SelfDecode

Clinical Overview Report

Report date: 28 July 2025

Client Information

Name:	Sample Client
Age:	40
Sex:	Male
Height:	5ft 9" / 175.0cm
Weight:	165lb / 75.0kg

Table of Contents

Medical Reports

Your DNA is like an instruction manual — it contains a lot of information. You can think of it as a blueprint for your body. Genetic variants are parts of DNA that differ from person to person. Some can make you more vulnerable to certain health issues. We use artificial intelligence and machine learning to analyze all this information. We then summarize your results as a risk score. **In total, we analyze up to 83 million genetic variants.**

When we give a risk score, the risk icon tells you if you are at a higher or lower risk compared to other people:

⚠ **High & Impactful**   ！ **High**   ✓ **Typical**   ✓ **Low**

When applicable, we also list top evidence-based recommendations that may help lower your risk. The focus is on recommendations that may be of benefit to you, based on your genetics.

Our recommendations come in four categories: lifestyle, diet, supplements and drugs. The following icons tell you which category a recommendation falls into:

🌱 **Dietary**   💓 **Lifestyle**   ⚗ **Supplement**   💊 **Drug**

Our team of scientists also ranks each recommendation. We rank based on impact and the strength of evidence in the medical literature.

Impact shows how strongly a recommendation will affect your health in a certain area. Evidence is how much scientific support there is for the recommendation. Rankings are from 1 to 5 (low to high).

Risks Overview

Risk	Condition
⚠️	Prostate Cancer
⚠️	Glaucoma
⚠️	Bipolar Disorder
⚠️	Colorectal Cancer
⚠️	Parkinson's Disease
✓	Coronary Artery Disease
✓	Crohn's Disease
✓	Psoriasis
✓	Melanoma
✓	Alzheimer's Disease
✓	Asthma
✓	Ulcerative Colitis
✓	Stroke
✓	Schizophrenia
✓	Venous Thromboembolism
✓	Cardiovascular Disease
✓	Multiple Sclerosis
✓	High Blood Pressure
✓	Type 2 Diabetes
✓	Age-Related Macular Degeneration
✓	Celiac Disease
✓	Rheumatoid Arthritis
✓	Atrial Fibrillation
✓	Osteoporosis

Risk	Condition
✓	Type 1 Diabetes
✓	Lupus

Recommendations Overview

#	Type	Recommendation	Helps With These
1		Mediterranean Diet	Colorectal Cancer, Coronary Artery Disease, Crohn's Disease, Parkinson's Disease, Prostate Cancer, Psoriasis
2		Methylfolate	Bipolar Disorder, Colorectal Cancer, Coronary Artery Disease, Crohn's Disease, Parkinson's Disease, Psoriasis
3		Strength Training	Colorectal Cancer, Coronary Artery Disease, Parkinson's Disease
4		Whole-Food Plant-Based Diet	Colorectal Cancer, Coronary Artery Disease, Prostate Cancer
5		Green Tea	Colorectal Cancer, Coronary Artery Disease, Prostate Cancer
6		Avoid Organochlorine Pesticide Exposure	Colorectal Cancer, Coronary Artery Disease, Parkinson's Disease, Prostate Cancer
7		Cruciferous Vegetables	Colorectal Cancer, Coronary Artery Disease, Prostate Cancer
8		Maintain Optimal Vitamin D Levels	Bipolar Disorder, Colorectal Cancer, Crohn's Disease, Parkinson's Disease, Prostate Cancer, Psoriasis
9		Walking	Bipolar Disorder, Colorectal Cancer, Coronary Artery Disease, Parkinson's Disease
10		Eat Fiber-Rich Foods	Colorectal Cancer, Coronary Artery Disease, Crohn's Disease
11		Garlic Supplement	Colorectal Cancer, Coronary Artery Disease, Prostate Cancer
12		Avoid Air Pollution	Colorectal Cancer, Coronary Artery Disease, Parkinson's Disease
13		Walnuts	Colorectal Cancer, Coronary Artery Disease, Prostate Cancer
14		Avoid Secondhand Smoke	Coronary Artery Disease, Prostate Cancer, Psoriasis
15		Tomato	Coronary Artery Disease, Prostate Cancer

Lab Results Overview

Risk	Lab Marker	Optimal Range	Result	Related To These
⚠	**Lead, Blood**	0 ug/L - 10 ug/L	**400 ug/L**	Coronary Artery Disease, High Blood Pressure, Osteoporosis, Rheumatoid Arthritis, Stroke
⚠	**Creatinine**	0.7 mg/dL - 1.1 mg/dL	**1.4 mg/dL**	Atrial Fibrillation, Coronary Artery Disease, Lupus
⚠	**Hematocrit**	40 % - 47 %	**57.5 %**	Coronary Artery Disease, Crohn's Disease, Ulcerative Colitis
⚠	**LDL/HDL Ratio**	0.5 x100% - 2.3 x100%	**7.13 x100%**	Coronary Artery Disease
⚠	**eGFR**	90 mL/min/1.73 m2 - 120 mL/min/1.73 m2	**50 mL/min/1.73 m2**	Atrial Fibrillation, Coronary Artery Disease
!	**AST**	17 IU/L - 30 IU/L	**14 IU/L**	Alzheimer's Disease, Type 2 Diabetes
!	**Lymphocytes (%)**	35 % - 55 %	**31 %**	Asthma
!	**Valine, Plasma**	180 mcmol/L - 313 mcmol/L	**143.43 mcmol/L**	Alzheimer's Disease, Coronary Artery Disease, High Blood Pressure
!	**Testosterone, Total**	500 ng/dL - 1100 ng/dL	**300 ng/dL**	Alzheimer's Disease, Celiac Disease, Coronary Artery Disease, Osteoporosis, Rheumatoid Arthritis, Stroke, Type 2 Diabetes
!	**Vitamin C**	0.85 mg/dL - 1.2 mg/dL	**0.262 mg/dL**	Alzheimer's Disease, Coronary Artery Disease, Type 2 Diabetes
!	**Platelet Count**	200 K/cumm - 270 K/cumm	**150 K/cumm**	Atrial Fibrillation, Coronary Artery Disease, High Blood Pressure, Lupus, Rheumatoid Arthritis, Stroke, Ulcerative Colitis
!	**LDL Cholesterol**	50 mg/dL - 100 mg/dL	**124 mg/dL**	Coronary Artery Disease, High Blood Pressure, Osteoporosis, Parkinson's Disease, Stroke, Type 2 Diabetes
!	**Triglycerides**	0 mg/dL - 90 mg/dL	**134 mg/dL**	Age-Related Macular Degeneration, Coronary Artery Disease, High Blood Pressure, Osteoporosis, Parkinson's Disease, Psoriasis
!	**SHBG**	30 nmol/L - 45 nmol/L	**51 nmol/L**	Asthma, Coronary Artery Disease, High Blood Pressure, Osteoporosis, Rheumatoid Arthritis, Schizophrenia, Stroke, Type 2 Diabetes
!	**RDW**	11.5 % - 13 %	**13.6 %**	Stroke

SelfDecode

Prostate Cancer

Your Lifetime Risk	⚠ High

Your 10-Year Risk	✓ Normal

Your results are indicating a High risk of developing in your lifetime and within the next decade. Monitor your risk by regularly checking your related labs and implementing the recommendations provided.

DNA Risk Factors

Polygenic Risk Score based on 1,049,413 variants	⚠ 94 percentile │ More Likely

Other Risk Factors

Lifestyle Risks	⚠ Detected
Question	**Answer**
Do you have a parent or sibling who has ever been diagnosed with prostate cancer?	**Yes**

Common Variants That Increase Risk

Gene	SNP	Genotype
FSTL5	**rs7691481**	**CC**

Summary

The exact cause of prostate cancer is not clearly understood, but several factors have been identified that increase the risk of developing this disease [R]:

- Age: The risk increases significantly after age 50, and it is most common in men over 65.
- Family history: Having a father or brother with prostate cancer more than doubles a man's risk.
- Race/Ethnicity: African-American men have a higher risk of prostate cancer than men of other races. They are also more likely to develop prostate cancer at an earlier age and have more aggressive tumors.
- Genetics: Genetic changes, including mutations in the *BRCA1* and *BRCA2* genes, which are also linked to breast and ovarian cancer in women, can increase risk.
- Diet: A diet high in red meat or high-fat dairy products and low in fruits and vegetables might increase the risk, although studies are not conclusive.

Treatment options vary depending on the stage of the cancer and other factors, including the patient's overall health and personal preferences [R]:

- Active surveillance: For low-risk cancers, monitoring the cancer closely with PSA tests, rectal exams, and ultrasounds may be recommended until tests show the cancer is growing.
- Surgery: Radical prostatectomy involves removing the prostate gland and some of the surrounding tissue.
- Radiation therapy: This can be used both as an initial treatment for cancer that has not spread beyond the prostate and as a way to relieve symptoms of advanced cancer.

- Hormone therapy: Also known as androgen deprivation therapy (ADT), aims to reduce levels of male hormones, androgens, which can stimulate the growth of prostate cancer cells.
- Chemotherapy: Used for more advanced prostate cancer that has spread to other parts of the body and does not respond to hormone therapy.
- Targeted therapy and immunotherapy: Newer forms of treatment that target specific aspects of cancer cells or utilize the body's immune system to fight the cancer.

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

SelfDecode

Glaucoma

Your Lifetime Risk	⚠ High

Your 10-Year Risk	✓ Normal

Your results are indicating a High risk of developing in your lifetime and within the next decade. Monitor your risk by regularly checking your related labs and implementing the recommendations provided.

DNA Risk Factors

Polygenic Risk Score based on 1,049,432 variants	⚠ 78 percentile ｜ More Likely

Other Risk Factors

Lab Risks		⚠ Detected
Lab Marker	**Optimal Range**	**Result**
Copper, Serum	70 mcg/dL - 105 mcg/dL	**107.15 mcg/dL**

Common Variants That Increase Risk

Gene	SNP	Genotype	Gene	SNP	Genotype
DISP3	rs149002763	**CC**	LOXL1	rs1550437	**CC**
CDKN2B	rs10120806	**CC**	AFAP1	rs6811851	**CC**
IKZF2	rs62186461	**TT**	FNDC3B	rs16845236	**GG**
VCAM1	rs148843724	**TT**	FOXC1	rs2745572	**AA**

Summary

Key Takeaways:

- Up to **50%** of the differences in people's chances of getting glaucoma may be due to genetics.
- Other risk factors include internal eye pressure, age, race, eye injury, and high corticosteroid use.
- About **3 million** Americans have glaucoma, and about **2.5%** of the world's population.
- Symptoms include loss of peripheral or central vision, headache, eye pain, halos, eye redness, and nausea.
- If your genetic risk is high, your overall risk is still low until older age, (about 1% at 40, up to 9% at 80).

Risk factors for glaucoma include the following [R]:

- High eye pressure (intraocular pressure)

- Age over 55
- African-American, Asian, or Hispanic race
- Medical conditions such as diabetes, migraines, and high blood pressure
- Eye injury or certain types of eye surgery
- Taking corticosteroid medicines, especially eye drops, for a long time
- **Genetics**

Up to **50%** of the differences in people's chances of getting glaucoma may be due to genetics [R].

SelfDecode

Bipolar Disorder

Your Lifetime Risk	⚠ High

Your 10-Year Risk	⚠ High

Your results are indicating a High risk of developing in your lifetime and within the next decade. Monitor your risk by regularly checking your related labs and implementing the recommendations provided.

DNA Risk Factors

Polygenic Risk Score based on 1,044,121 variants	⚠ 75 percentile │ More Likely

Other Risk Factors

Lab Risks		⚠ Detected
Lab Marker	**Optimal Range**	**Result**
Ferritin	80 ng/mL - 193 ng/mL	**230 ng/mL**
Copper, Serum	70 mcg/dL - 105 mcg/dL	**107.15 mcg/dL**

Common Variants That Increase Risk

Gene	SNP	Genotype	Gene	SNP	Genotype
DUSP28	**rs2953145**	**CC**	BCR	**rs131690**	**GG**
DCTN5	**rs420259**	**AA**	CMTM8	**rs4276227**	**CC**
THRA	**rs2314339**	**CC**	BCR	**rs131702**	**GG**
HES6	**rs2304672**	**GG**	TDRD9	**rs11622475**	**CC**

Summary

Key Takeaways:

- Up to **80%** of differences in people's chances of developing bipolar disorder may be due to genetics.
- Risk factors: being female, childhood bullying, excessive social media use, stressful events, and alcohol/drug abuse.
- If you have high genetic risk or symptoms, you may want to take action on modifiable risk factors to reduce your overall risk.
- Click the **Recommendations** tab for potential dietary and lifestyle changes and **next steps** for relevant labs.

Anger, sadness, and joy are everyday human experiences. It's normal to feel a wide range of emotions. **However, some people experience extreme changes in emotions that interfere with their lives.** These are called **mood swings**, and they can be a symptom of a deeper problem.

One cause of mood swings is bipolar disorder. This condition causes mood changes that are severe enough to affect daily life. It can also cause shifts in energy, focus, and ability to perform basic tasks [R, R, R].

People with bipolar disorder have periods of high energy and good mood followed by periods of low energy and poor mood. **These 'up' periods are called *manic* episodes, and the 'down' periods are called *depressive* episodes**. **Some people experience less extreme highs called *hypomanic* episodes** [R, R].

Other conditions that can cause mood swings include [R, R, R]:

- Personality disorders (e.g., borderline personality disorder)
- Premenstrual syndrome (PMS)

About **2-3%** of people may develop some form of bipolar disorder during their lifetime. Most people develop it as teens or young adults [R].

Women are more likely to develop bipolar disorder than men. Other risk factors include [R, R]:

- Childhood bullying
- Excessive social media use
- Stressful or traumatic events
- Alcohol or drug abuse
- **Genetics**

Bipolar disorder can have negative effects on a person's life. It can increase the risk of [R, R]:

- Alcohol or drug abuse
- Other health conditions (e.g., obesity, heart disease, or diabetes)
- Self-harm
- Relationship problems
- Financial issues
- Poor performance at work or school

It is important to work with your doctor and find appropriate ways to manage bipolar disorder. Management options include [R]:

- Medication
- Talk therapy
- Lifestyle changes, such as regular exercise
- Brain stimulation therapies

People with untreated mood disorders are considerably more likely to harm themselves and even die by suicide. If you are diagnosed with a mood disorder, it is essential to follow your doctor's treatment plan [R].

About 80% of differences in people's chances of developing bipolar disorder may be attributed to genetics. Genes involved in bipolar disorder may influence [R, R, R]:

- Brain activity (*DAOA*, *BDNF*, *CACNA1C*, *SCN2A*)
- Nerve inflammation (*CD47*)
- Serotonin levels (*SLC6A4*)

Genetically high levels of omega-3s may be causally associated with a lower risk of mood swings [R].

Colorectal Cancer

Your Lifetime Risk	⚠️ High

Your 10-Year Risk	✓ Normal

Your results are indicating a High risk of developing in your lifetime and within the next decade. Monitor your risk by regularly checking your related labs and implementing the recommendations provided.

DNA Risk Factors

Polygenic Risk Score based on 1,049,410 variants	⚠️ 73 percentile | More Likely

Common Variants That Increase Risk

Gene	SNP	Genotype	Gene	SNP	Genotype
ZPLD1	rs116265807	GG	MYL2	rs17550549	CC
MYH3	rs1078643	AA	PRICKLE1	rs11610543	GG
ACTL8	rs11203467	GG	LRP1	rs7398375	CC
COX7A2L	rs13426988	GG	GRM7	rs2163735	GG

Summary

While the exact cause of colorectal cancer is not fully understood, several factors increase the risk of developing this disease [R]:

- Age: The majority of cases occur in people aged 50 and older, though incidence rates are rising among younger populations.
- Family history: Having a family history of colorectal cancer or polyps increases one's risk.
- Personal history: Those with a history of inflammatory bowel disease (like Crohn's disease or ulcerative colitis), or who have had colorectal cancer or adenomatous polyps before are at higher risk.
- Genetic syndromes: Genetic mutations passed through generations, such as familial adenomatous polyposis (FAP) and hereditary non-polyposis colorectal cancer (Lynch syndrome), significantly increase the risk.
- Lifestyle factors: A diet high in red or processed meats, physical inactivity, obesity, smoking, and heavy alcohol use are known risk factors.
- Racial and ethnic background: African Americans have a higher incidence rate of colorectal cancer than other racial groups in the United States.

Treatment for colorectal cancer depends on the stage of the disease, the location of the tumor, and the patient's overall health [R]:

- Surgery: The primary treatment for localized cancer, surgery involves removing the tumor and surrounding tissue. For some cases, resection of part of the colon or rectum may be necessary.
- Chemotherapy: Used before or after surgery to shrink tumors and kill any cancer cells that may remain.
- Radiation therapy: Often used alongside chemotherapy, especially for rectal cancer, to reduce tumor size before surgery or eliminate remaining cells postoperatively.
- Targeted therapy: Drugs that target specific abnormalities in cancer cells. It's used for cancers that have specific gene mutations.
- Immunotherapy: Uses the body's immune system to fight cancer. It's typically reserved for advanced colorectal cancer.

Preventive measures include:

- Regular screening: Beginning at age 45 for average-risk adults, as recommended by the American Cancer Society.

- Diet and lifestyle: A diet rich in fruits, vegetables, and whole grains, limited red and processed meats, regular physical activity, maintaining a healthy weight, not smoking, and moderating alcohol intake can reduce risk.
- Genetic testing and counseling: Recommended for those with a family history indicative of genetic syndromes.

Colorectal cancer, when discovered early, is often treatable and frequently curable, highlighting the importance of regular screening and awareness of risk factors and symptoms.

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

Parkinson's Disease

Your Lifetime Risk	⚠ High

Your 10-Year Risk	✓ Normal

Your results are indicating a High risk of developing in your lifetime and within the next decade. Monitor your risk by regularly checking your related labs and implementing the recommendations provided.

DNA Risk Factors

Polygenic Risk Score based on 1,031,982 variants	⚠ 70 percentile │ More Likely

Other Risk Factors

Lab Risks		⚠ Detected
Lab Marker	**Optimal Range**	**Result**
LDL Cholesterol	50 mg/dL - 100 mg/dL	**124 mg/dL**
Triglycerides	0 mg/dL - 90 mg/dL	**134 mg/dL**
Copper, Serum	70 mcg/dL - 105 mcg/dL	**107.15 mcg/dL**

Common Variants That Increase Risk

Gene	SNP	Genotype	Gene	SNP	Genotype
MAPT	**rs17649553**	**CC**	FYN	**rs943437**	**AA**
STK39	**rs1474055**	**TT**	TMEM229B	**rs1555399**	**TT**
HLA-DQA2	**rs9275326**	**CC**	COQ8A	**rs4653767**	**TT**
MCCC1	**rs12637471**	**GG**	MED13	**rs6416935**	**GG**

Summary

Key Takeaways:

- About **20-40%** of the differences in people's chances to develop Parkinson's disease may be due to genetics.
- Other risk factors include age (over 60), being male, and toxin exposure.

- PD is an underdiagnosed disease, with about **90,000** diagnosed each year in the U.S.
- PD has no cure, but is managed better the earlier it is diagnosed.
- If you are at high genetic risk be aware of symptoms and talk to your doctor immediately if you notice any.

The causes of Parkinson's disease are not fully understood, but it likely involves a combination of **genetic and environmental factors**. These factors reduce the brain's ability to produce certain chemicals, mainly **dopamine** [R].

About **20-40%** of the differences in people's chances of developing Parkinson's disease may be due to **genetics**. Approximately **15%** of cases have a **family history** of the condition [R, R, R].

Genetically high betaine and choline levels may be causally associated with Parkinson's disease, while genetically high levels of DHA may be causally associated with a lower risk [R, R].

Beyond genetics, other risk factors for Parkinson's include [R]:

- Age: typically over 60
- Sex: men are at a higher risk
- Exposure to toxins like pesticides

Coronary Artery Disease

Your Lifetime Risk	✓ Normal

Your 10-Year Risk	✓ Normal

Your results are indicating a Normal risk of developing in your lifetime and within the next decade. Monitor your risk by regularly checking your related labs and implementing the recommendations provided.

DNA Risk Factors

Polygenic Risk Score based on 1,049,366 variants	⚠ 67 percentile │ More Likely

Other Risk Factors

Lab Risks		⚠ Detected

Lab Marker	Optimal Range	Result
Lead, Blood	0 ug/L - 10 ug/L	**400 ug/L**
Creatinine	0.7 mg/dL - 1.1 mg/dL	**1.4 mg/dL**
Hematocrit	40 % - 47 %	**57.5 %**
LDL/HDL Ratio	0.5 x100% - 2.3 x100%	**7.13 x100%**
eGFR	90 mL/min/1.73 m2 - 120 mL/min/1.73 m2	**50 mL/min/1.73 m2**
Valine, Plasma	180 mcmol/L - 313 mcmol/L	**143.43 mcmol/L**
Testosterone, Total	500 ng/dL - 1100 ng/dL	**300 ng/dL**
Vitamin C	0.85 mg/dL - 1.2 mg/dL	**0.262 mg/dL**
Platelet Count	200 K/cumm - 270 K/cumm	**150 K/cumm**
LDL Cholesterol	50 mg/dL - 100 mg/dL	**124 mg/dL**
Triglycerides	0 mg/dL - 90 mg/dL	**134 mg/dL**
SHBG	30 nmol/L - 45 nmol/L	**51 nmol/L**
ALP	30 IU/L - 60 IU/L	**74 IU/L**
Ferritin	80 ng/mL - 193 ng/mL	**230 ng/mL**

hs-CRP		0 mg/L - 0.5 mg/L	**0.91 mg/L**
Copper, Serum		70 mcg/dL - 105 mcg/dL	**107.15 mcg/dL**

Common Variants That Increase Risk

Gene	SNP	Genotype	Gene	SNP	Genotype
PCSK9	**rs11591147**	**GG**	SERPINA1	**rs112635299**	**GG**
ATG16L1	**rs10210302**	**TT**	ANGPTL4	**rs116843064**	**GG**
NKX2-3	**rs10883365**	**GG**	APOE	**rs7412**	**CC**
FHL3	**rs190569784**	**GG**	LDLR	**rs6511720**	**GG**

Summary

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

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]:

- 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].

Crohn's Disease

Your Lifetime Risk	✓ Normal

Your 10-Year Risk	✓ Normal

Your results are indicating a Normal risk of developing in your lifetime and within the next decade. Monitor your risk by regularly checking your related labs and implementing the recommendations provided.

DNA Risk Factors

Polygenic Risk Score based on 1,031,499 variants	⚠ 66 percentile │ More Likely

Other Risk Factors

Lab Risks		⚠ Detected
Lab Marker	**Optimal Range**	**Result**
Hematocrit	40 % - 47 %	**57.5 %**
Ferritin	80 ng/mL - 193 ng/mL	**230 ng/mL**
hs-CRP	0 mg/L - 0.5 mg/L	**0.91 mg/L**

Common Variants That Increase Risk

Gene	SNP	Genotype	Gene	SNP	Genotype
SLC23A1	rs10063949	CC	IL23R	rs11465804	TT
ATG16L1	rs2241880	GG	PPM1M	rs5743836	GG
STAT3	rs744166	AA	NKX2-3	rs10883365	GG
SLC22A5	rs12521868	TT	AGT	rs5051	TT

Summary

Aside from gastrointestinal symptoms, Crohn's Disease can also have systemic effects on the body, leading to issues such as anemia, skin rashes, arthritis, and eye inflammation. The cause of Crohn's Disease is not fully understood, but it involves an abnormal immune response to the microorganisms in the intestine, in genetically susceptible individuals.

There's no known cure for Crohn's Disease, but therapies can greatly reduce its signs and symptoms and even bring about long-term remission and healing of inflammation.

Psoriasis

Your Lifetime Risk	✓ Normal

Your 10-Year Risk	✓ Normal

Your results are indicating a Normal risk of developing in your lifetime and within the next decade. Monitor your risk by regularly checking your related labs and implementing the recommendations provided.

DNA Risk Factors

Polygenic Risk Score based on 1,049,035 variants	⚠ 65 percentile | More Likely

Other Risk Factors

Lab Risks		⚠ Detected
Lab Marker	**Optimal Range**	**Result**
Triglycerides	0 mg/dL - 90 mg/dL	**134 mg/dL**
hs-CRP	0 mg/L - 0.5 mg/L	**0.91 mg/L**

Common Variants That Increase Risk

Gene	SNP	Genotype	Gene	SNP	Genotype
TYK2	rs34536443	GG	IL12B	rs7709212	TT
IL23R	rs9988642	TT	GCA	rs17716942	TT
STAT2	rs2066819	CC	LCE3C	rs4845459	AA
RNF145	rs2082412	GG	IFNLR1	rs10794648	CC

Summary

Key Takeaways:

- Up to **90%** of differences in people's odds of developing psoriasis may be due to genetics.
- Psoriasis triggers include: infections, weather, skin injuries, stress, cigarette smoke, alcohol abuse, steroid withdrawal.
- About **2%** of Americans have psoriasis, mostly appearing in younger and older adults.
- Even though the condition is rare, people with high genetic risk should understand and be wary of potential triggers.
- Click the **Recommendations** tab for potential dietary and lifestyle changes.

Psoriasis is an autoimmune skin disease in which the body attacks its own skin cells. In response, skin cells begin to grow too quickly. New cells then begin to pile up on the skin's surface, forming plaques. The result is itchy, inflamed, scaly skin - the hallmark of psoriasis [R, R, R].

About 2% of Americans have psoriasis. It can appear at any age, but most cases develop between the ages of 15-20 or 55-60 [R].

People predisposed to psoriasis don't always have symptoms. In fact, **symptoms may only appear after contact with a "trigger"** [R].

Some common triggers include [R]:

- Throat and skin infections
- Dry and cold weather
- Skin injuries (like bug bites and sunburns)
- Stress
- Cigarette smoke
- Alcohol abuse
- Topical steroid withdrawal

Signs and symptoms of psoriasis include [R]:

- White scales covering patches of inflamed, itchy skin (often on the elbows, knees, scalp, and back)
- Joint stiffness
- Thickened or discolored nails

People with psoriasis also tend to have problems with their kidneys, heart, and joints. In fact, about 30% of patients have *psoriatic arthritis*. This painful condition mainly affects the fingers and toes [R].

As there is no cure for psoriasis, treatment aims to manage symptoms. Your doctor may suggest [R, R, R]:

- Light therapy
- Coal tar
- Medications that block the immune response
- Topical vitamin D
- Retinoids

Between 60-90% of differences in psoriasis may be attributed to genetics. Genes involved in psoriasis may influence [R, R, R]:

- Inflammation (*IL12B*, *IL23A*, *IL23R*, *NFKBIZ*)
- Immune response (*IFNLR1*, *NOS2*, *IFIH1*, *HLA-C*)

Genetically high neutrophil levels may be causally associated with a higher risk of psoriasis [R].

Melanoma

Your Lifetime Risk	✓ Normal

Your 10-Year Risk	✓ Normal

Your results are indicating a Normal risk of developing in your lifetime and within the next decade. Monitor your risk by regularly checking your related labs and implementing the recommendations provided.

DNA Risk Factors

Polygenic Risk Score based on 1,049,396 variants	◈ 76 percentile │ Typical Likelihood

Common Variants That Increase Risk

Gene	SNP	Genotype
SLC45A2	rs35407	GG

Summary

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

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

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

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

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

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

Alzheimer's Disease

Your Lifetime Risk	✓ Normal

Your 10-Year Risk	✓ Normal

Your results are indicating a Normal risk of developing in your lifetime and within the next decade. Monitor your risk by regularly checking your related labs and implementing the recommendations provided.

DNA Risk Factors

Polygenic Risk Score based on 1,049,157 variants	◆ 65 percentile │ Typical Likelihood

Other Risk Factors

Lab Risks	⚠ Detected

Lab Marker	Optimal Range	Result
AST	17 IU/L - 30 IU/L	**14 IU/L**
Valine, Plasma	180 mcmol/L - 313 mcmol/L	**143.43 mcmol/L**
Testosterone, Total	500 ng/dL - 1100 ng/dL	**300 ng/dL**
Vitamin C	0.85 mg/dL - 1.2 mg/dL	**0.262 mg/dL**
ALP	30 IU/L - 60 IU/L	**74 IU/L**
hs-CRP	0 mg/L - 0.5 mg/L	**0.91 mg/L**
Copper, Serum	70 mcg/dL - 105 mcg/dL	**107.15 mcg/dL**

Common Variants That Increase Risk

Gene	SNP	Genotype
CHRM2	**rs6962027**	**TT**
PICALM	**rs3851179**	**TT**
POLR2E	**rs12151021**	**AA**
ECHDC3	**rs7912495**	**GG**

Summary

Key Takeaways:

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

Some of the risk factors for Alzheimer's include [R]:

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

The following conditions may contribute to Alzheimer's disease [R]:

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

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

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

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

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

Asthma

Your Lifetime Risk	✓ Normal

Your 10-Year Risk	✓ Normal

Your results are indicating a Normal risk of developing in your lifetime and within the next decade. Monitor your risk by regularly checking your related labs and implementing the recommendations provided.

DNA Risk Factors

Polygenic Risk Score based on 1,049,396 variants	◈ 64 percentile │ Typical Likelihood

Other Risk Factors

Lifestyle Risks	⚠ Detected

Question	Answer
Do you have a parent or sibling who has ever been diagnosed with asthma?	**Yes**
Have you smoked tobacco for a total of 3 or more years?	**Yes**
How would you describe the environment you live in?	**Urban**
In a typical week, how many times do you participate in any physical activities or exercise for 30 minutes at a time? (such as walking, running, bike riding, weight training, yoga, etc.) *Note: longer exercise equals more sessions (e.g., 1 hour = 2 sessions)	**3-4**

Lab Risks	⚠ Detected

Lab Marker	Optimal Range	Result
Lymphocytes (%)	35 % - 55 %	**31 %**
SHBG	30 nmol/L - 45 nmol/L	**51 nmol/L**
hs-CRP	0 mg/L - 0.5 mg/L	**0.91 mg/L**

Common Variants That Increase Risk

Gene	SNP	Genotype	Gene	SNP	Genotype
STAT6	rs3024971	TT		rs115468973	TT
PTGER4	rs7720838	TT	/	rs201184533	CC
LPP	rs9865818	GG	/	rs116189786	AA
RUNX3	rs760805	TT	IL2RA	rs12722502	CC

Summary

Key Takeaways:

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

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

The symptoms of an asthma attack include [R, R]:

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

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

In people with asthma, attacks can be triggered by [R, R]:

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

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

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

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

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

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

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

SelfDecode

Ulcerative Colitis

Your Lifetime Risk	✓ Normal

Your 10-Year Risk	✓ Normal

Your results are indicating a Normal risk of developing in your lifetime and within the next decade. Monitor your risk by regularly checking your related labs and implementing the recommendations provided.

DNA Risk Factors

Polygenic Risk Score based on 1,049,227 variants	◈ 63 percentile │ Typical Likelihood

Other Risk Factors

Lab Risks	⚠ Detected

Lab Marker	Optimal Range	Result
Hematocrit	40 % - 47 %	**57.5 %**
Platelet Count	200 K/cumm - 270 K/cumm	**150 K/cumm**
hs-CRP	0 mg/L - 0.5 mg/L	**0.91 mg/L**

Common Variants That Increase Risk

Gene	SNP	Genotype	Gene	SNP	Genotype
STAT3	**rs744166**	**AA**	MFSD4B	**rs3851228**	**AA**
HNF4A	**rs6017342**	**CC**	NR5A2	**rs2816958**	**GG**
IL23R	**rs76418789**	**GG**	IL19	**rs1800872**	**GG**
/	**rs113653754**	**CC**	FCGR2A	**rs1801274**	**AA**

Summary

The exact cause of ulcerative colitis is not fully understood, but it is believed to be the result of an overactive immune system response that leads to inflammation in the colon. It can affect individuals at any age, though it often begins during adolescence and early adulthood.

The impact of the disease can range from mild to severe, with some patients experiencing life-threatening complications. Managing ulcerative colitis often requires a combination of medication, lifestyle changes, and potentially surgery to control symptoms and improve quality of life.

SelfDecode

Stroke

Your Lifetime Risk	✓ Normal

Your 10-Year Risk	✓ Normal

Your results are indicating a Normal risk of developing in your lifetime and within the next decade. Monitor your risk by regularly checking your related labs and implementing the recommendations provided.

DNA Risk Factors

Polygenic Risk Score based on 1,030,648 variants	◆ 63 percentile | Typical Likelihood

Other Risk Factors

Lab Risks	⚠ Detected

Lab Marker	Optimal Range	Result
Lead, Blood	0 ug/L - 10 ug/L	**400 ug/L**
Testosterone, Total	500 ng/dL - 1100 ng/dL	**300 ng/dL**
Platelet Count	200 K/cumm - 270 K/cumm	**150 K/cumm**
LDL Cholesterol	50 mg/dL - 100 mg/dL	**124 mg/dL**
SHBG	30 nmol/L - 45 nmol/L	**51 nmol/L**
RDW	11.5 % - 13 %	**13.6 %**
hs-CRP	0 mg/L - 0.5 mg/L	**0.91 mg/L**
Copper, Serum	70 mcg/dL - 105 mcg/dL	**107.15 mcg/dL**

Common Variants That Increase Risk

Gene	SNP	Genotype	Gene	SNP	Genotype
HP	rs879324	AA	PTPRF	rs6695915	AA
ALDH2	rs2238151	TT	LIPA	rs1412444	TT
ABCG5	rs76866386	TT	HTRA1	rs60401382	CC
ABO	rs532436	AA	SWAP70	rs10840293	AA

Summary

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

SelfDecode

Schizophrenia

Your Lifetime Risk	✓ **Normal**

Your 10-Year Risk	✓ **Normal**

Your results are indicating a Normal risk of developing in your lifetime and within the next decade. Monitor your risk by regularly checking your related labs and implementing the recommendations provided.

DNA Risk Factors

Polygenic Risk Score based on 1,033,405 variants	◆ **63 percentile** │ **Typical Likelihood**

Other Risk Factors

Lab Risks		⚠ **Detected**
Lab Marker	**Optimal Range**	**Result**
SHBG	30 nmol/L - 45 nmol/L	**51 nmol/L**
Copper, Serum	70 mcg/dL - 105 mcg/dL	**107.15 mcg/dL**

Common Variants That Increase Risk

Gene	SNP	Genotype	Gene	SNP	Genotype
COMT	**rs737865**	**AA**	CBLN3	**rs146732081**	**CC**
DPYD	**rs1702294**	**CC**	EFCAB6	**rs76365544**	**GG**
PILRB	**rs41295924**	**CC**	EDEM3	**rs78444298**	**GG**
H4C13	**rs140365013**	**GG**	ELAPOR2	**rs137881681**	**GG**

Summary

About **70-80%** of differences in people's schizophrenia rates may be due to **genetics**! Individuals with a close family member, like a parent or sibling with the disorder, are more likely to develop schizophrenia than those without a family history.

Genetically high fasting insulin and alpha-linolenic acid levels may be causally associated with schizophrenia. In contrast, genetically high levels of omega-3s may be causally associated with a lower risk [R, R, R, R].

Several genes are associated with an increased risk of schizophrenia, but no single gene causes the disorder by itself. It's believed that a complex interplay of genetics and one's environment contributes to the development of the disorder.

Factors that might increase the risk of developing schizophrenia include:

- Increased immune system activation, such as from inflammation or infections.
- Complications during birth.
- Psychosocial stresses during early adulthood.
- Psychoactive drug use during adolescence.
- Some pregnancy and birth complications, like malnutrition or exposure to viruses.

Please note: This report accounts for only a fraction of schizophrenia's genetic component. Even if your risk is higher, it doesn't mean you are likely to have or develop the condition.

Venous Thromboembolism

Your Lifetime Risk	✓ Normal

Your 10-Year Risk	✓ Normal

Your results are indicating a Normal risk of developing in your lifetime and within the next decade. Monitor your risk by regularly checking your related labs and implementing the recommendations provided.

DNA Risk Factors

Polygenic Risk Score based on 1,049,243 variants	◈ 61 percentile │ Typical Likelihood

Common Variants That Increase Risk

Gene	SNP	Genotype	Gene	SNP	Genotype
F5	**rs4524**	**TT**	HLA-DQA2	**rs17202393**	**GG**
ABO	**rs657152**	**AA**	NRG3	**rs1649936**	**CC**
MCMBP	**rs12767946**	**TT**	NCAM2	**rs62207434**	**CC**
KIF26B	**rs1756912**	**GG**	CSGALNACT1	**rs62496681**	**CC**

Summary

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.

Cardiovascular Disease

Your Lifetime Risk	✓ Normal

Your 10-Year Risk	✓ Normal

Your results are indicating a Normal risk of developing in your lifetime and within the next decade. Monitor your risk by regularly checking your related labs and implementing the recommendations provided.

DNA Risk Factors

Polygenic Risk Score based on 1,049,427 variants	◈ 60 percentile ⎪ Typical Likelihood

Common Variants That Increase Risk

Gene	SNP	Genotype	Gene	SNP	Genotype
PCSK9	rs11591147	GG	SERPINA1	rs112635299	GG
ATG16L1	rs10210302	TT	ANGPTL4	rs116843064	GG
NKX2-3	rs10883365	GG	APOE	rs7412	CC
FHL3	rs190569784	GG	LDLR	rs6511720	GG

Summary

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.

Multiple Sclerosis

Your Lifetime Risk	✓ Normal

Your 10-Year Risk	✓ Normal

Your results are indicating a Normal risk of developing in your lifetime and within the next decade. Monitor your risk by regularly checking your related labs and implementing the recommendations provided.

DNA Risk Factors

Polygenic Risk Score based on 1,019,187 variants	◆ 60 percentile	Typical Likelihood

Other Risk Factors

Lab Risks	⚠ Detected

Lab Marker	Optimal Range	Result
hs-CRP	0 mg/L - 0.5 mg/L	**0.91 mg/L**

Common Variants That Increase Risk

Gene	SNP	Genotype	Gene	SNP	Genotype
EBPL	rs9591325	TT	TAPBPL	rs12832171	CC
IL2RA	rs2104286	TT	JAK1	rs72922276	GG
RBM17	rs11256593	TT	RGS1	rs1323292	AA
TYK2	rs34536443	GG	RTEL1-TNFRSF6B	rs6742	CC

Summary

About **50%** of the differences in people's MS rates may be due to **genetics** [R].

While no single gene has been identified as the cause of MS, certain genetic variants have been linked to an increased risk of the disease. Having a close family member with MS can increase one's risk, suggesting a hereditary component.

Moreover, a genetically high leukocyte count may be causally associated with MS susceptibility [R].

Other factors that might increase the risk of developing multiple sclerosis include:

- Age: MS is most commonly diagnosed in people between the ages of 20 and 50.
- Sex: Women are about two to three times more likely than men to develop MS.

- Certain infections, like Epstein-Barr virus.
- Climate: MS is more common in countries with temperate climates.
- Autoimmune diseases: If you have thyroid disease, type 1 diabetes, or inflammatory bowel disease, you might have an increased risk of developing MS.
- Smoking.

SelfDecode

High Blood Pressure

Your Lifetime Risk	✓ Normal

Your 10-Year Risk	✓ Normal

Your results are indicating a Normal risk of developing in your lifetime and within the next decade. Monitor your risk by regularly checking your related labs and implementing the recommendations provided.

DNA Risk Factors

Polygenic Risk Score based on 1,035,787 variants	◆ 51 percentile │ Typical Likelihood

Other Risk Factors

Lab Risks		⚠ Detected
Lab Marker	**Optimal Range**	**Result**
Lead, Blood	0 ug/L - 10 ug/L	**400 ug/L**
Valine, Plasma	180 mcmol/L - 313 mcmol/L	**143.43 mcmol/L**
Platelet Count	200 K/cumm - 270 K/cumm	**150 K/cumm**
LDL Cholesterol	50 mg/dL - 100 mg/dL	**124 mg/dL**
Triglycerides	0 mg/dL - 90 mg/dL	**134 mg/dL**
SHBG	30 nmol/L - 45 nmol/L	**51 nmol/L**
hs-CRP	0 mg/L - 0.5 mg/L	**0.91 mg/L**
Copper, Serum	70 mcg/dL - 105 mcg/dL	**107.15 mcg/dL**

Common Variants That Increase Risk

Gene	SNP	Genotype
AGT	**rs699**	**GG**
ADRB1	**rs1801253**	**CC**
BCL2	**rs12454712**	**TT**
APOE	**rs7412**	**CC**

Gene	SNP	Genotype
TNNT3	**rs1973765**	**CC**

Summary

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]
- 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].

Type 2 Diabetes

Your Lifetime Risk	✓ Normal

Your 10-Year Risk	✓ Normal

Your results are indicating a Normal risk of developing in your lifetime and within the next decade. Monitor your risk by regularly checking your related labs and implementing the recommendations provided.

DNA Risk Factors

Polygenic Risk Score based on 1,048,858 variants	◆ 49 percentile	Typical Likelihood

Other Risk Factors

Lifestyle Risks ⚠ Detected

Question	Answer
How many cups of coffee do you drink on a typical day?	**1-2**
In a typical week, how many times do you participate in any physical activities or exercise for 30 minutes at a time? (such as walking, running, bike riding, weight training, yoga, etc.) *Note: longer exercise equals more sessions (e.g., 1 hour = 2 sessions)	**3-4**
What is your waist size?	**35-39 (89-99 cm)**
How many servings of whole grains do you eat in a typical day? Please click here for more information on whole grain servings	**Less than 3**
How many times in a typical day do you eat refined grains (e.g., white pasta, white rice, white bread, etc.)?	**3 or more**

Lab Risks ⚠ Detected

Lab Marker	Optimal Range	Result
AST	17 IU/L - 30 IU/L	**14 IU/L**
Testosterone, Total	500 ng/dL - 1100 ng/dL	**300 ng/dL**
Vitamin C	0.85 mg/dL - 1.2 mg/dL	**0.262 mg/dL**
LDL Cholesterol	50 mg/dL - 100 mg/dL	**124 mg/dL**
SHBG	30 nmol/L - 45 nmol/L	**51 nmol/L**

hs-CRP	0 mg/L - 0.5 mg/L	**0.91 mg/L**
Copper, Serum	70 mcg/dL - 105 mcg/dL	**107.15 mcg/dL**

Common Variants That Increase Risk

Gene	SNP	Genotype
SLC30A8	**rs13266634**	**CC**
GCKR	**rs780093**	**CC**
CDKN2A	**rs10811661**	**TT**
ADCY5	**rs11708067**	**AA**

Gene	SNP	Genotype
BCL2	**rs12454712**	**TT**
CCND2	**rs76895963**	**TT**
PEMT	**rs12325817**	**CC**

Summary

Key Takeaways:

- Almost **1 in 3 Americans** are at risk of developing type 2 diabetes.
- Up to **80%** of the differences in people's risk for getting type 2 diabetes may be due to genetics.
- Even with high genetic risk, blood sugar issues are highly modifiable through diet, exercise, and lifestyle changes.
- Risk factors include: obesity, high sugar diet, lack of exercise, age over 45, smoking, and family history. Even with low genetic risk, these factors can raise your overall risk, so take action now!
- Click the **Recommendations** tab for potential dietary and lifestyle changes and **next steps** for relevant labs.

You've probably heard about the dangers of high blood sugar (glucose). It puts **almost 1 in 3 Americans at risk of developing type 2 diabetes** [R].

Type 2 diabetes is a common and dangerous disease. In older adults, it can cause heart disease, stroke, kidney damage, and more. If diabetes isn't treated, it can be fatal [R].

If you're at risk of diabetes, your doctor may recommend weight loss and diet changes. **Eating less sugar is usually the first step.** If your blood sugar (glucose) is very high, your doctor may also prescribe medications [R, R].

To understand how blood sugar rises and falls, we first need to understand how insulin works.

When blood sugar is high, the pancreas releases insulin. Insulin is responsible for lowering blood sugar. It signals your liver and muscles to store sugar [R, R].

Insulin levels rise when you eat sugary foods. If insulin stays high for a long time, your body can stop responding to it. This is called insulin resistance [R].

Insulin resistance often leads to higher than normal blood sugar levels, or *prediabetes*. **If you don't take steps to fix it, prediabetes can develop into type 2 diabetes** [R].

Prediabetes is hard to spot because it doesn't have obvious symptoms. However, blood tests can help diagnose it [R].

A doctor might order blood sugar tests if any of the following risk factors apply to you [R]:

- Obesity
- A diet high in sugar and refined carbs
- Lack of exercise
- Age over 45
- Polycystic ovary syndrome (PCOS)
- Smoking
- Family history of diabetes
- Black, Hispanic, Asian, or Native American ethnicity

Up to 80% of the differences in people's chances of getting type 2 diabetes can be attributed to genetics. Genes that may contribute to high blood sugar influence [R]:

- Sensitivity to insulin (*TCF7L2*, *FTO*, *PPARG*)
- Insulin production & release (*KCNJ11*, *SLC30A8*)
- Liver function (*HNF4A*)

Genetically high levels of the following markers may be causally associated with a higher risk of type 2 diabetes [R, R, R, R, R]:

- IGF-1
- Neutrophils
- Leucine

In contrast, genetic predisposition to the following high markers may be causally linked to a lower risk of type 2 diabetes [R, R, R, R, R, R]:

- Testosterone (in men)
- Betaine
- Choline
- Alpha-linolenic acid

- Sensitivity to insulin (*TCF7L2*, *FTO*, *PPARG*)
- Insulin production & release (*KCNJ11*, *SLC30A8*)
- Liver function (*HNF4A*)

SelfDecode

Age-Related Macular Degeneration

Your Lifetime Risk	✓ Normal

Your 10-Year Risk	✓ Normal

Your results are indicating a Normal risk of developing in your lifetime and within the next decade. Monitor your risk by regularly checking your related labs and implementing the recommendations provided.

DNA Risk Factors

Polygenic Risk Score based on 1,049,359 variants	44 percentile │ Typical Likelihood

Other Risk Factors

Lab Risks		⚠ Detected
Lab Marker	**Optimal Range**	**Result**
Triglycerides	0 mg/dL - 90 mg/dL	**134 mg/dL**
hs-CRP	0 mg/L - 0.5 mg/L	**0.91 mg/L**

Common Variants That Increase Risk

Gene	SNP	Genotype	Gene	SNP	Genotype
NECTIN2	**rs2075650**	**AA**	NLRC5	**rs1864163**	**GG**
MBL2	**rs6480975**	**CC**	PRDM1	**rs7750345**	**AA**
	rs5749482	**GG**	VEGFA	**rs943080**	**TT**
APOE	**rs4420638**	**AA**	MCUB	**rs4698775**	**GG**

Summary

Key Takeaways:

- About **45-70%** of differences in people's chances of having AMD may be due to genetics.
- Other potential risk factors include age (60+), smoking, diet, race, high blood pressure, heart disease, and excessive sun exposure.
- About **8.5%** of people aged **45-85** have some degree of AMD. Symptoms include difficulty reading, blurred vision, and dark spots in the field of vision. Talk to your eye doctor if you notice any symptoms.
- If you have a high genetic risk, be aware of symptoms and have your eyes checked regularly, especially after age 60.

- Click the **Recommendations** tab for potential dietary and lifestyle changes.

Age-related macular degeneration (AMD) is a progressive eye disease that affects the macula, the part of the retina responsible for central vision. It is the leading cause of blindness in people over the age of 60.

About **45-70%** of differences in people's chances of having AMD may be due to **genetics** [R].

For example, genetically high ApoB levels may be causally associated with a lower risk of the intermediate and geographic atrophy (GA) subtypes of AMD [R].

Other factors that play a role in AMD development include:

- Age (60+)
- Smoking
- Excessive sun exposure
- Unhealthy diet
- Race (European)
- High blood pressure
- Heart disease

Celiac Disease

Your Lifetime Risk	✓ Normal

Your 10-Year Risk	✓ Normal

Your results are indicating a Normal risk of developing in your lifetime and within the next decade. Monitor your risk by regularly checking your related labs and implementing the recommendations provided.

DNA Risk Factors

Polygenic Risk Score based on 1,019,187 variants	◆ 42 percentile │ Typical Likelihood

Other Risk Factors

Lab Risks	⚠ Detected

Lab Marker	Optimal Range	Result
Testosterone, Total	500 ng/dL - 1100 ng/dL	**300 ng/dL**
Ferritin	80 ng/mL - 193 ng/mL	**230 ng/mL**

Common Variants That Increase Risk

Gene	SNP	Genotype

Summary

Personalized For Your Genes

Based on the variants that we looked at, you may have typical sensitivity to gluten. You may be less likely to experience digestive symptoms after eating foods containing gluten.

Feel free to enjoy gluten-containing foods in moderation if they are not causing you unpleasant symptoms.

Key Takeaways:

- It's estimated that 1-2% of the population has gluten sensitivity. The most likely risk factor is genetics.
- If you have symptoms, diet restriction may indicate whether you have the sensitivity or not. You should speak to a healthcare professional if symptoms persist.
- Symptoms include diarrhea/constipation, fatigue, weight loss, gut pain/bloating, and nausea.
- Celiac disease is rare, so even with high genetic risk, your overall risk is still low.
- Click the **next steps** tab for relevant labs.

Gluten is a protein found in grains such as wheat, rye, spelt, barley, and triticale. Some people cannot properly digest gluten. In fact, their immune systems may react to gluten as if it is dangerous. To make matters worse, gluten is similar to a normal protein in the intestine. Sometimes, the immune system will attack both. People with this type of reaction have celiac disease [R, R, R].

Researchers aren't completely sure why some people are sensitive to gluten. Infections in the gut may play a role. However, a major risk factor is probably genetic [R, R, R].

The most important genes involved in celiac disease are *HLA* genes. These genes help make HLA proteins, which sit on the surface of white blood cells. They help the immune system attack and remove dangerous invaders like bacteria and viruses. In people with celiac disease, HLA proteins may attack gluten by mistake and damage the gut barrier [R, R].

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

Rheumatoid Arthritis

Your Lifetime Risk	✓ Normal

Your 10-Year Risk	✓ Normal

Your results are indicating a Normal risk of developing in your lifetime and within the next decade. Monitor your risk by regularly checking your related labs and implementing the recommendations provided.

DNA Risk Factors

Polygenic Risk Score based on 1,049,410 variants	◈ 40 percentile │ Typical Likelihood

Other Risk Factors

Lab Risks	⚠ Detected

Lab Marker	Optimal Range	Result
Lead, Blood	0 ug/L - 10 ug/L	**400 ug/L**
Testosterone, Total	500 ng/dL - 1100 ng/dL	**300 ng/dL**
Platelet Count	200 K/cumm - 270 K/cumm	**150 K/cumm**
SHBG	30 nmol/L - 45 nmol/L	**51 nmol/L**
hs-CRP	0 mg/L - 0.5 mg/L	**0.91 mg/L**
Copper, Serum	70 mcg/dL - 105 mcg/dL	**107.15 mcg/dL**

Common Variants That Increase Risk

Gene	SNP	Genotype	Gene	SNP	Genotype
CTLA4	**rs3087243**	**GG**	WDFY4	**rs2671692**	**AA**
ANKRD55	**rs7731626**	**GG**	RASGRP1	**rs8032939**	**CC**
UBASH3A	**rs1893592**	**AA**	RUNX1	**rs8133843**	**AA**
ETS1	**rs73013527**	**CC**	COG6	**rs9603618**	**CC**

Summary

Key Takeaways:

- Up to 65% of differences in people's chances of developing rheumatoid arthritis may be due to genetics.
- Other risk factors include obesity and smoking.
- Rheumatoid arthritis affects about **1%** of people around the world. This means even a high genetic risk is still a low overall risk.
- Click the **Recommendations** tab for potential dietary and lifestyle changes and **next steps** for relevant labs.

Rheumatoid arthritis is an autoimmune condition in which the body attacks its own joints. This causes inflammation, tissue damage, and pain [R].

Rheumatoid arthritis affects about 1% of people around the world. Researchers have found big differences between populations. North America has the highest rate, with the lowest rates in South America and Asia [R].

According to one estimate, about **1.3 million Americans** have this condition [R].

Rheumatoid arthritis usually affects small joints in the hands and feet. Its signs and symptoms include [R, R]:

- Joint pain and tenderness
- Heat and swelling in the affected joints
- Joint stiffness

Many people have periods of worsening symptoms called "flares." These flares may be triggered by [R]:

- Stress
- Too much movement
- A change in medication

Rheumatoid arthritis may lead to complications outside the joints. They can include heart disease, nerve problems, and infections [R].

There is no cure for rheumatoid arthritis. Instead, patients and doctors work to control symptoms. Some ways to manage the condition include [R, R]:

- Medications
- Surgery (e.g., joint replacement surgery)
- Exercise
- Supplements to reduce inflammation and support bone health

The exact cause of rheumatoid arthritis is unknown. Risk factors include [R, R, R]:

- Cigarette smoking
- Obesity
- **Genetics**

In fact, up to 65% of differences in people's chances of developing rheumatoid arthritis may be attributed to genetics. Genes involved in this condition may influence [R, R]:

- Immune function (*HLA-DRB1*, *PSORS1C1*)
- Inflammation (*STAT4*, *IL10*, *PTPN2*)

Genetically high testosterone and omega-3s levels may be causally associated with a high risk of rheumatoid arthritis [R, R].

SelfDecode

Atrial Fibrillation

Your Lifetime Risk	✓ Normal

Your 10-Year Risk	✓ Normal

Your results are indicating a Normal risk of developing in your lifetime and within the next decade. Monitor your risk by regularly checking your related labs and implementing the recommendations provided.

DNA Risk Factors

Polygenic Risk Score based on 1,049,356 variants	39 percentile │ Typical Likelihood

Other Risk Factors

Lab Risks	⚠ Detected

Lab Marker	Optimal Range	Result
Creatinine	0.7 mg/dL - 1.1 mg/dL	**1.4 mg/dL**
eGFR	90 mL/min/1.73 m2 - 120 mL/min/1.73 m2	**50 mL/min/1.73 m2**
Platelet Count	200 K/cumm - 270 K/cumm	**150 K/cumm**
Ferritin	80 ng/mL - 193 ng/mL	**230 ng/mL**
hs-CRP	0 mg/L - 0.5 mg/L	**0.91 mg/L**

Common Variants That Increase Risk

Gene	SNP	Genotype
HP	**rs2359171**	**AA**
PITX2	**rs143269342**	**CC**
TBX5	**rs883079**	**TT**
GJA5	**rs79187193**	**GG**

Gene	SNP	Genotype
PITX2	**rs3853445**	**TT**
PITX2	**rs17570669**	**AA**

Summary

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].

SelfDecode

Osteoporosis

Your Lifetime Risk	✓ Normal

Your 10-Year Risk	✓ Normal

Your results are indicating a Normal risk of developing in your lifetime and within the next decade. Monitor your risk by regularly checking your related labs and implementing the recommendations provided.

DNA Risk Factors

Polygenic Risk Score based on 1,031,189 variants	◈ 33 percentile │ Typical Likelihood

Other Risk Factors

Lab Risks	⚠ Detected

Lab Marker	Optimal Range	Result
Lead, Blood	0 ug/L - 10 ug/L	**400 ug/L**
Testosterone, Total	500 ng/dL - 1100 ng/dL	**300 ng/dL**
LDL Cholesterol	50 mg/dL - 100 mg/dL	**124 mg/dL**
Triglycerides	0 mg/dL - 90 mg/dL	**134 mg/dL**
SHBG	30 nmol/L - 45 nmol/L	**51 nmol/L**
ALP	30 IU/L - 60 IU/L	**74 IU/L**

Common Variants That Increase Risk

Gene	SNP	Genotype	Gene	SNP	Genotype
MARCO	rs115242848	**CC**	VARS2	rs9262558	**CC**
COPB1	rs10741657	**GG**	SP7	rs144680237	**CC**
CPNE1	rs143383	**AA**	CPED1	rs3779381	**AA**
HLA-DQA1	rs2071805	**CC**	ZBTB40	rs34414754	**AA**

Summary

Key Takeaways:

- Up to **65%** of differences in people's BMD may be attributed to genetics.
- Other risk factors for osteoporosis include: age, menopause, underweight, steroid use, smoking, and alcohol abuse.
- Over **200 million** people have osteoporosis. If your genetic risk is high or you are getting older, you may want to take precautions.
- Click the **Recommendations** tab for potential dietary and lifestyle changes and **next steps** for relevant labs.

Bone health is most often measured through **bone mineral density** (**BMD**). This is the amount of calcium and other minerals in your bones [R, R, R].

Higher BMD tends to mean stronger, healthier bones. BMD peaks between the ages of 25 and 30 and then decreases as we age. Lower BMD for your age may put you at risk of fractures and *osteoporosis* [R, R].

Osteoporosis is a disease that develops when BMD is dangerously low. Its name means *porous bones*. As the name implies, the structure of the bones changes in people with this condition. Their bones lose mass and strength, leaving gaps and holes [R, R, R].

People with osteoporosis are much more likely to break their bones. They may even be at risk of fracture from doing normal day-to-day activities [R].

According to one estimate, **over 200 million people currently have osteoporosis**. **About 1 in 3 women and 1 in 5 men over 50 will break a bone due to this condition** [R].

Risk factors for weak bones include [R, R]:

- Older age
- Menopause
- Low body weight
- Steroid medications
- Cigarette smoking
- High alcohol intake
- **Genetics**

It's impossible to tell if you have low BMD without a doctor's help. This is because low BMD on its own doesn't have any obvious symptoms. Many people have no idea they have osteoporosis until they break a bone [R].

To support bone health and prevent fractures, your doctor may recommend [R]:

- Exercising
- Getting more calcium and vitamin D
- Avoiding cigarettes
- Limiting alcohol

Once osteoporosis is diagnosed, treatment may also include medication [R, R, R].

Up to 65% of differences in people's BMD may be attributed to genetics. Genes involved in BMD may influence [R, R, R]:

- Bone formation and repair (*DAAM2*, *BICC1*, *LGR4*, *NPR3*)
- Gene activity (*HMGA2*)
- Vitamin D activity (*VDR*)

Genetically high IGF-1, free testosterone (in men), and alpha-linolenic acid may be causally associated with higher bone density. In contrast, genetically high total testosterone may be causally associated with lower bone density and a higher risk of osteoporosis [R, R, R, R].

SelfDecode

Type 1 Diabetes

Your Lifetime Risk	✓ Normal

Your 10-Year Risk	✓ Normal

Your results are indicating a Normal risk of developing in your lifetime and within the next decade. Monitor your risk by regularly checking your related labs and implementing the recommendations provided.

DNA Risk Factors

Polygenic Risk Score based on 1,047,920 variants	◈ 15 percentile │ Typical Likelihood

Common Variants That Increase Risk

Gene	SNP	Genotype	Gene	SNP	Genotype
RASGRP1	rs72727394	TT	TYK2	rs34536443	GG
CTSH	rs34593439	GG	IL2RA	rs61839660	CC
IL7R	rs11954020	GG	INS-IGF2	rs689	TT
MAPT	rs1052553	AA	IL2RA	rs41295121	CC

Summary

Risk factors for type 1 diabetes include [R]:

- Family history: Having a parent or sibling with type 1 diabetes.
- Age: Type 1 diabetes can occur at any age but is more commonly diagnosed from infancy to the late 30s.
- Geography: The incidence of type 1 diabetes tends to increase as you travel away from the equator.
- **Genetics**

Up to **88%** of the differences in people's risk of type 1 diabetes may be genetic. Multiple genes contribute to its risk, particularly those affecting immune system function [R, R].

Currently, there is no known way to prevent type 1 diabetes. The condition requires careful management and a multidisciplinary healthcare approach that includes [R]:

- Lifelong insulin therapy: Insulin injections or an insulin pump to regulate blood sugar levels.
- Blood sugar monitoring: Regular monitoring is essential for managing insulin dosing.
- Healthy eating: Paying attention to food choices, particularly carbohydrates.
- Regular exercise: Exercise is a crucial part of diabetes management.
- Routine health checks: Regular check-ups are vital to monitor for complications.

SelfDecode

Lupus

Your Lifetime Risk	✓ Normal

Your 10-Year Risk	✓ Normal

Your results are indicating a Normal risk of developing in your lifetime and within the next decade. Monitor your risk by regularly checking your related labs and implementing the recommendations provided.

DNA Risk Factors

Polygenic Risk Score based on 1,049,429 variants	◇ 12 percentile │ Typical Likelihood

Other Risk Factors

Lab Risks	⚠ Detected

Lab Marker	Optimal Range	Result
Creatinine	0.7 mg/dL - 1.1 mg/dL	**1.4 mg/dL**
Platelet Count	200 K/cumm - 270 K/cumm	**150 K/cumm**
Ferritin	80 ng/mL - 193 ng/mL	**230 ng/mL**
hs-CRP	0 mg/L - 0.5 mg/L	**0.91 mg/L**
Copper, Serum	70 mcg/dL - 105 mcg/dL	**107.15 mcg/dL**

Common Variants That Increase Risk

Gene	SNP	Genotype	Gene	SNP	Genotype
IRF8	**rs11644034**	**GG**	LYST	**rs9782955**	**CC**
NADSYN1	**rs3794060**	**CC**	ZFP36L1	**rs4902562**	**AA**
PRDM1	**rs6568431**	**AA**			
IKZF1	**rs4917014**	**TT**			

Summary

Key Takeaways:

- Up to **65%** of differences in people's chances of having lupus may be due to genetics.
- Other risk factors include being female, non-white, and young.
- It's often triggered by sunlight, medications, or infections.
- If you have a high genetic risk, being aware of symptoms and living a healthy lifestyle are important factors. Contact a healthcare professional if you develop symptoms.
- Click the **Recommendations** tab for potential dietary and lifestyle changes, and **next steps** for relevant labs.

The main risk factors for lupus include [R,R]:

- **Sex**: lupus is far more prevalent in women
- **Age**: most new cases are falling in the 15-44 age range
- **Race**: it's 2-3 times less likely in white people
- **Genetics**: Up to **65%** of differences in people's chances of having lupus may be due to genetics

Lupus can be triggered by sunlight, medications, or infections.

Medication Check (Pharmacogenomics)

Our Pharmacogenomic (PGx) Report provides information about how a patient's genetic makeup affects their response to medication. Genetic variants may speed or slow down the metabolism of certain drugs and may also make a patient more or less prone to side effects associated with the use of certain drugs.

This report provides information about 50+ drugs, based on the guidelines developed by specialized organizations such as the Clinical Pharmacogenetics Implementation Consortium (CPIC), US Food and Drug Administration (FDA), Pharmacogenomics Knowledge Base (PharmGKB), and the Dutch Pharmacogenetics Working Group (DPWG).

The information in this report is provided for:
- a specific genetic variant, denoted by its rsID
- a metabolizer type, based on a set of variants found in a certain gene. Each patient carries a combination of two alleles, denoted by a star (e.g. *1*2) which are mapped to a specific metabolizer type as defined by CPIC and PharmGKB

Evidence levels

Variant-drug or metabolizer-drug information carries the following evidence levels:

⭐⭐⭐ These are variant-drug combinations that include known pharmacogenes and have variant-specific prescribing guidance available in a current clinical guideline annotation or an FDA-approved drug label annotation with at least one publication in addition to the clinical guideline OR have high level of evidence supporting the association with at least two independent publications.

⭐⭐ These variant-drug combinations include known pharmacogenes and have a moderate level of evidence supporting the association, with at least two independent publications. For example, the association may be found in multiple cohorts, but there may be a minority of studies that do not support the majority assertion.

⭐ These are variant-drug combinations with a low level of evidence supporting the association. This association may be based on a single study or there may be several studies that failed to replicate the association. The annotation may also be based on preliminary evidence (e.g., a case report, non-significant study, or in vitro, molecular, or functional assay evidence). Alternatively, this is an association involving a rare genotype that has been extrapolated based on information available for more common and well studied genotypes.

Prescribing guidance

Recommendations about drug dosages and administration are obtained from CPIC, FDA, or DPWG guideline publications.

Based on the available information and recommendations, drugs are classified into two categories:

> ✓ **Standard precautions:** The patient may respond well to this drug, with a low risk of side effects. Often, the standard recommended dosage is advised for this drug.

> ⓘ **Use with caution:** The patient may be at an increased risk of adverse events and may need monitoring for efficacy or side effects. Standard dosage may not be adequate for certain drugs, and, in some cases, an alternative drug may be recommended.

Pharmacogenomic Guidance Overview

Category	Drug Class	✓ Standard Precautions	⚠ Use With Caution
Anesthesiology	**Anesthetic**	Volatile Anesthetics (Desflurane, Enflurane, Halothane, Isoflurane, Methoxyflurane, Sevoflurane)	
	Skeletal Muscle Relaxant	Succinylcholine	
Cardiovascular	**Antiplatelets & Anticoagulants**	Acenocoumarol Clopidogrel	**Phenprocoumon** **Warfarin**
	Statins		**Atorvastatin** **Fluvastatin** **Lovastatin** **Pitavastatin** **Pravastatin** **Rosuvastatin** **Simvastatin**
Gastrointestinal	**Proton Pump Inhibitors**	Rabeprazole	**Dexlansoprazole** **Lansoprazole** **Omeprazole** **Pantoprazole**
Immunology, Rheumatology & Oncology	**Antihyperuricemic & Antigout Agents**	Allopurinol	
	Antimetabolites	Capecitabine Fluorouracil Mercaptopurine Tegafur	
	Immunosuppressants	Azathioprine Siponimod Tacrolimus	**Methotrexate**
Infections	**Antifungals**	Voriconazole	
	Antivirals	Nevirapine	**Efavirenz** **Triple therapy (peginterferon alfa-2a/b & ribavirin)**
Pain	**NSAIDs**	Celecoxib Flurbiprofen Ibuprofen Lornoxicam Meloxicam Piroxicam Tenoxicam	
	Opioids	Methadone	

SelfDecode

Category	Drug Class	✓ Standard Precautions	⚠ Use With Caution
Psychotropic	**Antidepressants**	Amitriptyline Bupropion Citalopram Clomipramine Doxepin Escitalopram Imipramine Trimipramine	**Sertraline**
	Antiepileptic	Phenytoin	
	Antipsychotics	Quetiapine	

Detailed prescribing guidance

Atorvastatin
Cardiovascular / Statins

Gene	Genotype	Phenotype
SLCO1B1	*1*15	Intermediate Metabolizer

The SLCO1B1*1 allele is assigned as a normal function allele by CPIC. The SLCO1B1*15 allele is assigned as a no function allele by CPIC. Patients with the *1*15 genotype (intermediate metabolizers) **may have increased atorvastatin concentrations** as compared to patients with two normal function alleles. ⭐⭐⭐

Patients with the *1*15 genotype **may have a higher risk of atorvastatin-related myopathy when treated with atorvastatin** as compared to patients with two normal function alleles. However, conflicting evidence has been reported. ⭐⭐⭐

CPIC recommends prescribing ≤40 mg as a starting dose and adjusting doses of atorvastatin based on disease-specific guidelines. Prescriber should be aware of possible increased risk for myopathy especially for 40mg dose. If dose >40 mg needed for desired efficacy, consider combination therapy (i.e., atorvastatin plus non-statin guideline directed medical therapy).

Other genetic and clinical factors may also affect atorvastatin pharmacokinetics and toxicity.

Dexlansoprazole
Gastrointestinal / Proton Pump Inhibitors

Gene	Genotype	Phenotype
CYP2C19	*1*1	Normal Metabolizer

The CYP2C19*1 allele is assigned as a normal function allele by CPIC. Patients with the *1*1 genotype (normal metabolizers) **may have increased metabolism of dexlansoprazole** compared to patients with a decreased or no function allele. ⭐⭐⭐

CPIC recommends initiating therapy with the standard starting daily dose and considering increasing dose by 50-100% for the treatment of H. pylori infection and erosive esophagitis. The daily dose may be given in divided doses. Monitor for efficacy.

Other genetic and clinical factors may also influence dexlansoprazole metabolism.

SelfDecode

⚠ ## Efavirenz

Infections / Antivirals

Gene	Genotype	Phenotype
CYP2B6	*1*6	Likely Intermediate Metabolizer

The CYP2B6*1 allele is assigned as a normal function allele by CPIC. The CYP2B6*6 allele is assigned as a decreased function allele by CPIC. Patients with the *1*6 genotype (likely intermediate metabolizers) **may have decreased metabolism of efavirenz** as compared to patients with two normal function alleles. However, conflicting evidence has been reported. ⭐⭐⭐

Patients with the *1*6 genotype **may require a decreased dose of efavirenz** compared to patients with two normal function alleles. ⭐⭐⭐

Patients with the *1*6 genotype **may have an increased risk of adverse events (eg. liver toxicity or CNS side effects) when treated with efavirenz** compared to patients with two normal function alleles. However, conflicting evidence has been reported. ⭐⭐⭐

CPIC recommends initiating efavirenz therapy with a decreased dose of 400 mg/day.

Other genetic and clinical factors may also influence efavirenz metabolism, dose requirements and toxicity.

⚠ ## Fluvastatin

Cardiovascular / Statins

Gene	Genotype	Phenotype
CYP2C9	*1*1	Normal Metabolizer

The CYP2C9*1 allele is assigned as a normal function allele by CPIC. Patients carrying the CYP2C9*1*1 genotype (normal metabolizers) **may have increased metabolism of fluvastatin** as compared to patients carrying at least one copy of a decreased function or no function allele. ⭐⭐⭐

Patients carrying the CYP2C9*1*1 genotype **may have a decreased likelihood of adverse events when treated with fluvastatin** as compared to patients carrying at least one copy of a decreased function or no function allele. However, conflicting evidence has been reported. ⭐⭐⭐

CPIC recommends prescribing the desired starting dose and adjusting doses of fluvastatin based on disease-specific guidelines.

Other genetic and clinical factors may also influence fluvastatin metabolism and toxicity.

Gene	Genotype	Phenotype
SLCO1B1	*1*15	Intermediate Metabolizer

The SLCO1B1*1 allele is assigned as a normal function allele by CPIC. The SLCO1B1*15 allele is assigned as a no function allele by CPIC. Patients with the *1*15 genotype (intermediate metabolizers) **may have increased fluvastatin concentrations when treated with fluvastatin** as compared to patients with two normal function alleles. However, conflicting evidence has been reported. ⭐⭐⭐

Patients with the *1*15 genotype **may have a higher risk of fluvastatin-related myopathy when treated with fluvastatin** as compared to patients with two normal function alleles. However, conflicting evidence has been reported. ⭐⭐⭐

CPIC recommends prescribing the desired starting dose and adjusting doses of fluvastatin based on disease-specific guidelines. Prescriber should be aware of possible increased risk for myopathy especially for doses > 40mg per day.

Other genetic and clinical factors may also influence the metabolism and the toxicity of fluvastatin.

Lansoprazole

Gastrointestinal / Proton Pump Inhibitors

Gene	Genotype	Phenotype
CYP2C19	***1*1**	Normal Metabolizer

The CYP2C19*1 allele is assigned as a normal function allele by CPIC. Patients with the *1*1 genotype (normal metabolizers) **may have increased metabolism of lansoprazole** compared to patients with at least one decreased or no function allele but **decreased metabolism of lansoprazole** compared to patients with two increased function alleles or an increased function allele in combination with a normal function allele. However, conflicting evidence has been reported. ⭐⭐⭐

Patients with the *1*1 genotype **may have a reduced response to lansoprazole (greater % of time with intragastric pH < 4.0, a lower intragastric pH during a 24-hour time period, poorer healing or cure rate of gastroesophageal reflux disease, decreased likelihood of *H. pylori* eradication, among other parameters)** compared to patients with at least one decreased or no function allele. However, conflicting evidence has been reported. ⭐⭐⭐

CPIC recommends initiating therapy with the standard starting daily dose. Consider increasing the dose by 50-100% for the treatment of H. pylori infection and erosive esophagitis. The daily dose may be given in divided doses. Monitor for efficacy.

Other genetic and clinical factors may also influence lansoprazole metabolism and clinical response.

Lovastatin

Cardiovascular / Statins

Gene	Genotype	Phenotype
SLCO1B1	***1*15**	Intermediate Metabolizer

The SLCO1B1*1 allele is assigned as a normal function allele by CPIC. The SLCO1B1*15 allele is assigned as a no function allele by CPIC. Patients with the *1*15 genotype (intermediate metabolizers) **may have increased lovastatin concentrations** as compared to patients with two normal function alleles. ⭐⭐⭐

Patients with the *1*15 genotype **may have a higher risk of lovastatin-related myopathy when treated with lovastatin** as compared to patients with two normal function alleles. ⭐⭐⭐

CPIC recommends prescribing an alternative statin depending on the desired potency. If lovastatin therapy is warranted, CPIC recommends limiting the dose to ≤20 mg/day.

Other genetic and clinical factors may also influence a patient's lovastatin pharmacokinetics and toxicity.

Methotrexate

Immunology, Rheumatology & Oncology / Immunosuppressants

Gene	Genotype	Phenotype
MTHFR	rs1801133 **AA**	

Patients with the rs1801133 AA genotype and arthritis or cancer who are treated with methotrexate **may be at increased risk of toxicity** as compared to patients with the AG or GG genotype. However, conflicting evidence has been reported. ⭐⭐

Other genetic and clinical factors may also influence methotrexate toxicity.

Omeprazole

Gastrointestinal / Proton Pump Inhibitors

Gene	Genotype	Phenotype
CYP2C19	*1*1	Normal Metabolizer

The CYP2C19*1 allele is assigned as a normal function allele by CPIC. Patients with the *1*1 genotype (normal metabolizers) **may have increased metabolism of omeprazole** compared to patients with a decreased or no function allele but **decreased metabolism of omeprazole** compared to patients with two increased function alleles or an increased function allele in combination with a normal function allele. However, conflicting evidence has been reported. ⭐⭐⭐

Patients with the *1*1 genotype **may have a reduced response to omeprazole (greater % of time with intragastric pH < 4.0, a lower intragastric pH during a 24-hour time period, decreased likelihood of *H. pylori* eradication, among other parameters)** compared to patients with at least one no or decreased function allele. However, conflicting evidence has been reported. ⭐⭐⭐

CPIC recommends initiating therapy with the standard starting daily dose. Considering increasing the dose by 50-100% for the treatment of H. pylori infection and erosive esophagitis. The daily dose may be given in divided doses. Monitor for efficacy.

Other genetic and clinical factors may also influence omeprazole metabolism and response.

Pantoprazole

Gastrointestinal / Proton Pump Inhibitors

Gene	Genotype	Phenotype
CYP2C19	*1*1	Normal Metabolizer

The CYP2C19*1 allele is assigned as a normal function allele by CPIC. Patients with the *1*1 genotype (normal metabolizers) **may have increased metabolism of pantoprazole** compared to patients with at least one decreased or no function allele but **decreased metabolism of pantoprazole** compared to patients with two increased function alleles or an increased function allele in combination with a normal function allele. ⭐⭐⭐

Patients with the *1*1 genotype **may have a reduced response to pantoprazole (greater % of time with intragastric pH < 4.0, a lower intragastric pH during a 24-hour time period, decreased likelihood of *H. pylori* eradication, among other parameters)** compared to patients with at least one no or decreased function allele. However, conflicting evidence has been reported. ⭐⭐⭐

CPIC recommends initiating therapy with the standard starting daily dose. Consider increasing dose by 50-100% for the treatment of H. pylori infection and erosive esophagitis. The daily dose may be given in divided doses. Monitor for efficacy.

Other genetic and clinical factors may also influence pantoprazole metabolism and response.

SelfDecode

⚠ Phenprocoumon
Cardiovascular / Antiplatelets & Anticoagulants

Gene	Genotype	Phenotype
VKORC1	rs9923231 **TC** rs9934438 **AG**	

Patients with the rs9923231 CT genotype **may require a lower dose of phenprocoumon** as compared to patients with the CC genotype. ⭐⭐⭐

Patients with the rs9923231 CT genotype **may have an increased risk of adverse events (bleeding, over-anticoagulation or increased time above therapeutic range) when treated with phenprocoumon** as compared to patients with the CC genotype. ⭐⭐

Patients with the rs9934438 AG genotype **may require a decreased dose of phenprocoumon as compared to patients with the GG genotype, but an increased dose as compared to patients with the AA genotype**. ⭐⭐

Other clinical and genetic factors may also influence dose requirements and the risk of adverse events due to phenprocoumon.

⚠ Pitavastatin
Cardiovascular / Statins

Gene	Genotype	Phenotype
SLCO1B1	***1*15**	Intermediate Metabolizer

The SLCO1B1*1 allele is assigned as a normal function allele by CPIC. The SLCO1B1*15 allele is assigned as a no function allele by CPIC. Patients with the *1*15 genotype (intermediate metabolizers) **may have increased exposure to pitavastatin** as compared to patients with two normal function alleles. ⭐⭐⭐

CPIC recommends prescribing ≤2 mg as a starting dose and adjusting doses of pitavastatin based on disease-specific guidelines. Prescriber should be aware of possible increased risk for myopathy especially for doses >1 mg. If dose >2 mg is needed for desired efficacy, consider an alternative statin or combination therapy (i.e. pitavastatin plus non-statin guideline directed medical therapy).

Other genetic or clinical factors may also affect a patient's exposure to pitavastatin.

SelfDecode

⚠ Pravastatin
Cardiovascular / Statins

Gene	Genotype	Phenotype
SLCO1B1	*1*15	Intermediate Metabolizer

The SLCO1B1*1 allele is assigned as a normal function allele by CPIC. The SLCO1B1*15 allele is assigned as a no function allele by CPIC. Patients with the *1*15 genotype (intermediate metabolizers) **may have increased bioavailability of pravastatin** as compared to patients with two normal function alleles. ⭐⭐⭐

Patients with the *1*15 genotype **may have a higher risk of pravastatin-related myopathy when treated with pravastatin** as compared to patients with two normal function alleles. However, conflicting evidence has been reported. ⭐⭐⭐

CPIC recommends prescribing the desired starting dose and adjusting doses of pravastatin based on disease-specific guidelines. Prescriber should be aware of possible increased risk for myopathy with pravastatin especially with doses >40 mg per day.

Other genetic and clinical factors may also influence the pharmacokinetics and the toxicity of pravastatin.

⚠ Rosuvastatin
Cardiovascular / Statins

Gene	Genotype	Phenotype
SLCO1B1	*1*15	Intermediate Metabolizer

The SLCO1B1*1 allele is assigned as a normal function allele by CPIC. The SLCO1B1*15 allele is assigned as a no function allele by CPIC. Patients with the *1*15 genotype (intermediate metabolizers) **may have increased exposure to rosuvastatin** as compared to patients with two normal function alleles. ⭐⭐⭐

Patients with the *1*15 genotype **may have a higher risk of rosuvastatin-related myopathy or myalgia when treated with rosuvastatin** as compared to patients with two normal function alleles. However, conflicting evidence has been reported. ⭐⭐⭐

CPIC recommends prescribing the desired starting dose and adjusting doses of rosuvastatin based on disease-specific and specific population guidelines. Prescriber should be aware of possible increased risk for myopathy especially for doses >20 mg.

Other genetic and clinical factors may also influence rosuvastatin pharmacokinetics.

Gene	Genotype	Phenotype
ABCG2	rs2231142 **GG**	

Patients with the rs2231142 GG genotype **may have decreased plasma concentrations of rosuvastatin when treated with rosuvastatin** as compared to patients with the TT or GT genotype. ⭐⭐⭐

Patients with the rs2231142 GG genotype and who are treated with **rosuvastatin may have a reduced response to treatment** as determined by a lower reduction in LDL-C as compared to patients with the GT or TT genotypes. ⭐⭐

Patients with the rs2231142 GG genotype **may have a lower risk of statin-related myopathy when treated with rosuvastatin** as compared to patients with the TT or GT genotype. However, conflicting evidence has been reported. ⭐⭐⭐

Other genetic and clinical factors may also influence the metabolism, efficacy, and toxicity of rosuvastatin.

SelfDecode

⚠ Sertraline

Psychotropic / Antidepressants

Gene	Genotype	Phenotype
CYP2B6	*1*6	Likely Intermediate Metabolizer

The CYP2B6*1 allele is assigned as a normal function allele by CPIC. The CYP2B6*6 allele is assigned as a decreased function allele by CPIC. Patients with the *1*6 genotype (likely intermediate metabolizers) **may have increased concentrations of sertraline** as compared to patients with two normal function alleles. ⭐⭐⭐

CPIC recommends initiating therapy with the recommended starting dose. CPIC also recommends considering a slower titration schedule and lower maintenance dose than CYP2B6 normal metabolizers.

Other genetic and clinical factors may also influence metabolism of sertraline.

Gene	Genotype	Phenotype
CYP2C19	*1*1	Normal Metabolizer

The CYP2C19*1 allele is assigned as a normal function allele by CPIC. Patients with the *1*1 genotype (normal metabolizers) **may have increased metabolism of sertraline** compared to patients with at least one decreased or no function allele but **decreased metabolism of sertraline** compared to patients with two increased function alleles or an increased function allele in combination with a normal function allele. However, conflicting evidence has been reported. ⭐⭐⭐

CPIC recommends initiating therapy with the recommended starting dose.

Other genetic and clinical factors may also influence sertraline metabolism.

⚠ Simvastatin

Cardiovascular / Statins

Gene	Genotype	Phenotype
SLCO1B1	*1*15	Intermediate Metabolizer

The SLCO1B1*1 allele is assigned as a normal function allele by CPIC. The SLCO1B1*15 allele is assigned as a no function allele by CPIC. Patients with the *1*15 genotype (intermediate metabolizers) **may have increased simvastatin acid concentration when treated with simvastatin** as compared to patients with two normal function alleles. ⭐⭐⭐

Patients with the *1*15 genotype **may have a higher risk of simvastatin-related myopathy when treated with simvastatin** as compared to patients with two normal function alleles. However, conflicting evidence has been reported. ⭐⭐⭐

CPIC recommends prescribing an alternative statin depending on the desired potency. If simvastatin therapy is warranted, CPIC recommends limiting the dose to <20 mg/day.

Other genetic and clinical factors may also influence the metabolism and toxicity of simvastatin.

⚠ Triple therapy (peginterferon alfa-2a/b & ribavirin)

Infections / Antivirals

Gene	Genotype	Phenotype
IFNL3	rs11881222 **GA** rs12979860 **TC** rs8099917 **GT**	

Patients with the rs11881222 AG genotype and hepatitis C or HIV **may have a poorer response (SVR) to treatment with peginterferon alpha-2a/2b in combination with ribavirin** as compared to patients with the AA genotype. ⭐⭐

Patients with the s12979860 CT genotype and hepatitis C infection **may have an intermediate response (SVR) when administered peg interferon alpha-2a/2b in combination with ribavirin**, decreased compared to patients with the CC genotype but increased compared to patients with the TT genotype. However, conflicting evidence has been reported. ⭐⭐⭐

Patients with the rs12979860 CT genotype and hepatitis C infection **may have lower response rates (SVR) to triple therapy (telaprevir or boceprevir, peginterferon alfa-2a/b and ribavirin)** as compared to patients with the CC genotype. However, conflicting evidence has been reported. ⭐⭐⭐

According to CPIC, rs12979860 is the strongest baseline predictor of response to peginterferon and ribavirin therapy in patients with hepatitis C. Patients with this genotype have approximately a 30% chance for SVR after 48 weeks of treatment. If the regimen is combined with protease inhibitors, there is approximately 60% chance for SVR after 24-48 weeks of treatment and approximately 50% of patients are eligible for shortened therapy (24-48 weeks).

Patients with the rs8099917 GT genotype and chronic hepatitis C infection **may have a decreased response (SVR) to peginterferon alfa-2a/2b in combination with ribavirin** as compared to patients with the TT genotype. However, conflicting evidence has been reported. ⭐⭐⭐

Patients with the rs8099917 GT genotype and hepatitis C infection **may have lower response rates (SVR) to triple therapy (telaprevir, peginterferon alfa-2a/b, and ribavirin)** as compared to patients with the TT genotype. ⭐⭐

Other genetic and clinical factors may also influence response to triple therapy. Importantly, the impact of the genotype may be dampened in patients with prior treatment failure.

> ⚠️ # Warfarin
> Cardiovascular / Antiplatelets & Anticoagulants

Gene	Genotype	Phenotype
CYP2C9	***1*1**	Normal Metabolizer

The CYP2C9*1 allele is assigned as a normal function allele by CPIC. Patients carrying the CYP2C9*1*1 genotype (normal metabolizers) **may require a higher dose of warfarin** as compared to patients carrying at least one copy of a decreased function or no function allele. ⭐⭐⭐

Patients carrying the CYP2C9*1*1 genotype **may require less time to achieve a stable dose when treated with warfarin** as compared to patients carrying at least one copy of a decreased function or no function allele. ⭐⭐

Patients carrying the CYP2C9*1*1 genotype **may have a decreased risk of over-anticoagulation and bleeding when treated with warfarin** as compared to patients carrying at least one copy of a decreased function or no function allele. ⭐⭐⭐

Other genetic and clinical factors may also influence the required dose, efficacy, and toxicity of warfarin.

Gene	Genotype	Phenotype
CYP4F2	rs2108622 **CC**	

Patients with the rs2108622 CC genotype **may have decreased warfarin dosage requirements** as compared to patients with the CT or TT genotype. However, conflicting evidence has been reported. ⭐⭐⭐

Other genetic and clinical factors may also affect warfarin dosage requirements.

Gene	Genotype	Phenotype
VKORC1	rs2359612 **AG** rs2884737 **CA** rs61742245 **CC** rs7294 **CT** rs8050894 **GC** rs9923231 **TC** rs9934438 **AG**	

Patients with the rs2359612 AG genotype **may require a decreased dose of warfarin** as compared to patients with the GG genotype. ⭐⭐⭐

Patients with the rs2884737 AC genotype **may require a higher dose of warfarin** as compared to patients with the CC genotype. ⭐⭐

Patients with the rs61742245 CC genotype **may require a decreased dose of warfarin** as compared to patients with the AA or AC genotype. However, conflicting evidence has been reported. ⭐⭐

Patients with the rs7294 CT genotype **may require a higher dose of warfarin** as compared to patients with the CC genotype. However, conflicting evidence has been reported. ⭐⭐⭐

Patients with the rs8050894 CG genotype **may require a lower dose of warfarin** as compared to patients with the CC genotype. ⭐⭐⭐

Patients with the rs9923231 CT genotype **may require a decreased dose of warfarin** as compared to patients with the CC genotype or an increased dose as compared to patients with the TT genotype. They **may have an increased risk of over-anticoagulation when treated with warfarin** as compared with patients with the CC genotype. However, conflicting evidence has been reported. ⭐⭐⭐

Patients with the rs9923231 CT genotype **may have an increased risk of bleeding when treated with warfarin** as compared to patients with the CC genotype. However, conflicting evidence has been reported. ⭐⭐

Patients with the rs9923231 CT genotype **may require shorter time to therapeutic INR when treated with warfarin** and **may spend less time in the INR therapeutic range (TTR) when treated with warfarin** as compared with patients with the CC genotype. However, conflicting evidence has been reported. ⭐⭐

Patients with the rs9934438 AG genotype **may require a lower dose of warfarin as compared to patients with the GG genotype, and a higher dose as compared to patients with the AA genotype**. However, conflicting evidence has been reported. ⭐⭐⭐

Other genetic and clinical factors may also influence warfarin dose requirement and response to warfarin.

Family Planning (Carrier Status)

Our Carrier Status Reports provide information about variants that may not affect your health, but may increase your chances of having a child with a genetic disease. **Knowing your carrier status is valuable when you want to start a family.**

Report results

You may get the following results for your carrier status reports:

✓ **VARIANT NOT DETECTED**	Likely not a carrier. May still have a rare variant we didn't check for in the report.

⚠ **VARIANT DETECTED**	Likely a carrier. May pass a variant on to their child.

? **NOT DETERMINED**	Not determined. In rare cases, the report cannot provide a conclusive result.

Results Overview

Risk	Condition	Result
✓	Beta-Thalassemia and Related Hemoglobinopathies	Variant not detected
✓	Canavan Disease	Variant not detected
✓	Congenital Disorder of Glycosylation Type 1a (PMM2-CDG)	Variant not detected
✓	Cystic Fibrosis	Variant not detected
✓	Dihydrolipoamide Dehydrogenase Deficiency	Variant not detected
✓	Familial Dysautonomia	Variant not detected
✓	Familial Hyperinsulinism (ABCC8-Related)	Variant not detected
✓	Familial Mediterranean Fever	Variant not detected
✓	Fanconi Anemia Group C	Variant not detected
✓	Gaucher Disease Type 1	Variant not detected
✓	Glycogen Storage Disease Type Ia	Variant not detected
✓	Glycogen Storage Disease Type Ib	Variant not detected
✓	GRACILE Syndrome	Variant not detected
✓	Hereditary Fructose Intolerance	Variant not detected
✓	Leigh Syndrome, French Canadian Type	Variant not detected
✓	Limb-Girdle Muscular Dystrophy Type 2D	Variant not detected
✓	Limb-Girdle Muscular Dystrophy Type 2E	Variant not detected
✓	Limb-Girdle Muscular Dystrophy Type 2I	Variant not detected
✓	Maple Syrup Urine Disease Type 1B	Variant not detected

Risk	Condition	Result
✓	Mucolipidosis Type IV	Variant not detected
✓	Neuronal Ceroid Lipofuscinosis (PPT1-Related)	Variant not detected
✓	Niemann-Pick Disease Type 1A	Variant not detected
✓	Pendred Syndrome and DFNB4 Hearing Loss (SLC26A4-Related)	Variant not detected
✓	Phenylketonuria and Related Disorders	Variant not detected
✓	Pompe Disease	Variant not detected
✓	Primary Hyperoxaluria Type 2	Variant not detected
✓	Pyruvate Kinase Deficiency	Variant not detected
✓	Rhizomelic Chondrodysplasia Punctata Type 1	Variant not detected
✓	Salla Disease	Variant not detected
✓	Severe Junctional Epidermolysis Bullosa (LAMB3-Related)	Variant not detected
✓	Sickle Cell Anemia	Variant not detected
✓	Sjögren-Larsson Syndrome	Variant not detected
✓	Tay-Sachs Disease	Variant not detected
✓	Tyrosinemia Type I	Variant not detected
✓	Usher Syndrome Type 1F	Variant not detected
✓	Usher Syndrome Type 3A	Variant not detected
✓	Zellweger Spectrum Disorder (PEX1-Related)	Variant not detected
?	Agenesis of the Corpus Callosum with Peripheral Neuropathy	Not determined
?	Neuronal Ceroid Lipofuscinosis (CLN5-Related)	Not determined