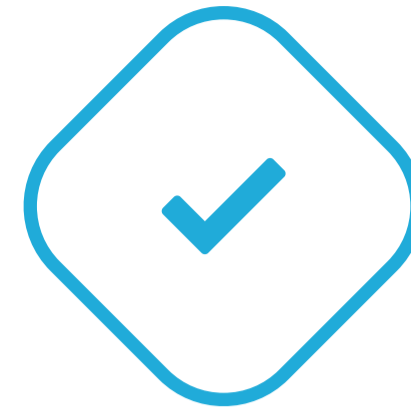
Magnesium

Some people may have higher magnesium levels than others. This may be partly due to genetics. Genes involved may influence:

- Magnesium transport in and out of cells [\[R, R, R, R\]](#)
- Magnesium metabolism [\[R, R\]](#)

Genetically higher magnesium levels may be causally associated with:

- Stroke [\[R\]](#)
- Bone health [\[R\]](#)
- Gout [\[R, R\]](#)
- Uric acid [\[R\]](#)
- Cataracts. [\[R\]](#)
- Mood Swings [\[R\]](#)
- Joint Inflammation [\[R\]](#)
- Atrial fibrillation [\[R\]](#)
- Heart Health [\[R\]](#)



TYPICAL NEED

Likely typical need for magnesium based on 31 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TRPM6	rs11144134	TT
MTMR7	rs3764796	TT
CSTA	rs1801725	GG
FGFR2	rs1219515	GG
RTL1	rs915364	CC
PAPSS2	rs1969821	GG
VIPR1	rs11718502	TC
THBS3	rs4072037	CT
PAPSS2	rs791888	GG
RALGDS	rs7032317	CT
CDKL2	rs6838240	CT
ALPK1	rs2074379	GA
C8ORF48	rs10888073	TC
CANT1	rs11891	GA
THBS3	rs4971100	GA
BORCS7	rs3740393	GC
CDKL2	rs6852678	TC
TRPM6	rs113607577	GG
HDHD2	rs117060920	GG
MPPED2	rs3925584	CC
SHROOM3	rs13146355	GG
SHROOM3	rs9993810	GA
MECOM	rs448378	AG
TRPM6	rs2274924	TT
ASAP1	rs72728275	AA
CAMK1D	rs2648708	CC
FGFR2	rs3135758	CC
CCDC136	rs1472147	TT
METTL21C	rs603894	CC

GENE	SNP	GENOTYPE
PHACTR2	rs2073214	CC
DLK1	rs4905994	CC
OR5BS1P	rs193153567	CC
PRMT7	rs7197653	GG
ATP2B1	rs7965584	AA

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

Lactose Intolerance

Lactose intolerance means a person cannot digest lactose, a sugar found in dairy. To be able to digest lactose, you need an enzyme called *lactase*. People with lactose intolerance may experience symptoms such as diarrhea, stomach cramps, nausea, bloating, and gas after eating dairy [R, R].

In people who are lactose intolerant, the gene that makes the enzyme lactase—*LCT*—gets "turned off" in adulthood. Without this enzyme, people may have trouble digesting dairy as adults [R, R].

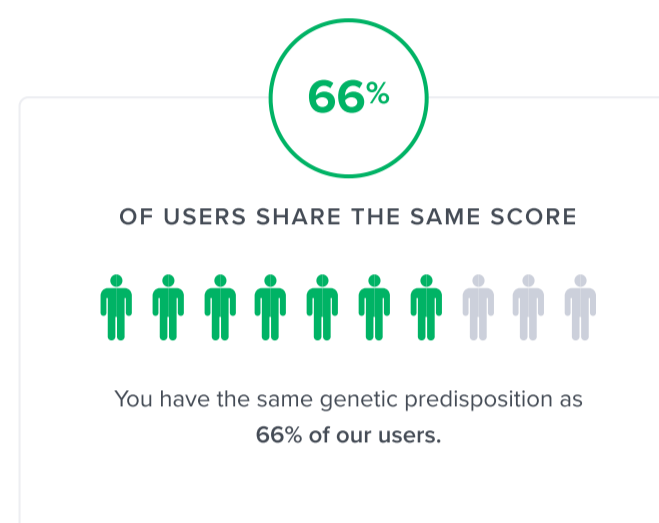
A common variant near the *LCT* gene (rs4988235 'A') is responsible for keeping the lactase enzyme "turned on." This variant is responsible for lactose tolerance in most people who are able to digest milk as adults. The 'T' allele of rs182549 has the same effect. Because these variants are usually inherited together, you will most likely have both variants or neither of them [R, R].

It's important to note that there are also other less common variants linked to lactose tolerance that we are not including in this report [R, R]. In addition, the way people respond to dairy may also depend on factors like diet, gut bacteria, and certain health conditions.



LIKELY TOLERANT

Likely lactose tolerant based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
LCT	rs4988235	GA

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

Binge Eating

Binge eating can be associated with:

- Psychological issues: Low self-esteem, body dissatisfaction, and significant stress can trigger binge eating.
- Biological factors: Genetic mutations may affect hunger and satiety, through alterations in brain chemistry that can predispose individuals to eating disorders.
- Social and cultural factors: Dysfunctional family dynamics, professions and activities that focus on appearance and dieting, and traumatic situations such as bullying or abuse can increase the risk of BED.
- Dieting: Some people with binge eating disorder have a history of dieting or restrictive eating, which may trigger an urge to binge eat.

Treatment of binge eating disorder may involve:

- Psychotherapy: Cognitive-behavioral therapy (CBT) is considered effective for treating BED. It involves teaching individuals to regulate their eating patterns and to replace unhealthy thoughts and behaviors with healthier ones.
- Medications: Antidepressants, antiepileptic drugs, and certain stimulants such as lisdexamfetamine are sometimes prescribed to help control symptoms of binge eating.
- Nutritional counseling: Working with a nutritionist can help create healthier eating habits and mend one's relationship with food.
- Support groups: These can provide a network of support and an environment to share experiences and coping strategies.

Moreover, the following lifestyle interventions may help:

- Regular eating schedules: Eating regular meals and avoiding skipping meals can prevent extreme hunger that might trigger a binge.
- Mindfulness practices: Techniques such as mindful eating can help improve awareness of hunger cues and feelings of fullness.
- Stress management: Learning and implementing stress reduction techniques can help manage the emotional component often associated with binge eating.



LESS LIKELY

Less likely to binge eat based on 13,449 genetic variants we looked at

7th

PERCENTILE



Your risk is greater than 7% of the population and lower than 93% of the population.

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NUP35	rs1950038	CT
CUBN	rs7904579	GC
BCKDHB	rs17810023	CC
MMADHC	rs182107583	AA
RGPD4	rs111940429	CC
SYNDIG1	rs76087671	CC
SLC25A26	rs145763646	GG
FTO	rs9939609	TT
/	rs73057489	AA
MC4R	rs17782313	TT
ARHGAP8	rs726170	CC
/	rs7337127	CC

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

Fat Metabolism

Your genes may affect your response to different levels of fat in a diet. Some people do better on a high-fat diet, and others on a low-fat diet, in terms of weight control and cholesterol levels [R, R].

Some of the genes responsible may also influence [R, R, R]:

- Fat metabolism
- Sugar metabolism
- Inflammation

Talk to your doctor before making big changes to your diet. Keto and other high-fat diets may increase the risk of some nutrient deficiencies. They may also affect the body's response to medication [R].



BETTER

Predisposed to better fat metabolism based on 53 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ADRB3	rs4994	AA
TCF7L2	rs7903146	TC
PPARA	rs135549	TT
APOA1	rs670	CC
ABCA1	rs2230806	CC
MC4R	rs2229616	CC
GIPR	rs2287019	CC
ACSL5	rs2419621	CC
STAT3	rs2293152	CG
STAT6	rs1799986	CT
CETP	rs5882	GA
MTTP	rs1800591	GT
AHSG	rs4917	CT
CD36	rs1984112	GA
CLOCK	rs1801260	AG
PEX11A	rs894160	CT
APOB	rs693	AG
IRS1	rs2943641	TC
CLOCK	rs3749474	TC
LPL	rs328	CC
LPL	rs1121923	GG
TLR4	rs5030728	GG
CLOCK	rs4580704	CC
APOA4	rs5110	CC
SIDT2	rs5070	GG
FABP1	rs2241883	TT
UCP3	rs1800849	GG
TCF7L2	rs12255372	TG
LPL	rs13702	CT

GENE	SNP	GENOTYPE
FCER1G	rs5082	AA
PPARG	rs1801282	CC
FTO	rs9939609	TT
PCSK7	rs662799	AA
FABP2	rs1799883	CC
RFC4	rs17300539	GG
APOC1	rs405509	GG
SIDT2	rs964184	CC
STAT3	rs8069645	AA
STAT3	rs744166	AA
APOE	rs7412	CC
CETP	rs708272	AA
AGT	rs699	GG
APOE	rs429358	TT
PPARG	rs3856806	CC
NSMAF	rs3808607	TT
MICB	rs361525	GG
ADAM10	rs2070895	GG
TNF	rs1800629	GG
ADAM10	rs1800588	CC
PPARA	rs1800206	CC

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



Phase I Detox

This section explores the genetic factors that influence your body's initial detoxification processes. Phase 1 detoxification involves enzymes that begin breaking down toxins, medications, and other foreign substances in your body, primarily in the liver.

Your genetic variations in key detoxification enzymes affect how efficiently your body processes everything from environmental toxins to common medications like acetaminophen. Understanding these genetic differences provides insights into your detoxification capacity and potential vulnerabilities to certain substances.


This analysis examines your unique genetic profile for Phase 1 detoxification pathways, helping you understand how your body handles toxin processing and what steps you can take to support optimal detoxification function based on your individual genetic makeup.

Topics include:

- CYP2D6
- CYP2C9
- CYP2B6
- Mold Sensitivity
- Acetaminophen Toxicity

 **TYPICAL METABOLIZER**
CYP2C9 (Detox)

Likely a typical metabolizer

 **TYPICAL**
Sensitivity to Foodborne Mold

Likely typical sensitivity to foodborne mold

 **TYPICAL ACTIVITY**
CYP2D6 (Mental Health, Detox)

Predisposed to a typical CYP2D6 activity

 **TYPICAL ACTIVITY**
CYP2B6 (Drug Metabolism)

Predisposed to a typical CYP2B6 activity

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].

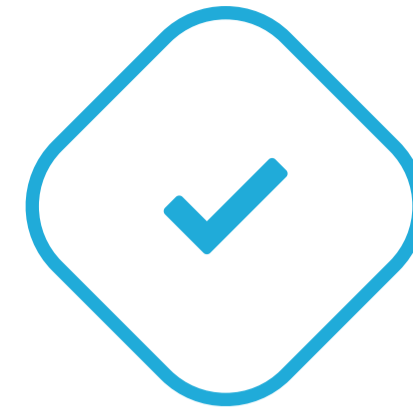
The two most common CYP2C9 gene polymorphisms decreasing enzyme activity are known as CYP2C9*2 and CYP2C9*3. Individuals with one copy of any of these alleles or two copies of the CYP2C9*2 allele are considered intermediate metabolizers, while those with one copy of each one or two copies of the CYP2C9*3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

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.

CYP2D6 (Mental Health, Detox)



TYPICAL ACTIVITY

PLEASE NOTE: This test did not evaluate for CYP2D6 copy number variations, such as gene deletions or duplications. CNVs are a primary factor influencing CYP2D6 enzyme activity and are essential for a complete assessment of metabolizer status. The results presented should be interpreted with this significant limitation in mind [R].

The *CYP2D6* gene has more than 100 known variants. These include [R, R]:

- Normal enzyme function variants (e.g., *1 and *2) [R, R]
- Reduced function variants (e.g., *9, *10, *17, and *41) [R, R]
- Non-functional variants (e.g., *3, *4, *5, *6, and *7) [R, R]

*CYP2D6*1* is the wild-type variant. The change from an 'A' to a 'G' at [rs16947](#) encodes *CYP2D6*2*. Although its activity is indistinguishable from that of *CYP2D6*1*, having several copies of this variant can result in an ultrafast phenotype. This variant has been associated with tyramine intolerance, suggesting a slightly lower metabolic efficiency for this substrate [R, R].

The 'T' allele of [rs3892097](#) corresponds to *CYP2D6*4*. This is the most frequent non-functional variant in Europeans and North Americans (18.0%), accounting for 70-90% of cases [R].

Another non-functional variant is *CYP2D6*6*, consisting of the deletion of an 'A' at [rs5030655](#). This variant is generally rare, but may be slightly more common in Europeans (around 1%) [R, R, R].

Finally, the 'G' allele of [rs5030867](#) encodes the non-functional *CYP2D6*7* variant. This allele is very rare but may be slightly more common in South East Asians (around 1%) [R].

The 'A' allele of [rs1065852](#) encodes the intermediate-activity *CYP2D6*10* variant. This variant is especially common in Thai (50%) and East Asians (42%) [R, R].

Another intermediate-activity variant is *CYP2D6*17*, encoded by the 'A' allele of [rs28371706](#). This variant is most common in Africans (20%-35%) [R, R].

Studies suggest that people with low CYP2D6 enzyme activity may be more prone to anxiety and less successful at socializing than extensive metabolizers. Reduced activity has also been associated with impulsivity or novelty seeking [R, R, R].

Alternatively, poor metabolizers may perform better in cognitive tasks that demand sustained attention or vigilance and spatial working memory. They may also have higher conscientiousness, responsibility, orderliness, and perseverance [R, R, R].

Low-activity variants have been associated with an increased risk of developing the following conditions:

- Alzheimer's disease [R]
- Parkinson's disease [R]
- Systemic sclerosis [R, R]
- Lupus [R]
- Ankylosing spondylitis [R]
- Pesticide toxicity [R]
- Adverse effects from antidepressants [R]

In contrast, they have been linked to a reduced risk of:

- Schizophrenia [R]
- Bulimia [R]

Predisposed to a typical CYP2D6 activity based on 4 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CYP2D6	rs5030867	TT
CYP2D6	rs5030655	CC
CYP2D6	rs3892097	CC
CYP2D6	rs28371706	GG
CYP2D6	rs1065852	GG
CYP2D6	rs16947	AG

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

- Heavy smoking [\[R\]](#)
- Adverse effects from codeine and tramadol [\[R\]](#)

CYP2B6 (Drug Metabolism)

There are over 100 known *CYP2B6* polymorphisms, with numerous complex haplotypes and distinct ethnic frequencies, making *CYP2B6* one of the most polymorphic cytochrome p450 genes in humans [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), and has been associated with slower metabolism of multiple drugs such as methadone, efavirenz, bupropion, cyclophosphamide, sertraline, ketamine, and nicotine [R, R, R, R, R, R, R].

Another low-function variant is the rare 'C' allele of [rs28399499](#), which encodes the *CYP2B6*18* variant. This variant has been associated with slower efavirenz and nevirapine metabolism, resulting in higher blood levels of these drugs [R, R].

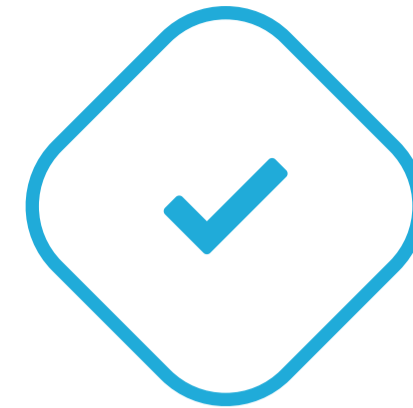
*CYP2B6*1* is the wild-type variant. The change from a 'C' to a 'T' at [rs8192709](#) encodes the *CYP2B6*2* variant. Although considered a normal-function variant, carriers may have slightly slower efavirenz and prasugrel metabolism [R, R, R].

Another polymorphism considered a normal-function variant is the 'T' allele of [rs3211371](#) (*CYP2B6*5*). Research on its effects on methadone metabolism has produced mixed results. Moreover, this variant has been associated with an increased risk of gastrointestinal cancer and relapse after autologous hematopoietic cell transplantation for lymphoma [R, R, R, R, R].

Finally, the 'G' allele of [rs2279343](#) encodes the *CYP2B6*4* variant. This polymorphism has been associated with slower metabolism of efavirenz. However, it has also been linked to faster metabolism of bupropion (and thus lower success rate of smoking cessation therapy with bupropion) and methadone [R, R, R, R].

The *CYP2B6* gene affects how you metabolize ketamine via the liver. Between 10 and 20% of people have a variant that causes them to clear the drug from their system half as fast as others.

These "slow metabolizers" should be extra cautious about taking ketamine, since they're more likely to have a long or intense trip, according to some resources. They also have a higher risk for adverse reactions to the drug — such as drowsiness, unpleasant hallucinations, and confusion — especially if they inject it rather than taking it orally or nasally.



TYPICAL ACTIVITY

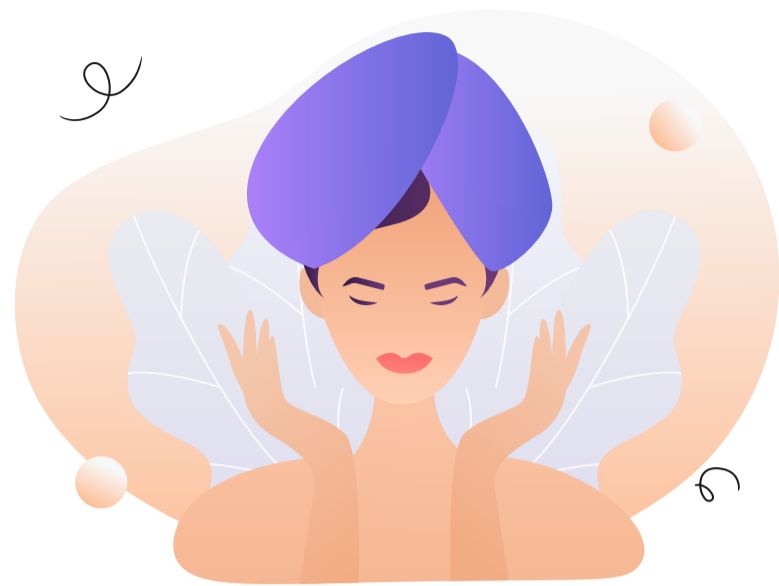
Predisposed to a typical CYP2B6 activity based on 4 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CYP2B6	rs3745274	GT
/	rs2279343	AG
CYP2B6	rs28399499	TT
CYP2B6	rs8192709	CC
CYP2B6	rs3211371	CC

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



Skin Health


This section examines the genetic factors that influence your skin's aging process, elasticity, and susceptibility to various skin conditions. Understanding these genetic variations provides insights into how your skin responds to environmental factors, sun exposure, and the natural aging process.

Your genetic profile affects fundamental aspects of skin health, from collagen production and glycation processes to melanin formation and cellular repair mechanisms. This analysis explores your predisposition to skin aging, elasticity changes, and specific skin conditions based on your unique genetic makeup.

This comprehensive skin genetics assessment empowers you to make informed decisions about skincare routines, sun protection strategies, and lifestyle choices that support optimal skin health throughout your lifetime, tailored to your individual genetic predispositions.

Topics include:


- Skin Aging
- Skin Conditions

 **MORE LIKELY**
Melanoma

More likely to get melanoma

 **TYPICAL**
Glycation


Predisposed to typical glycation

 **TYPICAL LIKELIHOOD**
Basal Cell Carcinoma

Typical likelihood of basal cell carcinoma

 **TYPICAL ACTIVITY**
MC1R (Pigmentation & Skin Damage)

Likely typical MC1R activity

 **TYPICAL**
Facial Wrinkles

Predisposed to a typical amount of facial wrinkles

Melanoma

Factors that may increase the risk of melanoma include [R]:

- High exposure to UV radiation: Exposure to UV radiation from the sun or tanning beds is the primary risk factor for melanoma.
- Fair skin: melanoma is most common in people with fair skin, hair, and eyes.
- History of sunburns: Severe, blistering sunburns, particularly in childhood, increase the risk.
- Multiple atypical moles: Having a large number of moles or atypical (dysplastic) moles increases the risk.
- Weakened immune system: Individuals with weakened immune systems, such as those who have had organ transplants, are at higher risk.
- Living closer to the Earth's equator or at high elevation
- Family history

Treatment options vary depending on the stage and may include [R]:

- Surgery: The primary treatment for early-stage melanoma, which involves removing the tumor along with a margin of healthy tissue.
- Lymph node dissection: If the melanoma has spread to nearby lymph nodes, these may be surgically removed.
- Immunotherapy: Drugs like pembrolizumab (Keytruda) or nivolumab (Opdivo) boost the body's immune system to fight the cancer.
- Targeted therapy: For melanomas with specific genetic mutations, drugs that target those mutations (e.g., BRAF inhibitors like vemurafenib) can be effective.
- Radiation therapy: May be used in cases where surgery is not possible or if the melanoma has spread.
- Chemotherapy: Less commonly used for melanoma, but may be considered in certain advanced cases.

The prognosis for melanoma depends on the stage at diagnosis. Early-stage melanomas that are detected and treated before they spread have a very high cure rate. However, once melanoma has spread to other parts of the body, it becomes more challenging to treat. Advances in immunotherapy and targeted therapy have improved outcomes for many patients with advanced melanoma.

Please note: This report is not diagnostic and can't be used to make any medical decisions. Most cancers are uncommon and have a strong environmental component. Even if your genetic predisposition is high, you will most likely not develop the disease. This report doesn't test for hereditary cancer syndromes or 'cancer genes'. These are usually caused by rare mutations that can't be analyzed by our test. If you're concerned about your risk of hereditary cancer, consider getting a specialized test at a reference laboratory.



MORE LIKELY

More likely to get melanoma based on 1,049,396 genetic variants we looked at



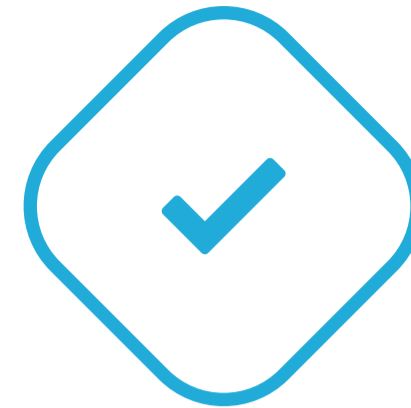
Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SLC45A2	rs35407	GG
TERT	rs139996880	AG
TYR	rs1393350	AG
FLACC1	rs13016963	GA
MX2	rs45430	TC
CCND1	rs498136	AC
AGR2	rs1636744	CT
SOX4	rs6914598	CT
LINC02218	rs187843643	CC
ASIP	rs910873	GG
SPATA2L	rs258322	GG
ASIP	rs1885120	GG
MC1R	rs1805007	CC
ASIP	rs6059655	GG
DBNDD1	rs4785763	CC
IRF4	rs62389423	GG
SLK	rs2995264	AA
IRX3	rs16953002	GG
CTSS	rs7412746	CC
/	rs10739221	CC
CYP1B1	rs6750047	GG

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

Glycation

A genetic study identified five variants (SNPs) associated with skin autofluorescence in non-diabetic individuals. These SNPs are located in or near genes such as **FANCA**, **MMP27**, **CYP1A1/2**, **ZNF276**, and **NAT2**, each with distinct potential roles in glycation mechanisms [R].



TYPICAL

Predisposed to typical glycation based on 5 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

- rs12931267 (FANCA, Chr16):** This SNP lies in an intronic region of the *FANCA* gene, which is involved in DNA repair. The G allele is associated with higher SAF. This locus also includes the *MC1R* gene, known for its role in skin pigmentation. This suggests a potential interplay between pigmentation and glycation levels.
- rs2846707 (MMP27, Chr11):** Located in the *MMP27* gene, this SNP encodes a missense variant (Met30Val) affecting matrix metalloproteinase activity. The T allele correlates with lower SAF, suggesting a protective role potentially linked to extracellular matrix remodeling.
- rs2470893 (CYP1A1/2, Chr15):** This SNP lies between *CYP1A1* and *CYP1A2*, genes involved in xenobiotic metabolism. The T allele is linked to higher SAF, with attenuation of the SNP effect when adjusted for **coffee consumption**, indicating environmental interactions.
- rs3764257 (ZNF276, Chr16):** Found upstream of *FANCA*, this SNP is associated with lower SAF. *ZNF276* encodes a zinc finger protein that may influence gene regulation in response to glycation stress.
- rs576201050 (NAT2, Chr8):** Located near *NAT2*, a gene encoding N-acetyltransferase, this SNP affects acetylator status (detox ability). The minor allele A is associated with lower SAF, highlighting its role in metabolic processing of AGEs.

GENE	SNP	GENOTYPE
NAT2	rs576201050	GG
ZNF276	rs3764257	CC
MMP27	rs2846707	TC
CSK	rs2470893	TC
FANCA	rs12931267	CC

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

Overall, the authors estimated about **20%** of the differences in glycation may be due to genetics. These findings demonstrate that SAF levels are influenced by complex genetic and environmental interactions [R].

Basal Cell Carcinoma

Unlike other forms of skin cancer, basal cell carcinoma rarely spreads (metastasizes) beyond the original tumor site. However, it can be disfiguring if not treated promptly and can cause considerable destruction and disfigurement by invading surrounding tissues.

It's important to monitor changes in the skin and seek medical advice if any suspicious growths or new lesions appear, especially in areas that receive a lot of sun exposure. Treatment options vary depending on the severity and can include surgical excision, topical medications, cryotherapy, laser therapy, or radiation, with the aim to remove or destroy the cancerous cells.



TYPICAL LIKELIHOOD

Typical likelihood of basal cell carcinoma based on 33,751 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SLC45A2	rs16891982	GG
RHOU	rs61824911	GA
LPP	rs2049218	TT
PADI6	rs12122129	AG
EDN2	rs2781249	CC
TYR	rs1126809	AG
FOXP1	rs35768603	CT
BNC2	rs10810657	TA
CTLA4	rs1427676	CT
RNASET2	rs4710154	TA
KANK1	rs1323262	CG
IRF4	rs62389423	GG
FANCA	rs12931267	CC
ASIP	rs56238684	GG
KRT5	rs11170164	CC
ASIP	rs17401449	AA
HLA-F	rs29243	GG
NDRG3	rs55804368	CC
EXO1	rs4149909	AA
CTSS	rs41271951	AA
NEK9	rs7145468	AA
OCA2	rs1800407	CC
NCR3	rs61447909	GG
HLA-DQB1	rs9268847	AA
/	rs2572140	GG
SOX4	rs55775505	CC
TICAM1	rs10425559	AA
CTSH	rs2289702	CC
CCDC88B	rs663743	GG

GENE	SNP	GENOTYPE
GRHL1	rs6741117	GG

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

MC1R (Pigmentation & Skin Damage)

A variant of *MC1R* has been associated with hair and skin color. Carriers of the minor 'T' allele of [rs1805007](#) are more likely to [\[R\]](#):

- Have red or blonde hair
- Have lighter skin
- Get sunburns

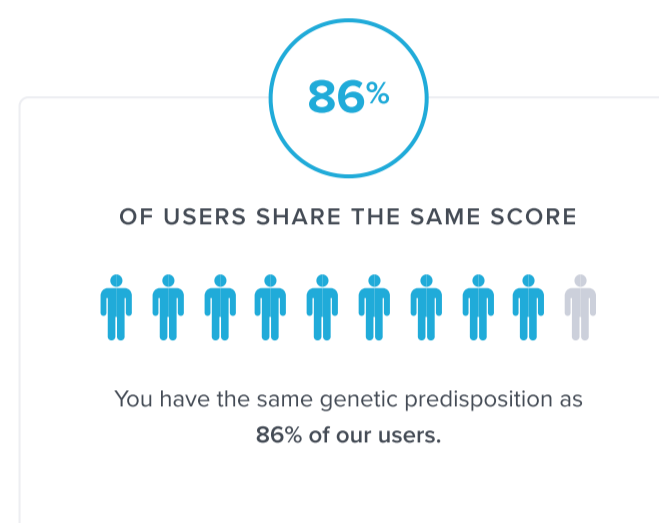
Unsurprisingly, this variant has also been associated with skin cancer, including melanoma [\[R, R\]](#).

This variant likely reduces the production and activity of MC1R, leading to lower eumelanin and higher pheomelanin levels [\[R\]](#).



TYPICAL ACTIVITY

Likely typical MC1R activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MC1R	rs1805007	CC

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

Facial Wrinkles

Wrinkles are small but visible folds in the skin. Wrinkles appear with age and develop over several decades. They are more evident in areas of the body that are usually exposed, such as the face, neck, forearms, and hands. **The most noticeable wrinkles are facial** [R, R, R].

Up to **55%** of differences in people’s facial wrinkling may be due to genetics. Involved genes may influence **skin color and health** [R, R, R].

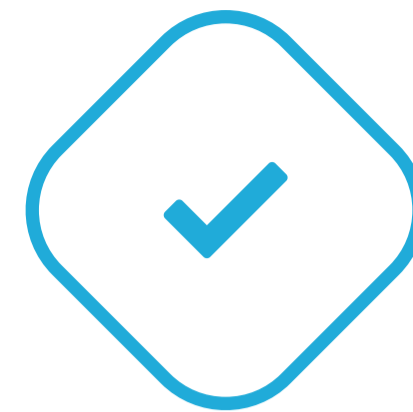
UV radiation also contributes to facial wrinkling. Excessive sunbathing or use of tanning beds exposes the skin to unhealthy levels of UV radiation and leads to premature aging. Hence, experts recommend wearing sunscreen [R, R, R].

Make sure to find the right balance. [Sunlight](#) or bright light during the day can benefit your body (by increasing vitamin D levels) and mind (by boosting mood) [R, R].

The color of your skin also influences facial wrinkling. Wrinkles appear more rapidly in people sensitive to UV radiation. Hence, white skin may wrinkle earlier than other skin types [R, R].

Others factors that may also contribute to facial wrinkling include [R, R, R]:

- Aging
- Smoking
- Very low weight
- Health conditions (e.g., depression)



TYPICAL

Predisposed to a typical amount of facial wrinkles based on 20 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SIK2	rs11213999	CC
TPGS2	rs78569750	GG
LRRC3B	rs116248825	CC
NUDT12	rs113322056	AA
HACD4	rs116873518	GG
SYNDIG1	rs184605088	CC
NUDT12	rs112608607	TT
LINGO2	rs117828793	CC
BBX	rs1283106	AC
BMP6	rs1225927	GT
/	rs11711327	AG
BMP6	rs382029	AT
MON1B	rs62047859	TT
DCSTAMP	rs147672305	TT
GLIS1	rs702491	CC
RESF1	rs1150997	AA
CA3	rs184880542	GG
/	rs72811030	GG

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



Fitness









This section explores the genetic factors that influence your athletic performance, recovery capacity, and injury susceptibility. Understanding these genetic variations provides insights into your body's natural strengths, optimal training approaches, and areas where you may need additional support or caution.

Your genetic profile affects key aspects of fitness including muscle fiber composition, cardiovascular endurance, recovery speed, and connective tissue integrity. This analysis examines how your genes influence your response to different types of exercise, your risk for specific injuries, and your body's ability to adapt to training stress.

This comprehensive fitness genetics assessment helps you optimize your training program, recovery strategies, and injury prevention approaches based on your unique genetic predispositions, enabling you to achieve better results while minimizing risk.

Topics include:

- Strength & Muscle
- Fatigue & Recovery
- Injuries

<p> LOWER Muscle Mass</p> <p>Predisposed to lower muscle mass</p>	<p> TYPICAL Muscle Recovery</p> <p>Predisposed to typical muscle recovery</p>	<p> TYPICAL Strength</p> <p>Predisposed to typical strength</p>
<p> TYPICAL ACTIVITY COL5A1 (Collagen)</p> <p>Likely typical COL5A1 activity</p>	<p> TYPICAL LIKELIHOOD Achilles Tendon Injury</p> <p>Typical likelihood of Achilles tendon injury</p>	<p> HIGHER Endurance</p> <p>Predisposed to higher endurance</p>
<p> HIGHER ACTIVITY COL1A1 (Collagen)</p> <p>Predisposed to higher COL1A1 activity</p>	<p> LESS LIKELY Tendon Injury</p> <p>Less likely to have tendinopathy</p>	

Muscle Mass

Muscle mass is the total amount of all the muscles in your body [R, R].

Your **physical activity and diet** have a great impact on your muscle mass. But some of it is also due to your genetics. Twin studies suggest **about 50-80% of people's differences in muscle mass are due to genes** [R, R, R].

Genes associated with muscle mass affect processes such as [R, R]:

- Muscle growth
- Growth and sex hormone levels
- Energy metabolism

Other factors that affect muscle mass include aging and certain health conditions [R, R, R].

To maintain and build your muscle mass:

- **Do regular resistance training**, such as weightlifting or bodyweight exercises (e.g. push-ups or squats). Aim for 2 or more sessions of strength training per week.
- **Get enough protein**. Make sure you get enough protein in your diet to support muscle growth and repair.
- **Be physically active**. Staying active throughout the day helps maintain muscle mass, beyond resistance training. Walking, cycling and other activities contribute to overall muscle health.
- **Get sufficient rest**. Muscles grow while you are resting. Getting enough sleep and recovery time between workouts supports muscle growth and repair.



LOWER

Predisposed to lower muscle mass based on 7,086,143 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
IL1B	rs1143634	GG
RPN1	rs111804884	TT
GJB2	rs73442383	TT
STEAP1B	rs10242595	GA
MTCH2	rs4752856	GA
RASSF10	rs543716802	GG
LDB2	rs573762110	GG
NDUFA12	rs529529785	GG
PPARGC1A	rs752744147	TT
AOAH	rs78185366	TT
LEO1	rs190878891	AA
USP34	rs183237934	TT
MARCHF1	rs150541056	TT
CCDC171	rs117635839	AA
ZMAT4	rs184622626	AA
AQP6	rs706798	AA
FTO	rs9939609	TT
ZNF648	rs190210789	AA
/	rs557373392	CC
/	rs774160312	GG
/	rs758954781	CC
PRELID2	rs574519712	CC
/	rs758566456	TT
DOCK3	rs551736560	GG
ATG7	rs193172711	GG
TSC22D1	rs554766983	GG
MYL12B	rs141372016	GG
DRD5	rs182591441	TT
ACTR3B	rs138921315	GG

GENE	SNP	GENOTYPE
LMX1B	rs144076261	CC
DLG2	rs558729974	TT
KCNH7	rs74698413	GG
FTO	rs9972653	GG
EPHA3	rs72913870	GG
METTL21A	rs111556242	CC
ZDHHC14	rs749547	GG
SVBP	rs111455041	CC
PRUNE2	rs11145045	CC

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

Muscle Recovery

Muscle recovery is when the **body repairs the muscle fibers damaged or strained during exercise**. It is during this time that muscles grow and become stronger.

The exact **genetic influences** on muscle recovery are still being researched. Several genes play a role in this process (e.g. ACTN3, CKM, IL6), through their effects on muscle growth, muscle repair and inflammation [R, R].

Other factors that affect muscle recovery, include [R, R]:

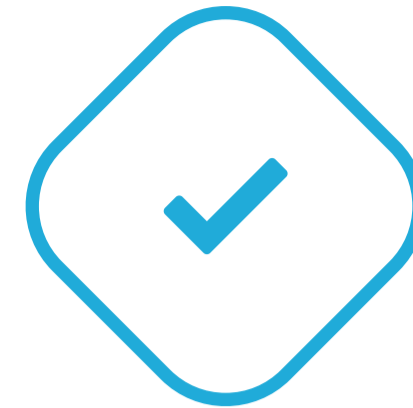
- Intensity and type of exercise
- Nutrition and hydration
- Sleep quality
- Stress
- Aging

Popular methods to aid muscle recovery include [R, R, R, R]:

- Light stretching
- Massage
- Applying heat or cold to the muscle
- Compression

Adequate nutrition and hydration are important for muscle health. Our bodies need enough protein to repair and build new muscle, and enough carbs to restore depleted energy stores [R, R, R].

Interestingly, drinking cherry juice several days before exercise may help improve muscle recovery [R].



TYPICAL

Predisposed to typical muscle recovery based on 15 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ACTN3	rs1815739	CT
CCR2	rs1799865	TT
IGF2	rs2230949	GG
SLC30A8	rs13266634	CC
IGF2	rs680	TC
IGF2	rs3213221	CG
COL2A1	rs2070739	TC
COL5A1	rs12722	TC
TRIM63	rs2275950	TC
SOD2	rs4880	GG
CCR2	rs3918358	AA
CCL2	rs3917878	CC
TNF	rs1800629	GG
IGF2	rs7924316	GG
IGF2	rs4244808	GG

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

Strength

Strength is the maximum force applied in one movement (e.g., in a single weight lift) [R].

Some people are stronger than others. Up to **85% of people's differences in muscle strength may be due to genetics.** Genes associated with strength affect [R, R, R, R, R, R]:

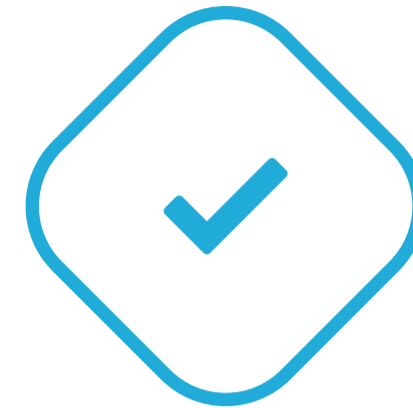
- Muscle composition (fast-twitch vs. slow-twitch muscle fibers)
- Muscle repair and regeneration
- Hormones and growth factors
- Energy metabolism

However, other factors, such as **fitness, diet and age**, also strongly affect your muscle power [R, R].

Strength training is important not just for your muscles, but also for your bones, cognition, and your overall health. Muscle strength is a predictor of healthy aging and longevity [R, R].

Some tips that may help you build stronger muscles include [R]:

- Resistance training. You can train with weights or do bodyweight exercises (e.g., pushups, planks). Aim for 2 or more sessions of strength training per week.
- Using a weight or resistance level that is heavy enough to tire your muscles after 12-15 repetitions. As your strength bulbs over time, you should be able to slowly increase the weight you are lifting.
- Eating a protein-rich diet to support muscle growth and repair
- Allowing plenty of rest for each muscle group to maximize strength gains.



TYPICAL

Predisposed to typical strength based on 7,133,253 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MTHFR	rs1801131	TT
SLC16A1	rs1049434	TT
LRPPRC	rs10186876	GG
PPARG	rs1801282	CC
LEMD2	rs12055409	GG
IL11RA	rs41274853	GG
BDNF	rs10501089	CC
HIF1A	rs11549465	CC
FTO	rs9939609	TT
VEGFA	rs2010963	GG
GABPB1	rs7181866	AA
/	rs9320823	TC
PITX3	rs2273555	AG
IGF2	rs680	TC
ACTN3	rs1815739	CT
ADRB2	rs1042713	AG
ADRB2	rs1042714	CG
BDKRB2	rs1799722	TC
AGT	rs699	GG
TBC1D7	rs6905419	CC
ZNF608	rs4626333	CC
ACVR1B	rs2854464	AA
AMPD1	rs17602729	GG
TRHR	rs16892496	AA

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

COL5A1 (Collagen)

Variants resulting in reduced type V collagen may increase the risk of injuries. This may be because carriers of these variants may have weaker ligaments, tendons, and muscles. One of such variants is [rs12722](#). Its minor 'T' allele has been associated with an increased risk of:

- Achilles tendon injury [\[R, R\]](#)
- Anterior cruciate ligament injury [\[R, R\]](#)
- Tennis elbow [\[R, R\]](#)
- Carpal tunnel syndrome [\[R\]](#)
- Muscle cramps [\[R\]](#)

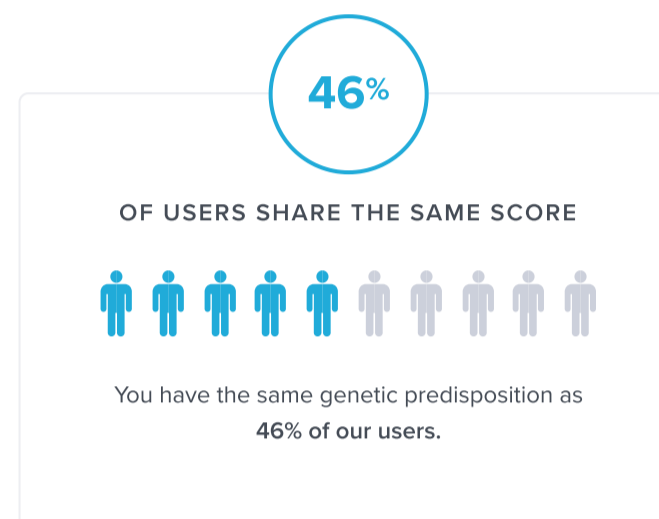
People with the 'TT' genotype may also have more severe (but not more frequent) muscle injuries. Probably due to the association of this variant with soft tissue injuries, carriers may have lower odds of elite rugby status [\[R, R\]](#).

On the bright side, this genotype has also been associated with improved endurance performance in runners. Moreover, the 'T' variant may reduce the risk of rotator cuff injury [\[R, R\]](#).



TYPICAL ACTIVITY

Likely typical COL5A1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
COL5A1	rs12722	TC

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

Achilles Tendon Injury

The **Achilles tendon** is a cord made of strong tissue. It is found in the lower back leg and connects the calf muscles to the heel bone. The Achilles tendon helps you move around. Some common injuries to this tendon include **tendonitis** (inflammation of the tendon, usually due to overuse or damage) and **rupture** (partial or complete tear of the tendon) [R, R, R].

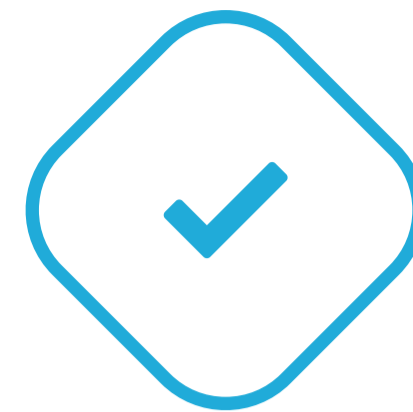
Some people may be more prone to Achilles tendon injuries than others. Risk factors include [R, R, R, R, R]:

- A “weekend warrior” exercise pattern (i.e. taking part in physical activity intermittently)
- Age (the risk tends to increase between age 30 and 40)
- Male sex
- Taking part in activities that include a lot of running, jumping, or sudden changes in movement (e.g., soccer, basketball, and tennis)
- Wearing improper shoes
- Lack of a warmup or cooldown
- Some medications
- Obesity
- **Genetics**

In fact, about 50% of differences in people’s chances of developing an Achilles tendon injury may be attributed to genetics [R].

Tips to help prevent an Achilles tendon injury include [R, R]:

- Stretching and strengthening your calf muscles
- Alternating high-impact sports with low-impact ones (e.g., alternating running with walking)
- Avoiding running on hard, uneven, or slippery surfaces
- Using proper equipment (e.g., well-fitting, cushioned running shoes)
- Slowly increasing the intensity of the activity



TYPICAL LIKELIHOOD

Typical likelihood of Achilles tendon injury based on 23 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MMP1	rs650108	AA
COL1A1	rs1800012	AC
MMP1	rs591058	CC
CPNE1	rs143383	AA
VEGFA	rs699947	AC
SOAT1	rs113435565	CA
FCN2	rs1134170	TT
COL5A1	rs12722	TC
MPP7	rs6481512	TT
MPP7	rs1249269	TT
MRPL43	rs4919510	CG
TNF	rs1800629	GG
CEP295NL	rs4789932	GG
TMEM158	rs183364169	CC
DPP6	rs4067493	CC
/	rs11960097	TT
TRIML1	rs60713544	GG
ZNF648	rs57104447	TT
MPP7	rs1937810	TT
SOX21	rs4454832	GG

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

Endurance

Endurance is the ability to produce low-intensity movements for a long period of time (e.g., cycling, running, swimming). It is made up of **cardiorespiratory endurance** (the ability of the heart and lungs to deliver enough oxygen to muscles during prolonged activity) and **muscular endurance** (the ability of muscles to contract over an extended period of time without fatigue) [R, R, R].

Some people have greater endurance than others. **Up to 70% of people's differences in endurance may be due to genetics.** Genes associated with endurance affect [R, R, R, R, R, R, R, R]:

- Muscle composition and efficiency (slow-twitch vs. fast-twitch muscle fibers)
- The body's ability to use oxygen
- Heart function

Some tips that may help you build endurance include:

- Getting at least 150 min of cardio per week
- Doing the same exercise regularly and letting your body adapt to it over time
- Slowly increasing the amount and intensity of your workouts over time
- Switching between periods of high-intensity training and resting. This is called high-intensity interval training (HIIT).

Great news is that **endurance can actually improve as we age!** For example, peak endurance age for ultra-marathoners often occurs in the late 30s and 40s, potentially even the 50s [R, R, R, R].



HIGHER

Predisposed to higher endurance based on 36 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HFE	rs1799945	CC
GABPB1	rs7181866	AA
GABPB1	rs8031031	CC
ACTN3	rs1815739	CT
SLC16A1	rs1049434	TT
TTN	rs10497520	CC
KDR	rs1870377	TT
PRDM1	rs10499043	CC
ADRB3	rs4994	AA
CHRNA3	rs4950	AA
GSTP1	rs1695	AA
HIF1A	rs11549465	CC
TSHR	rs7144481	TT
NFE2L2	rs35652124	TT
DES	rs7564856	GA
SATB1	rs4973706	TC
PPARGC1A	rs8192678	CT
GALNTL6	rs558129	GA
ADRB2	rs1042713	AG
ADRB2	rs1042714	CG
COL5A1	rs12722	TC
GNB3	rs5443	CT
BDKRB2	rs1799722	TC
RBFOX1	rs7191721	GA
CNDP2	rs6566810	TA
PKDREJ	rs4253778	CG
AGTR2	rs11091046	C
CKM	rs8111989	TC
DEF6	rs2016520	CT

GENE	SNP	GENOTYPE
PPARGC1A	rs7665116	CT
PPARGC1A	rs2970869	CT
NR1H3	rs7120118	TT
ACE	rs4343	AA
GABPB1	rs12594956	AA
MYBPC3	rs1052373	CC
CYFIP1	rs8029108	AA
NFIA	rs1572312	GG
TTC23	rs1464430	AA
KCNA4	rs1323860	GG
PPARA	rs1800206	CC
PPARGC1A	rs3774923	CC
HIF1A	rs2301113	AA

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

COL1A1 (Collagen)

The most well-researched *COL1A1* polymorphism is [rs1800012](#). Its minor 'A' allele promotes the expression of an alternative version of the protein with increased pro- α 1 chain levels, potentially increasing its mechanical strength and stability [\[R\]](#).

This variant has been associated with a decreased risk of:

- Sports-related tendon and ligament injuries [\[R\]](#)
- Musculoskeletal soft tissue injuries [\[R\]](#)
- Cervical insufficiency [\[R\]](#)
- Pelvic organ prolapse [\[R\]](#)

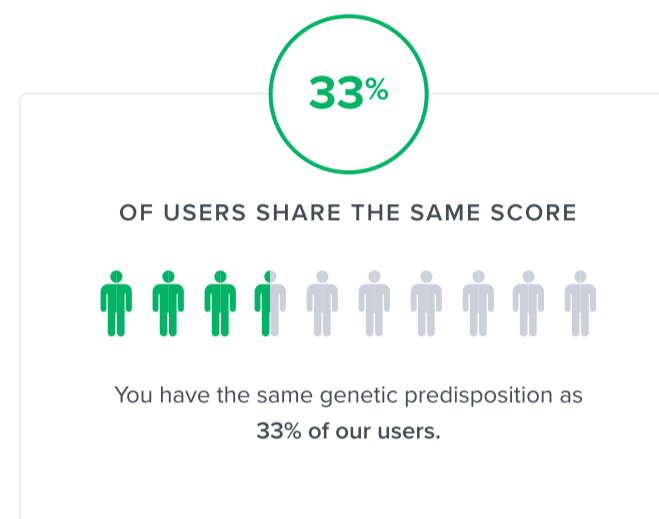
In contrast, it may increase the risk of osteoporosis and bone fractures [\[R\]](#), [\[R\]](#).

The 'A' allele may also affect athletic performance and has been found more commonly in competitive runners [\[R\]](#).



HIGHER ACTIVITY

Predisposed to higher COL1A1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
COL1A1	rs1800012	AC

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

Tendon Injury

Key Takeaways:

- Up to **40%** of the differences in people's chances of developing tendinopathy may be due to genetics.
- Other risk factors include intense physical activity or certain sports, using improper training technique or equipment, doing repetitive tasks, age (over 40), being female, some chronic health conditions and medications.
- About 16 million tendon and ligament injuries occur in the U.S. each year.
- If you have a high genetic risk, you may lower your overall risk by taking action on those factors that you can change.

Tendons are cords made of strong tissue. They attach muscle to bone. There are many tendons throughout the body. Conditions in which tendons become inflamed and painful are called **tendinopathies** [R, R].

Risk factors for tendinopathies include [R, R, R, R]:

- Taking part in intense physical activity or certain sports (e.g., baseball, golf)
- Using improper training technique or equipment
- Doing repetitive tasks (e.g., gardening, painting, typing)
- Age (over 40)
- Gender (women may be at a higher risk)
- Some chronic health conditions (e.g., obesity, diabetes)
- Some medications
- **Genetics**

Up to **40%** of the differences in people's chances of developing tendinopathy may be due to genetics. Involved genes may influence tendon function [R, R, R, R, R].

Interestingly, one study that examined the impact of genetics on tendon injuries was conducted on elite athletes from FC Barcelona [R].

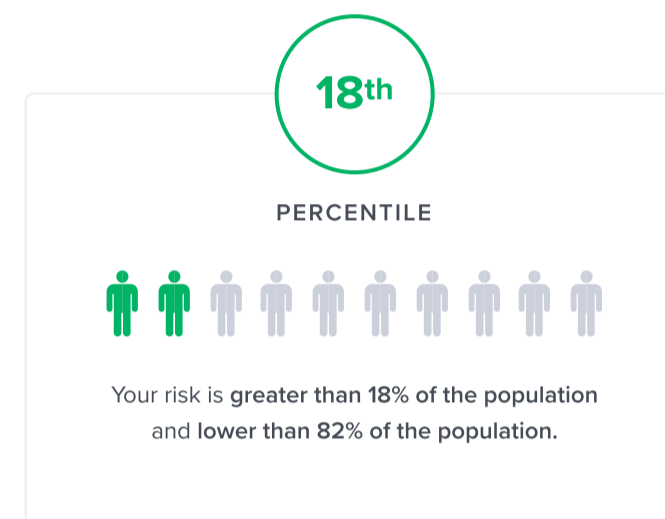
Tips to help prevent a tendon injury include [R, R]:

- Stretching and strengthening your muscles
- Alternating high-impact sports with low-impact ones (e.g., alternating running with walking)
- Avoiding running on hard, uneven, or slippery surfaces
- Using proper equipment (e.g., protective gloves, well-fitting shoes)
- Slowly increasing the intensity of the activity



LESS LIKELY

Less likely to have tendinopathy based on 6 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
POLE2	rs3218791	TG
/	rs11232681	AG
GJA1	rs11154027	CC
KLHL1	rs59988404	GG
TRIML1	rs60713544	GG
CERS5	rs57224706	AA

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



Brain Health


This section examines the genetic factors that influence cognitive function, mental health, and neurological well-being. Understanding these genetic variations provides insights into your brain's unique characteristics, including cognitive strengths, potential vulnerabilities, and responses to various substances and conditions.

Your genetic profile affects critical aspects of brain health from neurotransmitter function and cognitive processing to susceptibility to neurological conditions and mental health disorders. This analysis explores how your genes influence attention, memory, cognitive decline risk, and your brain's response to different compounds and stressors.


This comprehensive brain health genetics assessment empowers you to make informed decisions about cognitive enhancement strategies, mental health support, and lifestyle choices that promote optimal brain function throughout your lifetime, tailored to your individual genetic predispositions.

Topics include:


- Mental Effects of THC
- ADHD
- Cognition
- Cognitive Impairment and Decline
- Anxiety

WORSE
 **GAD1**
(Glutamate/GABA)


Likely worse GAD1 genetics

MORE LIKELY
 **THC and Psychosis**


More likely to experience psychotic symptoms from cannabis use

INCREASED
 **Psychedelic Effects of THC (Functional)**


Predisposed to increased psychedelic effects of THC

TYPICAL LIKELIHOOD
 **Anxiety**


Typical likelihood of anxiety

TYPICAL
 **Cognitive Function**


Predisposed to typical cognition

TYPICAL
 **Attention**

Typical likelihood of ADHD

TYPICAL
 **Memory Performance**

Predisposed to typical memory performance

TYPICAL LIKELIHOOD
 **Alzheimer's Disease**

Typical likelihood of Alzheimer's disease

E3/E3
 **APOE**

You carry two APOE ε3 variants



LESS LIKELY

Cognitive Decline

Less likely to have cognitive decline

GAD1 (Glutamate/GABA)

The most well-researched *GAD1* polymorphism is [rs3749034](#). Its major 'G' allele has been shown to reduce *GAD1* activity, resulting in lower GABA levels in the brain. In addition, this variant has been associated with an increased risk of [\[R\]](#):

- Schizophrenia [\[R, R, R\]](#)
- Hyperactive and impulsive symptoms in ADHD [\[R\]](#)

However, this variant has also been linked to lower odds of bipolar disorder and fewer respiratory symptoms in people with panic disorder [\[R, R\]](#).

Another variant associated with schizophrenia is the minor 'A' allele at [rs1978340](#). Surprisingly, this variant has been shown to reduce GABA levels. The fact that GABA acts as an inhibitory neurotransmitter in the mature brain but has the opposite activity during brain development may explain the complex effects of GABA levels on conditions such as schizophrenia and ADHD [\[R, R, R, R\]](#).

This variant has also been linked to:

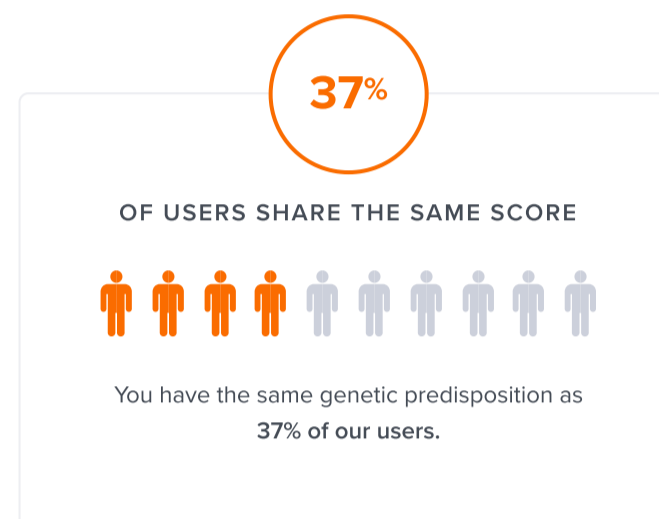
- Cocaine dependence [\[R\]](#)
- Panic disorder and respiratory symptoms in this condition [\[R, R\]](#)
- Earlier onset of alcohol dependence [\[R\]](#)

However, this allele was associated with a reduced risk of heroin addiction [\[R\]](#).



WORSE

Likely worse GAD1 genetics based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GAD1	rs3749034	GG
GAD1	rs1978340	AG

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

THC And Psychosis

Certain genetic variants may affect the risk and severity of THC-related psychosis. Carriers of several risk variants may want to consider avoiding cannabis use.

The [CNR1](#) gene encodes the type-1 cannabinoid receptor (CB1), whose activation improves mood and anxiety. The most studied SNP in this gene is [rs1049353](#) or 1359G/A. Its minor 'T' allele doesn't change the CB1 receptor structure but is believed to impair gene expression and receptor activity. This variant has been linked to worse psychotic symptoms in cannabis smokers and greater size reduction in a region of the brain (the caudate nucleus) after the first episode [\[R, R\]](#).

Another [CNR1](#) variant, 'T' at [rs12720071](#), has been associated with psychotic symptoms in cannabis users. However, the 'C' variant has been linked to greater white matter reduction in the brain from marijuana misuse [\[R, R\]](#).

The [FAAH](#) gene helps create an enzyme that breaks down certain compounds in the body, including endocannabinoids. The 'G' allele of [rs2295633](#), associated with increased FAAH activity, has been linked to reduced odds of requiring treatment with antipsychotics in cannabis users [\[R, R\]](#).

The [COMT](#) gene encodes an enzyme that helps break down neurotransmitters such as [dopamine](#), [norepinephrine](#), and epinephrine. The major 'G' variant of its main polymorphism [rs4680](#) has been associated with increased risk, earlier onset, and worse symptoms of psychosis and schizophrenia from cannabis use [\[R, R, R, R, R, R, R\]](#).

The [DRD2](#) gene helps make [dopamine](#) D2 receptors. Those are proteins on the surface of brain cells that bind dopamine. The minor 'A' allele of [rs1076560](#) has been associated with increased DRD2 signaling and a 3- to 5-fold higher risk of psychosis in cannabis users [\[R, R\]](#).

Finally, the [AKT1](#) gene encodes an enzyme involved in many cellular processes, whose dysregulation may promote schizophrenia. The 'C' allele of [rs2494732](#) has been associated with a 7-fold higher risk of psychosis in daily cannabis smokers [\[R, R\]](#).



MORE LIKELY

More likely to experience psychotic symptoms from cannabis use based on 6 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CNR1	rs1049353	TT
FAAH	rs2295633	AA
CNR1	rs12720071	TT
COMT	rs4680	GA
TTC12	rs1076560	CA
AKT1	rs2494732	TT

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

Psychedelic Effects Of THC (Functional)

The genetic predisposition to THC's psychedelic effects is shaped by variations in specific genes involved in the endocannabinoid system and neurotransmitter regulation.

Among these, the **CNR1** gene (**rs1049353**, **rs12720071**) plays a critical role. This gene encodes cannabinoid receptor CB1. Variants in this gene influence receptor functionality and density, which modulate how THC interacts with the endocannabinoid system to produce psychedelic effects [R, R].

Additionally, the **AKT1** gene (particularly the SNP **rs2494732**) is linked to an increased risk of experiencing psychosis-like effects from THC. AKT1 is involved in dopamine signaling pathways, which THC impacts, potentially enhancing susceptibility to altered perceptions [R].

Another key gene is **DRD2**, associated with the dopamine D2 receptor. The SNP **rs1076560** is linked to altered dopamine receptor activity, potentially influencing THC-induced euphoria and cognitive effects. Variants in this gene may also affect the likelihood of experiencing addiction or dependency on cannabis [R].

The **FAAH** gene, represented by SNP **rs324420**, is responsible for the breakdown of anandamide, a natural cannabinoid. Reduced FAAH activity leads to increased levels of anandamide, which may enhance THC's effects [R, R].

Finally, **COMT** is responsible for the breakdown of neurotransmitters such as dopamine, epinephrine, and norepinephrine in the brain. Its **rs4680** SNP may increase the risk of psychosis in cannabis users [R, R, R, R, R, R, R].

Understanding these genetic markers can provide valuable insights into personalized cannabis use, highlighting who might benefit from THC's therapeutic potential and who might be at risk for adverse effects.



INCREASED

Predisposed to increased psychedelic effects of THC based on 7 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CNR1	rs1049353	TT
FAAH	rs2295633	AA
COMT	rs4680	GA
TTC12	rs1076560	CA
CNR1	rs12720071	TT
FAAH	rs324420	AC
AKT1	rs2494732	TT

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

Anxiety

Key Takeaways:

- Up to **65%** of the differences in people's risk of getting anxiety may be due to genetics.
- Other risk factors include traumatic and stressful events, thyroid problems, heart problems, and substance use problems.
- If your genetic risk is high, managing stress and substance use may help reduce overall risk.
- Anxiety can cause issues with sleep, fatigue, the gut, stress, focus, and mood.
- Click the **Recommendations** tab for potential dietary and lifestyle changes and **next steps** for relevant labs.

It's completely normal to feel anxious about things from time to time.

Occasional anxiety can help us solve problems and make better life decisions. However, people with *anxiety disorders* often worry about normal activities, which impacts their daily life [R, R].

Two parts of your brain process threats [R, R, R]:

- The *amygdala* helps activate the "fight or flight" response
- Frontal areas of your brain override the amygdala and help you respond logically

People experience anxiety when they have too much activity in their amygdala or too little in frontal brain areas [R, R].

If you're anxious, you may experience [R]:

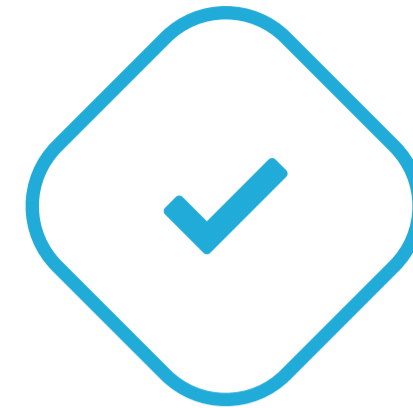
- Restlessness
- Fatigue
- Problems concentrating
- Short temper
- Muscle tension
- Heavy sweating
- Trembling
- Gut problems
- Heart rate changes
- Sleep problems

People are more likely to have these symptoms if they experience [R]:

- Traumatic or stressful events
- Thyroid problems
- Heart problems
- Substance use problems

Another important risk factor for anxiety is genetics. About 30-65% of the differences in people's chances of getting anxiety can be attributed to genetics. Genes linked to anxiety may influence the levels and activity of different brain chemicals, such as [R, R, R, R, R, R, R]:

- [Serotonin](#) and [dopamine](#), which make you feel happy ([SLC6A4](#), [HTR1A](#), [TPH2](#), [MAOA](#))
- [GABA](#), which calms the mind ([GABRG2](#))
- Stress hormones such as [cortisol](#) ([MC4R](#), [MAOA](#))
- Substances that promote new brain cell growth ([BDNF](#), [NGF](#))



TYPICAL LIKELIHOOD

Typical likelihood of anxiety based on 806,651 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ATP8B4	rs2413998	AA
/	rs16838980	GG
DNAH8	rs4714177	AA
NUP107	rs11177321	GG
FKBP4	rs2302729	CC
PREPL	rs1067327	CC
RNF180	rs6295	GG
IL20RB	rs17374749	GG
PID1	rs10498237	GG
/	rs10092548	AA
C6ORF118	rs9295300	AA
NOX4	rs17221829	CC
NOX4	rs10830352	GG
GABRG2	rs211037	TT
MARCHF4	rs955816	GG
IRX6	rs2397376	TT
HTR2A	rs12584920	GG
COMT	rs4680	GA
ERCC6L2	rs7867155	CC
COMT	rs4633	CT
SLC6A2	rs3785151	GC

GENE	SNP	GENOTYPE
CES1	rs1566652	TG
GAD1	rs3828275	CT
GAD1	rs701492	TC
GAD1	rs769407	CG
GAD1	rs3791878	TG
IL18R1	rs2058622	AG
GAD1	rs3791851	CT
ZPLD1	rs1709393	CT
DMD	rs921896	C
CAMTA1	rs11120917	TC
OR5P3	rs7112002	AC
SRBD1	rs2344662	CA
ADRB1	rs1034258	AG
SSH2	rs6354	TT
ESR1	rs9340799	AG
ESR1	rs2234693	TC
AKAP6	rs17406568	GG
OSCP1	rs906228	CA
AGPAT4	rs3798943	CC
CCNY	rs2086153	CT
COX7B2	rs6447514	TT
DDT	rs755622	GG
TULP1	rs3800373	AA
RGS2	rs10801153	GG
RNF220	rs12138940	GA
MC4R	rs10871777	AA
TBL1X	rs5934574	T
TACR1	rs3771841	GA
DSCAM	rs1040315	AG

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

Cognitive Function

Some people are really good at math, but bad at memorizing facts. Others excel at learning languages, but lack spatial awareness and get easily lost. Everyone’s brain works differently.

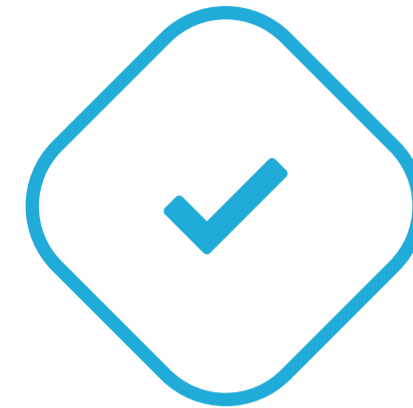
Your strengths and your weaknesses depend on your education, your life experiences and your genes. Up to **50-80%** of differences in cognitive ability may be due to **genetics**. Genes that affect our cognitive function tend to influence **brain development, structure and function** [R, R, R, R, R].

Other factors that affect our cognitive function include [R, R, R, R, R, R]:

- Lifestyle (e.g. physical activity, sleep quality)
- Education
- Physical and mental health
- Sensory loss (e.g. vision or hearing loss)
- Aging
- Social connectedness
- Environment (e.g. air pollution)
- Being cognitively active (e.g. reading, having intellectual-stimulating jobs)

To improve cognitive function focus on:

- Staying physically active [R, R, R]
- Getting enough good quality sleep [R, R]
- Staying social (in person) [R, R, R]
- Eating a healthy, balanced diet [R, R]
- Keeping the mind active (e.g., with puzzles, games of strategy or learning new skills) [R, R, R, R]



TYPICAL

Predisposed to typical cognition based on 7,159,700 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SNAP25	rs363043	CC
ARMC2	rs9384679	TT
CHRM2	rs8191992	TT
MAPRE1	rs406193	CC
CABP5	rs3936340	TT
PAM	rs35658696	AA
MTHFR	rs1801133	AA
GYPC	rs1550404	TT
PRMT6	rs12125971	CC
/	rs17813294	CC
TET2	rs2454205	TT
NEGR1	rs7531118	TT
/	rs2478286	CC
SCMH1	rs12035012	AA
UBA7	rs7613360	TT
CNR1	rs1049353	TT
UBA7	rs9855505	TT
BCL2	rs956572	GG
/	rs9388349	TT
ST8SIA6	rs7897269	TT
CHRM2	rs7799047	GG
REC114	rs7171755	GA
CLSTN2	rs6439886	AA
NR2F2	rs4984541	AA
SNAP25	rs363016	TC
CHRM2	rs2350786	AA
DPP4	rs1913808	GG
ST8SIA6	rs17141089	GG
PKN2	rs17130578	GG

GENE	SNP	GENOTYPE
DPYD	rs1702294	CC
SLC10A7	rs11737630	CC
SBNO1	rs1060105	CC
PPA2	rs2726491	AG
/	rs9320747	GT
AKR1C3	rs9423406	GA
BDNF	rs6265	CT
COMT	rs4680	GA
SNAP25	rs363050	AG
SNAP25	rs363039	GA
PLXNB2	rs28379706	TC
CHRM2	rs2350780	AG
TFAM	rs1937	GC
ARVCF	rs165599	GA
SCN2A	rs10174400	CT
NEGR1	rs12128707	AG
RBM6	rs13100903	CT
/	rs12211582	GT
NR1D2	rs6550835	AG
NEGR1	rs1486091	TC
ELAVL2	rs10733389	GA

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

Attention

Key Takeaways:

- Up to **80%** of differences in people's chances of developing ADHD may be due to genetics.
- About **6.4 million** American children aged 4-17 have ADHD, along with **4% of adults**.
- Risk factors include: smoking, drug or alcohol use during pregnancy, toxins, brain injuries, and being male.
- ADHD can lead to substance abuse and money problems in adults.
- Since the condition is rare in adults, a high genetic risk is not necessarily a reason to worry.
- Click the **Recommendations** tab for potential dietary and lifestyle changes and **next steps** for relevant labs.

We've all struggled to stay focused on an important task. However, some people have more trouble paying [attention](#) than others.

The most important part of the brain for attention and focus is the *prefrontal cortex*. This region also helps you plan and solve problems [\[R\]](#).

Other parts of the brain help to filter important information without having to think about it. This allows you to avoid distractions [\[R\]](#).

Problems in these brain regions may make it harder to stay focused. Some people have so much difficulty focusing that it interferes with their daily lives. This is a sign of *attention-deficit/hyperactivity disorder* (ADHD) [\[R\]](#).

ADHD affects millions of children and teenagers in the US. More boys are diagnosed with ADHD than girls [\[R\]](#).

Children and teens with ADHD tend to have trouble with school. They might also experience problems with relationships [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

Attention problems often continue into adulthood. Adults with ADHD are more likely to experience [\[R\]](#):

- Substance abuse
- Car accidents
- Money problems

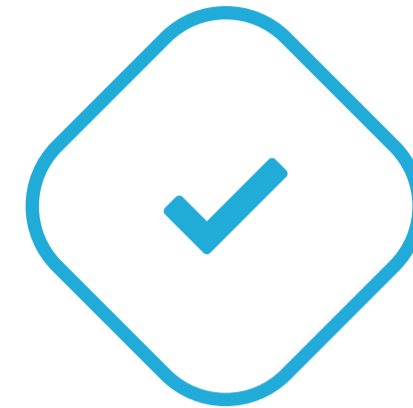
Treatment for ADHD usually includes talk therapy and medication [\[R\]](#).

The cause of ADHD is unknown. Risk factors include [\[R\]](#):

- Maternal use of cigarettes, drugs, or alcohol during pregnancy
- Environmental toxins
- Brain injuries
- Genetics

Up to 80% of differences in people's chances of developing ADHD may be attributed to genetics. Genes involved in ADHD may influence [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#):

- [Dopamine](#) ([DRD4](#), [DRD5](#), [COMT](#))
- [Serotonin](#) ([HTR1B](#), [SLC6A4](#), [SNAP25](#))
- Brain cell growth ([BAIAP2](#))



TYPICAL

Typical likelihood of ADHD based on 261,702 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
LPCAT1	rs27072	CC
DLC1	rs2410116	GG
COMT	rs4680	GA
TPH2	rs1843809	TT
MFHAS1	rs13439086	CT
AXIN2	rs8074751	AA
MUC15	rs10767556	AG
ANAPC4	rs28612433	CT
FBN3	rs35624673	CT
BDNF	rs56164415	GG
/	rs9545903	TC
CHRNA4	rs1044396	GG
CLPTM1L	rs11564750	GG
SLC26A5	rs144525	CC
PLXDC2	rs10828015	CT
BLOC1S2	rs35835615	CC
TPH2	rs1386497	AA
LGR4	rs11030104	AA
HTR2C	rs3813929	C
BDNF	rs6265	CT
ANKK1	rs1800497	GA

GENE	SNP	GENOTYPE
MAOA	rs6323	G
COMT	rs4633	CT
SLC6A2	rs3785151	GC
CES1	rs1566652	TG
DRD1	rs265981	AG
SFXN1	rs5326	CT
DRD1	rs686	GA
KCTD3	rs6540899	GA
PFKP	rs1537617	GA
SNAP25	rs362987	AC
CNTLN	rs10962864	TC
SNAP25	rs3746544	GT
EXOC1L	rs895614	AG
CNTLN	rs6475111	TC
ZNF584	rs35782676	CT
DTNBP1	rs760761	AG
ADA	rs73598374	TC
HTR1B	rs6296	CG
DTNBP1	rs2619528	TC
DTNBP1	rs2619522	CA
DTNBP1	rs1018381	AG
SNAP25	rs363039	GA
ESD	rs7984966	TT
IL10RB	rs77224013	GG
SLC6A2	rs3785143	CC
C21ORF62	rs112686226	AA
TNR	rs6686722	CC
SPATA7	rs61975260	CC
HERC2	rs4778174	GG

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

Memory Performance

Memory performance refers to how good your brain is at storing and recalling information [R, R, R].

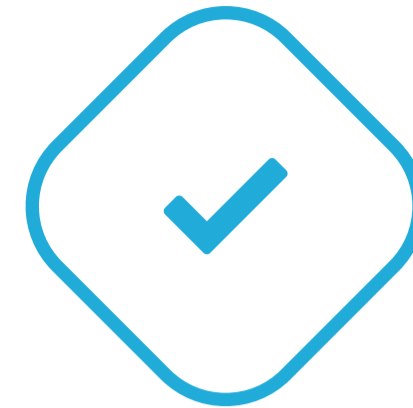
About 30-70% of differences in people’s memory performance may be due to genetics. Genes involved in memory may influence [R, R, R, R]:

- The way brain cells grow and develop
- Brain cell communication

Ways to boost your memory performance include [R, R, R, R]:

- Being physically active
- Getting 7-9 hours of good-quality sleep every night
- Eating a healthy diet
- Spending time with family and friends
- Engaging your mind (e.g, reading, solving puzzles, learning new skills)

If you want to go the extra mile, there are specific techniques you can use to train your memory (e.g. mind palace). There is even a world memory championship!



TYPICAL

Predisposed to typical memory performance based on 7,166,380 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HDGFL1	rs9466427	GG
HDGFL1	rs9348530	AA
HDGFL1	rs9358578	GG
HTATIP2	rs10741845	AT
/	rs9345419	TT
TMEM63A	rs6426075	AG
NT5DC2	rs4687625	CT
NT5DC2	rs2015971	CT
NT5DC2	rs11711421	CT
ITIH1	rs3774354	GA
ITIH1	rs1961958	AG
ITIH1	rs3774355	GA
ITIH1	rs6778844	TC
ITIH1	rs12487445	AC
ITIH1	rs6798246	GA
ITIH1	rs1961959	GC
PBRM1	rs17264436	TA
ITIH1	rs2289249	GA
GNL3	rs11177	GA
ITIH1	rs10865973	AT
ITIH1	rs2118540	TC
ITIH1	rs11717836	AG
ITIH1	rs6976	CT
ITIH1	rs2268027	GA
ITIH1	rs2239551	GA
CGGBP1	rs12492805	GT
ITIH1	rs2268025	AT
NEK4	rs1029871	GC
ITIH1	rs2286798	AC

GENE	SNP	GENOTYPE
TDRD3	rs4886229	TC
TEK	rs10757641	TC
TEK	rs633903	TG
TEK	rs581724	GT
MAT1A	rs4933327	GG
ZNF804A	rs1344706	CA
STARD3	rs879606	GG
COMT	rs4680	GA
DTNBP1	rs2619522	CA
TTC12	rs1076560	CA
SLC19A1	rs1051266	TC
SYNJ2	rs10945973	AG
LMX1A	rs4657412	AG
SYNJ2	rs2502601	AG
MAT1A	rs3851059	AG
SYNJ2	rs9356200	CT
SORL1	rs3824968	AT
COL4A2	rs4773144	AA
SYNJ2	rs10455935	GA
UBE2Z	rs15563	AA
WWC1	rs17070145	TT

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

Alzheimer's Disease

Key Takeaways:

- About **60-80%** of differences in people's chances of getting Alzheimer's disease may be due to genetics.
- Alzheimer's disease can wipe out cognitive abilities.
- **5.8 million** Americans have Alzheimer's disease, the vast majority of them being over 75 years of age.
- Other risk factors include old age, female sex, air pollution, alcohol abuse, and obesity.
- **This report doesn't take into account the APOE-e4 variant.**

Some of the risk factors for Alzheimer's include [\[R\]](#):

- Being over the age of 75
- Being female
- High exposure to air pollution
- Poor sleep patterns
- Alcohol abuse
- Sedentary lifestyle
- Low social interaction
- Low involvement in mentally stimulating activities

The following conditions may contribute to Alzheimer's disease [\[R\]](#):

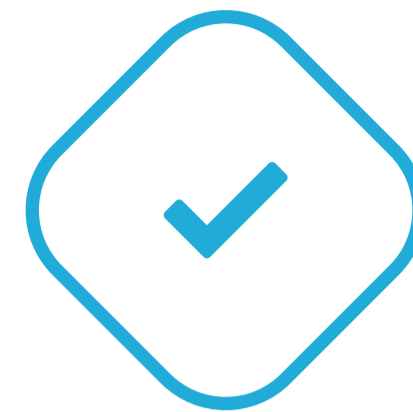
- Mild cognitive impairment
- Head trauma
- Obesity
- Diabetes
- High cholesterol
- Down syndrome

About **60-80%** of differences in people's chances of getting Alzheimer's disease may be due to genetics [\[R\]](#).

Genetically high fasting insulin, ApoB, and neutrophil levels may be causally associated with a higher risk of Alzheimer's disease [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

In contrast, genetic predisposition to high total testosterone and glucosamine supplement use may be causally associated with a lower risk [\[R\]](#), [\[R\]](#).

Please note: Genetic models analyzing a lot of variants (PRS models) usually don't take into account variants with large effects, such as **APOE-e4**. This variant is by far the strongest genetic factor for Alzheimer's disease. If you carry it, your predisposition to Alzheimer's disease is higher, regardless of your result for this report.



TYPICAL LIKELIHOOD

Typical likelihood of Alzheimer's disease based on 1,049,157 genetic variants we looked at

66th

PERCENTILE



Your risk is greater than 66% of the population and lower than 34% of the population.

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CHRM2	rs6962027	TT
PICALM	rs3851179	TT
GSK3B	rs334558	GA
CD55	rs3818361	GA
POLR2E	rs12151021	AA
HLA-DRB1	rs9271192	CA
CD55	rs679515	CT
ECHDC3	rs7912495	GG
SORT1	rs11102972	CT
CLNK	rs6846529	CT
COX7C	rs62374257	CT
CPSF3	rs72777026	AG
WDR81	rs35048651	DEL(GAG)T
IGHG3	rs7157106	GA
LILRB5	rs587709	CT
SNX1	rs3848143	AG
GC	rs2282679	GT
CLU	rs11136000	CC
APOE	rs429358	TT
TREM2	rs75932628	CC
PTGS2	rs20417	GG

GENE	SNP	GENOTYPE
RELN	rs528528	CC
SETD7	rs535347112	CC
BDNF	rs56164415	GG
SYPL2	rs17646665	AA
NGFR	rs2072446	CC
SLC20A1	rs1800587	GG
TREML1	rs60755019	AA
SORL1	rs11218343	TT
NCK2	rs143080277	TT
TREM2	rs143332484	CC
SORT1	rs141749679	TT
GPX4	rs3764650	TT
ABI3	rs616338	CC
WWC1	rs17070145	TT
ATP8B4	rs138799625	CC
PILRB	rs1476679	TT
BIN1	rs744373	AA
SORL1	rs74685827	TT
BIN1	rs6733839	CC
MME	rs61762319	AA
SHARPIN	rs34173062	GG
FOXF1	rs16941239	TT
C1QTNF4	rs10838725	TT
DBNDD1	rs56407236	GG
APH1B	rs117618017	CC
CD2AP	rs9349407	GG
STYX	rs17125924	AA
RASGEF1C	rs113706587	GG
OTULIN	rs112403360	TT

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

APOE

Key Takeaways:

- If you carry one or both **ε4** variants, your risk for Alzheimer's disease may be higher.
- The risk is greatest for late onset (after age 65) Alzheimer's disease.
- Even if your risk is higher due to the **ε4** variants, numerous other factors from your environment to lifestyle to other genetic variants impact overall risk.
- People with both variants may never get Alzheimer's, and some who have neither variant can get the disease.

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

ε2, ε3, and ε4 change the shape of the ApoE protein. This can impact how well ApoE functions [R, R].

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

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

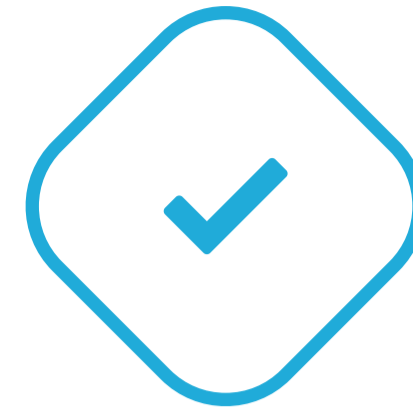
ε2 is another less common variant. It makes a protein that is better than ε3 at removing plaque from the brain, but not as good at removing fats from the blood. ε2 has been linked to a lower risk of Alzheimer's disease [R, R, R].

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

Did you know? The **ε4** variant was much more common among ancient hunter-gatherers. Scientists suggest this variant might have improved their [R]:

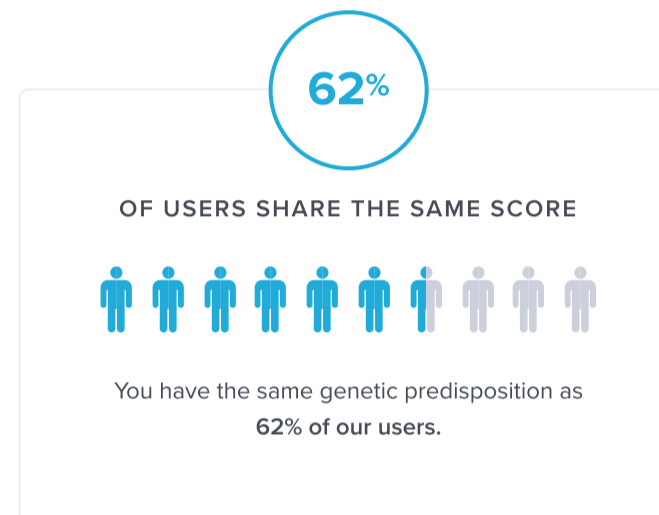
- Inflammatory response to germs in the wilderness
- Vitamin D status in less sunny European areas
- Aerobic endurance, crucial for a hunter-gatherer lifestyle

As humans largely switched to farming, some effects of this variant became useless or even harmful. For this reason, evolution strongly favored the **ε3** variant in ancient farmers and their modern descendants [R].



E3/E3

You carry two APOE ε3 variants based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
APOE	rs7412	CC
APOE	rs429358	TT

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

Cognitive Decline

Mild cognitive decline is a normal part of aging that can affect cognitive functions such as memory, attention, and problem-solving.

About **60-70%** of the differences in people’s cognitive decline may come from genetics. For example, genetically high total and bioavailable testosterone may be causally associated with larger gray matter volume in men [R, R, R].

Other risk factors for cognitive decline include [R]:

- Older age
- Female sex
- Lifestyle factors like smoking and being inactive
- Lower education level

Different health conditions may play a role in cognitive decline, including high cholesterol and blood pressure [R].



LESS LIKELY

Less likely to have cognitive decline based on 272,168 genetic variants we looked at

2nd

PERCENTILE



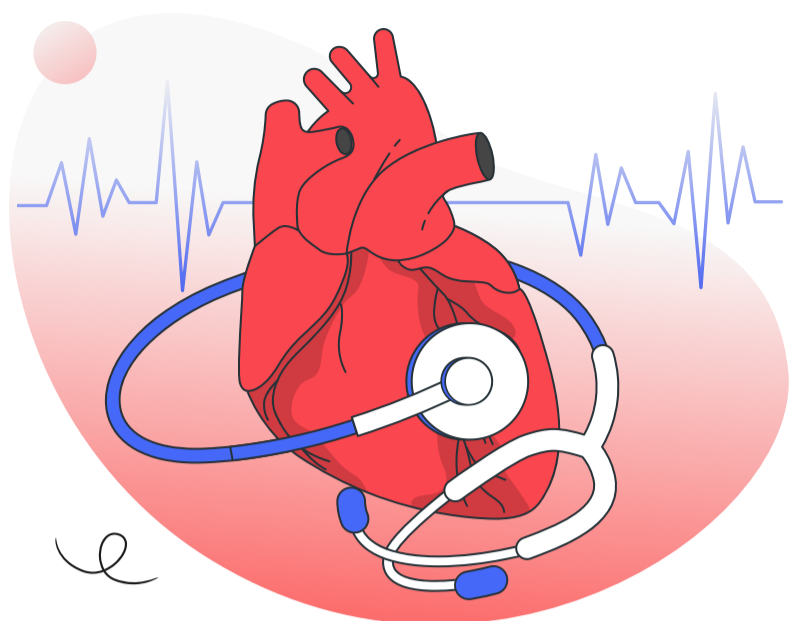
Your risk is greater than 2% of the population and lower than 98% of the population.

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CDCA7	rs182734936	CC
ANXA5	rs141005242	CC
/	rs200668351	GG
TEK	rs147486058	AA
DUSP15	rs6089150	CC
CTBP2	rs61869228	CC
HHEX	rs60320343	AA
CRP	rs1205	CC
FOXO3	rs4946936	CC
APOE	rs7412	CC
CLU	rs11136000	CC
KIF11	rs6583817	CC
MS4A6A	rs610932	GG
TRIM32	rs7852872	CC
LHFPL6	rs9315702	AA
DPP4	rs6741949	GG
/	rs11706133	TT
WDFY2	rs9535753	TT
LAMP3	rs630527	GG
FOXJ2	rs7138264	GG
OPCML	rs11606197	TT

GENE	SNP	GENOTYPE
/	rs72956174	TT
B3GALNT1	rs4455332	CC
C3ORF56	rs11716691	AA
IRX2	rs72720951	AA
ZNF799	rs4804181	AA
/	rs57169846	GG
BDNF	rs6265	CT
ALCAM	rs34476301	AG
SIRT1	rs3758391	CT
TNF	rs1799724	CT
SNRPB	rs2076650	TC
A2M	rs11609582	TA
APBB2	rs13133980	GC
BCHE	rs1803274	CT
PRR16	rs3991625	CT
CEMIP2	rs12237894	GC
SALL1	rs2075199	CT
MRPS18C	rs10004897	AG
SALL3	rs7231688	AG
CHD6	rs6072411	GA
HSD11B1	rs60686175	TC
/	rs10457441	TT
TMEM106B	rs1990622	AG
APOE	rs429358	TT
TNS1	rs13013766	GG
/	rs62477365	TT
BCL11A	rs6545794	GG
IFNL3	rs73050457	CC
ABCA2	rs908832	GG

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



Heart & Blood Vessels


This section explores the genetic factors that influence your heart health, blood vessel function, and overall cardiovascular system performance. Understanding these genetic variations provides insights into your risk profile for heart disease, arrhythmias, and other cardiovascular conditions.

Your genetic profile affects fundamental aspects of cardiovascular health including coronary artery disease susceptibility, heart rhythm regulation, and lipid metabolism. This analysis examines your predisposition to various cardiovascular conditions and how your genes influence your heart's structure and function throughout your lifetime.


This comprehensive cardiovascular genetics assessment helps you understand your unique risk factors and empowers you to make informed decisions about heart-healthy lifestyle choices, preventive measures, and monitoring strategies tailored to your individual genetic predispositions.

Topics include:

- CAD
- Atrial fibrillation
- Lp(a)
- Nitric oxide (NOS3)

 **MORE LIKELY**
Coronary Artery Disease


More likely to have coronary artery disease

 **TYPICAL ACTIVITY**
NOS3 (Cardiovascular)

Likely typical NOS3 activity

 **TYPICAL LEVELS**
Lipoprotein(a)

Predisposed to typical Lipoprotein(a) levels

 **TYPICAL LIKELIHOOD**
Atrial Fibrillation

Typical likelihood of atrial fibrillation

Coronary Artery Disease

Key Takeaways:

- Over **18 million** people have heart disease in the U.S. A third of deaths from heart disease are preventable.
- Up to **40%** of differences in people's chances of getting coronary artery disease may be due to genetics.
- Other risk factors include excess weight, stress, sedentary lifestyle, smoking, and more.
- If you have a high genetic risk, take action on modifiable risk factors. Even with a low genetic risk, having other risk factors will still make you prone to heart disease.
- Click the **next steps** tab for relevant labs and lifestyle factors.

In the US, 1 in 3 deaths from heart disease could be prevented. That's about 92,000 deaths each year. **Imagine if we could save all those lives by striving to prevent heart disease** [\[R\]](#)!

Coronary artery disease is the most common type of heart disease. It affects the coronary arteries -- the large blood vessels that feed the heart. When these vessels become narrowed or blocked, they can't deliver as much oxygen to the heart. Because of this, heart muscle tissue can start to die off [\[R\]](#), [\[R\]](#).

If a coronary artery is blocked suddenly, it can cause a heart attack. If the artery narrows slowly over a long period of time, it can cause chest pain and other problems [\[R\]](#).

Many factors can increase your risk of heart disease. These include [\[R\]](#), [\[R\]](#):

- Excess weight
- Unhealthy diet
- Stress
- Lack of exercise
- Smoking
- Air pollution
- Age
- High blood pressure
- High cholesterol
- Diabetes
- Genetics

According to the CDC, **over 18 million adults in the US have coronary artery disease**, and the rates keep increasing. However, death rates have been going down. This is likely due to improved diagnosis and treatment [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)!

Medications that doctors often prescribe for coronary artery disease include [\[R\]](#):

- Low doses of aspirin, to help prevent blood clots
- Statins, to reduce cholesterol and slow down fat buildup in blood vessels
- Beta-blockers, to lower blood pressure and relax the heart

It's much easier to prevent heart disease than to treat it. To avoid heart disease, experts recommend a "heart-healthy" lifestyle, which includes [\[R\]](#):

- Not smoking cigarettes
- Eating a healthy diet
- Staying physically fit
- Getting good-quality sleep



MORE LIKELY

More likely to have coronary artery disease based on 1,049,366 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NOS3	rs2070744	CT
PEMT	rs12936587	GA
COMT	rs4680	GA
PCSK9	rs11591147	GG
ATG16L1	rs10210302	TT
NKX2-3	rs10883365	GG
FHL3	rs190569784	GG
SERPINA1	rs112635299	GG
ANGPTL4	rs116843064	GG
APOE	rs7412	CC
IRGM	rs1000113	CT
LDLR	rs6511720	GG
IL23R	rs11805303	CT
/	rs72711827	GG
SORT1	rs12740374	GG
PHACTR1	rs9349379	GG
FBXL20	rs72823390	CC
PLPP3	rs17114046	AA
/	rs2457480	AA
ADO	rs10761659	AG
MCTP2	rs28607113	TT

Up to 40% of differences in people's chances of getting coronary artery disease may be attributed to genetics. Genes that may contribute to coronary artery disease influence [R]:

- Fat metabolism ([APOE](#), [APOB](#), [LPL](#), [LPA](#), [PCSK9](#))
- Inflammation ([IL5](#), [IL6R](#))
- Blood clotting ([SERPINA1](#))
- Blood vessel function ([NOS3](#), [TGFB1](#), [VEGFA](#), [ANGPTL4](#))

Genetically higher levels of the following markers are causally associated with a higher risk of heart disease [R, R, R, R, R, R, R, R, R, R, R]:

- White blood cells
- Fasting insulin
- IGF-1
- ApoB
- Neutrophils
- L-carnitine

In contrast, genetically high total testosterone and EPA may be causally associated with a lower risk of coronary heart disease [R, R].

GENE	SNP	GENOTYPE
PHOSPHO1	rs191896574	TC
FAM177B	rs17465982	AA
NOS3	rs3918226	TC
MRPS6	rs28451064	AG
LPA	rs73596816	AG
PEMT	rs7946	CT
TWIST1	rs2107595	GA
EDNRA	rs17612693	AT
TCF21	rs1966248	AT
DDI1	rs2128739	AC
FGD5	rs148880716	GG
LPA	rs140570886	TT
LPA	rs147555597	GG
PTGER4	rs17234657	TT
LPA	rs55730499	CC
SEH1L	rs2542151	TT
NOD2	rs17221417	CC
BSN	rs9858542	GG
MAP3K4	rs145099029	AA
CDKN2B	rs145542470	GG
NBEAL1	rs72934535	TT
SCAF11	rs1291621	GG
MTRNR2L7	rs4934855	AA
LPL	rs7011846	GG
SOX11	rs79576311	GG
SMIM11A	rs149487184	CC
BMP1	rs73225842	CC
BAG2	rs223290	CC
LRR25	rs11670056	CC

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

NOS3 (Cardiovascular)

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

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

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

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

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

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

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

The minor variants of [rs179983](#) and [rs2070744](#) have also been associated with:

- Worse [athletic performance](#) in power sports [\[R, R, R, R, R, R\]](#)
- Longer and more frequent [migraines](#) [\[R, R, R, R\]](#)

On the bright side, they are also linked to:

- Improved performance in aerobic sports and soccer [\[R, R, R\]](#)
- Greater decreases in triglycerides, cholesterol, and blood pressure in [response to unsaturated fats](#) such as [omega-3 fatty acids](#) and [extra virgin olive oil](#) [\[R, R, R, R, R\]](#)

Finally, the ‘T’ allele of [rs3918226](#) also results in lower *NOS3* levels and is associated with a higher risk and severity of heart and coronary events. Fortunately, this allele is extremely rare and most people (81-99%) have the ‘CC’ genotype [\[R, R, R\]](#).



TYPICAL ACTIVITY

Likely typical NOS3 activity based on 2 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NOS3	rs2070744	CT
NOS3	rs1549758	TC
NOS3	rs3918226	TC

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

Lipoprotein(A)

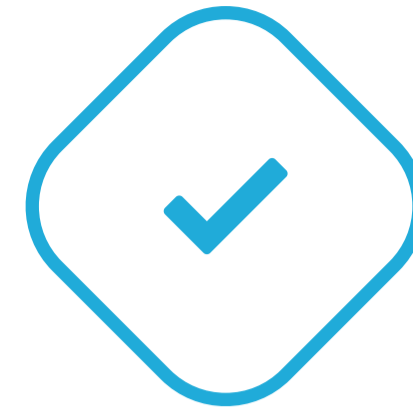
Lipoprotein(a), or Lp(a), is a type of LDL. It is made in the liver and carries fats such as cholesterol around the body. **Lp(a) may deposit on the artery walls.** This may lead to the formation of plaques that narrow the arteries. In line with this, higher Lp(a) levels have been associated with heart disease and stroke [R, R, R, R, R, R, R].

Genetics strongly influence Lp(a) levels. Up to **90%** of differences in people's Lp(a) levels may be due to genetics [R, R].

Genetically higher Lp(a) levels may be causally associated with:

- Heart health (cardiovascular diseases) [R, R, R, R, R]
- Stroke [R, R, R]
- Longevity (reduced) [R]
- Atrial fibrillation [R, R]
- Anemia [R]
- Prostate cancer [R]

The effect of diet on Lp(a) is still a matter of research [R].



TYPICAL LEVELS

Predisposed to typical Lipoprotein(a) levels based on 831 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
LPA	rs41267819	GG
FADS2	rs1535	AA
LPA	rs76144756	GG
LPA	rs143431368	TT
SLC22A1	rs182980975	CC
LPA	rs41267809	AA
LPA	rs73596816	AG
PLG	rs145535174	AA
LPA	rs41272114	CC
LPA	rs4708871	TT
CETP	rs5882	GA
APOB	rs693	AG
TCF7L2	rs7903146	TC
GCKR	rs780094	CT
SLC22A3	rs6919346	CT
LPA	rs74617384	AA
SLC22A3	rs3918291	TT
/	rs151135411	GG
LPA	rs142720914	GG
SLC22A3	rs117446263	GG
LPA	rs3798220	TT

GENE	SNP	GENOTYPE
AGPAT4	rs61735260	GG
MRPL18	rs146888147	GG
SIDT2	rs964184	CC
PCSK7	rs662799	AA
APOA5	rs3135506	GG
LPA	rs10455872	AA
SLC22A1	rs146534110	GG
SLC22A3	rs118133674	GG
PLG	rs4252152	TT
/	rs200865946	CC
LPA	rs41272112	CC
LPA	rs200376184	GG
SLC22A3	rs8187722	AA
PLG	rs41272078	CC
LPA	rs41264848	GG
SLC22A3	rs3127573	AA
PLG	rs4252128	CC
SLC22A1	rs2282143	CC
LPA	rs140306630	CC
IGF2R	rs12207188	CC
MRPL18	rs73020718	AA

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

Atrial Fibrillation

Key Takeaways:

- Up to **60%** of differences in people's chances of having atrial fibrillation may be due to genetics.
- Risk factors include age, heart disease, high blood pressure, lung disease, sleep apnea, and thyroid disease.
- If you have a high genetic risk, you may lower your overall risk by taking action on risk factors that you can change.
- Symptoms include palpitations, chest pain, fatigue, dizziness, shortness of breath, and weakness.
- Click the **Recommendations** tab for potential dietary and lifestyle changes, and **next steps** for relevant labs.

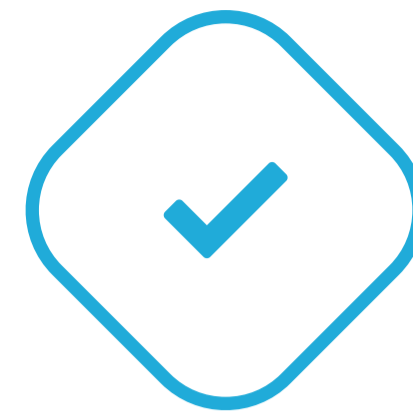
Some of the risk factors for AFib include [\[source\]](#):

- Older age
- Alcohol use
- Use of stimulants, including certain medications, caffeine, and tobacco
- Obesity
- Family history of AFib

The following conditions may contribute to AFib [\[source\]](#):

- Heart disease (coronary artery disease, heart attack, congenital heart defects, heart valve problems)
- High blood pressure
- Lung diseases
- Thyroid disease
- Chronic kidney disease
- Diabetes and metabolic syndrome
- Sleep apnea

Up to **60%** of differences in people's chances of having atrial fibrillation may be due to genetics [\[source\]](#).



TYPICAL LIKELIHOOD

Typical likelihood of atrial fibrillation based on 1,049,356 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HP	rs2359171	AA
PITX2	rs2129977	AG
PITX2	rs112599895	GA
PITX2	rs143269342	CC
TBX5	rs883079	TT
GJA5	rs79187193	GG
PITX2	rs75021220	TC
PITX2	rs3853445	TT
PITX2	rs6847935	TA
PITX2	rs6843082	GA
NEURL1	rs11598047	AG
PBXIP1	rs11264280	TC
PITX2	rs17570669	AA
SCN5A	rs7373065	TC
PITX2	rs13105878	CA
MYOZ1	rs6480708	AC
MYOZ1	rs60212594	CG
CLIC6	rs2834618	GT
UBE4B	rs187585530	GG
METTL11B	rs72700114	GG
TXNDC12	rs146518726	GG

GENE	SNP	GENOTYPE
C11ORF45	rs76097649	GG
PITX2	rs149829837	TT
RPL3L	rs140185678	GG
PITX2	rs2595104	GG
SH3PXD2A	rs35176054	TT
SELL	rs12122060	TT
FBXO32	rs78332318	CC
KCNN3	rs34292822	GG
FBXO32	rs62521286	AA

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



Miscellaneous


This section examines additional genetic factors that influence various aspects of your health and physiology beyond the primary categories. Understanding these genetic variations provides insights into your predisposition to specific conditions and metabolic processes that affect your overall well-being.

Your genetic profile affects diverse health areas including metabolic regulation, immune system function, and respiratory health. This analysis explores how your genes influence your susceptibility to conditions like insulin resistance, thyroid disorders, and asthma, providing a comprehensive view of your genetic health landscape.


This additional genetics assessment helps you understand important health considerations that significantly impact your daily life and long-term health outcomes, enabling you to make informed decisions about prevention and management strategies.

Topics include:

- Insulin Resistance
- Thyroid
- Asthma

 **MORE LIKELY**
Asthma

More likely to get asthma

 **TYPICAL**
Insulin Resistance

Predisposed to typical insulin resistance

 **TYPICAL LIKELIHOOD**
Hashimoto's Disease

Typical likelihood of Hashimoto's disease

 **LESS LIKELY**
Graves' Disease

Less likely to have Graves' disease

Asthma

Key Takeaways:

- Up to **70%** of differences in people's chances of developing asthma may be due to genetics.
- About **300 million** people worldwide are believed to have asthma, and many will develop it at a young age.
- Asthma triggers include viral infections, tobacco smoke, cold air, pollen, some medications, chemical fumes, and stress.
- A high genetic risk may mean greater susceptibility to triggers, so take actions to reduce your risk.
- Click the **Recommendations** tab for potential dietary and lifestyle changes and **next steps** for relevant labs.

Asthma is a chronic condition of the airway and lungs. In response to a trigger, the airway becomes inflamed. This reaction narrows the tubes that carry air into the body [\[R\]](#).

The symptoms of an asthma attack include [\[R\]](#), [\[R\]](#):

- Difficulty breathing
- Coughing
- Wheezing
- Tightness in the chest

About 300 million people are believed to have asthma worldwide. Asthma is more common in children than in adults. It is also much more common in people living below the poverty line [\[R\]](#), [\[R\]](#).

In people with asthma, attacks can be triggered by [\[R\]](#), [\[R\]](#):

- Viral infections
- Tobacco smoke
- Pollen
- Some medications
- Chemical fumes
- Stress
- Cold air

Asthma attacks can range from mild to severe. At their worst, they can close the airway and be fatal [\[R\]](#), [\[R\]](#).

Asthma has no known cure. If you have asthma, your doctor may prescribe an inhaler to control attacks. Your doctor can also help you recognize triggers that can lead to an attack. Then you can take steps to avoid them [\[R\]](#), [\[R\]](#), [\[R\]](#).

The exact cause of asthma is unknown, but genetics is thought to play a major role [\[R\]](#).

In fact, **up to 70% of differences in people's chances of developing asthma may be attributed to genetics.** Genes involved in asthma may influence [\[R\]](#), [\[R\]](#), [\[R\]](#):

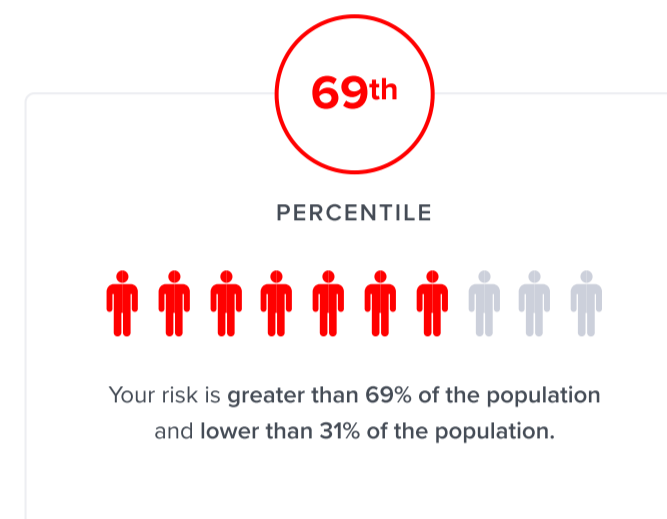
- Inflammation ([IL33](#), [IL1RL1](#), [TSLP](#), [ORMDL3](#))
- Autoimmune reactions ([HLA-DQA1](#), [HLA-DQB1](#), [HLA-DQA2](#))
- Lung cell death ([GSDML](#))

Genetically high white blood cell count may be causally associated with asthma in people with African ancestry. In contrast, genetically higher IGF-1 levels may be causally associated with a lower risk of asthma [\[R\]](#), [\[R\]](#).



MORE LIKELY

More likely to get asthma based on 1,049,396 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
STAT6	rs3024971	TT
PTGER4	rs7720838	TT
LPP	rs9865818	GG
RUNX3	rs760805	TT
LRRC32	rs2155219	GT
TSLP	rs1438673	TC
HLA-DQA1	rs6906021	TC
SH2B3	rs10774625	AG
GSDMB	rs9303280	CT
NFATC2	rs6021270	TC
GATA3	rs10795656	GA
/	rs115468973	TT
/	rs201184533	CC
/	rs116189786	AA
IL2RA	rs12722502	CC
IL18R1	rs78545931	AA
IL18RAP	rs72823641	TT
HLA-DRB5	rs1064713	GA
CCR7	rs112401631	TT
LINGO4	rs12123821	CC
KIAA1109	rs17454584	AA

GENE	SNP	GENOTYPE
IL33	rs144829310	GG
IL2RA	rs61839660	CC
IL1RL1	rs950880	CC
IRF4	rs11242709	CC
IL13	rs20541	GG
SERPINB10	rs12964116	AA
OVOL1	rs479844	AA
NOD2	rs2066844	CC
TLR1	rs17616434	CC
OR2B6	rs7767176	GG
SMAD3	rs17228058	AA
FLG	rs61816761	GG
KRT24	rs8067124	AA
LINGO4	rs115045402	GG
LRRC32	rs55646091	GG

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

Insulin Resistance

Insulin resistance is the reduction of the body’s ability to control blood sugar levels. It happens when the muscles, liver, and fat cells no longer respond to insulin and have trouble taking sugar up [R].

In response, the pancreas is forced to produce more insulin than normal to keep blood sugar in balance. Hence, people with insulin resistance may have high insulin levels. Blood sugar levels may also rise eventually, paving the way for diabetes [R, R].

Homeostatic model assessment ([HOMA-IR](#)) helps measure insulin resistance. It is calculated using your fasting glucose and fasting insulin. The higher your HOMA-IR, the more insulin resistant you are [R, R].

Insulin resistance is commonly caused by two factors: **overeating and lack of physical activity**. These can cause a buildup of fat in the liver and muscles that lead to insulin resistance [R, R, R].

Insulin resistance is associated with overweight and obesity, especially due to the accumulation of belly fat. However, normal-weight people may also have insulin resistance. Other health conditions may also lead to insulin resistance, including [R]:

- Sleep apnea [R]
- Thyroid disorders [R, R, R]
- Polycystic ovary syndrome (PCOS) [R, R]
- Pancreas disease [R, R]
- Acromegaly (too much growth hormone) [R]
- Cushing’s syndrome (excess of cortisol) [R]
- Rare genetic diseases [R, R, R, R]

Keep in mind that this report is not about the rare genetic disorders mentioned above. They are very rare and usually diagnosed in infancy.

The risk of insulin resistance may also increase due to:

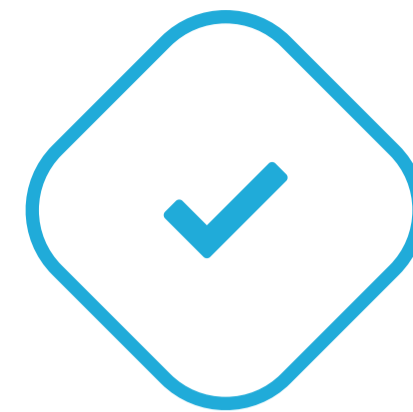
- Aging [R, R]
- Stress [R, R]
- Fasting [R, R, R]
- Western diet [R]
- Too little sleep [R, R, R, R]
- Pregnancy [R]
- Exposure to toxins (e.g., herbicides) [R, R, R]
- Some drugs (e.g., corticosteroids) [R, R]

Genetics also influences insulin resistance. Up to **65%** of differences in people’s insulin resistance may be due to genetics [R, R].

Insulin resistance may increase the risk of:

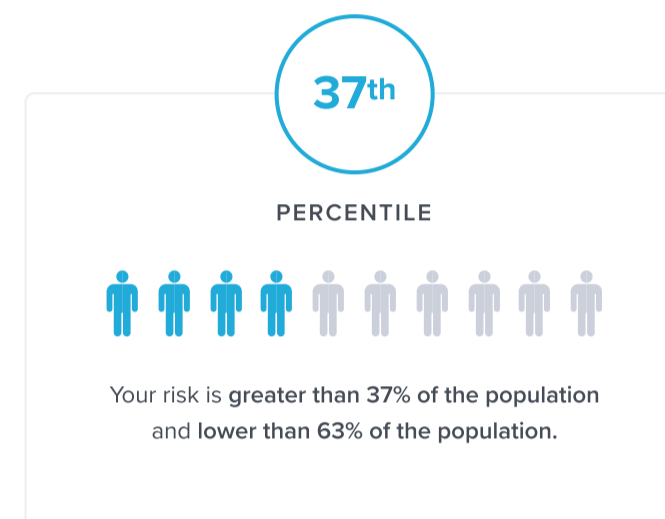
- Diabetes
- Liver disease
- Metabolic syndrome

Interestingly, insulin resistance may occur up to 15 years before diabetes develops. Read [this post](#) for a detailed list of tips to reduce insulin resistance [R].



TYPICAL

Predisposed to typical insulin resistance based on 2,420 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GAS1	rs9792548	AA
PPARG	rs1801282	CC
IRS1	rs2943641	TC
PPARG	rs3856806	CC
FOXO3	rs13217795	TT
FOXO3	rs2802288	AA
IGF1	rs35767	GG
NAT2	rs1208	AA
TIMP4	rs13081389	AA
KLHL2	rs17046216	AA
LEPR	rs1137101	AG
MRPS31	rs4581585	CC
ZC3H12C	rs475338	AA
FBXO21	rs2036313	GG
HAPLN1	rs1457105	CC
/	rs12969333	AA
DAAM2	rs4345393	GG
ME1	rs11967452	CC
KCNK17	rs10456469	GG
ORMDL3	rs939345	CC
ZIC2	rs7338383	GG

GENE	SNP	GENOTYPE
CSNK2A1	rs6053042	CC
RAB28	rs1197712	AA
ATP8B1	rs10439020	AA
MPC1	rs2281056	AA
MROH8	rs11698899	GG
RUNX3	rs803323	AA
TLR4	rs13290714	CC
SORCS1	rs7088188	CT
MDGA1	rs17589516	AA
CACNA1D	rs1401492	CC
SLC10A2	rs16962638	AA
ATP10A	rs6576507	TT
/	rs7043482	AC
CSMD1	rs2407314	CC
FTO	rs9939609	TT
TCERG1L	rs7077836	GG
ADRB3	rs4994	AA
FABP2	rs1799883	CC
FTO	rs1421085	TT
FTO	rs1121980	GG
BRD1	rs13057821	CC
KL	rs9535766	TT
UBR1	rs17776090	AA
BMP8A	rs710912	CC
/	rs2873975	GG
POLL	rs3730464	AA
RUNX1	rs17227476	GG
NINL	rs11698267	GG
FAM135B	rs10088248	CC

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

Hashimoto's Disease

Key Takeaways:

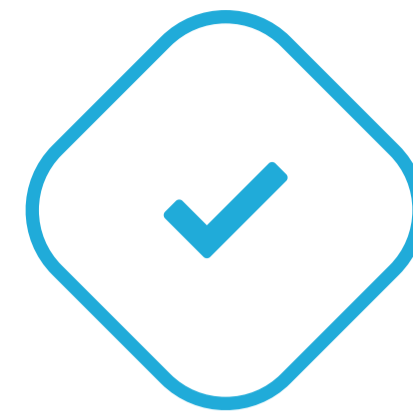
- Up to **65%** of differences in people's chances of having Hashimoto's disease may be due to genetics.
- Risk factors include being female, middle age, pregnancy, other autoimmune diseases, and excessive iodine intake.
- It affects 1 to 2 percent of people in the U.S., occurring more often in women than men.
- Click the **Recommendations** tab for potential dietary and lifestyle changes, and **next steps** for relevant labs.

Risk factors for Hashimoto's disease include [\[R\]](#):

- Being female
- Middle age
- Pregnancy
- Excessive iodine intake
- Radiation exposure
- Having another autoimmune disease
- **Genetics**

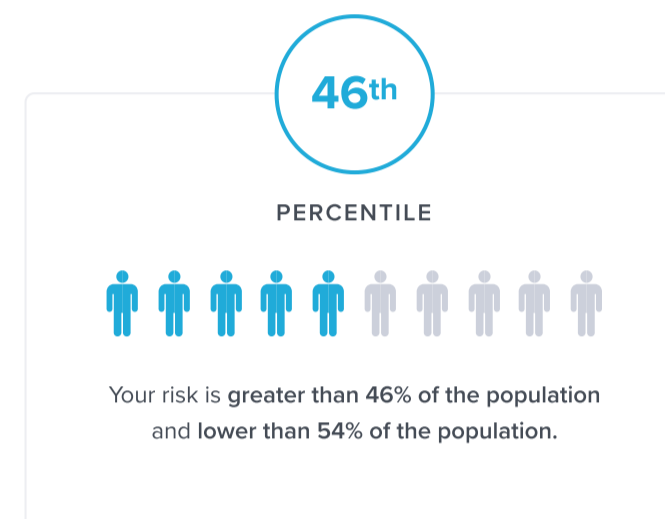
Up to **65%** of differences in people's chances of having Hashimoto's disease may be due to genetics [\[R\]](#).

Hashimoto's disease is typically treated with medications to help normalize thyroid hormone levels. **It's important for people with Hashimoto's disease to work closely with their healthcare provider** to manage their condition and prevent complications.



TYPICAL LIKELIHOOD

Typical likelihood of Hashimoto's disease based on 85 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CTLA4	rs3087243	GG
HLA-DPA1	rs9277768	TC
IL6	rs1800795	GG
CTLA4	rs34636506	AA
/	rs9271365	TG
VAV3	rs7537605	GA
TRIB2	rs1534422	GG
PDE8B	rs1993945	TA
SH2B3	rs653178	CT
STAT4	rs11889341	TC
TPO	rs11675434	CT
CTLA4	rs231775	GA
CD69	rs2110451	AG
RPS26	rs11611029	CT
NIPSNAP1	rs757024	CG
SESN3	rs4409785	TC
ZNF668	rs57348955	AG
TNFRSF14	rs2843403	CT
PTPN22	rs2476601	GG
SLC25A27	rs2270450	CC
VAV3	rs17020139	GG

GENE	SNP	GENOTYPE
TRMO	rs7030280	CC
PTPN22	rs1230666	GG
BACH2	rs10944479	GG
TNF	rs1799964	TT
AP4B1	rs12730735	TT
TNF	rs1800629	GG
CTLA4	rs11571297	TT
BACH2	rs7754251	GG
BACH2	rs72928038	GG
LPP	rs13093110	CC
IL2RA	rs706779	TC
GXYLT1	rs4768412	CC

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

Graves' Disease

Key Takeaways:

- Up to **80%** of differences in people's chances of getting Graves' disease may be due to genetics.
- Risk factors include young age, female sex, stress, smoking, and pregnancy.
- Symptoms include weight loss, rapid heartbeat, difficulty sleeping, eye bulging, and sexual dysfunction.
- If you have a high genetic risk, your overall risk is low due to its rarity. You can still improve this risk by taking action on those risk factors you can change.
- Click the **Recommendations** tab for potential dietary and lifestyle changes, and **next steps** for relevant labs.

Risk factors for Graves' disease include [\[R\]](#):

- **Genetics**
- Being female
- Age under 40
- Other autoimmune disorders
- Emotional or physical stress
- Pregnancy
- Smoking

Up to **80%** of differences in people's chances of getting Graves' disease may be due to genetics [\[R\]](#).



LESS LIKELY

Less likely to have Graves' disease based on 176 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CTLA4	rs3087243	GG
TSHR	rs179247	AA
TSHR	rs28414437	CA
SESN3	rs4409785	TC
CTLA4	rs231779	TC
TPO	rs11675434	CT
IGLV3-21	rs5751536	GA
TSHR	rs2300519	AT
CTLA4	rs231775	GA
CD40	rs1569723	CA
MAF	rs17689159	CT
FCRL3	rs1977710	AG
UHRF1BP1	rs9469899	AG
STAT4	rs12612769	CA
TMPRSS3	rs34544259	GA
RNASET2	rs13210649	TG
TRIB2	rs1534422	GG
TSHR	rs4903964	AG
SH2B3	rs653178	CT
ZNF668	rs57348955	AG
TNFRSF14	rs2843403	CT
IL2RA	rs706779	TC
CD40	rs1883832	TC
HLA-DPA1	rs9357156	AA
SLAMF6	rs12026490	TT
ALDH2	rs4646776	GG
HLA-DQA1	rs2187668	CC
PTPN22	rs2476601	GG
MAGT1	rs4826198	A

GENE	SNP	GENOTYPE
TNF	rs1799964	TT
MICB	rs361525	GG
TNF	rs1800629	GG
CTLA4	rs11571297	TT
BACH2	rs7754251	GG
BACH2	rs72928038	GG
FCRL3	rs3761959	CC
GXYLT1	rs4768412	CC
LPP	rs13093110	CC

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

Recommendations Details

1

Aerobic Exercise (Cardio)

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

TYPICAL STARTING DOSE**1 hour**

Helps with these Symptoms & Conditions:

Allergies

Anxiety

High Blood Pressure

Migraines

Helps with these Goals:

Energy

Immunity

Mood

Helps with these DNA Risks:

Asthma

Coronary Artery Disease

Melanoma

Helps with these Lifestyle Risks:

Attention Deficit Hyperactivity Disorder (ADHD)

Anxiety

Asthma

2

Strength Training

Engage in strength training exercises, such as weight lifting or bodyweight exercises, for 60 minutes per session, 2 to 3 times per week. Ensure you work all major muscle groups and rest each muscle group for at least 48 hours before exercising it again.

TYPICAL STARTING DOSE**1 hour**

Helps with these Symptoms & Conditions:

Anxiety

High Blood Pressure

Helps with these Goals:

Immunity

Mood

Muscle Growth

Helps with these DNA Risks:

⚠️ Coronary Artery Disease

⚠️ Muscle Mass

Helps with these Lifestyle Risks:

⚠️ Attention Deficit Hyperactivity Disorder (ADHD)

⚠️ Anxiety

3  **Omega-3 (Fish Oil)**

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

TYPICAL STARTING DOSE

500 mg

Helps with these Symptoms & Conditions:

Anxiety

High Blood Pressure

Migraines

Helps with these Goals:

Exercise Recovery

Immunity

Mood

Helps with these DNA Risks:

⚠️ Asthma

⚠️ Coronary Artery Disease

⚠️ Melanoma

⚠️ GAD1 (Glutamate/GABA)

Helps with these Lifestyle Risks:

⚠️ Attention Deficit Hyperactivity Disorder (ADHD)

⚠️ Anxiety

✅ Asthma

4  **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

500 mg

Helps with these Symptoms & Conditions:

Anxiety

High Blood Pressure

Helps with these Goals:

Immunity

Helps with these DNA Risks:

 Asthma

 Coronary Artery Disease

Helps with these Lifestyle Risks:

 Anxiety

 Asthma

5



Music Therapy

Engage in music therapy sessions for at least 30 minutes a day, three times a week. These sessions can involve listening to music, playing an instrument, singing, or writing songs, facilitated by a certified music therapist if possible.

TYPICAL STARTING DOSE

30 minutes

Helps with these Symptoms & Conditions:

Anxiety

High Blood Pressure

Migraines

Helps with these Goals:

Exercise Recovery

Mood

Helps with these DNA Risks:

 Asthma

 Coronary Artery Disease

Helps with these Lifestyle Risks:

 Attention Deficit Hyperactivity Disorder (ADHD)

 Anxiety

 Asthma

6



Mediterranean Diet

Incorporate a variety of primarily plant-based foods, such as fruits, vegetables, whole grains, nuts, and legumes, into every meal. Choose healthy fats, like olive oil, over saturated fats and consume fish and poultry at least twice a week. Limit red meat to a few times a month and include a moderate amount of dairy products. Opt for water and red wine in moderation as your beverages.

Helps with these Symptoms & Conditions:

Allergies

High Blood Pressure

Helps with these Goals:

Energy

Mood

Helps with these DNA Risks:

 Asthma

 Coronary Artery Disease

 Melanoma

Helps with these Lifestyle Risks:

 Attention Deficit Hyperactivity Disorder (ADHD)

 Asthma

7



Yoga

Practice yoga for at least 20 to 30 minutes a day, most days of the week. Choose a style that matches your fitness level and goals, and consider attending a class or using online resources to guide your practice.

TYPICAL STARTING DOSE

30 minutes

Helps with these Symptoms & Conditions:

Anxiety



High Blood Pressure

Migraines




Helps with these Goals:

- Energy
- Exercise Recovery
- Immunity
- Mood
- Muscle Growth


Helps with these DNA Risks:

-  Asthma
-  Coronary Artery Disease
-  Muscle Mass

Helps with these Lifestyle Risks:

-  Attention Deficit Hyperactivity Disorder (ADHD)
-  Anxiety
-  Asthma

8



Ginkgo

Take 120 mg of Ginkgo supplement daily, preferably with meals to aid absorption. This dosage is typically split into two 60 mg doses taken in the morning and evening for best results.

TYPICAL STARTING DOSE
120 mg




Helps with these Symptoms & Conditions:

- Allergies
- Anxiety
- Migraines


Helps with these DNA Risks:

-  Asthma
-  Coronary Artery Disease

Helps with these Lifestyle Risks:

-  Attention Deficit Hyperactivity Disorder (ADHD)
-  Anxiety
-  Asthma

9



Relaxation Techniques

Incorporate relaxation techniques such as deep breathing exercises, meditation, or yoga into your daily routine. Spend at least 15-30 minutes each day practicing one of these techniques, preferably in a quiet, comfortable space without interruptions.

TYPICAL STARTING DOSE
30 minutes

Helps with these Symptoms & Conditions:

- Anxiety
- High Blood Pressure
- Migraines

Helps with these Goals:

- Energy
- Immunity
- Mood

Helps with these DNA Risks:

- ⚠️ Coronary Artery Disease

Helps with these Lifestyle Risks:

- ⚠️ Attention Deficit Hyperactivity Disorder (ADHD)
- ⚠️ Anxiety

10  **Magnesium**

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

TYPICAL STARTING DOSE
250 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:

- ⚠️ Asthma
- ⚠️ Coronary Artery Disease

Helps with these Lifestyle Risks:

- ⚠️ Attention Deficit Hyperactivity Disorder (ADHD)
- ⚠️ Anxiety
- ✅ Asthma

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Tai Chi

Practice Tai Chi for 30 to 60 minutes at least twice a week. Choose a quiet, spacious area and follow along with a qualified instructor, either in person at a class or through an online video tutorial, to ensure proper technique and maximum benefit.

TYPICAL STARTING DOSE

1 hour

Helps with these Symptoms & Conditions:

Anxiety

High Blood Pressure

Helps with these Goals:

Energy

Mood

Helps with these DNA Risks:

Coronary Artery Disease

Helps with these Lifestyle Risks:

Attention Deficit Hyperactivity Disorder (ADHD)

Anxiety

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Avoid Air Pollution

Stay indoors on days when air quality indexes (AQI) indicate high pollution levels, which are often reported by weather services or government environmental agencies. **Install air purifiers** in your home, especially in bedrooms, to reduce indoor pollutants. Limit outdoor exercise when air pollution warnings are issued, opting for indoor activities instead.

Helps with these Symptoms & Conditions:

Allergies

Anxiety

High Blood Pressure

Helps with these Goals:


Immunity

Mood


Helps with these DNA Risks:

-  Asthma
-  Coronary Artery Disease

Helps with these Lifestyle Risks:

-  Anxiety
-  Asthma

13



Meditation

Set aside 10-20 minutes each day in a quiet space without distractions to practice meditation. Focus on your breath or perform guided meditation using an app or audio track.

TYPICAL STARTING DOSE
30 minutes


Helps with these Symptoms & Conditions:

- Anxiety
- Migraines

Helps with these Goals:

- Energy
- Immunity
- Mood


Helps with these DNA Risks:

-  Coronary Artery Disease

Helps with these Lifestyle Risks:

-  Attention Deficit Hyperactivity Disorder (ADHD)
-  Anxiety

14



Methylfolate

Take an L-methyl folate supplement (400-800 micrograms daily), ideally with a meal, to improve absorption. This dosage is recommended for adults, including pregnant women, to support overall health, especially to reduce the risk of neural tube defects in developing fetuses. Continue daily use as part of your regular supplement routine.

TYPICAL STARTING DOSE
400 mcg

Helps with these Symptoms & Conditions:

High Blood Pressure

Helps with these Goals:

Immunity

Mood

Helps with these DNA Risks:

⚠️ Coronary Artery Disease

⚠️ Melanoma

⚠️ MTHFR

⚠️ Folate (Vitamin B9)

15



Walking

Incorporate at least 30 minutes of brisk walking into your daily routine, aiming for a minimum of five days a week. This can be done in one continuous session or broken into shorter periods, such as three 10-minute walks throughout the day.

TYPICAL STARTING DOSE

30 minutes

Helps with these Symptoms & Conditions:

Anxiety

High Blood Pressure

Helps with these Goals:

Energy

Mood

Helps with these DNA Risks:

⚠️ Coronary Artery Disease

Helps with these Lifestyle Risks:

⚠️ Anxiety

Next Steps

Remember, your genes only tell one important part of your health story!

Now that you've seen your DNA-based results for this health topic, let's take a look at other contributing factors.

Your Lifestyle Assessments

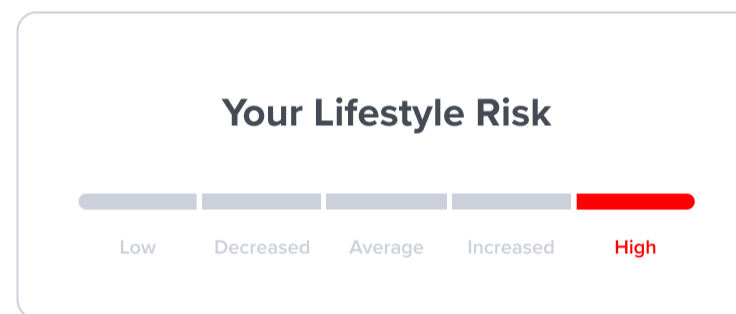
Ever heard of the term Nature vs. Nurture?

The thing is, both DNA and environment play a role in determining your health risks. The following assessments shows how much of an impact your lifestyle, environment and medical history are having on your health risks.



LIFESTYLE

You have an **increased risk** of Attention Deficit Hyperactivity Disorder (ADHD) based on the answers you provided.



Factors impacting your risk:

Has your household ever received social welfare (e.g., Medicaid, housing assistance, AFDC etc.)?

Yes

Increasing Risk

How many years of formal education did your biological mother complete?

13-14 years

Increasing Risk

How much sleep do you get in a typical night?

6 hours or less

Increasing Risk

Do you have a parent or sibling who has ever been diagnosed with ADHD?

Yes

Increasing Risk

Did you grow up in a single-parent household for most of your early childhood?

Yes

Increasing Risk

What is your sex?

Male

Increasing Risk

Did your mother smoke while pregnant with you?

No

Decreasing Risk

Were you born early (before 37 weeks)?

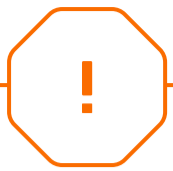
No

Decreasing Risk 

Has your biological mother ever suffered from depression?

Not sure

Decreasing Risk 



LIFESTYLE

You have a **slightly increased risk** of anxiety based on the answers you provided.



Factors impacting your risk:

Have either of your biological parents ever suffered from anxiety? Yes	Increasing Risk
What is your current marital status? Single or not living with partner	Increasing Risk
Do you consume at least 1000 mg caffeine per week (equivalent to 2 cups of coffee, 4 cups of tea, 6 cans of cola, or 1 energy drink per day)? Yes	Increasing Risk
What is your sex? Male	Increasing Risk
Do you regularly use drugs such as cannabis, cocaine, amphetamines, or opioids in a way that appreciably harms your health, social relationships, or occupational duties? Yes	Increasing Risk
Have you ever struggled with substance misuse? Yes	Increasing Risk
In a typical week, how many times do you participate in any physical activities or exercise for 30 minutes at a time? (such as walking, running, bike riding, weight training, yoga, etc.) 8 or more <small>*Note: longer exercise equals more sessions (e.g., 1 hour = 2 sessions)</small>	Decreasing Risk
Do you smoke tobacco? No, never	Decreasing Risk
Do you often experience periods of low mood? No	Decreasing Risk
How much alcohol do you drink on a typical day? Calculate your alcohol consumption in units here 0 units	Decreasing Risk
Did you ever suffer from physical abuse or physical bullying during your childhood? No	Decreasing Risk
Did you ever suffer from sexual abuse during your childhood? No	Decreasing Risk
Have you ever been diagnosed with rheumatoid arthritis (autoimmune joint inflammation)? No	Decreasing Risk

What is your ethnicity?

Other

Decreasing Risk 

Do you suffer from chronic pain?

No

Decreasing Risk 

What is your current employment status?

Self-employed

Decreasing Risk 

Did you ever suffer from emotional or physical neglect during your childhood?

No

Decreasing Risk 

Have you ever been diagnosed with alcohol use disorder?

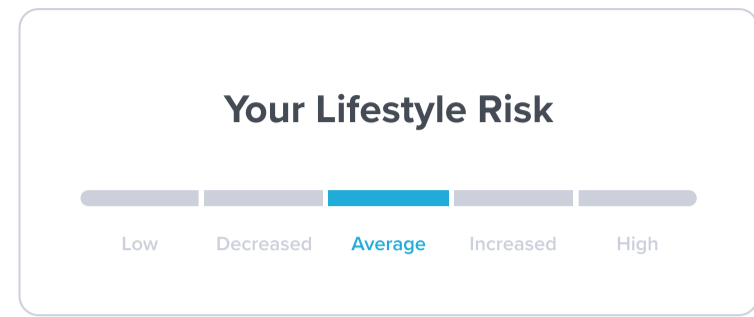
No

Decreasing Risk 



LIFESTYLE

You have an **average risk** of asthma based on the answers you provided.



Factors impacting your risk:

Your BMI: 30.77	Increasing Risk
Have you ever been diagnosed with atopic dermatitis (eczema)? Yes	Increasing Risk
In a typical week, how many times do you participate in any physical activities or exercise for 30 minutes at a time? (such as walking, running, bike riding, weight training, yoga, etc.) *Note: longer exercise equals more sessions (e.g., 1 hour = 2 sessions) 8 or more	Decreasing Risk
Did your mother smoke while pregnant with you? No	Decreasing Risk
Have you ever been diagnosed with allergic rhinitis (hay fever)? No	Decreasing Risk
Do you have a parent or sibling who has ever been diagnosed with asthma? No	Decreasing Risk
How would you describe the environment you live in? Suburban	Decreasing Risk
What is your height? 178 cm	No impact
What is your current weight? 97.5 kg	No impact



LIFESTYLE

You have a **slightly reduced risk** of hashimoto's disease based on the answers you provided.



Factors impacting your risk:

Your BMI: 30.77	Increasing Risk
Have you ever been diagnosed with multiple sclerosis? No	Decreasing Risk
Have you been diagnosed with psoriasis? No	Decreasing Risk
Have you ever been diagnosed with rheumatoid arthritis (autoimmune joint inflammation)? No	Decreasing Risk
Have you ever been diagnosed with lupus? No	Decreasing Risk
Have you ever been diagnosed with type 1 diabetes? No	Decreasing Risk
Do you have a parent or sibling who has been diagnosed with Hashimoto's disease (autoimmune underactive thyroid)? No	Decreasing Risk
What is your sex? Male	Decreasing Risk
What is your height? 178 cm	No impact
What is your current weight? 97.5 kg	No impact