UGENOME

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Inflammation & Autoimmunity

Summary Report

REPORT CATEGORY

INFLAMMATION & AUTOIMMUNITY

Sample Client

Report date: 30 April 2026

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DISCLAIMER

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

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

Personal information

NAME

Sample Client

SEX AT BIRTH

Male

HEIGHT

5ft 10" 178cm

WEIGHT

215lb 97.5kg

REPORT PROVIDED BY

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Summary

Inflammation and autoimmunity are key processes that affect the body's ability to maintain health and respond to threats. Inflammation is the body's natural response to injury or infection, but chronic inflammation can lead to a variety of health problems, from gut issues to systemic diseases. Autoimmune conditions occur when the immune system mistakenly attacks healthy tissue, creating inflammation and damaging the tissues and organs. Both inflammatory and autoimmune conditions have **strong genetic components**.

This report explores the genetic markers that may increase your risk for various autoimmune and inflammatory conditions. From gut inflammation to skin conditions like psoriasis and systemic issues like lupus, the report offers insights into your body's inflammatory and immune responses. Additionally, it examines key biomarkers and genes, including inflammation markers such as CRP and cytokines, and autoimmune-related genes like *HLA*.

By understanding your genetic predisposition to these conditions, you can take proactive steps to manage inflammation, support your immune system, and potentially reduce the risk of developing autoimmune diseases.

This summary report contains:

- **83** Genetic Results
- **15** Recommendations
- **2** Lifestyle Assessments

Overview of Your Results

Inflammatory Gut Conditions

MORE LIKELY

Ulcerative Colitis

More likely to have ulcerative colitis

MORE LIKELY

Crohn’s Disease

More likely to get Crohn's disease

TYPICAL LIKELIHOOD

Stomach Inflammation

Typical likelihood of gastritis

TYPICAL LIKELIHOOD

Celiac Disease

Typical likelihood of celiac disease

LESS LIKELY

Gut Inflammation

Less likely to have IBD

LESS LIKELY

Appendicitis

Less likely to have appendicitis

LESS LIKELY

Diverticular Disease

Less likely to have diverticular disease

Other Inflammatory Conditions

MORE LIKELY

Eczema

More likely to have eczema

MORE LIKELY

Pancreas Inflammation

More likely to get pancreas inflammation

MORE LIKELY

Nephritis

More likely to have nephritis

TYPICAL LIKELIHOOD

Chronic Lyme

Typical likelihood of having “chronic Lyme”

TYPICAL LIKELIHOOD

Tonsil Inflammation

Typical likelihood of tonsillitis

TYPICAL LIKELIHOOD

Myocarditis

Typical likelihood of myocarditis

TYPICAL LIKELIHOOD

Sepsis

Typical likelihood of sepsis

TYPICAL LIKELIHOOD

Actinic Keratosis

Typical likelihood of an actinic keratosis

TYPICAL LIKELIHOOD

Sarcoidosis

Typical likelihood of sarcoidosis

TYPICAL LIKELIHOOD

Rheumatic Fever

Typical likelihood of rheumatic fever

LESS LIKELY

Thyroid Inflammation

Less likely to have thyroid inflammation

LESS LIKELY

Eye Inflammation

Less likely to have uveitis

LESS LIKELY

Optic Nerve Inflammation

Less likely to have optic neuritis

LESS LIKELY

Urethra Inflammation

Less likely to have non-specific urethritis

LESS LIKELY

Pulmonary Fibrosis

Less likely to have pulmonary fibrosis

LESS LIKELY

Meningitis

Less likely to have meningitis

Autoimmune Joint Conditions

TYPICAL LIKELIHOOD

Rheumatoid Arthritis

Typical likelihood of rheumatoid arthritis

TYPICAL LIKELIHOOD

Lupus

Typical likelihood of lupus

TYPICAL LIKELIHOOD

Psoriatic Arthritis

Typical likelihood of psoriatic arthropathy

TYPICAL LIKELIHOOD

Ankylosing Spondylitis

Typical likelihood of ankylosing spondylitis

Autoimmune Skin Conditions

MORE LIKELY

Psoriasis

More likely to have psoriasis

TYPICAL LIKELIHOOD

Vitiligo

Typical likelihood of vitiligo

TYPICAL LIKELIHOOD

Alopecia Areata

Typical

TYPICAL LIKELIHOOD

Pemphigus Vulgaris

Typical likelihood of pemphigus vulgaris

TYPICAL LIKELIHOOD

Behcet’s Disease

Typical likelihood of Behcet's disease

LESS LIKELY

Systemic Sclerosis

Less likely to have systemic sclerosis

Other Autoimmune Conditions

TYPICAL LIKELIHOOD

Multiple Sclerosis

Typical likelihood of multiple sclerosis

TYPICAL LIKELIHOOD

Type 1 Diabetes

Typical likelihood of type 1 diabetes

TYPICAL LIKELIHOOD

IgA Nephropathy

Typical likelihood of IgA nephropathy

TYPICAL LIKELIHOOD

Myasthenia Gravis

Typical likelihood of myasthenia gravis

TYPICAL LIKELIHOOD

Antiphospholipid Syndrome

Typical likelihood of antiphospholipid syndrome

LOWER PREDISPOSITION

Amyotrophic Lateral Sclerosis (ALS)

Lower predisposition to ALS

LESS LIKELY

Sjogren's Syndrome

Less likely to have Sjögren's syndrome

Inflammation Markers

HIGHER LEVELS

IgE

Predisposed to higher IgE levels

LOWER LEVELS

IL-10

Predisposed to lower IL-10 levels

TYPICAL LEVELS

Inflammation (CRP)

Predisposed to typical CRP levels

TYPICAL LEVELS

TNF

Predisposed to typical TNF levels

TYPICAL LEVELS

White Blood Cells

Predisposed to typical WBC count

TYPICAL LEVELS

Basophils

Predisposed to typical basophil levels

TYPICAL LEVELS

Eosinophils

Predisposed to typical eosinophil levels

TYPICAL LEVELS

Monocytes

Predisposed to typical monocyte levels

TYPICAL LEVELS

Neutrophils

Predisposed to typical neutrophil levels

TYPICAL LEVELS

Erythrocyte Sedimentation Rate

Predisposed to typical ESR levels

TYPICAL LEVELS

IL-17 (Th17)

Predisposed to typical IL-17 levels

TYPICAL LEVELS

IL-6

Predisposed to typical IL-6 levels

HLA Genes

HIGHER ACTIVITY

HLA (Inflammation)

Likely higher HLA activity

HIGHER ACTIVITY

HLA-DOB (Inflammation)

Likely higher HLA-DOB activity

TYPICAL

HLA-B (Autoimmune)

Likely typical HLA-B genetics

TYPICAL ACTIVITY

HLA-DQ (Inflammation)

Likely typical HLA-DQ activity

TYPICAL

HLA-DRB1 (Autoimmunity)

Likely typical HLA-DRB1 genetics

TYPICAL

HLA-DQA2 (Inflammation)

Likely typical HLA-DQA2 genetics

Other Inflammation Genes

HIGHER ACTIVITY

NF-kB (Inflammation)

Likely higher NF-kB activity

HIGHER ACTIVITY

NLRP3 (Gut Inflammation)

Likely higher NLRP3 activity

LOWER ACTIVITY

SH2B3 (Autoimmunity)

Likely lower SH2B3 activity

LOWER ACTIVITY

HNF4A (Gut Inflammation)

Likely lower HNF4A activity

HIGHER ACTIVITY

IL6R (Weight)

Predisposed to higher IL6R activity

HIGHER ACTIVITY

ANKRD55 (Autoimmune)

Predisposed to higher ANKRD55 activity

LOWER ACTIVITY

CTLA4 (Autoimmunity)

Likely lower CTLA4 activity

TYPICAL ACTIVITY

TLR4 (Inflammation)

Likely typical TLR4 activity

TYPICAL

PTPN22 (Autoimmunity)

Likely typical PTPN22 genetics

TYPICAL ACTIVITY

DAO (Histamine)

Likely typical DAO activity

TYPICAL ACTIVITY

STAT3 (Th1/Th17)

Likely typical STAT3 activity

TYPICAL ACTIVITY

TNF Gene (Inflammation)

Likely typical TNF activity

TYPICAL ACTIVITY

IL13 (Allergies, Lung Function)

Predisposed to typical IL13 activity

TYPICAL ACTIVITY

IL4 (Allergies, Autoimmunity)

Likely typical IL4 activity

TYPICAL ACTIVITY

HRH4 (Allergies, Inflammation)

Predisposed to typical HRH4 activity

TYPICAL ACTIVITY

CRP Gene

Predisposed to typical CRP gene activity

TYPICAL ACTIVITY

PADI4 (Autoimmune)

Predisposed to typical PADI4 activity

TYPICAL ACTIVITY

IL10 Gene (Autoimmunity)

Predisposed to typical IL10 activity

TYPICAL GENETICS

SLC22A4 (Gut Inflammation)

Likely typical SLC22A4 genetics

LOWER ACTIVITY

IL1B (Inflammation/ Fatigue)

Likely lower IL1B activity

HIGHER ACTIVITY

ABCB1 (Chronic Lyme)

Likely higher ABCB1 activity

HIGHER ACTIVITY

HNMT (Histamine)

Likely higher HNMT activity

LOWER ACTIVITY

JAK2 (Gut Inflammation)

Likely lower JAK2 activity

LOWER ACTIVITY

IL8 (Inflammation)

Likely lower IL8 activity

LOWER ACTIVITY

IL21 (Autoimmunity & Allergies)

Predisposed to lower IL21 activity

Recommendations Overview

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

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

		DOSAGE
1	Probiotics	30 billion CFU
2	Maintain a Healthy Weight	30 minutes
3	Omega-3 (Fish Oil)	2000 mg
4	Maintain Optimal Vitamin D Levels	1000 iu
5	Relaxation Techniques	30 minutes
6	Dietary Omega-3 Fatty Acids	
7	Curcumin	500 mg
8	Aerobic Exercise (Cardio)	1 hour
9	Yoga	30 minutes
10	Mediterranean Diet	
11	Mindfulness	30 minutes
12	Acupuncture	1 hour
13	Guided Imagery	30 minutes
14	Avoid Food Triggers	
15	Avoid Processed Foods	

Your Results in Details

Inflammatory Gut Conditions

Gut health is deeply linked to the body's inflammatory responses, and genetic predispositions can make some individuals more susceptible to inflammatory gut conditions. This section examines the genetic markers associated with disorders such as Crohn's disease, ulcerative colitis, celiac disease, and more.

Understanding your genetic risk for these conditions can help you take preventive steps, such as dietary adjustments or early medical intervention, to maintain gut health and reduce inflammation. By identifying potential risks, you can better manage symptoms and support your digestive health.

MORE LIKELY

Ulcerative Colitis

More likely to have ulcerative colitis

MORE LIKELY

Crohn's Disease

More likely to get Crohn's disease

TYPICAL LIKELIHOOD

Stomach Inflammation

Typical likelihood of gastritis

TYPICAL LIKELIHOOD

Celiac Disease

Typical likelihood of celiac disease

LESS LIKELY

Gut Inflammation

Less likely to have IBD

LESS LIKELY

Appendicitis

Less likely to have appendicitis

LESS LIKELY

Diverticular Disease

Less likely to have diverticular disease

Ulcerative Colitis

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.

MORE LIKELY

More likely to have ulcerative colitis based on 1,049,227 genetic variants we looked at

70th

PERCENTILE

Your risk is **greater than 70% of the population** and **lower than 30% of the population.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
STAT3	rs744166	AA
HNF4A	rs6017342	CC
HLA-DQA2	rs2395185	GT
IL23R	rs76418789	GG
IL23R	rs11805303	CT
/	rs113653754	CC
MFSD4B	rs3851228	AA
NR5A2	rs2816958	GG
IL19	rs1800872	GG
FCGR2A	rs1801274	AA
IRF5	rs4728142	GA
RORC	rs4845604	GG
KIAA1841	rs7608910	GG
OTUD3	rs6426833	AG
SLC39A11	rs17780256	AA
ETS2	rs2836878	AG
IL19	rs3024505	AG
INAVA	rs7554511	CA
IL23R	rs2201841	AG
TNFAIP3	rs6920220	GA
IL12B	rs56167332	AC

GENE	SNP	GENOTYPE
TNFSF15	rs11554257	CT
CARD9	rs13300218	GA
CARD9	rs10781499	AG
IRGM	rs11741861	AG
ADO	rs10761659	AG
NCR3	rs3749946	CC
IL23R	rs80174646	GG
TLR4	rs4986790	AA
IL19	rs1800896	CT
JAK2	rs1830610	CC
LRRK2	rs12422544	CT
DLD	rs2158836	GG
MST1	rs3197999	GG
TMCO4	rs3806308	CC
MDM1	rs7134472	GG
NKX2-3	rs4409764	TT
TYK2	rs12720356	AA
CELSR3	rs9868809	CC
GPR35	rs3749171	CT
CIITA	rs4781011	GG
PDGFB	rs2413583	CC
PARK7	rs3766606	GG
GPR12	rs17085007	TT
IKZF3	rs12946510	CT
DLD	rs4380874	CC
GALC	rs8005161	CC
HSPG2	rs12568930	TT
CCR1	rs113010081	TT
NXPE4	rs561722	TT

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

Crohn’s Disease

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.

MORE LIKELY

More likely to get Crohn's disease based on 1,031,499 genetic variants we looked at

69th

PERCENTILE

Your risk is **greater than 69% of the population** and **lower than 31% of the population.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SLC23A1	rs10063949	CC
ATG16L1	rs2241880	GG
STAT3	rs744166	AA
SLC22A5	rs12521868	TT
JAK2	rs10758669	CA
IL23R	rs11465804	TT
IRGM	rs1000113	CT
PPM1M	rs5743836	GG
NKX2-3	rs10883365	GG
AGT	rs5051	TT
CUL1	rs7807268	GG
IL23R	rs11805303	CT
PTPN22	rs2476601	GG
IRGM	rs4958847	GA
IRGM	rs13361189	TC
ATG16L1	rs3828309	GG
LRRK2	rs11175593	TC
BTBD8	rs34856868	GG
SLC22A5	rs17622378	GG
NKX2-3	rs11190140	TT
IRF8	rs2361755	GG

GENE	SNP	GENOTYPE
PTGER4	rs1992660	TC
KLF6	rs6601764	CC
SLC22A5	rs2188962	TT
IRGM	rs11741861	AG
NRIP1	rs2823286	GG
ITLN1	rs2274910	CC
MFSD4B	rs3851228	AA
ADO	rs10995271	GC
IRGM	rs11747270	AG
RNASET2	rs2301436	CT
ADO	rs10761659	AG
IL12B	rs56167332	AC
CARD9	rs13300218	GA
CARD9	rs10781499	AG
CACNA1S	rs11584383	TC
IL19	rs3024505	AG
INAVA	rs7554511	CA
CUL2	rs17582416	TG
GPX4	rs2024092	AG
ICAM5	rs11879191	AG
CCL13	rs3091315	GA
CUL2	rs11010067	CG
HLA-DQB1	rs9469220	GA
IKZF3	rs2872507	GA
C1ORF141	rs17375018	GA
CDKAL1	rs6908425	CT
UBLCP1	rs10045431	CA
TRIB1	rs1551398	AG
NOD2	rs2066844	CC

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

Stomach Inflammation

Symptoms of gastritis vary among individuals and can range from mild discomfort to severe pain. Common symptoms include upper abdominal pain or burning, nausea, vomiting, bloating, and a feeling of fullness in the upper abdomen after eating. If left untreated, gastritis can lead to stomach ulcers and an increased risk of stomach bleeding, which may be life-threatening.

In some cases, chronic gastritis may increase the risk of developing stomach polyps or stomach tumors. Treatment for gastritis generally involves a combination of medications to reduce stomach acid and a change in diet or lifestyle habits that may be contributing to the inflammation.

TYPICAL LIKELIHOOD

Typical likelihood of gastritis based on 9,903 genetic variants we looked at

48th

PERCENTILE

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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MRPL13	rs7826906	AG
CCDC66	rs6445797	GG
MTBP	rs10955971	GA
CDC37	rs8112449	AG
TCP10L	rs11088226	CC

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

Celiac Disease

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].

TYPICAL LIKELIHOOD

Typical likelihood of celiac disease based on 1,019,187 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HLA-DRB5	rs2395182	GT
HLA-DQA2	rs7454108	TT
HLA-DQA1	rs2187668	CC

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

Gut Inflammation

Key Takeaways:

- Up to **75%** of differences in people's chances of developing IBD may be due to genetics.
- Risk factors include being under age 30, European ancestry, and smoking.
- IBD may cause: diarrhea, fatigue, abdominal pain, bloody stool, weight loss, inflammation, liver damage, and colon cancer.
- IBD only affects about **3 in 1000** people worldwide. So, even with high genetic risk, your overall risk is actually low.
- Click the **Recommendations** tab for potential dietary and lifestyle changes and **next steps** for relevant labs.

Our intestines do much more than absorb food. They can impact our immune system, mood, and more [R]!

Inflammatory bowel disease (IBD) is a group of gut diseases affecting **about 0.3% of people worldwide.** It's most common in North America, Europe, and Australia [R].

The exact causes of IBD are unknown. Possible risk factors include [R]:

- Age (most people develop IBD before the age of 30)
- European ancestry
- Cigarette smoking
- **Genetics**

There are two major types of IBD: ulcerative colitis and Crohn's disease. Ulcerative colitis involves inflammation in the large intestine, while Crohn's disease often affects both the large and small intestines [R, R, R].

In both types of IBD, the immune system reacts to normal gut bacteria as if they're dangerous. These immune reactions cause inflammation and damage to the gut lining [R].

This gut damage can cause signs and symptoms like [R, R, R]:

- Diarrhea
- Fatigue
- Abdominal pain and cramping
- Blood in the stool
- Low appetite
- Weight loss

Untreated IBD can have serious complications, including [R]:

- Skin, eye, and joint inflammation
- Bile duct and liver damage
- Blood clots
- Colon cancer

People with IBD typically need anti-inflammatory medications to control their disease [R, R].

Many people with IBD take supplements because their damaged guts have trouble absorbing certain nutrients. Some people may need to adhere to special diets as well [R, R].

IBD can be a disabling condition, and many turn to alternative and complementary strategies to help them manage their symptoms. Your DNA may help determine which of these strategies is likely to work best for you.

LESS LIKELY

Less likely to have IBD based on 1,671 genetic variants we looked at

10th

PERCENTILE

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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
FCGR2A	rs1801274	AA
ATG16L1	rs2241880	GG
STAT3	rs744166	AA
SLC22A5	rs12521868	TT
JAK2	rs10758669	CA
ADO	rs10761659	AG
HNF4A	rs6017342	CC
IL23R	rs11465804	TT
IL23R	rs11209026	GG
HLA-DQA2	rs2395185	GT
IL23R	rs10889677	CA
IRGM	rs1000113	CT
ATG16L1	rs10210302	TT
PPM1M	rs5743836	GG
TNFSF15	rs6478108	TC
IL23R	rs76418789	GG
IRGM	rs10065172	CT
NKX2-3	rs10883365	GG
TNF	rs1799724	CT
AGT	rs5051	TT
STAT3	rs4796793	CC

Up to 75% of differences in people's chances of developing IBD may be attributed to genetics. Genes involved in IBD may influence [R, R, R, R]:

- Inflammation (*JAK2*, *TNFSF15*, *SLAMF8*)
- Immune response (*TLR9*, *UBE2L3*, *BCL3*)

Moreover, genetically high betaine levels may be causally associated with a high risk of Crohn's disease. In contrast, genetically high levels of omega-3s may be causally associated with a lower risk [R, R, R, R].

GENE	SNP	GENOTYPE
CUL1	rs7807268	GG
STAT3	rs2293152	CG
IL23R	rs11805303	CT
CLEC4G	rs4804803	GG
PTPN22	rs2476601	GG
TNFSF8	rs6478109	GA
GLYCTK	rs352140	TT
IRGM	rs4958847	GA
NLRP3	rs10754558	CG
IRGM	rs13361189	TC
ATG16L1	rs3828309	GG
LRRK2	rs11175593	TC
IL23R	rs7517847	TT
GLYCTK	rs352139	CC
STAT3	rs9891119	AA
PDGFB	rs2413583	CC
NKX2-3	rs11190140	TT
PTGER4	rs1992660	TC
IL19	rs1800872	GG
IRF5	rs4728142	GA
NKX2-3	rs4409764	TT
ZNF365	rs7076156	AG
TNFSF15	rs4263839	GA
KLF6	rs6601764	CC
SLC22A5	rs2188962	TT
NRIP1	rs2823286	GG
TNFSF15	rs3810936	CT
IRF8	rs10521318	CC
LRRK2	rs11564258	AG

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

Appendicitis

Key Takeaways:

- Up to **56%** of differences in people's chances of developing appendicitis may be due to genetics.
- Other risk factors include being young and male.
- Appendicitis is not rare, happening to about 7-8% of people in their lifetime.
- If your genetic risk is high, know the symptoms and seek medical attention if you have them.
- Click the **Recommendations** tab for potential dietary and lifestyle changes and **next steps** for relevant labs.

The **appendix** is a small, finger-shaped pouch near the beginning of the large intestine. It is in the lower right of your abdomen [R, R].

The function of the appendix has been debated for many years. More recent studies suggest that the appendix is a safe house for good bacteria that live in the gut. If an illness wipes out large numbers of these bacteria in the gut, the ones from the appendix can help replace them [R].

Appendicitis is inflammation of the appendix. It is likely caused by something blocking the lining of the appendix, leading to an infection. If left untreated, the appendix can rupture and the infection can spread. This can be life-threatening [R].

Although anyone can develop appendicitis, it most often occurs in people between 10 and 30 years old. Men are slightly more likely to experience it than women [R, R].

The symptoms of appendicitis include [R]:

- Sudden pain in the lower right abdomen
- Sudden pain around the belly button that shifts to the lower right abdomen
- Pain that worsens if you move suddenly
- Nausea and vomiting
- Loss of appetite
- Fever
- Gut issues

The standard treatment for appendicitis is surgery to remove the appendix [R].

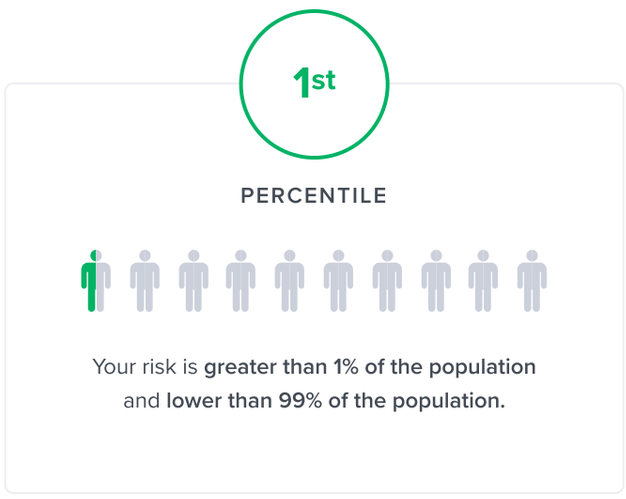
Up to 56% of differences in people's chances of developing appendicitis may be attributed to genetics. Involved genes may influence [R, R]:

- Gut development
- Gut function
- Inflammation

Genetically predicted higher levels of fasting insulin may be associated with appendicitis [R].

LESS LIKELY

Less likely to have appendicitis based on 805,324 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ENPEP	rs2129979	GT
TUB	rs72848490	TC
MTARC1	rs3738182	GG
PITX2	rs7697491	AT
PITX2	rs13121924	AG
LTBR	rs10849448	AG
DLEU7	rs201768	CT
/	rs77114860	TT
KRT73	rs146783619	AA
OSR1	rs56259011	CC
NKX2-3	rs41290504	CC
NKX2-3	rs7095491	TT

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

Diverticular Disease

When diverticula become inflamed or infected, the condition can cause more severe symptoms. These include severe abdominal pain, fever, nausea, and a marked change in bowel habits. Complications of diverticular disease include abscesses, perforation of the colon, peritonitis, fistulas, and intestinal obstruction.

Treatment typically involves a combination of antibiotics for infection and a liquid or low-fiber diet to allow the colon to heal. In some severe cases, surgery may be necessary to remove the affected portion of the colon. Lifestyle changes, such as increasing the intake of fiber, drinking plenty of fluids, and exercising regularly can help to manage symptoms and prevent future episodes.

LESS LIKELY

Less likely to have diverticular disease based on 996,903 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
POLR3A	rs139044580	GG
TBX5	rs2551395	TT
UPF2	rs78114187	CC
LYPLAL1	rs61823192	CC
SHANK1	rs113101273	GG
SOCS6	rs79586998	CC
ARHGAP15	rs6734367	GT
/	rs12814314	GA
LHFPL3	rs6980251	AC
YARS2	rs191699548	GG
/	rs190502327	CC
MGAT4A	rs148376933	CC
BHLHE23	rs6011570	GG
OMG	rs150700179	AA
SPDYE21	rs189921337	CC
FIBCD1	rs182799903	CC
INSIG1	rs138074947	TT
UBL4B	rs115490395	TT
NOC4L	rs73486316	GG
IFNAR2	rs138262539	AA
DIRAS2	rs78628486	GG
ROBO1	rs183318239	AA
VAPA	rs80261783	CC
EPB41L3	rs141727959	GG
SLCO5A1	rs117969400	GG
FAM180A	rs113477191	CC
MPP7	rs12219062	CC
ZNF28	rs140094156	CC
CIB4	rs74887298	GG

GENE	SNP	GENOTYPE
SLC35F3	rs4333882	AA
TMEM270	rs3823878	GG
TMEM101	rs71371957	AA
EFEMP1	rs1802575	GG
COL6A1	rs75434097	GG
IVD	rs71472433	AA

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

Other Inflammatory Conditions

Inflammation is not limited to the gut; it can affect many parts of the body, leading to conditions such as asthma, myocarditis, and even chronic infections like Lyme disease. This section explores your genetic predisposition to a variety of inflammatory conditions, including those affecting the thyroid, lungs, pancreas, and skin.

Understanding these risks can help you take steps to minimize chronic inflammation, improve overall health, and prevent potential complications. Proactive management of inflammation may include lifestyle changes, medical treatments, or therapies tailored to your specific genetic profile.

MORE LIKELY

Eczema

More likely to have eczema

MORE LIKELY

Pancreas Inflammation

More likely to get pancreas inflammation

MORE LIKELY

Nephritis

More likely to have nephritis

TYPICAL LIKELIHOOD

Chronic Lyme

Typical likelihood of having “chronic Lyme”

TYPICAL LIKELIHOOD

Tonsil Inflammation

Typical likelihood of tonsillitis

TYPICAL LIKELIHOOD

Myocarditis

Typical likelihood of myocarditis

TYPICAL LIKELIHOOD

Sepsis

Typical likelihood of sepsis

TYPICAL LIKELIHOOD

Actinic Keratosis

Typical likelihood of an actinic keratosis

TYPICAL LIKELIHOOD

Sarcoidosis

Typical likelihood of sarcoidosis

TYPICAL LIKELIHOOD

Rheumatic Fever

Typical likelihood of rheumatic fever

LESS LIKELY

Thyroid Inflammation

Less likely to have thyroid inflammation

LESS LIKELY

Eye Inflammation

Less likely to have uveitis

LESS LIKELY

Optic Nerve Inflammation

Less likely to have optic neuritis

LESS LIKELY

Urethra Inflammation

Less likely to have non-specific urethritis

LESS LIKELY

Pulmonary Fibrosis

Less likely to have pulmonary fibrosis

LESS LIKELY

Meningitis

Less likely to have meningitis

Eczema

Key Takeaways:

- Up to **75%** of differences in people's chances of developing eczema may be due to genetics.
- Eczema triggers include: allergens, cold, dry air, infections, skin irritants, and stress.
- It can affect your appearance and quality of life.
- If you have a high genetic risk, take special care to avoid potential triggers.
- Click the **Recommendations** tab for potential dietary and lifestyle changes.

Eczema is an inflammatory skin condition. It causes dry skin and itchy red rashes, usually on the elbow creases, neck, and back of the knees [R, R].

Up to 1 in 3 children experience eczema, usually in the first year of life. The condition is less common (2-10%) in adults [R].

Factors that tend to worsen eczema include [R, R]:

- Contact with allergens (pollen, mold, dust mites, or animals)
- Cold, dry air
- Infections like the flu
- Contact with skin irritants (chemicals or fabrics)
- Stress

People with eczema may be more prone to skin infections. Normally, the skin has a protective barrier that keeps out germs. Eczema can compromise this barrier, making it easier for infections to arise [R, R].

The symptoms of eczema can usually be managed at home with the help of [R]:

- Moisturizers
- Humidifiers
- Topical medications
- Trimming or covering fingernails (to limit scratching)
- Avoiding skin irritants

While the causes of eczema aren't completely clear, **genetics seems to play a major role**. What's more, the genetics of eczema, asthma, hay fever, and food allergies are very similar. This means that if you have one, you're more likely to have the others [R, R].

Up to 75% of differences in people's chances of developing eczema may be attributed to genetics. Genes involved in eczema may influence [R, R, R, R, R, R]:

- Skin barrier function (*FLG*, *OVOL1*, *KIF3A*)
- Inflammation (*IL13*, *IL4*)
- Immune response (*HLA-DQA1*, *EMSY*)

MORE LIKELY

More likely to have eczema based on 6,952 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
STMN3	rs3848669	TT
IL6R	rs61812598	AA
ADO	rs4372325	CC
PPP2R3C	rs2415269	GG
SATB1	rs4395418	CC
ID2	rs891058	GG
D2HGDH	rs34290285	GG
ARHGAP27	rs9895436	AA
LRRC32	rs7936434	GC
ACTL9	rs2918299	CT
TREH	rs10790275	CG
TRIB1	rs12334935	GA
PRR5L	rs10836538	TG
MDM1	rs2227491	CT
NCF4	rs4821564	CT
RUNX3	rs6672420	AT
TRAF3	rs12888955	GA
FLG	rs61816761	GG
FLG	rs138726443	GG
LINGO4	rs12123821	CC
ARRDC1	rs117137535	GG

GENE	SNP	GENOTYPE
CCR7	rs112401631	TT
LRRC32	rs55646091	GG
SLC22A5	rs60153262	CC
IL2RA	rs62626322	TT
OVOL1	rs10791824	AA
HLA-C	rs2844594	GG
TBKBP1	rs72833417	AA
KIAA1109	rs62323874	GG
SLC25A46	rs3853750	TT
PRR5L	rs6484847	CC
KIAA1109	rs1904522	GG
GNGT2	rs28406364	CC
TNFSF18	rs6691738	TT
ZBTB25	rs11625265	AA

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

Pancreas Inflammation

The pancreas is an organ located behind the stomach that releases crucial enzymes for carbs and fats digestion. **Pancreatitis** is inflammation of the pancreas, which can be acute or chronic.

Potential risk factors for pancreatitis include [R, R, R]:

- **Alcohol**
- Cigarette smoking
- Obesity
- High blood lipids
- Certain medications
- Genetics

Genetically predicted higher fasting insulin may be associated with acute and chronic pancreas inflammation. In contrast, genetically high testosterone levels may be causally associated with a lower risk of pancreas inflammation [R, R].

Health conditions that may contribute to pancreas inflammation include [R, R]:

- Gallstones
- Diabetes
- Infections
- Injury or trauma

MORE LIKELY

More likely to get pancreas inflammation based on 1,669 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PRSS1	rs10273639	CC
JCAD	rs2995271	TT
JAKMIP2	rs17107296	AA
JAKMIP2	rs150261364	CC
SPINK5	rs112861203	TT
STK32A	rs148849032	CC
MORC4	rs12688220	C
JAKMIP2	rs146303903	AA
CTRC	rs497078	CC
STK32A	rs142623619	AA
/	rs150176211	GG
ADRB2	rs17640347	GG
ABCG5	rs75331444	GG
SLC25A34	rs60816621	CC
COLEC10	rs11988997	CC
NUP62CL	rs12688091	G
TBC1D8B	rs12689287	G
RNF128	rs66491909	G
PWWP3B	rs379742	G
RADX	rs5916761	A
PWWP3B	rs67184230	G

GENE	SNP	GENOTYPE
TWIST2	rs4663946	CC
IGDCC4	rs75405617	TT
SYNJ1	rs61750217	CC
GBE1	rs28763904	AA
C4ORF50	rs138928790	CC
PLEKHH3	rs200210041	GG
NAA38	rs144163075	CC
PDLIM5	rs76352571	AA
FLNC	rs181067717	CC
SNRK	rs56104180	CC
FAM81A	rs111778770	GG
PCDHB1	rs17208383	CC
RAP1B	rs141697489	GG
TMEM222	rs150461998	CC
CAPG	rs62623452	CC
KRT76	rs149868801	CC
TMC3	rs150843673	GG
CFAP61	rs115175415	CC
RAD51C	rs28363317	AA

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

Nephritis

Symptoms of nephritis may vary depending on the type and severity of the condition but can include hematuria (blood in the urine), proteinuria (protein in the urine), high blood pressure, and edema (swelling of the body, especially in the legs, ankles, or face). Nephritis can arise due to various causes including autoimmune disorders, infections, toxins, or as a reaction to certain medications.

Prompt diagnosis and treatment are crucial in managing the condition to prevent further kidney damage and associated health complications.

MORE LIKELY

More likely to have nephritis based on 626,851 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PLA2R1	rs4664308	AA
HLA-DQA1	rs2187668	CC
MICB	rs3134792	TT
MICB	rs7775397	TT
MICB	rs389884	AA
MICB	rs7750641	CC
MICB	rs3130544	CC
HLA-DMB	rs1480380	CC
MICB	rs3115663	TT
MICB	rs3130618	CC
HLA-DQA2	rs1980493	TT
CCHCR1	rs2233956	TT
MICB	rs3132580	GG
C4A	rs652888	AA
PSORS1C1	rs3130564	CC
HLA-DQA2	rs3129939	AA
EGFL8	rs3096697	GG
PBX2	rs3134945	CC
RNF5	rs204999	AA
MICB	rs1265159	GG

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

Chronic Lyme

The risk of developing Lyme disease is mainly influenced by a person's chance of getting bitten by an infected tick. Relevant factors include:

- Location
- Time spent outdoors
- Season

Genetics doesn't seem to play a major role in susceptibility to Lyme disease. However, in some people, it may affect **chronic post-treatment symptoms**, especially joint pain. Involved genes play a role in [R, R, R, R, R, R]:

- Immune response and inflammation (*TLR1*, *TLR2*, *HLA-DRB1*)
- Removal of toxic bacterial products (*ABCB1*)

Keep in mind the research in this area is limited, and there have been some mixed results. Please take your results with a grain of salt until more research is done [R, R].

TYPICAL LIKELIHOOD

Typical likelihood of having "chronic Lyme" based on 5 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TLR2	rs5743708	GG
HLA-DQA2	rs660895	AG
HLA-DQA2	rs6910071	AG
DDAH2	rs9267658	TC
HLA-DQA2	rs17208888	GG
/	rs9271366	GA
TLR1	rs5743618	AA
ABCB1	rs1128503	GG
HLA-DOB	rs3817964	TT
C6ORF47	rs4947332	CC
HLA-DQA2	rs3763305	GG

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

Tonsil Inflammation

The course of treatment for tonsillitis depends on whether the cause is viral or bacterial. Viral tonsillitis typically does not require specific medical treatment other than symptomatic relief through pain relievers and rest, as it usually resolves on its own. Bacterial tonsillitis, often caused by Streptococcus pyogenes, can require antibiotic therapy to eliminate the infection.

Recurrent cases of bacterial tonsillitis may lead to a recommendation for tonsillectomy, which is the surgical removal of the tonsils. Adequate hydration and throat soothing remedies are also common supportive care measures regardless of the cause.

TYPICAL LIKELIHOOD

Typical likelihood of tonsillitis based on 351,312 genetic variants we looked at

30th

PERCENTILE

Your risk is **greater than 30% of the population** and **lower than 70% of the population.**

Myocarditis

Myocarditis can be caused by [R]:

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

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

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

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

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

TYPICAL LIKELIHOOD

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

Sepsis

While sepsis can occur in anyone with an infection, certain individuals are at higher risk:

- People with weakened immune systems, including those with conditions like HIV/AIDS, cancer, or those taking immunosuppressive medications.
- Very young children and older adults, whose immune systems are typically not as robust.
- Patients with chronic illnesses, such as diabetes, kidney or liver disease.
- People who have recently had surgery or have invasive devices like catheters or breathing tubes.
- Hospitalized patients, especially those in intensive care units.

Treatment of sepsis needs to be quick and aggressive, usually in a hospital setting. The mainstay of sepsis treatment includes:

- Antibiotics to fight infection. Initial broad-spectrum antibiotics are refined once the specific infection-causing agent is identified.
- Intravenous fluids to maintain blood pressure and circulation.
- Vasopressors if blood pressure cannot be maintained with fluids alone.
- Support for other affected organs may involve dialysis for kidney failure, mechanical ventilation for respiratory failure, or surgery to remove sources of infection such as abscesses or infected tissues.

TYPICAL LIKELIHOOD

Typical likelihood of sepsis based on 939,004 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GPM6A	rs79422343	TT
RIN3	rs149187226	GG
CHRM2	rs142021422	CC
KLHDC10	rs181021474	TT
RALGDS	rs140871186	CC
MAST4	rs577432066	GG
MAD1L1	rs147296048	AA

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

Actinic Keratosis

Frequent or intense exposure to ultraviolet (UV) rays is usually the cause of actinic keratoses. People who frequently lay in the sun or on tanning beds, live in a sunny place, or work outdoors may be at increased risk.

The following factors may further increase your likelihood of developing actinic keratoses [R]:

- Having red or blond hair and blue or light-colored eyes
- Tending to freckle or burn when exposed to sunlight
- Age older than 40
- Having a weakened immune system

While sun exposure is the primary cause of actinic keratoses, genetic factors may influence an individual's susceptibility. People with certain genetic traits, like fair skin and light hair, are more predisposed to developing actinic keratoses due to reduced melanin, which offers less protection from UV radiation.

An actinic keratosis sometimes disappears on its own but might return after more sun exposure. It's hard to tell which actinic keratoses will develop into skin cancer, so they're usually removed as a precaution. Treatment options include [R]:

- Cryotherapy (freezing the lesion with liquid nitrogen).
- Topical removal treatments such as fluorouracil, imiquimod, or diclofenac.
- Photodynamic therapy, which uses a combination of light and a special chemical to destroy cancer cells.
- Curettage, where the lesion is scraped off.

If treated early, actinic keratoses can be cleared up or removed. If left untreated, some of these spots might progress to squamous cell carcinoma. This is a type of cancer that usually isn't life-threatening if detected and treated early.

TYPICAL LIKELIHOOD

Typical likelihood of an actinic keratosis based on 1,675 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TYR	rs1126809	AG
BNC2	rs12350739	GA
HERC2	rs12916300	CT
FOXP1	rs7638354	AT
SLC45A2	rs16891982	GG
IRF4	rs12203592	CC
SPATA2L	rs35063026	CC
ASIP	rs6059655	GG
TRPC4AP	rs2425025	AA
DEF8	rs4268748	TT
HLA-DQA2	rs4455710	CC
TRPS1	rs7832568	AA

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

Sarcoidosis

Symptoms of sarcoidosis vary widely, depending on the organs involved and the severity of the inflammation. Many patients are asymptomatic. They do not have any visible symptoms and the disease may be discovered incidentally during a routine chest X-ray. However, when symptoms are present, they may include persistent dry cough, fatigue, shortness of breath, and chest pain.

Some patients may experience symptoms related to specific organ involvement, such as skin sores, blurred vision, or enlarged lymph nodes. The course of the disease can range from mild and self-limiting to chronic and progressive. In some cases, it can cause severe organ damage and significantly affect quality of life.

TYPICAL LIKELIHOOD

Typical likelihood of sarcoidosis based on 270 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ZNF439	rs76054429	TT
PRRG4	rs143440435	TT
/	rs78439249	GG
HLA-DMA	rs9276935	TT
HLA-DQA2	rs3101944	CC
HLA-DRB1	rs28589559	GG
ZNF578	rs73575927	TC
TNR	rs732292	AA
HLA-DRB1	rs9271346	CT
/	rs9269233	AC
HLA-DQA2	rs3129888	GA
ATP6V1G2	rs3131376	AG
HLA-DRB5	rs7451330	TC
IGFBP3	rs7791656	TC
SORBS1	rs185608970	CC
SLC9A9	rs146666669	GG
EPAS1	rs73926243	AA
CHD6	rs6124391	GG
GABRP	rs4868030	GG

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

Rheumatic Fever

The cardiac complications associated with ARF are especially concerning, often presenting as carditis or inflammation of the heart. This can lead to chronic health issues, including rheumatic heart disease which might result in long-term valve damage, heart failure, and other severe cardiac conditions. In addition, the disease can provoke Sydenham's chorea, a neurological disorder marked by rapid, involuntary movements, emotional instability, and muscle weakness.

Patients may also exhibit erythema marginatum, a rare skin condition characterized by a distinctive rash, and subcutaneous nodules. Effective diagnosis and treatment of acute rheumatic fever require a multidisciplinary approach, usually involving antibiotics to eradicate the streptococcus bacterium, and anti-inflammatory medications to manage the inflammatory symptoms. For those with carditis, long-term follow-up and prophylactic antibiotics are often necessary to prevent recurrence and mitigate the risk of further damage to the heart.

TYPICAL LIKELIHOOD

Typical likelihood of rheumatic fever based on 1,671 genetic variants we looked at

Thyroid Inflammation

The most common forms of thyroiditis include Hashimoto's thyroiditis, an autoimmune disease where the body's immune system mistakenly attacks the thyroid; postpartum thyroiditis, which can occur after childbirth; and subacute thyroiditis, which usually follows a viral infection. Diagnosis may involve blood tests to measure thyroid hormone levels, as well as antibody tests to detect autoimmune activity.

Treatment depends on the type and severity of thyroiditis, ranging from observation and symptom management to medications like levothyroxine for hypothyroidism or beta-blockers for hyperthyroidism symptoms.

LESS LIKELY

Less likely to have thyroid inflammation based on 348,866 genetic variants we looked at

3rd

PERCENTILE

Your risk is **greater than 3% of the population** and **lower than 97% of the population.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TRIB2	rs1534422	GG
SESN3	rs4409785	TC
ZNF668	rs57348955	AG
TNFRSF14	rs2843403	CT
PTPN22	rs2476601	GG
PTPN22	rs6679677	CC
CTLA4	rs11571297	TT
TNF	rs1800629	GG
TNF	rs1799964	TT
BACH2	rs72928038	GG
LPP	rs13093110	CC
IL2RA	rs706779	TC
GXYLT1	rs4768412	CC

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

Eye Inflammation

The management of uveitis involves addressing both the inflammation and the underlying cause if identified. Typical treatments include corticosteroids to reduce the inflammation and drugs that manage the immune system's response.

Careful monitoring by an ophthalmologist is critical to prevent complications such as glaucoma, cataracts, and macular edema, all of which could lead to loss of vision.

LESS LIKELY

Less likely to have uveitis based on 3,409 genetic variants we looked at

8th

PERCENTILE

Your risk is **greater than 8% of the population** and **lower than 92% of the population.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
EYS	rs665873	AA
IL23R	rs79755370	CC
ERAP1	rs2032890	AA
B3GNT2	rs4672507	TT
IL10	rs17351243	AA
INAVA	rs12132349	TA
IL18R1	rs10197284	GA
IL6R	rs6690230	GG

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

Optic Nerve Inflammation

The causes of optic neuritis are multifactorial, with potential links to autoimmune diseases like multiple sclerosis (MS) and neuromyelitis optica. In fact, optic neuritis can be the first indication of MS in many cases. Additionally, viral and bacterial infections can trigger the condition.

Recovery from optic neuritis starts spontaneously within a few weeks in most patients, and while vision often improves within a few months, some might experience residual effects or permanent vision changes. Treatment usually involves corticosteroids to reduce inflammation and accelerate the recovery of vision, though the response to medications can vary among individuals.

LESS LIKELY

Less likely to have optic neuritis based on 12,788 genetic variants we looked at

12th

PERCENTILE

Your risk is **greater than 12% of the population** and **lower than 88% of the population.**

Urethra Inflammation

Diagnosis of non-specific urethritis involves examining the patient's symptoms and ruling out specific bacterial infections through standard STI tests, as the symptoms can closely mimic those caused by sexually transmitted diseases. Treatment usually consists of antibiotics to cover a wide range of potential bacterial causes, and patients are advised to abstain from sexual activity until the infection is resolved to prevent the spread of possible undetected infections.

It's important to treat NSU promptly to avoid complications, including spread of infection to the reproductive organs, which could result in long-term problems with fertility or chronic pain.

LESS LIKELY

Less likely to have non-specific urethritis based on 26,897 genetic variants we looked at

1st

PERCENTILE

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

Pulmonary Fibrosis

Risk factors for pulmonary fibrosis include [R]:

- Age: Pulmonary fibrosis is more likely to affect middle-aged and older adults
- Current or past smoking
- Exposure to toxins and pollutants such as silica dust, asbestos fibers, hard metal dusts, coal dust, grain dust, or animal droppings
- Radiation and chemotherapy treatments
- Chronic conditions such as lupus, rheumatoid arthritis, or scleroderma.

While the majority of pulmonary fibrosis cases are sporadic, familial pulmonary fibrosis is recognized, and certain genetic mutations have been identified. These mutations might impact the lung's susceptibility to damage and scarring.

The lung damage caused by pulmonary fibrosis can't be repaired, but medications and therapies can sometimes help ease symptoms and improve quality of life. Treatment options include [R]:

- Medication: Two drugs, pirfenidone and nintedanib, are FDA-approved to treat IPF and can slow the progression of the disease.
- Oxygen therapy to ease the symptoms of shortness of breath.
- Pulmonary rehabilitation to help manage the symptoms and improve daily functioning.
- Lung transplant in severe cases or rapid progression of the disease.

LESS LIKELY

Less likely to have pulmonary fibrosis based on 17,440 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
IRF3	rs76246107	GG
NCF2	rs17849502	GT
GLS	rs13389408	CT
CCDC116	rs5754467	GA
IRF5	rs10954214	CT
PTTG1	rs2431098	GA
PRDX6	rs2422345	GA
TNFAIP3	rs58721818	CC
METTL27	rs193107685	TT
TNPO3	rs13238352	CC
PTPN22	rs6679677	CC
TRIM59	rs112846137	GG
BLK	rs2736337	TT
DNASE1L3	rs35677470	GG
TNIP1	rs4958880	CC
IDUA	rs13101828	AA
ALDH2	rs11066301	GA

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

Meningitis

The diagnosis of meningitis typically involves the analysis of cerebrospinal fluid (CSF) obtained through a procedure known as a lumbar puncture or spinal tap. In this process, the fluid surrounding the central nervous system is assessed for signs of inflammation, infection, and the presence of the causative organism.

Treatment strategies depend on the underlying cause of the meningitis and may include antimicrobial therapy for infections, corticosteroids to reduce inflammation, and supportive care. Preventative measures such as vaccines are available for some forms of bacterial meningitis, and prophylactic antibiotics may be given to close contacts of those with certain types of the disease.

LESS LIKELY

Less likely to have meningitis based on 698,252 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SGCD	rs13358188	AA
SIK3	rs116886525	CC
ISL2	rs188530871	CC
CD28	rs189257688	CC
ITPKB	rs17587821	TT
RCN1	rs61878814	TT
CACNA1E	rs116306652	GG
RERE	rs35608792	AA

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

Autoimmune Joint Conditions

The immune system can mistakenly target joint tissues, leading to various autoimmune conditions that cause inflammation, pain, and potential joint damage. This section explores genetic factors associated with joint-related autoimmune conditions including rheumatoid arthritis, psoriatic arthritis, lupus, and ankylosing spondylitis. Understanding these genetic predispositions can help inform personalized approaches to managing joint health and inflammatory responses.

TYPICAL LIKELIHOOD

Rheumatoid Arthritis

Typical likelihood of rheumatoid arthritis

TYPICAL LIKELIHOOD

Lupus

Typical likelihood of lupus

TYPICAL LIKELIHOOD

Psoriatic Arthritis

Typical likelihood of psoriatic arthropathy

TYPICAL LIKELIHOOD

Ankylosing Spondylitis

Typical likelihood of ankylosing spondylitis

Rheumatoid Arthritis

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*)

TYPICAL LIKELIHOOD

Typical likelihood of rheumatoid arthritis based on 1,049,410 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HLA-DQA2	rs6457617	CT
CTLA4	rs3087243	GG
ANKRD55	rs7731626	GG
UBASH3A	rs1893592	AA
ETS1	rs73013527	CC
WDFY4	rs2671692	AA
RASGRP1	rs8032939	CC
RUNX1	rs8133843	AA
COG6	rs9603618	CC
ZFP36L1	rs1950897	TT
/	rs6651252	TT
LBH	rs7579944	CC
PODXL	rs11761231	CC
HLA-DQA2	rs660895	AG
TLR3	rs3775291	CC
HLA-DQA2	rs9268839	AG
CXCL13	rs117605225	TT
TNFAIP3	rs17264332	AG
IL2RA	rs706778	TC
ARID5B	rs71508903	CT
TCTE1	rs2233424	TC

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

GENE	SNP	GENOTYPE
IKZF3	rs2872507	GA
PADI4	rs2240335	CA
POU3F1	rs883220	CA
STAT4	rs11889341	TC
IRF5	rs4728142	GA
YDJC	rs2298428	TC
CD28	rs1980422	TC
REL	rs34695944	TC
CD40	rs4239702	TC
CD101	rs624988	TC
EOMES	rs3806624	AG
TNFAIP3	rs6920220	GA
ANAPC4	rs3816587	TC
REL	rs13031237	GT
STAT4	rs7574865	TG
RNASET2	rs3093024	GA
NCF4	rs729749	CT
HLA-DQA2	rs6910071	AG
CD40	rs4810485	TG
GTF2I	rs113066392	ADEL(C)
TREH	rs10892279	GA
NOS3	rs2070744	CT
PTPN22	rs6679677	CC
DCTN3	rs11574914	GG
TRAF1	rs10985070	AA
TLE3	rs8026898	GG
AFF3	rs9653442	TT
IRF8	rs13330176	TT
FCRL3	rs3761959	CC

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

Lupus

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.

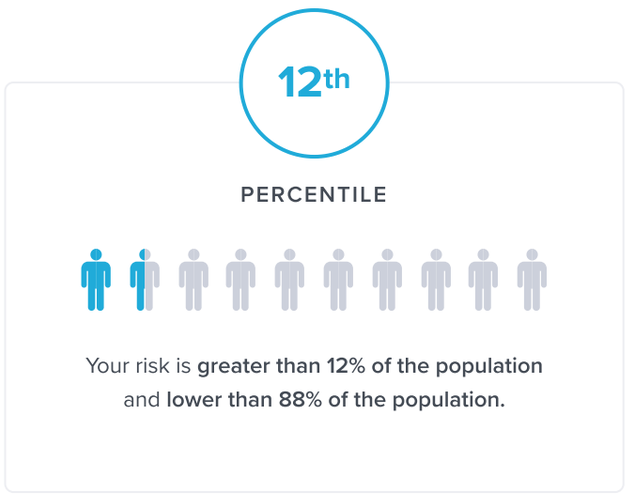
The main risk factors for lupus include [R,R]:

- **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.

TYPICAL LIKELIHOOD

Typical likelihood of lupus based on 1,049,429 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SMG7	rs17849501	CT
STAT4	rs11889341	TC
IRF8	rs11644034	GG
NADSYN1	rs3794060	CC
PRDM1	rs6568431	AA
IKZF1	rs4917014	TT
LYST	rs9782955	CC
ZFP36L1	rs4902562	AA
UBE2L3	rs7444	CT
SLU7	rs2431697	TC
TYK2	rs2304256	AC
TMEM80	rs12802200	CA
DEXI	rs9652601	GA
TASL	rs887369	C
FAP	rs2111485	AG
/	rs11908000	CC
MICB	rs1270942	AA
TNPO3	rs10488631	TT
TNFAIP3	rs6932056	TT
ITGAM	rs34572943	GG
SKP1	rs7726414	CC

GENE	SNP	GENOTYPE
PTPN22	rs2476601	GG
TNIP1	rs10036748	CC
IKZF3	rs2941509	CC
MECP2	rs1734787	A
ZC3H13	rs912784	CC
BLK	rs2736340	CC
PLD2	rs2286672	CC
IKZF2	rs3768792	AA
CD44	rs2732549	AG
TNFSF4	rs704840	TT
BANK1	rs10028805	AA
RPP25	rs2289583	CC
RPP14	rs9311676	TT
IL19	rs3024505	AG
SLC15A4	rs1059312	AA
WDFY4	rs2663052	GG
FCGR2A	rs1801274	AA
DDO	rs9398235	GG
JARID2	rs1267499	CT

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

Psoriatic Arthritis

The diagnostic process for psoriatic arthritis typically involves a multifaceted approach due to its diverse clinical presentations. A healthcare provider will often look for symptoms such as joint pain combined with psoriasis or nail changes, as well as a family history of psoriatic conditions. Sufferers may also experience symptoms beyond joint pain and skin lesions, including fatigue, nail pitting or separation, and eye inflammation like conjunctivitis.

Treatments for psoriatic arthritis focus on controlling inflammation to prevent joint damage and alleviate symptoms. Therapeutic strategies may include a combination of medication, such as nonsteroidal anti-inflammatory drugs (NSAIDs), immunosuppressants, or biologics, and lifestyle modifications, including physical therapy and exercise.

TYPICAL LIKELIHOOD

Typical likelihood of psoriatic arthropathy based on 306 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TYK2	rs34536443	GG
MICB	rs12191877	CT
MICB	rs13191343	CT
IL12B	rs4921482	TT
STAT2	rs2020854	TT
IL12B	rs62377586	GG
SLC22A5	rs848	CC
NXPE3	rs4683946	GG
CNTN4	rs17194140	TT
INSIG1	rs306281	GG
NAPG	rs11665266	GA
C1ORF141	rs12044149	GT
CTNNA3	rs12356475	CT
FAP	rs2111485	AG
TYK2	rs34725611	GA
REL	rs13017599	GA
SLC22A5	rs17622208	GA
LYRM9	rs3794767	TC
CCDC116	rs5754467	GA
RUNX3	rs1395621	TC
STX4	rs12924903	GA
TNFAIP3	rs610604	TG
INSL6	rs145699582	CC
TNIP1	rs76956521	AA
IL12B	rs12188300	AA
TRAF3IP2	rs33980500	CC
DEFB1	rs56677333	AA
FLRT2	rs76800961	CC
LGALS9	rs4795067	AA

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

Ankylosing Spondylitis

The fusion of the spine caused by ankylosing spondylitis can lead to a stooped-over posture, which is often a distinctive sign of the disease. Apart from the spinal symptoms, some individuals may experience symptoms in other areas such as the eyes (uveitis), heart, lungs, and even the skin.

The cause of ankylosing spondylitis is unknown, though genetic factors are thought to be involved, particularly the *HLA-B27* gene. Treatments typically focus on reducing pain and stiffness and preserving joint mobility, which may encompass medications, physical therapy, and exercise.

TYPICAL LIKELIHOOD

Typical likelihood of ankylosing spondylitis based on 355 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
IL23R	rs11209026	GG
ERAP2	rs10045403	AA
NKX2-3	rs11190133	CC
ERAP1	rs30187	CT
CARD9	rs1128905	CC
INAVA	rs41299637	TG
ETS2	rs2836883	AG
ERAP2	rs2910686	CT
ICAM5	rs35164067	AG
RUNX3	rs6600247	TC
IL23R	rs12141575	GA
NPEPPS	rs9901869	AG
UBE2L3	rs2283790	GA
IL12B	rs6871626	AC
B3GNT2	rs6759298	GG
GALC	rs11624293	TT
IL6R	rs4129267	TT
BACH2	rs17765610	AA
GPR35	rs4676410	GA
NOS2	rs2297518	GG
LTBR	rs1860545	AG
SH2B3	rs11065898	CC
NOS2	rs2531875	TT

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

Autoimmune Skin Conditions

Autoimmune responses affecting the skin can manifest in various ways, from changes in pigmentation to inflammation and tissue integrity. This section examines genetic variants linked to skin-related autoimmune conditions including vitiligo, psoriasis, lupus, and more. Knowledge of these genetic factors can provide insights into immune regulation and skin health management strategies.

MORE LIKELY

Psoriasis

More likely to have psoriasis

TYPICAL LIKELIHOOD

Vitiligo

Typical likelihood of vitiligo

TYPICAL LIKELIHOOD

Alopecia Areata

Typical

TYPICAL LIKELIHOOD

Pemphigus Vulgaris

Typical likelihood of pemphigus vulgaris

TYPICAL LIKELIHOOD

Behcet’s Disease

Typical likelihood of Behcet's disease

LESS LIKELY

Systemic Sclerosis

Less likely to have systemic sclerosis

Psoriasis

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].

MORE LIKELY

More likely to have psoriasis based on 1,049,035 genetic variants we looked at

65th

PERCENTILE

Your risk is **greater than 65% of the population** and **lower than 35% of the population.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TYK2	rs34536443	GG
IL23R	rs9988642	TT
STAT2	rs2066819	CC
RNF145	rs2082412	GG
IL12B	rs7709212	TT
GCA	rs17716942	TT
LCE3C	rs4845459	AA
IFNLR1	rs10794648	CC
SLC22A5	rs1295685	GG
PPP2R3C	rs8016947	GG
ZNF816	rs9304742	TT
DDX58	rs11795343	TT
IL13	rs20541	GG
TSC22D1	rs9533962	CC
POLI	rs545979	TT
ELMO1	rs2700987	AA
TNFAIP3	rs643177	CT
COG6	rs34394770	TT
TP63	rs28512356	CC
LYRM9	rs28998802	GA
STX1B	rs13708	AG

GENE	SNP	GENOTYPE
CCDC88B	rs645078	AA
IFIH1	rs1990760	CT
PUS10	rs62149416	TC
POLI	rs3730682	AG
SLC44A2	rs892085	GA
REL	rs842625	GA
STX1B	rs12445568	TC
EXOC2	rs9504361	AG
TNFAIP3	rs582757	TC
RUNX1	rs8128234	TC
TNFSF8	rs6478109	GA
CARD11	rs4722404	TC
CLIC6	rs9305556	AG
ZMIZ1	rs1250546	GA
EXOC2	rs3799296	TA
AK8	rs1076160	CT
STAT3	rs744166	AA
ANXA6	rs2233278	GG
TNIP1	rs17728338	GG
TRAF3IP2	rs33980500	CC
STAT2	rs2066807	CC
RNF145	rs3213094	CC
LGALS9	rs4795067	AA
PARK7	rs417065	CC
CAVIN1	rs56364076	TT
SPATA2	rs1056198	TT
SPATA2	rs7352944	CC
MFSD4B	rs240993	CC
POU2F3	rs2847500	GG

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

Vitiligo

Key Takeaways:

- Up to **80%** of differences in people's chances of developing vitiligo may be due to genetics, and many will see it develop before adulthood.
- About **1%** of the world's population has vitiligo, so even if you have a high genetic risk, the actual risk is still low.
- A high genetic risk may make vitiligo triggers such as an autoimmune response or a sunburn, more likely to trigger the condition.
- Click the **Recommendations** tab for potential dietary and lifestyle changes, and **next steps** for relevant labs.

Vitiligo is a condition in which the skin loses pigment. Normally, special skin cells called melanocytes produce a pigment called *melanin*. This pigment helps give the skin, hair, and eyes their color. In vitiligo, these skin cells are damaged or die off [R, R].

In the skin, pigment is often lost in patches. In 90% of cases, these patches appear on both sides of the body, in a symmetrical pattern. For example, if a white patch appears around the left eye, it will also appear around the right eye [R, R].

Vitiligo mostly affects the skin. However, it can also make hair go gray prematurely [R].

About 1% of people worldwide develop vitiligo. Anyone can get it, but it is more noticeable in people with darker skin [R, R].

The cause of vitiligo is unclear. It may happen due to [R, R, R]:

- An autoimmune condition
- A trigger event (e.g., stress, skin injury, severe sunburn, chemical contact)
- **Genetics**

Possible complications of this condition include [R, R]:

- Stress
- Sunburn
- Eye problems
- Hearing loss

Treatment options for vitiligo include [R]:

- Medication
- Light therapy
- Surgery

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

- The immune response (*HLA-DQB1*, *HLA-DQA1*, *PTPN22*)
- Skin pigmentation (*FGFR1OP*, *ZMIZ1*, *OCA2*)
- Cell death (*GZMB*, *SLC29A3*, *CASP7*)

TYPICAL LIKELIHOOD

Typical likelihood of vitiligo based on 509,196 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ASIP	rs6059655	GG
HLA-A	rs60131261	GG
DEF8	rs4268748	TT
/	rs148136154	CC
TEF	rs9611565	TT
IRF4	rs12203592	CC
SERPINB1	rs78521699	AA
FARP2	rs41342147	GG
TICAM1	rs4807000	AA
PTPRC	rs16843742	TT
HLA-DQA2	rs9271597	TA
TYR	rs1126809	AG
SESN3	rs11021232	TC
IL2RA	rs706779	TC
FAP	rs2111485	AG
SH2B3	rs10774624	GA
RNASET2	rs2247314	CT
GZMB	rs8192917	CT
FOXP1	rs34346645	AC
CASP7	rs12771452	GA
RERE	rs301807	GA
TG	rs2687812	AT
ARID5B	rs71508903	CT
CPVL	rs117744081	AA
IL1RAPL1	rs73456411	G
CFAP36	rs10200159	TT
PTPN22	rs2476601	GG
HERC2	rs1635168	CC
UBASH3A	rs12482904	TT

GENE	SNP	GENOTYPE
SUOX	rs2017445	TG
TNFSF18	rs78037977	AA
C1QTNF6	rs229527	CC
LPP	rs13076312	CC
BACH2	rs72928038	GG
CD44	rs1043101	AA
RAB5C	rs11079035	GG
PIGN	rs8083511	AA
FADS1	rs968567	CC
IRF3	rs2304206	AA

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

Alopecia Areata

Risk factors for alopecia areata include:

- Family history of alopecia areata or other autoimmune conditions.
- Having another autoimmune disorder, such as thyroiditis or vitiligo.
- Certain genetic markers related to the immune system.
- Stress, which may trigger or exacerbate the condition.

Alopecia areata has a strong genetic component, and multiple genes are involved, especially those linked to the immune system and inflammatory processes. People with a family history of autoimmune diseases are at a higher risk.

Currently, there's no known way to prevent alopecia areata. Managing stress and leading a healthy lifestyle might help reduce the risk of exacerbations.

The condition has no cure but hair often regrows on its own without treatment within a year. Treatments that can promote a faster hair growth include:

- Topical corticosteroids
- Topical immunotherapy
- Minoxidil
- Platelet-rich plasma injections

TYPICAL LIKELIHOOD

Typical based on 42 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PPARGC1A	rs16873952	AA
NTM	rs11600229	AA
CPVL	rs505532	TT
/	rs1431704	CT
REELD1	rs9997120	CC
ITPR2	rs10506012	GG
HLA-DQA2	rs9275572	AG
ARHGAP42	rs11224294	CT
DISP3	rs3099624	CC
BBS12	rs7664318	GA
HLA-DQA2	rs9268528	AG
ST8SIA5	rs9952976	AA
CCDC24	rs304303	GT
ST3GAL3	rs4660260	CT
B3GAT1	rs10791360	AA
CSMD1	rs718121	CT
NUP35	rs13409979	AG
KCNU1	rs10503991	GA
DPYSL4	rs9419187	TC
RBBP8	rs9954649	AG
DOCK5	rs2979742	TC
TERF1	rs4738296	AA
CXXC4	rs7657799	TT
TFF3	rs9982439	TT
RDH14	rs2345724	AA
IQSEC3	rs2270797	CC
RNF5	rs3115553	CC
UVSSA	rs4130791	GG
SRRM4	rs12228387	GG
UNC5C	rs17023881	CC

GENE	SNP	GENOTYPE
LDLRAD3	rs16928055	TT
LIPA	rs17479692	TT
BRD4	rs11666141	TT
CRIM1	rs2666138	AA
LURAP1L	rs7022183	TT
APOLD1	rs2110597	AA
OPCML	rs11223339	GG
ARHGAP42	rs1216476	AA
ACOX1	rs12430	GG
FNDC3B	rs6414541	TT
SNX2	rs2125856	TT
NUDT6	rs304650	TT
APCDD1	rs7228576	CC
UBE2E2	rs1692617	GG
HSD17B3	rs10512241	CT
PGM5	rs7036795	TT
ZNF217	rs2766671	CC
DCBLD1	rs916305	CC
DPH5	rs12403551	AA

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

Pemphigus Vulgaris

The management of pemphigus vulgaris typically involves the use of immunosuppressive agents to reduce the activity of the immune system to stop the formation of new blisters. Corticosteroids are commonly prescribed, often in combination with other drugs such as azathioprine or mycophenolate mofetil, to achieve better control of the condition with lower doses of steroids. The goal of treatment is to induce a long-term remission while minimizing side effects of the medication.

Despite treatment, people with pemphigus vulgaris may experience periods of flares and remission. Close monitoring by a team of medical professionals, including dermatologists, dental care providers, and other specialists is crucial for managing the disease effectively.

TYPICAL LIKELIHOOD

Typical likelihood of pemphigus vulgaris based on 36 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
EPHA5	rs1074532	AA
ANK2	rs362492	CC
UCN3	rs9423602	GG
SOX9	rs17246777	CC
CDH4	rs6093035	AA
ANKFN1	rs9899680	TT
PADI6	rs10888028	TT
STK32A	rs62377684	TT
CNTN4	rs67645795	AA
NRIP1	rs2823004	GG
DLC1	rs56061315	AA
PARD3	rs1545214	CA
SMAD3	rs12907183	AG
NRSN1	rs543017	GT
CADPS	rs6772352	AG
PELI2	rs4901619	AG
GPR63	rs4839863	GA
CETN3	rs117814792	GG
IPO7	rs12574246	GG
FHIT	rs17339157	AA
LAPTM4B	rs2512413	CC
RACGAP1	rs56232366	AA
THSD7B	rs16837853	AA
MED13L	rs11068077	TT
CSMD1	rs10104910	CC
MCPH1	rs17075940	GG
RABAC1	rs11878287	AA
B4GALT4	rs74280516	CC
KCNAB2	rs2235791	CC

GENE	SNP	GENOTYPE
LMNTD1	rs16928923	TT
COMMD10	rs77499388	TT
TTC13	rs12040904	AA
DNAJC15	rs61950435	CC
ITGBL1	rs11069452	GG
/	rs12460063	AA
/	rs11660027	AA

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

Behcet's Disease

The nature of Behçet's disease is episodic, meaning the symptoms may flare up intermittently followed by periods of remission. Since it's a chronic condition, the flares can recur over a person's lifetime. Diagnosis of Behçet's disease can be challenging as there is no specific test for it. Physicians usually diagnose it based on symptoms and ruling out other conditions.

Treatment often focuses on reducing the symptoms and preventing serious complications with medications that reduce inflammation and suppress the immune system. Because of its complexity, management of Behçet's disease often involves a team of specialists.

TYPICAL LIKELIHOOD

Typical likelihood of Behcet's disease based on 390 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CCR5	rs13092160	TT
CCR3	rs13075270	TT
PDGFRL	rs17633132	CC
YJU2	rs428253	GC
TNFAIP3	rs10499194	CC
MTHFD1L	rs12216229	AA
HPCAL4	rs11206377	GG
CPVL	rs317711	GC
CCDC180	rs2061634	GC
CD40	rs4810485	TG
CD40	rs1883832	TC
UBASH3B	rs4936742	CT
STAT3	rs2293152	CG
ERAP1	rs17482078	CC
POU5F1	rs4959053	GG
TFCP2L1	rs17006292	CC
HLA-C	rs76546355	GG
PSORS1C1	rs12525170	GG
CCR3	rs13084057	CC
IRF5	rs192829776	CC
GPR18	rs17575643	CC
GPR18	rs9517668	AA
UBAC2	rs9554581	CC
GPR18	rs727263	GG
GPR18	rs7332161	GG
REV3L	rs13210247	AA
TNF	rs1800629	GG
TRAPPC2L	rs79831785	TT
/	rs17810456	TT

GENE	SNP	GENOTYPE
GPR18	rs2892976	AA
IL12A	rs17810546	AA
UTS2	rs72633102	CC
IL12A	rs17753641	AA
TNFAIP3	rs9494885	TT
UBAC2	rs7999348	AA
UBAC2	rs9513584	AA
UBAC2	rs6491493	CC
UBAC2	rs9554573	GG
UBAC2	rs9517644	CC

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

Systemic Sclerosis

Risk factors for systemic sclerosis include [R]:

- Gender: Anyone can get systemic sclerosis, but it's more common in women.
- Age: Systemic sclerosis is typically diagnosed between the ages of 30 and 50.
- Environmental triggers: Exposure to certain viruses, drugs, or toxins can increase the risk of systemic sclerosis or trigger the symptoms. However, an environmental trigger is not identified for most people.
- Immune system problems: Systemic sclerosis is believed to be an autoimmune disease. People who have systemic sclerosis may also have symptoms of another autoimmune disease such as rheumatoid arthritis, lupus, or Sjögren's syndrome.

The precise genetic basis of systemic sclerosis is not fully understood, but genetic factors are believed to play a role, especially when combined with environmental triggers. Certain genetic markers have been associated with an increased risk of developing the disease.

There are no known preventive measures for systemic sclerosis. However, early diagnosis and management are crucial to prevent serious complications. Patients should regularly consult with rheumatologists and other specialists for comprehensive care and monitoring.

There is no treatment that can cure or stop the overproduction of collagen that is characteristic of systemic sclerosis. But a variety of treatments can help control symptoms and prevent complications. Examples of treatments include [R]:

- Medications to improve blood flow, suppress the immune system, or manage symptoms like acid reflux and high blood pressure.
- Physical therapy to maintain joint flexibility and strength.
- Stem cell transplants might be an option for people who have severe symptoms that haven't responded to more-common treatments.
- If the lungs or kidneys have been severely damaged, organ transplants might be considered.

LESS LIKELY

Less likely to have systemic sclerosis based on 12,699 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
IRF3	rs76246107	GG
NCF2	rs17849502	GT
GLS	rs13389408	CT
CCDC116	rs5754467	GA
IRF5	rs10954214	CT
PTTG1	rs2431098	GA
PRDX6	rs2422345	GA
TNFAIP3	rs58721818	CC
METTL27	rs193107685	TT
TNPO3	rs13238352	CC
PTPN22	rs6679677	CC
TRIM59	rs112846137	GG
BLK	rs2736337	TT
DNASE1L3	rs35677470	GG
TNIP1	rs4958880	CC
IDUA	rs13101828	AA
ALDH2	rs11066301	GA

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

Other Autoimmune Conditions

Autoimmune conditions occur when the immune system mistakenly targets healthy tissues, leading to a range of chronic diseases. This section focuses on genetic markers associated with autoimmune conditions such as multiple sclerosis, ALS, and type 1 diabetes.

Identifying your genetic predisposition to these conditions allows for early detection and targeted treatment strategies. By understanding your immune system's tendencies, you can work with healthcare professionals to manage autoimmune diseases and reduce their impact on your health through personalized care plans.

TYPICAL LIKELIHOOD

Multiple Sclerosis

Typical likelihood of multiple sclerosis

TYPICAL LIKELIHOOD

Type 1 Diabetes

Typical likelihood of type 1 diabetes

TYPICAL LIKELIHOOD

IgA Nephropathy

Typical likelihood of IgA nephropathy

TYPICAL LIKELIHOOD

Myasthenia Gravis

Typical likelihood of myasthenia gravis

TYPICAL LIKELIHOOD

Antiphospholipid Syndrome

Typical likelihood of antiphospholipid syndrome

LOWER PREDISPOSITION

Amyotrophic Lateral Sclerosis (ALS)

Lower predisposition to ALS

LESS LIKELY

Sjogren's Syndrome

Less likely to have Sjögren's syndrome

Multiple Sclerosis

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.

TYPICAL LIKELIHOOD

Typical likelihood of multiple sclerosis based on 1,019,187 genetic variants we looked at

60th

PERCENTILE

Your risk is **greater than 60% of the population** and **lower than 40% of the population.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HLA-DQA2	rs3129934	TC
HLA-DRB5	rs3135388	AG
EBPL	rs9591325	TT
IL2RA	rs2104286	TT
RBM17	rs11256593	TT
TYK2	rs34536443	GG
TAPBPL	rs12832171	CC
JAK1	rs72922276	GG
RGS1	rs1323292	AA
RTEL1-TNFRSF6B	rs6742	CC
RMI2	rs34947566	CC
MAF	rs17724508	TT
SP140	rs35540610	CC
CD58	rs10801908	CT
IL2RA	rs12722559	CC
IRF8	rs35703946	GG
TGFBR3	rs12133753	CC
SYPL1	rs73414214	CC
IL7R	rs6897932	CC
BCL10	rs35486093	GA
CD5	rs17824933	CG

GENE	SNP	GENOTYPE
TNFSF14	rs1077667	TC
POGLUT1	rs9843355	GA
EOMES	rs438613	CT
LTBR	rs1800693	CT
ELMO1	rs60600003	GT
PRXL2B	rs6670198	TC
TAGAP	rs1738074	CT
ETV7	rs1076928	TC
IMMP2L	rs868824	CT
STAT3	rs2293152	CG
TNFRSF1A	rs4149584	CC
CBLB	rs9657904	CC
APOA5	rs2727790	GG
ETV6	rs73277163	AA
PHGDH	rs12094392	TT
ERMP1	rs2150702	AA
BACH2	rs72928038	GG
PTGER4	rs11749040	GG
GTDC1	rs72855540	AA
IL22RA2	rs62420820	GG
RPAP2	rs58394161	TT
EVI5	rs11809700	CC
THRA	rs883871	GG
IL7R	rs10063294	AA
STAT3	rs744166	AA

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

Type 1 Diabetes

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.

TYPICAL LIKELIHOOD

Typical likelihood of type 1 diabetes based on 1,047,920 genetic variants we looked at

15th

PERCENTILE

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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
/	rs9272346	GA
RASGRP1	rs72727394	TT
CTSH	rs34593439	GG
SH2B3	rs653178	CT
ALDH2	rs17696736	GA
IL19	rs3024505	AG
ERBB3	rs11171739	CT
RTL1	rs56994090	CT
DEXI	rs12708716	AG
IL7R	rs11954020	GG
MAPT	rs1052553	AA
CLEC2D	rs3764021	CT
TYK2	rs34536443	GG
IL2RA	rs61839660	CC
INS-IGF2	rs689	TT
IL2RA	rs41295121	CC
RNLS	rs12416116	CC
BCL2L11	rs4849135	GG
DEXI	rs12927355	CT
IFIH1	rs35667974	TT
IFIH1	rs72871627	AA

GENE	SNP	GENOTYPE
CTLA4	rs3087243	GG
CCR1	rs113010081	TT
BCAR1	rs8056814	GA
/	rs75793288	GG
GSDMB	rs12453507	CG
PTPN2	rs12971201	GA
IL2RA	rs10795791	GA
ICOSLG	rs6518350	AG
CLECL1	rs917911	CA
TM9SF2	rs9585056	TC
CAMSAP2	rs6691977	CT
AFF3	rs13415583	TG
FAP	rs2111485	AG
PTPN22	rs6679677	CC
PTPN22	rs2476601	GG
/	rs1456988	TT
KIAA1109	rs6534347	GG
CLN3	rs151234	GG
RMI2	rs193778	AA
PTPN2	rs1893217	AA
CD226	rs1615504	CC
/	rs705705	GG
TLL1	rs2611215	GG
SMARCE1	rs757411	CC
BACH2	rs72928038	GG

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

IgA Nephropathy

Symptoms of IgA nephropathy can vary and may be mild or silent for many years, making initial detection difficult without specific testing. Common signs include hematuria (blood in the urine), which may be visible or detectable only by a urine test, and proteinuria (excess protein in the urine).

High blood pressure and swelling in the hands and feet are also possible indicators of the disease. Regular monitoring and various treatments, such as blood pressure medications, fish oil, and immunosuppressants, can be employed to manage symptoms and slow the progression of IgA nephropathy.

TYPICAL LIKELIHOOD

Typical likelihood of IgA nephropathy based on 2,048 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NRBF2	rs57917667	GG
OVOL1	rs10896045	AA
IRF4	rs12201499	CC
HP	rs10492815	AA
FAM91A1	rs34354351	TT
/	rs62123462	AA
RREB1	rs12530084	CC
GPN3	rs56838584	GA
PSD3	rs2410596	CC
PUS10	rs842638	TC
SH3BP2	rs4690002	CT
NT5M	rs57382045	AG
TMEM51	rs531901	GA
RBMS3	rs55711830	GA
THSD7A	rs17574506	CT
CA8	rs2611364	TG
FARS2	rs2142738	CT
INPP5B	rs4653337	AG
KLF6	rs10904115	GT
TPD52	rs9774752	AC
CX3CR1	rs12497322	AG
NFKBIZ	rs7625614	CT
TNFAIP3	rs58905141	AA
AFDN	rs111387965	TT
ANKRD34B	rs62364037	TT
DEFA3	rs2075836	GG
SCN9A	rs16852104	TT
WLS	rs10493441	GG
TMEM107	rs9913189	CC

GENE	SNP	GENOTYPE
PF4V1	rs6828610	AA
ESYT2	rs710423	CC
ETS1	rs7121743	TT
INPP5D	rs14243	AA
SEMA4D	rs4877094	GG
POU5F1B	rs6989575	CC
PPIL3	rs4035021	GG
TCF7	rs151822	AA
APOC4-APOC2	rs204474	TT
CTNNA3	rs10509258	AA

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

Myasthenia Gravis

Patients with myasthenia gravis may experience a range of symptoms, from mild to severe, affecting various muscle groups. Typical symptoms include drooping of one or both eyelids (*ptosis*), blurred or double vision due to weakness of the muscles that control eye movements, unstable or waddling gait, and weakness in the arms, hands, fingers, legs, and neck. Difficulty in swallowing, speaking, and breathing can also occur.

The symptoms can fluctuate throughout the day, often peaking towards the end of the day or after prolonged activity. Since the condition can be exacerbated by stress, high temperatures, and some medications, management often requires a combination of drug therapy, lifestyle adjustments, and sometimes surgical interventions.

TYPICAL LIKELIHOOD

Typical likelihood of myasthenia gravis based on 806,055 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HLA-DQB1	rs76815088	TT
PIGN	rs4574025	CC
RELCH	rs4263037	GG
HLA-DRB1	rs9270986	AC
CTLA4	rs231770	TC
HLA-B	rs9266277	AG
SESN3	rs4409785	TC
CHN1	rs35274388	GG
PTPN22	rs2476601	GG
SFMBT2	rs2245569	AA

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

Antiphospholipid Syndrome

Apart from clotting issues, antiphospholipid syndrome may cause additional problems such as long-term damage to organs like the kidneys due to poor blood supply, or complications in pregnancy including pre-eclampsia, intrauterine growth restriction, and fetal loss. Diagnosis of APS often involves blood tests to detect antiphospholipid antibodies, alongside a medical history of thrombosis or pregnancy complications.

Treatment typically focuses on reducing clotting risk through medications like anticoagulants and, during pregnancy, a combination of low-dose aspirin and heparin to manage the condition and safeguard mother and child.

TYPICAL LIKELIHOOD

Typical likelihood of antiphospholipid syndrome based on 15 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PTPRO	rs1024843	TT
SYCP2L	rs1225763	AG
SYCP2L	rs2788869	AG
MRPS23	rs1443267	AG
ID2	rs181132	AT
CDH18	rs12153263	CT
HLA-DQB1	rs2395166	CT
C1D	rs79154414	CC
CEP128	rs2288493	CC
NGF	rs145365907	AA
/	rs1020096	GG
FRMD4A	rs12570849	TT
RGS10	rs10886503	CC
SH3BP4	rs35176804	GG
HSDL2	rs6477918	GG

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

Amyotrophic Lateral Sclerosis (ALS)

Please note: Your genetic predisposition in this report is measured relative to the rest of the population. Even if your predisposition is higher, your absolute risk of ALS is still very low because it's a very rare condition. The vast majority of people with higher genetic predisposition won't develop ALS.

Most cases of ALS are sporadic, meaning they occur without a clear cause. However, about **5-10%** of cases are familial, implying **genetic inheritance**. Researchers have identified several genes associated with ALS, with C9orf72 and SOD1 being the most well-known [R].

Genetically high leukocyte count and ApoB levels may be associated with a lower risk of ALS [R, R].

Several risk factors are associated with ALS, such as:

- Age: more common between the ages of 40 and 70
- Being a male
- Exposure to heavy metals or pesticides

LOWER PREDISPOSITION

Lower predisposition to ALS based on 7,814 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
UNC13A	rs12608932	CA
TLE3	rs1971791	GA
ZNF142	rs2303565	CT
CNOT9	rs7607369	AG
CENPV	rs7477	AA
KIAA0513	rs8056742	TT
TSNARE1	rs4917300	CC
C9ORF72	rs3849942	CC
TSC22D1	rs9533799	CC
LAMA3	rs11082762	GG

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

Sjogren's Syndrome

The exact cause of Sjögren's Syndrome isn't entirely understood. It's believed to involve a combination of genetic and environmental factors.

About **50 %** of the differences in Sjögren's syndrome may be due to **genetics** [R, R].

Certain gene markers have been associated with the disease. It is also more common in families with rheumatic disease history. However, just having the genetic predisposition doesn't mean you'll get the disorder. The following factors might also play a role in its development:

- Gender: Women have a higher risk.
- Age: Most people are diagnosed after age 40.
- Infections: Certain viral or bacterial infections might act as a trigger in genetically predisposed individuals.
- Having rheumatic disease: more common for people who have rheumatoid arthritis or lupus

LESS LIKELY

Less likely to have Sjögren's syndrome based on 1,669 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
/	rs116232857	GA
RASGRP3	rs13425999	CC
STAT4	rs10553577	TT
DDX6	rs7119038	AA
HLA-DQA2	rs9271588	TC
IL12A	rs485497	AA
IRF5	rs3757387	TC
STAT4	rs10168266	TC
OLIG3	rs6933404	TC
CPEB4	rs359457	CT
PTTG1	rs2431098	GA
SH2B3	rs4766578	TA
PRDM1	rs526531	AG
IRF5	rs10954213	GA
/	rs115575857	AA
MICB	rs3135394	AA
GTF2IRD1	rs117026326	CC
SATB1	rs11915281	CC
TNFAIP3	rs5029939	CC
HLA-DPA1	rs4282438	TT
TNPO3	rs17339836	CC
TNIP1	rs6579837	GG
BLK	rs2736345	AA
HNRNPA3	rs1554770	CT
IDUA	rs3733346	TT

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

Inflammation Markers

Inflammation markers are proteins and cells in the blood that signal the presence of inflammation in the body. This section explores key markers such as **C-reactive protein (CRP), cytokines like TNF and IL-6**, and immune cells like white blood cells and eosinophils. Elevated levels of these markers can indicate chronic inflammation, which may lead to health problems.

By understanding your genetic predisposition to abnormal levels of these markers, you can take proactive measures to reduce inflammation, potentially preventing or managing inflammatory conditions more effectively.

HIGHER LEVELS
IgE
Predisposed to higher IgE levels

LOWER LEVELS
IL-10
Predisposed to lower IL-10 levels

TYPICAL LEVELS
Inflammation (CRP)
Predisposed to typical CRP levels

TYPICAL LEVELS
TNF
Predisposed to typical TNF levels

TYPICAL LEVELS
White Blood Cells
Predisposed to typical WBC count

TYPICAL LEVELS
Basophils
Predisposed to typical basophil levels

TYPICAL LEVELS
Eosinophils
Predisposed to typical eosinophil levels

TYPICAL LEVELS
Monocytes
Predisposed to typical monocyte levels

TYPICAL LEVELS
Neutrophils
Predisposed to typical neutrophil levels

TYPICAL LEVELS
Erythrocyte Sedimentation Rate
Predisposed to typical ESR levels

TYPICAL LEVELS
IL-17 (Th17)
Predisposed to typical IL-17 levels

TYPICAL LEVELS
IL-6
Predisposed to typical IL-6 levels

IgE

Immunoglobulin E (IgE) is a type of antibody. The main role of IgE is to protect the body from infections by parasitic worms. On the downside, it also contributes to **allergic diseases** [R, R].

Genetics influence IgE levels. Involved genes play a role in our bodies' response to IgE. For example, the gene *FCER1A* helps make an IgE receptor—cell protein that binds IgE [R, R].

The most common causes of increased IgE levels are parasitic infections and allergies [R, R].

Smoking and alcohol drinking may also increase IgE levels. People with some health conditions may also have **high IgE levels**, including [R, R]:

- Viral infections [R, R]
- Inflammatory bowel disease (IBD) [R]
- Kidney disease [R]
- Rare genetic disorders [R, R]

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

HIGHER LEVELS

Predisposed to higher IgE levels based on 16 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ERMP1	rs928413	GG
FCER1A	rs2251746	TT
FCER1A	rs2427827	CT
STAT6	rs1059513	TT
NQO1	rs6499255	AA
ACKR1	rs13962	GG
HLA-C	rs3130941	CG
HLA-DQB1	rs2858331	AA
HLA-A	rs2523809	GT
LPP	rs9290877	TC
IL13	rs20541	GG
SLC22A5	rs1800925	CC
ERMP1	rs1342326	AA
FCER1A	rs2298805	GG
FCER1A	rs4656784	AA
HLA-A	rs2571391	AA
IL4R	rs1801275	AA

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

IL-10

Interleukin-10 (IL-10) is an anti-inflammatory cytokine — a small protein involved in the communication between cells. **The main function of IL-10 is suppressing immune responses.** IL-10 helps our bodies recognize and not attack the proteins in our body (self-tolerance) and those that we eat (oral tolerance) [R, R, R].

Up to **50%** of differences in people's IL-10 levels may be due to genetics. Interestingly, women naturally have lower levels than men [R, R].

In addition to **smoking**, the following health conditions may also lead to **low IL-10 levels** [R, R]:

- Sleep apnea [R]
- Depression and anxiety [R, R, R, R]
- Irritable bowel syndrome (IBS) [R, R]
- Autoimmune disorders (e.g., rheumatoid arthritis, psoriasis, multiple sclerosis) [R, R]
- Type 2 diabetes [R]
- Lung and heart disease [R, R]

People with certain health conditions may have higher IL-10 levels. Genetically higher IL-10 levels may be linked to gastric cancer, while their role in other conditions is less clear [R].

In general, higher IL-10 levels tend to be better due to anti-inflammatory effects.

LOWER LEVELS

Predisposed to lower IL-10 levels based on 28 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NNT	rs140614282	GG
IL19	rs3024505	AG
IL19	rs1800896	CT
IKBIP	rs1048911	TT
VEGFA	rs4349809	GT
SHROOM3	rs143141511	GG
/	rs6937355	TT
ZNF516	rs9951418	CC
PDIA5	rs1530455	TT
/	rs10493718	CA
SERPINE2	rs282258	TC
BMP2	rs6085948	GA
VEGFA	rs3025021	TC
NFKBIE	rs6458375	CT
REEP3	rs7088799	GT
MACROD2	rs465757	AG
HOMER1	rs4345303	CT
VLDLR	rs2375980	GC
IL10	rs1800871	GG
/	rs140244749	AA
NEBL	rs45559637	TT
LYRM7	rs148438889	GG
LYRM7	rs191791704	CC
RAPGEF6	rs147320771	TT
FNIP1	rs115710902	TT
MEIKIN	rs115393715	GG
IL19	rs1800872	GG
H1-3	rs182422732	GG
FBRSL1	rs142425959	CC

GENE	SNP	GENOTYPE
MRPL37	rs11206302	TT
MIA3	rs3002131	CG
PREP	rs10457128	AA
SORCS2	rs111913416	AA
/	rs2086656	TT
PCP4	rs9981861	TT

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

Inflammation (CRP)

Key Takeaways:

- Chronic inflammatory diseases like diabetes and heart disease are responsible for **3 in 5** deaths worldwide.
- About **40-50%** of the differences in people's hs-CRP (inflammatory protein) levels may be due to genetics.
- Other factors are equally important. They include diet, exercise, and life satisfaction.
- Click the **next steps** tab for relevant labs.

Inflammation is an important biological process. It protects the body from disease and damage. When germs or other foreign substances enter the body, white blood cells rush to the site. The area then gets red, swollen, and warm. These changes help kill pathogens and prepare the tissue to heal [R, R].

A common marker that helps measure inflammation is **C-reactive protein** (**CRP**). **High sensitivity CRP** (**hs-CRP**) in particular helps measure low-grade inflammation.

CRP is produced in the liver. It helps recognize disease-causing microbes and damaged cells that need to be removed from the body. However, it may also play a role in autoimmune disease [R, R].

Short-term inflammation is helpful. However, too much inflammation can be a bad thing [R, R, R, R].

Chronic inflammation is linked to many diseases, including:

- Autoimmune conditions [R, R]
- Heart disease [R, R, R]
- Obesity [R, R]
- Type 2 diabetes [R, R]
- Fibromyalgia [R, R]
- Mental health conditions [R, R, R, R]
- Cancer [R, R, R, R, R]

In 2014, an estimated **60%** of Americans were living with at least one chronic inflammatory condition [R].

Factors that may influence chronic inflammation include [R, R, R]:

- Diet
- Exercise
- Life satisfaction
- **Genetics**

Common strategies for reducing low-grade inflammation include [R, R, R, R, R]:

- Lifestyle changes
- Diet changes
- Weight management
- Drugs targeting the underlying condition

Genetics may play an important role in inflammatory conditions. Genes involved in inflammation may influence [R, R, R, R, R, R]:

- Immune messengers (*STAT3*, *IL6*, *IL10*)
- Immune cell function (*HLA-DRB1*, *PTPN22*)
- Histamine levels (*AOC1*, *HNMT*)

Genetically high free testosterone levels may be causally associated with lower C-reactive protein [R].

TYPICAL LEVELS

Predisposed to typical CRP levels based on 8,937 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CRP	rs1205	CC
IL6R	rs2228145	CC
FUT2	rs601338	GA
IL1B	rs16944	GA
CTLA4	rs231775	GA
IL19	rs1800872	GG
IL19	rs3024505	AG
IL4R	rs1805011	AA
IL1RN	rs419598	TT
ATG16L1	rs10210302	TT
ATG16L1	rs2241880	GG
STEAP1B	rs1554606	GG
IL13	rs20541	GG
KLC1	rs8702	GG
STAT4	rs10181656	GC
ADRB2	rs1042713	AG
IL19	rs1800896	CT
CYP1B1	rs1056836	GG
TIMP4	rs3755724	CC
AOC1	rs1049793	CG
IFIH1	rs1990760	CT

GENE	SNP	GENOTYPE
IL21	rs6822844	GT
IL6	rs1524107	CT
IL6	rs2066992	GT
LRP6	rs2160525	AG
LRP6	rs2302685	CT
FUT2	rs492602	AG
FUT2	rs281377	TC
FUT2	rs602662	GA
LEPR	rs4394621	AA
SIRT1	rs12778366	TC
MICB	rs361525	GG
CRP	rs3093059	AA
RAD50	rs2069812	AA
SLC22A5	rs1800925	CC
TNF	rs1800629	GG
HLA-DQA1	rs2187668	CC
APOE	rs429358	TT
SRA1	rs2569191	TT
IL6	rs1800795	GG
SLC20A1	rs1800587	GG
IL1A	rs17561	CC
FADS2	rs174546	CC
IL1B	rs1143634	GG
CRP	rs10494326	CC
CFH	rs6677604	GG
TLR4	rs4986790	AA
SOD2	rs4880	GG
LGALS9	rs2248814	AA
TOM1	rs2071746	AA

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

TNF

About 50% of the differences in people's TNF levels may be due to genetics [R].

Some people might naturally make more TNF because of their genes. This means that their family's history might make them more likely to have higher levels of this protein. These genes can also affect how some medicines work, especially those that target TNF.

Other factors influencing TNF levels include:

- **Infections**: TNF is rapidly produced in response to infections and is crucial for defense against many pathogens. However, uncontrolled production can lead to septic shock in severe infections.
- **Autoimmune Diseases**: Conditions like rheumatoid arthritis, Crohn's disease, and psoriasis show elevated TNF levels contributing to inflammation and tissue damage.
- **Obesity**: Fat tissue can produce TNF, contributing to the low-grade inflammation often seen in obese individuals.
- **Stress**: Acute and chronic stress can influence immune function and potentially increase TNF production.
- **Aging**: Low-grade inflammation associated with aging (sometimes called "inflammaging") may involve elevated TNF levels.
- **Diet**: Certain foods or dietary patterns can either increase or decrease TNF production, influencing inflammation.
- **Physical Activity**: Regular exercise can modulate TNF production, generally reducing its levels and associated inflammation.

Gentically higher TNF levels may be causally associated with an increased risk of:

- Heart health [R]
- Stroke [R]
- Schizophrenia [R]

However, they may also be associated with a lower risk of certain cancers. [R]

Genetically higher levels are likely not associated with:

- Alzheimer's [R]
- Sleep apnea [R]
- Parkinson's [R]
- Heart failure [R]

TYPICAL LEVELS

Predisposed to typical TNF levels based on 5,001 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PHACTR4	rs188618085	CC
YTHDF2	rs185682149	CC
TMEM71	rs72725197	CC
CRISPLD2	rs118135095	GG
TRIB2	rs116434579	GG
TNF	rs1799724	CT
ROBO1	rs149420276	GA
GPX3	rs111332265	AG
/	rs7256693	CC
ATP10B	rs149126334	GG
ANO3	rs10834997	GG
TNF	rs1800629	GG
SPRY1	rs115669577	GG
ERICH1	rs138808635	CC
PSD3	rs79105320	GG
TNF	rs1799964	TT
/	rs72841564	TT
GTF2E1	rs10511404	GG
/	rs8121916	CC
RBM11	rs72490194	CC
GBA2	rs10814274	TT

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

White Blood Cells

White blood cells are immune cells that protect your body against specific types of invaders. The different types of white blood cells are neutrophils, lymphocytes, basophils, eosinophils, and monocytes. Your **white blood cell count** is the total number of all white blood cells in your blood [R].

A high white blood cell count usually means that your immune system is responding to something stressful. Common causes of a high white blood cell count include [R, R]:

- Infections from viruses, bacteria, or parasites
- Stress
- Inflammatory disorders (e.g., inflammatory bowel disease, rheumatoid arthritis)
- Allergies

Your white blood cell count generally returns to normal when the root cause is dealt with. If it stays high or reaches extremely high levels, then your doctor may order additional tests.

Anyone can have a temporarily low white blood cell count. It generally won't cause symptoms on its own. However, some conditions and treatments can cause a long-term decrease in white blood cell count. This can make infection more likely. Causes of low white blood cell count include [R]:

- Malnutrition or vitamin deficiencies
- HIV or other viruses
- Cancer treatment (chemo or radiation)
- Bone marrow damage

Most of the time, a low white blood cell count does not require specific treatment. If you're concerned about your white blood cell count, talk to your doctor about it.

About 50-60% of differences in white blood cell count may be attributed to genetics. Genes involved may influence [R, R, R]:

- White blood cell development in the bone marrow
- The immune response

TYPICAL LEVELS

Predisposed to typical WBC count based on 33,771 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
/	rs549579958	CC
LYST	rs1886654	CC
PTPN22	rs2476601	GG
TET2	rs199741557	AA
MICB	rs2524079	GA
SH2B3	rs3184504	TC
CXCR2	rs55799208	GG
NCLN	rs144284241	CC
/	rs201347186	GG
JAML	rs143034248	CC
TTC28	rs62237617	CC
IRF8	rs11642657	CC
IL17RA	rs140221307	TT
FLT3	rs76428106	TT
ACKR1	rs34599082	CC
DPH5	rs77046277	CC
FERMT3	rs142815441	CC
TEX15	rs116898861	CC
EHD3	rs184409696	GG
RC3H1	rs77941945	GG
ORMDL3	rs4795415	CC
CDK6	rs445	CC
PLEK	rs141250842	AA
ATF7	rs117788567	CC
CXCR4	rs77306654	AA
PREX1	rs149257976	CC
/	rs117255975	TT
GFI1	rs139795227	AA
TNFSF13B	rs374039502	TT

GENE	SNP	GENOTYPE
PLAUR	rs4760	AA
BCL2	rs17758695	CC
UBE2D3	rs56095122	AA
SESN3	rs75963851	AA
NAA38	rs74480102	GG
CREB5	rs56388170	GG
CXCL5	rs11733208	AA
ARAP2	rs28530750	GG
PAQR6	rs568036	AA
LRRC36	rs12928503	CC
PRTFDC1	rs11014291	TT

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

Basophils

Basophils are white blood cells that help protect against infections, but can also play a role in autoimmune diseases and allergies [R, R].

High basophil levels can be due to:

- Allergies [R, R, R]
- Infection [R]
- Inflammatory diseases, such as inflammatory bowel disease (IBD) and rheumatoid arthritis [R, R, R, R]

Genetics may also affect basophil levels [R].

Genetically higher basophil counts may be causally associated with a lower risk of narcolepsy. [R]

TYPICAL LEVELS

Predisposed to typical basophil levels based on 1,005,557 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PACC1	rs532279691	AA
/	rs370718489	GG
GATA2	rs6782812	AA
CEBPA	rs78744187	CT
CEBPG	rs12151289	GC
/	rs200688856	GC
LARP4B	rs11253511	TC
FCGR2B	rs2994672	CT
GFI1B	rs550065584	GG
TENT5A	rs559377462	CC
LPO	rs546552332	AA
CXCR2	rs16858768	AA
CDKN2D	rs3218221	GG
MAP4K1	rs143002957	GG
TFCP2	rs117053853	GG
/	rs535521164	GG
SPINT2	rs34158728	GG
/	rs562526020	CC
BCL2	rs17758695	CC
FCGR3A	rs533276421	GG
MPO	rs28730837	GG
MPO	rs56378716	AA
CDK6	rs445	CC
RNF212B	rs147453535	AA
P2RY2	rs74472890	TT
FCGR3B	rs116282955	CC
ATP5MC2	rs181969679	AA
FLT3	rs76428106	TT
GSE1	rs61751198	AA

GENE	SNP	GENOTYPE
PLEK	rs34338164	AA
DEFA3	rs140526216	GG
CXCR2	rs114050631	CC
FCGR3A	rs141772609	CC
L3MBTL3	rs139719552	TT
DEFA3	rs10086568	GG
SPIDR	rs45577137	AA
HLA-C	rs1065406	GG
RUNX1	rs138595256	CC
LMNB1	rs2271352	GG

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

Eosinophils

Eosinophils are white blood cells that help fight infections caused by parasites, but are also involved in allergies and inflammation [R].

Common causes of high eosinophil levels are:

- Allergic diseases such as asthma, eczema, or seasonal allergies [R, R, R, R]
- Parasitic infections, mainly due to worms [R, R]

Genetics may also affect eosinophil levels [R].

TYPICAL LEVELS

Predisposed to typical eosinophil levels based on 846,940 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GATA2	rs6782812	AA
SH2B3	rs7310615	GG
SLC22A5	rs2706334	TT
HLA-DQA2	rs28383314	CC
LGALS14	rs412884	CC
HBS1L	rs9389268	GG
IL18R1	rs9807989	TC
KLF3	rs73232890	GA
IKZF2	rs2170572	AG
ITGB8	rs34030463	ADEL(T)
NCF4	rs117582568	GG
/	rs536070968	CC
CCR3	rs138346219	AA
SHC1	rs8191981	GG
GFI1B	rs150813342	CC
ALOX15	rs71368508	CC
S1PR4	rs3746072	GG
IL17RA	rs140221307	TT
GATA1	rs146587548	G
CCR7	rs112401631	TT
BCL2	rs17758695	CC
ADORA1	rs61025910	GG
RUNX1	rs2242886	CC
ERMP1	rs992969	AA
NAA38	rs74480102	GG
RBM17	rs12722547	GG
BCL2L1	rs80054178	TT
/	rs76574427	GG
SPIDR	rs45577137	AA

GENE	SNP	GENOTYPE
BCL2L11	rs72836346	GG
ACKR2	rs2228467	TT
ERG	rs80109907	CC
ASB2	rs11555542	TT
/	rs367705866	AA
CD300LG	rs72836561	CC
CDK6	rs445	CC
SHARPIN	rs34173062	GG
NFKB1	rs113473633	AA
TRIB1	rs16900706	AA

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

Monocytes

Monocytes are white blood cells that protect against bacterial, viral, and other infections. Monocytes kill microbes, remove dead cells, and boost the immune response [R].

Higher monocyte levels most commonly occur due to [R, R]:

- **Infection**
- **Inflammation**
- Autoimmune conditions
- Heart disease

Genetics seems to play an important role, too. Up to **60%** of differences in people's monocyte levels may be due to genetics [R].

Genetically lower monocyte levels may be causally associated with:

- Alzheimer's [R]
- Deep vein thrombosis [R]

TYPICAL LEVELS

Predisposed to typical monocyte levels based on 837,771 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
LYST	rs10927074	CC
LPAR1	rs115162333	TG
B3GNTL1	rs9902102	CC
TET2	rs199741557	AA
CEBPG	rs12151289	GC
RPN1	rs4045811	TC
CKM	rs73036517	AG
IL17RA	rs140221307	TT
FLT3	rs76428106	TT
GFI1	rs150649461	GG
TNFRSF13B	rs34557412	AA
PRLR	rs186272630	GG
ACKR2	rs2228467	TT
ACOXL	rs150449635	TT
EHD3	rs184409696	GG
S1PR4	rs3746072	GG
LYZ	rs1800973	CC
ITGA4	rs10562650	DEL(TT)DEL(TT)
ARHGAP9	rs61758883	GG
TNFSF13B	rs374039502	TT
ARHGAP15	rs140397066	AA
APLF	rs190855339	AA
BCL2	rs17758695	CC
GSDMC	rs35389394	CC
THEMIS2	rs41284294	TT
S1PR2	rs117064827	AA
PLAGL1	rs149110519	CC
SESN3	rs75963851	AA
NFIX	rs1003393	CC

GENE	SNP	GENOTYPE
ST20	rs76648483	AA
CDK6	rs445	CC
HGSNAT	rs145097765	AA
MCL1	rs4970966	GG
NLRP12	rs10424405	AA
SPIDR	rs45577137	AA
CEBPB	rs17196752	CC
TRADD	rs12935169	CC
RASSF3	rs34927483	GG
ETS2	rs58030288	CC

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

Neutrophils

Neutrophils are the most abundant white blood cells in the body. They protect you from bacterial, fungal, and other infections [R].

A high neutrophil level may be a sign of:

- Infections caused by bacteria, fungi, viruses, and parasites [R]
- Inflammation [R, R, R]
- Smoking [R]
- Stress [R]
- Strenuous exercise [R]
- Pregnancy [R].

Low neutrophil levels, on the other hand, can be due to:

- Autoimmune disorders, such as lupus or rheumatoid arthritis [R, R, R, R, R]
- Bone marrow damage and disorders [R, R, R, R]
- Radiation therapy [R, R]
- Certain drugs [R, R, R, R]

Neutrophil levels are also partly affected by genetics. Genetically high neutrophils levels may be causally associated with:

- Chronic pain (lower risk) [R].
- Stroke [R, R]
- Heart disease (CHD) [R].
- Lung health [R].
- High blood pressure [R].
- Alzheimer Disease [R].
- Psoriasis [R].
- High blood sugar [R].

TYPICAL LEVELS

Predisposed to typical neutrophil levels based on 40,949 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ORMDL3	rs3826331	CC
LYST	rs1886654	CC
CSF3R	rs3917932	GG
NBR1	rs199625942	CC
MICB	rs2524079	GA
CXCR2	rs55799208	GG
NCLN	rs144284241	CC
JAML	rs143034248	CC
ACKR1	rs34599082	CC
TTC28	rs62237617	CC
TSPOAP1	rs138284624	CC
IFNA13	rs142938197	CC
FLT3	rs76428106	TT
ATF7	rs117788567	CC
RC3H1	rs77941945	GG
FGB	rs6054	CC
CDK6	rs445	CC
DARS1	rs11548872	GG
PREX1	rs144582521	TT
CXCL5	rs11733208	AA
PLAUR	rs4760	AA
CREB5	rs56388170	GG
CHD7	rs7846314	AA
ADGRL4	rs41313381	CC
ARAP2	rs28530750	GG
NAA38	rs74480102	GG
TNFSF13B	rs374039502	TT
RPN1	rs11359909	DEL(G)DEL(G)
HNF4A	rs1800961	CC

GENE	SNP	GENOTYPE
IFITM2	rs14408	TT
EHD4	rs72726038	CC
PTEN	rs59085061	AA
CDK2AP1	rs11057258	AA
GYPE	rs11735662	CC
PAQR6	rs568036	AA
BCL2	rs17758695	CC
LGI2	rs112783548	GG
LEPR	rs12067936	CC

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

Erythrocyte Sedimentation Rate

Increased ESR levels can be caused by:

- Chronic inflammation
- Pregnancy
- Anemia
- Autoimmune disorders, such as lupus or rheumatoid arthritis
- Some cancers
- Infections
- Kidney disease
- Older age

Factors leading to decreased ESR include:

- Polycythemia (an abnormal increase in red blood cells)
- Sickle cell anemia
- Leukocytosis (high white blood cell count)
- Certain protein abnormalities
- High blood sugar

Note that this report only looks at your genetics predisposition for ESR, which is only one of many contributing factors.

TYPICAL LEVELS

Predisposed to typical ESR levels based on 11,488 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CD55	rs12034383	AG

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

IL-17 (Th17)

Interleukin 17 (IL-17) is a proinflammatory cytokine, produced mainly by Th17 cells [R].

The main role of IL-17 is to defend us against harmful microbes. It also supports our [R, R, R]:

- Gut
- Skin
- Lungs
- Brain

However, excessive IL-17 may cause harmful inflammation and contribute to inflammatory disorders, such as [R, R, R, R, R, R]:

- Obesity
- Type 2 diabetes
- Liver disease
- Asthma
- Autoimmune diseases (e.g., multiple sclerosis, psoriasis, rheumatoid arthritis, Crohn's disease)
- Lung failure

The main factors that may influence IL-17 levels include health status and **genetics** [R].

TYPICAL LEVELS

Predisposed to typical IL-17 levels based on 13,199 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
/	rs117556572	CC
PTPMT1	rs139556855	AA
AMBRA1	rs148500124	AA
PTPRJ	rs185821266	GG
SLC1A1	rs7860087	GG
IKZF2	rs141312283	GG
/	rs187475560	CC
NAV3	rs184080173	TT
EPM2A	rs118117575	AA
/	rs148562661	CC
SHPRH	rs182530774	CC
EPM2A	rs187987903	GG
EPM2A	rs13215785	GG
GRM1	rs117785887	TT
GRM1	rs35548402	AA
MCFD2	rs1446499	TT
TRIB3	rs62191444	GG
/	rs78612928	TT
/	rs12735700	GG
/	rs11110094	GG
PCDH8	rs9568764	GC
GDNF	rs80004049	GG
ANKRD30A	rs146752254	AA
ANKRD30A	rs1148267	AA
EPHA8	rs45498698	GG
KLF7	rs17282552	TT
ZNF184	rs143334272	CC
H3C12	rs145168562	GG
EXTL3	rs149738638	TT

GENE	SNP	GENOTYPE
TCF25	rs11640734	CC
SPTLC2	rs17106604	CC
C1ORF174	rs57920188	TT

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

IL-6

Interleukin-6 (IL-6) is a cytokine with both pro- and anti-inflammatory properties. It's crucial in the defense against infections [R, R, R, R].

Up to 60% of differences in people's IL-6 levels may be due to genetics. Involved genes may influence our bodies' response to IL-6. For example, the *IL6R* gene helps make IL-6 receptors or proteins that bind IL-6 [R, R].

Normally, IL-6 is present in low levels. **An increase in its blood level has been linked to inflammatory conditions**, such as [R]:

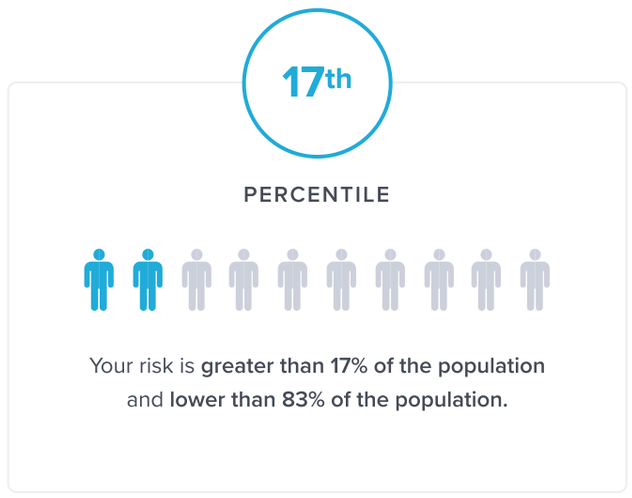
- Autoimmune disorders (e.g., IBS, psoriasis, lupus, systemic sclerosis, rheumatoid arthritis) [R, R, R, R, R, R, R]
- Obesity [R]
- Diabetes [R]
- Migraines [R]
- Infections [R, R, R]

Other factors linked to higher IL-6 levels include:

- Chronic stress [R, R, R]
- Coffee (>2 cups of coffee/day) [R, R]
- Smoking [R, R]
- Drinking alcohol [R, R]
- Intense, prolonged exercise like marathon (temporarily) [R, R, R]
- Older age [R, R, R, R]

TYPICAL LEVELS

Predisposed to typical IL-6 levels based on 616 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
IL6	rs1800795	GG
BTBD7	rs182261775	GG
NOS1	rs146828618	CC
P2RY1	rs114373846	CC
FBLN5	rs113207090	CC
ATP9A	rs73273528	CC
SOX4	rs185628618	GG
TBKBP1	rs72831623	GG
TBKBP1	rs113600793	CC
IL6R	rs4537545	TT
/	rs11110094	GG
AKNA	rs10982213	GG
SERPINE2	rs13412535	GA
/	rs148614378	CT
CASS4	rs1884910	GC
MTAP	rs2004627	CT
ATP2B2	rs4684700	CT
LRAT	rs2404476	AG
C17ORF64	rs3760332	CT
HLA-DQA2	rs660895	AG
GPC6	rs696931	TC

GENE	SNP	GENOTYPE
ZNF703	rs183298717	AA
RAP2B	rs75101555	CC
CDYL2	rs76856708	TT
KMT2E	rs62486616	CC
CAMSAP1	rs117146485	TT
RASEF	rs188644522	AA
KCNK2	rs12079357	AA
ARHGAP28	rs8089344	CC
CDKN2B	rs1333040	TT
IL6R	rs11265618	CC
IL6R	rs10796927	TT
/	rs7824087	AA
AQP10	rs1386821	TT
IL1RN	rs6734238	AA

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

HLA Genes

The Human Leukocyte Antigen (*HLA*) genes play a critical role in regulating immune responses and are strongly associated with autoimmune and inflammatory diseases. This section focuses on *HLA* genes like ***HLA-DQA2***, ***HLA-DOB***, ***HLA-B***, and ***HLA-DRB1***, which influence susceptibility to a range of conditions like celiac disease and rheumatoid arthritis.

Understanding your genetic profile in relation to *HLA* markers can help identify potential autoimmune risks and guide treatment strategies. These insights may also aid in the management of organ transplant rejection and other immune-related challenges.

HIGHER ACTIVITY

HLA (Inflammation)

Likely higher HLA activity

HIGHER ACTIVITY

HLA-DOB (Inflammation)

Likely higher HLA-DOB activity

TYPICAL

HLA-B (Autoimmune)

Likely typical HLA-B genetics

TYPICAL ACTIVITY

HLA-DQ (Inflammation)

Likely typical HLA-DQ activity

TYPICAL

HLA-DRB1 (Autoimmunity)

Likely typical HLA-DRB1 genetics

TYPICAL

HLA-DQA2 (Inflammation)

Likely typical HLA-DQA2 genetics

HLA (Inflammation)

HLA-B27 is a form of HLA-B that reatly increases the risk of certain autoimmune diseases that fall under the umbrella of spondyloarthritis. It is inherited in a dominant fashion; that is, you have the same risk of autoimmune diseases if you have one or two copies of the risk allele. The following rare alleles have been associated with HLA-B27 [R, R, R, R, R]:

- 'A' at rs4349859
- 'G' at rs13202464
- 'T' at rs116488202

Two alleles — DQA1*0501 and DQB1*0201 — form the DQ2.5 haplotype, which codes for the DQ2.5 receptor on white blood cells. The 'T' variant of rs2187668 serves as a genetic marker — it tags the DQ2.5 haplotype with high precision. This variant has been associated with:

- Celiac disease [R, R]
- Allergies to latex and fruits [R]
- Grave's disease [R, R, R, R]

The 'C' variant of rs9275596, located between *HLA-DQB1*, *HLA-DQA2*, and *HLA-DQB2*, causes structural changes in this DNA region that may affect its interaction with regulatory proteins. This variant has been linked to an increased risk of [R]:

- Peanut allergy [R, R]
- Shrimp allergy [R]
- Multiple sclerosis [R]
- Primary sclerosing cholangitis [R]

The 'G' allele at rs2395185 marks the allele HLA-**DRB1*1101** and is linked to a higher expression of the *HLA-DRB1* and *DQA1* genes. This variant has been associated with higher rates of ulcerative colitis in several studies, as well as with type 1 diabetes. On the bright side, it also predicts a better response to anti-TNF treatment [R, R, R, R, R, R, R].

Another polymorphism, rs9271366, marks HLA-**DRB1*1502** and may have a role in IBD by changing the HLA-DR structure. Its 'G' allele has been associated with higher rates of ulcerative colitis in different populations [R, R, R, R, R, R, R].

The 'C' allele of rs3763313 has also been associated with higher rates of Crohn's disease and ulcerative colitis. This variant has also been associated with an increased risk of tuberculosis but may be protective against dilated cardiomyopathy [R, R, R, R].

Two variants believed to increase or altere HLA activity that are always inherited together, 'G' at rs9275572 and 'C' at rs9275524, have been associated with increased odds of *alopecia areata* (patchy hair loss) [R, R].

Another variant, 'C' at rs6906021, is believed to increase *HLA-DQA1* activity and has been linked to increased allergic sensitization and hypothyroidism [R, R].

The 'C' variant of rs9271588 is also believed to increase *HLA-DQA1* activity and has been associated with:

- Allergy to wheat protein [R]
- Natural killer T-cell lymphoma [R]

People with the "C" allele at rs2516049 have 15% higher rates of hypothyroidism. The same SNP is also associated with [R, R, R, R]:

- Epstein-Barr virus infection
- Asthma
- Ulcerative colitis

HIGHER ACTIVITY

Likely higher HLA activity based on 15 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HLA-DQA2	rs9275596	CT
/	rs9271366	GA
HLA-DQA2	rs2395185	GT
HLA-DQA2	rs9275572	AG
HLA-DQA1	rs6906021	TC
HLA-DQA2	rs9271588	TC
HLA-DQA2	rs9275524	TC
HLA-DOB	rs10484565	GA
HLA-DQA1	rs9784858	GC
MICA	rs4349859	GG
HLA-DRB5	rs3763313	AA
HLA-DRB1	rs2516049	TT
HLA-DQA1	rs2187668	CC
HLA-C	rs13202464	AA
MICA	rs116488202	CC
TAP2	rs2071479	CC

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

Both the ‘A’ allele of rs10484565 and the ‘C’ allele of rs9784858 have been associated with an increased risk of rheumatoid arthritis, especially in smokers [R, R, R].

Finally, the ‘T’ allele of rs2071479 has been associated with higher blood sugar levels due to type 1 and type 2 diabetes. The ‘C’ allele of this variant may cause the production of proteins with decreased activity [R, R].

HLA-DOB (Inflammation)

Both the ‘A’ allele of rs10484565 and the ‘C’ allele of rs9784858 have been associated with an increased risk of rheumatoid arthritis, especially in smokers [R, R, R].

These variants are believed to increase HLA-DOB activity. In people with rheumatoid arthritis and other autoimmune conditions, HLA proteins may have increased activity or impaired structure. These changes cause them to target the body’s own components, such as joint cartilage, and trigger inflammation [R].

Alternatively, the ‘T’ allele of rs2071479 has been associated with higher blood sugar levels due to type 1 and type 2 diabetes. The ‘C’ allele of this variant may cause the production of proteins with decreased activity [R, R].

HIGHER ACTIVITY

Likely higher HLA-DOB activity based on the genetic variants we looked at

18%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **18% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HLA-DOB	rs10484565	GA
HLA-DQA1	rs9784858	GC
TAP2	rs2071479	CC

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

HLA-B (Autoimmune)

The HLA-B27 antigen is one of the hundreds of possible forms of HLA-B. It supports the antiviral immune response by "flagging" peptides from viruses—such as influenza, HIV, and Epstein-Barr—and presenting them to T-killer cells [R].

HLA-B27 greatly increases the risk of certain autoimmune diseases, such as [R]:

- Psoriasis
- Ankylosing spondylitis (spine deformation)
- IBD (inflammatory bowel disease), in combination with spondylitis
- Reactive arthritis (Reiter's syndrome)—inflammation of the joints, urethra, and eyes

These conditions fall under the umbrella of spondyloarthritis, with the most common form being ankylosing spondylitis (AS). In AS, inflammation gradually fuses the vertebrae in the spine, causing back pain and limited movement. It usually affects young men [R, R].

Despite the well-known link, it's still not clear how HLA-B27 provokes autoimmunity. According to the main theories, an error probably occurs in one of two processes [R, R, R, R]:

1. **The way it presents peptides to T-killer cells**: upon activation, T-killer cells are supposed to only flag the foreign peptide and not HLA-B27 itself. However, T-cells may mistakenly flag HLA-B27 fragments or other peptides as foreign and attack them.
2. **The biochemical properties (structure) of HLA-B27 itself**: the protein may misfold in such a way that causes inflammation within the cell and triggers an immune response.

Whatever the root cause, one thing is for sure: inflammation lurks behind all autoimmune disorders associated with HLA-B27 [R].

HLA-B27 is inherited in a dominant fashion; that is, you have the same risk of autoimmune diseases if you have one or two copies of the risk allele. The following rare alleles have been associated with HLA-B27 [R, R, R, R, R]:

- 'A' at rs4349859
- 'G' at rs13202464
- 'T' at rs116488202

Please note: To confirm the presence of this gene and detect the exact subtype, you may want to do the HLA-B27 blood test.

TYPICAL

Likely typical HLA-B genetics based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MICA	rs4349859	GG
HLA-C	rs13202464	AA
MICA	rs116488202	CC

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

HLA-DQ (Inflammation)

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

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

This variant was also linked to an increased risk of allergies to latex and fruits in a study of 78 patients [R].

The ‘G’ allele at rs2395185 marks the allele HLA-**DRB1*1101** and is linked to a higher expression of the *HLA-DRB1* and *DQA1* genes. This variant has been associated with higher rates of ulcerative colitis in several studies, as well as with type 1 diabetes. On the bright side, it also predicts a better response to anti-TNF treatment [R, R, R, R, R, R, R].

The ‘C’ variant of rs9275596, located between *HLA-DQB1*, *HLA-DQA2*, and *HLA-DQB2*, causes structural changes in this DNA region that may affect its interaction with regulatory proteins. This variant has been linked to an increased risk of [R]:

- Peanut allergy [R, R]
- Shrimp allergy [R]
- Multiple sclerosis [R]
- Primary sclerosing cholangitis [R]

A study of 4,332 participants associated the ‘G’ variant of rs9275572 with up to 5-fold higher odds of alopecia areata. This variant has also been associated with an increased risk of [R]:

- Liver cancer caused by HBV [R, R]
- Systemic lupus erythematosus [R]
- Lung cancer [R]

Another allele, ‘C’ at rs9275524, was associated with approximately twice the odds of alopecia areata in a study of 10,796 participants [R].

These two variants, believed to increase or altere HLA activity, are always inherited together, so they act as a single genetic factor. This means you will either have both ‘problematic’ genotypes or none of them.

Another variant, ‘C’ at rs6906021, is believed to increase *HLA-DQA1* activity and has been linked to increased allergic sensitization. Another study, of 29,349 subjects, associated this variant with hypothyroidism [R, R].

Finally, the ‘C’ variant of rs9271588 is also believed to increase *HLA-DQA1* activity and has been associated with:

- Allergy to wheat protein [R]
- Natural killer T-cell lymphoma [R]

TYPICAL ACTIVITY

Likely typical HLA-DQ activity based on 7 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HLA-DQA2	rs9275596	CT
HLA-DQA2	rs2395185	GT
HLA-DQA2	rs9275572	AG
HLA-DQA1	rs6906021	TC
HLA-DQA2	rs9271588	TC
HLA-DQA2	rs9275524	TC
HLA-DQA1	rs2187668	CC

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

HLA-DRB1 (Autoimmunity)

HLA-DRB1 plays vital roles by controlling the immune response against pathogens. It presents pathogenic peptides to white blood cells, enabling their removal. However, specific variants of this receptor may mistakenly flag our own peptides and thus contribute to autoimmune inflammation. This phenomenon is known as “molecular mimicry” and may lead to autoimmunity [R, R, R].

For instance, the ‘G’ allele at rs2395185 marks the allele HLA-**DRB1*1101** and is linked to a higher expression of the *HLA-DRB1* and *DQA1* genes. This variant has been associated with higher rates of ulcerative colitis in several studies, as well as with type 1 diabetes. On the bright side, it also predicts a better response to anti-TNF treatment [R, R, R, R, R, R, R].

Another polymorphism, rs9271366, marks HLA-**DRB1*1502** and may have a role in IBD by changing the HLA-DR structure. Its ‘G’ allele has been associated with higher rates of ulcerative colitis in different populations [R, R, R, R, R, R, R].

Finally, the ‘C’ allele of rs3763313 is associated with 19% higher rates of Crohn’s disease, according to a meta-analysis of three studies and over 15,000 European subjects. Another meta-analysis with nearly 9.5K European participants linked the same allele with ulcerative colitis. This variant has also been associated with an increased risk of tuberculosis. In contrast, it may be protective against dilated cardiomyopathy [R, R, R, R].

One of the earliest observed genetic associations with Graves' disease (autoimmune hyperthyroidism) refers to a so-called "DR3-DQ2" haplotype (a group of genetic variants). It includes three alleles — **DRB1*0301**, **DQB1*0201**, and **DQA1*0501** — and correlates with more than doubled GD rates. **DRB1*0301** is likely the lead variant responsible for the association with Graves’ disease [R, R, R, R].

Studying its connection with other autoimmune conditions, scientists have identified a “tag” SNP for DRB1*0301: the “T” allele at rs2187668 [R].

The above haplotype is among the crucial genetic factors of celiac disease in European descendants, confirming a strong connection between gluten intolerance and autoimmune thyroid conditions. Other associated conditions include [R, R, R, R]:

- Systemic lupus erythematosus (SLE)
- Type 1 diabetes
- Idiopathic membranous nephropathy (kidney disease)

A study of over 39,000 participants found a SNP in the HLA-DRB1 gene slightly associated with low thyroid hormones. People with the “C” allele at rs2516049 had 15% higher rates of hypothyroidism. The same SNP is also associated with [R, R, R, R]:

- Epstein-Barr virus infection
- Asthma
- Ulcerative colitis

TYPICAL

Likely typical HLA-DRB1 genetics based on 4 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HLA-DQA2	rs2395185	GT
/	rs9271366	GA
HLA-DRB5	rs3763313	AA
HLA-DRB1	rs2516049	TT
HLA-DQA1	rs2187668	CC

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

HLA-DQA2 (Inflammation)

Variants that increase or altere HLA-DQA2 activity may contribute to the autoimmune disruption of hair growth, as seen in alopecia areata. For instance, a study of 4,332 participants associated the ‘G’ variant of rs9275572 with up to 5-fold higher odds of alopecia areata. This variant has also been associated with an increased risk of [R]:

- Liver cancer caused by HBV [R, R]
- Systemic lupus erythematosus [R]
- Lung cancer [R]

Interestingly, the ‘G’ variant is protective against liver cancer caused by HCV. This suggests the rs9275572 polymorphism is involved in two different liver cancer-related pathways [R].

Another allele, ‘C’ at rs9275524, was associated with approximately twice the odds of alopecia areata in a study of 10,796 participants [R].

These two variants are almost always inherited together, **acting as a single genetic factor**. This means you will usually have either both ‘problematic’ genotypes or none of them.

TYPICAL

Likely typical HLA-DQA2 genetics based on the genetic variants we looked at

42%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **42% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HLA-DQA2	rs9275524	TC

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

Other Inflammation Genes

In addition to HLA genes, several other genes influence the body's inflammatory response. This section examines key inflammation-related genes, such as ***NF-kB***, ***IL6***, ***TNF***, and ***PTPN22***, which are linked to both inflammation and autoimmune diseases. It also covers genes involved in histamine regulation and gut inflammation.

Understanding your genetic predisposition related to these genes can help you manage inflammation, prevent flare-ups of chronic conditions, and explore personalized treatment options to support long-term health.

HIGHER ACTIVITY

NF-kB (Inflammation)

Likely higher NF-kB activity

HIGHER ACTIVITY

NLRP3 (Gut Inflammation)

Likely higher NLRP3 activity

LOWER ACTIVITY

SH2B3 (Autoimmunity)

Likely lower SH2B3 activity

LOWER ACTIVITY

HNF4A (Gut Inflammation)

Likely lower HNF4A activity

HIGHER ACTIVITY

IL6R (Weight)

Predisposed to higher IL6R activity

HIGHER ACTIVITY

ANKRD55 (Autoimmune)

Predisposed to higher ANKRD55 activity

LOWER ACTIVITY

CTLA4 (Autoimmunity)

Likely lower CTLA4 activity

TYPICAL ACTIVITY

TLR4 (Inflammation)

Likely typical TLR4 activity

TYPICAL

PTPN22 (Autoimmunity)

Likely typical PTPN22 genetics

TYPICAL ACTIVITY

DAO (Histamine)

Likely typical DAO activity

TYPICAL ACTIVITY

STAT3 (Th1/Th17)

Likely typical STAT3 activity

TYPICAL ACTIVITY

TNF Gene (Inflammation)

Likely typical TNF activity

TYPICAL ACTIVITY

IL13 (Allergies, Lung Function)

Predisposed to typical IL13 activity

TYPICAL ACTIVITY

IL4 (Allergies, Autoimmunity)

Likely typical IL4 activity

TYPICAL ACTIVITY

HRH4 (Allergies, Inflammation)

Predisposed to typical HRH4 activity

TYPICAL ACTIVITY

CRP Gene

Predisposed to typical CRP gene activity

TYPICAL ACTIVITY

PADI4 (Autoimmune)

Predisposed to typical PADI4 activity

TYPICAL ACTIVITY

IL10 Gene (Autoimmunity)

Predisposed to typical IL10 activity

TYPICAL GENETICS

SLC22A4 (Gut Inflammation)

Likely typical SLC22A4 genetics

LOWER ACTIVITY

IL1B (Inflammation/ Fatigue)

Likely lower IL1B activity

HIGHER ACTIVITY

ABCB1 (Chronic Lyme)

Likely higher ABCB1 activity

HIGHER ACTIVITY

HNMT (Histamine)

Likely higher HNMT activity

LOWER ACTIVITY

JAK2 (Gut Inflammation)

Likely lower JAK2 activity

LOWER ACTIVITY

IL8 (Inflammation)

Likely lower IL8 activity

LOWER ACTIVITY

IL21 (Autoimmunity & Allergies)

Predisposed to lower IL21 activity

NF-KB (Inflammation)

A large-scale study of over 1,700 people found that people who carried the minor *NFKBIL1* alleles 'T' at rs2230365 and 'C' at rs2255798 showed enhanced performance on several common tests of cognitive processing speed (such as the '*symbol search*' and '*digit-substitution*' tasks) [R].

These variants may decrease NF-kB activity, making the brain more resistant to inflammation [R].

Two rare *NFKBIE* variants resulting in increased NF-kB activity, 'G' at rs2233434 and 'T' at rs2233424, have been associated with an increased risk of rheumatoid arthritis [R, R, R, R].

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

HIGHER ACTIVITY

Likely higher NF-kB activity based on 4 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NFKBIE	rs2233434	GA
TCTE1	rs2233424	TC
HLA-C	rs2230365	CC
DDX39B	rs2255798	GG

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

NLRP3 (Gut Inflammation)

One of the main *NLRP3* variants is **rs10754558**. People with the "**GG**" **genotype** were about **2.5 times** more likely to have **ulcerative colitis** in one study. Interestingly, this variant has also been linked to severe food allergies [R, R, R].

Another well-researched variant is **rs35829419**. Its **"C" allele** is linked to higher odds of **IBD** and other inflammatory conditions like rheumatoid arthritis and eczema [R, R].

Other *NLRP3* variants linked to higher odds of IBD (Crohn's) include [R]:

- rs3806265-C
- rs4925648-T
- rs3738447-G

These variants likely increase *NLRP3* activity, contributing to excess inflammation [R, R].

HIGHER ACTIVITY

Likely higher NLRP3 activity based on 5 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NLRP3	rs35829419	CC
NLRP3	rs10754558	CG
NLRP3	rs3806265	CC
NLRP3	rs3738447	GG
NLRP3	rs4925648	CC

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

SH2B3 (Autoimmunity)

SH2B3 variants associated with autoimmune conditions probably reduce the activity of this gene, which then fails to suppress an autoimmune reaction. Further research should clarify the exact mechanisms.

For instance, the 'C' allele of **rs653178** is believed to promote the Th1 response and has been associated with autoimmune conditions such as [R, R, R]:

- Lupus [R]
- Celiac disease [R]
- Graves' disease and elevated thyroid peroxidase antibodies [R, R, R]
- Rheumatoid arthritis [R]

In contrast, this variant may protect against asthma [R].

Another variant, 'T' at **rs3184504**, decreases SH2B3 levels in the blood and has been linked to [R]:

- Celiac disease [R]
- Hashimoto's disease [R]

This variant may also raise blood pressure by increasing the levels of a protein called beta-2 microglobulin (β2M or B2M) and the inflammatory cytokines IL-6, IL-1b, and TNF-alpha [R, R, R, R, R, R].

Finally, the 'A' allele of **rs597808** has been associated with higher rates of lupus [R].

These variants are usually inherited together, meaning you will most likely carry none or all of them.

LOWER ACTIVITY

Likely lower SH2B3 activity based on the genetic variants we looked at

24%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **24% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SH2B3	rs653178	CT
SH2B3	rs3184504	TC
SH2B3	rs597808	AG

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

HNF4A (Gut Inflammation)

Several studies suggest that certain genetic variants of *HNF4A* may increase the risk of developing ulcerative colitis (UC) [R, R, R].

A genome study including over 7,000 people with European ancestry found that **the 'C' allele in rs6017342 is associated with greater rates of UC** [R].

Likewise, two more studies of over 2,000 Dutch and 1,900 Korean patients also found that those carrying a 'C' allele were significantly more likely to have UC [R, R].

The HNF4A protein helps maintain the mucosal barrier in the intestines. This barrier is responsible for protecting the lining of the intestines while allowing nutrients to be absorbed. Variants resulting in lower levels of this protein may disrupt this mucosal barrier [R, R, R].

LOWER ACTIVITY

Likely lower HNF4A activity based on the genetic variants we looked at

25%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **25% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HNF4A	rs6017342	CC

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

IL6R (Weight)

Three *IL6R* variants (rs2229238-T, rs4845623-G, and rs2228145-C) have been associated with weight gain in specific populations [R, R].

The rs2229238-T variant has been associated with increased abdominal fat in young Taiwanese girls and with increased BMI in indigenous Pima people (native to Arizona) [R, R].

This variant has also been associated with:

- Lower CRP levels [R]
- Increased risk of type 1 diabetes [R]
- Decreased risk of tuberculosis [R]

The rs4845623-G and rs2228145-C variants have been associated with increased BMI in indigenous Pima people only. No studies have investigated the relationship of these SNPs to weight in other populations so far [R].

Both variants have been associated with lower CRP levels [R, R].

In addition, the 'C' allele of rs2228145 has been associated with:

- Higher IL6R levels [R, R, R, R, R]
- Higher IL-6 levels [R]
- Higher iron levels [R]
- Higher relative monocyte count [R]
- Lower LDL cholesterol levels [R]
- Decreased risk of rheumatoid arthritis [R, R, R]
- Increased risk of eczema, hay fever, and asthma [R, R, R]

HIGHER ACTIVITY

Predisposed to higher IL6R activity based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
IL6R	rs4845623	GG
IL6R	rs2228145	CC
IL6R	rs2229238	CC

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

ANKRD55 (Autoimmune)

IL-6 has been heavily implicated in rheumatoid arthritis through its influence on pathological inflammation. Rheumatoid arthritis patients tend to have significantly higher IL-6 levels than healthy people, and this high IL-6 can lead to increased destruction of joint tissues [R, R].

In line with this, the following minor *ANKRD55* variants have been associated with a decreased risk of rheumatoid arthritis:

- 'A' of **rs71624119** [R, R]
- 'A' of **rs7731626** [R, R]
- 'T' of **rs10065637** [R, R, R]
- 'A' of **rs10040327** [R]

These variants may decrease the production of IL-6 and other inflammatory signals [R, R].

HIGHER ACTIVITY

Predisposed to higher ANKRD55 activity based on 4 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ANKRD55	rs71624119	GG
ANKRD55	rs7731626	GG
ANKRD55	rs10065637	CC
ANKRD55	rs10040327	CC

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

CTLA4 (Autoimmunity)

The best-researched *CTLA4* variant is **rs231775** (A49G). Its minor 'G' allele may decrease *CTLA4* gene expression and the levels of this protein in immune cells. This variant has been associated with an increased risk of autoimmune conditions such as [R, R, R]:

- Autoimmune thyroid disease (both Hashimoto's and Graves' disease) [R, R]
- Latent autoimmune diabetes [R, R]
- Type 1 diabetes [R, R]
- Rheumatoid arthritis [R, R]
- Addison's disease [R]
- Type 1 autoimmune hepatitis [R]
- Systemic lupus erythematosus [R]
- Alopecia areata [R]

Another well-researched variant is **rs3087243** (CT60). Its major 'G' allele has been associated with lower levels of the soluble CTLA4 isoform and an increased risk of [R]:

- Rheumatoid arthritis [R, R, R, R, R, R]
- Autoimmune thyroid disease (both Hashimoto's and Graves' disease) [R, R, R]
- Type 1 diabetes [R]
- Systemic lupus erythematosus [R]

Another minor allele, 'T' at ***rs231779***, has been associated with an increased risk of Graves' disease [R, R, R].

Finally, the major 'G' allele of **rs11571302** has been associated with an increased risk of rheumatoid arthritis [R, R].

LOWER ACTIVITY

Likely lower CTLA4 activity based on 4 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CTLA4	rs3087243	GG
CTLA4	rs231775	GA
CTLA4	rs11571302	GG
CTLA4	rs231779	TC

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

TLR4 (Inflammation)

The most well-researched TLR4 variant is rs4986791. Its minor 'T' allele is believed to increase TLR4 activation based on its pro-inflammatory effects [R].

This variant has been associated with an increased risk of:

- Crohn's disease and severe childhood IBD [R, R]
- Rheumatoid arthritis [R]
- Alzheimer's disease [R]
- Septic shock [R]
- Asthma [R]
- Tuberculosis [R]
- Gum disease [R]
- Glaucoma [R]
- Cancer [R, R]

However, this variant has also been linked to:

- Acne severity [R]
- Coronary artery disease risk [R]
- Type 2 diabetes risk [R]
- Susceptibility to HIV infection [R]

The other well-researched variant is rs4986790. Its 'G' allele is believed to increase the amount of TLR4 produced and has been associated with an increased risk of [R]

- PTSD [R]
- Atopic dermatitis [R]
- Type 2 diabetes [R]
- Glaucoma [R]
- Age-related macular degeneration [R]
- Urinary tract infections [R]
- *Helicobacter pylori* infection [R]
- Hepatitis C infection [R]
- Septic shock [R]
- Cancer [R, R]

However, this has also been associated with a reduced acne severity [R].

Both variants are usually inherited together, so you will most likely carry either both or neither of them.

TYPICAL ACTIVITY

Likely typical TLR4 activity based on the genetic variants we looked at

89%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **89% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TLR4	rs4986791	CC
TLR4	rs4986790	AA

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

PTPN22 (Autoimmunity)

According to a study of almost 40,000 people, two *PTPN22* variants—rs2476601 and rs6679677—are crucial genetic contributors to hypothyroidism. Carriers of minor 'A' alleles on one of these variations have a 36% higher risk of hypothyroidism, compared with people who carry major alleles (G and C, respectively). These variants are almost always inherited together, so you will most likely carry either both or neither of them [R].

The rs2476601 variant also showed a significant connection with Graves' disease. In two British studies of 2,700 participants, the 'A' allele was associated with 43-88% higher rates. Two studies of over 900 Polish subjects confirmed this link and found that people with the 'A' allele get diagnosed younger. The same allele correlated with 85% higher rates of Graves' disease in a Chinese meta-analysis [R, R, R, R, R].

The 'A' variants of these polymorphisms have also been associated with an increased risk of:

- Rheumatoid arthritis [R, R, R, R, R, R, R, R, R]
- Lupus [R, R, R, R, R, R, R]

Other conditions associated with the 'A' allele include [R, R, R, R, R, R]:

- Juvenile idiopathic arthritis
- Type 1 diabetes
- Vitiligo
- Myasthenia gravis
- Alopecia areata
- Drug-induced liver injury
- Addison's disease
- Idiopathic inflammatory myopathies
- Immune thrombocytopenia
- Anti-neutrophil cytoplasmic antibody-associated vasculitis

However, this allele showed a protective effect (around 16% lower odds) in the case of Crohn's disease [R].

Other *PTPN22* variants such as rs2488457 and rs33996649 are also associated with autoimmune diseases, epecially rheumatoid arthritis, type 1 diabetes, and ulcerative colitis.

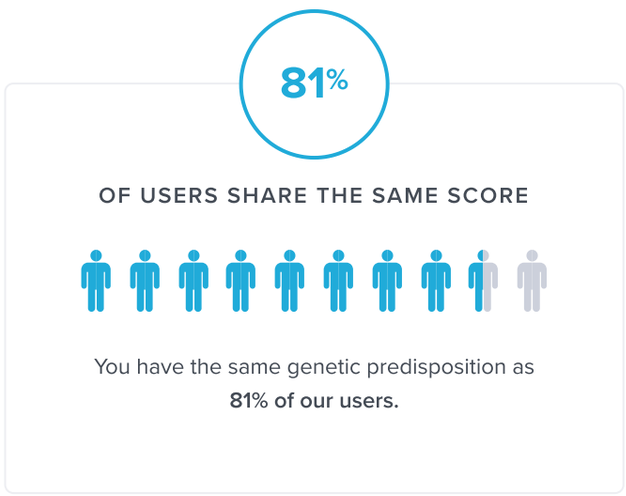
Scientists haven't figured out the exact mechanism by which these variations cause harm. The key may lie in defective PTP synthesis, which disables the protein and prevents the removal of self-attacking lymphocytes (T and B cells) [R, R].

Risk variants may contribute to autoimmunity by:

- Being less capable of fighting infections
- Increasing T cell, Th1 and Th17 activity
- Suppressing regulatory T cells (Tregs)
- Activating B cells and self-attacking B cells

TYPICAL

Likely typical PTPN22 genetics based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PTPN22	rs6679677	CC
PTPN22	rs2476601	GG

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

DAO (Histamine)

Four *AOC1* variants have been associated with lower levels of the DAO enzyme and higher incidence of migraine: rs1049793, rs2052129, rs10156191, and rs1049742 [R, R, R].

In almost every case, the minor allele reduces DAO activity, possibly by producing a non-functional or less functional version of the enzyme. Lower DAO enzyme activity reduces the body's capacity to break down and deactivate histamine, potentially leading to inflammation and pain [R, R, R].

The exception to the rule is rs2052129, at which the minor 'T' allele, associated with lower DAO levels, seems to be protective against migraines, but only in men. In women, there was no statistical difference between any genotype [R].

Two other minor variants, 'T' at rs2268999 and 'A' at rs2071514, have also been linked to lower DAO activity [R].

Some of these variants have also been associated with hypersensitive responses to non-steroidal anti-inflammatory drugs [R].

TYPICAL ACTIVITY

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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
AOC1	rs1049793	CG
AOC1	rs2071514	GA
AOC1	rs1049742	CC
AOC1	rs10156191	CC
AOC1	rs2268999	AA
AOC1	rs2052129	GG
AOC1	rs35070995	AA

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

STAT3 (Th1/Th17)

The most widely-studied *STAT3* polymorphism is rs744166. Although its mechanism remains unknown, some researchers have suggested that the minor 'G' variant may alter the structure of the STAT3 protein. This may favor its interaction with some cytokines, leading to an increased activation of the immune system [R].

Its minor allele has been associated with an increased risk of the following autoimmune diseases:

- Multiple sclerosis [R, R, R]
- Psoriasis and psoriatic arthritis [R, R, R]
- Vitiligo [R]
- Systemic lupus erythematosus [R]

However, the major 'A' variant has been associated with an increased risk of IBD (both Crohn's disease and ulcerative colitis). Cell-based research suggests that STAT3 activation in the gut lining promotes wound healing, kills infectious bacteria, and reduces inflammation, which might explain this association [R, R, R, R, R].

Similarly, this variant has been associated with a decreased risk of autoimmune thyroid disorders such as Hashimoto's thyroiditis and Grave's disease in some ethnicities but not in others [R, R, R].

Alternatively, the 'G' allele of this variant has been associated with abdominal obesity and increased weight gain from saturated fat intake [R].

Another polymorphism of this gene is rs2293152. Its minor 'G' allele was associated with increased frequency and severity of IBD (Crohn's disease, ulcerative colitis, or both) in three different studies, but not in a fourth one [R, R, R, R].

In contrast, the major 'C' allele was the one associated with multiple sclerosis in two studies (but only statistically significant in one of them). Although the mechanism of this variant is unknown, the fact that its effects are the opposite of those of rs744166 suggests the minor variant may decrease STAT3 activity [R, R].

The minor 'G' allele of another variant, rs4796793, reduces *STAT3* expression. In a Chinese population, this allele was less common among people with Crohn's disease. However, this variant was unrelated to ulcerative colitis [R, R, R, R].

The major 'T' allele of another *STAT3* polymorphism, rs3816769, was associated with both Hashimoto's and Graves' disease in a Polish study. People with autoimmune thyroid disorders had 'TT' over 6 times more frequently than 'CC' [R]!

Finally, the minor allele 'C' allele of rs1053005 was more common in people with either Hashimoto's or Graves' disease in a Chinese study. This allele increases STAT3 production, which could contribute to autoimmune thyroid issues by over-activating the immune system [R, R].

TYPICAL ACTIVITY

Likely typical STAT3 activity based on 5 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
STAT3	rs3816769	TT
STAT3	rs4796793	CC
STAT3	rs2293152	CG
STAT3	rs744166	AA
STAT3	rs1053005	TT

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

TNF Gene (Inflammation)

The **rs1800629** polymorphism (also known as *TNF* -308) is one of the most researched SNPs in the *TNF* gene. **The 'A' allele is associated with 6-7 times higher levels of TNF-alpha** [R].

In line with this, this variant has been associated with:

- IBD risk and severity[R, R, R]
- ARDS and sepsis [R, R]
- Chronic pain [R, R, R, R]
- Obesity [R, R, R, R]
- Hashimoto's disease [R]
- Acne [R]
- Insulin resistance and poor blood sugar control [R]
- Asthma and COPD [R, R]
- Heart disease [R]
- Rheumatoid arthritis [R, R]
- Systemic lupus erythematosus [R]
- Liver and digestive system cancers [R, R]

Probably due to its association with lower inflammation and a reduced risk of these conditions, the 'G' allele has been associated with a longer lifespan. In contrast, it has been linked to higher PTSD severity [R, R, R].

Another variant, 'C' at **rs1799964** (commonly referred to as TNF -1031), may increase TNF levels and has been associated with an increased risk of [R]:

- Crohn's disease [R, R]
- Hashimoto's disease [R]

Finally, the 'C' variant at **rs1799724** (commonly referred to as TNF -857C) may also increase TNF levels and has been associated with an increased risk of [R]:

- IBD [R, R, R]
- Acne [R]

People with variants linked to higher TNF-alpha levels may benefit more from interventions that counteract the negative effects of this cytokine, such as cold immersion for exercise recovery.

TYPICAL ACTIVITY

Likely typical TNF activity based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TNF	rs1799724	CT
TNF	rs1800629	GG
TNF	rs1799964	TT

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

IL13 (Allergies, Lung Function)

The most widely investigated IL13 variant is **rs20541**. Its minor 'A' allele has been linked to elevated IL-13 and IgE levels. The 'A' allele has also been associated with an increased risk of:

- Allergic rhinitis [R, R, R, R]
- Asthma [R, R]
- Alopecia areata [R]
- COPD (only in Caucasians) [R]
- ARDS in critically ill patients [R]
- Lupus [R]

However, this variant has been associated with a decreased risk of glioma [R].

The 'A' allele of **rs1295685** has also been associated with elevated IgE levels, as well as with an increased risk of asthma, psoriasis, eczema, and COPD [R, R, R, R, R, R, R].

The 'T' allele of **rs847** has been associated with an increased risk of COPD and eczema, as well as an increased severity of rosacea symptoms [R, R, R].

These variants are usually inherited together, meaning you will most likely have all or none of them.

Another well-researched allele, 'T' at **rs1800925**, may increase IL-13 levels. This variant has been associated with an increased risk of [R]:

- Asthma [R, R]
- COPD [R, R, R]

This variant may enhance the negative effects of cigarette smoke on lung function. On the bright side, it has been associated with a decreased risk of cancer, particularly glioma [R, R].

The 'T' allele of **rs1295686** has been associated with elevated IL-13 and IgE levels, as well as with an increased risk of food allergies, asthma, acute myeloid leukemia, and pain before breast cancer surgery [R, R, R, R, R].

Another variant usually inherited with the previous one, the 'A' allele of **rs848**, has been associated with elevated IgE levels and an increased risk and severity of asthma, COPD, eczema, and alopecia areata [R, R, R, R, R, R].

In contrast, this variant has been linked to a decreased risk of psoriatic arthritis [R, R].

Finally, the 'A' allele of **rs2066960** may increase IL13 activity based on its link to elevated IgE levels. This variant has been associated with an increased risk of asthma and wheezing [R, R, R].

TYPICAL ACTIVITY

Predisposed to typical IL13 activity based on 7 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
IL13	rs20541	GG
SLC22A5	rs1800925	CC
SLC22A5	rs1295686	CC
SLC22A5	rs848	CC
SLC22A5	rs1295685	GG
SLC22A5	rs2066960	CC
SLC22A5	rs847	CC

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

IL4 (Allergies, Autoimmunity)

The best-researched *IL4* variant is **rs2243250** (589C>T). People with the minor 'T' allele may have decreased Th2 cell activity and increased anti-inflammatory IL-4 levels [R].

This variant has been associated with an increased risk of allergic conditions such as:

- Hay fever [R]
- Eczema [R]
- Asthma [R, R, R]

Interestingly, one study found that a well-studied association between vitamin D and food allergies was altered by this variant. Vitamin D deficiency predicted food allergy only in people with the detrimental 'C' allele [R].

In contrast, the 'T' allele has been associated with a *decreased* risk of autoimmune diseases such as:

- Rheumatoid arthritis [R]
- Multiple sclerosis [R, R]
- Autoimmune thyroid disease [R]

The 'T' allele may also increase the risk of infections such as tuberculosis and hepatitis B and C. However, it has also been associated with a decreased risk of severe symptoms in people with this and other infectious diseases such as HIV and malaria and with a better response to the hepatitis B vaccine [R, R, R, R, R, R, R].

In a study of 430 Caucasian Russians, ARDS patients were less likely to have the high-producing 'T' allele [R].

TYPICAL ACTIVITY

Likely typical IL4 activity based on the genetic variants we looked at

70%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **70% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SLC22A5	rs2243250	CC

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

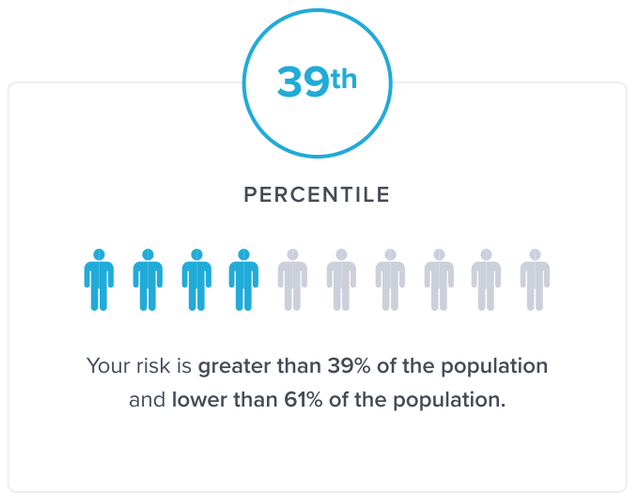
HRH4 (Allergies, Inflammation)

Several *HRH4* variants have been associated with allergic and inflammatory conditions. Although the effects of these variants on gene activity haven't been established, they may cause increased inflammation through enhanced histamine signaling. They include:

- The 'A' allele of **rs77485247**: associated with increased prevalence and severity of allergic rhinitis, eczema, and Ménière's disease and higher risk of adverse reactions from antihistamines [R, R, R].
- The rare 'T' allele of **rs77041280**: associated with increased prevalence of allergic rhinitis and eczema, higher risk of adverse reactions from antihistamines, and worse effectiveness of these drugs [R, R].
- The 'C' allele of **rs17187619**: associated with increased prevalence of asthma [R].
- The 'T' allele of **rs527790**: associated with increased prevalence of asthma [R].
- The 'C' allele of **rs487202:** associated with increased prevalence of asthma [R].
- The 'A' allele of **rs615283**: associated with increased prevalence and severity of psoriasis [R].
- The 'G' allele of **rs17203314**: associated with increased prevalence and severity of psoriasis [R].
- The 'C' allele of **rs524149**: associated with increased prevalence and severity of psoriasis [R].
- The 'C' allele of **rs17797945**: associated with increased prevalence and severity of psoriasis [R].
- The 'A' allele of **rs657132**: associated with increased prevalence of ankylosing spondylitis [R].

TYPICAL ACTIVITY

Predisposed to typical HRH4 activity based on 8 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HRH4	rs615283	AA
IMPACT	rs524149	CC
HRH4	rs17797945	CC
HRH4	rs657132	GG
OSBPL1A	rs527790	CC
OSBPL1A	rs487202	GG
IMPACT	rs17203314	AA
HRH4	rs17187619	TT

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

CRP Gene

The main SNP in the CRP gene is **rs1205** (also known as “1846 G>A”). The **‘C’ allele** is linked to **higher CRP** levels, as well as [R]:

- Heart disease [R]
- High blood sugar [R]
- Pneumonia [R]
- Overall and cardiovascular mortality [R, R].

However, a study found **lower** non-cardiovascular mortality in people with this variant, particularly from cancer [R].

Another well-known CRP variant is **rs1130864**. The **‘A’ allele** is linked to [R, R, R, R, R]:

- Higher CRP levels
- Obesity
- Pneumonia
- PTSD

Other notable CRP variants include:

- rs1800947: the 'G' allele is associated with lower CRP levels and lower odds of pneumonia, while the evidence for heart disease is mixed [R, R, R, R, R].
- rs3091244: the ‘A’ allele is linked to higher CRP levels and pneumonia [R].
- rs3093059: the ‘G’ allele is linked to higher CRP levels and a reduced risk of myocardial infarction [R, R].

TYPICAL ACTIVITY

Predisposed to typical CRP gene activity based on 4 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CRP	rs1205	CC
CRP	rs1800947	CC
DUSP23	rs1130864	GG
CRP	rs3093059	AA
DUSP23	rs3091244	GG

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

PADI4 (Autoimmune)

The following *PADI4* variants have been associated with rheumatoid arthritis:

- 'C' of **rs2240335** [R]
- 'G' of **rs2301888** [R, R, R]
- 'C' of **rs2240336** [R]

Similar to the effects of IL-6, these variants may increase the production or activity of PADI4, resulting in the excessive citrullination of joint proteins and the production of autoantibodies [R].

TYPICAL ACTIVITY

Predisposed to typical PADI4 activity based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PADI4	rs2240335	CA
PADI4	rs2301888	GA
PADI4	rs2240336	CT

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

IL10 Gene (Autoimmunity)

IL10 gene variants are linked to different inflammatory and autoimmune conditions, especially [R, R, R, R, R, R, R]:

- IBD
- Type 1 diabetes
- Lupus

These variants likely **reduce** anti-inflammatory IL10 activity, enabling a stronger autoimmune response.

TYPICAL ACTIVITY

Predisposed to typical IL10 activity based on 666 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
IL19	rs3024498	TT
FCMR	rs3024493	AC
IL19	rs3024505	AG
IL19	rs3024496	GA
IL19	rs1800896	CT

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

SLC22A4 (Gut Inflammation)

The main SLC22A4 variant is **rs1050152**. Its **“T” allele** is linked to **higher odds of IBD**, especially Crohn’s disease [R].

An interesting aspect of SLC22A4's function involves transporting **ergothioneine**, a compound abundant in **mushrooms**. A study has revealed that this variant can increase ergothioneine transport by approximately 50%. While ergothioneine typically acts as an antioxidant, this increased absorption may **exacerbate IBD symptoms** in people with this variant [R].

This interaction represents a clear example of how genetic variations can affect dietary tolerances and potentially impact disease management strategies.

Evolutionary perspective: Early farming communities ate fewer mushrooms than hunter-gatherers. The SLC22A4 variant may have helped European farmers adapt by absorbing more nutrients from the limited mushrooms in their diet.

TYPICAL GENETICS

Likely typical SLC22A4 genetics based on the genetic variants we looked at

48%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **48% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SLC22A4	rs1050152	CT

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

IL1B (Inflammation/ Fatigue)

One of the most highly-studied SNPs in the *IL1B* gene is rs16944, also known as the '*C-511/T*' polymorphism. Its 'A' allele is associated with higher IL-1β levels. In line with this, the variant is associated with multiple inflammatory conditions such as [R, R, R, R]:

- Rheumatoid arthritis [R]
- Acute pancreatitis [R]
- Severe reactions to common infectious illnesses, such as the flu [R]

In addition, the variant has been linked to:

- Depressive and anxiety symptoms of PTSD [R, R]
- Chronic fatigue syndrome [R]
- Various types of cancer (including cervical, prostate, gastric, bone marrow, and breast cancers) [R, R, R, R, R, R, R]
- Mortality from systemic infections, such as septic shock [R]
- Reduced longevity (in males only) [R]

Other variants that may increase *IL1B* activity include:

- 'C' at rs1143633 [R]
- 'T' at rs1143643 [R]
- 'A' at rs1143627 [R]
- 'A' at rs1143634 [R, R]
- 'C' at rs1143623 [R]

These variants have been associated with

- Asthma [R]
- Allergies [R]
- Rheumatoid arthritis [R, R]
- Cavities and gum disease [R, R, R, R]
- Metabolic syndrome and higher waist circumference [R]
- Acute pancreatitis [R]
- Preeclampsia [R]

On the bright side, they may be linked to a lower risk of:

- Obesity and high body fat [R, R, R, R, R, R]
- PTSD and depressive symptoms in this condition [R, R]

LOWER ACTIVITY

Likely lower IL1B activity based on 6 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
IL1A	rs1143633	CC
IL1B	rs16944	GA
IL1A	rs1143627	AG
IL1A	rs1143623	CG
IL1B	rs1143643	CC
IL1B	rs1143634	GG

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

ABCB1 (Chronic Lyme)

According to a study, the more P-gp-reducing variants you have in your *ABCB1* gene, the more likely you are to suffer from chronic Lyme after an acute *Borrelia* infection [R].

The single SNP with the greatest impact is rs1128503, which has a significant effect on chronic Lyme risk on its own. Its 'A' variant, which decreases *ABCB1* activity, is associated with an increased risk of chronic Lyme [R].

Two other variants, at rs2235067 and rs4148740, also increase the risk of chronic Lyme. These variants are usually inherited together, so you will typically have either both or neither of them [R].

HIGHER ACTIVITY

Likely higher ABCB1 activity based on the genetic variants we looked at

33%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **33% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ABCB1	rs1128503	GG

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

HNMT (Histamine)

Different *HNMT* gene variants have been linked to histamine-related conditions, such as [R, R, R, R, R]:

- Allergies
- Asthma
- Eczema
- Migraines
- ADHD

In one interesting study, increased brain histamine worsened behavioral symptoms in children with ADHD. The children with the 'AA' genotype at rs1050891 were more likely to have worse symptoms when they were exposed to artificial food colorings and additives (sunset yellow, carmoisine, tartrazine, ponceau 4R, quinoline yellow, Allura red AC, and sodium benzoate) that raise brain histamine [R].

Another variant, the 'T' allele at rs11558538, is associated with decreased HNMT activity and increased risk of schizophrenia, ADHD, and migraine [R].

HIGHER ACTIVITY

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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HNMT	rs1050891	AG
HNMT	rs11558538	CC

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

JAK2 (Gut Inflammation)

The most widely-studied *JAK2* polymorphism is **rs10758669**. **Its minor variant "C" increases the production and activity of the JAK2 protein**, leading to an enhanced immune response and inflammation [R].

As a result, it's linked to higher odds of **IBD — both Crohn's disease and ulcerative colitis**. The link may be stronger for Crohn's disease, especially in people of European ancestry [R, R, R, R, R, R, R, R].

Another important JAK2 variant, **rs12340895-G,** has shown similar associations. It's often inherited with rs10758669-C, meaning that many people will either have none or both of them [R, R, R].

LOWER ACTIVITY

Likely lower JAK2 activity based on the genetic variants we looked at

60%

OF USERS SHARE THE SAME SCORE

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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
JAK2	rs10758669	CA
JAK2	rs12340895	CC

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

IL8 (Inflammation)

The main IL8 polymorphism is **rs4073**. Its 'A' allele increases *IL8* expression in white blood cells, which may contribute to inflammatory conditions. In line with this, this variant has been associated with an increased risk of [R, R, R]:

- Acne [R]
- ARDS [R, R]
- Acute liver injury [R]
- Stomach inflammation [R, R]
- Acute pancreatitis [R, R]
- Systemic lupus erythematosus [R]
- Chronic gum disease [R]
- Pelvic pain in people with endometriosis [R]
- IBD [R, R]
- Systemic inflammatory response to wasp stings [R]

LOWER ACTIVITY

Likely lower IL8 activity based on the genetic variants we looked at

33%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **33% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PF4V1	rs4073	TT

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

IL21 (Autoimmunity & Allergies)

Several variants in the IL21 gene have been studied for their potential links to allergies and autoimmunity. One variant of particular interest is **rs6822844**. Its minor **“T” allele** is linked to **lower odds** of [R]:

- Celiac disease
- Inflammatory bowel disease
- Asthma and allergic conditions

This variant is almost always inherited with **rs13119723-G**, meaning that most people will either have none or both of them [R].

Two more IL21 variants, **rs907715-T and rs2221903-T**, have shown similar associations. They are also linked to lower odds of thyroid problems and type 1 diabetes, respectively [R, R].

On the other hand, a variant **rs6848139-C** may be linked to **higher odds** of the above conditions and alopecia areata (autoimmune spot baldness) [R].

Finally, **rs7682241 and rs7682481** also showed links with alopecia areata and other autoimmune conditions, but the results are mixed [R, R].

Protective variants may reduce IL21 activity, weakening allergic and autoimmune responses.

LOWER ACTIVITY

Predisposed to lower IL21 activity based on 5 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
IL21	rs6822844	GT
IL21	rs907715	CT
KIAA1109	rs13119723	AG
IL2	rs6848139	AA
KIAA1109	rs2221903	TT

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

Recommendations Details

1 Probiotics

Take a probiotic supplement containing 10 billion or more live cultures once daily, preferably with a meal or as directed by the packaging or a healthcare provider.

TYPICAL STARTING DOSE

30 billion CFU

Helps with these Symptoms & Conditions:

Allergies | Anxiety

Helps with these Goals:

Immunity | Mood | Muscle Growth

Helps with these DNA Risks:

Crohn's Disease | Ulcerative Colitis | Eczema | IgE | NLRP3 (Gut Inflammation) | Pancreas Inflammation | SH2B3 (Autoimmunity)

2 Maintain a Healthy Weight

Engage in at least 150 minutes of moderate aerobic exercise or 75 minutes of vigorous exercise weekly, along with strength training exercises for all major muscle groups on 2 or more days a week. Follow a balanced diet, rich in vegetables, fruits, whole grains, and lean proteins while controlling calorie intake to prevent excessive weight gain. Regularly monitor body fat percentage through methods like bioelectrical impedance analysis (BIA) scales, skinfold measurements, or DEXA scans to ensure it remains below 25%.

TYPICAL STARTING DOSE

30 minutes

Helps with these Symptoms & Conditions:

Allergies | High Blood Pressure | Migraines

Helps with these Goals:

Immunity

Helps with these DNA Risks:

Psoriasis | Ulcerative Colitis | Eczema | Pancreas Inflammation

3 Omega-3 (Fish Oil)

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

TYPICAL STARTING DOSE

2000 mg

Helps with these Symptoms & Conditions:

Anxiety | High Blood Pressure | Migraines

Helps with these Goals:

Exercise Recovery | Immunity | Mood

Helps with these DNA Risks:

Crohn's Disease | Psoriasis | Ulcerative Colitis | Eczema | IL6R (Weight) | NF-kB (Inflammation) | Pancreas Inflammation | SH2B3 (Autoimmunity)

4 Maintain Optimal Vitamin D Levels

Check your vitamin D levels, they should ideally be in the 30-66 ng/mL range. If your levels are lower than that, take a vitamin D supplement, 1000-4000 IU daily, to reach an optimal range.

TYPICAL STARTING DOSE

1000 iu

Helps with these Symptoms & Conditions:

Allergies | Anxiety | High Blood Pressure | Migraines

Helps with these Goals:

Energy | Immunity | Mood | Muscle Growth

Helps with these DNA Risks:

Crohn's Disease | Psoriasis | Ulcerative Colitis | Eczema | HLA-DOB (Inflammation) | IL6R (Weight) | Pancreas Inflammation | SH2B3 (Autoimmunity)

5 Relaxation Techniques

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

TYPICAL STARTING DOSE

30 minutes

Helps with these Symptoms & Conditions:

Anxiety | High Blood Pressure | Migraines

Helps with these Goals:

Energy | Immunity | Mood

Helps with these DNA Risks:

Crohn's Disease | Psoriasis | Ulcerative Colitis | Eczema

6 Dietary Omega-3 Fatty Acids

Incorporate foods high in omega-3 fatty acids into your diet daily. This includes eating fish such as salmon, mackerel, and sardines at least twice a week. Alternatively, include a tablespoon of flaxseed oil or chia seeds in your daily diet.

Helps with these Symptoms & Conditions:

High Blood Pressure | Migraines

Helps with these Goals:

Immunity

Mood

Helps with these DNA Risks:

Crohn's Disease

Ulcerative Colitis

HLA (Inflammation)

IgE

IL-10

Nephritis

NF-kB (Inflammation)

Pancreas Inflammation

7 Curcumin

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

TYPICAL STARTING DOSE

500 mg

Helps with these Symptoms & Conditions:

Allergies

Anxiety

High Blood Pressure

Helps with these Goals:

Energy

Exercise Recovery

Immunity

Mood

Helps with these DNA Risks:

Crohn's Disease

Psoriasis

Ulcerative Colitis

ANKRD55 (Autoimmune)

HLA (Inflammation)

HLA-DOB (Inflammation)

NF-kB (Inflammation)

NLRP3 (Gut Inflammation)

Pancreas Inflammation

SH2B3 (Autoimmunity)

8 Aerobic Exercise (Cardio)

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

TYPICAL STARTING DOSE

1 hour

Helps with these Symptoms & Conditions:

Allergies | Anxiety | High Blood Pressure | Migraines

Helps with these Goals:

Energy | Immunity | Mood

Helps with these DNA Risks:

⚠ Crohn's Disease | ⚠ Psoriasis | ⚠ Ulcerative Colitis

9 Yoga

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

TYPICAL STARTING DOSE

30 minutes

Helps with these Symptoms & Conditions:

Anxiety | High Blood Pressure | Migraines

Helps with these Goals:

Energy | Exercise Recovery | Immunity | Mood | Muscle Growth

Helps with these DNA Risks:

⚠ Crohn's Disease | ⚠ Ulcerative Colitis | Pancreas Inflammation

10 Mediterranean Diet

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

Helps with these Symptoms & Conditions:

Allergies | High Blood Pressure

Helps with these Goals:

Energy | Mood

Helps with these DNA Risks:

Crohn's Disease | Psoriasis | Ulcerative Colitis | HLA (Inflammation)

11 Mindfulness

Set aside 5-10 minutes each day to practice mindfulness meditation. Find a quiet place, assume a comfortable seated position, close your eyes, focus on your breathing, and observe your thoughts and sensations without judgment.

TYPICAL STARTING DOSE

30 minutes

Helps with these Symptoms & Conditions:

Anxiety | Migraines

Helps with these Goals:

Energy | Mood

Helps with these DNA Risks:

Crohn's Disease | Ulcerative Colitis | Eczema

12 Acupuncture

Visit a licensed acupuncturist for a session, typically lasting between 30 to 60 minutes, once or twice a week. Depending on your specific condition, a course of treatment might range from a few weeks to several months.

TYPICAL STARTING DOSE

1 hour

Helps with these Symptoms & Conditions:

Allergies | High Blood Pressure | Migraines

Helps with these Goals:

Energy | Mood

Helps with these DNA Risks:

Eczema | IgE | Pancreas Inflammation

13 Guided Imagery

Find a quiet, comfortable place to sit or lie down where you can spend 20 to 30 minutes without interruptions. Close your eyes and take deep breaths to relax. Then, listen to a guided imagery audio recording or follow a script where you imagine a peaceful scene or scenario in detail. Do this practice daily, ideally at the same time each day, to reduce stress and improve well-being.

TYPICAL STARTING DOSE

30 minutes

Helps with these Symptoms & Conditions:

Anxiety | High Blood Pressure

Helps with these Goals:

Energy | Mood

Helps with these DNA Risks:

Crohn's Disease | Psoriasis | Ulcerative Colitis | IgE

14 Avoid Food Triggers

Identify foods that trigger negative reactions in your body, such as digestive issues, allergies, or headaches, and eliminate them from your diet entirely. This may require keeping a food diary for a few weeks to observe patterns and might involve cutting out common triggers like dairy, gluten, soy, or certain additives. Continuously monitor and adjust your diet to avoid these triggers as long as the symptoms persist.

Helps with these Symptoms & Conditions:

Migraines

Helps with these DNA Risks:

Crohn's Disease | Ulcerative Colitis | Eczema | SH2B3 (Autoimmunity)

15

Avoid Processed Foods

Eliminate foods such as packaged snacks, sugary drinks, ready-made meals, and anything with ingredients you can't pronounce from your daily diet. Focus on consuming whole foods like fruits, vegetables, lean meats, and whole grains instead.

Helps with these DNA Risks:

Crohn's Disease | Ulcerative Colitis | Eczema

Next Steps

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

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

Your Lifestyle Assessments

Ever heard of the term Nature vs. Nurture?

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

LIFESTYLE

You have a slightly reduced risk of eczema based on the answers you provided.

Factors impacting your risk:

What is your age?

41

Increasing Risk

Do you have a parent or sibling who has ever suffered from eczema?

Yes

Increasing Risk

Your BMI:

30.77

Increasing Risk

Have you ever been diagnosed with asthma?

No

Decreasing Risk

Do you smoke tobacco?

No, never

Decreasing Risk

Did your mother smoke while pregnant with you?

No

Decreasing Risk

Have you ever been diagnosed with allergic rhinitis (hay fever)?

No

Decreasing Risk

Did your mother have an infection while pregnant with you?

No

Decreasing Risk

Do you have a parent or sibling who has ever been diagnosed with allergic rhinitis (hay fever)?

No

Decreasing Risk

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

No

Decreasing Risk

What is your sex?

Male

Decreasing Risk

What is your height?

178 cm

No impact

What is your current weight?

97.5 kg

No impact

LIFESTYLE

You have a reduced risk of lupus based on the answers you provided.

Factors impacting your risk:

Have you ever been diagnosed with atopic dermatitis (eczema)?

Yes

Increasing Risk

What is your age?

41

Increasing Risk

Have you ever been diagnosed with asthma?

No

Decreasing Risk

Do you smoke tobacco?

No, never

Decreasing Risk

Have you ever been diagnosed with allergic rhinitis (hay fever)?

No

Decreasing Risk

Have you ever been diagnosed with Epstein-Barr virus (EBV) infection?

No

Decreasing Risk

What is your ethnicity?

Other

Decreasing Risk

What is your sex?

Male

Decreasing Risk