UGENOME

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Metabolic Health

Summary Report

REPORT CATEGORIES

- THYROID
- WEIGHT & BODY FAT
- BLOOD SUGAR CONTROL

Sample Client

Report date: 30 April 2026

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DISCLAIMER

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

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

Personal information

NAME

Sample Client

SEX AT BIRTH

Male

HEIGHT

5ft 10" 178cm

WEIGHT

215lb 97.5kg

REPORT PROVIDED BY

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Summary

Is your metabolism optimal? Maybe, but probably not. A recent study indicated that only about **1 in 8 people** in the U.S. have optimal metabolic health [R].

Why so bad? Well, your metabolic health is about much more than weight. **It is a system designed to process nutrients for energy, maintain temperature, detox your body, and so much more!** If any part of that system gets out of balance, it can mess up the whole thing. This can lead to problems like diabetes, obesity, and underactive thyroid.

Knowing your genetic predispositions can help you determine where the risk factors for your metabolism may lie, and what actionable steps you can take to optimize metabolic health. This report covers a number of related topics, including:

- Weight control
- Blood sugar control
- Thyroid health

This summary report contains:

60 Genetic Results

15 Recommendations

3 Lifestyle Assessments

Overview of Your Results

Weight Control

HIGHER LEVELS

Visceral Fat

Predisposed to more visceral fat

LOWER

Weight Loss from Calorie Restriction

Predisposed to lower weight loss from calorie restriction

TYPICAL LIKELIHOOD

Weight Regain

Typical likelihood of weight regain

LOWER

Metabolic Rate

Predisposed to lower metabolic rate

LESS LIKELY

Overweight

Less likely to be overweight or obese

Blood Sugar Control

LOWER LEVELS

GLP-1

Predisposed to lower GLP-1 levels

TYPICAL LEVELS

Fasting Glucose

Predisposed to typical fasting glucose levels

TYPICAL

Insulin Resistance

Predisposed to typical insulin resistance

TYPICAL LEVELS

HbA1c

Predisposed to typical HbA1c levels

TYPICAL LIKELIHOOD

Type 2 Diabetes

Typical likelihood of type 2 diabetes

TYPICAL LEVELS

Insulin

Predisposed to typical insulin levels

LESS LIKELY

Low Blood Sugar

Less likely to have low blood sugar

LESS LIKELY

Metabolic Syndrome

Less likely to have metabolic syndrome

LOWER LEVELS

Postprandial Glucose

Likely lower postprandial glucose levels

Thyroid Health

TYPICAL LIKELIHOOD

Underactive Thyroid

Typical likelihood of hypothyroidism

TYPICAL LIKELIHOOD

Overactive Thyroid

Typical likelihood of hyperthyroidism

TYPICAL LIKELIHOOD

Hashimoto's Disease

Typical likelihood of Hashimoto's disease

TYPICAL LEVELS

TSH

Predisposed to typical TSH levels

TYPICAL LEVELS

T3 (Triiodothyronine)

Predisposed to typical T3 levels

TYPICAL LEVELS

T4 (Thyroxine)

Predisposed to typical T4 levels

TYPICAL LEVELS

Free T4

Predisposed to typical free T4 levels

TYPICAL LEVELS

Reverse T3 (rT3)

Predisposed to typical rT3 levels

TYPICAL LEVELS

Free T3 (fT3)

Predisposed to typical free T3 levels

LESS LIKELY

Graves' Disease

Less likely to have Graves' disease

Miscellaneous

MORE LIKELY

Heavy Sweating

More likely to have hyperhidrosis

TYPICAL LIKELIHOOD

Gout

Typical likelihood of gout

TYPICAL LEVELS

Uric Acid

Predisposed to typical uric acid levels

TYPICAL LEVELS

Ketone Bodies

Predisposed to typical levels of ketone bodies

TYPICAL LEVELS

Lactate

Predisposed to typical lactate levels

TYPICAL LEVELS

Creatine

Likely typical creatine levels

Metabolism Genes

LOWER ACTIVITY

SLC30A8 (Zinc & Blood Sugar)

Predisposed to lower SLC30A8 activity

LOWER ACTIVITY

UCP1 (Weight)

Likely lower UCP1 activity

WORSE GENETICS

ADRB2 (Weight)

Likely worse ADRB2 genetics

HIGHER ACTIVITY

FOXO1 (Blood Sugar)

Likely higher FOXO1 activity

LOWER ACTIVITY

CDKN2B (Blood Sugar)

Likely lower CDKN2B activity

LOWER ACTIVITY

PPM1K (Blood Sugar/Diet)

Likely lower PPM1K activity

LOWER ACTIVITY

MADD (Blood Sugar & Insulin)

Predisposed to lower MADD activity

HIGHER ACTIVITY

MTNR1B (Diet & Blood Sugar)

Predisposed to higher MTNR1B activity

TYPICAL ACTIVITY

LEPR (Weight/Leptin Resistance)

Likely typical LEPR activity

TYPICAL GENETICS

ADIPOQ (Weight/ Blood Sugar)

Likely typical ADIPOQ genetics

TYPICAL ACTIVITY

UCP3 (Weight)

Likely typical UCP3 activity

TYPICAL ACTIVITY

ADRB3 (Weight)

Likely typical ADRB3 activity

TYPICAL ACTIVITY

CD36 (Fat Preference/Weight)

Likely typical CD36 activity

TYPICAL ACTIVITY

TFAP2B (Weight/Diet)

Likely typical TFAP2B activity

TYPICAL ACTIVITY

PPARG (Metabolism)

Likely typical PPARG activity

TYPICAL ACTIVITY

APOA2 (Weight, Blood Lipids)

Likely typical APOA2 activity

TYPICAL ACTIVITY

MC3R (Weight)

Likely typical MC3R activity

TYPICAL

TPO (Thyroid)

Likely typical TPO genetics

TYPICAL ACTIVITY

PPARGC1A (Fitness/Blood Sugar)

Likely typical PPARGC1A activity

TYPICAL

SLC2A2 (Sugar Intake)

Predisposed to typical sugar intake

BALANCED PREFERENCES

FGF21 (Carbs vs Fats)

Likely balanced macronutrient preferences

LOWER ACTIVITY

FABP2 (Blood Sugar/ Cardiovascular)

Likely lower FABP2 activity

TYPICAL ACTIVITY

GCKR (Blood Sugar)

Likely typical GCKR activity

TYPICAL ACTIVITY

GIPR (Blood Sugar)

Likely typical GIPR activity

TYPICAL ACTIVITY

PPARA (Keto Diet)

Likely typical PPARA activity

BETTER

FTO (Weight)

Likely better FTO genetics

HIGHER ACTIVITY

MC4R (Weight/ Blood Sugar)

Likely higher MC4R activity

BALANCED ACTIVITY

UCP2 (Weight)

Likely balanced UCP2 activity

HIGHER ACTIVITY

DIO1 (Thyroid)

Likely higher DIO1 activity

HIGHER ACTIVITY

DIO2 (Thyroid)

Likely higher DIO2 activity

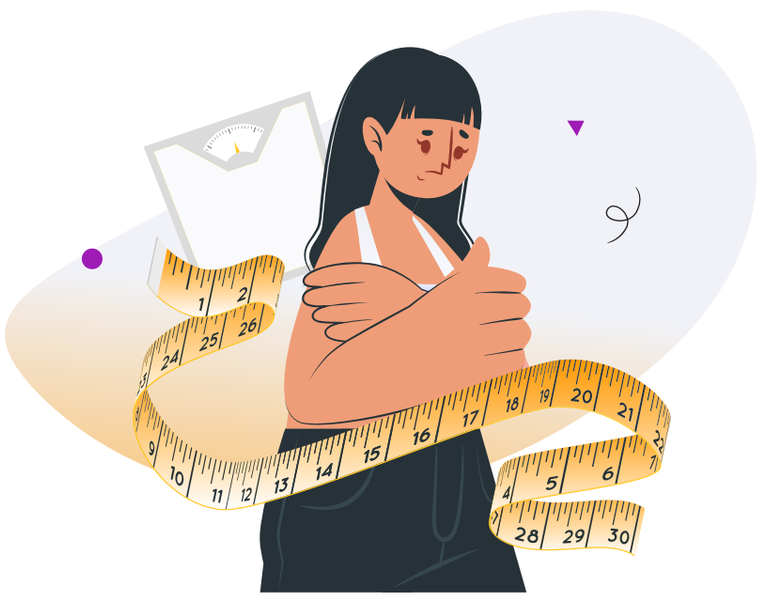
Recommendations Overview

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

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

		DOSAGE
1	Maintain a Healthy Weight	30 minutes
2	Avoid Sugary Foods & Drinks	
3	Maintain Optimal Vitamin D Levels	1000 iu
4	Aerobic Exercise (Cardio)	1 hour
5	Mediterranean Diet	
6	Limit Calorie Intake	
7	Relaxation Techniques	30 minutes
8	Whole Grains	
9	Green Tea	400 mg
10	Probiotics	30 billion CFU
11	Yoga	30 minutes
12	Strength Training	1 hour
13	Eat Fiber-Rich Foods	
14	Intermittent Fasting	
15	Cinnamon	1 g

Your Results in Details



Weight Control

This morning, you thought about ice cream and gained two pounds. Your friend ate an entire pint and the scale didn't even move. In what world is this remotely fair? Controlling weight is an ongoing struggle for many, and **your DNA has a lot to say in the matter**.

Your metabolic rate, how your body stores and burns fat, what affects your appetite are all affected by DNA. **Knowing these genetic predispositions can help you better control weight by making smarter diet and lifestyle choices**.

HIGHER LEVELS

Visceral Fat

Predisposed to more visceral fat

LOWER

Weight Loss from Calorie Restriction

Predisposed to lower weight loss from calorie restriction

TYPICAL LIKELIHOOD

Weight Regain

Typical likelihood of weight regain

LOWER

Metabolic Rate

Predisposed to lower metabolic rate

LESS LIKELY

Overweight

Less likely to be overweight or obese

Visceral Fat

Key Takeaways:

- About 40% of the differences in levels of visceral fat may be due to genetic factors
- High visceral fat levels have been related to high blood pressure, heart disease, and type 2 diabetes.
- Risk factors include a diet high in saturated fat and sugar, a lack of physical activity, stress, aging, and menopause.
- If your genetic risk is high or you have a low risk but more visceral fat than you want, take action on factors that you can change.

If we asked you to picture body fat, the first thing that would likely come to mind is *subcutaneous fat*. This fat is found under the skin of the belly, thighs, and other areas. However, there is another type of body fat called *visceral fat*. This type of body fat hides in the abdomen and surrounds organs like the liver, stomach, and intestines. It may have a greater impact on health than subcutaneous fat [R, R].

Factors that may increase the amount of visceral fat include [R, R, R, R]:

- A diet high in saturated fat and added sugar
- A lack of physical activity
- Long-term stress
- Aging
- Menopause
- **Genetics**

In fact, **about 40% of the differences in levels of visceral fat may be due to genetic factors** [R].

For example, genetically high bioavailable testosterone may be causally associated with lower hip circumference in men and genetically high free testosterone may be causally associated with lower body fat [R, R].

HIGHER LEVELS

Predisposed to more visceral fat based on 813,584 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ADRB2	rs1042714	CG
MC4R	rs2229616	CC
ADARB1	rs76040172	GG
SH3YL1	rs62106258	CT
/	rs114593013	AA
ADH1B	rs1229984	CT
RSPO3	rs9482772	CC
SH3YL1	rs13393304	AG
AS3MT	rs3740390	CT
SEC16B	rs539515	CA
SNX11	rs113866544	TC
PARD3B	rs4482463	CA
PDE4C	rs4808762	TC
NRXN3	rs7156625	GA
NCKAP5L	rs7132908	GA
LINGO2	rs17770336	CT
LRFN2	rs9471333	CT
UHRF1BP1	rs9469899	AG
VEGFA	rs11967262	CG
FABP2	rs1799883	CC
FCER1G	rs5082	AA

GENE	SNP	GENOTYPE
PPARG	rs1801282	CC
FTO	rs56094641	AA
GSTM4	rs7550711	CC
MC4R	rs538656	GG
TFAP2B	rs72892910	GG
/	rs62277680	CC
FUBP1	rs71658797	TT
DLK1	rs28473022	GG
KCNH5	rs79733879	CC
SLC39A8	rs13135092	AA
LIN7C	rs11030112	GG
MEF2C	rs2304608	CC
MFSD10	rs362307	CC
GNPDA2	rs10938398	GG
SH2B1	rs7498665	AA
ADCY3	rs10182458	AA
NEBL	rs10740991	CC
PPARG	rs7649970	CC
BPTF	rs62084234	AA
SLIT2	rs9985922	GG
SEMA4A	rs61813293	GG

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

Weight Loss From Calorie Restriction

Certain genetic variants may influence the effectiveness of weight-loss interventions based on low-calorie diets.

FTO is one of the best-studied genes when it comes to body weight and obesity. The 'A' allele of rs9939609 has shown a robust association with obesity. However, people with this variant may lose more weight from interventions including calorie restriction [R, R, R, R, R].

The *UCP1* encodes a mitochondrial protein also called *thermogenin* because it helps generate heat by a process called *non-shivering thermogenesis.* Because it prevents excess energy from being stored as body fat (and instead turns it into heat), UCP1 may help prevent obesity. The 'CC' genotype of rs1800592 (A3826G) has been associated with lower energy burning at rest. This suggests that carriers should limit their calorie intake to prevent weight gain [R, R].

The *UCP2* gene encodes. The 'GG' genotype of rs660339 (866G>A or Ala55Val) may promote greater weight loss from calorie restriction [R, R].

The *UCP3* gene encodes a protein believed to break down fatty acids and remove them from the inner side of the mitochondria to reduce oxidative damage and preserve mitochondrial function. People with the 'G' allele of rs1800849 (-55CT) may lose more weight on a low-calorie diet [R, R, R].

The *PPARG* gene encodes a protein that acts as a master regulator between nutrient intake, weight control, inflammation, fat burning, and insulin sensitivity. People with the 'G' allele of rs1801282 (Pro12Ala) may gain more weight from increased calorie intake [R, R, R].

Five other *PPARG* alleles, 'T' at rs2959272, 'G' at rs1386835, 'G' at rs709158, 'A' at rs1175540, and 'T' at rs1175544, were associated with greater weight loss from calorie restriction over 14 weeks in a study of 95 overweight women [R].

The *ADRB2* gene encodes a receptor that binds catecholamines, especially adrenaline. Catecholamines control fat burning and energy expenditure, especially during caloric restriction. The 'G' allele of rs1042714 (Gln27Glu) has been associated with obesity, but also with greater weight loss from a partial meal-replacement hypocaloric diet [R, R].

The *ACSL5* gene encodes an enzyme called long-chain fatty-acid-coenzyme A ligase 5 that plays a key role in lipid synthesis and breakdown. People with the 'T' allele of rs2419621 may lose more weight and improve metabolic parameters more from a partial-meal replacement intervention [R].

The *VEGFA* gene encodes a protein that initiates blood vessel formation and plays a critical role in both normal and abnormal blood vessel growth (*angiogenesis*). The 'T' allele of rs1358980 impaired weight loss during a dietary intervention in a study of 707 participants[R, R, R].

The *GNAS* gene encodes a protein belonging to the *guanine nucleotide-binding protein* family, which is involved in signaling. Specifically, GNAS stimulates an enzyme called adenylate cyclase that regulates the activity of several glands. In a trial of 110 participants, people with the 'GG' genotype of rs6123837 (-A1211G) lost more weight from a low-calorie diet [R, R, R, R].

The *LCT* gene encodes lactase, the enzyme that helps digest the milk sugar lactose. The 'G' allele of rs4988235 was

LOWER

Predisposed to lower weight loss from calorie restriction based on 22 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
FTO	rs9939609	TT
PPARG	rs1175544	CC
PPARG	rs1175540	CC
PPARG	rs709158	AA
IFNGR1	rs13201877	AA
ACSL5	rs2419621	CC
PPARG	rs1801282	CC
GNAS	rs6123837	AA
ADPGK	rs7164727	TT
RPTOR	rs12940622	GG
ADRB2	rs1042714	CG
LCT	rs4988235	GA
RETN	rs1862513	CG
LMX1B	rs10733682	AG
ETS2	rs2836754	TC
UCP1	rs1800592	TC
FUCA1	rs3123554	GA
UCP2	rs660339	AG
VEGFA	rs1358980	CT
PPARG	rs2959272	TT
CNR1	rs1049353	TT
UCP3	rs1800849	GG

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

associated with greater loss from a low-calorie, high-protein diet in a study [R, R, R].

The *RETN* gene encodes resistin, a hormone released by fat cells and linked to obesity and type 2 diabetes. People with the ‘’ allele of rs1862513 lost more weight and triglycerides after 3 months on a low-fat, low-calorie diet in a study [R, R].

The *CNR1* gene encodes the type-1 cannabinoid receptor (CB1) of the endocannabinoid system. People with the minor ‘T’ allele of rs1049353 (1359G/A) lost more weight and LDL cholesterol after 3 months on a low-fat, low-calorie diet in a study [R, R, R, R].

The *CNR2* gene encodes another cannabinoid receptor (CB2) that is most abundant in the immune system. The minor ‘A’ allele of rs3123554 has been associated with higher obesity rates and lower weight loss from different hypocaloric diets [R].

Finally, a study of 1198 obese children associated the ‘G’ allele of rs13201877 (*LOC107986544*), the ‘A’ allele of rs10733682 (*LMX1B*), and the ‘C’ allele of rs2836754 (*ETS2*) with a greater weight reduction from a lifestyle intervention while the ‘T’ allele of rs7164727 (LOC100287559) and the ‘G’ allele of rs12940622 (*RPTOR*) were associated with lower weight reduction [R].

Weight Regain

A lot of people who are overweight focus on losing weight by dieting or exercising. However, many people who have lost weight while on a diet will tell you it's hard to keep the weight off once the diet is over. In fact, it's very common to regain up to 50% of weight lost within a year after losing it [R]!

Different people may find it easier or harder to keep the weight off. Some of those differences may be genetic.

Genes linked to weight regain may influence [R, R, R, R, R, R, R]:

- The way fat is stored in the body
- Inflammation
- Feelings of hunger
- Feelings of reward from eating food

TYPICAL LIKELIHOOD

Typical likelihood of weight regain based on 54 genetic variants we looked at

72nd

PERCENTILE

Your risk is **greater than 72% of the population** and **lower than 28% of the population.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
LEP	rs4731426	GG
ALOX5AP	rs4769873	CC
ALOX5AP	rs9578196	CC
LEP	rs2071045	TT
TUB	rs7396690	GC
GCG	rs3761656	GT
BDNF	rs6265	CT
ALOX5AP	rs9315051	GA
TFAP2B	rs987237	AA
ADRB2	rs1042714	CG
ADRB2	rs1042713	AG
APP	rs2242682	TC
LEPR	rs4655537	AG
TUB	rs4385931	CG
GHRL	rs2619507	GA
ALOX5AP	rs3885907	CA
GHRL	rs1617161	TC
APP	rs466448	AG
BHLHE40	rs908078	CT
APP	rs2830054	TG
APP	rs9976453	GC

GENE	SNP	GENOTYPE
PEX11A	rs894160	CT
ANKK1	rs1800497	GA
SH3YL1	rs6548238	TC
KCTD15	rs29941	AG
FTO	rs9939609	TT
POMC	rs7565877	AA
POMC	rs3769671	AA
PPARG	rs1801282	CC
PPARGC1A	rs2932976	GG
APP	rs1876063	GG
ADIPOQ	rs17366568	GG
FAM241A	rs17044137	TT
CCK	rs11571842	TT
CCK	rs10865918	AA

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

Metabolic Rate

Key Takeaways:

- Being high or low metabolism is not inherently problematic. Knowing what yours is allows you to adjust various diet, exercise, and lifestyle choices to properly manage it.
- Your metabolic rate influences movement, thinking, breathing, body temperature, and healing rate.
- High metabolic rate may be affected by being younger, being bigger, as well as more active. Low metabolic rate tends to be affected by the opposite of these. Your genetics may impact the influence of these factors.

Your metabolic rate is the number of calories you burn in a day to maintain bodily functions. A lower metabolic rate or a "slower metabolism" means your body needs fewer calories to do basic functions. Others may need to burn more calories to support these functions. These people have a higher metabolic rate or a "faster metabolism" [R,R,R].

Is metabolism related to body weight? If so, is it possible to burn more calories by boosting your metabolic rate?

Differences in metabolic rate may be due to both genetic and environmental factors. Factors that can contribute to a slower metabolism include [R, R, R, R, R]:

- Being smaller
- Having less muscle mass
- Being older
- Being less active
- Not getting enough sleep

People with slower metabolism need fewer calories to get them through the day. They also tend to gain weight more easily.

Factors that can contribute to a faster metabolism include [R, R, R, R]:

- Being larger
- Having more muscle mass
- Being younger
- Being more active

People with faster metabolism need more calories to get them through the day. They also find it harder to gain weight.

Metabolic rate may not change much from age 20 to 60. While you may not change how many calories your body needs to perform automatic functions, **you can burn more calories by being more active**. Regular exercise can help maintain a healthy weight and support overall health [R, R, R, R, R].

If you're concerned about your weight or you think your metabolism is too slow or too fast, talk with your doctor.

LOWER

Predisposed to lower metabolic rate based on 136,918 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
LEPR	rs1805096	GG
PPARGC1A	rs8192678	CT
LEPR	rs1137101	AG
ADRB2	rs1042713	AG
ADRB2	rs1042714	CG
CLOCK	rs1801260	AG
LEPR	rs1137100	AG
UCP1	rs1800592	TC
UCP2	rs659366	TC
CCND2	rs76895963	TT
TP53	rs78378222	TT
PARD3B	rs1470545	CC
SH3YL1	rs62106258	CT
L3MBTL3	rs7740107	AA
HMGA2	rs1351394	CC
DLG5	rs117543413	TC
ZNF628	rs147110934	GT
DCAF16	rs1472852	CA
TEFM	rs6505216	TG
FTO	rs9939609	TT
ADRB3	rs4994	AA
MC4R	rs17782313	TT
UCP3	rs1800849	GG
CCND3	rs33966734	CC
MGA	rs117183161	AA
MC4R	rs76227980	CC
ZBTB26	rs369508364	CC
CDKN1C	rs143840904	CC
FANCC	rs370727606	GG

GENE	SNP	GENOTYPE
ACAN	rs28584580	AA
PAM	rs78408340	CC
ADAMTS10	rs62621197	CC
COQ5	rs76929617	AA
PPA2	rs143847362	AA
ASPRV1	rs35986233	AA
RPS20	rs72656010	TT
IL11	rs4252548	CC
RAD9A	rs7952436	CC
ADAL	rs148076268	AA
DLEU7	rs3118914	GG
HCAR1	rs147730268	GG
MROH8	rs73094911	TT
LTBP1	rs116072427	GG
COPZ2	rs62064921	CC
WDR35	rs113386058	AA
ZNF469	rs76520574	CC
GALR1	rs74540285	AA
UVSSA	rs111391498	AA
DRAM1	rs17032220	TT
ZFHX4	rs61729527	CC

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

Overweight

Key Takeaways:

- Up to **70%** of people's differences in weight may be due to genetics.
- Up to **42% of adults** and **19% of children** in the US meet the medical criteria for obesity.
- Weight gain affects conditions like high blood sugar and heart disease. However, it is highly modifiable by diet and exercise. So, even if your genetic risk is high, there's a lot you can do to reduce its impact.
- Click the **Recommendations** tab for useful weight control tips and **next steps** for relevant labs.

People are finding it harder than ever to manage their weight. **Global obesity rates have skyrocketed** [R, R, R].

Some health experts even say we're in an "obesity epidemic." **Up to 42% of adults and 19% of children in the US meet the medical criteria for obesity** [R, R, R].

Doctors can use *body mass index* (BMI) to tell if someone is obese. To calculate your BMI, divide your weight by the square of your height (kg/m^2). There are many online calculators that can help you do this [R, R].

In Western countries, people with a **BMI of 25 and over** are considered **overweight**. A **BMI of 30 or greater** is considered **obese**. In some Asian countries, a BMI of 25 and over is considered obese [R, R, R].

BMI isn't the only important measure of healthy weight, however. Body composition is also important because muscle is more dense than fat. Thus, a muscular athlete and an obese person can have similar BMIs [R, R].

For this reason, doctors and researchers often use other body weight measurements, including [R, R]:

- Waist circumference (WC)
- Waist-to-hip ratio (WHR)
- Percentage of body fat (%BF)
- Lean (muscle) mass

Some people worry about body weight because they value how they look. However, **body weight impacts both mental and physical health**. Obesity may increase the risk of [R, R]:

- High blood pressure
- High cholesterol
- Heart disease
- High blood sugar
- Reproductive issues and erectile dysfunction
- Breathing problems during sleep
- Joint and bone disorders
- Some cancers

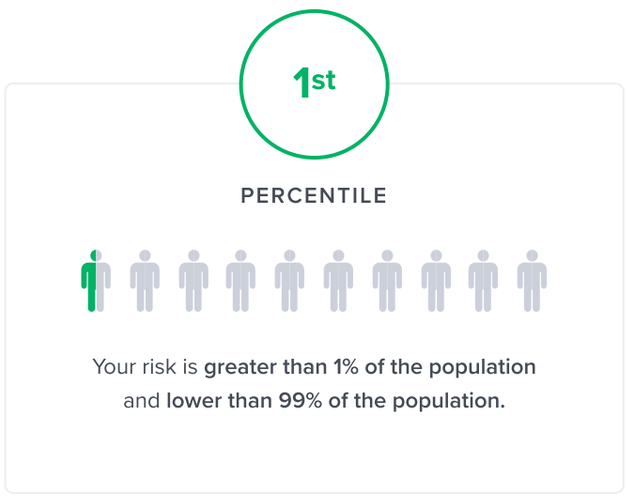
In theory, you gain weight when you consume more calories than you burn. Your body stores the extra energy as fat [R, R, R].

In reality, it's more complicated than that. To stick to a healthy weight, you'll need to manage many factors, including [R, R, R]:

- **Diet**. Pay attention to the amount and type of food you eat, meal timing, and portion size.
- **Lifestyle**. It's better to live an "active" lifestyle than a "sedentary" one and to allow your body to get the sleep it

LESS LIKELY

Less likely to be overweight or obese based on 455,232 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
LEPR	rs1805096	GG
MC4R	rs2229616	CC
TCF7L2	rs7903146	TC
LPCAT2	rs2285053	CC
LEPR	rs1137101	AG
SEC16B	rs591120	CC
NPY	rs16147	TT
POMC	rs6713532	TT
NEGR1	rs3101336	CC
ADRB2	rs1042713	AG
PEX11A	rs894160	CT
CLOCK	rs1801260	AG
UCP1	rs1800592	TC
UCP2	rs659366	TC
ANKK1	rs1800497	GA
NEGR1	rs2815752	AA
GIPR	rs2287019	CC
CDKAL1	rs2206734	CC
GP2	rs12597579	CC
KLF9	rs11142387	CC
RFC4	rs17300539	GG

needs.
- **Environment**. What are your family habits? Do you have social support? What is your stress level? These things have a surprising effect on weight management.
- **Medical conditions**. Anything that changes your metabolism or ability to exercise can also affect body weight.
- **Genetics**. Some gene variants may make it easier or harder to manage your weight.

Doctors may recommend a variety of strategies to help reach and maintain a healthy weight. These include [R]:

- Reducing how much food you eat
- Choosing low-calorie foods
- Choosing more plant-based foods
- Exercising
- Counseling or support groups

Your genes may help determine how well you respond to these strategies.

Rarely, obesity can become a serious health problem. In these cases, doctors may prescribe weight loss drugs or surgery [R].

Up to 70% of differences in weight may be attributed to genetics. Genes that may contribute to body weight influence [R, R, R, R, R, R, R, R, R]:

- Food choices (*FTO*, *IRX4*)
- Appetite (*LEP*, *POMC*, *MC4R*, *NPY*)
- Meal timing (*CLOCK*)
- Fat and sugar metabolism (*FTO*, *UCP2*, *TCF7L2*)

Genetically high bioavailable testosterone may be causally associated with a high risk of obesity (in women). In contrast, genetically high choline, omega-3 fatty acids, and DHA may be causally associated with a lower risk of obesity [R, R].

GENE	SNP	GENOTYPE
TAS1R2	rs35874116	TT
NPC1	rs1808579	CT
MTCH2	rs10838738	AG
NPC1	rs1805081	TC
KCTD15	rs29941	AG
SH3YL1	rs6548238	TC
LEPR	rs1137100	AG
ADRB2	rs1042714	CG
GNB3	rs5443	CT
IL6R	rs4845623	GG
IL6R	rs2228145	CC
LEPR	rs11208659	TT
LEP	rs2167270	AA
LEP	rs3828942	GG
LEP	rs10244329	TT
SOCS3	rs9892622	AA
CRP	rs1205	CC
STEAP1B	rs10242595	GA
STAT3	rs9891119	AA
PYY	rs162431	GG
PGS1	rs4969170	GA
TMC8	rs4969168	GA
UCP2	rs2075577	AG
GNPDA2	rs16858082	TT
UCP2	rs647126	GA
RFC4	rs266729	GC
UCP2	rs660339	AG
STMN4	rs140901272	CC
IL1B	rs1143634	GG

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

Blood Sugar Control

At the morning meal, your child proclaims: "*Because my ancestors were hunter-gatherers, I can no longer eat oatmeal for breakfast and will be eating bacon instead*". Amusing as it might be, your child could technically be right. Your DNA might affect your ability to digest complex carbohydrates and many more aspects of your blood sugar control.

Controlling blood sugar is vital to overall health. Too high or low levels can lead to serious health issues. A number of factors can impact the control of blood sugar, from diet to lifestyle or your DNA. **Knowing your genetic predispositions can help you make smart health decisions and thus lower the risk of issues like high blood sugar and insulin resistance.**

LOWER LEVELS
GLP-1
Predisposed to lower GLP-1 levels

TYPICAL LEVELS
Fasting Glucose
Predisposed to typical fasting glucose levels

TYPICAL
Insulin Resistance
Predisposed to typical insulin resistance

TYPICAL LEVELS
HbA1c
Predisposed to typical HbA1c levels

TYPICAL LIKELIHOOD
Type 2 Diabetes
Typical likelihood of type 2 diabetes

TYPICAL LEVELS
Insulin
Predisposed to typical insulin levels

LESS LIKELY
Low Blood Sugar
Less likely to have low blood sugar

LESS LIKELY
Metabolic Syndrome
Less likely to have metabolic syndrome

LOWER LEVELS
Postprandial Glucose
Likely lower postprandial glucose levels

GLP-1

GLP-1 levels can vary widely among individuals, depending on factors such as [R]:

- Time of day
- Nutritional status
- Meals consumed
- Health conditions
- **Genetics**

During fasting GLP-1 levels remain low. Other causes of low GLP-1 include [R]:

- Obesity
- Diabetes
- Fatty liver
- Polycystic ovary syndrome (PCOS)

Healthy people with low GLP-1 levels may have a higher risk of developing diabetes [R].

High GLP-1 levels can be caused by a recent meal. Bioactive GLP-1 levels may increase two- to threefold after 20-30 minutes after a meal according to the meal size and composition of it. Food components such as MUFAs and fructose may be particularly effective at increasing GLP-1 levels. Moreover, these may increase GLP-1 levels [R, R, R, R]:

- Gastric bypass surgery
- Medications (e.g., DPP-4 inhibitors)

LOWER LEVELS

Predisposed to lower GLP-1 levels based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
RAPGEF4	rs150495482	CC
POLR1C	rs201320592	GG
GPT	rs139849083	CC
ADAP1	rs1568773	AA

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

Fasting Glucose

Glucose is a type of sugar. **Fasting glucose –or fasting blood sugar – is the measurement of one's blood sugar level after 8-12 hours of avoiding food and drinks**. Fasting glucose levels help show how your body deals with dietary sugar. Doctors may order a fasting glucose test to check if someone is diabetic [R, R].

Your fasting glucose levels are partly dependent on your genes! Up to 65% of differences in people's fasting glucose levels may be attributed to genetics. Genes involved in fasting glucose may influence [R, R]:

- Pancreas development and function
- Insulin activity
- Glucose breakdown

Genetically high bioavailable testosterone levels may be causally associated with lower fasting glucose in men [R].

However, keep in mind that your diet and lifestyle may also contribute to your fasting glucose levels. If you have a genetic predisposition for higher fasting glucose levels, the following lifestyle changes may help [R]:

- Exercising
- Maintaining a healthy weight
- Avoiding cigarette smoke and alcohol
- Following a healthy diet

Genetically higher glucose levels may play a role in [R, R, R, R, R, R, R, R, R, R, R, R, R, R, R, R, R, R, R, R, R, R]:

- Glaucoma
- Gum disease
- High blood sugar
- Heart health
- Heart attack
- Alzheimer's

TYPICAL LEVELS

Predisposed to typical fasting glucose levels based on 954,309 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TCF7L2	rs7903146	TC
SLC30A8	rs13266634	CC
GCKR	rs780093	CC
CDKN2A	rs10811661	TT
CETP	rs708272	AA
FOXO3	rs2802292	GG
PPARG	rs1801282	CC
PPARA	rs1800206	CC
RFC4	rs17300539	GG
MRPS31	rs10507486	GG
ADCY5	rs11708067	AA
BCL2	rs12454712	TT
HMGA2	rs2261181	CT
IMPDH1	rs791595	AG
TNF	rs2857605	CT
SLC38A11	rs10195252	CT
TSPAN3	rs7177055	GA
DGKB	rs2191349	GT
PPARGC1A	rs8192678	CT
UCP2	rs659366	TC
ADRB2	rs1042714	CG
ADRB2	rs1042713	AG
CRY2	rs11605924	CA
IGF2BP2	rs1470579	CA
CCND2	rs76895963	TT
CDKAL1	rs7756992	AA
FTO	rs9939609	TT
MC4R	rs12970134	GG
JAZF1	rs1635852	CC

GENE	SNP	GENOTYPE
TAP2	rs2071479	CC
SLC2A2	rs5400	GG
FCER1G	rs5082	AA
IRS1	rs2943641	TC
DIO2	rs225014	TT
IRS1	rs1801278	CC
TNF	rs1800629	GG
FABP2	rs1799883	CC
GIPR	rs10423928	TT
KCNJ11	rs5215	TT
/	rs184660829	TT
/	rs569511541	AA
/	rs562386202	AA
/	rs543786825	CC
/	rs759111467	GG
CCDC68	rs76197067	AA
RNASEH2A	rs755734872	CC
NEDD1	rs557027608	GG
/	rs533172266	CC
QSER1	rs528122639	GG
BPTF	rs558308082	GG

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

Insulin Resistance

Insulin resistance is the reduction of the body's ability to control blood sugar levels. It happens when the muscles, liver, and fat cells no longer respond to insulin and have trouble taking sugar up [R].

In response, the pancreas is forced to produce more insulin than normal to keep blood sugar in balance. Hence, people with insulin resistance may have high insulin levels. Blood sugar levels may also rise eventually, paving the way for diabetes [R, R].

Homeostatic model assessment (HOMA-IR) helps measure insulin resistance. It is calculated using your fasting glucose and fasting insulin. The higher your HOMA-IR, the more insulin resistant you are [R, R].

Insulin resistance is commonly caused by two factors**: overeating and lack of physical activity**. These can cause a buildup of fat in the liver and muscles that lead to insulin resistance [R, R, R].

Insulin resistance is associated with overweight and obesity, especially due to the accumulation of belly fat. However, normal-weight people may also have insulin resistance. Other health conditions may also lead to insulin resistance, including [R]:

- Sleep apnea [R]
- Thyroid disorders [R, R, R]
- Polycystic ovary syndrome (PCOS) [R, R]
- Pancreas disease [R, R]
- Acromegaly (too much growth hormone) [R]
- Cushing's syndrome (excess of cortisol) [R]
- Rare genetic diseases [R, R, R, R]

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

The risk of insulin resistance may also increase due to:

- Aging [R, R]
- Stress [R, R]
- Fasting [R, R, R]
- Western diet [R]
- Too little sleep [R, R, R, R]
- Pregnancy [R]
- Exposure to toxins (e.g., herbicides) [R, R, R]
- Some drugs (e.g., corticosteroids) [R, R]

Genetics also influences insulin resistance. Up to **65%** of differences in people's insulin resistance may be due to genetics [R, R].

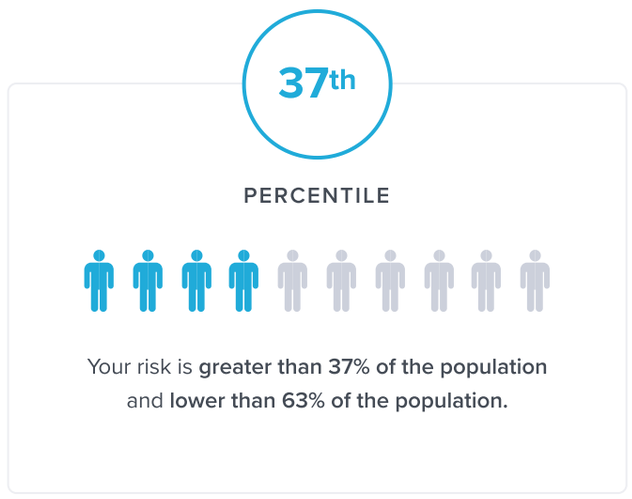
Insulin resistance may increase the risk of:

- Diabetes
- Liver disease
- Metabolic syndrome

Interestingly, insulin resistance may occur up to 15 years before diabetes develops. Read this post for a detailed list of tips to reduce insulin resistance [R].

TYPICAL

Predisposed to typical insulin resistance based on 2,420 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GAS1	rs9792548	AA
PPARG	rs1801282	CC
IRS1	rs2943641	TC
PPARG	rs3856806	CC
FOXO3	rs13217795	TT
FOXO3	rs2802288	AA
IGF1	rs35767	GG
NAT2	rs1208	AA
TIMP4	rs13081389	AA
KLHL2	rs17046216	AA
LEPR	rs1137101	AG
MRPS31	rs4581585	CC
ZC3H12C	rs475338	AA
FBXO21	rs2036313	GG
HAPLN1	rs1457105	CC
/	rs12969333	AA
DAAM2	rs4345393	GG
ME1	rs11967452	CC
KCNK17	rs10456469	GG
ORMDL3	rs939345	CC
ZIC2	rs7338383	GG

GENE	SNP	GENOTYPE
CSNK2A1	rs6053042	CC
RAB28	rs1197712	AA
ATP8B1	rs10439020	AA
MPC1	rs2281056	AA
MROH8	rs11698899	GG
RUNX3	rs803323	AA
TLR4	rs13290714	CC
SORCS1	rs7088188	CT
MDGA1	rs17589516	AA
CACNA1D	rs1401492	CC
SLC10A2	rs16962638	AA
ATP10A	rs6576507	TT
/	rs7043482	AC
CSMD1	rs2407314	CC
FTO	rs9939609	TT
TCERG1L	rs7077836	GG
ADRB3	rs4994	AA
FABP2	rs1799883	CC
FTO	rs1421085	TT
FTO	rs1121980	GG
BRD1	rs13057821	CC
KL	rs9535766	TT
UBR1	rs17776090	AA
BMP8A	rs710912	CC
/	rs2873975	GG
POLL	rs3730464	AA
RUNX1	rs17227476	GG
NINL	rs11698267	GG
FAM135B	rs10088248	CC

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

HbA1c

Sugar in the blood can stick to **hemoglobin**, a protein that helps red blood cells transport oxygen around the body. Hemoglobin with sugar stuck to it is called **glycated hemoglobin** (**HbA1c** or A1c). HbA1c percentage reflects the average amount of sugar in your blood over the past 8-12 weeks. The higher the percentage, the higher your blood sugar [R, R, R].

A doctor might order blood sugar tests like HbA1c if any of the following risk factors apply to you [R, R]:

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

If you're at risk of diabetes, your doctor may advise you to lose weight and change your diet. **Eating less sugar is usually the first step.** If your blood sugar is very high, your doctor may also prescribe medications [R, R].

Up to 75% of the differences in people's HbA1c levels can be attributed to genetics. Genes involved in glucose metabolism may contribute to higher HbA1c levels [R, R, R].

TYPICAL LEVELS

Predisposed to typical HbA1c levels based on 962,206 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CCND2	rs76895963	TT
ANKH	rs146886108	CC
H2BC5	rs144861591	CC
HNRNPUL1	rs180958600	AA
G6PC2	rs560887	CC
SOS2	rs72681869	GG
UBE2V2	rs113440580	CC
GYPC	rs111631066	GG
KEL	rs4987703	CC
GLP1R	rs10305492	GG
MSTN	rs191148279	GG
THADA	rs41382648	CC
NUDCD3	rs1985469	TA
SLC30A8	rs13266634	CC
ADCY5	rs11708067	AA
CCND1	rs74606104	GG
SLC22A23	rs112136863	GA
PFKM	rs4760682	CA
TCF7L2	rs35198068	CT
FN3KRP	rs9909940	TC
MTNR1B	rs10830963	CG
PIEZO1	rs837763	TC
OCEL1	rs12984096	GC
HK1	rs17476364	TT
PFKL	rs17850433	TT
SPTA1	rs2852635	GG
ATP11A	rs76533333	AA
PAX4	rs2233580	CC
ANK1	rs4737010	GG

GENE	SNP	GENOTYPE
ARPC1B	rs117370443	TT
PDE3B	rs539505257	GG
UBXN2A	rs72781679	GG
MAP3K15	rs56212339	T
GSN	rs1560980	GG
CDKAL1	rs35261542	CC

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

Type 2 Diabetes

Key Takeaways:

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

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

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

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

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

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

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

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

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

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

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

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

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

TYPICAL LIKELIHOOD

Typical likelihood of type 2 diabetes based on 1,048,858 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TCF7L2	rs7903146	TC
SLC30A8	rs13266634	CC
GCKR	rs780093	CC
CDKN2A	rs10811661	TT
ADCY5	rs11708067	AA
BCL2	rs12454712	TT
HMGA2	rs2261181	CT
IMPDH1	rs791595	AG
TNF	rs2857605	CT
SLC38A11	rs10195252	CT
TSPAN3	rs7177055	GA
DGKB	rs2191349	GT
IGF2BP2	rs1470579	CA
CCND2	rs76895963	TT
TCF7L2	rs4506565	TA
PEMT	rs12325817	CC
WSB2	rs7973260	GA
IGF2BP2	rs11927381	CT
IGF2BP2	rs11705701	AG
MC4R	rs12970134	GG
JAZF1	rs1635852	CC

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

- IGF-1
- Neutrophils
- Leucine

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

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

GENE	SNP	GENOTYPE
TAP2	rs2071479	CC
GIPR	rs10423928	TT
KCNJ11	rs5215	TT
CDKAL1	rs7756992	AA
/	rs184660829	TT
/	rs569511541	AA
/	rs562386202	AA
/	rs543786825	CC
/	rs759111467	GG
CCDC68	rs76197067	AA
RNASEH2A	rs755734872	CC
NEDD1	rs557027608	GG
/	rs533172266	CC
CDKAL1	rs9465871	TT
QSER1	rs528122639	GG
BPTF	rs558308082	GG
ZC3H11B	rs553014999	TT
ABCC8	rs67254669	AA
INS	rs571342427	TT
FAM13A	rs576406049	CC
/	rs745903616	GG
FTO	rs9939609	TT

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

Insulin

Key Takeaways:

- Insulin is the key hormone for blood sugar control.
- High insulin levels may play a role in diabetes, obesity, heart disease, and cancer.
- Low insulin levels may result from type 1 diabetes and pancreas conditions.
- Up to 55% of differences in people's insulin levels may be due to genetics.

Insulin is a hormone that increases the uptake and storage of sugar in muscles, liver, and fat cells for energy production. By doing this, insulin lowers blood sugar levels [R, R, R, R, R].

Between **30%-55%** of differences in people's insulin levels may be due to genetics [R, R, R].

Besides genetics, factors linked to **high insulin levels** include:

- Insulin resistance [R]
- Type 2 Diabetes [R]
- Weight change and obesity [R, R, R, R]
- Insulinomas (usually benign pancreatic tumors) [R, R]

Low insulin levels may result from:

- Type 1 diabetes [R, R]
- Inflammation of the pancreas (pancreatitis) [R]
- Pancreas removal [R]

TYPICAL LEVELS

Predisposed to typical insulin levels based on 462 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HDAC7	rs111264094	CC
PPARG	rs11712037	CC
PPARG	rs1801282	CC
RFC4	rs17300539	GG
CETP	rs708272	AA
MRPS31	rs10507486	GG
FOXO3	rs2802292	GG
PPARA	rs1800206	CC
IGF1	rs860598	AA
SPX	rs6487237	AA
BCL2	rs12454712	TT
/	rs200678953	TT
BMP2	rs979012	TT
PPARGC1A	rs8192678	CT
TCF7L2	rs7903146	TC
ADRB2	rs1042713	AG
ADRB2	rs1042714	CG
UCP2	rs659366	TC
TMEM60	rs848494	AA
ZC3H11B	rs6674544	GA
ARL15	rs4865796	AG
/	rs77935490	AT
CDHR4	rs9819511	TC
STC1	rs13258890	CT
PDGFC	rs6855363	TC
VEGFA	rs998584	CA
CHRDL1	rs12007422	T
PDE3A	rs12369443	AG
EVI5L	rs4804833	AG

GENE	SNP	GENOTYPE
GLIS3	rs4339696	GT
HMGA1	rs116141873	GG
INSR	rs1799815	GG
TSC22D2	rs62271373	TT
FTO	rs9939609	TT
SLC2A2	rs5400	GG
FCER1G	rs5082	AA
DIO2	rs225014	TT
IRS1	rs1801278	CC
TNF	rs1800629	GG
FABP2	rs1799883	CC
IRS1	rs17508368	CC
DPYSL5	rs61007968	GG
BLK	rs12541800	GG
RBL2	rs2024449	CC

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

Low Blood Sugar

Key Takeaways:

- Up to **65%** of differences in people's blood sugar may be due to genetics.
- Other non-diabetic risk factors include age, drinking alcohol, certain medications, severe infections, problems with your adrenal or pituitary gland, liver, kidney, or pancreas.
- Other diabetic risk factors include taking too much diabetes medication, not eating enough or skipping meals, increasing physical activity without adjusting diet or medications, and drinking alcohol.
- If you have high genetic risk, you may lower overall risk by taking action on those factors that you can change. If you have diabetes, make sure to follow your diet, exercise, and medication regimen.
- Click the **Recommendations** tab for potential dietary and lifestyle changes and **next steps** for relevant labs.

Glucose is a sugar that our body uses to make energy. **Insulin is a hormone that causes blood sugar (glucose) levels to drop.** People with diabetes don't make enough insulin and have to take insulin or other medication to help regulate their blood sugar levels. **However, too much medication can cause low blood sugar (hypoglycemia)** [R, R, R].
In people without diabetes, hypoglycemia may be caused by [R]:

- Too much alcohol
- Eating disorders
- Kidney or liver problems
- Hormone problems
- Medication
- Gut surgeries

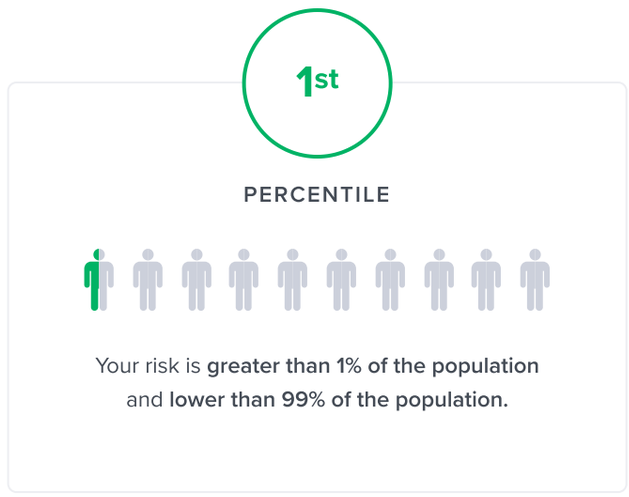
For most people, fasting blood sugar levels below 70 mg/dL or 3.9 mmol/L are considered too low. However, these numbers may differ in people with certain medical conditions. Talk to your doctor if you are concerned about your blood sugar levels [R, R].
People with hypoglycemia may have symptoms like [R]:

- Fast or skipping heartbeat
- Fatigue
- Pale skin
- Shaking
- Sweating
- Tingling or numbness of the face
- Lightheadedness or fainting

Any low blood sugar episode that produces symptoms should be treated immediately. Untreated, hypoglycemia can be life-threatening [R, R].
Immediate treatment of hypoglycemia involves getting some sugar into the body. Fruit juice, regular soft drinks, and sugary candy are good first options because they work quickly. Then, a proper meal or snack should be eaten. In extreme cases, medication may be needed [R, R, R].
If you have recurrent hypoglycemia, work with your doctor to manage the condition [R].

LESS LIKELY

Less likely to have low blood sugar based on 125,784 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MTNR1B	rs10830963	CG
GCK	rs4607517	AG
URAD	rs11619319	AA
TOP1	rs6072275	GG
PROX1	rs340874	TT
GCKR	rs780094	CT
FBRSL1	rs10747083	GG
PEAK1	rs7178572	AA
CRY2	rs11605924	CA
ZBED3	rs7708285	AA
TCF7L2	rs4506565	TA
KL	rs576674	AG
GLIS3	rs7034200	AC
PDE6C	rs2785137	GA
G6PC2	rs560887	CC
TMEM245	rs16913693	TT
FOXA2	rs6113722	GG
ADRA2A	rs10885122	GG
SLC30A8	rs11558471	AA
RPL22L1	rs11920090	TT
ZBTB38	rs11919595	TT

GENE	SNP	GENOTYPE
MADD	rs7944584	AA
ADCY5	rs11708067	AA
CDKN2A	rs10811661	TT
STARD10	rs11603334	GG
FADS2	rs174550	TT
SLC25A29	rs3783347	GG
DUS2	rs8044995	GG
CARD9	rs3829109	GG
C2CD4B	rs11071657	AA
MANBA	rs223486	GC
QPCTL	rs2302593	GC
IGF1R	rs6598541	AG
AMT	rs11715915	CC
RGS17	rs1281962	CC

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

Metabolic Syndrome

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

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

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

LESS LIKELY

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

5th

PERCENTILE

Your risk is **greater than 5% of the population** and **lower than 95% of the population.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TCF7L2	rs7903146	TC
MTNR1B	rs10830963	CG
LPL	rs328	CC
WSB2	rs7973260	GA
MLXIPL	rs12056034	GA
TRIB1	rs2980888	CT
GALNT2	rs2281721	CT
INO80E	rs3814883	TT
GSR	rs10954772	TT
VEGFA	rs998584	CA
C1QTNF4	rs7124681	CA
GCKR	rs1260326	CT
ATP1B2	rs1143015	AG
HLA-C	rs9378248	GA
NAT2	rs4921913	TC
HLA-DQA2	rs5021727	AG
NCKAP5L	rs7138803	GA
ADRB3	rs4994	AA
MC4R	rs17782313	TT
SIDT2	rs964184	CC
CD300LG	rs72836561	CC

GENE	SNP	GENOTYPE
PCSK7	rs662799	AA
SIDT2	rs651821	TT
ADAL	rs139974673	TT
ARAP2	rs73123462	CC
ZDHHC18	rs114165349	GG
HNF4A	rs1800961	CC
RSPO3	rs577721086	TT
CLPTM1	rs483082	GG
ILRUN	rs11754773	AA
PPP1R3B	rs9987289	GG
HMGA1	rs76376137	TT
FADS2	rs1535	AA
SLC39A8	rs13107325	CC
FTO	rs56094641	AA
MC4R	rs66922415	AA
PABPC4	rs11206374	GG
PLG	rs11751347	CC
CMIP	rs2925979	CC
KLF14	rs10260148	CC
SEC16B	rs10913469	TT
BPTF	rs11871285	GG
SNX15	rs35661464	CC
TUBG2	rs12945575	CC
LIN7C	rs56133711	GG
SNX10	rs1534696	AA
RPL17	rs1105654	AA
TRPS1	rs3808439	GG
GAD1	rs12472667	CC
MLLT10	rs9971210	CC

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

Postprandial Glucose

The postprandial glucose test measures how effectively the body's insulin response manages the spike in blood glucose after eating. In healthy individuals, insulin ensures that cells can absorb the glucose and either use it for energy or store it appropriately, thereby returning blood sugar levels to their normal range within a couple of hours.

However, if the body cannot respond to insulin properly, as in the case of insulin resistance or in diabetes, blood sugar levels remain elevated longer than they should. This sustained high level of blood sugar, or hyperglycemia, if left unchecked, can lead to a variety of chronic health issues, including nerve damage, kidney disease, and cardiovascular problems.

LOWER LEVELS

Likely lower postprandial glucose levels based on 374 genetic variants we looked at

Thyroid Health

Thyroid hormones are key players in your metabolic health. They affect your metabolic rate, body temperature, energy production, breathing, and more. Needless to say, if your thyroid is out of balance, your metabolism is going to suffer.

Thyroid issues are something to discuss with your doctor if you suspect anything. Your genetic predispositions may indicate particular aspects of thyroid health to focus on and help reduce the risk of potential problems.

TYPICAL LIKELIHOOD

Underactive Thyroid

Typical likelihood of hypothyroidism

TYPICAL LIKELIHOOD

Overactive Thyroid

Typical likelihood of hyperthyroidism

TYPICAL LIKELIHOOD

Hashimoto's Disease

Typical likelihood of Hashimoto's disease

TYPICAL LEVELS

TSH

Predisposed to typical TSH levels

TYPICAL LEVELS

T3 (Triiodothyronine)

Predisposed to typical T3 levels

TYPICAL LEVELS

T4 (Thyroxine)

Predisposed to typical T4 levels

TYPICAL LEVELS

Free T4

Predisposed to typical free T4 levels

TYPICAL LEVELS

Reverse T3 (rT3)

Predisposed to typical rT3 levels

TYPICAL LEVELS

Free T3 (fT3)

Predisposed to typical free T3 levels

LESS LIKELY

Graves' Disease

Less likely to have Graves' disease

Underactive Thyroid

Key Takeaways:

- Up to **65%** of differences in thyroid hormone levels may be due to genetics.
- Other risk factors for underactive thyroid include: autoimmune conditions, too much/little iodine, and radiation treatment.
- It can cause fatigue, sensitivity to cold, constipation, goiter, weight gain, voice changes, dry skin, and puffy face.
- Up to **1 in 10** people may have an underactive thyroid, and half of those don't know they have it.
- Be aware of the factors and symptoms, even if your genetic risk is low.
- Click the **Recommendations** tab for potential dietary and lifestyle changes and **next steps** for relevant labs.

The thyroid is a gland found in the front of the neck. It produces hormones T3 and T4, which affect [R]:

- Heart function
- Energy production
- Breathing rate
- Bone growth
- Alertness
- Reproductive health

If the thyroid does not produce enough of these hormones, the whole body may suffer ill effects. This condition is known as *hypothyroidism* (underactive thyroid) [R, R, R].

Up to 10% of people may have an underactive thyroid. Of these, about half don't know they have it [R].

Hypothyroidism can have a number of causes. These include [R, R, R]:

- Autoimmune conditions like *Hashimoto's disease*
- Too much or too little iodine
- Thyroid inflammation (*thyroiditis*)
- Surgery that removes all or part of the thyroid gland
- Radiation treatment
- Some medications
- **Genetics**

If your doctor suspects hypothyroidism, they may look for signs and symptoms like [R, R, R]:

- Fatigue
- Sensitivity to cold
- Constipation
- Enlarged thyroid gland (*goiter*)
- Weight gain
- Voice changes
- Dry skin
- Puffy face

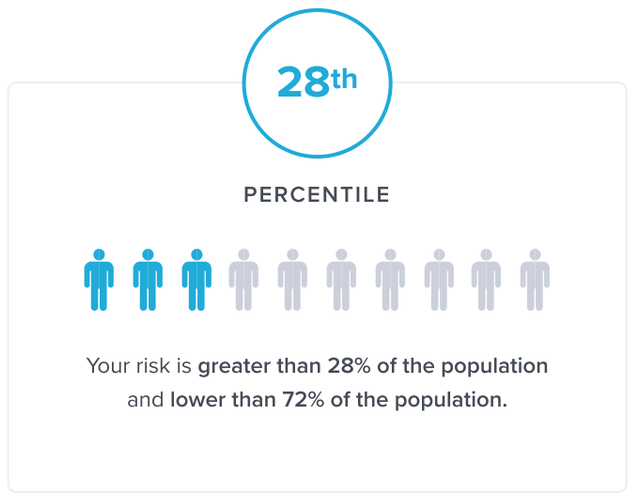
Diagnosis is confirmed with blood tests. These tests check for hormone levels that indicate the thyroid is not as active as it should be [R].

If you have an underactive thyroid (hypothyroidism), treatment will depend on your hormone levels, medical history, and your signs and symptoms.

The standard treatment involves a daily dose of synthetic thyroid hormone medication that can restore thyroid hormone

TYPICAL LIKELIHOOD

Typical likelihood of hypothyroidism based on 875 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CTLA4	rs3087243	GG
PDE8B	rs4704397	AG
TPO	rs11675434	CT
VAV3	rs7537605	GA
FCRL3	rs7522061	TC
TSHR	rs12101261	TC
SH2B3	rs653178	CT
MICB	rs2517532	AG
TYK2	rs34536443	GG
/	rs9271365	TG
RASGRP1	rs12593201	AA
TPO	rs732609	AC
SESN3	rs4409785	TC
CLECL1	rs370475698	DEL(A)T
PDE8B	rs1479565	AG
ARID5B	rs71508903	CT
TPO	rs11675342	CT
SESN1	rs1364450	CA
PLGRKT	rs911760	CA
SASH1	rs9497965	TC
FAP	rs2111485	AG

levels and reverse the signs and symptoms. But keep in mind that it may take some time to adjust the dosage of thyroid hormones so they are right for you [R].

It is extremely important to treat hypothyroidism according to your doctor's instructions. Left untreated, hypothyroidism can lead to *myxedema coma*. This condition is a medical emergency. Even with treatment at a hospital, up to 60% of these cases can lead to death [R].

Up to 67% of differences in thyroid hormone levels may be attributed to genetics. Genes that may affect thyroid function include [R, R]:

- *PDE8B*
- *DIO1*
- *CAPZB*
- *TSHR*
- *FOXE1*

GENE	SNP	GENOTYPE
IL2RA	rs3118469	TA
TRMO	rs925489	CC
NBL1	rs10917477	AA
PTPN22	rs6679677	CC
PTPN22	rs2476601	GG
FOXE1	rs1867277	AA
TNF	rs1800629	GG
DPH5	rs77046277	CC
ADCY7	rs78534766	CC
FLT3	rs76428106	TT
/	rs187707293	TT
TRMO	rs7030280	CC
BACH2	rs6908626	GG
ACAP1	rs61759532	CC
C1QTNF6	rs229528	CC
CD44	rs736374	GG
CBLB	rs13090803	GG
TNFRSF14	rs2234167	GG
RAB5C	rs9902341	CC
RBPJ	rs7441808	AA
DIO1	rs2235544	CC

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

Overactive Thyroid

Key Takeaways:

- Up to **65%** of differences in thyroid hormone levels may be due to genetics.
- Risk factors include: Graves' disease, goiter, too much/little iodine, thyroiditis, pituitary or thyroid gland tumors.
- It can cause: weight loss, increased appetite, irritability, irregular heartbeat, goiter, heart, bone, and muscle problems.
- Hyperthyroidism is fairly rare, mostly due to Graves' disease or iodine deficiency. If your genetic risk is high, the overall risk is still low due to its rarity, but be aware of symptoms.
- Click the **next steps** tab for relevant labs.

The thyroid is a gland found in the front of the neck. It produces T3 and T4, thyroid hormones that affect [R]:

- Heart function
- Energy production
- Breathing rate
- Bone growth
- Alertness
- Reproductive health

In some people, the thyroid produces too much of these hormones. This condition is called *hyperthyroidism* (overactive thyroid) [R, R, R].

Potential causes of overactive thyroid include [R, R]:

- **Autoimmune conditions like *Graves' disease***
- **Thyroid nodules (goiter)**
- Too much or too little iodine
- Thyroid inflammation (*thyroiditis*)
- Pituitary or thyroid gland tumors

Hyperthyroidism is fairly rare. In countries with iodine deficiency, goiter is a common cause. In developed countries like the United States, most people get enough iodine and Graves' disease is a more common cause [R, R].

When the thyroid is overactive, it may produce signs and symptoms like [R]:

- Weight loss
- Increased appetite
- Nervousness or irritability
- Rapid or irregular heartbeat
- Shaking
- Intolerance to heat
- Enlarged thyroid (*goiter*)

Treatment for hyperthyroidism may be different for each person. A doctor may recommend [R]:

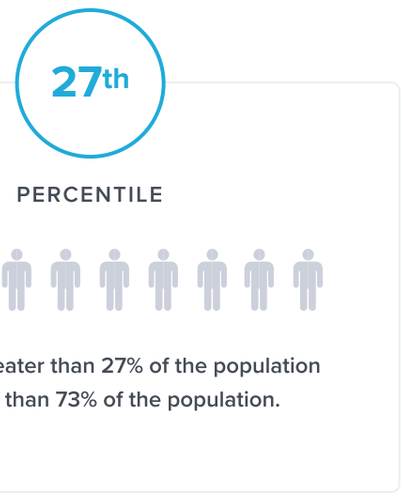
- Medication
- Radiation therapy
- Surgery

Diet changes may also help manage some cases. For example, if you have an autoimmune thyroid condition, you may need to avoid iodine-rich foods like seaweed [R].

It is extremely important to treat hyperthyroidism according to your doctor's instructions. Left untreated, an overactive thyroid can cause [R]:

TYPICAL LIKELIHOOD

Typical likelihood of hyperthyroidism based on 466 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CTLA4	rs3087243	GG
FCRL3	rs7522061	TC
TSHR	rs12101261	TC
SH2B3	rs653178	CT
CD40	rs1883832	TC
MICB	rs2517532	AG
FAM227B	rs17477923	TT
PDE10A	rs2983514	GG
LRRC6	rs118039499	AA
MAF	rs140851213	TT
PDE8B	rs2046045	GT
TSHR	rs2160215	CT
SYT13	rs11038357	AT
SOX9	rs8077245	GT
VEGFA	rs66760320	TC
RNASET2	rs385863	CG
CD40	rs6131010	AG
MYC	rs2466028	TT
TSHR	rs28414437	CA
CTLA4	rs231779	TC
SESN3	rs4409785	TC

- Heart problems
- Bone and muscle problems
- Eye problems
- Fertility problems

Up to 67% of differences in thyroid hormone levels may be attributed to genetics. Genes involved in hyperthyroidism may influence [R, R]:

- Thyroid hormones (*PDE8B*, *DIO1*, *CAPZB*, *TSHR*)
- Immune function (*HLA-DPB1*, *PTPN22*, *CTLA4*)

GENE	SNP	GENOTYPE
CD40	rs1569723	CA
MAF	rs17689159	CT
FCRL3	rs1977710	AG
UHRF1BP1	rs9469899	AG
STAT4	rs12612769	CA
TMPRSS3	rs34544259	GA
PTPN22	rs2476601	GG
TNF	rs1800629	GG
PRLR	rs143210911	GG
HLA-DQA2	rs1794280	AA
TRIM27	rs3135293	TT
TRMO	rs925488	GG
FAM227B	rs4338740	TT
BACH2	rs604912	AA
HLA-DPA1	rs9357156	AA
SLAMF6	rs12026490	TT
ALDH2	rs4646776	GG
MAGT1	rs4826198	A

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

Hashimoto's Disease

Key Takeaways:

- Up to **65%** of differences in people's chances of having Hashimoto's disease may be due to genetics.
- Risk factors include being female, middle age, pregnancy, other autoimmune diseases, and excessive iodine intake.
- It affects 1 to 2 percent of people in the U.S., occurring more often in women than men.
- Click the **Recommendations** tab for potential dietary and lifestyle changes, and **next steps** for relevant labs.

Risk factors for Hashimoto's disease include [R]:

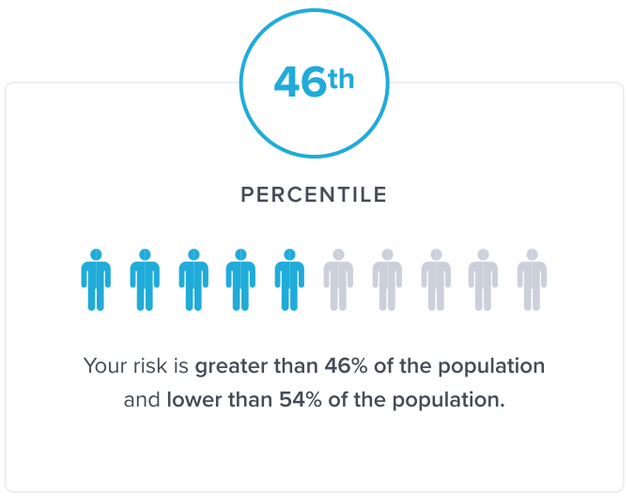
- Being female
- Middle age
- Pregnancy
- Excessive iodine intake
- Radiation exposure
- Having another autoimmune disease
- **Genetics**

Up to **65%** of differences in people's chances of having Hashimoto's disease may be due to genetics [R].

Hashimoto's disease is typically treated with medications to help normalize thyroid hormone levels. **It's important for people with Hashimoto's disease to work closely with their healthcare provider** to manage their condition and prevent complications.

TYPICAL LIKELIHOOD

Typical likelihood of Hashimoto's disease based on 85 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CTLA4	rs3087243	GG
HLA-DPA1	rs9277768	TC
IL6	rs1800795	GG
CTLA4	rs34636506	AA
/	rs9271365	TG
VAV3	rs7537605	GA
TRIB2	rs1534422	GG
PDE8B	rs1993945	TA
SH2B3	rs653178	CT
STAT4	rs11889341	TC
TPO	rs11675434	CT
CTLA4	rs231775	GA
CD69	rs2110451	AG
RPS26	rs11611029	CT
NIPSNAP1	rs757024	CG
SESN3	rs4409785	TC
ZNF668	rs57348955	AG
TNFRSF14	rs2843403	CT
PTPN22	rs2476601	GG
SLC25A27	rs2270450	CC
VAV3	rs17020139	GG

GENE	SNP	GENOTYPE
TRMO	rs7030280	CC
PTPN22	rs1230666	GG
BACH2	rs10944479	GG
TNF	rs1799964	TT
AP4B1	rs12730735	TT
TNF	rs1800629	GG
CTLA4	rs11571297	TT
BACH2	rs7754251	GG
BACH2	rs72928038	GG
LPP	rs13093110	CC
IL2RA	rs706779	TC
GXYLT1	rs4768412	CC

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

TSH

Thyroid-stimulating hormone (TSH), also known as thyrotropin, is a hormone produced by the pituitary gland — a small gland at the base of the brain. **TSH stimulates the thyroid gland** to produce thyroid hormones (T3 and T4). These hormones affect several processes, including energy production, heart function, and reproductive health [R].

Around **65%** of people's differences in TSH levels may be due to genetics [R, R, R].

Even though higher TSH levels may indicate an underactive thyroid, **genetically higher TSH** levels are linked to [R, R, R, R, R, R, R, R, R, R]:

- Reduce mortality, especially from respiratory infections
- Reduce the rate of some types of heart disease and stroke
- Reduce diabetes rates
- Fractures in men
- Alzheimer's in certain groups
- Reduce blood pressure

On the other hand, genetically lower TSH levels are linked to lower cholesterol, gaining weight [R, R, R, R].

TYPICAL LEVELS

Predisposed to typical TSH levels based on 92 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
LRRC6	rs117764941	GG
LRRC6	rs118039499	AA
NKX2-1	rs116909374	CC
NFIA	rs334725	AA
PDE8B	rs2928167	AA
NR3C2	rs11732089	TT
TBX2	rs1157994	GG
CERS6	rs62174422	TT
PDE8B	rs1479567	AG
FAM227B	rs17477923	TT
VEGFA	rs1317983	CT
TNP1	rs13020935	AG
CDK17	rs10735341	GG
MAF	rs58722186	TC
VEGFC	rs4571283	AA
VEGFA	rs9381266	CT
CAPZB	rs12027702	GT
/	rs3104389	CA
FOXA2	rs1203949	TC
INSR	rs4804416	GT
GATA3	rs11592436	GC
C6ORF163	rs2242602	TA
/	rs121908872	GG
CEP128	rs141751376	TT
B4GALNT3	rs145153320	CC
CCDC77	rs546738875	CC
LTA4H	rs61938844	GG
PDE10A	rs2983511	CC
VAV3	rs17020122	CC

GENE	SNP	GENOTYPE
ASXL2	rs6721104	AA
ITPK1	rs6575306	AA
VEGFA	rs34046483	GG
DPH6	rs74888443	CC
TRMO	rs925488	GG
HLA-B	rs1265091	CC
SOX9	rs1042678	GG
ARL17A	rs116956554	GG
THAP4	rs6717283	AA
GNG7	rs72978712	TT
MAL2	rs72682433	TT

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

T3 (Triiodothyronine)

The thyroid is a gland found in the front of the neck that produces thyroid hormones. **T3 (triiodothyronine) is the active thyroid hormone.**

Up to **65%** of the differences in people's T3 levels may be due to **genetics**. Involved genes play a role in thyroid function and immune response [R, R].

Other factors that may affect T3 levels include [R, R, R, R]:

- Autoimmunity
- Stress
- Sleep problems
- Dietary iodine
- Dietary goitrogens (substances that reduce thyroid function)

TYPICAL LEVELS

Predisposed to typical T3 levels based on 20,697 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SLK	rs2475217	CC
INSIG1	rs12534332	GA
FBLL1	rs590784	CA
SERPINA7	rs12687280	T
EPHB2	rs67142165	CC
RAB38	rs116951285	TT
PRKCE	rs10192064	TT
MOV10L1	rs2066773	GG
VPS37B	rs76465767	TT
AGPAT2	rs7020640	CC
CD200R1	rs145944228	GG
TIAM2	rs4482989	CC
ZNF616	rs749618	AA
GALNT13	rs80190198	AA
ERBB4	rs13428799	CC
AGBL1	rs72752186	GG

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

T4 (Thyroxine)

The thyroid is a gland found in the front of the neck that produces thyroid hormones. **T4 (thyroxine)** is a more abundant but less active thyroid hormone. Its breakdown releases active T3.

About **40-55%** of the differences in people's T4 levels may be due to **genetics**. Involved genes play a role in thyroid function and immune response [R, R].

Other factors that may affect T4 levels include [R, R, R, R]:

- Autoimmunity
- Stress
- Sleep problems
- Obesity
- Dietary iodine

TYPICAL LEVELS

Predisposed to typical T4 levels based on 2,581 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
QSOX2	rs7860634	AG
LPCAT2	rs6499766	AA
AADAT	rs11726248	AG
MC4R	rs56069042	AA
/	rs7240777	AA
AADAT	rs7694879	TC
SEPHS1	rs72783371	AA
CA8	rs67583169	CC
ILRUN	rs73405691	AA
LRRC42	rs12127960	AT
H2BC1	rs9356988	AA
DIO2	rs225014	TT
QSOX2	rs11103377	GA
NCOR1	rs11078333	AA
DIO3	rs11626434	GC
CPPED1	rs8063103	GC
GLIS3	rs10119187	CT
SLCO1B1	rs4149056	TC
RNF144B	rs10946313	TC
USP3	rs12907106	CG
MTCH2	rs11039355	CT
NUCKS1	rs951366	TC
CCNT2	rs4954192	TC
INSIG1	rs12534332	GA
DIO1	rs2235544	CC
SERPINA7	rs1804495	C
TRMO	rs7045138	CC
PWWP3B	rs139669326	T
B4GALT6	rs113107469	CC

GENE	SNP	GENOTYPE
UGT1A6	rs6722076	GG
SIM1	rs17185536	CC
SOX2	rs6785807	GG
NEK6	rs10818937	CC
EPHB2	rs67142165	CC
MOV10L1	rs2066773	GG
VPS37B	rs76465767	TT
AGPAT2	rs7020640	CC
CD200R1	rs145944228	GG
TIAM2	rs4482989	CC
ZNF616	rs749618	AA

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

Free T4

Free T4 is a small fraction of the thyroid hormone thyroxine not bound to proteins.

About **40-65%** of the differences in people's free T4 levels may be due to **genetics**. Involved genes play a role in thyroid function and immune response [R, R].

A high or low Free T4 usually indicates over- or underactive thyroid, respectively. A range of factors may affect thyroid function and free T4 levels, including [R, R, R, R, R, R]:

- Autoimmunity
- Obesity
- Exercise
- Toxins like BPA
- Dietary iodine and iron

Genetically higher free T4 levels may be associated with [R, R, R, R, R, R]:

- Lower LDL/Total cholesterol
- High cholesterol
- High blood pressure
- Heart health
- High blood sugar
- Mood swings
- HDL cholesterol
- Age-related macular degeneration
- Gallstones

TYPICAL LEVELS

Predisposed to typical free T4 levels based on 26 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ZGRF1	rs6834538	TC
JAZF1	rs7785730	GG
MC4R	rs56069042	AA
AADAT	rs7694879	TC
SEPHS1	rs72783371	AA
CA8	rs67583169	CC
ILRUN	rs73405691	AA
H2BC1	rs9356988	AA
DIO2	rs225014	TT
QSOX2	rs11103377	GA
NCOR1	rs11078333	AA
DIO3	rs11626434	GC
CPPED1	rs8063103	GC
GLIS3	rs10119187	CT
SLCO1B1	rs4149056	TC
RNF144B	rs10946313	TC
MTCH2	rs11039355	CT
USP3	rs12907106	CG
NUCKS1	rs951366	TC
CCNT2	rs4954192	TC
B4GALT6	rs113107469	CC
/	rs7951105	GG
DIO1	rs2235544	CC
SIM1	rs17185536	CC
SOX2	rs6785807	GG
NEK6	rs10818937	CC

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

Reverse T3 (RT3)

The following factors can elevate rT3 [R, R, R]:

- Stress
- Aging
- Weight loss
- Critical illness
- Liver diseases
- Iron deficiency
- Certain medications

Reverse T3 is also partly affected by **genetics**. Carrying a variant of an enzyme involved in thyroid hormone metabolism is associated with higher rT3 levels and a lower T3/rT3 ratio [R].

TYPICAL LEVELS

Predisposed to typical rT3 levels based on the genetic variants we looked at

50%

OF USERS SHARE THE SAME SCORE

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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
DIO1	rs11206244	CC

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

Free T3 (FT3)

About **40-50%** of people's differences in free T3 levels may be due to **genetics** [R].

Other factors that can change free T3 levels include:

- **Thyroid Issues**: Conditions like hyperthyroidism (an overactive thyroid) can increase free T3 levels, while hypothyroidism (an underactive thyroid) can decrease them.
- **Medications**: Some medicines, especially those for thyroid problems, can affect free T3 levels.
- **Diet**: Not getting enough iodine, a mineral found in foods like fish and dairy, can impact our thyroid and free T3 levels.
- **Pregnancy**: Women might see changes in their free T3 levels during and after pregnancy.
- **Illness**: Some illnesses, especially severe ones, can temporarily affect free T3 levels.
- **Age**: As we get older, our thyroid might not work as efficiently, which can affect free T3.

TYPICAL LEVELS

Predisposed to typical free T3 levels based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
KCNMB4	rs11178277	AG
SERPINA7	rs12687280	T
LMO7	rs7320337	TT

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

Graves' Disease

Key Takeaways:

- Up to **80%** of differences in people's chances of getting Graves' disease may be due to genetics.
- Risk factors include young age, female sex, stress, smoking, and pregnancy.
- Symptoms include weight loss, rapid heartbeat, difficulty sleeping, eye bulging, and sexual dysfunction.
- If you have a high genetic risk, your overall risk is low due to its rarity. You can still improve this risk by taking action on those risk factors you can change.
- Click the **Recommendations** tab for potential dietary and lifestyle changes, and **next steps** for relevant labs.

Risk factors for Graves' disease include [R]:

- **Genetics**
- Being female
- Age under 40
- Other autoimmune disorders
- Emotional or physical stress
- Pregnancy
- Smoking

Up to **80%** of differences in people's chances of getting Graves' disease may be due to genetics [R].

LESS LIKELY

Less likely to have Graves' disease based on 176 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CTLA4	rs3087243	GG
TSHR	rs179247	AA
TSHR	rs28414437	CA
SESN3	rs4409785	TC
CTLA4	rs231779	TC
TPO	rs11675434	CT
IGLV3-21	rs5751536	GA
TSHR	rs2300519	AT
CTLA4	rs231775	GA
CD40	rs1569723	CA
MAF	rs17689159	CT
FCRL3	rs1977710	AG
UHRF1BP1	rs9469899	AG
STAT4	rs12612769	CA
TMPRSS3	rs34544259	GA
RNASET2	rs13210649	TG
TRIB2	rs1534422	GG
TSHR	rs4903964	AG
SH2B3	rs653178	CT
ZNF668	rs57348955	AG
TNFRSF14	rs2843403	CT
IL2RA	rs706779	TC
CD40	rs1883832	TC
HLA-DPA1	rs9357156	AA
SLAMF6	rs12026490	TT
ALDH2	rs4646776	GG
HLA-DQA1	rs2187668	CC
PTPN22	rs2476601	GG
MAGT1	rs4826198	A

GENE	SNP	GENOTYPE
TNF	rs1799964	TT
MICB	rs361525	GG
TNF	rs1800629	GG
CTLA4	rs11571297	TT
BACH2	rs7754251	GG
BACH2	rs72928038	GG
FCRL3	rs3761959	CC
GXYLT1	rs4768412	CC
LPP	rs13093110	CC

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

Miscellaneous

This section covers different aspects of your metabolic health, from sweating to detox to muscle metabolism. Knowing your genetic predisposition in these areas will give you a clearer picture of your metabolic health.

MORE LIKELY

Heavy Sweating

More likely to have hyperhidrosis

TYPICAL LIKELIHOOD

Gout

Typical likelihood of gout

TYPICAL LEVELS

Uric Acid

Predisposed to typical uric acid levels

TYPICAL LEVELS

Ketone Bodies

Predisposed to typical levels of ketone bodies

TYPICAL LEVELS

Lactate

Predisposed to typical lactate levels

TYPICAL LEVELS

Creatine

Likely typical creatine levels

Heavy Sweating

Key Takeaways:

- Genes that affect excessive sweating may influence nerve function and chemical messengers.
- Excessive sweating can impact quality of life and cause undue stress and anxiety. If you are at high genetic risk, take action on your risk factors to help lower overall risk.
- Up to 5% of people in the U.S. may have hyperhidrosis. If you have symptoms, you may want to consult your doctor to rule out other conditions.
- Click the **next steps** tab for relevant lab tests.

Hyperhidrosis is the scientific term for heavy sweating [R].

It's normal to sweat a lot because of exercise, heat, or stress. In the absence of these conditions, a lot of sweat might be a sign that something is wrong [R].

Sweating turns from normal to worrisome if it [R]:

- Changes the way you live your daily life
- Causes anxiety or social problems
- Suddenly gets much worse for no apparent reason
- Suddenly starts while sleeping (night sweats) for no apparent reason

Up to 5% of people in the United States may have hyperhidrosis. Many people do not realize it is a treatable medical condition. For this reason, they often do not bring up symptoms with their doctors. **Only about 1 in 2 people who have it will be diagnosed** [R, R].

Most cases of heavy sweating are caused by a nerve problem. Simply put, the nerves that control the sweat glands are too active. This condition is called *primary focal hyperhidrosis*. It may be treated with [R, R, R, R]:

- Topical medication
- Antiperspirants
- Surgery
- Botulinum toxin therapy

Heavy sweating can also be caused by another health condition. This is called *secondary hyperhidrosis*. Underlying conditions that may cause this include [R]:

- Diabetes
- Menopause
- Thyroid problems
- Low blood sugar
- Infection

Researchers suggest that genetics plays a role in the development of heavy sweating. Genes involved in heavy sweating may influence [R]:

- Chemical messenger activity (*BCHE*, *PSEN2*, *DARS*)
- Nerve function (*PPP3R1*, *PPP1CB*, *ITPR2*)

MORE LIKELY

More likely to have hyperhidrosis based on 103 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
LONP2	rs6500380	GG
SLC6A16	rs149876322	GC
PPP1CB	rs56089836	CC
PPP1CB	rs1534480	CC
DLG2	rs12280544	CC
TLN2	rs139024759	AA
TUSC1	rs117093392	AA
UBLCP1	rs143772159	CC
CADM1	rs144975908	GG
FZD8	rs190252627	CC
/	rs75470475	CC
LRRC7	rs113867145	GG
SETD7	rs183414800	TT
LRRC7	rs113992293	GG
KRT72	rs61740873	GG
/	rs74837903	TT
LRRC7	rs111398942	CC
LRRC7	rs113434595	CC
HNRNPA1P48	rs117324726	CC
UBLCP1	rs77247779	TT
GATA3	rs80243082	GG

GENE	SNP	GENOTYPE
FBXO10	rs142695379	GG
CPNE2	rs7184935	CC
/	rs143053510	TT
ANKH	rs150150334	AA
PITPNM1	rs144939807	CC
GATA2	rs56099243	CC
QSOX1	rs142528261	CC
ADAMTS12	rs140640237	TT
EGFLAM	rs77788652	CC
TMC2	rs147782137	AA
CENPF	rs147733826	CC
/	rs115295459	CC
TBCA	rs140260005	CC
SRRM4	rs113353314	AA
GP2	rs145309364	AA
LYPD6B	rs7586963	GG
ITGA1	rs77066279	GG

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

Gout

Key Takeaways:

- About **30%** of differences in people's chances of developing gout may be due to genetics. It is most common in middle age.
- Risk factors include a diet rich in purines, fructose, alcohol, high blood pressure, overweight, diabetes, kidney disease, and genetics.
- If you are at high genetic risk or have symptoms, take action now on your modifiable risk factors to help lower overall risk.
- Click the **next steps** tab for relevant labs and lifestyle factors.

Gout is a common type of arthritis. It is caused by urate crystals building up in the joints [R, R].

Urate crystals are formed from *uric acid*, a waste product. The body makes the most uric acid when it breaks down *purines*. These are compounds found in our cells and in many foods, such as meat and seafood [R].

The major symptom of gout is pain and swelling in the joints. The most commonly affected joint is the big toe. However, gout can also occur in other joints, such as the elbows, wrists, and fingers [R, R].

Gout comes and goes in cycles called flares. Flare-ups are often sudden and tend to occur at night. Afterward, the joint may be uncomfortable for days or weeks [R, R].

Left untreated, gout can cause [R]:

- *Tophi* (crystals around the joints and other parts of the body, just under the skin)
- Joint deformities
- Loss of bone
- Osteoarthritis

This condition is most common in middle-aged people. Younger people do not usually get gout, but if they do, it tends to be severe [R, R].

In addition to age, other risk factors for gout include [R, R]:

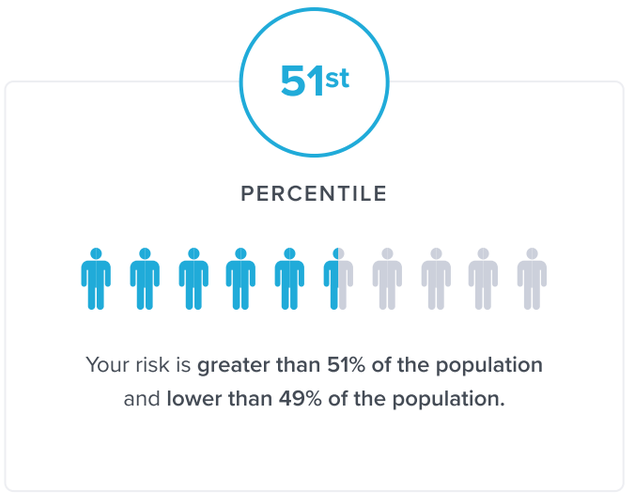
- A diet rich in purines (e.g., from red meat and shellfish)
- Fructose (fruit sugar)
- Alcohol (especially beer)
- Some medication
- High blood pressure
- Being overweight or obese
- Certain health conditions (e.g., diabetes, heart disease, and kidney disease)
- **Genetics**

Gout is a manageable condition with well-established treatments. For people with gout, a doctor may recommend [R, R, R, R]:

- Medication
- A low-purine diet
- Drinking more water
- Avoiding alcohol
- Weight management
- Exercise

TYPICAL LIKELIHOOD

Typical likelihood of gout based on 231 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SLC2A9	rs16890979	CC
SLC2A9	rs6449213	TT
SLC2A9	rs12498742	AA
SLC2A9	rs75341455	CC
SLC2A9	rs13129697	GT
ADH1B	rs1229984	CT
SLC2A9	rs6839820	CC
SPP1	rs2728127	AA
TMEM171	rs520007	CC
TRMT112	rs2078267	CT
GCKR	rs1260326	CT
INHBC	rs2229357	AG
MTX1	rs760077	AT
PRKAG2	rs10224002	GA
MAN2C1	rs1394125	AG
CARMIL1	rs9461183	AG
ABCG2	rs2231142	GG
NUDT9	rs114791459	GG
SPP1	rs114580333	GG
PPM1K	rs4693211	TT
RPS6KA4	rs77085155	TT

About **30%** of differences in people's chances of developing gout may be attributed to genetics. Genes involved in gout may influence [R, R]:

- The immune response (*HLA-B*)
- Uric acid levels (*ABCG2*, *SLC2A9*, *SLC22A11*, *SLC22A12*, *SLC17A1*)

Moreover, genetically higher fasting insulin may be causally associated with a higher risk of gout. In contrast, genetically high testosterone levels may be causally associated with a lower risk of gout in men [R, R, R].

GENE	SNP	GENOTYPE
HOXD1	rs72929103	CC
ABCG2	rs72552713	GG
NRXN2	rs471618	TT
NUDT17	rs1967017	AG
OVOL1	rs11227299	CC
MLXIPL	rs2286276	TT
RREB1	rs11755724	GG
SFMBT1	rs2581778	GG
PRSS16	rs3800307	TT
IDH2	rs28508560	GG
PNPLA3	rs738409	GG
MLXIP	rs28548845	CC
LRP2	rs2075252	CC

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

Uric Acid

Uric acid is made in the liver as an end product of the breakdown of **purines** (chemicals found in our cells and in foods such as meat and seafood). If too much uric acid is produced or not enough is removed by the kidneys, it can build up in the blood and urine. Uric acid crystals can deposit in the body, causing kidney stones or gout [R, R, R, R].

Blood uric acid increases with age. Men tend to have higher levels than women, and are therefore at greater risk of developing gout. This may be because estrogen helps eliminate uric acid [R, R].

Causes of elevated uric acid levels include:

- A diet high in purines (e.g., from meat and seafood) [R, R]
- A diet high in sugar [R, R, R]
- Obesity [R]
- Heavy drinking [R]

Genetically higher uric acid levels may be causally associated with:

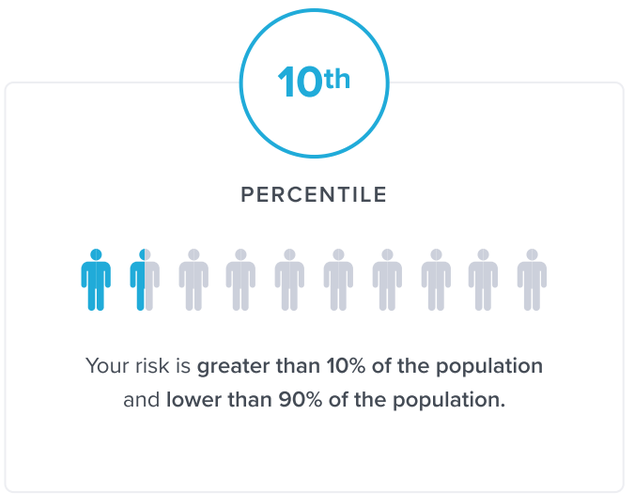
- Gout [R, R]
- High Blood Pressure [R, R, R, R]
- Stroke [R]
- Deep vein thrombosis [R]

Up to 70% of differences in people's uric acid levels may be attributed to genetics. Genes involved may influence [R]:

- How kidneys clear uric acid
- How much uric acid is created in the liver

TYPICAL LEVELS

Predisposed to typical uric acid levels based on 11,615 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HMGCS2	rs150147865	AA
SOS2	rs72681869	GG
ACVR1	rs186905001	AA
HPRT1	rs73560966	C
SLC17A1	rs2762353	GG
CCND2	rs76895963	TT
ABCA6	rs77542162	AA
PIP5KL1	rs56379622	GG
ZKSCAN5	rs34670419	GG
UNCX	rs13230509	CC
SLC39A8	rs13107325	CC
AAK1	rs12987661	TT
NRG4	rs4886755	GG
SLC16A9	rs1171614	CT
TBX2	rs9895661	TT
INHBC	rs2229357	AG
ADAM15	rs11264341	CT
SH3YL1	rs62106258	CT
VEGFA	rs1317983	CT
PRKAG2	rs6464165	CT
PKD2	rs45499402	GG

GENE	SNP	GENOTYPE
ZDHHC18	rs114165349	GG
HCRTR2	rs4715517	CC
CUBN	rs45551835	GG
HOXD10	rs187355703	CC
ASAH2B	rs10994860	CC
PPP2R5A	rs2788144	AA
DNAJC13	rs113177823	GG
EZH2	rs148190310	GG
GSTM3	rs17024258	CC
MLXIPL	rs13247874	TT
HNF4G	rs2941484	CC
SFMBT1	rs2581824	CC
CYB5D1	rs78671965	AA

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

Ketone Bodies

As mentioned, the production of ketone bodies depends on sugar and fat metabolism. In line with this, genetic variants affecting sugar and fat metabolism may also influence the levels of ketone bodies [R, R].

For example, the variant rs780094-C is linked to higher levels of ketone bodies. This variant belongs to the *GCKR* gene, which plays a major role in sugar metabolism [R].

When the body can't use carbs well (e.g., due to diabetes), it 'burns' more fat and makes ketone bodies to compensate. The above variant is linked to higher odds of diabetes but lower blood fat levels. In other words, people with this variant may not burn carbs well, but they may be better at burning fat [R, R, R].

Keep in mind that your diet, environment, and other genetic variants also influence your levels of ketone bodies.

TYPICAL LEVELS

Predisposed to typical levels of ketone bodies based on 7 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SFT2D1	rs150515955	CC
GCKR	rs780094	CT
MLXIP	rs144305620	GG
OXCT1	rs11745373	AT
GALNT2	rs4846915	CA
SIDT2	rs964184	CC

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

Lactate

Lactate is a byproduct that your body normally produces, especially during exercise. When exercise becomes intense, your body starts burning sugar without using oxygen (anaerobic). This process spikes lactate levels. Professional athletes use blood lactate as markers of **fatigue and exercise performance** [R, R].

Besides exercise and conditions like diabetes, genetics may also influence your lactate levels. Most of the involved genes play a role in sugar metabolism [R].

For example, variants near the *GCKR* gene are linked to lactate levels, diabetes, and insulin resistance. This gene helps make a crucial enzyme for sugar metabolism [R, R].

Keep in mind that your diet, lifestyle, environment, and other genetic variants also influence your lactate levels.

TYPICAL LEVELS

Predisposed to typical lactate levels based on 11,956 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HBS1L	rs9376091	TT
SH2B3	rs7137828	CT
GCKR	rs1260326	CT
ZFPM2	rs6993770	AT
PFKP	rs10794972	CA
SPATS2L	rs59273177	AA
ALDH2	rs7138688	TT
TMX4	rs1473698	TT

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

Creatine

Up to 60% of differences in people's creatine levels may be due to genetics. Involved genes influence creatine synthesis and transport [R, R, R].

A low-protein or creatine-deficient diet, particularly in vegetarians or vegans, may lead to low creatine levels [R].

Low creatine levels may also indicate:

- Muscle diseases such as dystrophies or myopathies [R]
- Liver diseases such as cirrhosis or hepatitis [R]
- Genetic disorders such as GAMT or SLC6A8 deficiency [R, R]

Because creatine is stored in the muscles, people with a high muscle mass may have higher levels. Intense physical activity may also raise its levels due to the release of creatine from the muscles into the blood. This can also happen from trauma or conditions that cause muscle damage or wasting such as rhabdomyolysis [R, R].

Elevated creatine levels may also arise from excessive supplementation [R].

TYPICAL LEVELS

Likely typical creatine levels based on 11,877 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CPS1	rs1047891	AC
GATM	rs2486274	TG
CCDC77	rs7307754	CT

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

Metabolism Genes

While diet and lifestyle have a major influence on metabolism, genetics also plays a role by affecting appetite, fat storage, thyroid function, and insulin sensitivity. Understanding your genetic predispositions can help you tailor your approach to nutrition, exercise, and weight management for long-term metabolic health.

This section explores key metabolism-related genes, including those linked to appetite and weight regulation (such as FTO, LEPR, ADIPOQ, UCP1, and ADRB2), thyroid hormone production and metabolism (TPO, DIO1, DIO2), and blood sugar control and insulin response (such as FOXO1, TCF7L2, PPARGC1A, and FABP2). By gaining insight into your genetic profile, you can make more informed decisions to optimize your metabolism and overall well-being.

LOWER ACTIVITY

SLC30A8 (Zinc & Blood Sugar)

Predisposed to lower SLC30A8 activity

LOWER ACTIVITY

UCP1 (Weight)

Likely lower UCP1 activity

WORSE GENETICS

ADRB2 (Weight)

Likely worse ADRB2 genetics

HIGHER ACTIVITY

FOXO1 (Blood Sugar)

Likely higher FOXO1 activity

LOWER ACTIVITY

CDKN2B (Blood Sugar)

Likely lower CDKN2B activity

LOWER ACTIVITY

PPM1K (Blood Sugar/Diet)

Likely lower PPM1K activity

LOWER ACTIVITY

MADD (Blood Sugar & Insulin)

Predisposed to lower MADD activity

HIGHER ACTIVITY

MTNR1B (Diet & Blood Sugar)

Predisposed to higher MTNR1B activity

TYPICAL ACTIVITY

LEPR (Weight/Leptin Resistance)

Likely typical LEPR activity

TYPICAL GENETICS

ADIPOQ (Weight/ Blood Sugar)

Likely typical ADIPOQ genetics

TYPICAL ACTIVITY

UCP3 (Weight)

Likely typical UCP3 activity

TYPICAL ACTIVITY

ADRB3 (Weight)

Likely typical ADRB3 activity

TYPICAL ACTIVITY

CD36 (Fat Preference/Weight)

Likely typical CD36 activity

TYPICAL ACTIVITY

TFAP2B (Weight/Diet)

Likely typical TFAP2B activity

TYPICAL ACTIVITY

PPARG (Metabolism)

Likely typical PPARG activity

TYPICAL ACTIVITY

APOA2 (Weight, Blood Lipids)

Likely typical APOA2 activity

TYPICAL ACTIVITY

MC3R (Weight)

Likely typical MC3R activity

TYPICAL

TPO (Thyroid)

Likely typical TPO genetics

TYPICAL ACTIVITY

PPARGC1A (Fitness/Blood Sugar)

Likely typical PPARGC1A activity

TYPICAL

SLC2A2 (Sugar Intake)

Predisposed to typical sugar intake

BALANCED PREFERENCES

FGF21 (Carbs vs Fats)

Likely balanced macronutrient preferences

LOWER ACTIVITY

FABP2 (Blood Sugar/ Cardiovascular)

Likely lower FABP2 activity

TYPICAL ACTIVITY

GCKR (Blood Sugar)

Likely typical GCKR activity

TYPICAL ACTIVITY

GIPR (Blood Sugar)

Likely typical GIPR activity

TYPICAL ACTIVITY

PPARA (Keto Diet)

Likely typical PPARA activity

BETTER

FTO (Weight)

Likely better FTO genetics

HIGHER ACTIVITY

MC4R (Weight/ Blood Sugar)

Likely higher MC4R activity

BALANCED ACTIVITY

UCP2 (Weight)

Likely balanced UCP2 activity

HIGHER ACTIVITY

DIO1 (Thyroid)

Likely higher DIO1 activity

HIGHER ACTIVITY

DIO2 (Thyroid)

Likely higher DIO2 activity

SLC30A8 (Zinc & Blood Sugar)

One of the most studied *SLC30A8* variants is the SNP **rs13266634**. Research suggests that the 'C' allele is strongly linked to impaired zinc transport and type 2 diabetes [R, R, R].

For instance, a systematic review of 39 studies including data from over 165,000 individuals found that the 'C' allele was associated with a higher risk of type 2 diabetes in Asian and European populations. Those carrying one 'C' allele had a 16.5% higher risk of this condition and those carrying two copies had a 33% higher risk compared to those without this allele [R].

The **rs11558471** polymorphism may also affect zinc transport activity and the risk of type 2 diabetes. Three studies on Chinese, Punjabi, and Malay participants concluded that the 'A' allele is strongly associated with a higher risk of type 2 diabetes. According to one of these studies, patients were almost twice as likely to have the 'AA' genotype compared to those without diabetes [R, R].

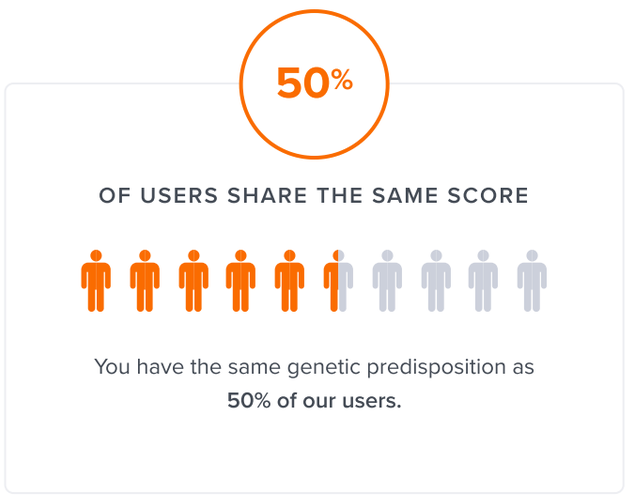
Based on a study of nearly 3,000 Chinese individuals, each copy of the 'G' allele of **rs3802177** is associated with a 17% higher risk of type 2 diabetes. The 'G' allele is also linked to impaired zinc transport and a higher risk of type 2 diabetes in Japanese and European groups, according to another study [R, R].

The three variants are usually inherited together, so you will typically carry all risk variants or none of them.

Some studies suggest that these variants reduce the effectiveness of the ZnT8 transporter. This would reduce the amount of zinc moving into insulin-producing structures. Genetic variants that decrease zinc transport activity may ultimately lead to lower insulin levels, higher insulin resistance, and a higher risk of type 2 diabetes [R, R].

LOWER ACTIVITY

Predisposed to lower SLC30A8 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SLC30A8	rs11558471	AA
SLC30A8	rs3802177	GG

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

UCP1 (Weight)

One of the best-studied SNPs in the *UCP1* gene is rs1800592 (also known as the "-3826 A>G" polymorphism). It helps determine how your body uses and stores the energy that you get from food [R].

The 'T' allele is linked to increased activity of the *UCP1* gene. It's associated with a higher resting metabolic rate, higher body heat production, and less weight gain. According to some researchers, this variant helps turn more of the energy from food into heat instead of body fat (white fat) [R, R].

Conversely, the 'C' allele is linked to *decreased* activity of the *UCP1* gene. It's associated with a lower resting metabolic rate, lower body heat production, higher weight gain, and a higher BMI. If less of the energy acquired from food is turned into heat, then more of it would get stored as body fat [R, R].

According to several studies, the 'C' allele (and especially the 'CC' genotype) is associated with increased weight gain as well as a higher chance of being obese [R, R, R, R, R, R, R, R].

For example, people with the 'CC' genotype were found to have lower basal metabolic rates than people with the 'T' allele. In other words, they burned less energy when resting. In fact, one study reported that 'C' carriers may burn as much as 200 fewer calories per day than people with the 'TT' genotype [R, R]!

Apart from burning less energy when resting, people with the 'CC' genotype also produced less heat when exposed to cold [R, R].

We all lose brown fat as we age. However, in one study, people with the 'CC' genotype had less brown fat at a younger age than people with the 'T' allele [R].

Several studies link the 'C' allele and the 'CC' genotype to metabolic disturbances commonly associated with being overweight. In various studies, the 'C' allele has been associated with elevated blood pressure, greater insulin resistance, and higher LDL cholesterol and triglycerides [R, R, R, R, R, R].

Fun fact: worldwide, about 30% of people have the 'TT' genotype, which is associated with higher resting metabolism and increased heat production. But this genotype is much more frequent in Europe, where 58% of people have it! Many researchers believe that the *UCP1* gene and the rs1800592 SNP are in part responsible for human adaptation to colder climates [R].

However, although today we consider the 'T' allele beneficial in terms of its potential effect on body weight, this allele is essentially linked to lower metabolic efficiency. In other words, people with this allele may "waste" more of the energy that they get from food on generating body heat. It is plausible that the more efficient 'C' allele may be advantageous when food is scarce and the climate is warm [R].

LOWER ACTIVITY

Likely lower UCP1 activity based on the genetic variants we looked at

42%

OF USERS SHARE THE SAME SCORE

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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
UCP1	rs1800592	TC

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

ADRB2 (Weight)

The **rs1042714** variant (also known as Q27E or Gln27Glu) has been most widely researched when it comes to weight. Its minor **'G' allele** (Glu, E) was associated with approximately 20% higher odds of **obesity** in a meta-analysis of 18 studies [R].

An older meta-analysis came to a similar conclusion. However, the authors observed a significant link between rs1042714-G and obesity only in populations with lower frequencies of this allele, such as **Asians and Native Americans**. In two studies with 150 women, those with the 'G' allele had more fat mass and impaired burning [R, R, R].

This variant has also been linked to:

- Reduced exercise performance and VO2 max [R]
- Increased risk of cardiovascular events in coronary artery disease patients [R]
- Increased risk of Graves' disease in Caucasians [R]

On the bright side, people with this variant tend to **lose weight more easily** when they reduce calorie intake. However, given the link of this variant with obesity under regular conditions, there is a potential risk of **weight regain** ("yo-yo" effect) after dieting [R, R].

This variant may be linked to lower asthma odds in adults and children [R].

Normally, leptin helps burn excess fat stores by stimulating the sympathetic activity in fat tissue. However, according to one clinical trial, this pathway may be suppressed in people with the above SNP. The 'G' allele carriers had higher leptin levels, indicating leptin resistance [R].

Interestingly, this SNP doesn't seem to alter the function of beta-2 receptors. Scientists are still looking for the exact mechanism behind its effects on sympathetic activity and fat burning. It may be just a marker for another variant with functional consequences [R].

WORSE GENETICS

Likely worse ADRB2 genetics based on the genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ADRB2	rs1042714	CG

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

FOXO1 (Blood Sugar)

The ‘GG’ genotype of rs10507486 has been associated with 27% higher odds of developing insulin resistance compared to ‘AA’. Additionally, the ‘A’ variant has been linked to decreased risk of atherosclerosis [R, R].

Two other variants, ‘A’ at rs7986407 and ‘C’ rs4581585, have been associated with an increased risk of type 2 diabetes. Compared to rs10507486, rs7986407 and rs4581585 are much more strongly linked to insulin resistance, each capable of more than doubling your risk [R].

A more recent study associated the ‘A’ variant of rs17446614 with an increased risk of diabetes, as well as with higher fasting glucose and HbA1c [R].

These variant may have increased FOXO1 activity. According to a series of mouse studies, over-active FOXO1 prevents the pancreas from producing enough insulin and responding appropriately to changes in blood glucose [R, R].

Variants of this gene have been associated with increased longevity. However, the results are mixed and may depend on the ethnicity [R, R, R].

HIGHER ACTIVITY

Likely higher FOXO1 activity based on 4 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MRPS31	rs17446614	GG
MRPS31	rs7986407	AA
MRPS31	rs4581585	CC
MRPS31	rs10507486	GG

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

CDKN2B (Blood Sugar)

Two studies (the largest one with 62,892 type 2 diabetes cases and 596,424 controls) done in individuals of European ancestry identified the minor ‘C’ allele of **rs10811661** as decreasing fasting glucose and the risk of type 2 diabetes. Another study on 48,437 individuals of South Asian ancestry and 20,298 of European descent confirmed these associations. In line with this, the major variant has been associated with impaired insulin release and glucose tolerance [R, R, R, R].

Moreover, people with the ‘C’ allele may experience a far greater benefit to blood sugar when they exercise than people without it [R, R].

This variant has also been associated with a decreased risk of:

- Obesity [R]
- Breast cancer [R]
- Intracranial aneurysm [R]

One of the above-mentioned meta-analyses also identified the minor ‘G’ allele of **rs1063192** among those decreasing fasting glucose and the risk of type 2 diabetes [R].

This variant has also been linked to a reduced risk of:

- Glaucoma [R]
- Myocardial infarction [R]

Based on their association with lower blood sugar and risk of diabetes, these variants may increase *CDKNB2* activity.

LOWER ACTIVITY

Likely lower CDKN2B activity based on the genetic variants we looked at

61%

OF USERS SHARE THE SAME SCORE

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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CDKN2A	rs10811661	TT
CDKN2B	rs1063192	AA

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

PPM1K (Blood Sugar/Diet)

Studies show that certain genetic variants of *PPM1K* may have negative effects on the body.

More specifically, research suggests that the ‘C’ allele in **rs1440581** may be associated with an increased risk of type 2 diabetes and poor response to certain diets [R, R, R].

According to researchers, this occurs because the ‘C’ allele may impair the body’s ability to metabolize BCAAs, leading to a higher-than-normal level of BCAAs. High levels of BCAAs in the blood have been strongly linked to insulin resistance and type 2 diabetes in a number of studies [R, R, R].

A study of 6,000 Chinese non-diabetic participants found that an increase in BMI resulted in greater increases in insulin levels and insulin resistance in those with the ‘C’ allele in rs1440581. The researchers also found that each copy of the ‘C’ allele was associated with a 20% higher risk of type 2 diabetes [R].

In the POUNDS LOST trial, subjects who were on a calorie-restricted, high-fat diet and carried the ‘C’ allele in rs1440581 had less weight loss and smaller improvements in insulin resistance compared to those with the ‘T’ allele [R].

Similarly, those carrying the ‘C’ allele experienced *worse* improvements in weight and insulin resistance while on a calorie-restricted high-fat diet in the NUGENOB trial [R].

To sum up, the ‘C’ allele of rs1440581 is associated with higher insulin levels, insulin resistance, and risk of type 2 diabetes. Carriers may lose less weight from a calorie-restricted, high-fat diet.

LOWER ACTIVITY

Likely lower PPM1K activity based on the genetic variants we looked at

26%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **26% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PPM1K	rs1440581	CC

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

MADD (Blood Sugar & Insulin)

The main MADD variant is rs7944584. Its **“A” allele** is linked to **higher** blood sugar levels and diabetes risk [R].

The MADD gene plays a critical role in the regulation of insulin secretion and proinsulin processing in pancreatic beta cells. Proinsulin, the precursor to insulin, must be cleaved into mature insulin and C-peptide to enable proper glucose regulation.

The A allele of rs7944584 may **reduce MADD expression or function**, impairing the efficient conversion of proinsulin to insulin. This could result in **elevated proinsulin** relative to insulin, a hallmark of beta-cell dysfunction often seen in type 2 diabetes (T2D) [R, R].

On the other hand, this variant is linked to lower blood pressure and better mood [R].

The mechanism behind these contradictory findings isn’t yet clear. The MADD gene has tissue-specific roles. In pancreatic beta cells, this variant may impair insulin production. However, the same variant might have detrimental effects in vascular or neural tissues, such as increasing blood pressure or altering mood regulation.

This highlights the concept of **pleiotropy**, where a single gene influences multiple, seemingly unrelated traits.

LOWER ACTIVITY

Predisposed to lower MADD activity based on the genetic variants we looked at

54%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **54% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MADD	rs7944584	AA

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

MTNR1B (Diet & Blood Sugar)

Recent years have brought fascinating insights into how our internal clock affects metabolism, particularly through the melatonin receptor gene MTNR1B. One genetic variant in this gene - **rs10830963** - has emerged as a key player in the connection between sleep timing and blood sugar control.

The **minor "G" allele** has one of the strongest links with **high blood sugar and type 2 diabetes**. It increases the expression of melatonin receptors in pancreatic beta cells. These beta cells release insulin, and when they have more melatonin receptors, they become more sensitive to melatonin's signals [R, R].

Here's where timing becomes crucial: **Melatonin naturally suppresses insulin release** - a useful feature during our normal sleeping hours when we're not eating. However, people carrying the G allele have heightened sensitivity to this effect. For these individuals, **eating late at night can lead to a reduced insulin response and higher blood sugar levels** [R].

New research has uncovered something unexpected: carriers of this variant produce melatonin for about 41 minutes longer than non-carriers, and their melatonin offset (when melatonin levels drop in the morning) is **delayed by about 80 minutes** [R].

This finding has important implications, particularly for early risers. If you carry this variant and wake up early, you might still have elevated melatonin levels when you eat breakfast. Since melatonin suppresses insulin release, this could lead to **higher blood sugar levels during your morning meal**, contributing to diabetes [R].

Taken together, these studies suggest that people with rs10830963-G may particularly benefit from **intermittent fasting**. By avoiding late dinners and early breakfasts, you lessen the negative impact of melatonin on blood sugar control [R].

HIGHER ACTIVITY

Predisposed to higher MTNR1B activity based on the genetic variants we looked at

50%

OF USERS SHARE THE SAME SCORE

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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MTNR1B	rs10830963	CG

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

LEPR (Weight/Leptin Resistance)

Many *LEPR* variants are currently under investigation for their possible link to leptin resistance and obesity. The most important one is **rs1137101**.

At rs1137101, the 'GG' genotype is associated with higher weight, higher BMI, and increased daily intake of calories. People with the 'GG' genotype at rs1137101 are also likely to have higher cholesterol, higher blood sugar, and insulin resistance [R].

A recent meta-analysis has confirmed a link between this variant and obesity. The risk was **19% higher per each "G" allele** [R].

This variant likely reduces the number or activity of leptin receptors, potentially contributing to leptin resistance [R, R].

Studies have linked another variant, **rs12405556-T**, to higher weight and blood lipid levels. This variant is often inherited together with rs1137101-G, meaning many people carry either both or none of them [R].

Other LEPR SNPs—like rs1137100, rs11208659, and rs11804091—have been linked to weight gain and obesity as well. However, the supporting evidence is weaker [R, R].

TYPICAL ACTIVITY

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

46%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **46% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
LEPR	rs1137101	AG

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

ADIPOQ (Weight/ Blood Sugar)

The minor **'T' variant of rs1501299** was associated with **increased BMI and risk of obesity** in several studies on different populations, especially those of Caucasian ethnicity. Similarly, obese carriers of this variant had **higher fat percentage** in a Swedish study [R, R, R, R, R, R, R, R, R, R, R].

However, some studies didn't find this link [R, R, R, R, R, R].

The 'T' variant has been associated with increased **insulin resistance, type 2 diabetes, and metabolic syndrome** in Italian, Indian, and Spanish studies [R, R, R, R].

Similarly, the **'A' allele of rs17300539** showed a link with impaired weight and blood sugar control but with some mixed evidence [R, R, R, R, R, R, R, R].

The minor **'G' variant of the rs2241766** polymorphism is linked to:

- Reduced glucose tolerance and high blood sugar [R, R]
- Type 2 diabetes [R, R, R]
- Metabolic syndrome [R, R]

Some studies found a link between this variant and obesity and belly fat in different populations. However, many other studies found opposite effects or no effects [R, R, R, R, R, R, R, R, R].

Another **rs266729** variant has been studied for its links with weight and blood sugar control, but the studies showed mixed results. Some studies found the negative effects of the **"G" allele,** but many found no effects or opposite effects [R, R, R, R, R, R, R, R].

Studies on the link between these variants and adiponectin levels also showed inconsistent results. It's unclear whether the observed effects are due to changes in adiponectin levels, and in which direction [R, R, R, R, R, R, R].

TYPICAL GENETICS

Likely typical ADIPOQ genetics based on 4 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ADIPOQ	rs2241766	GT
RFC4	rs266729	GC
ST6GAL1	rs1501299	GG
RFC4	rs17300539	GG

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

UCP3 (Weight)

The most widely-investigated *UCP3* polymorphism by far is rs1800849, also known as -55C/T. A study in Pima Indians found that the minor allele 'A' increases *UCP3* expression, and possibly the levels of the protein, in the muscles [R].

Some studies suggest that the minor variant at this polymorphism may protect from obesity, especially in Caucasians. The mixed results observed may be due to differences in the diet, physical activity, and genetic background of the different populations. Importantly, different *UPC2* and *UPC3* variants are inherited together and their combinations may influence their effects on weight [R, R, R, R].

This variant has been associated with a higher waist circumference, waist to hip ratio, and abdominal fat in multiple populations. However, a Spanish study found no relationship between this polymorphism and fat mass or distribution in obese. Another study on American women associated it with increased lean mass and calorie intake [R, R, R, R, R, R].

Two meta-analyses confirmed the association of the 'A' variant with increased susceptibility to type 2 diabetes in Asians but not in Europeans [R, R].

The minor variants at the rs647126 and rs2075577 polymorphisms, which are usually inherited together, were associated with increased BMI in Dutch men. Based on their effect, the authors of the study speculated that these variants may reduce UCP3 activity [R].

TYPICAL ACTIVITY

Likely typical UCP3 activity based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
UCP3	rs1800849	GG
UCP2	rs647126	GA
UCP2	rs2075577	AG

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

ADRB3 (Weight)

Over 100 studies have examined the relationship between one *ADRB3* variant—**rs4994** or Trp64Arg— and body-weight measures. The A>G switch at rs4994 changes one amino acid in the beta-3 receptor structure. The “mutant” receptor had a reduced ability to produce cAMP and burn fat in test tubes [R, R, R].

One meta-analysis included 97 studies, involving 44,800 participants. Among East Asians, those with the "G" allele had, on average, 0.31 units higher body mass index, which would equal 0.8-1 kg. In European descendants, the difference was four times smaller and wasn't statistically significant [R].

The same group of authors conducted the largest study of 4,854 European (UK) subjects and confirmed the lack of association between this SNP and BMI [R].

In a meta-analysis of 16 studies and 12,500 children and adolescents, rs4994-G correlated with 23% higher obesity rates. Once again, the effect stemmed from East Asian subjects, who had 47% higher odds of obesity per copy of the "G" allele [R].

A study of 329 adults from Saudi Arabia found a significant link between this SNP and obesity. People with the "G" allele also had a higher waist-hip ratio, blood lipids, leptin, and insulin levels [R].

Research has associated the same variant with other conditions, such as:

- Diabetes [R, R]
- High blood pressure [R]
- Heart disease [R]

Those genetic effects were also more pronounced in East Asians.

TYPICAL ACTIVITY

Likely typical ADRB3 activity based on the genetic variants we looked at

85%

OF USERS SHARE THE SAME SCORE

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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ADRB3	rs4994	AA

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

CD36 (Fat Preference/Weight)

The CD36 receptor has a complicated relationship with diet and may affect everything from taste preferences to fat metabolism [R].

The ‘G’ allele at **rs1761667** is associated with a number of metabolic changes compared to the ‘AA’ genotype. For example, people with at least one copy have increased appetite and food intake. However, the ‘G’ allele is also associated with lower BMI [R, R, R].

The ‘A’ allele, meanwhile, is associated with reduced production of the CD36 receptor, increased preference for fatty foods, and increased BMI [R, R].

TYPICAL ACTIVITY

Likely typical CD36 activity based on the genetic variants we looked at

48%

OF USERS SHARE THE SAME SCORE

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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CD36	rs1761667	GA

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

TFAP2B (Weight/Diet)

Variations in the *TFAP2B* gene have been associated with increased weight and belly fat and type 2 diabetes. The majority of obesity and diet-related research focused on a SNP labeled **rs987237**. Its minor "G" allele may increase *TFAP2B* expression in fat cells [R, R, R].

In a huge meta-analysis of over 195,000 people, the "G" allele was associated with a higher body mass index (BMI) [R].

The "G" allele was associated with 24% obesity rates in a meta-analysis that included 34,600 European participants. Another large review of 16 trials found a link between this variant and waist circumference, which is a measure of abdominal obesity [R, R].

In a study of 642 obese adults, those with the "GG" genotype lost 2.6 kg more on a high-fat diet, compared with a low-fat diet. The "AG" carriers also preferred a high-fat diet, but the effect was less significant. On the other hand, people with the "AA" did better on a low-fat diet. The authors also discovered a similar but weaker pattern for waist size reduction [R].

One trial analyzed weight maintenance in 468 European people who completed a weight loss program. Those following a high-protein diet regained 1.84 kg more per each "G" allele. On the other hand, the "AA" carriers did better on a high vs. low-protein diet [R].

To sum up, the "G" allele is associated with obesity. As opposed to those with the "AA" genotype, "G" carriers may do better on a high-fat diet compared to a high-protein diet for losing weight and maintaining weight loss.

TYPICAL ACTIVITY

Likely typical TFAP2B activity based on the genetic variants we looked at

67%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **67% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TFAP2B	rs987237	AA

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

PPARG (Metabolism)

Out of the different SNPs in the *PPARG* gene, researchers have mostly focused on rs1801282 (*Pro12Ala,* referred to as rs1805192 in some studies). Its 'G' allele changes one amino acid in the PPAR-γ structure, reducing its ability to activate target genes [R, R].

In a large meta-analysis of 75 studies and 49,000 subjects, the 'G' allele correlated with a slightly higher BMI. The link was more robust in European populations. A 2015 meta-analysis of 56 trials came to a similar conclusion. Another meta-analysis associated the 'G' allele with 55% higher obesity rates [R, R, R].

However, some studies failed to confirm a relationship between this variant and body weight, and some even observed a protective effect of the 'G' allele [R, R, R, R, R].

Among 978 elderly subjects, rs1801282-G correlated with 66% higher obesity rates. The lack of physical activity and increased intake of carbs amplified this genetic effect [R].

Interestingly, three studies found that people with rs1801282-G respond better to physical activity when it comes to metabolic improvements [R, R, R].

This variant may also affect weight loss from the Mediterranean diet. Several studies found that 'G' carriers lost more weight when eating a Mediterranean diet rich in MUFAs, PUFAs, and extra virgin olive oil but lost less or even gained it when eating a diet low in these and high in saturated fats [R, R, R, R, R].

PPAR-γ can be a double-edged sword when it comes to regulating blood sugar levels. While its activity in the liver and pancreas enhances glucose metabolism and insulin sensitivity, excess PPAR-γ activity in fat cells can accumulate fatty acids and other factors that contribute to insulin resistance and elevated blood sugar [R, R, R, R].

This may explain why the 'C' variant has been associated with higher blood sugar and increased rates of type 2 diabetes. In one of the studies, obesity further increased the risk of type 2 diabetes associated with this variant [R, R, R, R].

Two other variants, 'C' at rs1899951 and 'C' at rs17036160, have also been associated with high blood sugar and type 2 diabetes. However, they act as a single genetic factor because the three variants are usually inherited together [R, R].

Finally, the 'G' variant of rs1801282 has also been associated with reduced rates and severity of acne [R, R].

TYPICAL ACTIVITY

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

77%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **77% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PPARG	rs1801282	CC

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

APOA2 (Weight, Blood Lipids)

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

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

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

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

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

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

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

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

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

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

TYPICAL ACTIVITY

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

85%

OF USERS SHARE THE SAME SCORE

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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
FCER1G	rs5082	AA

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

MC3R (Weight)

Scientists have identified two major SNPs in the MC3R gene with a potential impact on body-weight measures: **rs3746619** (Thr6Lys) and **rs3827103** (Val81Ile). They are almost always inherited together, and the studies often refer to them in pairs.

The minor **"A" alleles** at rs3746619 and rs3827103 correlate with **higher weight and fat mass** in children, mostly of African and Asian descent [R, R, R, R].

Among 237 African American adults, those with the pair of "AA" genotypes had higher average BMI (37.6 vs. 35.2) and fat mass (45 vs. 39.7 kg) [R].

However, other studies have mostly **failed to establish a connection** between these SNPs and obesity in adults [R, R, R, R].

The obesity-associated "AA" genotypes are **scarce among European descendants**, so most trials had insufficient sample sizes to investigate these variants.

The "AA" genotypes at rs3746619 and rs3827103 change two amino acids in the MC3R structure. In test tubes, scientists found that this combination reduces receptor expression and activity [R, R].

Clinical studies have confirmed the link between the "A" alleles, **excess leptin, and suppressed fat oxidation** [R, R, R, R].

Did you know? The reduction in MC3R activity has likely brought an evolutionary advantage to people during starvation. They were able to build fat stores more efficiently, which increased their chances of survival. This phenomenon is called the thrifty gene hypothesis and may be responsible for a much higher frequency of the "A" allele in African populations [R].

TYPICAL ACTIVITY

Likely typical MC3R activity based on the genetic variants we looked at

75%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **75% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MC3R	rs3746619	CC
MC3R	rs3827103	GG

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

TPO (Thyroid)

In a trial of 500 European subjects, one SNP in the *TPO* gene showed a significant association with Hashimoto's disease (HD). This form of autoimmune hypothyroidism was 31% more common among people with the minor 'T' allele at rs11675434 [R].

In 1400 Polish subjects, the same variant correlated with 64% higher rates of eye complications in Graves' disease patients (Graves' ophthalmopathy). This group of authors conducted further research on over 2,300 and found the significant impact of rs11675434 only in older male patients [R, R].

Comprehensive studies have confirmed the link between this variant and anti-TPO antibody positivity. In over 18,000 European subjects, those with rs11675434-T were 21% more likely to be positive and also had higher antibody levels [R].

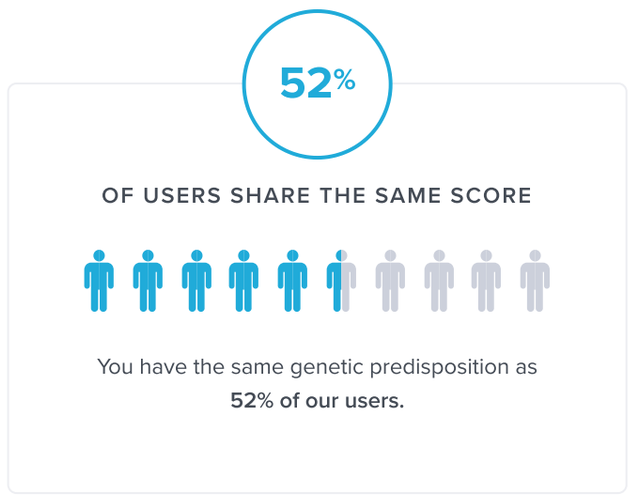
A clinical trial of 400 Indian subjects identified another *TPO* variant in connection with thyroid health. Carriers of the 'C' allele at rs732609 (Thr725Pro) had 45% higher rates of hypothyroidism [R].

Doctors from Iran came to a similar conclusion after conducting a small study of 150 participants. The 'C' allele correlated with subclinical or "hidden" hypothyroidism [R].

The 'C' allele of rs732609 changes one amino acid (threonine to proline) in the TPO enzyme, which may interrupt its secondary structure. The immune system can mistakenly flag such enzymes as foreign structures and produce antibodies to destroy them [R].

TYPICAL

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TPO	rs11675434	CT
TPO	rs732609	AC

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

PPARGC1A (Fitness/Blood Sugar)

The most well-researched *PPARGC1A* polymorphism is **rs8192678**. Its minor 'T' allele decreases *PPARGC1A* expression and PGC-1α levels in the muscles [R, R, R].

This variant has been associated with a decreased overall athletic ability and sports performance, especially in endurance sports such as long-distance running and cycling. Moreover, carriers may benefit less from aerobic exercise for improving their aerobic capacity, gaining muscle mass, and lowering LDL cholesterol [R, R, R].

This variant has been associated with an increased risk of type 2 diabetes in European, Indian, and Chinese populations, as well as with higher blood pressure in individuals younger than 50 years old [R, R, R, R].

It may also be linked to **worse cold adaptation** due to reduced PGC-1α levels and impaired mitochondrial function.

In contrast, a study of 161 Caucasian athletes from Russia and Lithuania found an increased prevalence of the 'TT' genotype among powerlifters [R].

TYPICAL ACTIVITY

Likely typical PPARGC1A activity based on the genetic variants we looked at

45%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **45% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PPARGC1A	rs8192678	CT

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

SLC2A2 (Sugar Intake)

A study of two distinct populations linked the SLC2A2 variant **rs5400** with appetite for sugary foods.

The first group of individuals, 194 men and women over 45 with pre-diabetic symptoms, had **increased sugar intake if they carried an "A" allele** [R].

This finding was replicated in a group of 700 healthy men and women aged 20-29 [R].

This variant is also linked to [R]:

- Higher cholesterol and triglyceride levels
- Liver health issues (higher GGT and bilirubin)
- Higher body weight (BMI)

Interestingly, it is also linked to **lower** blood sugar levels and lower odds of diabetes [R].

A potential explanation for these seemingly contradictory findings is that this variant might have different effects on GLUT2 activity in different tissues. In the brain, it may reduce its activity and thus make sugar "less sweet", increasing a person's hunger for it [R].

On the other hand, the variant might increase GLUT2 activity in the liver and pancreas, leading to enhanced glucose uptake and metabolism. This could explain lower blood sugar but higher blood lipids, liver enzymes, and body weight.

However, scientists have yet to confirm the exact mechanism behind these findings.

TYPICAL

Predisposed to typical sugar intake based on the genetic variants we looked at

75%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **75% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SLC2A2	rs5400	GG

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

FGF21 (Carbs Vs Fats)

A 2018 study analyzed the data from over 450,000 people and found a significant association between one *FGF21* SNP and macronutrient preference. Carriers of the minor “A” allele at **rs838133** consumed less fat and more carbohydrates. Additionally, these subjects had lower body fat but higher waist-hip ratio and blood pressure [R].

Another trial of 38,000 participants confirmed the link between rs838133 and lower fat/higher carb preference [R].

One more SNP in this gene showed a similar trend in a study of 33,500 subjects. People with the minor “G” allele at **rs838145** consumed less fat and more carbs [R].

BALANCED PREFERENCES

Likely balanced macronutrient preferences based on the genetic variants we looked at

30%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **30% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
FGF21	rs838133	GA
FGF21	rs838145	AG

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

FABP2 (Blood Sugar/ Cardiovascular)

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

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

This variant has also been associated with:

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

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

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

LOWER ACTIVITY

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

56%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **56% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
FABP2	rs1799883	CC

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

GCKR (Blood Sugar)

The main *GCKR* variants are:

- **rs1260326**
- **rs780093**
- **rs780094**

They are almost always inherited together, meaning you will most likely have either all or none of them. Their **"C" alleles** may be linked to **higher blood sugar levels and diabetes** [R, R, R].

On the positive side, these variants are linked to lower levels of cholesterol and other blood lipids [R].

These variants may **increase** the production or activity of the glucokinase regulator, and thus inhibit the actions of glucokinase. This can result in elevated blood sugar levels — and lower blood lipids — by **decreasing insulin release** in response to meals [R, R].

TYPICAL ACTIVITY

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

50%

OF USERS SHARE THE SAME SCORE

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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GCKR	rs780093	CC
GCKR	rs1260326	CT
GCKR	rs780094	CT

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

GIPR (Blood Sugar)

The main variants in the *GIPR* gene are **rs2287019 and rs10423928**. They are almost always inherited together, meaning you will most likely have either both or none of them.

The minor alleles, rs2287019–**T** and rs10423928–**A**, are strongly linked to [R, R, R, R]:

- **Type 2 diabetes**
- **Insulin resistance**
- **Gestational diabetes**

On the other hand, they have shown associations with reduced body weight [R, R].

These variants may **reduce insulin secretion** after a meal. This may impair blood sugar control and contribute to diabetes but also promote fat burning [R, R].

Interestingly, in one study, people with these variants did better on a **low-fat, high-carb, high-fiber diet**. Researchers are unsure about the underlying mechanism behind this finding [R].

TYPICAL ACTIVITY

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

64%

OF USERS SHARE THE SAME SCORE

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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GIPR	rs2287019	CC
GIPR	rs10423928	TT

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

PPARA (Keto Diet)

So far, the most well-studied PPARA SNP in nutrigenomics is rs1800206. **Its minor 'G' allele (also known as the *L162V polymorphism*) reduces PPAR-α activity**, according to the available data [R].

Carrying this allele has been associated with [R]:

- Increased heart disease risk in whites, with higher levels of triglycerides, total cholesterol, LDL, apoA1, and apoB, and decreased levels of HDL
- Possible, but uncertain impact on diabetes development

The negative effects of this genotype seem to be more pronounced in men than in women, and in whites than in Asians. Data on other ethnicities are sparse [R].

Moreover, **people carrying at least one 'G' allele who consumed high amounts of saturated fat had smaller LDL particles than those with lower intakes** in a study. Based on this, **they don't seem to be well suited for a ketogenic diet high in saturated fat** [R].

The opposite may be true for 'CC' carriers. Among people with this genotype, those with higher saturated fat intakes had larger LDL particles than those with lower saturated fat intakes [R].

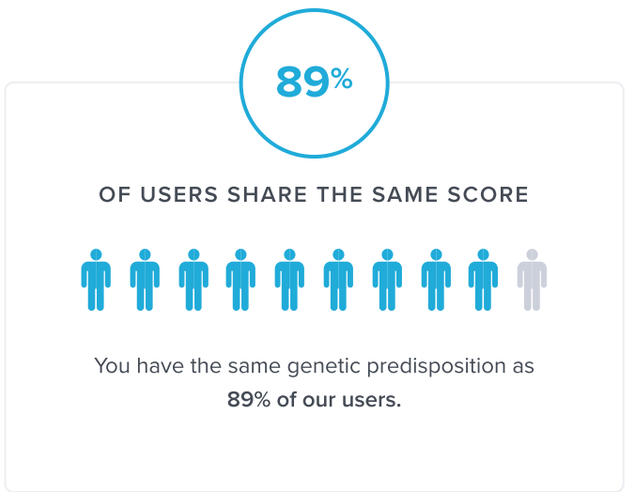
A study of 2373 participants found an association between the 'G' allele and higher total and LDL cholesterol in men and apolipoprotein B in both genders. The LDL cholesterol link was even stronger in carriers of the "good" E2 *APOE* allele [R].

A follow-up study of 2106 people from the same cohort concluded that **'G'-allele carriers had greater triglyceride and apoC-III levels when they consumed a low-PUFA diet.** But when their PUFA intake was high, they had *lower* triglyceride and apoC-III levels. The authors pointed out that the more PUFAs 'G'-allele carriers ate, the more their triglycerides and apoC-III levels dropped--and vice versa [R, R].

Theoretically, a higher PUFA intake might make up for lower PPARA activity in 'G'-allele carriers. A diet high in omega-3 PUFAs is still the healthiest choice even for people carrying the more common 'CC' genotype, but these subjects might be less prone to triglyceride spikes if their diet happens to be a bit lower in PUFAs, as most diets that rely on animal fat are [R].

TYPICAL ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PPARA	rs1800206	CC

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

FTO (Weight)

An SNP in this gene, rs9939609, has shown a robust association with obesity across different ages and ethnic groups. Carriers of the minor 'A' allele tend to gain more weight and have higher rates of obesity [R, R, R, R].

Many human studies suggest that the 'A' allele at rs9939609 is associated with:

- Higher levels of *ghrelin* or the "hunger hormone" [R]
- Higher food intake [R, R, R]
- Increased preference for higher-calorie foods [R, R, R]
- Increased enjoyment of food [R]
- Not feeling full after meals [R, R, R]
- Eating in the absence of hunger [R, R]
- Food cravings [R]
- Emotional and binge eating [R]

In contrast, the 'T' allele is linked to normal body weight, more satiety after meals, and possibly healthier dietary choices [R, R, R].

In addition to its potential influence on appetite and hunger control, this *FTO* SNP may also have metabolic effects that affect how the body actually processes the food we eat. For example, several studies suggest that the 'A' allele of rs9939609 may be linked with higher insulin resistance and blood sugar [R, R, R, R].

Following on this, a meta-analysis of data from over 40,000 Scandinavians found that people diagnosed with type-2 diabetes were more likely to carry the ‘A’ allele for this SNP. The authors also concluded that having the ‘A’ allele may make a person more likely to develop type-2 diabetes over time. However, another meta-analysis of over 150,000 people found this association significant only in some populations [R, R].

The good news is that **healthy dietary choices may reduce and even cancel out the harmful effects of the ‘A’ variant!**

Multiple studies have associated an increased intake of dietary fat, specifically saturated and trans fat, with obesity in carriers of the ‘A’ allele [R, R, R, R, R, R, R].

Carriers of this variant eating a low-fat diet or replacing saturated fat with healthy fats such as PUFA and MUFA may see greater reductions in [R, R, R, R, R]:

- Weight and abdominal fat
- Total cholesterol
- LDL
- CRP (inflammation)
- Insulin resistance

Additionally, carriers of the ‘A’ variant may benefit from increasing their intake of dietary protein. While a low-protein diet has been associated with higher BMI and waist circumference in people with the ‘AA’ genotype, ‘A’ carriers eating a high-protein diet may see greater improvements in their [R, R, R]:

- Appetite and food craving control
- Total cholesterol
- Triglycerides
- LDL
- Insulin resistance

However, two meta-analyses failed to confirm these associations. One of them even came to the opposite conclusion: the link between FTO variants and BMI was **stronger**

BETTER

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

37%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **37% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
FTO	rs9939609	TT

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

in those who consumed more protein. Further analysis of different populations confirmed this finding only in whites [R, R].

A 3-year-long study concluded that people with the 'AA' genotype may lose weight better on a Mediterranean diet compared to people with the ‘TT’ genotype. In addition, according to another study, this type of diet may protect people with the 'AA' genotype against diabetes [R, R].

MC4R (Weight/ Blood Sugar)

The most studied SNP near the *MC4R* gene is **rs17782313**.
The **"C" allele** is linked to:

- Higher BMI (8%) and obesity rates (12-30%) [R, R]
- Increased hunger, snacking, and overeating [R]
- Eating high-calorie foods high in fat [R, R]

Another important *MC4R* variant is **rs12970134**. The **“A” allele** is linked to:

- Obesity, higher BMI, and waist circumference [R, R, R]
- Food cravings and increased beverage consumption [R]
- High blood sugar and insulin resistance [R, R, R, R]

Other *MC4R* variants like **rs6567160** and **rs663129** have shown similar associations, They are almost always inherited together with the two main variants, so they all represent a single genetic factor [R, R, R, R, R].

“Bad” *MC4R* variants likely **reduce gene expression or receptor activity**, thus increasing food intake and hindering glucose and fat metabolism.

Learn more about the link between MC4R variants, weight, and food intake.

Learn more about the link between MC4R variants and blood sugar.

HIGHER ACTIVITY

Likely higher MC4R activity based on the genetic variants we looked at

55%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **55% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MC4R	rs17782313	TT
MC4R	rs12970134	GG

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

UCP2 (Weight)

The minor ‘T’ variant of the rs659366 increases *UCP2* expression in the fatty tissue, pancreas, and liver [R, R, R].

This allele showed a protective effect from obesity in studies on Austrian and Iranian adults, and Turkish children. Similarly, the major ‘C’ allele was associated with obesity in Danish and Egyptian adults, and Korean, Hungarian, and Spanish children [R, R, R, R, R, R, R, R].

However, no association between this polymorphism and obesity was found in some studies on Danish and Italian adults, and German children. The ‘T’ variant was even associated with *increased* obesity rates and body fat in Danish, Egyptian, Russian, Spanish, Mexican, Balinese, Indonesian, and Indian adults, and Spanish children [R, R, R, R, R, R, R, R, R, R, R, R, R].

Despite these discrepancies, all meta-analyses agree that the ‘T’ variant is associated with lower obesity rates and BMI in European but not in Asian populations [R, R, R, R].

However, this variant may have negative effects on blood lipids, glucose control, and diabetic outcomes [R, R, R, R, R, R, R, R, R].

Another variant usually inherited with this one (‘A’ at rs660339) has been associated with obesity in Asians but seems to have the opposite effect in Europeans [R, R, R].

BALANCED ACTIVITY

Likely balanced UCP2 activity based on the genetic variants we looked at

40%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **40% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
UCP2	rs659366	TC

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

DIO1 (Thyroid)

Based on the data from over 3,000 individuals, the researchers identified one variant, rs2235544, that correlates with low thyroid hormones. More precisely, carriers of the “A” allele have lower levels of T3, which is the active form of thyroid hormones [R].

Carriers of the "A" allele on rs2235544 have reduced expression of *DIO1* (compared with the “C” allele), which may lower the activity of thyroid hormones. People with low T3 can experience the symptoms of hypothyroidism despite having normal, or even slightly elevated T4 levels [R, R, R].

Studies have identified another variation on the same gene, rs11206244, but its effects are less significant and stem from a correlation with the SNP mentioned above [R, R].

HIGHER ACTIVITY

Likely higher DIO1 activity based on the genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
DIO1	rs2235544	CC

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

DIO2 (Thyroid)

At first glance, one might think DIO2 doesn't have much of an impact on thyroid health. Multiple studies failed to make a connection between variations in this gene and thyroid hormone levels [R, R].

However, many people have symptoms of low thyroid hormones despite their lab results being in the normal range ('hidden hypothyroidism'). That *might be* because their thyroid hormones don't function well on a cellular and tissue level, which cannot be measured by blood tests.

One *DIO2* variant may be involved in this phenomenon. The less common "C" allele on rs225014 (Thr92Ala) is associated with:

- Poor response to thyroid meds [R, R]
- Obesity and insulin resistance [R]
- Inadequate blood sugar control [R]
- Impaired cognitive development (lower IQ) [R]

All of the above may indicate low thyroid hormones in DIO2 target tissues such as the brain, fat tissue, and muscles.

The same DIO2 variant (rs225014) is associated with an inadequate response to thyroid meds in some people. Out of 45 patients, those who carried at least one "C" allele didn't respond as well to standard T4 treatment and were significantly more depressed. They preferred a combination of T4 and T3 (liothyronine) instead [R].

A larger trial came to a similar conclusion, though the effects were significant only for patients who had both copies of the "C" allele [R].

A Dutch study with over 12,600 participants found no connection between DIO2 and thyroid treatment response. However, they didn't investigate the effects of the T4+T3 combination [R].

HIGHER ACTIVITY

Likely higher DIO2 activity based on the genetic variants we looked at

45%

OF USERS SHARE THE SAME SCORE

You have the same genetic predisposition as **45% of our users.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
DIO2	rs225014	TT

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

Recommendations Details

1 Maintain a Healthy Weight

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

TYPICAL STARTING DOSE

30 minutes

Helps with these Symptoms & Conditions:

- Allergies
- High Blood Pressure
- Migraines

Helps with these Goals:

- Immunity

Helps with these DNA Risks:

- GLP-1
- PPM1K (Blood Sugar/Diet)
- Visceral Fat

2 Avoid Sugary Foods & Drinks

To avoid sugary foods, eliminate or significantly reduce consumption of foods and beverages high in added sugars such as sodas, candies, baked goods, and sugary cereals from your diet. Instead, opt for natural sugar sources like fruits. Aim to do this daily for ongoing health benefits.

Helps with these Goals:

- Energy
- Mood

Helps with these DNA Risks:

- ADRB2 (Weight)
- FOXO1 (Blood Sugar)
- Heavy Sweating
- PPM1K (Blood Sugar/Diet)
- SLC30A8 (Zinc & Blood Sugar)
- Visceral Fat

3 Maintain Optimal Vitamin D Levels

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

TYPICAL STARTING DOSE

1000 iu

Helps with these Symptoms & Conditions:

- Allergies
- Anxiety
- High Blood Pressure
- Migraines

Helps with these Goals:

- Energy
- Immunity
- Mood
- Muscle Growth

Helps with these DNA Risks:

- Visceral Fat

4 Aerobic Exercise (Cardio)

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

TYPICAL STARTING DOSE

1 hour

Helps with these Symptoms & Conditions:

- Allergies
- Anxiety
- High Blood Pressure
- Migraines

Helps with these Goals:

- Energy
- Immunity
- Mood

Helps with these DNA Risks:

- ADRB2 (Weight)
- UCP1 (Weight)
- Visceral Fat

5 Mediterranean Diet

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

Helps with these Symptoms & Conditions:

- Allergies
- High Blood Pressure

Helps with these Goals:

- Energy
- Mood

Helps with these DNA Risks:

- Visceral Fat

6 Limit Calorie Intake

Consume fewer calories than your body needs for maintenance. Calculate your daily caloric needs using an online calculator based on your sex, age, weight, height, and activity level, then reduce that number by 500-1000 calories per day to safely lose 1-2 pounds per week. Adjust the caloric intake as needed based on your progress.

Helps with these Symptoms & Conditions:

- High Blood Pressure

Helps with these Goals:

- Mood

Helps with these DNA Risks:

- ADRB2 (Weight)
- FOXO1 (Blood Sugar)
- UCP1 (Weight)
- Visceral Fat

7 Relaxation Techniques

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

TYPICAL STARTING DOSE

30 minutes

Helps with these Symptoms & Conditions:

- Anxiety
- High Blood Pressure
- Migraines

Helps with these Goals:

- Energy
- Immunity
- Mood

Helps with these DNA Risks:

- Heavy Sweating
- Visceral Fat

8 Whole Grains

Incorporate at least three servings of whole grains into your daily diet. This can include consuming foods such as whole grain bread, brown rice, whole grain pasta, and oats. Aim to replace refined grains with whole grains at each meal for optimal benefits.

Helps with these Symptoms & Conditions:

- High Blood Pressure

Helps with these DNA Risks:

- Visceral Fat

9 Green Tea

Consume 400 mg of green tea extract daily. This can be taken in the form of capsules or tablets available that specify the amount of green tea extract. Ensure the supplement is taken according to the product's specific

TYPICAL STARTING DOSE

instructions, usually once a day with water.

400 mg

Helps with these Symptoms & Conditions:

Anxiety | High Blood Pressure

Helps with these Goals:

Energy | Immunity | Mood

Helps with these DNA Risks:

FOXO1 (Blood Sugar) | Visceral Fat

10 Probiotics

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

TYPICAL STARTING DOSE

30 billion CFU

Helps with these Symptoms & Conditions:

Allergies | Anxiety

Helps with these Goals:

Immunity | Mood | Muscle Growth

Helps with these DNA Risks:

GLP-1 | Visceral Fat

11 Yoga

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

TYPICAL STARTING DOSE

30 minutes

Helps with these Symptoms & Conditions:

Anxiety | High Blood Pressure | Migraines

Helps with these Goals:

Energy | Exercise Recovery | Immunity | Mood | Muscle Growth

Helps with these DNA Risks:

Heavy Sweating | Visceral Fat

12 Strength Training

Engage in strength training exercises, such as weight lifting or bodyweight exercises, for 60 minutes per session, 2 to 3 times per week. Ensure you work all major muscle groups and rest each muscle group for at least 48 hours before exercising it again.

TYPICAL STARTING DOSE

1 hour

Helps with these Symptoms & Conditions:

Anxiety | High Blood Pressure

Helps with these Goals:

Immunity | Mood | Muscle Growth

Helps with these DNA Risks:

ADRB2 (Weight) | Visceral Fat

13 Eat Fiber-Rich Foods

Incorporate foods high in fiber, such as fruits, vegetables, whole grains, and legumes, into your daily meals. Aim for a total dietary fiber intake of 25 to 30 grams per day, spread out over all meals.

Helps with these DNA Risks:

GLP-1 | Visceral Fat

14 Intermittent Fasting

Limit your daily eating to a specific window of time, typically within an 8-hour period such as from 12 pm to 8 pm, and fast for the remaining 16 hours of the day. Repeat this daily or for at least 3-4 days per week.

Helps with these DNA Risks:

ADRB2 (Weight) | FOXO1 (Blood Sugar) | MTNR1B (Diet & Blood Sugar) | Visceral Fat

15 Cinnamon

Take a 1 g cinnamon supplement once daily, ideally with a meal to aid absorption. This can be in the form of a capsule or tablet. Continue this regimen as long as it aligns with your health goals and under the guidance of a healthcare provider.

TYPICAL STARTING DOSE

1 g

Helps with these Symptoms & Conditions:

High Blood Pressure | Migraines

Helps with these DNA Risks:

GLP-1 | Visceral Fat

Next Steps

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

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

Your Lifestyle Assessments

Ever heard of the term Nature vs. Nurture?

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

LIFESTYLE

You have a slightly reduced risk of hashimoto's disease based on the answers you provided.

Factors impacting your risk:

Your BMI:

30.77

Increasing Risk

Have you ever been diagnosed with multiple sclerosis?

No

Decreasing Risk

Have you been diagnosed with psoriasis?

No

Decreasing Risk

Have you ever been diagnosed with rheumatoid arthritis (autoimmune joint inflammation)?

No

Decreasing Risk

Have you ever been diagnosed with lupus?

No

Decreasing Risk

Have you ever been diagnosed with type 1 diabetes?

No

Decreasing Risk

Do you have a parent or sibling who has been diagnosed with Hashimoto's disease (autoimmune underactive thyroid)?

No

Decreasing Risk

What is your sex?

Male

Decreasing Risk

What is your height?

178 cm

No impact

What is your current weight?

97.5 kg

No impact

LIFESTYLE

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

Factors impacting your risk:

How much alcohol do you drink on a typical day?
Calculate your alcohol consumption in units here

0 units

Increasing Risk

How many servings of whole grains do you eat in a typical day?
Please click here for more information on whole grain servings

Less than 3

Increasing Risk

How many cups of coffee do you drink on a typical day?

0

Increasing Risk

Your BMI:

30.77

Increasing Risk

In a typical week, how many times do you participate in any physical activities or exercise for 30 minutes at a time? (such as walking, running, bike riding, weight training, yoga, etc.)

*Note: longer exercise equals more sessions (e.g., 1 hour = 2 sessions)

8 or more

Decreasing Risk

Do you smoke tobacco?

No, never

Decreasing Risk

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

No

Decreasing Risk

What is your waist size?

Less than 35 (less than 89 cm)

Decreasing Risk

How many times in a typical day do you eat refined grains (e.g., white pasta, white rice, white bread, etc.)?

0-2

Decreasing Risk

How much sleep do you get in a typical night?

7-8 hours

Decreasing Risk

Have you recurrently been diagnosed with high triglycerides?

No

Decreasing Risk

What is your ethnicity?

Other

Decreasing Risk

What is your height?

178 cm

No impact

What is your current weight?

97.5 kg

No impact

What is your sex?

Male

No impact

LIFESTYLE

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

Factors impacting your risk:

What is your age?

41

Increasing Risk

What is your current marital status?

Single or not living with partner

Decreasing Risk

How would you describe the environment you live in?

Suburban

Decreasing Risk

On a scale of 1 to 5, how would you rate the amount of stress in your life in the past month (at home and at work)?

2

Decreasing Risk

In a typical week, how many times do you participate in any physical activities or exercise for 30 minutes at a time? (such as walking, running, bike riding, weight training, yoga, etc.)

*Note: longer exercise equals more sessions (e.g., 1 hour = 2 sessions)

8 or more

Decreasing Risk