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Cardiovascular Health

Summary Report



REPORT CATEGORY

HEART & BLOOD VESSELS

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Table of Contents

03 **Summary**

04 **Overview of Your Results**

10 **Your Results in Details**

10 Heart Health

35 Brain Vessels

43 Veins & Other Vessels

59 Heart Rate

75 Blood Pressure

84 Heart Health Genes

111 Cholesterol & Lab Markers

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.

Personal information

NAME

SEX AT BIRTH

Female

REPORT PROVIDED BY

Summary

“Oh, my god! That will give you a heart attack!” said every cliche grandmother ever written. There is a notoriously long list for what “that” is, from high cholesterol to stress to weight. Unfortunately, people have a tendency to not worry about a health issue until it becomes a problem. So, it should be no surprise that **heart disease is the number one cause of death worldwide**!

On the bright side, **a third of deaths related to heart disease could be prevented**! Because heart disease is impacted by so many variables, it should come as no surprise that your genetics has a major impact on heart health and your ability to take preventive action.

This comprehensive report analyzes your genetic predisposition to different aspects of heart health, including:

- Heart and blood vessels
- Blood pressure
- Blood lipids and lab markers

This summary report contains:

89 **Genetic Results**

Overview of Your Results

Heart Health

MORE LIKELY

Heart Failure

More likely to have heart failure

MORE LIKELY

Aortic Valve Calcification

More likely to have aortic valve calcification

TYPICAL LIKELIHOOD

Artery Hardening

Typical likelihood of atherosclerosis

TYPICAL LIKELIHOOD

Coronary Artery Disease

Typical likelihood of coronary artery disease

TYPICAL LIKELIHOOD

Heart Attack

Typical likelihood of a heart attack

TYPICAL LIKELIHOOD

Rheumatic Heart Disease

Typical likelihood of rheumatic heart disease

TYPICAL LIKELIHOOD

Cardiovascular Disease

Typical likelihood of cardiovascular disease

TYPICAL LIKELIHOOD

Myocarditis

Typical likelihood of myocarditis

TYPICAL LIKELIHOOD

Aortic Stenosis

Typical likelihood of having aortic stenosis

TYPICAL LIKELIHOOD

Dilated Cardiomyopathy

Typical likelihood of dilated cardiomyopathy

TYPICAL LIKELIHOOD

Aortic Aneurysm

Typical likelihood of aortic aneurysm

LESS LIKELY

Mitral Valve Prolapse

Less likely to have mitral valve prolapse

LESS LIKELY

Hypertrophic Cardiomyopathy

Less likely to have hypertrophic cardiomyopathy

LESS LIKELY

Chest Pain (Angina)

Less likely to have angina

Brain Vessels

TYPICAL LIKELIHOOD

Stroke

Typical likelihood of stroke

TYPICAL LIKELIHOOD

Cerebral Small Vessel Disease

Typical likelihood of cerebral small vessel disease

TYPICAL LIKELIHOOD

Brain Ischemia

Typical likelihood of chronic cerebral ischemia

LESS LIKELY

Brain Aneurysm

Less likely to have a brain aneurysm

LESS LIKELY

Subarachnoid Hemorrhage

Less likely to have subarachnoid hemorrhage

LESS LIKELY

Brain Hemorrhage

Less likely to have a brain hemorrhage

Veins & Other Vessels

MORE LIKELY

Varicose Veins

More likely to have varicose veins

TYPICAL LIKELIHOOD

Venous Thromboembolism

Typical likelihood of venous thromboembolism

TYPICAL LIKELIHOOD

Deep Vein Thrombosis

Typical likelihood of getting DVT

TYPICAL LIKELIHOOD

Peripheral Artery Disease

Typical likelihood of peripheral artery disease

TYPICAL LIKELIHOOD

Blood Spots

Typical likelihood of purpura

TYPICAL LIKELIHOOD

Cold Hands and Feet (Raynaud's)

Typical likelihood of having Raynaud's

TYPICAL LIKELIHOOD

Blood Clotting

Typical likelihood of having thrombosis

LESS LIKELY

Chronic Venous Insufficiency

Less likely to have chronic venous insufficiency

LESS LIKELY

Peripheral Vascular Disease

Less likely to have peripheral vascular disease

LESS LIKELY

Vasculitis

Less likely to have vasculitis

LESS LIKELY

Retinal Vein Occlusion

Less likely to have retinal vein occlusion

Heart Rate

SLOWER

Heart Rate Recovery

Predisposed to lower HRR

TYPICAL

Heart Rate

Predisposed to typical heart rate

TYPICAL LIKELIHOOD

Tachycardia

Typical likelihood of tachycardia

TYPICAL LIKELIHOOD

Atrial Fibrillation

Typical likelihood of atrial fibrillation

TYPICAL LIKELIHOOD

POTS

Typical likelihood of POTS

TYPICAL LIKELIHOOD

Heart Arrhythmia

Typical likelihood of heart arrhythmia

HIGHER

Heart Rate Variability

Predisposed to higher HRV

LESS LIKELY

Palpitations

Less likely to have palpitations

LESS LIKELY

Bradycardia

Less likely to have bradycardia

Blood Pressure

MORE LIKELY

Low Blood Pressure

More likely to have low blood pressure

TYPICAL

Salt Sensitivity

Likely typical sensitivity to salt

TYPICAL LIKELIHOOD

High Blood Pressure

Typical likelihood of hypertension

TYPICAL LIKELIHOOD

Preeclampsia

Typical likelihood of preeclampsia

TYPICAL LIKELIHOOD

Pulmonary Hypertension

Typical likelihood of pulmonary hypertension

Heart Health Genes

E3/E4

APOE

You carry one ε3 and one ε4 APOE variant

HIGHER ACTIVITY

CETP (Cholesterol/ Longevity)

Likely higher CETP activity

HIGHER ACTIVITY

FABP2 (Blood Sugar/ Cardiovascular)

Likely higher FABP2 activity

LOWER ACTIVITY

NOS3 (Cardiovascular)

Likely lower NOS3 activity

HIGHER ACTIVITY

PCSK9 (Cholesterol)

Likely higher PCSK9 activity

HIGHER ACTIVITY

CELSR2-PSRC1-SORT1 (Cardiovascular)

Likely higher CELSR2-PSRC1-SORT1 activity

LOWER ACTIVITY

CPS1 (Cardiovascular, Kidney Health)

Likely lower CPS1 activity

HIGHER ACTIVITY

AGT (Cardiovascular & Fitness)

Likely higher AGT activity

HIGHER ACTIVITY

SOAT1 (Cholesterol/ Cognition)

Likely higher SOAT1 activity

HIGHER ACTIVITY

ADRB1 (Cardiovascular)

Predisposed to higher ADRB1 activity

LOWER ACTIVITY

IRS1 (Metabolic Health)

Predisposed to lower IRS1 activity

TYPICAL ACTIVITY

APOA5 (Cardiovascular)

Predisposed to typical APOA5 activity

TYPICAL ACTIVITY

ABCG8 (Cholesterol & Gallstones)

Predisposed to typical ABCG8 activity

TYPICAL ACTIVITY

APOC3 (Blood Lipids/ Longevity)

Likely typical APOC3 activity

TYPICAL ACTIVITY

LIPC (Cardiovascular)

Likely typical LIPC activity

TYPICAL ACTIVITY

MLXIPL (Triglycerides, Cardiovascular)

Likely typical MLXIPL activity

TYPICAL ACTIVITY

HMGCR (Cholesterol)

Likely typical HMGCR activity

TYPICAL ACTIVITY

APOA2 (Weight, Blood Lipids)

Likely typical APOA2 activity

TYPICAL ACTIVITY

APOB Gene (Cardiovascular)

Likely typical APOB activity

TYPICAL ACTIVITY

LPA (Blood Lipids & Heart Health)

Predisposed to typical LPA activity

TYPICAL ACTIVITY

LDLR (Cholesterol, Cardiovascular)

Predisposed to typical LDLR activity

LOWER ACTIVITY

ACE (Fitness/ Cardiovascular)

Likely lower ACE activity

HIGHER ACTIVITY

GCH1 (Cardiovascular)

Likely higher GCH1 activity

HIGHER ACTIVITY

DOCK7 (Blood Lipids)

Likely higher DOCK7 activity

BETTER GENETICS

ABCA1 (Cholesterol)

Likely better ABCA1 genetics

Cholesterol & Lab Markers

MORE LIKELY

High Cholesterol

More likely to have high cholesterol

HIGHER LEVELS

Total Cholesterol

Predisposed to higher cholesterol levels

HIGHER LEVELS

LDL Cholesterol

Predisposed to higher levels of "bad" cholesterol

HIGHER LEVELS

TMAO

Predisposed to higher TMAO levels

TYPICAL

LDL Particle Size

Predisposed to typical LDL particle size

TYPICAL LEVELS

HDL Cholesterol

Predisposed to typical HDL levels

TYPICAL LEVELS

Lipoprotein(a)

Predisposed to typical Lipoprotein(a) levels

TYPICAL LEVELS

Triglycerides

Predisposed to typical triglyceride levels

TYPICAL EFFECTS

Effects of Omega-3s on Triglycerides

Predisposed to typical effects of omega-3s on triglycerides

TYPICAL LEVELS

VLDL Cholesterol

Predisposed to typical VLDL levels

TYPICAL

LDL Particle Number (LDL-P)

Predisposed to typical LDL-P

TYPICAL LEVELS

Homocysteine

Predisposed to typical homocysteine levels

TYPICAL LEVELS

Lp-PLA2

Predisposed to typical Lp-PLA2 levels

TYPICAL

Platelet Aggregation

Predisposed to typical platelet aggregation

TYPICAL LEVELS

Platelets

Predisposed to typical platelet count

TYPICAL

Response to Ezetimibe

Likely typical response to ezetimibe

LOWER LEVELS

ApoB

Predisposed to lower ApoB levels

LESS LIKELY

Metabolic Syndrome

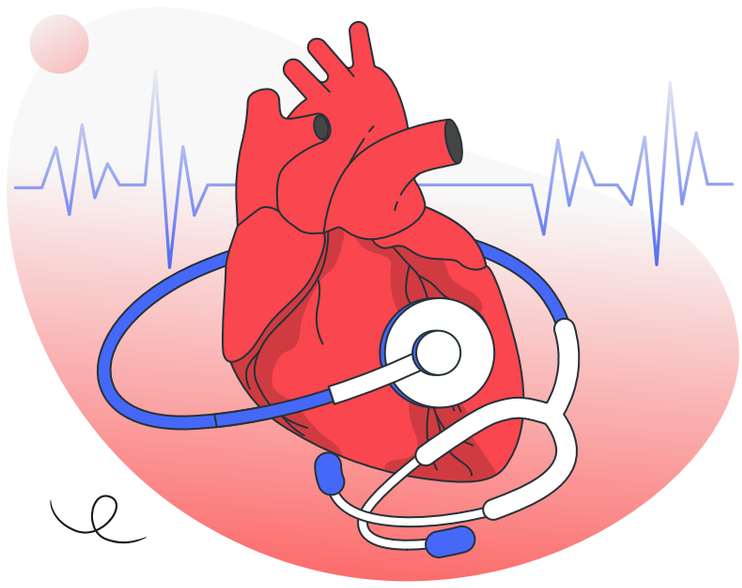
Less likely to have metabolic syndrome

BETTER RESPONSE

Response to Statins (Functional)

Predisposed to better response to statins

Your Results in Details



Heart Health

Yes, modern diets have done a lot to promote health issues related to the heart. However, other factors like your lifestyle and your DNA also have a significant impact. After all, ancient mummies have shown signs of artery hardening, and odds are good they weren't chowing down on twinkies!

Keeping the heart healthy is vital to avoiding long-term health issues. **This section dives into your genetic predispositions to things like heart disease, artery hardening, and arrhythmia**. This information can help you take the best course of actions over the long term.

MORE LIKELY

Heart Failure

More likely to have heart failure

MORE LIKELY

Aortic Valve Calcification

More likely to have aortic valve calcification

TYPICAL LIKELIHOOD

Artery Hardening

Typical likelihood of atherosclerosis

TYPICAL LIKELIHOOD

Coronary Artery Disease

Typical likelihood of coronary artery disease

TYPICAL LIKELIHOOD

Heart Attack

Typical likelihood of a heart attack

TYPICAL LIKELIHOOD

Rheumatic Heart Disease

Typical likelihood of rheumatic heart disease

TYPICAL LIKELIHOOD

Cardiovascular Disease

Typical likelihood of cardiovascular disease

TYPICAL LIKELIHOOD

Myocarditis

Typical likelihood of myocarditis

TYPICAL LIKELIHOOD

Aortic Stenosis

Typical likelihood of having aortic stenosis

TYPICAL LIKELIHOOD

Dilated Cardiomyopathy

Typical likelihood of dilated cardiomyopathy

TYPICAL LIKELIHOOD

Aortic Aneurysm

Typical likelihood of aortic aneurysm

LESS LIKELY

Mitral Valve Prolapse

Less likely to have mitral valve prolapse

LESS LIKELY

Hypertrophic Cardiomyopathy

Less likely to have hypertrophic cardiomyopathy

LESS LIKELY

Chest Pain (Angina)

Less likely to have angina

Heart Failure

Key Takeaways:

- Up to **25%** of differences in people's chances of having heart failure may be due to genetics.
- Key risk factors: high blood pressure, diabetes, coronary artery disease, arrhythmias, and infections.
- Heart failure affects about **26 million** people worldwide, with about **6 million** of those in the U.S.
- If your genetic risk is high, you may lower your overall risk by taking action on those risk factors that you can change.
- Make sure to click the **Next Steps** tab for relevant labs.

Up to **25%** of differences in people's chances of having heart failure may be due to genetics. For example, genetically high betaine and L-carnitine may be causally associated with a high risk of heart failure. In contrast, genetic predisposition to glucosamine supplement intake may be causally associated with a lower risk of heart failure [R, R, R, R].
A variety of health conditions can contribute to heart failure, including [R]:

- Coronary artery disease
- High blood pressure
- Diabetes
- Previous heart attacks
- Viral infections
- Heart inflammation (myocarditis)
- Heart rhythm problems
- Over/under-active thyroid



MORE LIKELY

More likely to have heart failure based on 35,698 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
FTO	rs56094641	GG
PITX2	rs7680240	AA
SRR	rs216193	AA
KLHL3	rs11745324	GG
CDK6	rs2282979	TT
MYOZ1	rs4746140	GC
BAG3	rs17617337	TC
SH2B3	rs10774624	GA
PITX2	rs1823290	TA
CHMP3	rs4832298	TC
USP36	rs2306527	TC
DMRTA2	rs116626164	TT
PITX2	rs17513625	GG
TXNDC12	rs80061532	CC
LPA	rs55730499	CC
PITX2	rs1906592	TT
CTAGE4	rs117540300	CC
ABO	rs600038	TT
SPDYE5	rs6944634	CC
ZFHX3	rs12325072	TT
PITX2	rs981150	AA
GNPDA2	rs10938398	GG
HSD17B12	rs7936836	AA
TM2D1	rs1997997	GG
NPC1	rs1788826	AA
STRN	rs7605601	GG
TBX3	rs35432	TT
CACNB2	rs1757223	AA
FAM177A1	rs1712355	GG

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

Aortic Valve Calcification

Calcification and stenosis generally affect older adults, as calcium builds up over time. When it occurs in younger people, it's often caused by [R]:

- A heart defect that's present at birth (congenital heart defect)
- Other illnesses, such as kidney failure

Other factors further increasing the risk of aortic valve calcification include [R]:

- High blood pressure
- High cholesterol levels
- Smoking
- History of rheumatic fever
- Family history of heart valve disease

There may be genetic components to aortic valve calcification, especially in individuals who develop it at a younger age. Specific genetic factors can predispose individuals to a higher risk of calcification and aortic valve disease.

Some possible ways to prevent aortic valve stenosis include:

- Controlling cardiovascular risk factors like high blood pressure, cholesterol, and not smoking.
- Regular physical activity and a heart-healthy diet.
- Timely treatment of any condition that can contribute to valve calcification, like rheumatic fever or kidney disease.
- Regular medical check-ups, especially for those with risk factors or a family history of heart disease.

Treatment for aortic valve calcification depends on the symptoms and the severity of the condition. If you have mild aortic valve symptoms or none, you may only need regular checkups by a healthcare provider. The provider may recommend healthy lifestyle changes and medications to treat valve disease symptoms or reduce the risk of complications. Severe cases may require surgery to repair or replace the aortic valve [R].



MORE LIKELY

More likely to have aortic valve calcification based on 26 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ACTR2	rs6715876	CC
RNMT	rs12605367	TT
DENND4C	rs10124723	GG
TREML4	rs7748777	AA
PALMD	rs7543039	CT
GLIPR1	rs11180610	TC
TACSTD2	rs170828	TG
STEAP1B	rs2069832	GA
ALPL	rs12141569	CT
PRRX1	rs2150026	CT
NAV1	rs665770	AG
ME3	rs11234705	GC
MECOM	rs2421649	AG
/	rs7593336	GA
HSD17B12	rs7937669	TG
EMX2	rs12264978	CC
ISG20	rs4932408	GG
/	rs113277183	GG
CADM1	rs112277963	TT
RND3	rs114314058	GG
LPA	rs10455872	AA
/	rs114795211	GG
STT3B	rs73056108	CC
FBRSL1	rs117206641	CC
ARHGAP24	rs17010961	TT
TPO	rs12465180	GG

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

Artery Hardening

Key Takeaways:

- About 60% of differences in people's chances of having atherosclerosis may be due to genetics
- Risk factors: being male, high blood pressure and cholesterol, diabetes, obesity, smoking, lack of exercise, poor diet, and age.
- If you are at high genetic risk, you may lower your overall risk by taking action on risk factors that you can change.
- Artery hardening begins at a young age and your genetics, lifestyle, diet, and exercise may all impact how much and how fast it develops over the course of your lifetime.

Click the **Recommendations** tab for potential dietary and lifestyle changes and **next steps** for relevant labs.

The arteries are blood vessels that carry oxygen and nutrients from the heart to the rest of the body. Normally, they are elastic, able to expand and contract as blood flows through them [R, R].

Over time, some people's arteries may lose that flexibility. The blood vessel walls become thick and stiff. This is called artery hardening (*arteriosclerosis*) [R].

The most common type of artery hardening is atherosclerosis. In this condition, fatty substances build up on the blood vessel walls. This buildup is called plaque. It can narrow the arteries, which reduces blood flow. It can also burst and cause a blood clot [R, R, R].

Atherosclerosis is the underlying cause of about 50% of all deaths in the Western world[R].

Men may be more likely to develop atherosclerosis than women. The risk tends to increase with age [R, R].

Other risk factors for atherosclerosis include [R]:

- High blood pressure
- High cholesterol
- Underlying conditions (e.g., diabetes, obesity, sleep apnea)
- Cigarette smoking
- Lack of exercise
- Unhealthy diet
- **Genetics**

In most cases, atherosclerosis won't cause any symptoms. In fact, most people don't have symptoms until a blood vessel is so narrow that an organ stops receiving enough blood. Symptoms of moderate or severe atherosclerosis can vary depending on the artery affected. They can include [R]:

- Chest pain (for blood vessels feeding the heart)
- Weakness, loss of vision, slurred speech (for blood vessels feeding the brain)
- Leg or arm pain (for blood vessels feeding the limbs)
- High blood pressure or kidney failure (for blood vessels feeding the kidneys)

To prevent artery hardening, doctors recommend [R]:

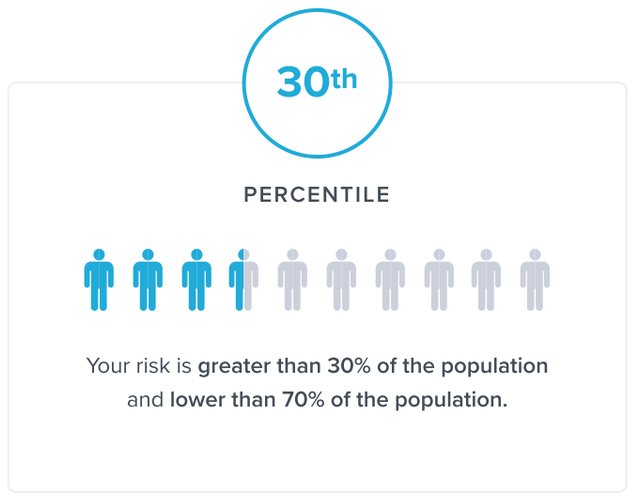
- Quitting smoking
- Eating healthy foods
- Staying fit

Once the condition progresses, treatment options can include [R]:



TYPICAL LIKELIHOOD

Typical likelihood of atherosclerosis based on 51,588 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PEMT	rs12936587	GG
NOS3	rs2070744	TC
CDKN2A	rs4977574	GG
CDKN2B	rs10757278	GG
PCSK9	rs11591147	GG
PCSK9	rs562556	AA
PCSK9	rs28362286	CC
COMT	rs4633	CC
COMT	rs4680	GA
CETP	rs5882	GA
ICAM1	rs5498	GA
FOXO3	rs2802292	GT
LRIG1	rs17045031	GG
LDLR	rs6511720	GG
CDKN2B	rs9632884	GC
APOC1	rs445925	AG
FAM167A	rs6601530	GA
ZHX2	rs11781551	GA
ZNF668	rs9923231	TT
CYP1A2	rs762551	AA
LPA	rs3798220	TT

- Medication
- Surgery
- Lifestyle changes

It's important to prevent, manage, or treat atherosclerosis. Left untreated, it can cause [R]:

- Heart attack
- Stroke
- Poor circulation in the arms and legs (peripheral artery disease)
- Bulges in blood vessels (aneurysms)
- Chronic kidney disease

Around 60% of differences in people's chances of artery hardening may be attributed to genetics. Genes involved in artery hardening may influence [R, R]:

- Cholesterol (*LRP6*)
- Inflammation (*ALOX5AP*, *LTA4H*)

Moreover, genetically high testosterone levels may be causally associated with a lower risk of atherosclerosis in men [R].

GENE	SNP	GENOTYPE
EDNRA	rs1878406	CC
CCDC71L	rs17398575	GG
CCDC71L	rs17477177	TT
STEAP1	rs259140	GG
FMN1	rs4779614	CC
BTN3A2	rs4712972	GG

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

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



TYPICAL LIKELIHOOD

Typical likelihood of 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
PEMT	rs12936587	GG
NOS3	rs2070744	TC
COMT	rs4680	GA
PCSK9	rs11591147	GG
PTGER4	rs17234657	GT
FHL3	rs190569784	GG
SERPINA1	rs112635299	GG
PHOSPHO1	rs191896574	TT
ANGPTL4	rs116843064	GG
APOE	rs7412	CC
NOD2	rs17221417	GC
LDLR	rs6511720	GG
IL23R	rs11805303	CT
ATG16L1	rs10210302	TC
/	rs72711827	GG
SORT1	rs12740374	GG
FBXL20	rs72823390	CC
PLPP3	rs17114046	AA
/	rs2457480	AA
NKX2-3	rs10883365	GA
ADO	rs10761659	GA

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
MCTP2	rs28607113	TT
PHACTR1	rs9349379	AG
PEMT	rs7946	TC
FAM177B	rs17465982	GA
DDI1	rs2128739	CA
FGD5	rs148880716	GG
LPA	rs140570886	TT
LPA	rs147555597	GG
LPA	rs55730499	CC
SEH1L	rs2542151	TT
IRGM	rs1000113	CC
BSN	rs9858542	GG
MAP3K4	rs145099029	AA
CDKN2B	rs145542470	GG
NBEAL1	rs72934535	TT
SCAF11	rs1291621	GG
NOS3	rs3918226	CC
MTRNR2L7	rs4934855	AA
MRPS6	rs28451064	GG
LPA	rs73596816	GG
LPL	rs7011846	GG
SOX11	rs79576311	GG
SMIM11A	rs149487184	CC
BMP1	rs73225842	CC
BAG2	rs223290	CC
LRRC25	rs11670056	CC
TTC32	rs16986953	GG
TRIM5	rs11601507	CC
TWIST1	rs2107595	GG

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

Heart Attack

Key Takeaways:

- **50-60%** of differences in people's chances of having a heart attack may be due to genetics.
- About **3 million people** around the world die from heart attacks every year.
- If you have a high genetic risk, you may lower your overall risk by taking action on risk factors that you can change.
- Other risk factors include age (45+ for men, 55+ for women), smoking, high blood pressure, obesity, diabetes, stress, and high cholesterol.
- Click the **Recommendations** tab for potential dietary and lifestyle changes, and **next steps** for relevant labs.

Risk factors for a heart attack include [R]:

- Age (over 45 for men and over 55 for women)
- Exposure to cigarette smoke
- Sedentary lifestyle
- Unhealthy diet
- Stress
- Recreational drug use
- **Genetics**

For example, genetically high ApoB, betaine, choline, and L-carnitine may be causally associated with a higher risk of myocardial infarction [R, R, R, R].

The following health conditions may contribute to a heart attack [R]:

- High blood pressure
- High cholesterol or triglycerides
- Obesity
- Diabetes
- Autoimmune conditions

About **50-60%** of differences in people's chances of having a heart attack may be due to genetics [R].



TYPICAL LIKELIHOOD

Typical likelihood of a heart attack based on 888,414 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CDKN2A	rs2891168	GG
GGT5	rs180803	GG
PLPP3	rs9970807	CC
SORT1	rs7528419	AA
SMARCA4	rs55791371	AA
COL4A1	rs11617955	TT
GUCY1A1	rs72689147	GG
SAYSD1	rs1544935	TT
PCSK9	rs11206510	TT
SFXN2	rs1004467	AA
SMAD3	rs72743461	CC
ZC3HC1	rs11556924	CC
VAMP8	rs10176176	TT
PHACTR1	rs9349379	AG
ATP5MC1	rs35895680	CC
IL6R	rs12118721	TT
MIA3	rs35700460	AG
SH2B3	rs653178	CT
LIPA	rs1332329	AC
COL4A2	rs55940034	AG
EDNRA	rs4593108	CG
DDI1	rs2019090	TA
CTSH	rs7165042	GC
SRR	rs9914266	CT
HHIPL1	rs10139550	CG
LPA	rs10455872	AA
PLG	rs2315065	CC
MRPS6	rs28451064	GG
ABO	rs532436	GG

GENE	SNP	GENOTYPE
APOE	rs56131196	GG
POC1B	rs2681472	AA
ZEB2	rs17678683	TT
TTC32	rs16986953	GG
BCAS3	rs7212798	TT
FES	rs2521501	AA
ZNF32	rs1870634	TT
JCAD	rs2505083	TT
TRIB1	rs2001846	CC

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

Rheumatic Heart Disease

Symptoms of rheumatic heart disease can range from mild to severe, including heart murmurs, chest pain, shortness of breath, and fatigue. The condition more commonly affects children and young adults, especially in areas of the world without easy access to healthcare.

Without proper care, including antibiotics to treat the initial streptococcal infection and medications to reduce inflammation, individuals with rheumatic heart disease may face increased risk of adverse health outcomes, including stroke or heart attack. It is also possible for infected individuals to experience recurrent episodes of rheumatic fever, which could further damage the heart valves.



TYPICAL LIKELIHOOD

Typical likelihood of rheumatic heart disease based on 1,064,485 genetic variants we looked at

Cardiovascular Disease

Cardiovascular disease is influenced by a combination of genetic, lifestyle, and environmental factors. Understanding these causes and risk factors is essential for prevention and effective management.

Genetic factors play a significant role in the development of cardiovascular disease. About 40-60% of differences in people's odds of heart disease may be due to genetics [R].

A family history of heart disease increases an individual's risk, as certain genetic mutations can affect cholesterol metabolism, blood pressure regulation, and the function of heart and blood vessels. For example, mutations in genes such as LDLR (low-density lipoprotein receptor) can lead to familial hypercholesterolemia, a condition characterized by high cholesterol levels and an increased risk of coronary artery disease.

Several lifestyle choices can significantly impact cardiovascular health:

- **Unhealthy Diet**: Diets high in saturated fats, trans fats, salt, and sugar can contribute to the development of atherosclerosis and hypertension.
- **Physical Inactivity**: A sedentary lifestyle increases the risk of obesity, hypertension, and diabetes, all of which are risk factors for CVD.
- **Smoking**: Tobacco use damages blood vessels, reduces oxygen in the blood, and raises blood pressure, significantly increasing the risk of heart disease.
- **Excessive Alcohol Consumption**: Drinking too much alcohol can lead to high blood pressure, heart failure, and stroke.

Other Risk Factors

- **Age**: The risk of cardiovascular disease increases with age, particularly after the age of 65.
- **Gender**: Men are generally at higher risk of developing CVD earlier in life compared to women, although post-menopausal women's risk increases.
- **High Blood Pressure**: Hypertension is a major risk factor as it puts extra strain on the heart and blood vessels.
- **High Cholesterol**: Elevated levels of LDL cholesterol contribute to the buildup of fatty deposits in arteries.
- **Diabetes**: Diabetes significantly increases the risk of CVD as high blood glucose levels can damage blood vessels.
- **Obesity**: Excess body weight, particularly around the abdomen, is associated with higher risk factors for CVD.



TYPICAL LIKELIHOOD

Typical likelihood of cardiovascular disease based on 1,049,427 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PEMT	rs12936587	GG
NOS3	rs2070744	TC
COMT	rs4680	GA
PCSK9	rs11591147	GG
PTGER4	rs17234657	GT
FHL3	rs190569784	GG
SERPINA1	rs112635299	GG
PHOSPHO1	rs191896574	TT
ANGPTL4	rs116843064	GG
APOE	rs7412	CC
NOD2	rs17221417	GC
LDLR	rs6511720	GG
IL23R	rs11805303	CT
ATG16L1	rs10210302	TC
/	rs72711827	GG
SORT1	rs12740374	GG
FBXL20	rs72823390	CC
PLPP3	rs17114046	AA
/	rs2457480	AA
NKX2-3	rs10883365	GA
ADO	rs10761659	GA

GENE	SNP	GENOTYPE
MCTP2	rs28607113	TT
PHACTR1	rs9349379	AG
PEMT	rs7946	TC
FAM177B	rs17465982	GA
DDI1	rs2128739	CA
FGD5	rs148880716	GG
LPA	rs140570886	TT
LPA	rs147555597	GG
LPA	rs55730499	CC
SEH1L	rs2542151	TT
IRGM	rs1000113	CC
BSN	rs9858542	GG
MAP3K4	rs145099029	AA
CDKN2B	rs145542470	GG
NBEAL1	rs72934535	TT
SCAF11	rs1291621	GG
NOS3	rs3918226	CC
MTRNR2L7	rs4934855	AA
MRPS6	rs28451064	GG
LPA	rs73596816	GG
LPL	rs7011846	GG
SOX11	rs79576311	GG
SMIM11A	rs149487184	CC
BMP1	rs73225842	CC
BAG2	rs223290	CC
LRRC25	rs11670056	CC
TTC32	rs16986953	GG
TRIM5	rs11601507	CC
TWIST1	rs2107595	GG

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

Myocarditis

Myocarditis can be caused by [R]:

- Viral infections, such as those caused by coxsackievirus, parvovirus, and adenovirus
- Bacterial infections, such asLyme disease
- Parasitic infections, such as those caused by *Trypanosoma cruzi* and toxoplasma
- Autoimmune diseases like lupus and rheumatoid arthritis
- Exposure to certain toxins, including alcohol, cocaine, and certain chemotherapy drugs
- Allergic reactions to medications

There is some evidence to suggest a genetic predisposition to myocarditis, particularly in cases where the condition recurs or is associated with other autoimmune diseases. Genetic factors might influence how the immune system responds to infections and other triggers of myocarditis.

Preventing myocarditis involves managing risk factors, such as avoiding infections, controlling autoimmune diseases, and avoiding toxic substances. Regular medical check-ups can help identify early signs of myocarditis, especially in people with symptoms of a recent viral infection and chest pain or palpitations.

Often, myocarditis improves on its own or with treatment. Myocarditis treatment focuses on the cause and the symptoms, and may include [R]:

- Medications to manage heart failure, control arrhythmias, and reduce inflammation
- Rest to reduce the workload on the heart during the acute phase of the disease
- Regular follow-up and monitoring for potential progression to dilated cardiomyopathy
- Avoiding alcohol and regular exercise until the inflammation has resolved
- Hospitalization in severe cases
- Ventricular assist device (VAD) or heart transplant in case of heart failure



TYPICAL LIKELIHOOD

Typical likelihood of myocarditis based on 16,734 genetic variants we looked at

Aortic Stenosis

About **50%** of the differences in aortic valve stenosis may be due to **genetics** [R].

Factors that might increase the risk of developing aortic valve stenosis include:

- Advanced age.
- A history of infections that can affect the heart.
- Chronic kidney disease.
- Radiation therapy to the chest.
- **Genetics**

Certain genetic factors and conditions increase the risk of developing aortic valve stenosis. For example, being born with a bicuspid aortic valve (which has two valve flaps instead of the normal three).

Additionally, certain genetic conditions, such as familial hypercholesterolemia, can lead to earlier and more severe valve calcification and stenosis.



TYPICAL LIKELIHOOD

Typical likelihood of having aortic stenosis based on 1,670 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ACTR2	rs6715876	CC
RNMT	rs12605367	TT
TREML4	rs7748777	AA
DENND4C	rs10124723	GG
PALMD	rs7543039	CT
PALMD	rs7543130	CA
TACSTD2	rs170828	TG
GLIPR1	rs11180610	TC
STEAP1B	rs2069832	GA
PRRX1	rs2150026	CT
/	rs1830321	TC
ALPL	rs12141569	CT
PALMD	rs11166276	CT
HSD17B12	rs7937669	TG
/	rs7593336	GA
NAV1	rs665770	AG
MECOM	rs2421649	AG
ME3	rs11234705	GC
/	rs2246363	AG
STEAP1B	rs13311155	CA
FLNB	rs1522388	CT
LPA	rs10455872	AA
EMX2	rs12264978	CC
ISG20	rs4932408	GG
/	rs113277183	GG
CADM1	rs112277963	TT
RND3	rs114314058	GG
/	rs114795211	GG
STT3B	rs73056108	CC

GENE	SNP	GENOTYPE
FBRSL1	rs117206641	CC
ARHGAP24	rs17010961	TT
TPO	rs12465180	GG
ELAVL3	rs10406522	TT
BLK	rs11250135	GG

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

Dilated Cardiomyopathy

The exact cause of dilated cardiomyopathy is often unclear, but several factors can contribute to or trigger its development:

- Genetic factors: A significant number of cases are inherited, though the specific genetics can be complex.
- Viral infections: Viruses that inflame the heart muscle, such as the Coxsackie virus, can lead to DCM.
- Alcohol and toxins: Chronic abuse of alcohol and exposure to certain toxic substances, including chemotherapy drugs and substances like cobalt, can damage the heart muscle.
- Autoimmune diseases: Conditions where the immune system mistakenly attacks the body's tissues, including the heart.
- Endocrine and metabolic disorders: Diseases such as diabetes or thyroid disorders can contribute to DCM.
- Neuromuscular disorders: Diseases like muscular dystrophy that affect the muscles and nerves can also impact heart muscle function.

There is no cure for dilated cardiomyopathy, but treatments can help manage symptoms and prevent complications:

- Medications: Such as ACE inhibitors, beta-blockers, diuretics, and aldosterone antagonists, which are used to manage blood pressure, prevent fluid build-up, and reduce the workload on the heart.
- Lifestyle changes: Including diet modifications, exercise, and quitting smoking.
- Devices and surgical procedures: In some cases, devices like pacemakers and implantable cardioverter-defibrillators (ICDs) are necessary to correct irregular heartbeats. In severe cases, a heart transplant may be considered.
- Monitoring and management: Regular follow-up visits with a cardiologist to monitor heart function and adjust treatment as necessary.

The prognosis for dilated cardiomyopathy varies widely depending on its severity, the effectiveness of treatment, and individual factors such as age and overall health. Some people live long, stable lives, while others may experience worsening heart failure or sudden cardiac death. Early detection and consistent treatment are crucial for improving the quality of life and outcomes for those affected by this condition.



TYPICAL LIKELIHOOD

Typical likelihood of dilated cardiomyopathy based on 498,517 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
BOC	rs1846594	AA
/	rs6556795	GG
MGAM	rs4341082	CT
IL19	rs17016480	AC
VEPH1	rs2649734	AG
ALG10B	rs11182052	TC
CDH10	rs4701446	AG
JPH3	rs8051448	CT
YTHDF1	rs73308970	AA
CACNB4	rs150793926	GG
ATXN7L3B	rs7486169	AG
RFTN2	rs75330306	AA
MARS2	rs139236944	GG
SPP1	rs72654150	TT
VEGFC	rs114108584	TT
/	rs10272945	CG
/	rs11742119	TT
VMP1	rs115624974	AA
CCDC149	rs2244757	CC
DNAH17	rs78212518	CC
TBX5	rs149932627	TT
CCND1	rs145355401	TT
RALY	rs140035275	CC
OR2F2	rs73462438	AA
RAG2	rs77698332	AA
FAM120B	rs149215314	GG
THSD7A	rs74676849	AA
PARD3B	rs4487073	TT
/	rs35548982	INS(T)INS(T)

GENE	SNP	GENOTYPE
ADAM7	rs17052317	AA
TPSD1	rs11862795	TT
DAOA	rs202209255	TT
B3GALT2	rs61815623	GG
SULF2	rs114241858	GG
TBC1D7	rs192183165	CC
TASOR	rs7627580	TT
WWOX	rs8047442	TT
PODXL	rs11765910	CC
/	rs142971575	CDEL(TTAAT)

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

Aortic Aneurysm

Several factors can increase the risk of developing an aortic aneurysm, including:

- Aging: The risk increases with age, particularly after 65.
- Tobacco use: Smoking is a significant risk factor, dramatically increasing the chances of developing an aneurysm.
- High blood pressure: Elevated blood pressure can damage the aortic wall, leading to an aneurysm.
- Genetic factors: A family history of aortic aneurysm increases the risk.
- Atherosclerosis: The build-up of plaques in the artery walls can weaken them and contribute to aneurysm formation.
- Gender: Men are more likely to develop aortic aneurysms than women.

Treatment depends on the size and rate of growth of the aneurysm:

- Small, slow-growing aneurysms: These may be monitored regularly without immediate surgery, with efforts focused on controlling blood pressure and reducing other risk factors.
- Large or fast-growing aneurysms: Surgical repair may be required. This can involve replacing the affected section of the aorta with a synthetic tube (graft), which is done through open surgery or a less invasive procedure called endovascular surgery.

Preventive strategies involve regular medical check-ups, especially for those with risk factors, managing blood pressure, quitting smoking, and maintaining a healthy lifestyle to minimize the chances of an aneurysm developing or worsening.



TYPICAL LIKELIHOOD

Typical likelihood of aortic aneurysm based on 960,609 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CDKN2B	rs7866503	TT
RBBP8	rs8087799	AA
/	rs143235478	GG
CDKN2A	rs2891168	GG
SORT1	rs12740374	GG
IL6R	rs12133641	AA
FGF9	rs12869493	CC
DAB2IP	rs5900493	TT
CTAGE1	rs34316398	DEL(C)G
DAB2IP	rs10985349	TC
SMYD2	rs1795061	TC
ERG	rs2836411	CT
LDAH	rs13382862	AG
FBN1	rs595244	CC
ANKRD44	rs919433	GG
LPA	rs10455872	AA
PCSK7	rs662799	AA
CHRNA5	rs58365910	TT
SMYD2	rs1660371	AA
LDLR	rs6511720	GG
CDKN2A	rs10757274	GG
PLTP	rs58749629	GG
FGF9	rs9316871	AA
SORT1	rs602633	GG
MCM8	rs6516091	GG
STAT6	rs1385526	GG

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

Mitral Valve Prolapse

Risk factors for mitral valve prolapse include [R]:

- Being female
- Connective tissue disorders, like Marfan syndrome or Ehlers-Danlos syndrome
- Scoliosis or other skeletal problems

MVP can sometimes run in families, suggesting a genetic component to the condition. It is also associated with genetic connective tissue disorders like Marfan syndrome, which is caused by mutations in the *FBN1* gene.

Most people with MVP don't need treatment unless the condition is severe or causing significant symptoms. Management of the condition often includes [R]:

- Regular monitoring, including echocardiograms to assess the mitral valve's condition.
- Medications such as beta-blockers for palpitations or blood thinners to reduce the risk of blood clots.
- In severe cases of mitral regurgitation, surgery to repair or replace the mitral valve may be necessary.

There's no certain way to prevent MVP. However, managing risk factors and keeping regular appointments with a healthcare provider for echocardiograms can help monitor the condition and its progression. Maintaining a healthy lifestyle, including regular exercise and a balanced diet, can also support overall heart health.



LESS LIKELY

Less likely to have mitral valve prolapse based on 1,668 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
KDM5B	rs199723025	AA
MSRA	rs56028519	AA
LTBP2	rs888414	GG
/	rs57355895	CC
TBX5	rs1946299	GG
CCN2	rs11962845	AA
SPTBN1	rs12713274	AA
TBX5	rs2555005	AA
LTBP2	rs11852134	AA
SRR	rs216205	CT
PINX1	rs12676417	AG
SIPA1L1	rs17767392	CT
GLIS1	rs1879734	TC
MEOX1	rs55674920	GA
BAG3	rs17099139	GC
ERCC4	rs13334552	AG
PDCD4	rs12573386	GC
CAND2	rs34871776	CT
SH2B3	rs10774625	AG
TGFB2	rs12406058	GG
LMCD1	rs171408	AA
TNP1	rs7595393	CC
SRR	rs112258894	CC
TNP1	rs12465515	GG
DMWD	rs4802272	GG
TNP1	rs34909633	GG
MN1	rs11705555	AA
SETD4	rs62229266	AA
NMB	rs35828350	GG

GENE	SNP	GENOTYPE
LMCD1	rs165177	CC
TNP1	rs13399995	TT

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

Hypertrophic Cardiomyopathy

Hypertrophic cardiomyopathy is often inherited. Approximately 60-70% of individuals with HCM have a family history of the condition. These genetic mutations affect the proteins in the heart muscle, leading to abnormal thickening. More than 1,500 mutations in at least 11 different genes have been associated with HCM, most of which are related to the sarcomere, the contractile unit of the heart muscle.

While genetics play a crucial role, other factors can contribute to the development and progression of HCM. High blood pressure, aging, and other cardiovascular conditions can exacerbate the thickening of the heart muscle. Additionally, lifestyle factors such as excessive alcohol consumption, lack of exercise, and poor diet can increase the risk of developing HCM or worsen existing symptoms. It is important for individuals with a family history of HCM to undergo regular medical check-ups and genetic counseling to assess their risk and take preventive measures.

Understanding the causes and risk factors of HCM is essential for effective management and prevention. By recognizing the genetic components and other contributing factors, individuals can take proactive steps to monitor their heart health, seek timely medical advice, and adopt lifestyle changes to mitigate the impact of this condition. Early diagnosis and personalized treatment plans are key to improving the quality of life for those affected by hypertrophic cardiomyopathy.



LESS LIKELY

Less likely to have hypertrophic cardiomyopathy based on 19,408 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
FLNC	rs2291569	GG
CLCNKA	rs10927875	CC
TTN	rs2042995	TT
CDKN1A	rs4713999	GG
BAG3	rs2234962	CT
MTSS1	rs12541595	GT
SVIL	rs2182400	GA
XPC	rs6807275	AG
CCDC149	rs2244757	CC
DNAH17	rs78212518	CC
TBX5	rs149932627	TT
CCND1	rs145355401	TT
RALY	rs140035275	CC
OR2F2	rs73462438	AA
RAG2	rs77698332	AA
FAM120B	rs149215314	GG
THSD7A	rs74676849	AA
PARD3B	rs4487073	TT
SMARCB1	rs2186370	AA
NMB	rs1051168	GG
FDFT1	rs13265989	AA
PRKCA	rs9892651	TT
FHOD3	rs2303510	AA

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

Chest Pain (Angina)

Factors that might increase the risk of developing angina or coronary artery disease include:

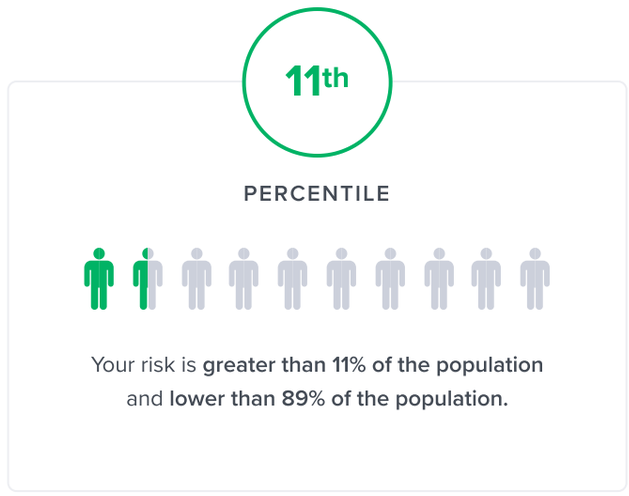
- Age: Men over 45 and women over 55 are at increased risk.
- Tobacco use.
- Diabetes.
- High blood pressure.
- High cholesterol levels.
- Family history of heart disease.
- Obesity or being overweight.
- Sedentary lifestyle.
- Unhealthy diet.
- Chronic stress or short episodes of severe stress.
- Genetics

There is a genetic component to the susceptibility to coronary artery disease and, consequently, angina. Specific genetic mutations and familial patterns have been identified that increase the risk of developing heart disease. Families with a history of early heart disease or angina may carry a higher risk due to shared genetic and environmental factors.



LESS LIKELY

Less likely to have angina based on 709,266 genetic variants we looked at

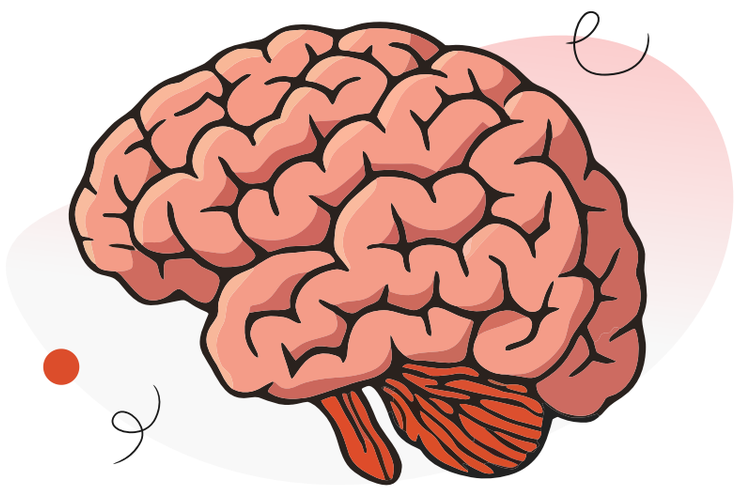


Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CDKN2B	rs1537371	AA
SORT1	rs646776	TT
PRRT1	rs3130283	CC
SRR	rs4790881	AA
APOE	rs429358	CT
PHACTR1	rs9349379	AG
TAF1A	rs1909196	TC
MAP3K11	rs11227229	AG
CTAGE1	rs1893250	AC
/	rs7873013	GT
CFDP1	rs4146810	GA
PHACTR2	rs191867719	CC
/	rs112735431	GG
/	rs191650849	TT
LPA	rs10455872	AA
/	rs13306206	GG
NAA25	rs11066132	CC
MRPS6	rs28451064	GG
BET1L	rs73392700	GG
TWIST1	rs2107595	GG
TTC32	rs16986953	GG

GENE	SNP	GENOTYPE
CCDC71L	rs12705390	GG
ZNF32	rs1870635	CC
FGF5	rs36034102	GG
STOML1	rs11072452	GG
DHX38	rs12325142	GG

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



Brain Vessels

Your brain relies on a steady supply of oxygen and nutrients, all delivered through an intricate network of blood vessels. When something disrupts this system—whether through blockages, weakened vessel walls, or bleeding—the consequences can be severe. While lifestyle factors play a role, genetics also influences your risk of vascular issues in the brain.

This section explores your genetic predisposition to conditions like **stroke, brain aneurysm, or cerebral small vessel disease**. Understanding these risks can help you take proactive steps to support brain vessel health and reduce potential complications.

TYPICAL LIKELIHOOD

Stroke

Typical likelihood of stroke

TYPICAL LIKELIHOOD

Cerebral Small Vessel Disease

Typical likelihood of cerebral small vessel disease

TYPICAL LIKELIHOOD

Brain Ischemia

Typical likelihood of chronic cerebral ischemia

LESS LIKELY

Brain Aneurysm

Less likely to have a brain aneurysm

LESS LIKELY

Subarachnoid Hemorrhage

Less likely to have subarachnoid hemorrhage

LESS LIKELY

Brain Hemorrhage

Less likely to have a brain hemorrhage

Stroke

Key Takeaways:

- A stroke is a serious emergency condition that requires immediate medical care.
- The most common symptom is sudden weakness or numbness on one side of the body.
- Up to **40%** of differences in people's stroke rates may be due to genetics.
- Obesity, smoking, alcohol, and a lack of physical activity are among risk factors for stroke.

Up to **40%** of differences in people's stroke rates may be due to genetics [R].
Genetically predicted high levels of the following markers may be causally associated with a higher risk of stroke [R, R, R, R, R]:

- Fasting insulin
- Apolipoprotein B
- Neutrophils
- Testosterone

In contrast, genetically high alpha-linolenic acid levels may be causally associated with a lower risk [R, R].
Other risk factors for stroke include:

- Age: the risk of stroke increases with age.
- Sex: men have a slightly higher risk of stroke than women.
- Race: African Americans have a higher risk of stroke than Caucasians.
- Personal or family history
- Chronic stress
- Smoking
- A lack of physical activity
- Being overweight or obese
- Excessive alcohol consumption
- Using recreational drugs, such as cocaine and amphetamines

Health conditions that may contribute to stroke include:

- High blood pressure
- Diabetes
- High cholesterol
- Heart disease



TYPICAL LIKELIHOOD

Typical likelihood of stroke based on 1,030,648 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SPSB4	rs16851055	GG
ABCG5	rs76866386	TT
ALDH2	rs2238151	TC
FN1	rs17517928	CC
FGD5	rs748431	GG
FLT1	rs1924981	TT
IL6R	rs11265613	TT
SWAP70	rs10840293	AA
COL4A1	rs9521634	CC
PTPRF	rs6695915	GA
LIPA	rs1412444	CT
SH2B3	rs10774625	AG
HTRA1	rs60401382	CT
MMP8	rs12792912	TG
ASB3	rs13407662	CC
TWIST1	rs2107595	GG
PITX2	rs6843082	AA
LPA	rs55730499	CC
HP	rs879324	GG
ABO	rs532436	GG
EDNRA	rs1878406	CC

GENE	SNP	GENOTYPE
FES	rs8027450	CC
DACH1	rs339800	CC
MPO	rs2632512	CC
JCAD	rs2487928	GG
NEDD4	rs12442374	CC

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

Cerebral Small Vessel Disease

The symptoms of cerebral small vessel disease can be subtle and may include cognitive decline, difficulty with walking and balance, mood changes, and in some cases, stroke-like events known as lacunar infarcts. Over time, SVD can lead to a more noticeable impact on cognitive function and mobility, contributing to vascular dementia and increasing the risk of falls.

The disease is progressive and although there is no cure, management focuses on controlling the risk factors through lifestyle changes and medication to lower blood pressure, manage diabetes, and reduce cholesterol levels. The aim of treatment is to prevent further vascular damage and to alleviate symptoms when possible.



TYPICAL LIKELIHOOD

Typical likelihood of cerebral small vessel disease based on 2 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
/	rs117338591	GG
CAAP1	rs77691192	AA

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

Brain Ischemia

The underlying causes of this condition are often systemic and can include atherosclerosis, where plaque buildup narrows arteries, chronic hypertension, or other vascular diseases that compromise the integrity of blood vessels. The diminished blood supply can result in cognitive impairment, memory problems, and difficulties in executive functions, reflecting the widespread impact of chronic ischemia on brain health.

Diagnosis usually involves imaging techniques such as MRI or CT scans which can reveal areas of reduced blood flow and potential brain injury. Treatment aims to address the cause of the reduced blood flow, often through medication, lifestyle changes, and, in some cases, surgical interventions to improve cerebral circulation and prevent further damage.



TYPICAL LIKELIHOOD

Typical likelihood of chronic cerebral ischemia based on 364,751 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SPSB4	rs16851055	GG
ABCG5	rs76866386	TT
ALDH2	rs2238151	TC
FN1	rs17517928	CC
FGD5	rs748431	GG
FLT1	rs1924981	TT
IL6R	rs11265613	TT
SWAP70	rs10840293	AA
COL4A1	rs9521634	CC
PTPRF	rs6695915	GA
LIPA	rs1412444	CT
SH2B3	rs10774625	AG
HTRA1	rs60401382	CT
MMP8	rs12792912	TG
ASB3	rs13407662	CC
TWIST1	rs2107595	GG
PITX2	rs6843082	AA
LPA	rs55730499	CC
HP	rs879324	GG
ABO	rs532436	GG
EDNRA	rs1878406	CC
FES	rs8027450	CC
DACH1	rs339800	CC
MPO	rs2632512	CC
JCAD	rs2487928	GG
NEDD4	rs12442374	CC

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

Brain Aneurysm

Risk factors for brain aneurysm typically contribute to weakness in an artery wall and high blood pressure. They include:

- Age between 30 and 60
- Being female
- Cigarette smoking
- Drug use, particularly cocaine
- Heavy alcohol use
- Inherited connective tissue disorders, such as Ehlers-Danlos syndrome
- Polycystic kidney disease
- A narrowing (coarctation) of the aorta
- Brain arteriovenous malformation
- Family history of brain aneurysm

Repairing a ruptured aneurysm requires surgery or endovascular treatment. You may also be given treatments to relieve the symptoms. If you have an unruptured aneurysm, talk with your healthcare provider about possible treatments. Discuss whether the risk of leaving the aneurysm alone is greater than the risk of treating it [R].



LESS LIKELY

Less likely to have a brain aneurysm based on 21,086 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
EDNRA	rs6841581	GG
NT5C2	rs79780963	CC
CDKN2A	rs1537373	GG
RBBP8	rs11661542	CC
PDE3A	rs11044991	GG
SLC22A5	rs4705938	TT
POC1B	rs2681472	AA
CFDP1	rs7184525	GA
FGD6	rs7137731	TC
SLC24A3	rs4814863	AG
NOC3L	rs11187838	GA
BET1L	rs2280543	CC
MTMR3	rs39713	CC
RP1	rs62516550	CC
UFL1	rs11153071	GG
STARD13	rs3742321	TT
IREB2	rs8034191	TT

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

Subarachnoid Hemorrhage

The clinical management of subarachnoid hemorrhage demands immediate medical attention and often necessitates neurosurgical intervention to stop the bleeding and repair the blood vessel. Diagnosing SAH typically involves a CT scan of the head to detect the presence of blood in the subarachnoid space, which may be followed by a lumbar puncture if the CT scan is inconclusive.

Prompt diagnosis and treatment are critical to improving outcomes, with interventions potentially including surgical clipping or endovascular coiling to secure the aneurysm. Post-treatment care is equally vital and focuses on preventing complications such as secondary strokes, brain swelling, and hydrocephalus, among other potential challenges in the recovery process.



LESS LIKELY

Less likely to have subarachnoid hemorrhage based on 17,939 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CDKN2A	rs10738606	TT
CDKN2B	rs4977575	GG

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

Brain Hemorrhage

Factors contributing to brain hemorrhage can include uncontrolled high blood pressure, aneurysms, blood vessel abnormalities, or blood or bleeding disorders. Symptoms can be sudden and severe, often including a sudden severe headache, loss of consciousness, paralysis or weakness on one side of the body, difficulty speaking or understanding language, and vision problems.

The diagnosis often involves neuroimaging tests such as CT scans or MRIs to locate the source of the bleeding. Treatment may involve surgical intervention to relieve pressure on the brain and repair damaged blood vessels, alongside medications to control blood pressure and prevent seizures.



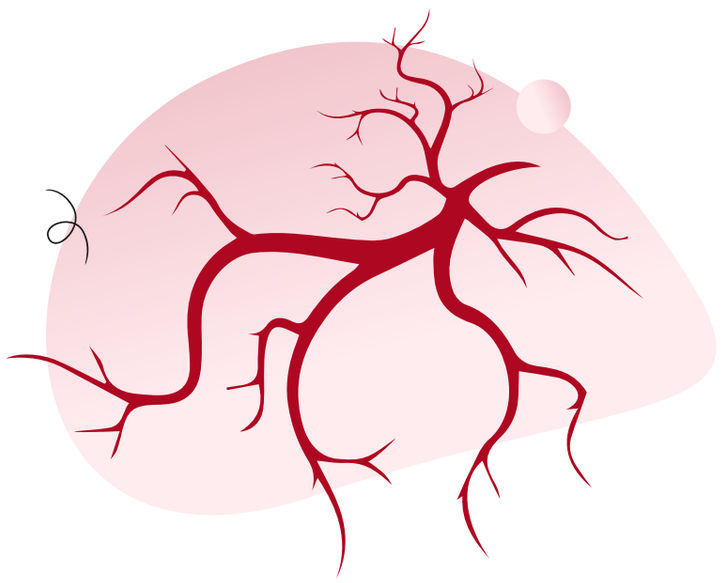
LESS LIKELY

Less likely to have a brain hemorrhage based on 16,196 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CUX2	rs12229654	TT
DPH1	rs34290270	TT
OR52E4	rs11823828	GT
FAM160A1	rs2709828	CT
SEMA4A	rs2984613	TC
FAM117B	rs72932727	GG
COL4A2	rs9515201	AA
SEMA4A	rs2758605	CG
ADGRG1	rs200646658	AA
DAB1	rs116161367	CC

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



Veins & Other Vessels

Your blood vessels, including veins, arteries, and capillaries, are essential for maintaining a healthy circulation and overall bodily function. While arteries tend to grab the spotlight, veins also face their own set of challenges, from weakened walls to blockages. Genetics can influence your susceptibility to various vascular issues, particularly those affecting blood flow and clotting.

This section explores your genetic predispositions to conditions like **varicose veins, venous thromboembolism, or peripheral vascular disease**. Understanding these risks can help you manage circulation health and take preventive steps for better long-term wellness.

MORE LIKELY

Varicose Veins

More likely to have varicose veins

TYPICAL LIKELIHOOD

Venous Thromboembolism

Typical likelihood of venous thromboembolism

TYPICAL LIKELIHOOD

Deep Vein Thrombosis

Typical likelihood of getting DVT

TYPICAL LIKELIHOOD

Peripheral Artery Disease

Typical likelihood of peripheral artery disease

TYPICAL LIKELIHOOD

Blood Spots

Typical likelihood of purpura

TYPICAL LIKELIHOOD

Cold Hands and Feet (Raynaud's)

Typical likelihood of having Raynaud's

TYPICAL LIKELIHOOD

Blood Clotting

Typical likelihood of having thrombosis

LESS LIKELY

Chronic Venous Insufficiency

Less likely to have chronic venous insufficiency

LESS LIKELY

Peripheral Vascular Disease

Less likely to have peripheral vascular disease

LESS LIKELY

Vasculitis

Less likely to have vasculitis

LESS LIKELY

Retinal Vein Occlusion

Less likely to have retinal vein occlusion

Varicose Veins

Key Takeaways:

- Up to **30%** of differences in people's chances of developing varicose veins may be due to genetics. Up to **1 in 3** people over the age of 70 may have varicose veins.
- Risk factors include: age, being female, overweight, sitting or standing most of the day, pregnancy, and childbirth.
- If you have a high genetic risk, you may reduce overall risk by taking action on risk factors that you can change.
- Click the **next steps** tab for relevant labs and lifestyle factors.

Normal, healthy veins have many valves that keep the blood flowing in the right direction. Over time or as a result of physical stress, these valves can become damaged. Damaged valves allow the blood to flow backwards or pool. This is called **venous reflux**, or **chronic venous insufficiency** [R, R, R].

Chronic venous insufficiency is a major cause of **varicose veins**. These are swollen veins that lie just under the skin. They may twist or bulge and appear purple or blue in color. They usually develop in the legs but can affect other parts of the body as well [R, R, R].

Chronic venous insufficiency may also cause [R, R]:

- Swelling, due to a buildup of fluid in the limbs (*edema*)
- Open wounds on the skin (*venous ulcers*)
- Itchy skin
- Pain

Chronic vein problems are more common in older people. In fact, up to **1 in 3 people** over the age of 70 may have varicose veins [R, R].

Besides age, risk factors for chronic vein problems include [R, R, R]:

- Female sex
- Overweight or obesity
- Pregnancy and childbirth
- Sitting or standing for most of the day
- **Genetics**

Ways to manage chronic vein problems include [R, R, R, R]:

- Keeping the legs elevated
- Lifestyle adjustments (e.g., avoiding sitting or standing for long periods, managing weight)
- Compression therapy (using stockings or bandages)
- Laser-based or injectable treatments
- Surgery

The risk of developing chronic vein problems can depend on genes. For example, up to 30% of differences in people's chances of developing varicose veins may be attributed to genetics. Genes involved in varicose veins may influence [R]:

- Blood vessel function (*CASZ1*, *STIM2*)
- Blood flow (*CASZ1*, *PIEZO1*, *EBF1*)
- Immune response (*PPP3R1*, *GATA2*, *NFATC2*)
- Blood iron levels (*HFE*)



MORE LIKELY

More likely to have varicose veins based on 81,137 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
FKBP10	rs550216862	GG
NELFCD	rs76602912	TT
ARID5B	rs3740357	TT
/	rs4561414	GG
NFATC2	rs12625547	TT
SRPX	rs35318931	GG
UBE2H	rs17559301	TT
C20ORF204	rs6062618	TT
SOX7	rs75731123	AA
HLA-G	rs2524005	GG
UNC5B	rs7086901	CC
SLC12A2	rs6860245	GC
DAOA	rs1549061	CC
TMEM87B	rs4849007	AA
PARK7	rs7524424	TT
WDR92	rs7569914	AG
EDN1	rs111797764	CT
HDAC7	rs56389811	TC
SLC38A2	rs4768737	TT
TNFSF8	rs10817784	GA
ATF1	rs10783387	TC

GENE	SNP	GENOTYPE
RREB1	rs675209	TC
CPNE3	rs62512472	AG
SRL	rs4786476	AG
SBF2	rs1372809	CG
NEK8	rs199933041	GG
RAB7A	rs13325550	GG
ITGA6	rs10169716	TT
CDC42SE2	rs2189759	GG
DOCK8	rs78216177	GG
ABO	rs635634	CC
CLDN11	rs496104	GG
ADAM15	rs11589479	GG
SVBP	rs58062906	CC
NFIL3	rs6479353	GG
SGK1	rs228433	GG
NDP	rs5906307	AA
PRKAR1B	rs9719461	CC

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

Venous Thromboembolism

VTE is influenced by several risk factors and underlying conditions [R, R]:

- Immobility: Prolonged sitting or bed rest can lead to blood pooling in the legs, increasing the risk of clot formation.
- Surgery: Especially orthopedic or major surgeries such as hip or knee replacements, which can affect blood flow.
- Trauma: Injuries that affect the veins can lead to clot formation.
- Cancer: Certain cancers and chemotherapy treatments increase the risk of VTE.
- Pregnancy: The risk of VTE increases during pregnancy due to increased pressure in the veins of the pelvis and legs and changes in blood clotting factors.
- Birth control pills or hormone replacement therapy (HRT): These can increase the likelihood of clotting.
- Obesity: Excess weight increases pressure on the veins in the pelvis and legs.
- Smoking: Contributes to blood clot formation and reduced blood flow.
- Genetic factors: Inherited blood clotting disorders can significantly increase the risk.

The primary goals for treating VTE are to stop the clot from getting bigger, prevent the clot from breaking loose and causing a PE, and reduce the chances of another VTE. Treatment usually involves [R, R]:

- Anticoagulants (blood thinners): These medications are the main treatment for VTE. They can prevent new clots from forming and stop existing clots from growing.
- Thrombolytics (clot busters): Used in life-threatening situations to quickly dissolve a large clot.
- Compression stockings: Reduce the swelling associated with DVT and help prevent post-thrombotic syndrome.
- Filters: In some cases, particularly where anticoagulants are not suitable, a filter may be inserted into the inferior vena cava to catch clots before they reach the lungs.



TYPICAL LIKELIHOOD

Typical likelihood of venous thromboembolism based on 1,049,243 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ITGB3	rs5918	CT
ABO	rs657152	AA
KIF26B	rs1756912	GG
GP6	rs1671152	GT
GP6	rs1613662	AG
CYP4V2	rs2289252	CT
HLA-DQA2	rs17202393	GG
NRG3	rs1649936	CC
NCAM2	rs62207434	CC
F5	rs4524	TC
MCMBP	rs12767946	TC
CSGALNACT1	rs62496681	CC
CYP2C19	rs4244285	AG
KNG1	rs710446	CT
/	rs72755680	AC
PSG8	rs59559305	GA
F5	rs6025	CC
F2	rs1799963	GG
PROCR	rs867186	AA
ENPP1	rs72983636	AA
RNASEL	rs55897462	TT

GENE	SNP	GENOTYPE
TAS2R5	rs145241704	TT
ABCD3	rs3917643	TT
POLE4	rs74965230	TT
FAM174A	rs115887893	CC
NME7	rs16861990	AA
SELL	rs1018827	GG
/	rs687289	AA
PLRG1	rs6825454	TT

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

Deep Vein Thrombosis

Key Takeaways:

- About 60% of the differences in people's chances of getting DVT may be due to genetics.
- Other risk factors include age (60+), sedentary lifestyle, injury or surgery involving a deep vein, pregnancy, birth control pills, hormone replacement therapy, being overweight, and smoking.
- If you have a high genetic risk, you can lower overall risk by taking action on risk factors that you can change.
- As many as 900,000 people are likely affected by DVT each year in the United States.
- Click the **Recommendations** tab for potential dietary and lifestyle changes, and **next steps** for relevant labs.

About **60%** of the differences in people's chances of getting DVT may be due to **genetics.** Unsurprisingly, involved genes play a role in blood clotting [R].

Genetically high levels of EPA may be causally associated with a high risk of venous thromboembolism. In contrast, genetically high alpha-linolenic acid levels may be causally associated with a lower risk of DVT [R, R].

Besides genetics, other risk factors for DVT include [R]:

- Age (over 60)
- Lack of movement (due to lifestyle, long travels, surgery, illness, etc.)
- Injury or surgery involving a deep vein
- Pregnancy
- Birth control pills and hormone replacement therapy
- Being overweight or obese
- Smoking

The following health conditions may contribute to DVT [R]:

- Cancer
- Heart failure
- Inflammatory bowel disease
- Genetic conditions that affect blood clotting



TYPICAL LIKELIHOOD

Typical likelihood of getting DVT based on 1,671 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
/	rs687289	AA
CAPN9	rs145470028	GG
TSPAN15	rs78707713	TT
CYP4V2	rs4253421	AG
SLC44A2	rs4548995	CC
ABO	rs8176749	CT
CYP4V2	rs2289252	CT
ZFPM2	rs4541868	CC
F11	rs2036914	TC
F5	rs4524	TC
GRK5	rs10886430	GA
PLCG2	rs12445050	CT
F5	rs6025	CC
LRP4	rs191945075	GG
ABO	rs2519093	CC
ABO	rs579459	TT
PLRG1	rs2066864	GG
C4BPA	rs2842700	CC
PROCR	rs867186	AA

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

Peripheral Artery Disease

Patients with peripheral artery disease often have other co-occurring conditions such as diabetes, obesity, or high blood pressure, which contribute to the progression of PAD. The risk of peripheral artery disease increases with age, especially after 50 years. Smokers or those with a history of smoking have a higher risk of developing PAD.

The complications of PAD can be serious, including an increased risk of coronary heart disease, heart attack, stroke, and in extreme cases, leading to limb amputation. Lifestyle changes, such as exercising regularly, quitting smoking, and maintaining a healthy diet, along with medications, can help manage PAD symptoms and slow the progression of the disease.



TYPICAL LIKELIHOOD

Typical likelihood of peripheral artery disease based on 747,296 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HLA-DQA1	rs9271393	CC
CDKN2B	rs10757271	AG
CDKN2B	rs1537372	TT
SMARCA4	rs138294113	CC
SORT1	rs7528419	AA
ABO	rs505922	CC
SMOC1	rs55784307	AA
COL4A1	rs1975514	CC
STEAP1B	rs4722172	AG
BPTF	rs62084752	CG
LPL	rs322	CA
PHF21A	rs7476	AC
PAWR	rs4842266	AG
ALDH2	rs11066301	GA
LPA	rs10455872	AA
TRIM10	rs3132625	AA
TWIST1	rs57301765	GG
LPA	rs118039278	GG
F5	rs6025	CC
MMP3	rs566125	CC
TWIST1	rs2107595	GG
MICB	rs3130968	CC
TCF7L2	rs7903146	CC
IREB2	rs10851907	GG

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

Blood Spots

There are various types of purpura, each with different implications and causes. Thrombocytopenic purpura is associated with a low platelet count, which can result from autoimmune diseases, bone marrow disorders, or certain medications.

Non-thrombocytopenic purpuras occur despite a normal platelet count and may be due to blood vessel inflammation (vasculitis), aging skin, certain infections, or a response to medications. It's essential to diagnose the cause accurately, as treatment strategies are diverse, ranging from observation and addressing the underlying issue to more aggressive interventions when significant bleeding is involved.



TYPICAL LIKELIHOOD

Typical likelihood of purpura based on 873,823 genetic variants we looked at

39th

PERCENTILE



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

Cold Hands And Feet (Raynaud's)

Key Takeaways:

- About **55-65%** of the differences in developing Raynaud's may be due to genetics.
- Other risk factors include smoking, injury, certain medicines or chemical exposure, and prolonged repetitive motions.
- Raynaud's affects 3-5% people worldwide, so even if your genetic risk is high, the overall risk is relatively low.
- Click the **Recommendations** tab for potential dietary and lifestyle changes.

Raynaud's is a rare disorder of the blood vessels. It causes the blood vessels to narrow when you are cold or feeling stressed, and usually affects the fingers and toes.

About **55-65%** of the differences in developing Raynaud's may be due to genetics [R].

Risk factors for secondary Raynaud's include [R]:

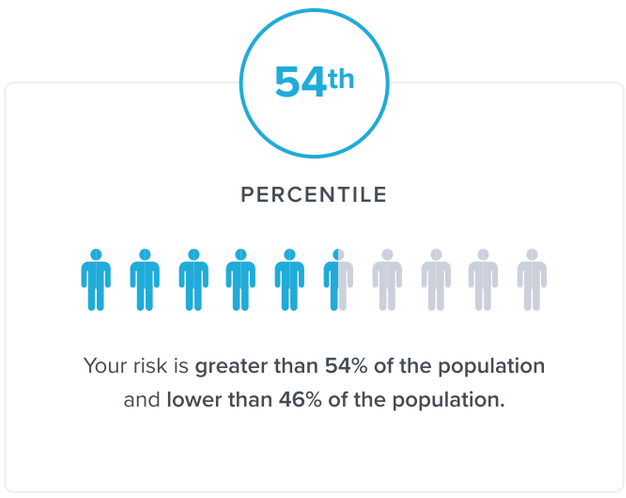
- Autoimmune diseases (scleroderma, lupus, rheumatoid arthritis)
- Issues with blood vessels
- Exposure to certain medicines or chemicals
- Smoking
- Age (over 30)
- Injury or trauma
- Prolonged repetitive motions

If you experience symptoms, talk to your healthcare professional about possible actions to take.



TYPICAL LIKELIHOOD

Typical likelihood of having Raynaud's based on 996,273 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
KIAA1217	rs7069430	AA
/	rs140991804	CC
/	rs570208750	GG
NEBL	rs190575139	CC
FAM171A1	rs143100064	CC
PRPF18	rs57572383	AA
/	rs140673014	AA
MALRD1	rs146119830	CC
SKIDA1	rs141961273	TT
PRKCQ	rs112942698	GG
KIAA1217	rs182841690	AA
KIAA1217	rs146561629	GG
KLF6	rs185109474	AA
SFMBT2	rs78967739	TT
KIAA1217	rs145536431	GG
TUBAL3	rs143549736	CC
PLXDC2	rs112408375	CC
MCM10	rs117278045	CC
ADARB2	rs117804762	GG
PRPF18	rs141694090	AA
CELF2	rs147411784	GG

GENE	SNP	GENOTYPE
NSUN6	rs80162025	AA
PLXDC2	rs11599807	TT
CCDC3	rs138336556	CC
NSUN6	rs79253631	AA
ADARB2	rs142207383	CC
SPAG6	rs1170547	GG
PFKP	rs77345563	CC
/	rs56372212	AA
STAM	rs11254703	GG
COMMD3-BMI1	rs7099367	CC
AKR1E2	rs114725652	GG
/	rs117036760	AA
PIP4K2A	rs11013083	AA
AKR1E2	rs143293957	CC
ADARB2	rs112448126	GG
KIAA1217	rs183347830	GG
CELF2	rs144272580	TT
CALML5	rs117143307	GG
ITGA8	rs140551139	CC
MALRD1	rs183014160	TT
ADARB2	rs72772247	TT
CAMK1D	rs139224231	AA
MALRD1	rs192852937	CC
ITGA8	rs145054943	AA
PRKCQ	rs117533148	TT
/	rs369714188	CC
MALRD1	rs150972735	GG
PLXDC2	rs77930811	AA
USP6NL	rs180921710	CC

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

Blood Clotting

About **50%** of the differences in thrombosis may be due to **genetics** [R].

Other factors that might influence the risk of thrombosis include:

- Prolonged immobility (e.g., bed rest, long flights)
- Surgery, especially joint replacement surgery
- Injuries, especially those involving fractures or muscle damage
- Certain medications(e.g., oral contraceptives, hormone replacement therapy, chemotherapy drugs)
- Smoking
- Age, especially being over 60
- Pregnancy

Some health conditions may increase the risk of thrombosis, including:

- Obesity
- Chronic inflammatory diseases (e.g., lupus, inflammatory bowel disease)
- Blood clotting disorders
- Cancer



TYPICAL LIKELIHOOD

Typical likelihood of having thrombosis based on 1,669 genetic variants we looked at

46th

PERCENTILE



Your risk is **greater than 46% of the population** and **lower than 54% of the population.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ITGB3	rs5918	CT
ABO	rs657152	AA
KIF26B	rs1756912	GG
GP6	rs1671152	GT
GP6	rs1613662	AG
CYP4V2	rs2289252	CT
HLA-DQA2	rs17202393	GG
NRG3	rs1649936	CC
NCAM2	rs62207434	CC
F5	rs4524	TC
MCMBP	rs12767946	TC
CSGALNACT1	rs62496681	CC
/	rs72755680	AC
PSG8	rs59559305	GA
F5	rs6025	CC
F2	rs1799963	GG
PROCR	rs867186	AA
ENPP1	rs72983636	AA
RNASEL	rs55897462	TT
TAS2R5	rs145241704	TT
POLE4	rs74965230	TT

GENE	SNP	GENOTYPE
FAM174A	rs115887893	CC
NME7	rs16861990	AA
SELL	rs1018827	GG
/	rs687289	AA
PLRG1	rs6825454	TT

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

Chronic Venous Insufficiency

Factors that can increase the likelihood of developing CVI include:

- Older age
- Female sex
- Obesity
- Pregnancy
- Family history
- Physical inactivity

Treatment aims to improve blood flow in the leg veins and manage symptoms. It can include:

- Compression therapy: Wearing compression stockings is often the first approach to help veins move blood efficiently.
- Medication: Diuretics may be used to reduce swelling, but they should be used cautiously as they can also lower blood volume and make venous pressure worse.
- Exercise: Regular physical activity, especially walking, can help pump blood up the legs.
- Elevating the legs: Raising the legs above the heart several times a day can decrease swelling and improve circulation.
- Sclerotherapy and surgery: For severe cases or cosmetic reasons, procedures to close off or remove damaged veins may be considered.



LESS LIKELY

Less likely to have chronic venous insufficiency based on 8,325 genetic variants we looked at

37th

PERCENTILE



Your risk is **greater than 37% of the population** and **lower than 63% of the population.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
KCNH8	rs727139	AA
HOXA2	rs2030136	CT
EFEMP1	rs17278665	CC

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

Peripheral Vascular Disease

Factors that can increase your risk of developing PVD include:

- Age, especially after 50.
- Smoking
- Diabetes
- Obesity
- High blood pressure
- High cholesterol
- Physical inactivity
- Family history

Treatment for PVD involves lifestyle changes, medications, and sometimes surgery. Key aspects include:

- Smoking cessation
- Exercise
- Healthy diet
- Medications (cholesterol-lowering drugs, high blood pressure medications, medications to control blood sugar, and medications to prevent blood clots)
- Compression therapy
- Surgical treatments: angioplasty or surgery to bypass or open blocked arteries, or correct valve dysfunctions.



LESS LIKELY

Less likely to have peripheral vascular disease based on 791,959 genetic variants we looked at

57th

PERCENTILE



Your risk is **greater than 57% of the population** and **lower than 43% of the population.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CDKN2B	rs1537372	TT
SMARCA4	rs138294113	CC
SORT1	rs7528419	AA
ABO	rs505922	CC
SMOC1	rs55784307	AA
COL4A1	rs1975514	CC
STEAP1B	rs4722172	AG
BPTF	rs62084752	CG
ALDH2	rs11066301	GA
PAWR	rs4842266	AG
LPL	rs322	CA
PHF21A	rs7476	AC
LPA	rs118039278	GG
F5	rs6025	CC
TWIST1	rs2107595	GG
MICB	rs3130968	CC
MMP3	rs566125	CC
TCF7L2	rs7903146	CC
IREB2	rs10851907	GG

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

Vasculitis

The exact cause of vasculitis is often unknown, but it can result from an autoimmune response where the body's immune system mistakenly attacks the blood vessels. Alternatively, it may follow an infection or another disease. The condition can be acute or chronic and range from mild to life-threatening, depending on the severity and the extent of vessel involvement.

Treatment for vasculitis often involves suppressing the immune system with medications such as corticosteroids and cytotoxic drugs to reduce inflammation. Close monitoring and management of symptoms is essential to prevent complications. In some cases, vasculitis may resolve on its own.



LESS LIKELY

Less likely to have vasculitis based on 252,730 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ETS2	rs2242944	GG
TSLP	rs1837253	TC
CCR3	rs2087726	AA
PTK2B	rs73223431	TC
P4HA2	rs128738	TG
IL12B	rs7725339	GT
PCGF3	rs4690319	GA
OSBP2	rs136301	AA
/	rs9540128	TC
CFL2	rs76457959	CT
RPS9	rs34629529	AG
BCL2L11	rs72836352	CC
CTLA4	rs62184865	GG
PLG	rs4252120	TT
IL23R	rs11209039	GG
PPP2R2B	rs142314071	CC
GADL1	rs74712413	GG
PLCG2	rs12596533	AA
IGFBP4	rs76139923	CC
CAV3	rs115155040	TT
CACNA2D3	rs12492780	AA
CCKAR	rs113569359	GG
APCDD1	rs176237	GG

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

Retinal Vein Occlusion

Treatment for retinal vein occlusion focuses on managing the underlying risk factors and addressing the complications arising from the blockage. Interventions may include intraocular injections of anti-VEGF drugs to reduce swelling and improve vision, laser therapy to seal leaking vessels, or corticosteroids to reduce inflammation.

Long-term management often involves monitoring and controlling conditions that contribute to the risk of RVO, such as hypertension, diabetes, and hyperlipidemia. The prognosis of RVO varies depending on the severity and the individual's response to treatment, but timely medical attention can help preserve vision and prevent more significant loss.



LESS LIKELY

Less likely to have retinal vein occlusion based on 26,525 genetic variants we looked at

13th

PERCENTILE



Your risk is **greater than 13% of the population** and **lower than 87% of the population.**



Heart Rate

Heart rate parameters are various measures related to a person's heart rate. Besides heart rate, which is the number of beats per minute (bpm), the parameters measure how much heart rate varies and how fast it can return to its resting rate.

They help evaluate an individual's **heart health, fitness, health status, and response to things like stress and exercise**. Your genetics affects all heart rate parameters — **dive into this section for the results and what they mean for your health and wellbeing**!

SLOWER

Heart Rate Recovery

Predisposed to lower HRR

TYPICAL

Heart Rate

Predisposed to typical heart rate

TYPICAL LIKELIHOOD

Tachycardia

Typical likelihood of tachycardia

TYPICAL LIKELIHOOD

Atrial Fibrillation

Typical likelihood of atrial fibrillation

TYPICAL LIKELIHOOD

POTS

Typical likelihood of POTS

TYPICAL LIKELIHOOD

Heart Arrhythmia

Typical likelihood of heart arrhythmia

HIGHER

Heart Rate Variability

Predisposed to higher HRV

LESS LIKELY

Palpitations

Less likely to have palpitations

LESS LIKELY

Bradycardia

Less likely to have bradycardia

Heart Rate Recovery

Heart rate recovery (HRR) is the difference between your peak heart rate during exercise and your heart rate soon after you stop. **Normal HRR after one minute of rest is typically 18 bpm or higher**.

About **60%** of the differences in people's HRR may be due to genetics. Many involved genes play a role in nerve function [R, R].

Factors that can affect your ability to recover heart rate after exercise include [R, R, R, R]:

- Fitness level
- Age
- Sleep quality

HRR can also depend on the exercise and resting methods [R].

Talk to your healthcare provider if you have a poor HRR. While it's been linked to certain health problems, it may simply mean you need more physical activity.



SLOWER

Predisposed to lower HRR based on 704,947 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
RGS6	rs17180489	GG
SYT10	rs6488162	TC
SOX5	rs4963772	GA
CAV1	rs1997571	GA
GIGYF1	rs221789	CC
C19ORF12	rs55954001	GG
TGM2	rs6127466	AG
INPPL1	rs7130652	GG
NEGR1	rs61765646	TT
NEGR1	rs12740789	GG
GRIK2	rs2224202	GG
SATB1	rs73043051	TT
SNX2	rs4836027	CC
SERINC2	rs11589125	TC
PAX2	rs10748799	TC
PRDM6	rs151283	AC
/	rs12906962	TC
/	rs13022107	TC
GNG11	rs180253	AA
TBX3	rs61928421	CC
TFPI2	rs180238	TT
ACHE	rs3757868	GG
CHRM2	rs17168815	GG
CAPS	rs8108862	CC
RNF220	rs272564	AA

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

Heart Rate

Your heart is a pump that drives blood through your body at a rate needed to maintain oxygen supply. The healthier and stronger your heart is, the less effort it needs to accomplish this task, and thus the fewer times it has to beat per minute.

There are several risk factors for an abnormal heart rate, including:

- **Heart disease**: conditions such as hypertension, coronary artery disease, and heart valve problems
- **Metabolic disorders**: diabetes, thyroid disease, and other metabolic disorders
- **Medications**: certain medications, such as beta-blockers and anti-arrhythmic drugs
- **Lifestyle factors**: stress, smoking, excessive alcohol consumption, and a sedentary lifestyle
- **Older age**

Up to **30%** of differences in people's heart rate may be due to **genetics**. Involved genes control the production of substances that contract and relax the heart muscle [R].

It's important to consult with a doctor if you have symptoms of abnormal heart rate such as **palpitations, fainting, dizziness, or shortness of breath.**

Genetically higher resting heart rate may be causally associated with:

- High blood sugar [R, R, R, R]
- Atrial fibrillation (lower risk) [R]
- High blood pressure (diastolic) [R]
- Triglycerides [R]
- CRP [R]
- Overweight [R]



TYPICAL

Predisposed to typical heart rate based on 1,026,255 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TRAPPC14	rs140367586	TT
FHOD3	rs61735998	GG
GRINA	rs56233017	GG
HSF2	rs1320761	TC
MICAL2	rs112421686	AA
GJA1	rs3792943	TT
SOX5	rs4963772	GA
FBXL17	rs9326726	AA
SLC35F1	rs3951016	TA
ZHX3	rs17265513	CC
FADS1	rs174536	CA
RASSF3	rs867400	TC
FRMD4B	rs1483890	AG
PPIL1	rs236349	GA
SCN10A	rs6599255	AC
MEIS1	rs62144050	TC
CCDC141	rs10497529	GG
FKBP7	rs151041685	GG
LZIC	rs182770070	AA
MYH6	rs422068	TT
PLN	rs10457327	GG
CD46	rs41317993	GG
ACHE	rs17881696	GG
ARHGEF40	rs12889267	AA
RBBP8	rs117159291	AA
SYT10	rs1994135	TT
CHRM2	rs73158705	AA
RNF220	rs272564	AA
CALCRL	rs62172372	AA

GENE	SNP	GENOTYPE
MYH11	rs3915499	GG
PPARGC1A	rs12501032	CC
MIX23	rs11920570	GG
RBM6	rs3749237	GG
GPATCH2	rs11454451	CC
TP53I11	rs12576326	AA
MFF	rs4608502	TT
MAP3K10	rs16974196	GG
DSP	rs2744375	AA

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

Tachycardia

Key Takeaways:

- Up to **60%** of differences in people's chances of having tachycardia may be due to genetics.
- Other risk factors include: stress, excessive caffeine intake, smoking, high/low blood pressure & electrolyte imbalance.
- If you are at high genetic risk, you may lower the overall risk by taking action on risk factors that you can change.
- Symptoms include palpitations, shortness of breath, chest pain, dizziness, and fainting.
- Click the **Recommendations** tab for potential dietary and lifestyle changes, and **next steps** for relevant labs.

Tachycardia can be caused by various factors, including [R]:

- Stress
- Excessive caffeine intake
- Smoking
- Certain medications
- Heavy alcohol use or alcohol withdrawal
- Use of illegal drugs

Conditions that may contribute to tachycardia include [R]:

- High or low blood pressure
- Electrolyte imbalance
- Heart disease
- Overactive thyroid (hyperthyroidism)
- Anemia

Up to **60%** of differences in people's chances of having tachycardia may be due to genetics [R].



TYPICAL LIKELIHOOD

Typical likelihood of tachycardia based on 186,698 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PITX2	rs143269342	CC
TBX5	rs883079	TT
PBXIP1	rs11264280	TT
GJA5	rs79187193	GG
PITX2	rs3853445	TT
PITX2	rs13105878	CC
PITX2	rs17570669	AA
CLIC6	rs2834618	TT
PITX2	rs2595104	TG
SELL	rs12122060	AT
KCNN3	rs34292822	CG
MYOZ1	rs6480708	CA
MYOZ1	rs60212594	GC
UBE4B	rs187585530	GG
PITX2	rs2129977	GG
PITX2	rs112599895	AA
PITX2	rs75021220	CC
METTL11B	rs72700114	GG
PITX2	rs6847935	AA
PITX2	rs6843082	AA
HP	rs2359171	TT
TXNDC12	rs146518726	GG
NEURL1	rs11598047	AA
C11ORF45	rs76097649	GG
PITX2	rs149829837	TT
SCN5A	rs7373065	CC
RPL3L	rs140185678	GG
SH3PXD2A	rs35176054	TT
FBXO32	rs78332318	CC

GENE	SNP	GENOTYPE
FBXO32	rs62521286	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
PITX2	rs143269342	CC
TBX5	rs883079	TT
PBXIP1	rs11264280	TT
GJA5	rs79187193	GG
PITX2	rs3853445	TT
PITX2	rs13105878	CC
PITX2	rs17570669	AA
CLIC6	rs2834618	TT
PITX2	rs2595104	TG
SELL	rs12122060	AT
KCNN3	rs34292822	CG
MYOZ1	rs6480708	CA
MYOZ1	rs60212594	GC
UBE4B	rs187585530	GG
PITX2	rs2129977	GG
PITX2	rs112599895	AA
PITX2	rs75021220	CC
METTL11B	rs72700114	GG
PITX2	rs6847935	AA
PITX2	rs6843082	AA
HP	rs2359171	TT

GENE	SNP	GENOTYPE
TXNDC12	rs146518726	GG
NEURL1	rs11598047	AA
C11ORF45	rs76097649	GG
PITX2	rs149829837	TT
SCN5A	rs7373065	CC
RPL3L	rs140185678	GG
SH3PXD2A	rs35176054	TT
FBXO32	rs78332318	CC
FBXO32	rs62521286	AA

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

POTS

The exact cause of POTS is not fully understood, but it is thought to involve several factors:

- Viral infections
- Physical deconditioning following an illness or extended period of inactivity
- Blood volume deficiencies and nerve dysfunction in the legs
- **Genetics:** POTS can run in families and certain variants in genes such as *GNB3*, *SLC6A2*, and *NOS3* have been associated with this condition [R, R, R]

There is no cure for POTS, but various treatments and lifestyle adjustments can help manage symptoms [R]:

- **Increased salt and fluid intake** to boost blood volume and improve blood pressure control.
- **Medications** such as beta-blockers (to lower heart rate), fludrocortisone (to increase blood volume), or midodrine (to raise blood pressure).
- **Compression garments** to prevent blood pooling in the legs.
- **Exercise programs** to improve physical conditioning without exacerbating symptoms.
- **Dietary changes** such as small, frequent meals to prevent postprandial hypotension (drop in blood pressure after eating).
- **Consistent sleep schedule** to improve overall autonomic function.
- **Avoiding triggers** such as prolonged standing, extreme temperatures, and large meals that divert blood to the stomach.
- **Elevating the head of the bed** to improve blood volume distribution.
- **Mindfulness and stress management** to help cope with the impact of symptoms on mental health.



TYPICAL LIKELIHOOD

Typical likelihood of POTS based on 4 genetic variants we looked at

78th

PERCENTILE



Your risk is **greater than 78% of the population** and **lower than 22% of the population.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GNB3	rs5443	TT
MMP2	rs7194256	CT
NOS3	rs2070744	TC

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

Heart Arrhythmia

Arrhythmias can be caused by several factors, including [R]:

- Coronary artery disease: the most common cause of heart rhythm issues.
- Heart attack: scarring of the heart tissue from a heart attack can disrupt the heart's electrical signals.
- Changes in heart structure, such as from cardiomyopathy.
- Electrolyte imbalances that affect the heart's electrical impulses.
- High blood pressure
- Infection or fever
- Alcohol, caffeine, tobacco, or drug use
- Certain medications
- Stress

Treatment depends on the type and severity of the arrhythmia and may include [R]:

- Medications: to control the heart rate or restore a normal heart rhythm.
- Cardioversion: electric shocks or drugs to reset the heart to its regular rhythm.
- Catheter ablation: radiofrequency energy to destroy small areas of heart tissue that may be causing the arrhythmia.
- Pacemaker: a device implanted under the skin to help manage slow heart rhythms.
- Implantable cardioverter defibrillator (ICD): a device similar to a pacemaker that can correct life-threatening arrhythmias by delivering shocks.
- Lifestyle changes: diet and exercise modifications, reducing alcohol and caffeine intake, and managing stress.



TYPICAL LIKELIHOOD

Typical likelihood of heart arrhythmia based on 689,990 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GORAB	rs588837	GG
/	rs34443138	TT
/	rs34109091	AA
VNN3	rs6941949	TT
TBX5	rs883079	TT
PPFIA4	rs871298	GC
CAV1	rs729949	AG
HAND2	rs4615152	CT
LRMDA	rs10458661	CG
NEURL1	rs373205748	CC
/	rs141301535	CC
PITX2	rs12644625	CC
ATP5MK	rs12253987	TT
PITX2	rs12506083	AA
HP	rs2359171	TT
PITX2	rs3853445	TT
HAND2	rs3822127	GG
CUX2	rs3809297	GG
NEBL	rs2296610	GG
ZFHX3	rs9921081	TT
PTCHD1	rs73205368	TT

GENE	SNP	GENOTYPE
KCND3	rs12129789	TT
MAPT	rs242557	GG

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

Heart Rate Variability

Key Takeaways:

- HRV reflects how adaptable our bodies are.
- People with higher HRV tend to be more relaxed, healthier, and in better shape.
- Up to **60%** of differences in people's HRV may be due to genetics.
- Besides genetics, our emotions, fitness level, physical and mental health, and age influence HRV.
- The relevance of HRV as a health indicator is still being studied.

The heart doesn't keep the same rhythm from one heartbeat to the next. Instead, the time between heartbeats is constantly changing. HRV is a measure of those changes [R].

Up to **60%** of differences in people's HRV may be due to genetics. Involved genes may also influence heart rate and blood pressure [R, R, R].
Genetically higher HRV may be causally associated with [R, R, R]:

- Atrial fibrillation
- Heart attack
- High blood pressure (diastolic)

Many other internal and external factors influence HRV, including [R, R, R]:

- Emotions
- Personality
- Physical fitness
- Race
- Health condition

Factors linked to **lower HRV** include:

- Overtraining [R, R]
- Stress [R, R, R]
- Sleep disturbances [R, R, R]
- Alcohol consumption and smoking [R, R, R, R]
- Air pollution [R, R, R, R]
- Some drugs (e.g., antidepressants) [R, R, R, R]
- Anxiety and depression [R, R]
- Many health conditions (e.g., diabetes and thyroid disorders) [R, R, R]
- Inflammation and obesity [R, R, R]
- Aging [R]

Thankfully, there are a number of ways to increase HRV. One thing to keep in mind is that you should focus on improving your physical health, mental health, and fitness in general. HRV is just a marker that will reflect those changes.



HIGHER

Predisposed to higher HRV based on 1,286 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SOX5	rs10842383	CT
PPIL1	rs236349	GA
NEO1	rs2680344	GA
NEO1	rs1812835	AA
RGS6	rs2529471	CC
SYT10	rs1351682	GA
TGM2	rs6123471	TC
RBFOX1	rs4786125	AA
SYT10	rs1384598	TA
CAPS	rs12974440	GG
CAPS	rs12974991	GG
GNG11	rs4262	TT
RGS6	rs36423	GG
TMPRSS4	rs677652	CC
NDUFA11	rs12980262	GG
SYT10	rs7980799	CC
RGS6	rs2052015	CC
TFPI2	rs180238	TT
RGS6	rs4899412	CC
CCDC141	rs13004438	CC

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

Palpitations

Key Takeaways:

- While no heritability percentage exists yet for this condition, genes involved may influence various heart functions.
- Other risk factors include stress, anxiety, pregnancy, age, stimulants, overactive thyroid, and heart problems.
- Experiencing palpitations are not uncommon, particularly because they can result from causes unrelated to heart conditions, like anxiety.
- If your genetic risk is high, you can lower overall risk by taking action on risk factors that you can change.
- Click the **next steps** tab for relevant labs and lifestyle factors.

When the heart is working normally, it beats in a steady rhythm. Things like exercise or stress can speed it up temporarily [R, R].

Palpitations are feelings that the heart is racing, pounding, fluttering, or skipping beats. They can be felt in the chest or throat [R].

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

- Stress
- Anxiety
- Pregnancy
- Aging
- Stimulants (e.g., in coffee, cigarettes, and some medications)
- Overactive thyroid
- Heart problems

While palpitations can be scary, they are usually harmless. In those with palpitations due to heart problems, complications can occur. Seek emergency care if you have palpitations and [R, R]:

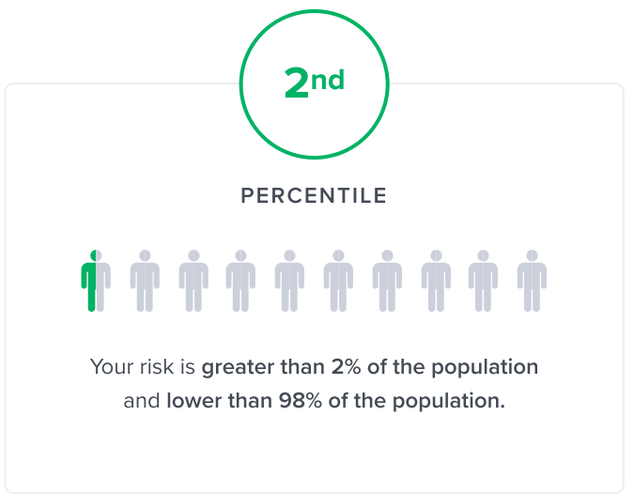
- Chest pain
- Severe shortness of breath
- Fainting
- Severe dizziness

Genetics may play a role in people's chances of having palpitations. Genes involved in palpitations may influence heart function [R, R].



LESS LIKELY

Less likely to have palpitations based on 1,209,944 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TRIM33	rs6693430	GG
TRIM33	rs576432148	AA
TRIM33	rs2336579	TT
VANGL1	rs146633921	CC
NTNG1	rs113586420	CC
/	rs112002694	AA
PEX14	rs111303544	GG
/	rs113879811	TT
PRPF38B	rs150830373	CC
KIF1B	rs374430851	GG
/	rs372345235	CC
/	rs111640537	CC
/	rs12070100	CC
PIFO	rs187038126	TT
FBXO44	rs369701158	CC
COL11A1	rs80206932	CC
PIFO	rs150153130	CC
/	rs530195853	GG
/	rs12057505	GG
PRDM2	rs141161525	CC
/	rs11184531	CC

GENE	SNP	GENOTYPE
UBE4B	rs61782898	CC
/	rs11184526	CC
PRMT6	rs143823403	GG
PERM1	rs138543362	GG
ZNF697	rs113542321	TT
TENT5C	rs142254460	CC
KIF1B	rs60490329	CC
ST7L	rs192255766	TT
MTOR	rs551629035	GG
/	rs9919305	TT
/	rs148678515	TT
SPAG17	rs138576102	CC
SPAG17	rs57221566	GG
COL11A1	rs542303135	GG
OLFM3	rs113591435	GG
RAP1A	rs116289484	CC
CD58	rs141148236	GG
MTOR	rs112840392	TT

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

Bradycardia

The causes of bradycardia vary and can include changes in the heart resulting from aging, damage to heart tissues from heart disease or a heart attack, congenital heart defects, electrolyte imbalances, or certain medications that can affect the heart's rhythm such as beta-blockers. When bradycardia is symptomatic, it may necessitate medical intervention.

Treatment options are based on the underlying cause and may range from medication adjustments to the implantation of a pacemaker, which serves to ensure the heart rate does not fall below a predetermined threshold, thus maintaining an adequate heart rate for proper bodily function.



LESS LIKELY

Less likely to have bradycardia based on 1,673 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GJA1	rs17052912	AA
ENPEP	rs33966350	GG
BCO1	rs143238312	GG
BIN2	rs142121602	CC
EPS8L1	rs144463157	GG
RPGRIP1L	rs139974543	GG
CGN	rs41272459	GG
DHX38	rs11554765	CC
URB2	rs41310553	GG
CACNA1S	rs142356235	CC
ZNF100	rs138292237	GG
PKD1	rs147350387	GG
CD109	rs148575660	AA
FOXM1	rs28990715	GG
DYNC2H1	rs144717489	GG
LRRC69	rs72666050	TT
EPRS1	rs114015346	TT
TRPM1	rs141540242	GG
RBL2	rs76818213	GG
VPREB1	rs11089977	AA
SCUBE2	rs72547298	GG
SNCAIP	rs140850272	GG
PNRC1	rs2231267	CC
GFM2	rs35080306	GG
BMPER	rs10249320	CC
DSG1	rs117656447	TT
RRP9	rs115426717	TT
RFX7	rs33984059	AA
POLQ	rs41540016	CC

GENE	SNP	GENOTYPE
CNTN6	rs41293401	GG
UEVLD	rs61752314	TT
ATRN	rs118065662	AA
KIAA1755	rs41282820	GG
RRS1	rs34077648	GG
TBX15	rs61730011	AA
SDCBP2	rs35367003	CC
RDH11	rs80140987	CC
ENTPD2	rs34618694	GG
FGD5	rs144177006	CC
ENDOG	rs200885264	GG

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



Blood Pressure

“That guy is going to burst a blood vessel!” The phrase is a reference to high blood pressure, typically induced by stress. On the other hand, if you get dizzy from getting up too fast, that could be due to low blood pressure.

Your circulatory system is set up to maintain a certain amount of pressure in order to keep blood flowing properly. **High blood pressure can be dangerous in particular**; it’s hard to notice and plays a major role in heart disease, stroke, and more. **These reports look at your genetic predispositions for high or low blood pressure, as well as the contributing effects of salt**.

MORE LIKELY

Low Blood Pressure

More likely to have low blood pressure

TYPICAL

Salt Sensitivity

Likely typical sensitivity to salt

TYPICAL LIKELIHOOD

High Blood Pressure

Typical likelihood of hypertension

TYPICAL LIKELIHOOD

Preeclampsia

Typical likelihood of preeclampsia

TYPICAL LIKELIHOOD

Pulmonary Hypertension

Typical likelihood of pulmonary hypertension

Low Blood Pressure

Key Takeaways:

- Genetics may play a role by influencing nervous system function, kidney function, and hormone activity.
- Other risk factors include certain medications and health conditions.
- It is estimated that up to **25%** of adults may have low blood pressure.
- Symptoms are uncommon, so if you do experience symptoms, speak to a healthcare professional. Managing symptoms may involve things like drinking more water, increasing salt intake, avoiding alcohol, changing positions slowly, diet changes, exercising, and wearing compression stockings.
- Click the **next steps** tab for relevant labs.

When your heart beats, it pumps blood to your entire body through your blood vessels. As blood circulates, it pushes against the inner walls of these blood vessels. Your blood pressure is a measurement of how hard your blood is pushing on these walls. **Blood pressure drops when the blood vessels widen or when the heart pumps slower** [R, R].
When a doctor measures your blood pressure, they give you two numbers. The first number describes the force when your heart beats (*systolic* blood pressure). The second number describes the force between heartbeats (*diastolic* blood pressure) [R].

A reading below 120/80 is generally considered normal. However, **a reading below 90/60 is considered to be low** [R, R].

Up to 25% of older adults may have low blood pressure. It may be less common in young people [R, R, R].

Besides older age, other risk factors for low blood pressure include some [R]:

- Medications
- Health conditions (e.g., Parkinson's disease, heart problems)

Two of the most common types of low blood pressure are [R]:

- **Orthostatic hypotension** (also called **postural hypotension**): Occurs almost immediately after standing up
- **Postprandial hypotension**: Occurs 1-2 hours after eating a meal

Sudden drops in blood pressure may also be a sign of an underlying health problem. This may be the case if you experience [R]:

- Fainting
- Blurry vision
- Nausea
- Fatigue
- Loss of coordination

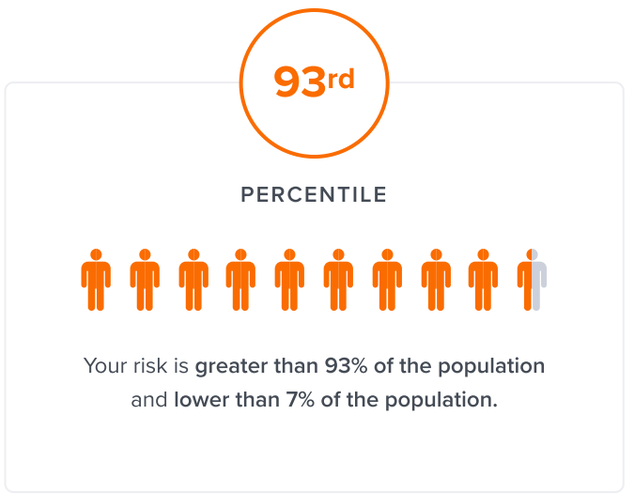
Ways to manage low blood pressure include [R]:

- Drinking more water
- Increasing salt intake
- Avoiding alcohol
- Changing positions slowly (e.g., slowly getting up from a chair or from bed)
- Eating smaller, low-carb meals more often
- Exercising
- Wearing compression stockings



MORE LIKELY

More likely to have low blood pressure based on 54,555 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
STAR	rs16887217	TT
ACTBL2	rs6892553	TT
/	rs6736587	CA
WRNIP1	rs4959677	GC
PIK3AP1	rs7098785	AG
NEGR1	rs116746813	GG
DNAH14	rs74517776	GG
/	rs140537647	TT
DNAH14	rs76052697	CC
DNAH14	rs141112742	CC
DNAH14	rs115273007	CC
DENND1B	rs116306513	GG
DNAH14	rs115906048	CC
DNAH14	rs114806783	AA
DNAH14	rs143344867	GG
PLXNA2	rs114552486	GG
DNAH14	rs562197190	TT
CNIH3	rs115958935	AA
DNAH14	rs114841428	CC
LBR	rs79075305	CC
/	rs201392883	GG

If these strategies don't help, your doctor may prescribe medication [R].

Genetics may play a role in blood pressure by influencing [R, R, R]:

- Nervous system function
- Kidney function
- Hormone activity

GENE	SNP	GENOTYPE
HFM1	rs183176641	AA
FASLG	rs2639625	GG
NEGR1	rs76297070	GG
LBR	rs116561851	AA
TAF12	rs60327514	AA
LBR	rs115372030	GG
LBR	rs149016006	GG
USH2A	rs553114711	GG
FGGY	rs188687527	GG
OR14C36	rs115632439	GG
FASLG	rs140884462	GG
HMGB4	rs114694562	TT
LMO4	rs114165625	TT
USP24	rs184142173	GG
AMY1C	rs72983604	CC
GADD45A	rs12090085	GG
/	rs148689075	GG
RNF19B	rs181818032	GG

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
NR2F2	rs2398162	AA
ACE2	rs184874220	TT
SGK1	rs9389154	GG
PRKG1	rs7905063	TT
FGF5	rs16998073	AA
ACE2	rs714205	GC
ACE2	rs2285666	TC
GSKIP	rs11847625	GC
PRKG1	rs7897633	CA
SLC4A5	rs7571842	GA
SCNN1G	rs7404408	CC
SCNN1G	rs5735	TT
SCNN1G	rs4299163	GG
SCNN1G	rs4073930	TT
SCNN1G	rs4073291	AA
CLGN	rs2567241	CC
RAD52	rs2301880	CT
WNK1	rs12828016	GT
SLC8A1	rs11893826	AG
SLC4A5	rs10177833	CA
GC	rs4254735	CT
BCAT1	rs7961152	CC
RENBP	rs78377269	GG
AGT	rs699	GG
ACE	rs4343	AA
SLC24A3	rs3790261	AA
POC1B	rs2681472	AA
SGK1	rs9376026	CC
RAD52	rs880054	TC

GENE	SNP	GENOTYPE
CPA3	rs75367686	AA
SLC8A1	rs434082	CC
CSTF2T	rs12414562	GG
CSTF2T	rs10997916	GG
HYAL1	rs10510755	CC
ADRB2	rs1042714	CC
SLC4A4	rs10022637	TT
KL	rs9536314	TT
SCNN1A	rs4764586	CC
SCNN1G	rs4499238	CC
SCNN1A	rs3741914	CC
TNFRSF1A	rs11614164	AA

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

High Blood Pressure

Key Takeaways:

- About 50% of people's differences in blood pressure may be due to genetics.
- Risk factors include age, ethnicity, diet, weight, activity levels, and stress which are all highly modifiable.
- If your genetic risk is high or you already have high blood pressure, you can take steps now to help reduce overall risk and improve your health.
- High blood pressure rarely causes symptoms, but it raises the risk for stroke and heart attack. Nine out of 10 Americans develop high blood pressure at some point in their lives.
- Click the **next steps** tab for relevant labs and lifestyle factors.

There are two major types of high blood pressure.

The first one is slow-developing and without an underlying cause. Doctors call this *primary* or *essential hypertension*. The majority of people will develop this type of high blood pressure.

Several factors can contribute to primary hypertension [R]:

- Age
- Being overweight or obese
- Not getting enough physical activity
- Tobacco use
- A diet high in salt (sodium)
- A diet low in potassium
- Alcohol abuse
- Stress
- Ethnicity (African ancestry)
- **Genetics**

Sometimes, high blood pressure is the result of a known underlying cause. Doctors call this *secondary* hypertension. Some examples of things that can cause secondary hypertension include [R]:

- Abuse of recreational drugs, such as cocaine and amphetamines
- Some medications, such as birth control pills and painkillers
- Conditions such as obstructive sleep apnea, kidney disease, and blood vessel defects

High blood pressure usually doesn't produce any symptoms. Most people don't realize they have it until they visit their doctor for a routine checkup [R]!

The danger is that high blood pressure increases your chances of heart attack and stroke. In 2018, high blood pressure contributed to the death of almost 500,000 Americans [R, R].

The good news is that high blood pressure is easy to detect and treat. Your doctor will work with you to reduce your blood pressure. They may recommend medication, a low-sodium diet, exercise, and other lifestyle changes [R].

Some strategies and recommendations may work better for some people than others. This is partly due to genetics, which may account for up to 50% of differences in blood pressure [R, R].

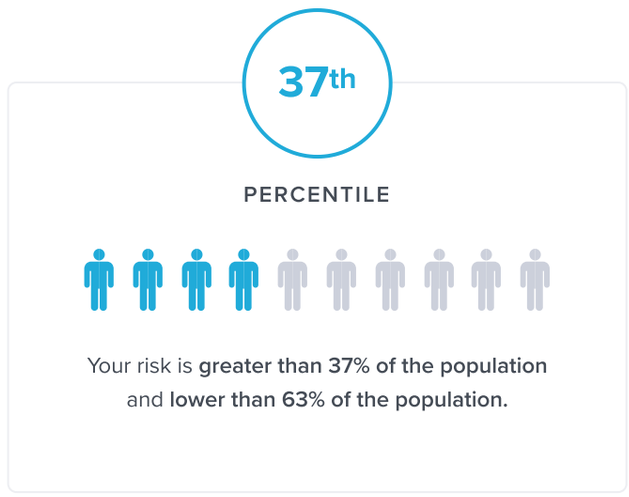
Genes that influence blood pressure can affect:

- Blood volume (*SCNN1A*, *NPR3*, *CSK*, *AGT*, and *ACE2*) [R, R, R, R, R]



TYPICAL LIKELIHOOD

Typical likelihood of hypertension based on 1,035,787 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
AGT	rs699	GG
ABO	rs579459	TT
ADRB1	rs1801253	CC
APOE	rs7412	CC
ACE2	rs2285666	TC
EPAS1	rs10168349	CG
NPR3	rs1173771	AG
SH2B3	rs3184504	TC
BCL2	rs12454712	CT
ULK3	rs6495122	CA
ARHGAP42	rs633185	GC
KCNK3	rs35021474	CC
NT5C2	rs112913898	GG
TNNT3	rs1973765	CC
NT5C2	rs11191593	TT
NT5C2	rs11191580	TT
NT5C2	rs11191548	TT
NT5C2	rs12219304	GG
CASZ1	rs880315	CT
FGF5	rs13149993	GA
ACE	rs4343	AA

- Blood vessel width (*AGT*, *ACE2*, and *NOS3*) [R, R, R]
- Stress response (*ADRB1* and *ADRB2*) [R, R]
- Breakdown of blood pressure-raising compounds, such as caffeine (*CYP1A2*) [R, R]

AGT and *ACE2* genes raise your blood pressure. They do this by increasing the amount of blood and making your blood vessels smaller. ACE inhibitors are blood pressure-lowering drugs that can counteract this [R, R, R].

Moreover, genetic predisposition to high levels of the following markers may be causally associated with high blood pressure [R, R, R, R, R, R]:

- Free testosterone
- Lymphocyte count
- Neutrophil count
- L-carnitine
- Alpha-linolenic acid

In contrast, genetically high IGF-1 and EPA levels may be causally associated with lower blood pressure [R, R].

It's important to remember that genetics isn't everything. Your lifestyle and environment account for about 50% of blood pressure differences [R].

GENE	SNP	GENOTYPE
TNNT3	rs4980379	CC
TCF7L2	rs34872471	TT
ULK3	rs2472299	GG
TWIST1	rs2107595	GG
ACE2	rs1978124	CC
NGF	rs11466111	CC
PRKAG2	rs10224002	AA
RPTOR	rs139293840	GG
BMP3	rs17004869	AA
FGF5	rs10857147	AA
CACNA1D	rs3774427	CC
CACNA1D	rs9814480	CC
FGF5	rs16998073	AA
FGF5	rs11099098	GG
ST7L	rs10776752	GG
FGF5	rs1458038	CC
ST7L	rs3790604	CC
EML6	rs72806698	CC
FES	rs17514846	CC
PRDM8	rs1902859	TT
ST7L	rs12129649	GG
EML6	rs17046380	TT

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

Preeclampsia

Preeclampsia can affect any pregnant woman, but certain factors may increase the risk [R, R, R]:

- First pregnancy.
- A history of preeclampsia in previous pregnancies.
- Chronic hypertension, kidney disease, or diabetes.
- Obesity or being overweight.
- Maternal age over 35 or under 20.
- Carrying twins or multiples.
- Family history of preeclampsia (e.g., mother or sister).
- Autoimmune disorders, such as lupus.
- Genetic predisposition.

Genetics accounts for about **50%** of the differences in blood pressure and preeclampsia rates [R, R].

Management of preeclampsia depends on the severity and stage of pregnancy [R, R, R]:

- Mild cases may require blood pressure monitoring, medication, and bed rest.
- Severe cases may necessitate hospitalization, early delivery, or emergency cesarean section.

Early diagnosis and intervention are critical to improving outcomes for both mother and baby.
Consumer Summary

- Preeclampsia is a pregnancy complication marked by high blood pressure and organ problems, often involving the liver or kidneys. It typically happens after 20 weeks of pregnancy but can occur earlier or even after delivery.
- Symptoms include very high blood pressure, severe headaches, swelling in the face or hands, sudden weight gain, blurry vision, pain under the ribs, shortness of breath, and low urine output. Recognizing these signs early can prevent serious complications.
- Risk factors include being pregnant for the first time, having a history of preeclampsia or other chronic conditions, being over 35 or under 20, being overweight, carrying twins, and genetics.
- Mild cases may need close monitoring, medications, and rest, while severe cases might require hospitalization or early delivery to ensure the safety of both the mother and baby. Early diagnosis and treatment are essential for better outcomes.



TYPICAL LIKELIHOOD

Typical likelihood of preeclampsia based on 7,287,096 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
FGL1	rs2653414	AC
NELFCD	rs259983	CC
ANKRD13C	rs482843	GA
AUH	rs5899121	INS(G)INS(G)
PGR	rs2508372	AA
RGL3	rs167479	GG
FTO	rs1421085	CC
WNT3A	rs708119	CC
MECOM	rs9855086	AA
SH2B3	rs10774624	GA
FLT1	rs7318880	CT
MICB	rs2442752	TC
UPB1	rs17572606	CC
FGF5	rs16998073	AA

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

Pulmonary Hypertension

Genetics influences the risk of PAH. Around **6–10%** of people with a diagnosis of PAH have a family history of the disease [R].

Factors that might increase the risk of developing PAH include:

- Certain drugs (e.g., diet drugs, drugs for irregular heartbeat, methamphetamines, cocaine)
- Living at a high altitude.

The following health conditions may also increase the risk:

- Connective tissue disorders (e.g., scleroderma)
- Liver disease
- HIV
- Congenital heart disease
- Sickle cell anemia.
- History of pulmonary embolism or blood clots.



TYPICAL LIKELIHOOD

Typical likelihood of pulmonary hypertension based on 22,528 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SOX17	rs13266183	CC
DNAJC10	rs71427857	GG
CBLN2	rs2217560	AA
SOX17	rs10103692	AA
HLA-DPA1	rs2856830	TT

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



Heart Health Genes

Your heart health is deeply influenced by both lifestyle and genetic factors, with specific genes playing a pivotal role in regulating cholesterol levels, blood pressure, and vascular function. While we all share a common set of heart health risks, your unique genetic makeup can determine how your body processes fats, manages blood flow, and responds to various cardiovascular stressors.

This section delves into key **heart health genes** such as **APOE, CETP, NOS3, ACE, and PCSK9**, which are involved in lipid metabolism, nitric oxide production, and the regulation of blood pressure and cholesterol. By understanding your genetic predispositions, you can make more informed decisions to support your heart health and prevent potential cardiovascular issues.

E3/E4

APOE

You carry one ε3 and one ε4 APOE variant

HIGHER ACTIVITY

CETP (Cholesterol/ Longevity)

Likely higher CETP activity

HIGHER ACTIVITY

FABP2 (Blood Sugar/ Cardiovascular)

Likely higher FABP2 activity

LOWER ACTIVITY

NOS3 (Cardiovascular)

Likely lower NOS3 activity

HIGHER ACTIVITY

PCSK9 (Cholesterol)

Likely higher PCSK9 activity

HIGHER ACTIVITY

CELSR2-PSRC1-SORT1 (Cardiovascular)

Likely higher CELSR2-PSRC1-SORT1 activity

LOWER ACTIVITY

CPS1 (Cardiovascular, Kidney Health)

Likely lower CPS1 activity

HIGHER ACTIVITY

AGT (Cardiovascular & Fitness)

Likely higher AGT activity

HIGHER ACTIVITY

SOAT1 (Cholesterol/ Cognition)

Likely higher SOAT1 activity

HIGHER ACTIVITY

ADRB1 (Cardiovascular)

Predisposed to higher ADRB1 activity

LOWER ACTIVITY

IRS1 (Metabolic Health)

Predisposed to lower IRS1 activity

TYPICAL ACTIVITY

APOA5 (Cardiovascular)

Predisposed to typical APOA5 activity

TYPICAL ACTIVITY

ABCG8 (Cholesterol & Gallstones)

Predisposed to typical ABCG8 activity

TYPICAL ACTIVITY

APOC3 (Blood Lipids/ Longevity)

Likely typical APOC3 activity

TYPICAL ACTIVITY

LIPC (Cardiovascular)

Likely typical LIPC activity

TYPICAL ACTIVITY

MLXIPL (Triglycerides, Cardiovascular)

Likely typical MLXIPL activity

TYPICAL ACTIVITY

HMGCR (Cholesterol)

Likely typical HMGCR activity

TYPICAL ACTIVITY

APOA2 (Weight, Blood Lipids)

Likely typical APOA2 activity

TYPICAL ACTIVITY

APOB Gene (Cardiovascular)

Likely typical APOB activity

TYPICAL ACTIVITY

LPA (Blood Lipids & Heart Health)

Predisposed to typical LPA activity

TYPICAL ACTIVITY

LDLR (Cholesterol, Cardiovascular)

Predisposed to typical LDLR activity

LOWER ACTIVITY

ACE (Fitness/ Cardiovascular)

Likely lower ACE activity

HIGHER ACTIVITY

GCH1 (Cardiovascular)

Likely higher GCH1 activity

HIGHER ACTIVITY

DOCK7 (Blood Lipids)

Likely higher DOCK7 activity

BETTER GENETICS

ABCA1 (Cholesterol)

Likely better ABCA1 genetics

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/E4

You carry one ε3 and one ε4 APOE variant based on the genetic variants we looked at

22%

OF USERS SHARE THE SAME SCORE



You have the same genetic predisposition as **22% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
APOE	rs429358	CT
APOE	rs7412	CC

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

CETP (Cholesterol/ Longevity)

Certain variants in *CETP* are associated with longevity in some studies. According to researchers, this life-extending effect may be due to improved cholesterol levels, which may help prevent a number of heart conditions [R].

Longevity research has focused on two particular variants. The 'GG' genotype in **rs5882** (also known as the "I405V" polymorphism) and the 'AA' genotype in **rs708272** (also called the "TaqIB" polymorphism) have each been associated with lower CETP activity and longer lifespan [R].

These variants have also been associated with a more favorable blood lipid profile characterized by [R, R, R, R, R, R]:

- Higher HDL
- Larger HDL and LDL particle size
- Higher apolipoprotein A-I
- Lower triglycerides



HIGHER ACTIVITY

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

52%

OF USERS SHARE THE SAME SCORE



You have the same genetic predisposition as **52% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CETP	rs708272	GG
CETP	rs5882	GA

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

FABP2 (Blood Sugar/ Cardiovascular)

The most widely researched polymorphism is **rs1799883**, also called Ala54Thr. Its minor ‘T’ allele encodes a protein with an amino acid substitution that increases its affinity for long-chain fatty acids. As a result, the mutated version of this protein increases intestinal fatty acid absorption while reducing insulin sensitivity [R].

In line with its link to reduced insulin sensitivity, this variant has been associated with an increased risk of type 2 diabetes, especially in Asians [R, R, R, R].

This variant has also been associated with:

- High blood pressure [R]
- High triglycerides [R]
- High BMI, body weight, and hip circumference [R, R]
- Metabolic syndrome [R]
- Coronary artery disease [R]
- Ischemic stroke [R, R]

Moreover, women with the ‘C’ variant lowered their total cholesterol, triglycerides, fasting glucose, and HbA1c more after following a low-glycemic-index diet for 4-5 weeks in a study on 165 patients with type 2 diabetes. However, this variant didn’t modify the effectiveness of the intervention in men [R].

Alternatively, carriers of the ‘T’ allele increased their insulin resistance and decreased their HDL to total cholesterol ratio more when consuming a diet high in saturated fat in a trial of 2148 participants [R].



HIGHER ACTIVITY

Likely higher FABP2 activity based on the genetic variants we looked at

44%

OF USERS SHARE THE SAME SCORE



You have the same genetic predisposition as **44% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
FABP2	rs1799883	CT

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, 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].



LOWER ACTIVITY

Likely lower 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	TC
NOS3	rs1549758	CT
NOS3	rs3918226	CC

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

PCSK9 (Cholesterol)

One PCSK9 variant, **rs562556-G**, is linked to **lower LDL cholesterol levels and lower odds of heart disease** [R, R].

In a large study, two rare PCSK9 variants have shown similar protective associations [R]:

- **rs11591147-T (R46L)**: 15% lower LDL and 2 times lower odds of heart disease in people of European ancestry.
- **rs28362286-A (C679X)**: 28% lower LDL and 9 times lower odds of heart disease in people of African ancestry.

The second variant is extremely rare and exists only in people of African ancestry.

These variants reduce PCSK9 activity, which then increases the number of LDL receptors and enhances cholesterol metabolism.



HIGHER ACTIVITY

Likely higher PCSK9 activity based on the genetic variants we looked at

63%

OF USERS SHARE THE SAME SCORE



You have the same genetic predisposition as **63% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PCSK9	rs562556	AA
PCSK9	rs11591147	GG
PCSK9	rs28362286	CC

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

CELSR2-PSRC1-SORT1 (Cardiovascular)

The best-characterized polymorphism within this cluster is **rs599839**. Its minor 'A' allele may increase *PSRC1* and *SORT1* expression. This variant has been associated with higher total cholesterol, LDL cholesterol, ApoB, CRP, IL-1beta, and TNF-alpha levels, as well as with an increased risk of coronary artery disease and myocardial infarction [R, R, R, R, R, R, R, R, R].

Another well-researched polymorphism is **rs646776**. Its minor 'T' allele may increase *PSRC1* and *SORT1* expression. This variant has been associated with higher total cholesterol, LDL cholesterol, and ApoB levels, as well as an increased risk of cardiovascular disease, including coronary artery disease, peripheral artery disease, and myocardial infarction [R, R, R, R, R].

These variants are usually inherited together, meaning you will most likely have both or neither of them.



HIGHER ACTIVITY

Likely higher CELSR2-PSRC1-SORT1 activity based on the genetic variants we looked at

61%

OF USERS SHARE THE SAME SCORE



You have the same genetic predisposition as **61% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SORT1	rs599839	AA
SORT1	rs646776	TT

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

CPS1 (Cardiovascular, Kidney Health)

The best-characterized *CPS1* polymorphism is **rs1047891** (T1405N), formerly known as rs7422339. Its minor 'A' allele has been associated with lower urea levels in the blood and ammonia levels in the liver, suggesting lower enzyme activity. This variant has also been associated with higher LDL cholesterol, homocysteine, and glycine levels but lower HDL cholesterol and apoA1 levels, as well as with [R, R, R, R]:

- Hyperammonemia in epilepsy patients taking valproic acid [R, R, R, R, R, R]
- Increased risk of coronary artery disease [R]
- Increased risk of migraine and CKD [R, R]
- Lower platelet count [R]
- Lower eGFR [R]

However, this variant has been associated with a decreased risk of liver scarring in people with NAFLD and lower odds of necrotizing enterocolitis [R, R, R].

Another variant believed to decrease *CPS1* expression, 'C' of **rs715**, has been associated with:

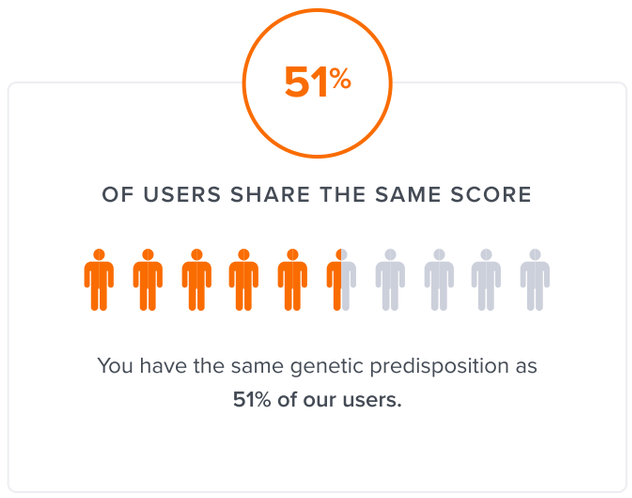
- Higher glycine and lower citrulline and TMAO levels [R, R]
- Lower eGFR [R]
- Higher BMI [R]
- Decreased risk of macular telangiectasia type 2 [R]
- Decreased risk of severe coronary artery disease [R]

This variant is usually inherited together with rs1047891, meaning you will most likely have both or neither of them.



LOWER ACTIVITY

Likely lower CPS1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CPS1	rs1047891	CA
CPS1	rs715	TC

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

AGT (Cardiovascular & Fitness)

The main *AGT* polymorphism is **rs699**, commonly called M235T in the literature. Its 'G' allele may increase angiotensinogen production, thereby promoting the production of angiotensin II and aldosterone [R].

This variant has been associated with an increased risk of:

- Hypertension [R, R, R]
- Preeclampsia [R, R, R]
- Ischemic stroke [R, R, R]
- Coronary heart disease [R, R, R, R, R]
- Heart failure [R, R]
- Heart attack [R, R]
- Diabetic nephropathy [R]
- End-stage kidney disease [R]

Moreover, carriers of this variant may see a weaker reduction of their blood pressure in response to exercise [R].

On the bright side, this variant may confer greater athletic performance in power sports [R, R].



HIGHER ACTIVITY

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

64%

OF USERS SHARE THE SAME SCORE



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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
AGT	rs699	GG

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

SOAT1 (Cholesterol/ Cognition)

The main *SOAT1* (ACAT-1) variant is **rs1044925**. Its **“C” allele is generally considered “bad”** due to its link with higher SOAT1 activity, which may imply increased cholesterol buildup.

Studies have linked this variant to [R, R, R, R]:

- Higher cholesterol levels in men
- Higher blood pressure
- Alzheimer’s disease
- Chagas disease (tropical disease that may involve heart problems)

However, some studies didn’t find a link between this variant and blood lipids or Alzheimer’s. One study even found a protective effect on heart health due to higher HDL cholesterol levels [R, R, R, R].

Other SOAT1 variants include:

- rs11545566-G: A study linked it to heart disease and artery hardening [R]
- rs2247071-C: May be linked to Alzheimer’s disease [R]
- rs13306731-G: Studies have linked it to heart problems [R]

Lower SOAT1 (ACAT-1) activity means less conversion of free cholesterol to cholesterol esters. This reduces cholesterol storage in cells and may help prevent foam cell formation in arteries. It may also reduce the formation of amyloid plaques in the brain [R, R].



HIGHER ACTIVITY

Likely higher SOAT1 activity based on 4 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
AXDND1	rs1044925	AC
AXDND1	rs11545566	GG
TOR3A	rs2247071	TC
SOAT1	rs13306731	AA

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

ADRB1 (Cardiovascular)

The best-researched *ADRB1* polymorphism is **rs1801253** (Arg389). Its minor 'C' allele may increase the activation of the beta-1 adrenergic receptors and has been associated with [R]:

- Higher blood pressure [R, R, R, R, R]
- Increased risk of cardiovascular disease [R, R, R, R]
- Increased risk of sudden cardiac death [R]
- Higher LDL cholesterol levels [R]
- Lower training-induced exercise tolerance [R]
- Increased risk of postoperative pain [R]

On the bright side, carriers may have a decreased risk of adverse effects in response to blood pressure medication (beta blockers) [R, R].

Another variant, 'A' of **rs1801252** (Ser49Gly), may increase ADRB1 stability and has been associated with [R]:

- Higher blood pressure [R]
- Increased risk of cardiovascular disease [R]
- Increased risk of sudden cardiac death [R]
- Lower odds of LVEF recovery in heart failure patients [R]
- Lower renin levels [R]

However, this variant has also been associated with a better response to beta blockers. Moreover, people with resistant hypertension are less likely to carry this variant [R, R].

Finally, the 'T' allele of **rs10787516** has been associated with relatively higher blood pressure. This variant may also increase ADRB1 activity, leading to increased heart rate and contraction pressure [R, R].



HIGHER ACTIVITY

Predisposed to higher ADRB1 activity based on 3 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ADRB1	rs1801253	CC
ADRB1	rs1801252	AA
ADRB1	rs10787516	TT

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

IRS1 (Metabolic Health)

One of the most studied IRS1 variants is **rs2943641**. People with the **“T” allele** may have [R]:

- Lower odds of heart disease and diabetes
- Lower levels of fasting glucose, insulin, and triglycerides
- Higher levels of HDL or “good” cholesterol

Interestingly, a study linked this variant to improved insulin resistance and weight loss on a high-carb, low-fat diet. However, another study reached the opposite conclusion [R, R].

Another IRS1 variant, **rs7578326-G**, showed similar associations as rs2943641-T. These two variants are sometimes inherited together, so some people will carry either none or both of them [R, R].

One rare variant, **rs1801278** (G972R), changes the structure of the IRS1 protein. Its **“T” allele** is linked to:

- Type 2 diabetes [R]
- Gestational diabetes (diabetes in pregnancy) [R]
- Higher cholesterol [R]

According to limited evidence, this variant also seems to favor high-carb diets [R].

Variants linked to better metabolic outcomes may boost IRS1 activity, improving insulin receptor activation. This stimulates the downstream signaling pathways essential for glucose uptake and lipid metabolism, directly impacting blood sugar and fat storage [R].



LOWER ACTIVITY

Predisposed to lower IRS1 activity based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
IRS1	rs2943641	CC
IRS1	rs7578326	AA
IRS1	rs1801278	CC

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

APOA5 (Cardiovascular)

Different *APOA5* variants are linked to impaired metabolic and cardiovascular health, more precisely [R, R, R, R, R, R, R, R]:

- Higher triglycerides, LDL, and apoB levels
- Lower HDL and vitamin D levels
- Heart problems

They may reduce **APOA5 activity**, impairing the removal of excess fat and cholesterol from the blood [R].



TYPICAL ACTIVITY

Predisposed to typical APOA5 activity based on 6 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PAFAH1B2	rs12272004	CC
SIDT2	rs2266788	GA
APOA5	rs2075291	CC
PCSK7	rs662799	AA
SIDT2	rs651821	TT
SIDT2	rs964184	CC
APOA5	rs3135506	GG

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

ABCG8 (Cholesterol & Gallstones)

ABCG8 promotes the removal of excess cholesterol through bile. This effect is good in moderation but may contribute to gallstones if too much cholesterol is processed [R].

In line with this, variants that increase ABCG8 activity are linked to **lower total and LDL cholesterol but higher odds of gallstones**. They include:

- **rs11887534-C**
- rs6544713-C
- rs4148217-A



TYPICAL ACTIVITY

Predisposed to typical ABCG8 activity based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ABCG8	rs6544713	CT
ABCG8	rs4148217	AC
ABCG8	rs11887534	GG

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

APOC3 (Blood Lipids/ Longevity)

The main *APOC3* variants, **rs5128** and **rs2542052**, may influence the metabolism of blood lipids. Their **“C” alleles** are linked to [R, R, R]:

- Lower triglycerides
- Lower LDL or “bad” cholesterol levels
- Higher HDL or “good” cholesterol levels

However, their impact on cardiovascular health is less clear. A meta-analysis of 79 studies looked at the link between stroke and APOC3, along with many other related genes. According to the results, rs5128 and other *APOC3* SNPs have no association with stroke [R].

When it comes to direct links with longevity, a study looked at rs2542052 in Ashkenazi Jews. The results suggest that the **“CC” genotype** may be more common in people who live past 100 years old. They also had better cardiovascular health and glucose metabolism [R].

However, one study looked at 749 American Caucasians who have exceptionally long lifespans and found no association between rs2542052 and longevity [R].

Finally, the rare **“A”** allele of **rs138326449** encodes a version of the protein with impaired function. This variant has been associated with decreased triglyceride and VLDL cholesterol but increased HDL cholesterol levels [R, R, R, R].



TYPICAL ACTIVITY

Likely typical APOC3 activity based on the genetic variants we looked at

50%

OF USERS SHARE THE SAME SCORE



You have the same genetic predisposition as **50% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SIDT2	rs2542052	AA
APOC3	rs138326449	GG
PCSK7	rs5128	CC

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

LIPC (Cardiovascular)

The most well-researched LIPC polymorphism is rs1800588. Its 'T' allele encodes an enzyme with lower activity and has been associated with higher total cholesterol, HDL cholesterol, apolipoprotein AI, and triglyceride levels. The HDL-cholesterol and triglyceride profile may be more unfavorable in 'T' carriers who eat a diet high in saturated fats [R, R, R].

This variant has also been associated with an increased risk of [R, R, R, R, R]:

- Type 2 diabetes
- Hypertriglyceridemia
- Coronary artery disease
- Coronary artery calcification
- Vitamin D insufficiency

On the bright side, aerobic exercise may improve HDL-cholesterol more in carriers of this allele. In contrast, people with two copies of the 'C' allele may benefit more from a high-carbohydrate diet for decreasing their apoB100/apoAI ratio [R, R].

The minor 'A' allele of another polymorphism, rs2070895 (250A/G), has been associated with higher LDL cholesterol and lower HDL cholesterol, as well as with an increased risk of [R]:

- Coronary artery disease [R]
- Artery hardening [R]
- Diabeic dyslipidemia [R]
- Hypertension, especially in alcohol drinkers [R, R]
- Peripheral artery disease [R]

However, this variant may be protective for stroke and type 2 diabetes [R, R].

Interestingly, carriers of this variant may lower their total and LDL cholesterol levels more if they adopt a low-fat diet and raise their HDL cholesterol level more if they exercise [R, R].

Another well-researched *LIPC* variant is rs10468017. Its minor 'T' allele may increase enzyme activity in the liver and has been linked to higher HDL and triglycerides, and lower LDL cholesterol levels. This variant has also been associated with a decreased risk of age-related macular degeneration and choroidal neovascularization [R, R, R, R, R, R].

Finally, the 'T' allele of rs493258 may also be linked to increased LIPC activity in the liver and has been associated with a decreased risk of age-related macular degeneration and higher plasma zeaxanthin levels [R, R, R].



TYPICAL ACTIVITY

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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ALDH1A2	rs10468017	CC
ADAM10	rs1800588	CC
ADAM10	rs2070895	GG
ALDH1A2	rs493258	TT

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

MLXIPL (Triglycerides, Cardiovascular)

The best-researched *MLXIPL* polymorphism is **rs3812316** (G771C). The rare 'G' allele has been associated with lower triglyceride levels due to the production of an unstable version of the protein. This allele has also been associated with a decreased risk of [R, R, R, R, R, R, R]:

- Coronary artery disease [R, R, R, R]
- Myocardial infarction [R]
- NAFLD [R]
- Obesity [R]
- Metabolic syndrome [R]
- Elevated uric acid levels [R]

Interestingly, the beneficial effects of this variant may be enhanced by the Mediterranean diet [R].



TYPICAL ACTIVITY

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

78%

OF USERS SHARE THE SAME SCORE



You have the same genetic predisposition as **78% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MLXIPL	rs3812316	CC

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

HMGCR (Cholesterol)

Summary
Variants that increase HMGCR activity are linked to higher cholesterol levels and adverse brain and cardiovascular health outcomes. On the bright side, people with those variants may see greater improvements from statin therapy for high cholesterol.

Details
The most well-researched *HMGCR* polymorphism is rs3846662. Its 'G' allele results in an increased production of HMGCR and has been associated with **higher LDL, apoB, and total cholesterol** but lower HDL cholesterol levels. On the bright side, carriers of this variant may lower their LDL cholesterol more in response to statins [R, R, R, R, R, R, R, R, R, R, R, R, R].

This allele has also been associated with an increased risk of:

- Alzheimer's disease [R, R, R, R, R]
- Mild cogntive impairment [R]
- Myocardial infarction [R]
- Overweight, especially in those with low soy intake [R]

Another well-researched polymorphism is rs12916. Its 'C' allele has been associated with higher total and LDL cholesterol levels and increased risk of ovarian cancer, but also with an increased effectiveness of statin treatment [R, R, R, R, R].

This variant has also been associated with:

- Insomnia [R]
- Prostate cancer [R]
- Aortic aneurysm [R]
- Premature triple-vessel coronary disease and residual cholesterol risk [R, R]

The 'T' allele of rs17244841 has been associated with a reduced effectiveness of statin therapy at lowering LDL cholesterol. Interestingly, a study of 306 patients with Parkinson's disease found that those carrying this allele had higher HDL cholesterol levels [R, R, R, R, R].

Finally, the 'G' allele at rs17238540 has been associated with a reduced effectiveness of statin therapy at lowering cholesterol and triglycerides, as well as with [R, R, R]:

- Greater triglyceride decrease in response to a low-fat, high-fiber diet [R]
- Higher blood pressure and increased stroke risk [R]

Because this variant and rs17244841 are usually inherited together, you will most likely carry either both or neither of them.



TYPICAL ACTIVITY

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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HMGCR	rs3846662	GA
HMGCR	rs12916	CT
ANKRD31	rs17244841	AA
HMGCR	rs17238540	TT

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

APOA2 (Weight, Blood Lipids)

Scientists have observed the association between one *APOA2* variation, rs5082, and obesity across different ethnic groups. **People with the “GG” genotype have significantly higher BMIs and obesity rates** [R, R].

According to two major trials, the “GG” genotype at rs5082 lowers apo A-II levels and correlates with increased calorie intake. Detailed analyses have confirmed the role of apo A-II in appetite control [R, R, R].

One study gathered data from three populations (3,462 total participants) and found a robust association between rs5082, obesity, and saturated fat (SF) intake. The "GG" carriers had 84% higher obesity rates compared with other genotypes, but only when their SF intake was high. **In cases of low SF intake, *APOA2* didn't correlate with obesity** [R].

Another trial of 4,600 Asian and Mediterranean subjects came to the same conclusion. Additionally, the “GG” allele was associated with insulin resistance in Chinese and Indian people who consumed more SF [R].

One study analyzed dairy intake in two populations (n=2,071) with a proven link between rs5082 and saturated fat. In both groups, the “GG” carriers who consumed more high-fat dairy had significantly higher BMIs [R].

Among 180 diabetic patients, unsaturated fatty acids positively affected those with the "GG" genotype. Increased intake of omega-3 and MUFA was associated with lower inflammatory markers (IL-18 and CRP) and stronger antioxidant defense (SOD) [R, R].

This *APOA2* variant may increase the levels of ghrelin or the “hunger hormone.” A study of 1,225 obese subjects found that people with the “GG” genotype who consume more SF have higher ghrelin levels. In other words, SF fails to satiate their hunger [R].

On the bright side, this variant has been associated with a **better blood lipid profile**.

By reducing *APOA2* expression, rs5082 can stimulate VLDL and triglyceride metabolism. Indeed, in a study of 88 participants, those with the “GG” genotype had lower triglyceride and cholesterol levels in response to a high-fat meal [R, R, R, R].

Among 700 diabetes patients, the “GG” carriers also had significantly lower triglycerides and total cholesterol but not HDL. In a trial of 982 Australian subjects, people with this variant had nearly two times lower rates of heart disease [R, R].



TYPICAL ACTIVITY

Likely typical APOA2 activity based on the genetic variants we looked at

85%

OF USERS SHARE THE SAME SCORE



You have the same genetic predisposition as **85% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
FCER1G	rs5082	GA

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

APOB Gene (Cardiovascular)

The best-studied *APOB* polymorphism is **rs693**, also known as XbaI. Its minor 'A' allele may increase APOB activity, leading to higher total cholesterol, LDL cholesterol, VLDL cholesterol, apoB, and triglycerides but lower levels of HDL cholesterol. This variant has been associated with an increased risk of [R, R, R, R, R, R, R, R]:

- Coronary artery disease [R]
- Metabolic syndrome [R]
- Calcific aortic stenosis [R]
- Ischemic stroke [R, R, R]
- Gallstone disease [R, R]

Another well-characterized *APOB* variant is **rs515135**. Its minor 'T' allele has been associated with higher total and LDL cholesterol levels, as well as with an increased risk of coronary artery disease [R, R, R, R, R, R].

Another variant linked to increased total cholesterol, LDL cholesterol, and ApoB levels, 'T' at **rs1801701**, has been associated with an increased risk of [R]:

- Myocardial infarction [R]
- Coronary artery disease [R]
- Ischemic stroke [R]

Finally, the minor 'A' allele of **rs934197** increases *APOB* expression and has been associated with higher total and LDL cholesterol levels [R, R, R, R, R].

TYPICAL ACTIVITY

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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
APOB	rs515135	TT
APOB	rs693	GG
APOB	rs1801701	CC
APOB	rs934197	GG

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

LPA (Blood Lipids & Heart Health)

In line with the *LPA* gene roles, its variants are linked to [R, R]:

- Lipoprotein(a) levels
- Total and LDL cholesterol levels
- Heart disease (CAD)

These variants likely **increase LPA activity**, resulting in higher Lp(a) levels and negative downstream effects.

In one large study, low-dose aspirin canceled out the cardiovascular risk from the *LPA* gene variant rs3798220-C [R].



TYPICAL ACTIVITY

Predisposed to typical LPA activity based on 7 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
LPA	rs41272114	CC
LPA	rs1853021	GG
SLC22A3	rs9364559	AA
LPA	rs10455872	AA
LPA	rs3798220	TT
SLC22A3	rs10755578	CC
LPA	rs6415084	CC

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

LDLR (Cholesterol, Cardiovascular)

The best-researched *LDLR* polymorphism is **rs688**. Its minor 'T' allele may decrease LDLR production and has been associated with higher total and LDL cholesterol levels, as well as with an increased risk of [R, R, R, R, R, R, R]:

- Coronary artery disease [R, R]
- Ischemic stroke [R, R, R, R, R]
- Cardiovascular disease in end-stake kidney disease patients [R]

Similarly, the minor 'C' allele of **rs5925** has been associated with a decreased *LDLR* expression, higher total cholesterol, LDL cholesterol, and triglyceride levels, and an increased risk of ischemic stroke and coronary artery disease [R, R, R, R, R, R].

These variants are usually inherited together, meaning you will most likely have both or neither of them.

Another well-characterized polymorphism is **rs6511720**. Its minor 'T' allele may increase *LDLR* expression by creating binding sites for proteins that enhance it in the gene sequence. Multiple studies have associated this allele with lower LDL cholesterol levels, as well as with a decreased risk of [R, R, R, R, R, R, R, R, R, R]:

- Coronary heart disease [R, R, R]
- Ischemic stroke [R]
- Myocardial infarction [R]
- Athersoclerosis [R]

Another variant, the minor 'T' allele of **rs1122608,** has been associated with lower total cholesterol and LDL cholesterol levels, as well as with a decreased risk of [R]:

- Coronary artery disease [R, R]
- Myocardial infarction, especially in people who smoke or drink alcohol [R, R]
- Ischemic stroke [R]

Interestingly, this variant may decrease the expression of a nearby gene (*SRSF3*), resulting in lower levels of the IL-1β cytokine [R].

Finally, the minor 'T' allele of **rs2228671**, has also been associated with lower LDL cholesterol levels and a decreased risk of coronary artery disease [R, R, R].



TYPICAL ACTIVITY

Predisposed to typical LDLR activity based on 5 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
LDLR	rs6511720	GG
LDLR	rs688	CT
LDLR	rs5925	TC
SMARCA4	rs1122608	GG
LDLR	rs2228671	CT

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

ACE (Fitness/ Cardiovascular)

The two main ACE gene variants, **rs4341 and rs4343**, influence gene and enzyme activity. Their **“G” alleles** may increase ACE activity and levels. In line with this, they are linked to **high blood pressure and heart disease** [R, R, R, R].

Regarding athletic performance, higher ACE activity may favor short, high-intensity bursts of activity. This makes it advantageous for **power-based sports** [R, R, R].

Conversely, **rs4341-C and rs4343-A** are linked to lower ACE activity, which may offer some protection against hypertension and cardiovascular conditions. Lower ACE activity can enhance **endurance** by improving blood flow and oxygen delivery to muscles during prolonged physical activity [R, R, R, R].

Please note: Some people's genetic files don't contain the rs4341 variant, so we didn't include it in the model. However, this variant is almost always inherited with rs4343, so one of them is sufficient to estimate your ACE genetics.



LOWER ACTIVITY

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

15%

OF USERS SHARE THE SAME SCORE



You have the same genetic predisposition as **15% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ACE	rs4343	AA

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

GCH1 (Cardiovascular)

The following three variants may **reduce GCH1 activity**. They have been linked to **lower BH4 levels,** especially in people with both copies [R]:

- **rs10483639-C**
- **rs3783641-A**
- **rs8007267-T**

People with these variants may be prone to **blood vessel issues**, especially if they have diabetes or heart disease. Lower BH4 levels mean less nitric oxide, a substance that widens the blood vessels and keeps them healthy [R, R].

On the other hand, BH4 is involved in pain signalling, and these variants are linked to **reduced pain sensitivity** [R, R, R].

Note that these variants are often inherited together, meaning you will likely have either all or none.



HIGHER ACTIVITY

Likely higher GCH1 activity based on the genetic variants we looked at

60%

OF USERS SHARE THE SAME SCORE



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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GCH1	rs10483639	GG
GCH1	rs8007267	CC
GCH1	rs3783641	TT

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

DOCK7 (Blood Lipids)

One of the most widely researched *DOCK7* polymorphism is **rs10889353**. Its minor 'C' allele has been associated with higher *DOCK7* expression, lower total cholesterol, LDL cholesterol, and triglyceride levels, and a decreased risk of coronary heart disease [R, R, R, R, R].

Another well-characterized polymorphism is **rs2131925**. Its minor 'G' allele has been associated with lower total cholesterol, LDL cholesterol, and triglyceride levels, higher vitamin D levels, and an a decreased risk of hypertension [R, R, R, R, R, R, R, R].

These variants are usually inherited together in people of Europena ancestry, meaning you will most likely have both or neither of them.



HIGHER ACTIVITY

Likely higher DOCK7 activity based on the genetic variants we looked at

50%

OF USERS SHARE THE SAME SCORE



You have the same genetic predisposition as **50% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
DOCK7	rs10889353	AC
DOCK7	rs2131925	TG

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

ABCA1 (Cholesterol)

A variant in the ABCA1 gene, **rs1883025-T**, is linked to **lower levels of cholesterol (all subtypes) and lower heart disease rates** [R].

This variant is also linked to a lower risk of **age-related macular degeneration** (AMD), an eye condition that can cause vision loss. It may help remove cholesterol from cells in the macula, a critical part of the retina [R].

Another notable ABCA1 variant is **rs2230808**, with the “T” allele linked to lower cholesterol levels and lower inflammation [R].

ABCA1 variants have robust links with HDL or “good” cholesterol. **Despite the reduction in HDL, the net effect on heart health is protective**, likely due to LDL reduction [R, R].

A rare variant, rs9282541-A (R230C), was linked to lower HDL levels and poor metabolic profile in Mexican women who consumed a high-carb diet. Women with this variant on a high-fat diet had higher LDL and better metabolic profiles [R].

BETTER GENETICS

Likely better ABCA1 genetics based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ABCA1	rs1883025	TC
ABCA1	rs2230808	TC
ABCA1	rs9282541	GG

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



Cholesterol & Lab Markers

Cholesterol and triglycerides (blood lipids) are key lab markers when it comes to heart health! Your body actually requires fats and cholesterol to store energy and help cells function. So, like many other things in the body, they require balance. Too much can increase the risk for health issues like heart disease.

How your body handles fats and cholesterol depends on a variety of factors, including your genetics.

Besides blood lipids, several other lab markers can tell a lot about heart health and potential risks. **Reports in this section look at your genetic predispositions for all lab markers relevant to heart health.**

MORE LIKELY

High Cholesterol

More likely to have high cholesterol

HIGHER LEVELS

Total Cholesterol

Predisposed to higher cholesterol levels

HIGHER LEVELS

LDL Cholesterol

Predisposed to higher levels of "bad" cholesterol

HIGHER LEVELS

TMAO

Predisposed to higher TMAO levels

TYPICAL

LDL Particle Size

Predisposed to typical LDL particle size

TYPICAL LEVELS

HDL Cholesterol

Predisposed to typical HDL levels

TYPICAL LEVELS

Lipoprotein(a)

Predisposed to typical Lipoprotein(a) levels

TYPICAL LEVELS

Triglycerides

Predisposed to typical triglyceride levels

TYPICAL EFFECTS

Effects of Omega-3s on Triglycerides

Predisposed to typical effects of omega-3s on triglycerides

TYPICAL LEVELS

VLDL Cholesterol

Predisposed to typical VLDL levels

TYPICAL

LDL Particle Number (LDL-P)

Predisposed to typical LDL-P

TYPICAL LEVELS

Homocysteine

Predisposed to typical homocysteine levels

TYPICAL LEVELS

Lp-PLA2

Predisposed to typical Lp-PLA2 levels

TYPICAL

Platelet Aggregation

Predisposed to typical platelet aggregation

TYPICAL LEVELS

Platelets

Predisposed to typical platelet count

TYPICAL

Response to Ezetimibe

Likely typical response to ezetimibe

LOWER LEVELS

ApoB

Predisposed to lower ApoB levels

LESS LIKELY

Metabolic Syndrome

Less likely to have metabolic syndrome

BETTER RESPONSE

Response to Statins (Functional)

Predisposed to better response to statins

High Cholesterol

Key Takeaways:

- **65%** of people's differences in cholesterol levels may be due to genetics.
- Other risk factors include a diet high in saturated fat, obesity, lack of exercise, older age, smoking, and diabetes.
- If you have high genetic risk, monitoring your cholesterol levels and taking action on modifiable risk factors can reduce your overall risk. If your genetic risk is low but you are testing high or have several risk factors, you may want to take action now.
- Click the **Recommendations** tab for potential dietary and lifestyle changes and **next steps** for relevant labs.

People think of cholesterol as being bad, but it's actually essential. Cholesterol gives shape to all of your cells. Your body also uses it to make vitamin D and some types of hormones [R].

There are two major types of cholesterol: LDL and HDL. We call HDL "good cholesterol" because it helps your liver get rid of excess cholesterol. On the other hand, LDL cholesterol is "bad" because it can stick to your blood vessels. This can cause heart problems [R, R].

If your doctor has ever told you that you have high cholesterol, they were either referring to [R]:

- LDL cholesterol
- Total cholesterol (LDL + HDL)

Some risk factors for high LDL cholesterol include [R, R]:

- A diet high in saturated fat
- Obesity
- Lack of exercise
- Older age
- Smoking
- Diabetes
- Family history (genetics)

If your cholesterol levels rise, your doctor will recommend strategies for lowering them. These may include [R]:

- A diet low in saturated fat
- The Mediterranean diet
- Exercise
- Losing weight

How well you respond to these strategies may depend on your genes.

Up to 65% of differences in cholesterol levels may be attributed to genetics. Genes that may contribute to high cholesterol influence [R, R, R, R, R, R]:

- Cholesterol production (*LPL*, *LIPC*, *HMGCR*)
- Cholesterol transport (*APOB*)
- HDL and LDL cholesterol balance (*CETP*)



MORE LIKELY

More likely to have high cholesterol based on 1,171,067 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
APOE	rs7412	CC
PCSK9	rs28362286	CC
PCSK9	rs11591147	GG
PCSK9	rs562556	AA
APOE	rs141622900	GG
APOE	rs7254892	GG
SIDT2	rs651821	TT
APOE	rs12721109	GG
LDLR	rs72658867	GG
APOE	rs62117160	GG
ABCA1	rs188308962	AA
APOE	rs429358	CT
IL6	rs1800795	GC
CETP	rs5882	GA
FABP2	rs1799883	CT
ABCG5	rs141828689	TC
TOMM40	rs394819	TG
LPL	rs328	CC
CETP	rs708272	GG
PCSK7	rs662799	AA
PCSK7	rs5128	CC

GENE	SNP	GENOTYPE
LIPG	rs77960347	AA
/	rs12713559	GG
/	rs151135411	GG
SNX8	rs144787122	AA
MICB	rs361525	GG
LIPG	rs117623631	CC
APOC4	rs140526515	AA
/	rs150401285	AA
NECTIN2	rs138914864	CC
NECTIN2	rs117310449	CC
NECTIN2	rs144261139	CC
NECTIN2	rs76366838	GG
APOE	rs769449	GG
APOE	rs4420638	AA
CLPTM1	rs12691088	GG
NECTIN2	rs138607350	TT
NECTIN2	rs34095326	GG
PVR	rs139267469	CC
LIPG	rs80175721	AA
SLC22A3	rs3918291	TT
CLPTM1	rs79429216	GG
MAFB	rs2207132	GG
NECTIN2	rs41289512	CC
LPA	rs3798220	TT
APOC1	rs60049679	GG
BCL3	rs114036675	GG
/	rs145030841	CC
ABCA6	rs77542162	AA
MAFB	rs1883711	GG

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

Total Cholesterol

If your doctor has ever told you that you have high cholesterol, they were either referring to [R]:

- LDL cholesterol
- Total cholesterol (LDL, HDL, and VLDL)

Some risk factors for high cholesterol include [R, R]:

- A diet high in saturated fat
- Obesity
- Lack of exercise
- Older age
- **Genetics**

If your cholesterol levels rise, your doctor will recommend strategies for lowering them. These may include [R]:

- A diet low in saturated fat (such as the Mediterranean diet)
- Exercise
- Losing excess weight
- Cholesterol-lowering medication

How well you respond to these strategies may, in part, depend on your genes.

Up to 65% of differences in cholesterol levels may be attributed to genetics. Genes that may contribute to high cholesterol influence [R, R, R, R, R, R, R]:

- Cholesterol production (*HMGCR*)
- Cholesterol transport (*APOB*)
- HDL and LDL cholesterol balance (*CETP*, *LIPC*, *LPL*)



HIGHER LEVELS

Predisposed to higher cholesterol levels based on 1,290,924 genetic variants we looked at

99th

PERCENTILE



Your risk is **greater than 99% of the population** and **lower than 1% of the population.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
APOE	rs7412	CC
PCSK9	rs11591147	GG
SIDT2	rs651821	TT
SLC16A11	rs186021206	GG
APOE	rs429358	CT
SMARCA4	rs142158911	GG
DNAJB14	rs140280172	CC
IL6	rs1800795	GC
CETP	rs5882	GA
FABP2	rs1799883	CT
MINK1	rs79202680	GG
SORT1	rs12740374	GG
HNF4A	rs1800961	CC
FLT3	rs76428106	TT
PPP1R3B	rs9987289	GG
KIF13B	rs117139027	GG
ABCA1	rs2740488	AA
CD300LG	rs72836561	CC
SEZ6	rs72817635	CC
SLC39A8	rs13107325	CC
GATA6	rs79120103	AA

GENE	SNP	GENOTYPE
LCAT	rs4986970	AA
HMGCR	rs12916	CT
IRF2BP2	rs508293	AG
LPL	rs328	CC
CETP	rs708272	GG
PCSK7	rs662799	AA
PCSK7	rs5128	CC
MICB	rs361525	GG
LIPG	rs77960347	AA
SNX8	rs144787122	AA
/	rs145030841	CC
ABCA6	rs77542162	AA
MAFB	rs1883711	GG
FGB	rs6054	CC
APOB	rs562338	AA
SLC33A1	rs76440173	CC
LPA	rs55730499	CC
ADAM10	rs1800588	CC
ABO	rs2519093	CC
APOH	rs149394327	GG
SGMS1	rs80276949	GG
HP	rs34042070	CC
NLRC5	rs56156922	TT
HAVCR2	rs12657266	CC
HLA-DQA2	rs6689	AA
NPC1L1	rs10260606	GG

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

LDL Cholesterol

Cholesterol in the blood is carried by proteins, mainly LDL, HDL, and VLDL. We call cholesterol carried by LDL “bad” cholesterol because it can stick to your blood vessels. This can cause heart problems [R, R].

Some risk factors for high LDL cholesterol include [R, R]:

- A diet high in saturated fat
- Obesity
- Lack of exercise
- Older age
- **Genetics**

If your cholesterol levels rise, your doctor will recommend strategies for lowering them. These may include [R]:

- A diet low in saturated fat (such as the Mediterranean diet)
- Exercise
- Losing excess weight
- Cholesterol-lowering medication

How well you respond to these strategies may depend on your genes.

Genetically higher LDL cholesterol levels may play a role in:

- Heart Health [R, R, R, R, R, R, R, R, R, R, R, R, R, R]
- High Blood Sugar [R, R, R, R, R, R, R, R]
- Stroke [R, R, R, R, R]
- Kidney Health/eGFR/Artery Hardening [R].
- Bone Health [R, R, R]
- Joint Pain [R]
- Parkinson’s Disease [R]
- Longevity [R, R, R, R]
- High Blood Pressure [R]

Up to 65% of differences in cholesterol levels may be attributed to genetics. Genes that may contribute to high cholesterol influence [R, R, R, R, R, R]:

- Cholesterol production (*HMGCR*)
- Cholesterol transport (*APOB*)
- HDL and LDL cholesterol balance (*CETP*, *LPL*, *LIPC*)

Genetically high testosterone levels may be causally associated with a high risk of increased LDL-cholesterol [R].



HIGHER LEVELS

Predisposed to higher levels of "bad" cholesterol based on 1,366,051 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
APOE	rs7412	CC
APOE	rs141622900	GG
APOE	rs7254892	GG
NLRC5	rs1800775	CC
PCSK9	rs11591147	GG
APOE	rs12721109	GG
APOE	rs62117160	GG
LDLR	rs72658867	GG
CEACAM20	rs200628672	GG
BCAM	rs28399654	GG
LDLR	rs688	CT
CETP	rs5882	GA
LDLR	rs6511720	GG
USP24	rs72660594	TT
CLPTM1	rs490243	TC
NECTIN2	rs365653	GA
ABCG5	rs141828689	TC
APOE	rs429358	CT
NECTIN2	rs11668327	GC
TOMM40	rs394819	TG
APOC1	rs389261	AG

GENE	SNP	GENOTYPE
LDLR	rs73015030	GA
APOB	rs693	GG
SIDT2	rs964184	CC
/	rs12713559	GG
/	rs151135411	GG
APOC4	rs140526515	AA
NECTIN2	rs138914864	CC
NECTIN2	rs117310449	CC
APOE	rs769449	GG
/	rs150401285	AA
NECTIN2	rs144261139	CC
NECTIN2	rs76366838	GG
APOE	rs4420638	AA
CLPTM1	rs12691088	GG
APOB	rs5742904	CC
SLCO1B1	rs4149056	TT
OLR1	rs12316150	AA
NECTIN2	rs34095326	GG
NECTIN2	rs41289512	CC
NECTIN2	rs138607350	TT
SNX8	rs144787122	AA
SLC22A3	rs3918291	TT
LPA	rs3798220	TT
PVR	rs139267469	CC
MAFB	rs2207132	GG
APOC1	rs60049679	GG
BCL3	rs114036675	GG

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

TMAO

Key Takeaways:

- Up to **30%** of differences in people's TMAO levels may be due to genetics.
- Other factors that may lead to high TMAO include L-carnitine, choline, and histidine supplements, certain foods, sleep deprivation, aging, kidney disease, and diabetes.
- If you have a high genetic risk or other risk factors, you may lower your overall risk by taking action now on factors that you can change.
- Click the **next steps** tab for relevant labs and lifestyle changes.



HIGHER LEVELS

Predisposed to higher TMAO levels based on 9 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PHACTR4	rs114145653	AA
UBE2G1	rs75116832	GG
RPA2	rs148553452	AA
IFNK	rs143482172	CC
PLN	rs75363923	CC
ENPP4	rs146839869	GG
TENM3	rs114755225	CC
AK9	rs143831173	AA
RHOBTB2	rs6557607	GG
FMO3	rs2266782	AA
FMO3	rs1736557	GG

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

TMAO (trimethylamine N-oxide) is an oxidation product of our gut microbiome, generated from the breakdown of foods. It can also be found in some foods, especially fish [R, R].

Up to **30%** of differences in people's TMAO levels may be due to **genetics** [R, R].

Our diet and, thereby, our gut microbiome play a key role in TMAO production. Consuming animal products rich in TMAO, choline, betaine, lecithin, and carnitine may increase TMAO levels. Some examples include [R, R, R, R, R, R, R, R, R]:

- Fish and seafood
- Eggs
- Dairy
- Red meat

In line with this, the following diets may raise TMAO levels:

- Western-like and high-fat diets [R, R]
- Red meat-rich diets (e.g., Paleo diet, low-carbohydrate diet) [R]

If your TMAO levels are elevated due to high fish and seafood intake, there is likely no need for concern. Many studies link fish and seafood consumption to lower heart disease risk. Fish is also an excellent source of omega-3 fatty acids [R, R].

Other factors that may lead to **high TMAO** include:

- L-carnitine, choline, and histidine supplements [R, R, R]
- Sleep deprivation [R]
- Aging [R]
- Kidney disease [R, R, R]
- Diabetes [R]

Genetically higher levels of TMAO may be causally associated with:

- Gut inflammation [R]
- High blood pressure (systolic) [R]

LDL Particle Size

Research suggests that small LDL more easily goes into blood vessel walls, where it deposits as plaque, while larger particles tend to bounce off blood vessel walls. That's why small LDL may be worse, even though it carries less cholesterol than the larger particles [R, R, R, R, R].

Up to **50%** of differences in people's LDL particle size may be due to genetics [R].

Other factors that can raise small LDL levels include:

- Obesity [R]
- Physical inactivity [R, R]
- A diet high in simple carbs and saturated fat [R, R]



TYPICAL

Predisposed to typical LDL particle size based on 57 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ANGPTL4	rs116843064	GG
LPA	rs56393506	CC
APOH	rs1801689	AA
APOB	rs577584	TT
ADAM10	rs1077835	AA
NLRC5	rs12446515	CC
TMEM116	rs73412716	GG
SAMM50	rs3747207	GG
MLXIPL	rs71556736	CC
PKD2L1	rs603424	AA
SMARCA4	rs10412048	GA
APOE	rs157594	TT
IRS1	rs2943654	TT
KLF14	rs553015785	AA
TRIB1	rs2954025	CT
POLK	rs767676	CT
PLTP	rs4810479	TC
SLC38A11	rs13389219	TC
ABCA1	rs1883025	TC
TNXB	rs1061808	GT
MFHAS1	rs7012814	AG
PGS1	rs10642898	INS(CCG)C
APOE	rs7412	CC
PCSK9	rs11591147	GG
CD300LG	rs72836561	CC
PIGV	rs79598313	CC
SIDT2	rs964184	CC
SORT1	rs12740374	GG
VEGFB	rs11231698	CC

GENE	SNP	GENOTYPE
GCKR	rs1260326	CC
ALDH1A2	rs1601935	GG
SOAT2	rs28883710	TT
TRPS1	rs13262459	TT
BLK	rs34962960	GG
DSTN	rs2618566	GG

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

HDL Cholesterol

Cholesterol in the blood is carried by proteins, mainly LDL, HDL, and VLDL. We call cholesterol carried by HDL “good” cholesterol because it helps your liver get rid of excess cholesterol [R, R].

Normal to high levels of HDL cholesterol are linked to a lower risk of heart disease. Causes of low HDL cholesterol include [R, R, R]

- Being overweight or obese
- Smoking
- Metabolic syndrome
- A diet rich in trans fats
- Low physical activity

However, research has shown that medications that increase HDL cholesterol don't necessarily decrease one's risk of heart disease. In addition, genetic studies suggest that low HDL cholesterol levels probably don't cause heart disease on their own [R, R, R, R, R, R, R, R].

This is why doctors will often suggest lifestyle and diet strategies that can improve both your “good” and your “bad” cholesterol, as well as your heart health. These strategies include [R, R]:

- A healthy diet rich in fiber, and low in saturated fat and trans fat
- Exercise
- Losing weight
- Stopping tobacco use

Up to 65% of differences in cholesterol levels may be attributed to genetics. Genes that may affect HDL cholesterol influence [R, R, R, R, R, R]:

- Cholesterol production (*LPL*, *LIPC*, *HMGCR*)
- Cholesterol transport (*APOB*)
- HDL and LDL cholesterol balance (*CETP*)

Genetically high fasting insulin and total, bioavailable, and free testosterone may be causally associated with low HDL cholesterol [R, R, R].



TYPICAL LEVELS

Predisposed to typical HDL levels based on 916,929 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
/	rs200748895	TT
SCARB1	rs921919	AA
IRS1	rs2138161	CC
APOE	rs429358	CT
GPAM	rs2792751	CC
WDR11	rs10886863	CC
PLTP	rs6073958	TC
FADS1	rs174567	GA
APOB	rs2678379	AG
DGAT2	rs1219550	CT
/	rs71926466	DEL(T)G
ETV5	rs57912727	CA
CD300LG	rs72836561	CC
LPL	rs328	CC
ADAM10	rs2070895	GG
MCUB	rs189866430	TT
HNF4A	rs1800961	CC
ACAD10	rs11066015	GG
ADAL	rs150844304	AA
ARID1A	rs193084249	AA
SLC39A8	rs13107325	CC
ABCA1	rs2740488	AA
CYP27A1	rs17572799	AA
BTNL8	rs188238483	TT
ALPK2	rs41292412	CC
FLT3	rs76428106	TT
PLG	rs571848809	GG
TCF15	rs151235402	CC
ZNF653	rs737338	CC

GENE	SNP	GENOTYPE
/	rs56271783	GG
ABCA8	rs112001035	GG
RSPO3	rs72959041	GG
NELFCD	rs76602912	TT
PABPC4	rs17513135	CC
PPARG	rs12485478	AA
TSC22D2	rs9844972	GG
CNIH4	rs56105022	GG
UBE2L3	rs12158299	CC
KANSL1	rs117499775	TT
VEGFA	rs998584	CC
ST3GAL4	rs112771035	CC

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, 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 839 genetic variants we looked at

52nd

PERCENTILE



Your risk is **greater than 52% of the population** and **lower than 48% of the population.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
LPA	rs41267819	GG
LPA	rs76144756	GG
LPA	rs143431368	TT
SLC22A1	rs182980975	CC
LPA	rs41267809	AA
PLG	rs4252152	GG
PLG	rs145535174	AA
LPA	rs41272114	CC
SLC22A3	rs6919346	CC
LPA	rs4708871	TT
FADS2	rs1535	GA
CETP	rs5882	GA
LPA	rs74617384	AA
SLC22A3	rs3918291	TT
/	rs151135411	GG
LPA	rs142720914	GG
SLC22A3	rs117446263	GG
LPA	rs73596816	GG
LPA	rs3798220	TT
AGPAT4	rs61735260	GG
MRPL18	rs146888147	GG

GENE	SNP	GENOTYPE
SIDT2	rs964184	CC
TCF7L2	rs7903146	CC
GCKR	rs780094	CC
APOB	rs693	GG
PCSK7	rs662799	AA
APOA5	rs3135506	GG
LPA	rs10455872	AA
SLC22A1	rs146534110	GG
SLC22A3	rs118133674	GG
/	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.

Triglycerides

Triglycerides are the most common type of fat in the body. Some triglycerides are needed for the body to function. However, high levels can lead to health problems like artery hardening, stroke, heart disease, and pancreas inflammation [R, R, R, R, R].

About 1 in 3 adults in the US may have high triglycerides. This estimate is slightly higher in older adults. Over 4 in 10 people over 60 may be affected [R].

Many health conditions can lead to high triglycerides. These include [R, R]:

- Overweight or obesity
- Low thyroid hormones (*hypothyroidism*)
- Metabolic syndrome
- Diabetes
- Chronic kidney disease
- Autoimmune diseases like lupus
- HIV infection

To help lower triglycerides, doctors may recommend [R, R]:

- Diet changes
- Avoiding alcohol
- Exercise
- Weight loss
- Omega-3s
- Medication

Genetically higher triglyceride levels are likely causally associated with:

- Heart health [R, R, R, R]
- High blood pressure [R]
- Heart attack [R]
- Low mood [R, R]
- Gout [R]
- Kidney Health [R, R, R]
- Parkinson's (lower risk) [R]
- Bone health [R]
- Pancreas inflammation [R, R]
- Fatty liver [R]
- Age-related macular degeneration (lower risk) [R, R]
- Joint pain [R]
- Psoriasis [R]

Up to 45% of differences in people's triglyceride levels may be attributed to genetics. Genes involved in high triglycerides may influence fat metabolism. They include [R, R]:

- *BUD13*
- *APOC3*
- *APOA5*
- *GCKR*
- *LPL*
- *ZPR1*

Genetically higher fasting insulin may be causally associated with high triglycerides [R].

TYPICAL LEVELS

Predisposed to typical triglyceride levels based on 17,845 genetic variants we looked at

10th

PERCENTILE

Your risk is **greater than 10% of the population** and **lower than 90% of the population.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NLRC5	rs1800775	CC
APOC3	rs147210663	GG
FADS2	rs174546	TC
CETP	rs5882	GA
ACACB	rs149793040	AA
ANGPTL4	rs116843064	GG
APOA4	rs12721043	CC
PAFAH1B2	rs186808413	CC
LPL	rs75218485	CC
LPL	rs17091905	GG
LPL	rs117604010	GG
MLXIPL	rs71556711	CC
LPL	rs17489373	GG
MAU2	rs58542926	CC
MLXIPL	rs113296769	AA
APOB	rs533617	TT
DNAJC30	rs13242693	CC
CLPTM1	rs483082	TG
APOE	rs429358	CT
SIDT2	rs964184	CC
PCSK7	rs662799	AA

GENE	SNP	GENOTYPE
/	rs201079485	GG
/	rs149808404	GG
/	rs118204057	GG
PCSK7	rs5128	CC
LPL	rs268	AA
APOA5	rs3135506	GG
PLA2G12A	rs41278045	AA
APOA4	rs12721041	CC
BACE1	rs116987336	GG
LPL	rs1801177	GG
WSB2	rs7973260	GG
MAP1A	rs55707100	CC
LPL	rs186868868	CC
BUD13	rs117794084	GG
APOE	rs141622900	GG
A1CF	rs41274050	CC
APOE	rs7412	CC
EIF3J	rs151291132	AA
APOE	rs7254892	GG
SOST	rs76868109	AA
SLC30A3	rs116170113	GG
GTF3C2	rs149117895	CC
BUD13	rs114594921	TT
NECTIN2	rs138607350	TT

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

Effects Of Omega-3s On Triglycerides

Genetic studies revealed why omega-3 supplements may work better for some people than others. They identified key genetic variants influencing individuals' response to fish oil supplementation [R, R, R, R].

The studies found that variants in several genes - particularly GJB2, SLC12A3, ABCA6, MLXIPL, and APOE - affect how blood lipid levels change in response to fish oil [R, R, R].

Additionally, researchers developed a genetic risk score based on multiple variants that could partly predict **which individuals would respond positively to fish oil supplementation**. This suggests that genetic testing could help determine whether omega-3 supplements benefit a given individual [R].

These findings point toward more personalized approaches to nutrition, where genetic profiles could guide supplementation recommendations. However, the researchers note that additional studies are needed to fully validate these genetic markers across different populations and dosing protocols.



TYPICAL EFFECTS

Predisposed to typical effects of omega-3s on triglycerides based on 25 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SIK3	rs144018203	GG
SLIT2	rs2952724	CC
TADA2A	rs1714987	CC
CD36	rs1984112	AA
APOE	rs7412	CC
JADE1	rs931681	AA
ALDH8A1	rs6920829	CT
GJB2	rs112803755	AA
NELL1	rs752088	GA
PLA2G4A	rs1569480	GA
MAU2	rs141844019	CC
BAZ1B	rs117788606	TT
LPL	rs142084074	GG
NXPH1	rs6463808	GG
IQCJ	rs61332355	CC
MAP1A	rs55707100	CC
IQCJ	rs2621308	GG
JADE1	rs1216352	CC
IQCJ	rs1449009	AA
DDX39B	rs909253	AA
NT5C3B	rs8071753	GG
CD36	rs1761667	GG
JADE1	rs1216365	TT

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

VLDL Cholesterol

About 35% of the differences in people's VLDL levels may be due to genetics [R].

Other factors that can impact VLDL levels include:

- **Diet**: Diets high in refined sugars and saturated fats can lead to increased production of VLDL in the liver.
- **Physical Activity**: Regular exercise can help lower VLDL levels by burning triglycerides as energy.
- **Alcohol**: While moderate alcohol consumption might lower VLDL, excessive drinking can increase its levels.
- **Medications**: Some medications, like steroids, can elevate VLDL levels.
- **Health Conditions**: Conditions such as hypothyroidism, kidney disease, or poorly controlled diabetes can increase VLDL.
- **Obesity**: Carrying excess weight, especially around the abdomen, can lead to increased VLDL production.



TYPICAL LEVELS

Predisposed to typical VLDL levels based on 508 genetic variants we looked at

1st

PERCENTILE

Your risk is **greater than 1% of the population** and **lower than 99% of the population.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
LPA	rs140570886	TT
PCSK9	rs11591147	GG
APOE	rs1065853	GG
/	rs555766713	CC
LPA	rs142231215	GG
SLC16A11	rs186021206	GG
ANGPTL4	rs116843064	GG
MAU2	rs58542926	CC
BORCS8	rs150057262	CC
LPA	rs147555597	GG
LPA	rs76735376	GG
SORT1	rs12740374	GG
LPL	rs10096633	TC
MLXIPL	rs55747707	GG
TRIB1	rs112875651	GA
PCCB	rs34894639	CC
DOCK7	rs12239736	TA
ITIH4	rs13059141	GG
ALDH1A2	rs261290	TC
GLI1	rs10649122	AA
HP	rs7202323	TT

GENE	SNP	GENOTYPE
ABCG8	rs4299376	TG
SLC38A11	rs13389219	TC
ALDH1A2	rs1973688	TC
DOK7	rs13108218	AG
CCDC92	rs11057397	CT
CITED2	rs632057	TG
NCOR2	rs11057601	AG
TTC32	rs907866	GA
SIK3	rs45611741	CC
SIDT2	rs964184	CC
ADAM10	rs261342	CC
GCKR	rs1260326	CC
NSMAF	rs10504255	AA
CYP26C1	rs2478236	AA

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

LDL Particle Number (LDL-P)

Several factors can impact LDL-P levels:

- Diet: Diets high in saturated fats and trans fats can elevate LDL-P levels. Conversely, diets rich in fiber and unsaturated fats can help modulate them.
- Physical Activity: Engaging in regular cardiovascular exercise can positively influence LDL-P, leading to lower numbers of small, dense LDL particles, which are considered more atherogenic.
- Weight: Obesity, especially visceral fat, can be associated with increased LDL particle numbers.
- Insulin Resistance: Insulin resistance can lead to higher LDL-P levels, often seen in conditions like metabolic syndrome and type 2 diabetes.
- Medications: Statins and other lipid-lowering medications can influence LDL particle number and size. It's crucial to monitor these numbers when on such medications.
- Other Health Conditions: Hypothyroidism, chronic kidney disease, and certain inflammatory conditions can influence LDL-P levels.
- Genetics: There is a notable genetic component to LDL-P levels. Certain genetic variants and mutations have been linked to differences in LDL particle number and size among individuals.



TYPICAL

Predisposed to typical LDL-P based on 5 genetic variants we looked at

75th

PERCENTILE



Your risk is **greater than 75% of the population** and **lower than 25% of the population.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CD36	rs1761665	CC
GNAT3	rs6970109	AC
CD36	rs7755	GA
CD36	rs3211842	AA
CD36	rs1722507	AA

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

Homocysteine

Key Takeaways:

- About 55% of differences in people's homocysteine levels may be due to genetics.
- High homocysteine is usually caused by the lack of vitamins B12, B9 (folate), and B6.
- People with heart disease and cognitive problems tend to have higher homocysteine.
- High homocysteine may not be a risk factor for heart disease.

Homocysteine is a metabolic byproduct linked to heart disease and cognitive decline. It's cleared out of our bodies with the aid of **vitamins B12, B9 (folate), and B6** [R].

A deficiency of vitamins B12, B9 (folate), or B6 is the most common cause of high homocysteine levels [R].

Homocysteine can also be increased by:

- Stress [R, R, R]
- Cigarette smoke [R, R]
- Alcohol [R, R]
- Certain medications [R, R, R, R, R, R, R]

About 55% of differences in people's homocysteine levels may be due to genetics. Unsurprisingly, involved genes like *MTHFR* play a role in the metabolism of folate and other B vitamins [R].

Gene variants that increase homocysteine levels may also play a role in schizophrenia and some types of cancer [R, R, R, R, R].

If you are worried about your genetic results, make sure to check your homocysteine lab marker to see your actual levels. It may also be a good idea to check your genetics and levels of folate and other B vitamins.



TYPICAL LEVELS

Predisposed to typical homocysteine levels based on 24 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MTRR	rs1801394	GA
NOX4	rs7130284	CC
SLC19A1	rs1051266	CC
C12ORF43	rs2251468	CC
TCN2	rs1801198	CC
SPATA2L	rs154657	GA
NOX4	rs957140	GG
CPS1	rs1047891	CA
MTR	rs2275565	GT
TRDMT1	rs12780845	GA
CBS	rs2851391	CC
ZDHHC20	rs17356983	AG
COLEC12	rs621636	CT
RNF175	rs2404916	AG
MTHFR	rs1801133	GG
C1ORF167	rs12134663	AA
FANCA	rs12921383	TT
CBS	rs234709	TT
H2BC5	rs548987	GG
MTR	rs28372871	TT
CUBN	rs1801222	GG
MMUT	rs9369898	GG
AKR1A1	rs4660306	CC
FGF21	rs838133	GG
GTPBP10	rs42648	AA
SYT6	rs79079833	TT
CSMD1	rs17394429	GG
TAFA5	rs13054085	GG
EDNRA	rs1429107	GG

GENE	SNP	GENOTYPE
NOX4	rs10830265	GG

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

Lp-PLA2

Up to **35%** of the differences in Lp-PLA2 may be due to **genetics** [R].

Some individuals may have a genetic predisposition to produce higher amounts of Lp-PLA2. This can contribute to a higher risk of cardiovascular diseases. However, the precise genetic factors influencing Lp-PLA2 levels are still under research.

Other factors influencing Lp-PLA2 levels include:

- **Age**: Levels might increase with age
- **Smoking**

Additionally, the following health conditions are also associated with elevated Lp-PLA2 levels:

- **Artery hardening (atherosclerosis)**
- High cholesterol
- Inflammation
- Diabetes
- High blood pressure



TYPICAL LEVELS

Predisposed to typical Lp-PLA2 levels based on 16 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
LDLR	rs6511720	GG
APOB	rs6413458	GG
APOC1	rs445925	AG
SORT1	rs7528419	AA
SORT1	rs12740374	GG
SCARB1	rs11057841	TT
PLA2G7	rs1805017	CT
MS4A6A	rs600550	CC
PLA2G7	rs7756935	CA
PLA2G7	rs1362931	AC
GCKR	rs1260326	CC
VMP1	rs11650106	TC
APOE	rs4420638	AA
SIDT2	rs964184	CC
NLRC5	rs247616	CC
NLRC5	rs3764261	CC

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

Platelet Aggregation

About 45-60% of the differences in platelet aggregation may be due to genetics [R].

Various gene variants have been linked to differences in platelet function, reactivity, and aggregation propensity. Heritability studies suggest a considerable genetic influence on platelet aggregation and responsiveness to antiplatelet therapies.

Other factors that can affect platelet aggregation include:

- Medications: Antiplatelet drugs, like aspirin and clopidogrel, are specifically designed to inhibit platelet aggregation. Conversely, certain medications might enhance platelet activity.
- Diet: Foods rich in omega-3 fatty acids, like fish, can reduce platelet aggregation. On the other hand, excessive alcohol or caffeine might increase it.
- Smoking: Tobacco smoking can increase platelet aggregation and is a risk factor for cardiovascular diseases.
- Stress: Acute or chronic stress can promote platelet aggregation.
- Medical Conditions: Diseases like diabetes and conditions associated with inflammation can increase platelet aggregation.
- Surgery or Trauma: Any form of vascular injury can stimulate platelet aggregation as a natural response to halt bleeding.
- Hormonal Changes: Some studies suggest that hormones, such as those fluctuating during menstrual cycles, can influence platelet function.



TYPICAL

Predisposed to typical platelet aggregation based on 16 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CSMD2	rs115780313	TT
IRAG1	rs4909945	CC
GP6	rs1671152	GT
REEP3	rs10761731	AA
CCDC71L	rs342293	GG
REEP3	rs10761779	AA
IRAG1	rs7940646	CC
ADRA2A	rs869244	AG
DTWD2	rs2914908	CC
SHH	rs6943029	GA
FGFR1	rs7832232	AG
TSSK2	rs1052763	TC
ADRA2A	rs4545476	TC
PEAR1	rs12566888	GG
MICB	rs9267673	CC
HLA-B	rs2263316	GG

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

Platelets

Common reasons for low platelets include [R, R]:

- Some viral infections
- Liver disease
- Medications
- Some autoimmune disease

Having low platelets will usually not cause symptoms. If platelet levels become low enough, you may notice [R]:

- Nosebleeds
- Bleeding gums
- Heavy menstrual periods
- Easy bruising
- Tiny red or purple dots on the lower legs that resemble a rash (*petechiae*)

Common reasons for high platelets include [R]:

- Iron deficiency
- Infections
- Inflammatory disorders
- Injury or surgery

Having high platelets usually doesn't cause symptoms. It is often noticed by chance on lab tests. High platelet levels may increase the risk of developing blood clots [R].

Most of the time, treating the underlying condition will normalize high or low platelets [R, R].

Up to 85% of differences in people's platelet levels may be due to genetics. Genes involved may influence [R]:

- Communication between platelets
- Platelet shape
- Bone marrow function



TYPICAL LEVELS

Predisposed to typical platelet count based on 47,854 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
KCNT2	rs577885731	TT
ANKRD16	rs188131767	GG
NEK7	rs148230727	TT
RHOU	rs73100748	TT
GPR83	rs116420142	CC
IZUMO1R	rs115902890	TT
RNF2	rs149846493	TT
LRRTM3	rs143620141	GG
NEXN	rs150511446	TT
RBM14-RBM4	rs574184969	TT
SWT1	rs187723693	GG
DPH5	rs145201041	CC
LRRTM3	rs116282985	TT
RBBP5	rs16855138	TT
GCSAML	rs56043070	GG
CTNNA3	rs189718953	CC
GCSAML	rs149685951	GG
USP24	rs115527178	AA
PTPRJ	rs115849281	CC
C11ORF49	rs142924457	GG
DDB2	rs115443197	CC
OR2W3	rs142455309	TT
ACTA2	rs115020109	GG
ZNF496	rs143904230	GG
EEF1AKNMT	rs78795075	TT
NNMT	rs111371029	CC
CH25H	rs77086884	CC
DNM3	rs76257386	GG
DNM3	rs58679315	GG

GENE	SNP	GENOTYPE
VAMP4	rs563948913	GG
TMCC2	rs147728054	CC
DSTYK	rs148495065	GG
JMJD1C	rs61853560	GG
DSTYK	rs116125187	GG
EEF1AKNMT	rs79744378	GG
LPXN	rs145169535	CC
DSTYK	rs149878760	TT
DSTYK	rs143419712	GG
JMJD1C	rs11593336	CC
JMJD1C	rs563511831	AA

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

Response To Ezetimibe

The response to ezetimibe can vary based on several factors:

- Adherence to treatment: As with all medications, consistent adherence to the prescribed treatment regimen is crucial for effectiveness.
- Diet: A diet high in cholesterol can counteract the effects of ezetimibe, as the drug works by reducing the absorption of dietary cholesterol.
- Use with other medications: The cholesterol-lowering effects of ezetimibe are enhanced when combined with statins or other lipid-lowering treatments.
- Genetics

Variants in the *NPC1L1* gene may affect the effectiveness of ezetimibe at lowering cholesterol levels. For instance, a study of 101 dyslipidemic subjects tested the effects of three different polymorphisms on response to ezetimibe [R]:

- rs2072183
- rs217428
- rs217434

The study found that 1 out of 8 participants were homozygous for the minor variant of all three polymorphisms ('C' at rs2072183, 'G' at rs217428, and 'G' at rs217434). People with this genotype lowered their LDL cholesterol levels in response to ezetimibe by 49% more (35.2 vs 23.6 mg/dL) compared to those with any other genotypes.

Please note: The combination of ezetimibe with statins is not recommended in people with liver disease or those with increased liver transaminase levels. There are no clinical studies with ezetimibe in pregnant or breastfeeding women, or children younger than 10 years old. Therefore, this drug is not recommended in these situations. Always consult your doctor before starting or stopping any medication [R, R, R, R, R, R, R].



TYPICAL

Likely typical response to ezetimibe based on the genetic variants we looked at

88%

OF USERS SHARE THE SAME SCORE



You have the same genetic predisposition as **88% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NUDCD3	rs217428	TT
NUDCD3	rs217434	AA
NPC1L1	rs2072183	CG

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

ApoB

Key takeaways:

- ApoB is a protein that forms part of "bad cholesterol".
- High ApoB levels are linked to heart disease.
- Genetically high ApoB may be involved in the development of heart disease and Alzheimer's disease.
- Up to **70%** of differences in people's ApoB levels may be due to **genetics.**
- Besides genetics, different lifestyle factors, health conditions, and drugs can affect ApoB levels.

Apolipoprotein B (ApoB) is a large protein that helps build three major lipoproteins, collectively called "bad cholesterol" [R].

Up to **70%** of differences in people's ApoB levels may be due to **genetics**. Involved genes may influence ApoB production [R, R, R].

Some factors that may lead to **high ApoB** include:

- High-sugar foods [R, R]
- Diets high in saturated fats and cholesterol [R, R, R]
- Smoking [R, R, R]
- Drinking coffee (including decaf) [R, R, R]
- Sleep deprivation [R, R]
- Some drugs (e.g., steroids, chemotherapy) [R, R, R, R]

ApoB levels may increase during pregnancy and menopause. Some health conditions may also lead to high ApoB levels, including [R, R, R]:

- Obesity [R, R, R]
- Underactive thyroid [R, R, R]
- Diabetes [R, R, R]
- Liver and kidney disease [R, R, R, R, R]
- Rare genetic disorders [R, R, R, R]

Low ApoB levels may result from:

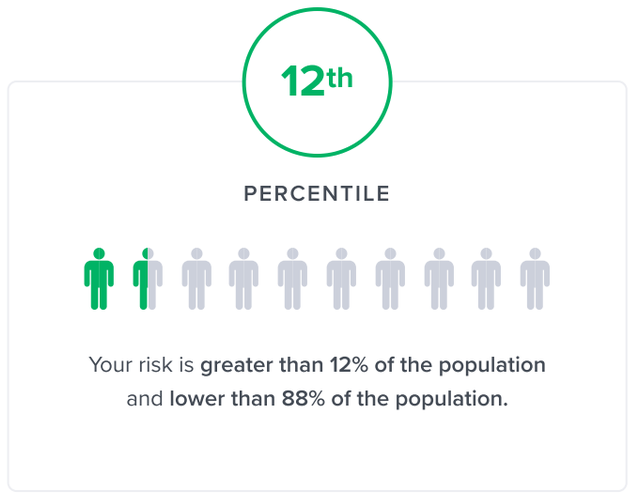
- Overactive thyroid [R, R, R]
- Liver disease [R, R, R, R]
- Some drugs (e.g., cholesterol-lowering drugs) [R, R, R, R, R, R, R]
- Rare genetic disorders [R, R, R, R, R]

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



LOWER LEVELS

Predisposed to lower ApoB levels based on 6,389 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CLPTM1	rs490243	TC
TOMM40	rs394819	TG
TOMM40	rs157587	GA
APOC1	rs389261	AG
NECTIN2	rs28399637	GA
APOB	rs550619	GA
/	rs12713559	GG
APOC4	rs140526515	AA
NECTIN2	rs138914864	CC
/	rs151135411	GG
APOE	rs769449	GG
NECTIN2	rs117310449	CC
NECTIN2	rs144261139	CC
NECTIN2	rs76366838	GG
APOE	rs4420638	AA
CLPTM1	rs12691088	GG
NECTIN2	rs34095326	GG
NECTIN2	rs41289512	CC
NECTIN2	rs138607350	TT
PVR	rs35959395	GG
PVR	rs139267469	CC

GENE	SNP	GENOTYPE
APOC2	rs10424663	GG
APOC1	rs60049679	GG
BCL3	rs114036675	GG
LPA	rs3798220	TT
SNX8	rs144787122	AA
SLC22A3	rs3918291	TT
APOE	rs157599	AA
MAFB	rs2207132	GG
LPA	rs74617384	AA
TRAPPC6A	rs142501705	AA
A1CF	rs41274050	CC
CLPTM1	rs79429216	GG
APOA5	rs3135506	GG
TDRD15	rs113588790	CC
ABCA5	rs75016991	CC
SLC22A1	rs2282143	CC
APOA4	rs12721041	CC
TDRD15	rs116157399	GG
TDRD15	rs111548358	TT

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

Metabolic Syndrome

Factors that might increase the risk of developing metabolic syndrome include:

- Age: Risk increases with age.
- Obesity, particularly abdominal obesity.
- Insulin resistance.
- A history of diabetes in one's family.
- A history of gestational diabetes or having given birth to a baby weighing more than 9 pounds.
- Other diseases: A history of nonalcoholic fatty liver disease, polycystic ovary syndrome, or having had a cardiovascular disease or stroke.
- Hormonal imbalance, like low testosterone in men.
- Lack of physical activity.
- An unhealthy diet high in fats and sugars.
- Genetics

Genetics plays a significant role in metabolic syndrome. Specific genetic factors might make certain individuals more susceptible to the conditions that contribute to metabolic syndrome. Family history, particularly if parents or siblings have had diabetes, heart disease, or a stroke, can be an indicator of increased risk.



LESS LIKELY

Less likely to have metabolic syndrome based on 636,870 genetic variants we looked at

15th

PERCENTILE



Your risk is **greater than 15% of the population** and **lower than 85% of the population.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MC4R	rs17782313	CT
LPL	rs328	CC
GALNT2	rs2281721	CC
FTO	rs56094641	GG
TRIB1	rs2980888	TC
CLPTM1	rs483082	TG
SNX10	rs1534696	CC
HMGA1	rs76376137	TG
FADS2	rs1535	GA
GNGT2	rs11655056	CC
MC4R	rs66922415	GA
C1QTNF4	rs7124681	AC
KLF14	rs10260148	TC
BPTF	rs11871285	TG
HLA-C	rs9378248	GA
GSR	rs10954772	TC
RPL17	rs1105654	GA
MLLT10	rs9971210	CG
TCF7L2	rs7903146	CC
ADRB3	rs4994	AA
MTNR1B	rs10830963	CC

GENE	SNP	GENOTYPE
SIDT2	rs964184	CC
CD300LG	rs72836561	CC
PCSK7	rs662799	AA
SIDT2	rs651821	TT
ADAL	rs139974673	TT
ARAP2	rs73123462	CC
ZDHHC18	rs114165349	GG
HNF4A	rs1800961	CC
WSB2	rs7973260	GG
MLXIPL	rs12056034	AA
RSPO3	rs577721086	TT
ILRUN	rs11754773	AA
PPP1R3B	rs9987289	GG
SLC39A8	rs13107325	CC
VEGFA	rs998584	CC
PABPC4	rs11206374	GG
PLG	rs11751347	CC
CMIP	rs2925979	CC
GCKR	rs1260326	CC
ATP1B2	rs1143015	GG
SEC16B	rs10913469	TT
SNX15	rs35661464	CC
TUBG2	rs12945575	CC
LIN7C	rs56133711	GG
NAT2	rs4921913	TT
INO80E	rs3814883	CC
TRPS1	rs3808439	GG
GAD1	rs12472667	CC
HLA-DQA2	rs5021727	AA

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

Response To Statins (Functional)

The following factors may affect the effectiveness of statins in lowering cholesterol, as well as the risk of adverse effects from these medications:

- **Statin type and dosage:** Different types of statins (e.g., atorvastatin, simvastatin, rosuvastatin) vary in potency, half-life, and side-effect profile. For instance, high-dose atorvastatin or rosuvastatin may provide more effective cholesterol reduction compared to older statins like simvastatin. Adjusting the statin dose can also impact individual responses, as higher doses may offer greater cholesterol reduction but also increase the likelihood of side effects like muscle pain (myopathy) or liver enzyme abnormalities.
- **Age and gender:** Older adults may have a different response to statins due to age-related changes in liver function and metabolism. In addition, some studies suggest that women may experience more frequent or severe side effects compared to men, possibly due to differences in muscle mass, hormonal influences, or pharmacokinetics.
- **Diet and lifestyle:** Dietary factors, particularly the intake of fats and alcohol, can affect how well statins work. A diet rich in healthy fats and low in processed sugars can enhance the effectiveness of statins, while excessive alcohol consumption or poor diet can increase the likelihood of adverse effects. Regular exercise is also associated with a better response to statin therapy.
- **Health status:** Individuals with other health conditions such as diabetes, liver disease, or kidney dysfunction may have altered responses to statins. For example, people with diabetes may have a less favorable lipid-lowering effect from statins, and those with liver or kidney impairments may be at higher risk for adverse reactions.
- **Genetics:** Genetic variations play a significant role in how effectively a person responds to statins. Polymorphisms in genes involved in cholesterol metabolism, such as *HMGCR*, *SLCO1B1,* and *PCSK9,* can influence statin efficacy and the risk of side effects. For example, variations in the *SLCO1B1* gene can affect statin uptake in the liver, leading to reduced drug efficacy or increased risk of muscle-related side effects.



BETTER RESPONSE

Predisposed to better response to statins based on 19 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
APOE	rs7412	CC
APOC1	rs445925	AG
SORT1	rs7528419	AA
DZIP1	rs12428035	CC
CEMIP	rs11638450	CC
KIF6	rs20455	GG
LPL	rs1801177	GG
LPA	rs3124784	GG
PCSK9	rs505151	AA
PCSK9	rs11591147	GG
LPA	rs10455872	AA
SORT1	rs646776	TT
SLCO1B1	rs73079476	AA
SLCO1B1	rs58310495	CC
LIMCH1	rs7696430	GG
APOB	rs1713222	AA
LDLR	rs67337506	CC
SLCO1B1	rs4149056	TT
LPA	rs41267807	TT
SLCO1B1	rs2900478	TT
HMGCR	rs17238540	TT
RNF175	rs981844	GG

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