SelfDecode™

TNFSF15 (Gut Inflamamtion)

Gene Report

REPORT CATEGORIES

GUT HEALTH

INFLAMMATION & AUTOIMMUNITY

Sample Client

Report date: 15 January 2026

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DISCLAIMER

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

Personal information

NAME

Sample Client

SEX AT BIRTH

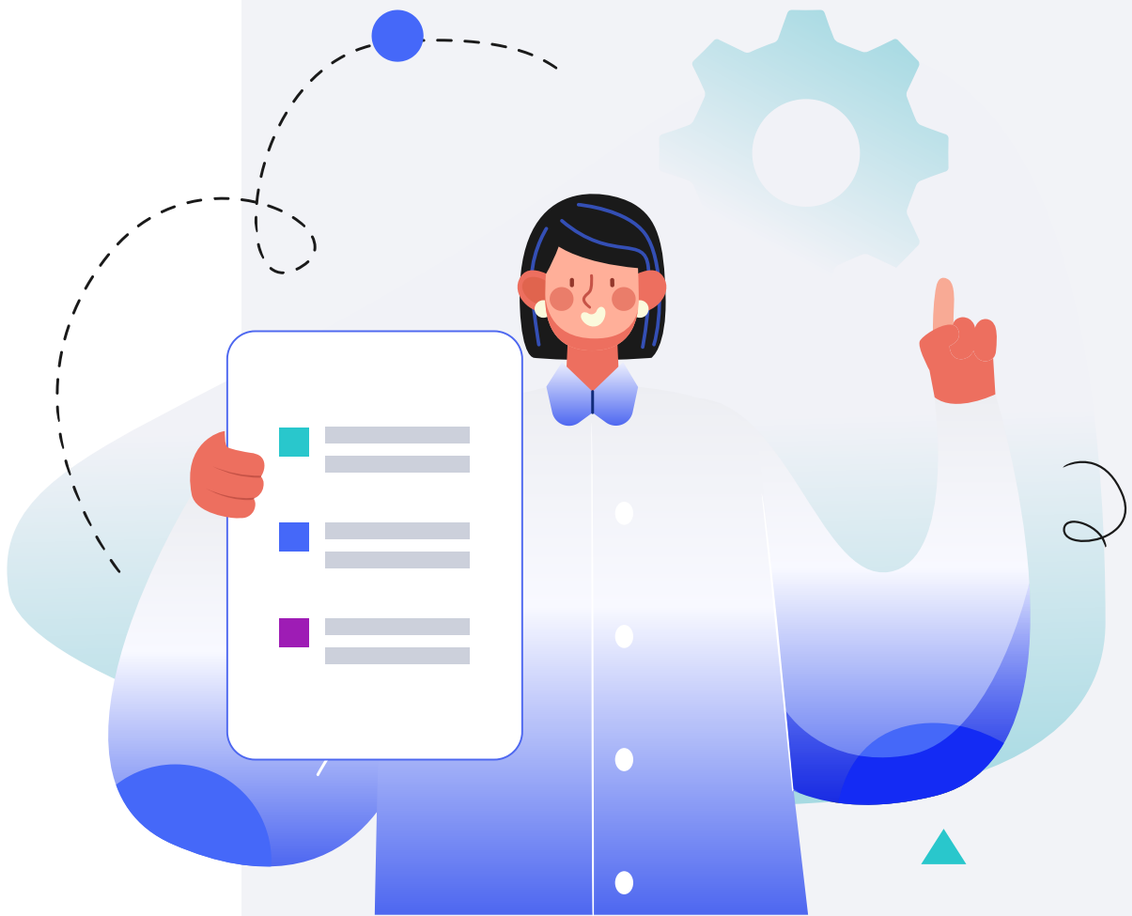
Male

HEIGHT

5ft 5" 165cm

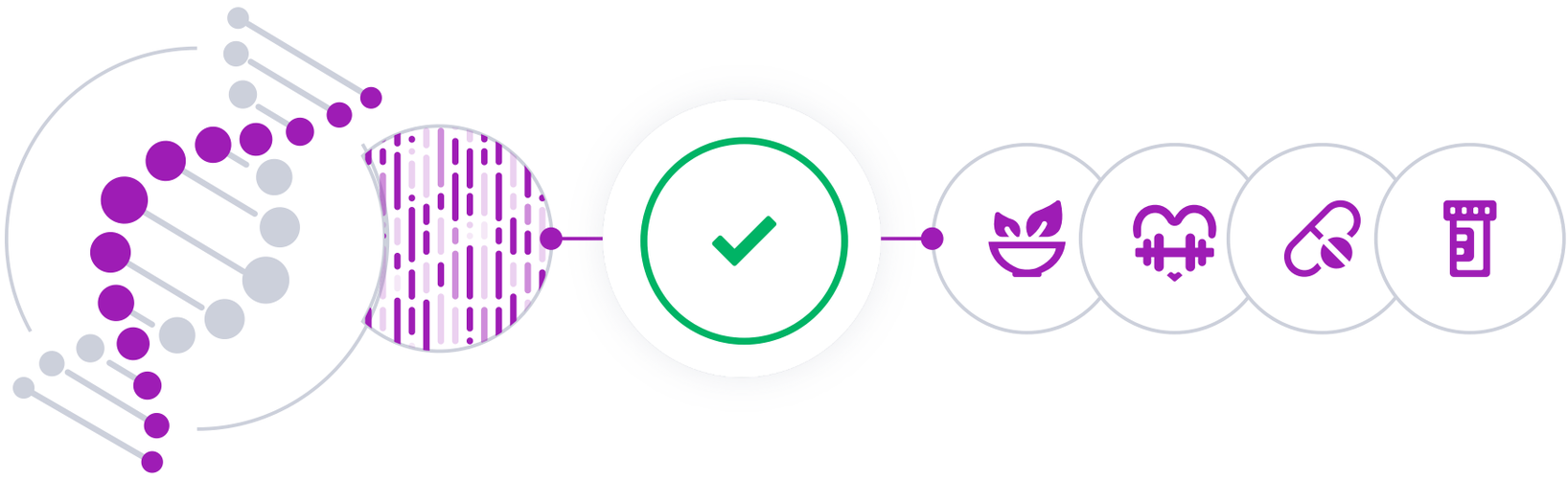
WEIGHT

137lb 62kg



How this works

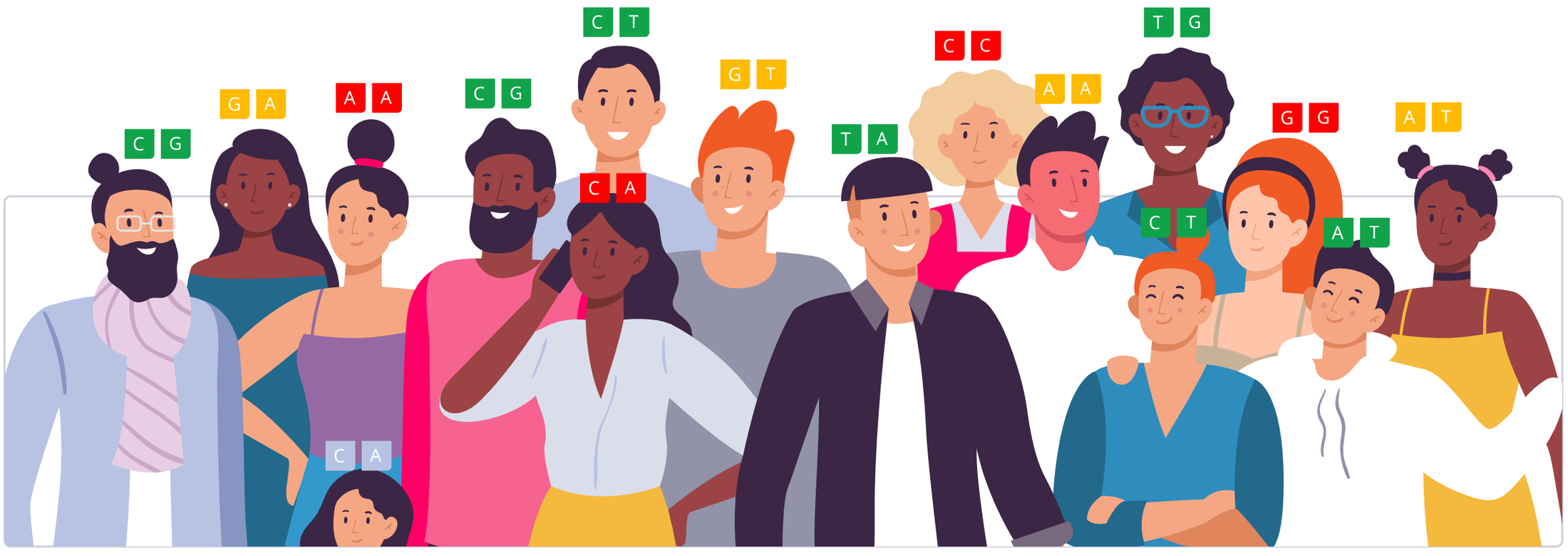
Our Wellness Reports analyze how your DNA influences your health.
We then use this analysis to give you personalized risk estimates and recommendations.



Similarly, our Trait Reports look at how your DNA influences your traits.

Your DNA is like an instruction manual — it contains a lot of information.
You can think of it as a blueprint for your body.

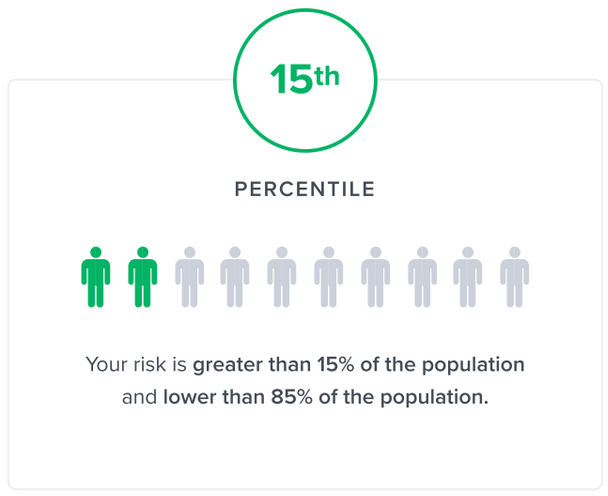
Genetic variants are parts of DNA that differ from person to person. Some can make you more vulnerable to certain health issues, while others may influence traits such as eye color.



We use artificial intelligence and machine learning to analyze all this information. We then summarize your results as a risk score or display it on a gauge.

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

In total, we analyze up to 83 million genetic variants.



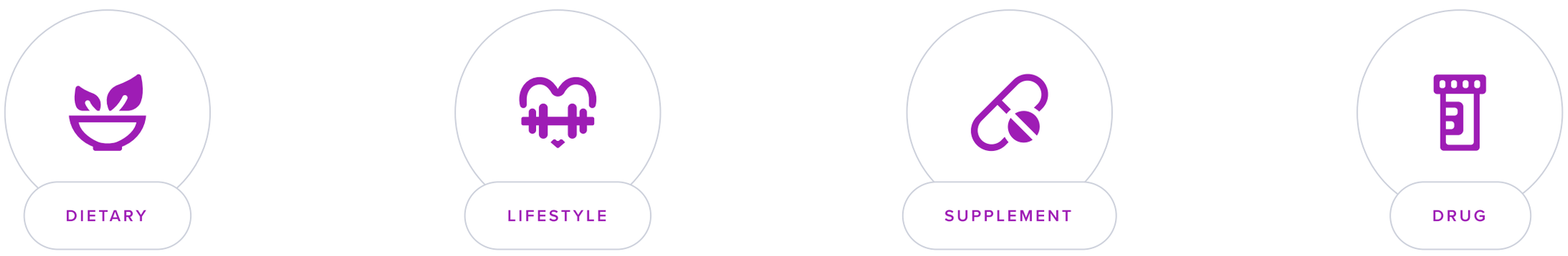
Genotype color info: AA You don't have any risk alleles | AA You have 1 risk allele | AA You have 2 risk alleles

Your risk is also displayed as a percentile. This will tell you how your risks compare to our sample population. The lower your percentile number, the lower your risk. The "50th percentile" would be an average risk.

Similarly, the gauge tells you your relative risk score compared to our sample population, or it indicates a specific trait or haplotype you are more likely to have based on your genetic variants.

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

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



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

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

Impact

Impact scores range from 1-5. These scores reflect how much of an effect each recommendation can have. An impact score of 5 predicts the biggest effect.

When a recommendation affects something we can measure, we use those measurements to assign the impact score. For example, a recommendation that decreases cholesterol by 20% will have a higher impact score than one that decreases it by 5%.

Some recommendations affect things that we cannot directly measure, like stress or mood. For these, the impact score is based on how well they work relative to other recommendations and standard treatments. The best ones get the highest scores.

If there is a lot of research that shows a recommendation works especially well for your genotype, the impact score gets increased.

Recommendation Evidence

5 / 5

Recommendations that are considered effective and generally recommended by experts and medical bodies.

4 / 5

Recommendations that are considered likely effective and that have multiple independent meta-analyses and a great many studies supporting them.

3 / 5

Recommendations that are considered possibly effective and have many studies supporting them

2 / 5

Recommendations that have insufficient evidence, with two or several clinical trials supporting them, or many studies but with ambiguous results.

1 / 5

Recommendations that have insufficient evidence, with a single clinical trial, or with many studies most of which didn't find support for the recommendation.

0 / 5

No evidence in humans.

Genotype-specific Evidence

High-quality

Direct evidence that a recommendation helps more in people with your gene variant (many clinical trials, afew large clinical trials, or a meta-analysis).

Medium-quality

Direct evidence that a recommendation helps more in people with your gene variant (a few clinical trials or one large clinical trial).

Low-quality

Direct evidence that a recommendation helps more in people with your gene variant (a single clinical trial or more trials with inconsistent results).

Indirect

A recommendation may help more in people with your gene variant because it targets a specific gene or protein affected by your variant (e.g., MTHFR, dopamine).

In theory

A recommendation may help more in people with your gene variant because it targets a specific mechanism affected by your variant (e.g., inflammation, oxidative stress).

Some things to keep in mind:

- Genetics doesn’t play a considerable role in a condition or a trait.
- There is not enough research available to estimate a genetic predisposition.
- There are technical limitations to estimating or presenting a genetic predisposition.
- The topic is sensitive, and a genetic predisposition should only be estimated and presented by a healthcare professional.

Introduction

The *TNFSF15* gene encodes a cytokine from the tumor necrosis factor (TNF) family called '*TNF superfamily member 15* (TNFSF15)'. The protein is also commonly known as TL1A ('*Tumor necrosis factor-like cytokine 1A*') and VEGI ('*Vascular endothelial growth inhibitor*') [R, R].

TNFSF15 is expressed at very low levels by tissue-lining cells and inactive immune cells. Upon recognizing inflammatory cytokines, antibodies, and foreign microbes, immune cells such as T cells, macrophages, and dendritic cells increase their production of this protein [R, R, R, R].

TNFSF15 exerts its function by binding to DR3 ('*Death receptor 3*'), a protein mainly produced by activated T cells, B cells, and natural killer cells [R, R, R, R].

By enhancing the effects of the cytokines that stimulate these processes, the TNFSF15/DR3 interaction promotes the proliferation, maturation, and function of the following immune cells:

- Th1 [R, R]
- Th2 [R, R]
- Th9 [R, R]
- Th17 [R, R]
- Tregs [R, R]
- NK cells [R, R]

As a result, TNFSF15 helps fight off infections by amplifying the immune response. Unfortunately, its excessive or uncontrolled activation is associated with inflammatory and autoimmune diseases such as IBD, rheumatoid arthritis, ankylosing spondylitis, and primary biliary cirrhosis [R, R].

TNFSF15 Genetics

Four *TNFSF15* polymorphisms have been most widely studied:

- **rs3810936**: its major allele ‘C’ is associated with higher TNFSF15 levels and activity [R, R].
- **rs6478108**: its major variant ‘T’ is associated with increased TNFSF15 levels and activity in macrophages, colon cancer cells, and gut lining of people with IBD [R, R, R].
- **rs6478109**: its minor variant ‘A’ has been associated with higher TNFSF15 levels in immune cells, but ‘G’ increased them in the gut lining of people with IBD and colon cancer cells [R, R, R, R].
- **rs7848647**: its minor variant ‘T’ was associated with lower blood TNFSF15 levels in people with rheumatoid arthritis, while ‘C’ increased the levels of this protein in the gut lining of people with IBD [R, R].

The minor variants at these polymorphisms protected from IBD (especially Crohn’s disease) in several studies [R, R, R, R, R, R, R, R, R, R, R, R].

In contrast, the major ‘G’ allele of rs7848647 was associated with an earlier onset of Crohn’s diagnosis (under 16 years old) [R].

The minor allele ‘A’ of **rs4263839**, associated with reduced protein levels in the blood and gut lining, protected against Crohn’s and ulcerative colitis in 3 studies. However, this variant was also associated with an increased progression of the disease in a study [R, R, R, R].

The minor ‘C’ allele at **rs7869487** was protective against Crohn’s disease in 3 studies and against both Crohn’s and ulcerative colitis in one [R, R, R, R].

Inheriting several of the major variants together has been associated with higher levels of the TNFSF15 protein and more severe IBD symptoms in different populations [R, R, R, R].

In addition, two cases of minor *TNFSF15* variants conferring increased susceptibility to Crohn’s disease have been

TYPICAL ACTIVITY

Predisposed to a typical TNFSF15 activity based on 8 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TNFSF15	rs4263839	GA
TNFSF15	rs7869487	TT
TNFSF8	rs6478109	GA
TNFSF15	rs6478108	TC
TNFSF15	rs3810936	CT
TNFSF8	rs7848647	CT
TNFSF8	rs6478106	CC
TNFSF8	rs4979462	CC

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

identified:

- The ‘T’ variant of **rs4979462**, which increases *TNFSF15* expression [R, R, R, R]
- The ‘T’ variant of **rs6478106** [R, R]