

Inflammation & Autoimmunity

Summary Report

REPORT CATEGORY —



INFLAMMATION & AUTOIMMUNITY

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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.

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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:

55 Genetic Results

Overview of Your Results

Inflammatory Gut Conditions

 TYPICAL LIKELIHOOD Appendicitis Typical likelihood of appendicitis	 TYPICAL LIKELIHOOD Stomach Inflammation Typical likelihood of gastritis	 LESS LIKELY Gut Inflammation Less likely to have IBD
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Other Inflammatory Conditions

 MORE LIKELY Eczema More likely to have eczema	 MORE LIKELY Pancreas Inflammation More likely to get pancreas inflammation	 TYPICAL LIKELIHOOD Chronic Lyme Typical likelihood of having “chronic Lyme”
 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 Tonsil Inflammation Less likely to have tonsillitis	 LESS LIKELY Urethra Inflammation Less likely to have non-specific urethritis	

 Autoimmune Skin Conditions



TYPICAL LIKELIHOOD

Alopecia Areata

Typical

 Inflammation Markers

 HIGHER LEVELS Inflammation (CRP) Predisposed to higher CRP levels	 HIGHER LEVELS TNF Predisposed to higher TNF levels	 HIGHER LEVELS White Blood Cells Predisposed to higher WBC count
 HIGHER LEVELS Eosinophils Predisposed to higher eosinophil levels	 HIGHER LEVELS Monocytes Predisposed to higher monocyte levels	 HIGHER LEVELS IL-17 (Th17) Predisposed to higher IL-17 levels
 HIGHER LEVELS IL-6 Predisposed to higher IL-6 levels	 TYPICAL LEVELS Basophils Predisposed to typical basophil levels	 HIGHER LEVELS Neutrophils Predisposed to higher neutrophil levels
 TYPICAL LEVELS Erythrocyte Sedimentation Rate Predisposed to typical ESR levels	 TYPICAL LEVELS IgE Predisposed to typical IgE levels	 TYPICAL LEVELS IL-10 Predisposed to typical IL-10 levels

 HLA Genes

 TYPICAL ACTIVITY HLA (Inflammation) Likely typical HLA activity	 TYPICAL ACTIVITY HLA-DOB (Inflammation) Likely typical 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

 LOWER ACTIVITY SH2B3 (Autoimmunity) Likely lower SH2B3 activity	 HIGHER ACTIVITY IL13 (Allergies, Lung Function) Predisposed to higher IL13 activity	 HIGHER ACTIVITY IL4 (Allergies, Autoimmunity) Likely higher IL4 activity
 HIGHER ACTIVITY ANKRD55 (Autoimmune) Predisposed to higher ANKRD55 activity	 LOWER ACTIVITY IL10 Gene (Autoimmunity) Predisposed to lower IL10 activity	 TYPICAL ACTIVITY NF-kB (Inflammation) Likely typical NF-kB activity
 TYPICAL ACTIVITY IL1B (Inflammation/ Fatigue) Likely typical IL1B activity	 TYPICAL ACTIVITY TLR4 (Inflammation) Likely typical TLR4 activity	 TYPICAL PTPN22 (Autoimmunity) Likely typical PTPN22 genetics

 TYPICAL ACTIVITY STAT3 (Th1/Th17) Likely typical STAT3 activity	 TYPICAL ACTIVITY NLRP3 (Gut Inflammation) Likely typical NLRP3 activity	 TYPICAL ACTIVITY TNF Gene (Inflammation) Likely typical TNF activity
 INTERMEDIATE ACTIVITY HNF4A (Gut Inflammation) Likely intermediate HNF4A activity	 TYPICAL IL21 (Autoimmunity & Allergies) Predisposed to typical IL21 activity	 TYPICAL ACTIVITY HRH4 (Allergies, Inflammation) Predisposed to typical HRH4 activity
 TYPICAL ACTIVITY CRP Gene Predisposed to typical CRP gene activity	 TYPICAL GENETICS IL6R (Weight) Likely typical IL6R genetics	 TYPICAL ACTIVITY PADI4 (Autoimmune) Predisposed to typical PADI4 activity
 TYPICAL ACTIVITY CTLA4 (Autoimmunity) Likely typical CTLA4 activity	 HIGHER ACTIVITY ABCB1 (Chronic Lyme) Likely higher ABCB1 activity	 HIGHER ACTIVITY HNMT (Histamine) Likely higher HNMT activity
 HIGHER ACTIVITY DAO (Histamine) Likely higher DAO activity	 LOWER ACTIVITY JAK2 (Gut Inflammation) Likely lower JAK2 activity	 LOWER ACTIVITY IL8 (Inflammation) Likely lower IL8 activity
 BETTER GENETICS SLC22A4 (Gut Inflammation) Likely better SLC22A4 genetics		

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.



TYPICAL LIKELIHOOD

Appendicitis

Typical likelihood of appendicitis



TYPICAL LIKELIHOOD

Stomach Inflammation

Typical likelihood of gastritis



LESS LIKELY

Gut Inflammation

Less likely to have IBD

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



TYPICAL LIKELIHOOD

Typical likelihood of appendicitis based on 809,853 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

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

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

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 [\[R\]](#).

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



Your top variants that most likely impact your genetic predisposition:

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



LESS LIKELY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HLA-DQA2	rs2395185	GG
JAK2	rs10758669	CA
FCGR2A	rs1801274	AG
ADO	rs10761659	GA
IL23R	rs11465804	TT
IL23R	rs11209026	GG
ATG16L1	rs2241880	GA
IL23R	rs10889677	AC
IRGM	rs1000113	CT
STAT3	rs744166	GA
IL23R	rs76418789	GG
IRGM	rs10065172	CT
HNF4A	rs6017342	AC
TNF	rs1799724	TC
AGT	rs5051	TT
CUL1	rs7807268	GG
IL23R	rs11805303	TC
ATG16L1	rs10210302	TC
PPM1M	rs5743836	GA
PTPN22	rs2476601	GG
NKX2-3	rs10883365	AG
IRGM	rs4958847	GA

- 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.

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
IL23R	rs1004819	AG
NLRP3	rs10754558	CG
IRGM	rs13361189	TC
IL23R	rs7517847	TT
DLD	rs2158836	AA
PDGFB	rs2413583	CC
STAT3	rs4796793	GC
IRF5	rs4728142	GA
ETS2	rs2836878	GG
ZNF365	rs7076156	GA
INAVA	rs7554511	CC
JAK2	rs1830610	TC
NRIP1	rs2823286	GG
IKZF3	rs12946510	TT
TNFSF15	rs3810936	CT
IRF8	rs10521318	CC
TNFSF18	rs9286879	GG
RORC	rs4845604	GG
ITLN1	rs2274910	CC
CRTC3	rs7495132	CC
IKZF3	rs2872507	AA
PHACTR2	rs12199775	AA
ATG16L1	rs3828309	GA
IRGM	rs11741861	AG
IRGM	rs11747270	AG
CDKAL1	rs6908425	CC
UBLCP1	rs10045431	CC
GLYCTK	rs352139	CT

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	 TYPICAL LIKELIHOOD Chronic Lyme Typical likelihood of having “chronic Lyme”
 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 Tonsil Inflammation Less likely to have tonsillitis	 LESS LIKELY Urethra Inflammation Less likely to have non-specific urethritis	

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



MORE LIKELY

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



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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
OVOL1	rs10791824	GG
STMN3	rs3848669	TT
TREH	rs10790275	CC
ADO	rs4372325	CC
PPP2R3C	rs2415269	GG
SATB1	rs4395418	CC
IL2RA	rs62626322	GT
ID2	rs891058	GG
TRAF3	rs12888955	AA
TRIB1	rs12334935	AG
PRR5L	rs10836538	GT
MDM1	rs2227491	TC
NCF4	rs4821564	TC
RUNX3	rs6672420	AT
D2HGDH	rs34290285	AG
ZBTB25	rs11625265	GA
FLG	rs61816761	GG
FLG	rs138726443	GG
LINGO4	rs12123821	CC
ARRDC1	rs117137535	GG
CCR7	rs112401631	TT
LRRC32	rs55646091	GG

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*)

GENE	SNP	GENOTYPE
SLC22A5	rs60153262	CC
LRRC32	rs7936434	GG
ACTL9	rs2918299	CC
HLA-C	rs2844594	GG
TBKBP1	rs72833417	AA
KIAA1109	rs62323874	GG
IL6R	rs61812598	GG
SLC25A46	rs3853750	TT
PRR5L	rs6484847	CC
KIAA1109	rs1904522	GG
GNGT2	rs28406364	CC
ARHGAP27	rs9895436	GG
TNFSF18	rs6691738	TT

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
MORC4	rs12688220	CT
CTRC	rs497078	CT
TBC1D8B	rs12689287	AA
SLC25A34	rs60816621	CG
NUP62CL	rs12688091	GA
JCAD	rs2995271	TC
RNF128	rs66491909	AG
RADX	rs5916761	GA
PWWP3B	rs67184230	CG
TWIST2	rs4663946	CT
SYNJ1	rs61750217	TC
TMEM222	rs150461998	CT
JAKMIP2	rs17107296	AA
JAKMIP2	rs150261364	CC
SPINK5	rs112861203	TT
STK32A	rs148849032	CC
JAKMIP2	rs146303903	AA
STK32A	rs142623619	AA
/	rs150176211	GG
ADRB2	rs17640347	GG
ABCG5	rs75331444	GG
PRSS1	rs10273639	TT

GENE	SNP	GENOTYPE
COLEC10	rs11988997	CC
PWWP3B	rs379742	AG
IGDCC4	rs75405617	TT
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
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.

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-DOB	rs3817964	AT
HLA-DQA2	rs6910071	GA
HLA-DQA2	rs3763305	AG
HLA-DQA2	rs17208888	GG
/	rs9271366	AG
TLR1	rs5743618	AA
ABCB1	rs1128503	GG
HLA-DQA2	rs660895	AA
DDAH2	rs9267658	CC
C6ORF47	rs4947332	CC

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

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



Your top variants that most likely impact your genetic predisposition:

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

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



Your top variants that most likely impact your genetic predisposition:

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

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



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.



LESS LIKELY

Less likely to have tonsillitis based on 351,312 genetic variants we looked at



Urethra Inflammation

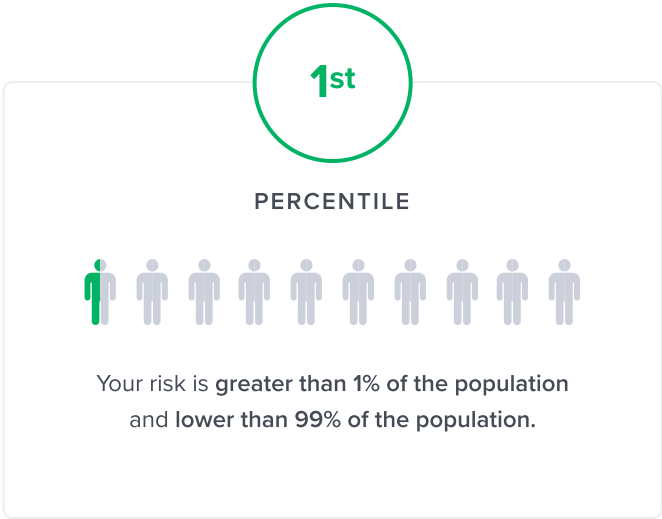
Diagnosis of non-specific urethritis involves examining the patient's symptoms and ruling out specific bacterial infections through standard STI tests, as the symptoms can closely mimic those caused by sexually transmitted diseases. Treatment usually consists of antibiotics to cover a wide range of potential 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





TYPICAL LIKELIHOOD

Alopecia Areata

Typical



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.

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 based on 42 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CCDC24	rs304303	GG
NTM	rs11600229	AA
RDH14	rs2345724	AG
CPVL	rs505532	TT
FNDC3B	rs6414541	CC
UNC5C	rs17023881	TC
HLA-DQA2	rs9275572	AG
DPYSL4	rs9419187	CC
DISP3	rs3099624	CC
HLA-DQA2	rs9268528	GA
DOCK5	rs2979742	CC
LURAP1L	rs7022183	TC
OPCML	rs11223339	GT
ST3GAL3	rs4660260	TC
B3GAT1	rs10791360	AA
CSMD1	rs718121	TC
ARHGAP42	rs1216476	AG
REELD1	rs9997120	CT
SNX2	rs2125856	TC
KCNU1	rs10503991	GA
RBBP8	rs9954649	GA
ST8SIA5	rs9952976	AG
DPH5	rs12403551	AG
TERF1	rs4738296	AA
CXXC4	rs7657799	TT
TFF3	rs9982439	TT
IQSEC3	rs2270797	CT
/	rs1431704	TT
RNF5	rs3115553	CC
UVSSA	rs4130791	GG
SRRM4	rs12228387	GG

GENE	SNP	GENOTYPE
ARHGAP42	rs11224294	TT
LDLRAD3	rs16928055	TT
LIPA	rs17479692	TT
BBS12	rs7664318	AA
BRD4	rs11666141	TT
CRIM1	rs2666138	GG
APOLD1	rs2110597	AA
PPARGC1A	rs16873952	GG
ACOX1	rs12430	AG
ITPR2	rs10506012	AA
NUDT6	rs304650	TC
NUP35	rs13409979	GG
APCDD1	rs7228576	CC
UBE2E2	rs1692617	GA
HSD17B3	rs10512241	CC
PGM5	rs7036795	TT
ZNF217	rs2766671	CC
DCBLD1	rs916305	CC

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

Inflammation (CRP)

Predisposed to higher CRP levels



HIGHER LEVELS

TNF

Predisposed to higher TNF levels



HIGHER LEVELS

White Blood Cells

Predisposed to higher WBC count



HIGHER LEVELS

Eosinophils

Predisposed to higher eosinophil levels



HIGHER LEVELS

Monocytes

Predisposed to higher monocyte levels



HIGHER LEVELS

IL-17 (Th17)

Predisposed to higher IL-17 levels



HIGHER LEVELS

IL-6

Predisposed to higher IL-6 levels



TYPICAL LEVELS

Basophils

Predisposed to typical basophil levels



HIGHER LEVELS

Neutrophils

Predisposed to higher neutrophil levels



TYPICAL LEVELS

Erythrocyte Sedimentation Rate

Predisposed to typical ESR levels



TYPICAL LEVELS

IgE

Predisposed to typical IgE levels



TYPICAL LEVELS

IL-10

Predisposed to typical IL-10 levels

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 [\[R\]](#).



HIGHER LEVELS

Predisposed to higher CRP levels based on 9,023 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
FUT2	rs601338	AA
RAD50	rs2069812	GG
CRP	rs1205	TC
IL1B	rs16944	AG
SLC22A5	rs1800925	CT
IL4R	rs1805011	AA
TOM1	rs2071746	TT
LGALS9	rs2248814	GG
KLC1	rs8702	GG
LRP6	rs2160525	GG
LRP6	rs2302685	TT
FUT2	rs492602	GG
FUT2	rs602662	AA
ADRB2	rs1042713	GA
IL19	rs1800872	GT
FADS2	rs174546	CT
APOE	rs429358	TC
IL6	rs1800795	CG
IL1RN	rs419598	CT
SRA1	rs2569191	TC
IFIH1	rs1990760	TC
ATG16L1	rs10210302	TC

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 [\[R\]](#).

GENE	SNP	GENOTYPE
ATG16L1	rs2241880	GA
CFH	rs6677604	GA
STEAP1B	rs2069840	CG
STEAP1B	rs1554606	TG
LEPR	rs4394621	AA
FOXO3	rs2802292	TG
TIMP4	rs3755724	TC
APCS	rs12745083	TC
UNC119B	rs7305618	CT
MICB	rs361525	GG
CRP	rs3093059	AA
IL19	rs3024505	GG
CTLA4	rs231775	AA
TNF	rs1800629	GG
IL6R	rs2228145	AA
HLA-DQA1	rs2187668	CC
IL19	rs1800896	TT
STAT4	rs10181656	CC
SLC20A1	rs1800587	GG
IL1A	rs17561	CC
IL1B	rs1143634	GG
CRP	rs10494326	CC
IL21	rs6822844	GG
TLR4	rs4986790	AA
SOD2	rs4880	GG
FUT2	rs281377	CC
IL6	rs2069849	CC
IL6	rs2069837	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 [\[R\]](#)



Predisposed to higher 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
/	rs72841564	CC
TNF	rs1799724	TC
ATP10B	rs149126334	GG
GBA2	rs10814274	CC
/	rs8121916	AC
ANO3	rs10834997	AG
TNF	rs1800629	GG
SPRY1	rs115669577	GG
ERICH1	rs138808635	CC
PSD3	rs79105320	GG
TNF	rs1799964	TT
ROBO1	rs149420276	AA
GPX3	rs111332265	AA
/	rs7256693	TT
GTF2E1	rs10511404	GG
RBM11	rs72490194	CC

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

- Sleep apnea [\[R\]](#)
- Parkinson’s [\[R\]](#)
- Heart failure [\[R\]](#)

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



HIGHER LEVELS

Predisposed to higher WBC count based on 34,015 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
/	rs549579958	CC
ACKR1	rs34599082	TC
PLAUR	rs4760	GG
LYST	rs1886654	CC
PTPN22	rs2476601	GG
TET2	rs199741557	AA
ORMDL3	rs4795415	TC
SH2B3	rs3184504	TC
CREB5	rs56388170	GT
PAQR6	rs568036	GA
PRTFDC1	rs11014291	CT
CXCR2	rs55799208	GG
NCLN	rs144284241	CC
/	rs201347186	GG
JAML	rs143034248	CC
TTC28	rs62237617	CC
IRF8	rs11642657	CC
IL17RA	rs140221307	TT
FLT3	rs76428106	TT
DPH5	rs77046277	CC
FERMT3	rs142815441	CC
TEX15	rs116898861	CC
EHD3	rs184409696	GG
RC3H1	rs77941945	GG
CDK6	rs445	CC
PLEK	rs141250842	AA
ATF7	rs117788567	CC
MICB	rs2524079	GG
CXCR4	rs77306654	AA
PREX1	rs149257976	CC

GENE	SNP	GENOTYPE
/	rs117255975	TT
GFI1	rs139795227	AA
TNFSF13B	rs374039502	TT
BCL2	rs17758695	CC
UBE2D3	rs56095122	AA
SESN3	rs75963851	AA
NAA38	rs74480102	GG
CXCL5	rs11733208	AA
ARAP2	rs28530750	GG
LRRC36	rs12928503	CC

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].



HIGHER LEVELS

Predisposed to higher eosinophil levels based on 851,736 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SH2B3	rs7310615	GG
ERMP1	rs992969	GG
ITGB8	rs34030463	AA
HLA-DQA2	rs28383314	CC
LGALS14	rs412884	CC
TRIB1	rs16900706	GA
NCF4	rs117582568	GG
/	rs536070968	CC
CCR3	rs138346219	AA
SHC1	rs8191981	GG
GFI1B	rs150813342	CC
ALOX15	rs71368508	CC
S1PR4	rs3746072	GG
IL17RA	rs140221307	TT
GATA2	rs6782812	GG
GATA1	rs146587548	GG
CCR7	rs112401631	TT
BCL2	rs17758695	CC
ADORA1	rs61025910	GG
RUNX1	rs2242886	CC
SLC22A5	rs2706334	CC
IL18R1	rs9807989	TT
NAA38	rs74480102	GG
RBM17	rs12722547	GG
BCL2L1	rs80054178	TT
KLF3	rs73232890	GG
/	rs76574427	GG
SPIDR	rs45577137	AA
BCL2L11	rs72836346	GG
ACKR2	rs2228467	TT

GENE	SNP	GENOTYPE
ERG	rs80109907	CC
IKZF2	rs2170572	AA
ASB2	rs11555542	TT
/	rs367705866	AA
CD300LG	rs72836561	CC
CDK6	rs445	CC
SHARPIN	rs34173062	GG
NFKB1	rs113473633	AA
HBS1L	rs9389268	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]



Predisposed to higher monocyte levels based on **842,556** genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GSDMC	rs35389394	TT
LYST	rs10927074	CC
LPAR1	rs115162333	GT
TET2	rs199741557	AA
MCL1	rs4970966	GT
CKM	rs73036517	GA
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
THEMIS2	rs41284294	TT
S1PR2	rs117064827	AA
PLAGL1	rs149110519	CC
SESN3	rs75963851	AA
CEBPG	rs12151289	GG
NFIX	rs1003393	CC
ST20	rs76648483	AA
CDK6	rs445	CC

GENE	SNP	GENOTYPE
RPN1	rs4045811	CC
HGSNAT	rs145097765	AA
NLRP12	rs10424405	AA
SPIDR	rs45577137	AA
CEBPB	rs17196752	CC
TRADD	rs12935169	CC
RASSF3	rs34927483	GG
B3GNTL1	rs9902102	GG
ETS2	rs58030288	CC

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** [\[R\]](#).



HIGHER LEVELS

Predisposed to higher 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
/	rs78612928	TT
/	rs12735700	GG
/	rs11110094	GG
TRIB3	rs62191444	TG
GDNF	rs80004049	GG
ANKRD30A	rs146752254	AA
ANKRD30A	rs1148267	AA
EPHA8	rs45498698	GG
KLF7	rs17282552	TT
ZNF184	rs143334272	CC
H3C12	rs145168562	GG
EXTL3	rs149738638	TT
TCF25	rs11640734	CC
SPTLC2	rs17106604	CC

GENE	SNP	GENOTYPE
C1ORF174	rs57920188	TT
PCDH8	rs9568764	GG

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 [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)



HIGHER LEVELS

Predisposed to higher 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	CG
BTBD7	rs182261775	GG
NOS1	rs146828618	CC
P2RY1	rs114373846	CC
FBLN5	rs113207090	CC
ATP9A	rs73273528	CC
SOX4	rs185628618	GG
TBKBP1	rs72831623	GG
TBKBP1	rs113600793	CC
SERPINE2	rs13412535	GG
/	rs11110094	GG
AKNA	rs10982213	GG
/	rs148614378	CC
MTAP	rs2004627	CC
CASS4	rs1884910	GC
LRAT	rs2404476	AG
IL6R	rs11265618	CT
ZNF703	rs183298717	AA
RAP2B	rs75101555	CC
CDYL2	rs76856708	TT
KMT2E	rs62486616	CC
CAMSAP1	rs117146485	TT

GENE	SNP	GENOTYPE
RASEF	rs188644522	AA
KCNK2	rs12079357	AA
ARHGAP28	rs8089344	CC
IL6R	rs4537545	CC
CDKN2B	rs1333040	TT
ATP2B2	rs4684700	TT
C17ORF64	rs3760332	TT
HLA-DQA2	rs660895	AA
IL6R	rs10796927	TT
/	rs7824087	AA
AQP10	rs1386821	TT
GPC6	rs696931	CC
IL1RN	rs6734238	AA

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 1,005,560 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PACC1	rs532279691	AA
/	rs370718489	GG
/	rs200688856	CC
DEFA3	rs10086568	AA
FCGR2B	rs2994672	TT
CEBPA	rs78744187	TC
LMNB1	rs2271352	CG
GF1B	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
CEBPG	rs12151289	GG
PLEK	rs34338164	AA

GENE	SNP	GENOTYPE
DEFA3	rs140526216	GG
CXCR2	rs114050631	CC
FCGR3A	rs141772609	CC
L3MBTL3	rs139719552	TT
GATA2	rs6782812	GG
LARP4B	rs11253511	CC
SPIDR	rs45577137	AA
HLA-C	rs1065406	GG
RUNX1	rs138595256	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 [\[R\]](#).



HIGHER LEVELS

Predisposed to higher neutrophil levels based on 41,236 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ACKR1	rs34599082	TC
PLAUR	rs4760	GG
LYST	rs1886654	CC
ORMDL3	rs3826331	TC
RPN1	rs11359909	AA
IFITM2	rs14408	CC
NBR1	rs199625942	CC
CREB5	rs56388170	GT
PAQR6	rs568036	GA
LEPR	rs12067936	CA
CXCR2	rs55799208	GG
NCLN	rs144284241	CC
JAML	rs143034248	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
MICB	rs2524079	GG
CHD7	rs7846314	AA
ADGRL4	rs41313381	CC
ARAP2	rs28530750	GG
NAA38	rs74480102	GG
TNFSF13B	rs374039502	TT

GENE	SNP	GENOTYPE
HNF4A	rs1800961	CC
EHD4	rs72726038	CC
PTEN	rs59085061	AA
CSF3R	rs3917932	CC
CDK2AP1	rs11057258	AA
GYPE	rs11735662	CC
BCL2	rs17758695	CC
LGI2	rs112783548	GG

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	GG

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

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.



TYPICAL LEVELS

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

Your top variants that most likely impact your genetic predisposition:

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



Predisposed to typical 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
IL10	rs1800871	GA
VEGFA	rs4349809	TT
IKBIP	rs1048911	TT
IL19	rs1800872	GT
SHROOM3	rs143141511	GG
BMP2	rs6085948	AA
ZNF516	rs9951418	CC
VLDLR	rs2375980	CC
PDIA5	rs1530455	TT
/	rs2086656	CC
/	rs10493718	CA
SERPINE2	rs282258	TC
PREP	rs10457128	GA
MACROD2	rs465757	GA
IL19	rs3024505	GG
IL19	rs1800896	TT
/	rs140244749	AA
NEBL	rs45559637	TT
LYRM7	rs148438889	GG
LYRM7	rs191791704	CC
RAPGEF6	rs147320771	TT
FNIP1	rs115710902	TT
MEIKIN	rs115393715	GG
H1-3	rs182422732	GG
FBRSL1	rs142425959	CC
MRPL37	rs11206302	TT
MIA3	rs3002131	GG
/	rs6937355	CC
VEGFA	rs3025021	CC

GENE	SNP	GENOTYPE
NFKBIE	rs6458375	CC
REEP3	rs7088799	TT
SORCS2	rs111913416	AA
HOMER1	rs4345303	TT
PCP4	rs9981861	TT

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.

 TYPICAL ACTIVITY HLA (Inflammation) Likely typical HLA activity	 TYPICAL ACTIVITY HLA-DOB (Inflammation) Likely typical 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



TYPICAL ACTIVITY

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

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

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 alter 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

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 [\[R\]](#), [\[R\]](#), [\[R\]](#).

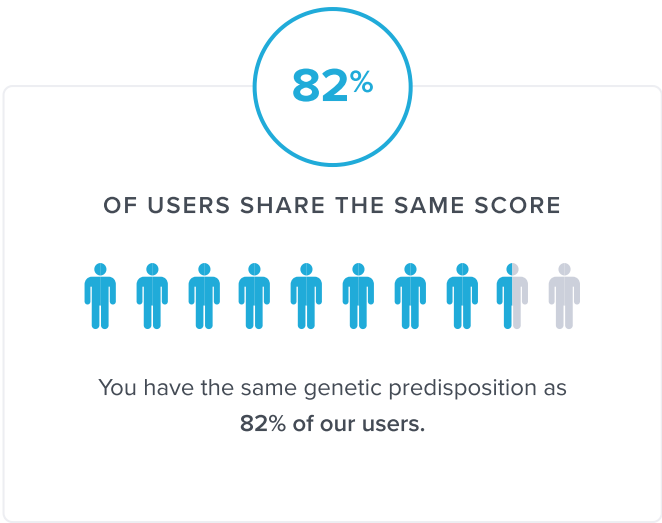
These variants are believed to increase HLA-DOB activity. In people with rheumatoid arthritis and other autoimmune conditions, HLA proteins may have increased activity or impaired structure. These changes cause them to target the body’s own components, such as joint cartilage, and trigger inflammation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

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 [\[R\]](#), [\[R\]](#).



TYPICAL ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

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

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



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.

- ‘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.

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]



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	rs2395185	GG
HLA-DQA2	rs9275596	CT
HLA-DQA2	rs9275572	AG
HLA-DQA1	rs6906021	TC
HLA-DQA2	rs9275524	TC
HLA-DQA1	rs2187668	CC
HLA-DQA2	rs9271588	TT

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

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 alter 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]

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



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	GG
/	rs9271366	AG
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.

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

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 [\[R\]](#).

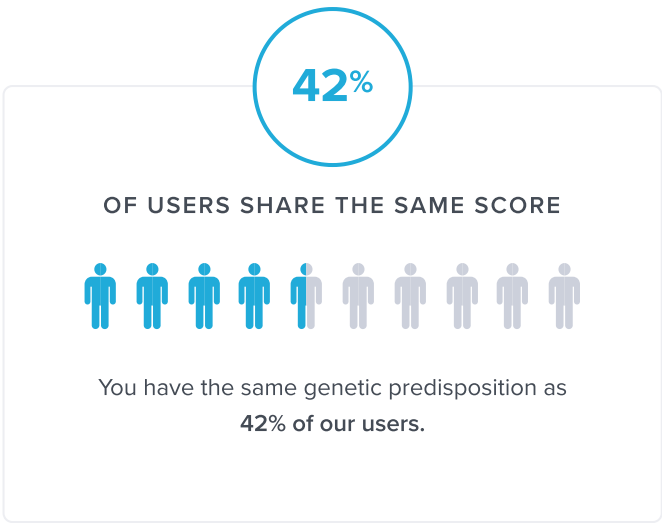
Another allele, ‘C’ at rs9275524, was associated with approximately twice the odds of alopecia areata in a study of 10,796 participants [\[R\]](#).

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



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.

 LOWER ACTIVITY SH2B3 (Autoimmunity) Likely lower SH2B3 activity	 HIGHER ACTIVITY IL13 (Allergies, Lung Function) Predisposed to higher IL13 activity	 HIGHER ACTIVITY IL4 (Allergies, Autoimmunity) Likely higher IL4 activity
 HIGHER ACTIVITY ANKRD55 (Autoimmune) Predisposed to higher ANKRD55 activity	 LOWER ACTIVITY IL10 Gene (Autoimmunity) Predisposed to lower IL10 activity	 TYPICAL ACTIVITY NF-kB (Inflammation) Likely typical NF-kB activity
 TYPICAL ACTIVITY IL1B (Inflammation/ Fatigue) Likely typical IL1B activity	 TYPICAL ACTIVITY TLR4 (Inflammation) Likely typical TLR4 activity	 TYPICAL PTPN22 (Autoimmunity) Likely typical PTPN22 genetics
 TYPICAL ACTIVITY STAT3 (Th1/Th17) Likely typical STAT3 activity	 TYPICAL ACTIVITY NLRP3 (Gut Inflammation) Likely typical NLRP3 activity	 TYPICAL ACTIVITY TNF Gene (Inflammation) Likely typical TNF activity



INTERMEDIATE ACTIVITY

HNF4A (Gut Inflammation)

Likely intermediate HNF4A activity



TYPICAL

IL21 (Autoimmunity & Allergies)

Predisposed to typical IL21 activity



TYPICAL ACTIVITY

HRH4 (Allergies, Inflammation)

Predisposed to typical HRH4 activity



TYPICAL ACTIVITY

CRP Gene

Predisposed to typical CRP gene activity



TYPICAL GENETICS

IL6R (Weight)

Likely typical IL6R genetics



TYPICAL ACTIVITY

PADI4 (Autoimmune)

Predisposed to typical PADI4 activity



TYPICAL ACTIVITY

CTLA4 (Autoimmunity)

Likely typical CTLA4 activity



HIGHER ACTIVITY

ABCB1 (Chronic Lyme)

Likely higher ABCB1 activity



HIGHER ACTIVITY

HNMT (Histamine)

Likely higher HNMT activity



HIGHER ACTIVITY

DAO (Histamine)

Likely higher DAO activity



LOWER ACTIVITY

JAK2 (Gut Inflammation)

Likely lower JAK2 activity



LOWER ACTIVITY

IL8 (Inflammation)

Likely lower IL8 activity



BETTER GENETICS

SLC22A4 (Gut Inflammation)

Likely better SLC22A4 genetics

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](#) [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

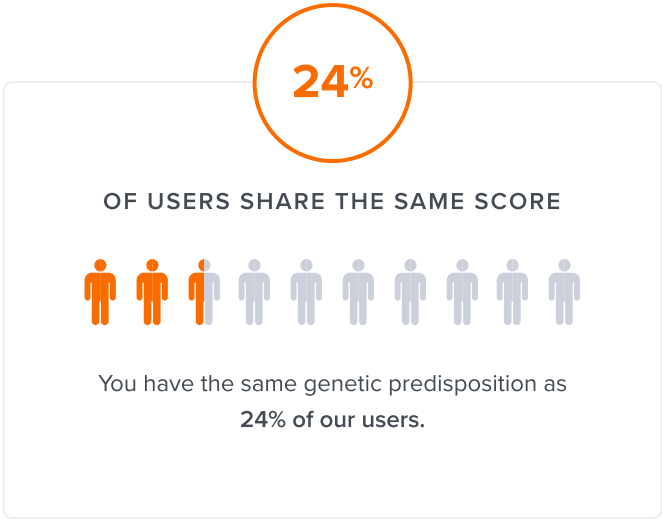
Finally, the ‘A’ allele of **rs597808** has been associated with higher rates of lupus [\[R\]](#).

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



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.

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



HIGHER ACTIVITY

Predisposed to higher 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	AA
SLC22A5	rs1295685	AA
SLC22A5	rs848	AA
SLC22A5	rs1800925	CT
SLC22A5	rs1295686	TC
SLC22A5	rs847	TT
SLC22A5	rs2066960	CC

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

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].

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]

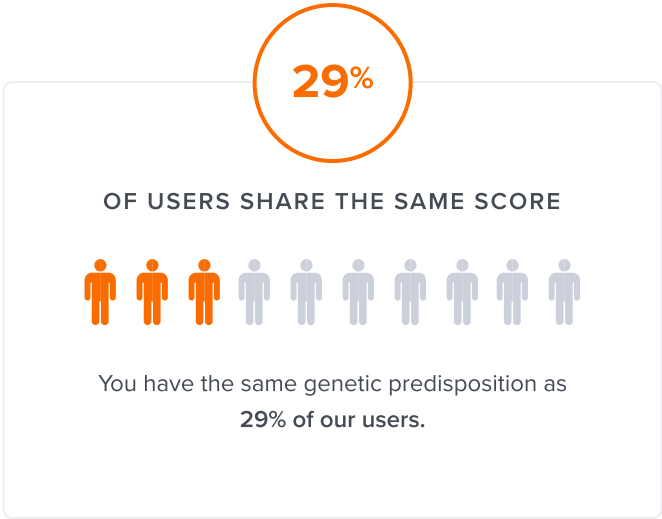
The ‘T’ allele may also increase the risk of infections such as tuberculosis and hepatitis B and C. However, it has also been associated with a decreased risk of severe symptoms in people with this and other infectious diseases such as HIV and malaria and with a better response to the hepatitis B vaccine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

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



HIGHER ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SLC22A5	rs2243250	TC

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].



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.

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.



LOWER ACTIVITY

Predisposed to lower 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	CC
IL19	rs3024505	GG
IL19	rs3024496	AA
IL19	rs1800896	TT

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

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.



TYPICAL ACTIVITY

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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
DDX39B	rs2255798	GG
HLA-C	rs2230365	CT
NFKBIE	rs2233434	AA
TCTE1	rs2233424	CC

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](#)



TYPICAL ACTIVITY

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

Your top variants that most likely impact your genetic predisposition:

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

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]

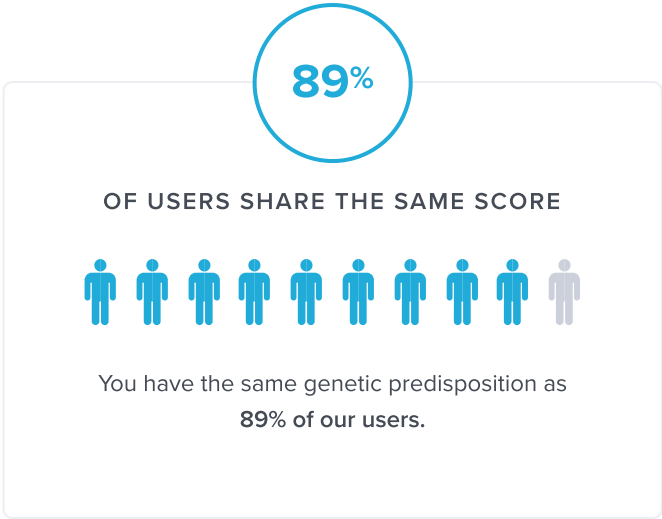
However, this has also been associated with a reduced acne severity [R].

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



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

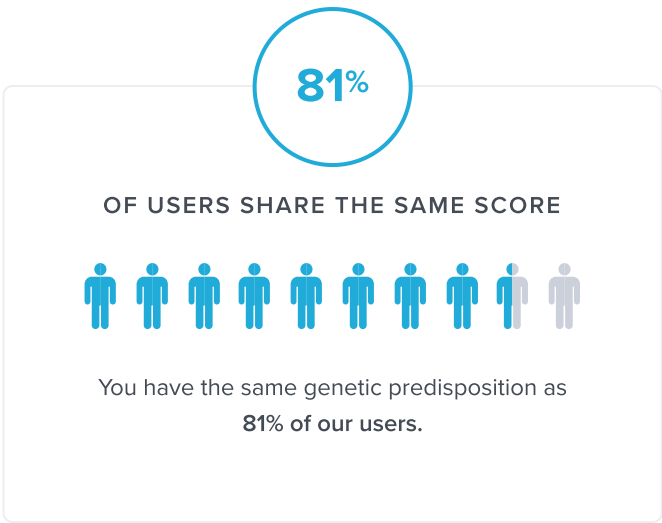
However, this allele showed a protective effect (around 16% lower odds) in the case of Crohn’s disease [R].

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



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.

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

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].



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	rs744166	GA
STAT3	rs3816769	CT
STAT3	rs4796793	GC
STAT3	rs2293152	CC
STAT3	rs1053005	TT

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

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 [\[R\]](#), [\[R\]](#).

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].



TYPICAL ACTIVITY

Likely typical 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	CT
NLRP3	rs3738447	AG
NLRP3	rs4925648	CC

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	TC
TNF	rs1800629	GG
TNF	rs1799964	TT

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** [\[R\]](#).

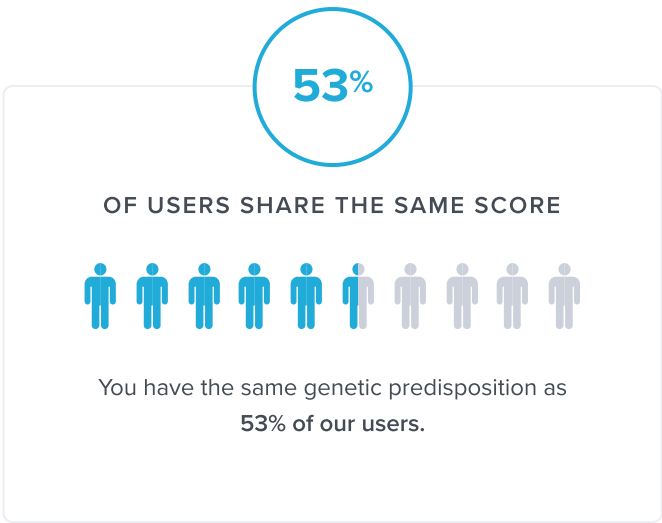
Likewise, two more studies of over 2,000 Dutch and 1,900 Korean patients also found that those carrying a ‘C’ allele were significantly more likely to have UC [\[R\]](#), [\[R\]](#).

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 [\[R\]](#), [\[R\]](#), [\[R\]](#).



INTERMEDIATE ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HNF4A	rs6017342	AC

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.



TYPICAL

Predisposed to typical 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	GG
KIAA1109	rs13119723	AA
IL21	rs907715	CT
IL2	rs6848139	AA
KIAA1109	rs2221903	TT

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	rs17187619	CT
OSBPL1A	rs527790	CT
OSBPL1A	rs487202	GC
IMPACT	rs524149	CC
HRH4	rs17797945	CT
HRH4	rs657132	GG
HRH4	rs615283	GG
IMPACT	rs17203314	AA

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	rs1800947	CC
CRP	rs1205	TC
DUSP23	rs1130864	GG
CRP	rs3093059	AA
DUSP23	rs3091244	GG

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]



TYPICAL GENETICS

Likely typical IL6R genetics based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
IL6R	rs2229238	TC
IL6R	rs4845623	AA
IL6R	rs2228145	AA

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.

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 [\[R\]](#), [\[R\]](#).



TYPICAL ACTIVITY

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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CTLA4	rs231775	AA
CTLA4	rs3087243	AA
CTLA4	rs231779	CC
CTLA4	rs11571302	TT

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].

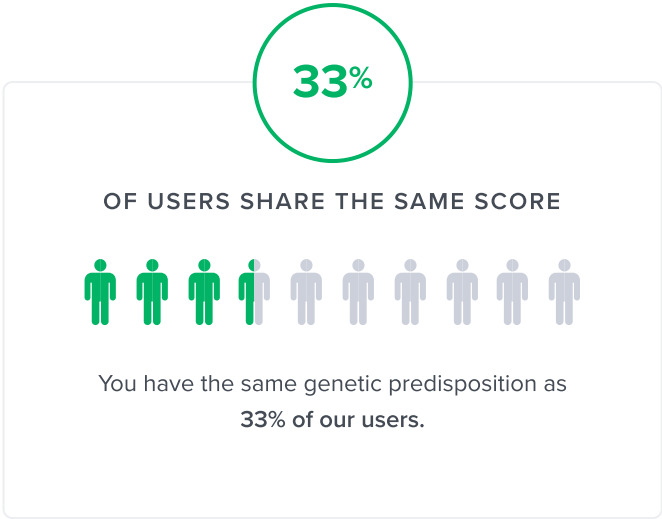
The single SNP with the greatest impact is rs1128503, which has a significant effect on chronic Lyme risk on its own. Its ‘A’ variant, which decreases *ABCB1* activity, is associated with an increased risk of chronic Lyme [R].

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



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	GA
HNMT	rs11558538	CC

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 [\[R\]](#).



HIGHER ACTIVITY

Likely higher 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	CC
AOC1	rs1049742	CC
AOC1	rs10156191	CC
AOC1	rs2268999	AA
AOC1	rs2071514	GG
AOC1	rs2052129	GG
AOC1	rs35070995	AA

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].

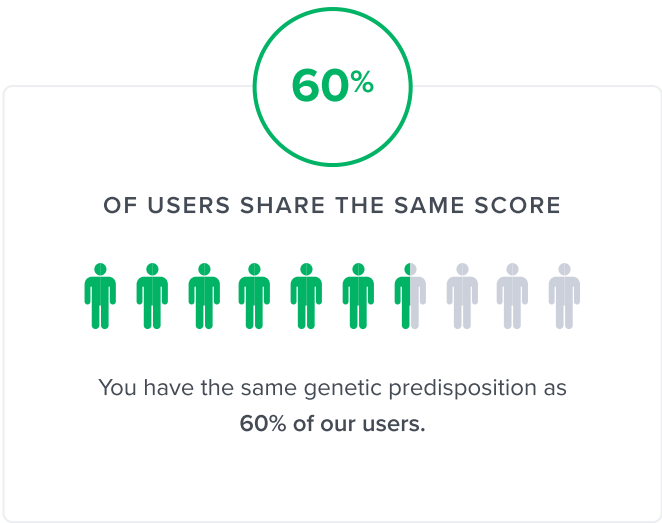
As a result, it’s linked to higher odds of **IBD — both Crohn’s disease and ulcerative colitis**. The link may be stronger for Crohn’s disease, especially in people of European ancestry [R, R, R, R, R, R, R, R, R].

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



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)

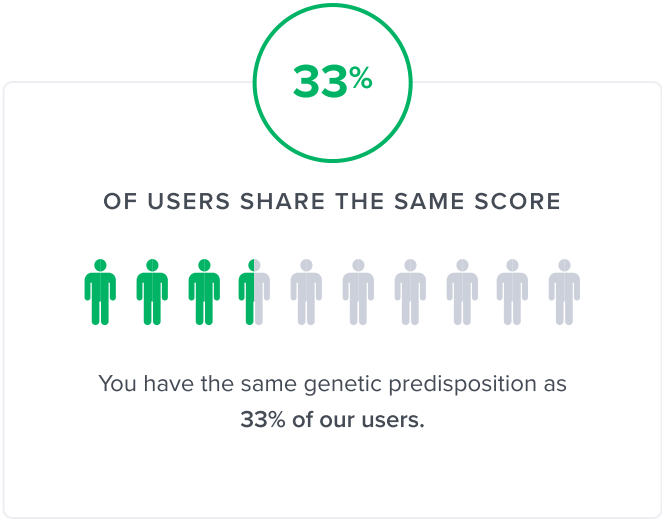
The main IL8 polymorphism is **rs4073**. Its ‘A’ allele increases *IL8* expression in white blood cells, which may contribute to inflammatory conditions. In line with this, this variant has been associated with an increased risk of [\[R\]](#), [\[R\]](#), [\[R\]](#):

- 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



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.

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].

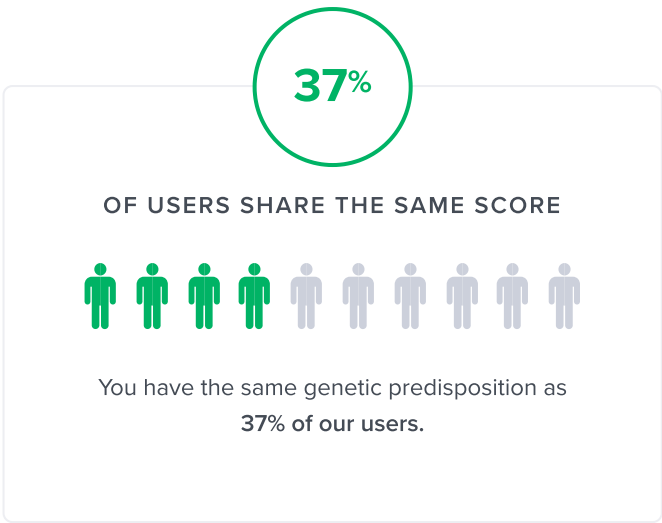
This interaction represents a clear example of how genetic variations can affect dietary tolerances and potentially impact disease management strategies.

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.



BETTER GENETICS

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SLC22A4	rs1050152	CC

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