

100x Smart Gene Functional Report

Health Report

REPORT CATEGORIES —



COGNITION



GUT HEALTH



NUTRITION



FOOD SENSITIVITIES



HEART & BLOOD
VESSELS



THYROID



SLEEP



INFLAMMATION &
AUTOIMMUNITY



IMMUNITY &
INFECTIONS



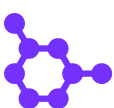
WEIGHT & BODY FAT



BLOOD SUGAR
CONTROL



DETOX



SEX HORMONES



FITNESS

Sample Client

Report date: 28 July 2025

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Recommendations Details

DISCLAIMER

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

Personal information

NAME

Sample Client

SEX AT BIRTH

Male

HEIGHT

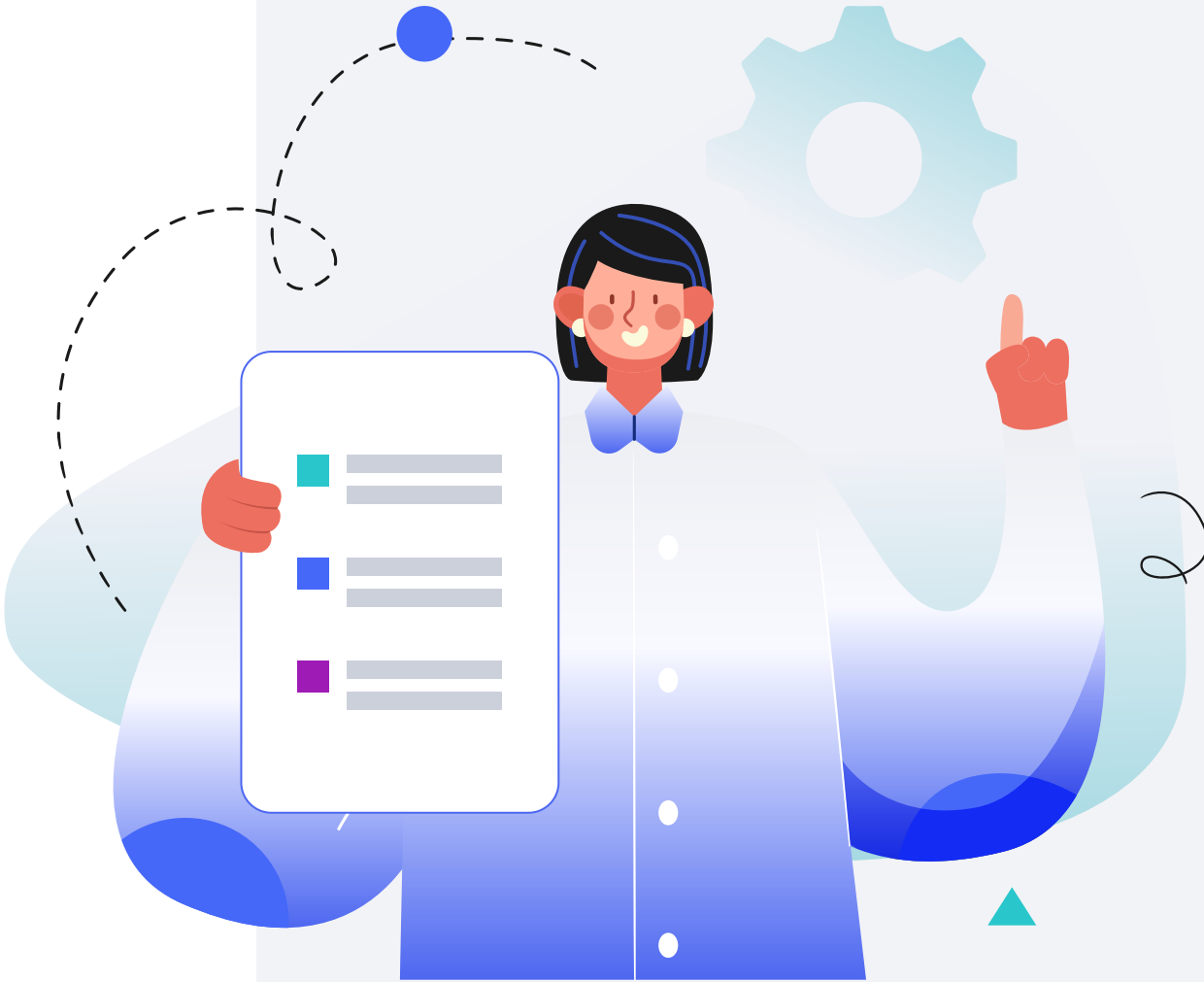
5ft 9"

175.0cm

WEIGHT

165lb

75.0kg



Summary

It’s easy to ignore subtle signs of health until they become a problem. The truth is, many chronic conditions develop quietly over time, influenced by a complex mix of lifestyle, environment, and genetics. Understanding your unique genetic makeup can help you stay ahead by identifying risks before they manifest.

This comprehensive report dives into the genes affecting your predispositions across a wide range of health areas that shape your overall well-being, including cardiovascular disease risk, metabolism, inflammation, gut health, hormonal balance, and more. By exploring how your genes influence these critical systems, you gain valuable insights that empower you to make personalized lifestyle and health decisions.

In this report, you’ll learn about your genetics in areas such as:

- Cardiovascular disease risk and lipid metabolism
- Metabolic syndrome, diabetes, and obesity
- Inflammatory disorders and gut microbiome health
- Nutrient metabolism
- Detoxification and oxidative stress management
- Hormone regulation
- Brain function and stress response
- Physical fitness

With this knowledge, you can take proactive steps to optimize your health and wellness on a deeper, personalized level.

This summary report contains:

247 Genetic Results

50 Recommendations

Overview of Your Results

 Cardiovascular Health

 HIGHER ACTIVITY AGT (Cardiovascular & Fitness) Likely higher AGT activity	 HIGHER ACTIVITY AGTR1 (Blood Pressure) Predisposed to higher AGTR1 activity	 HIGHER ACTIVITY ITGB3 (Cardiovascular) Predisposed to higher ITGB3 activity
 WORSE GENETICS PHACTR1 (Cardiovascular) Predisposed to a worse PHACTR1 genetics	 LOWER ACTIVITY CDKN2B-AS (Cardiovascular) Predisposed to a lower CDKN2B-AS activity	 LOWER ACTIVITY PITX2 (Cardiovascular) Predisposed to a lower PITX2 activity
 HIGHER ACTIVITY ADRB1 (Cardiovascular) Predisposed to higher ADRB1 activity	 LOWER ACTIVITY CPS1 (Cardiovascular, Kidney Health) Likely lower CPS1 activity	 HIGHER ACTIVITY CELSR2-PSRC1-SORT1 (Cardiovascular) Likely higher CELSR2-PSRC1-SORT1 activity
 TYPICAL ACTIVITY NOS3 (Cardiovascular) Likely typical NOS3 activity	 TYPICAL ACTIVITY EDN1 (Blood Pressure) Predisposed to a typical EDN1 activity	 TYPICAL ACTIVITY ACYP2 (Cardiovascular, Cancer) Likely typical ACYP2 activity
 TYPICAL ACTIVITY ACMSD (Cardiovascular) Predisposed to typical ACMSD activity	 TYPICAL ACTIVITY BPIFB4 (Cardiovascular & Longevity) Predisposed to typical BPIFB4 activity	 TYPICAL GENETICS SCARB1 (Cardiovascular) Predisposed to a typical SCARB1 genetics



TYPICAL ACTIVITY

FN1
(Neurodegeneration,
Cardiovascular)

Predisposed to typical FN1 activity



TYPICAL GENETICS

CORIN (Cardiovascular)

Predisposed to a typical CORIN genetics



LOWER ACTIVITY

**ACE (Fitness/
Cardiovascular)**

Likely lower ACE activity



HIGHER ACTIVITY

GCH1 (Cardiovascular)

Likely higher GCH1 activity



HIGHER ACTIVITY

GLUL (Cardiovascular)

Predisposed to a higher GLUL activity

Lipid Metabolism



HIGHER ACTIVITY

PCSK9 (Cholesterol)

Likely higher PCSK9 activity



LOWER ACTIVITY

DOCK7 (Blood Lipids)

Likely lower DOCK7 activity



TYPICAL ACTIVITY

**APOC3 (Blood Lipids/
Longevity)**

Likely typical APOC3 activity



E3/E3

APOE

You carry two APOE ε3 variants



TYPICAL ACTIVITY

HMGCR (Cholesterol)

Likely typical HMGCR activity



TYPICAL ACTIVITY

**APOA2 (Weight, Blood
Lipids)**

Likely typical APOA2 activity



TYPICAL ACTIVITY

**ABCG8 (Cholesterol &
Gallstones)**

Predisposed to typical ABCG8 activity



TYPICAL ACTIVITY

**APOB Gene
(Cardiovascular)**

Likely typical APOB activity



TYPICAL GENETICS

ABCA1 (Cholesterol)

Likely typical ABCA1 genetics

 TYPICAL GENETICS ABCA6 (Cholesterol) Likely typical ABCA6 genetics	 TYPICAL ACTIVITY APOA5 (Cardiovascular) Predisposed to typical APOA5 activity	 TYPICAL ACTIVITY LPA (Blood Lipids & Heart Health) Predisposed to typical LPA activity
 TYPICAL ACTIVITY LDLR (Cholesterol, Cardiovascular) Predisposed to typical LDLR activity	 LOWER ACTIVITY FABP2 (Blood Sugar/ Cardiovascular) Likely lower FABP2 activity	 TYPICAL GENETICS SCARB1 (Cardiovascular) Predisposed to a typical SCARB1 genetics
 TYPICAL ACTIVITY DHCR7 (Cholesterol) Predisposed to typical DHCR7 activity	 TYPICAL ACTIVITY APOA1 (Blood Lipids) Predisposed to a typical APOA1 activity	 LOWER ACTIVITY CETP (Cholesterol/ Longevity) Likely lower CETP activity
 HIGHER ACTIVITY LIPC (Cardiovascular) Likely higher LIPC activity	 LOWER ACTIVITY MLXIPL (Triglycerides, Cardiovascular) Likely lower MLXIPL activity	 LOWER ACTIVITY SOAT1 (Cholesterol/ Cognition) Likely lower SOAT1 activity

 Metabolic Syndrome & Diabetes

 LOWER ACTIVITY WFS1 (Blood Sugar) Predisposed to a lower WFS1 activity	 WORSE GENETICS TCF7L2 (Blood Sugar/Diet) Likely worse TCF7L2 genetics	 LOWER ACTIVITY ACSL1 (Blood Sugar) Predisposed to a lower ACSL1 activity

 LOWER ACTIVITY SLC30A8 (Blood Sugar) Predisposed to lower SLC30A8 activity	 LOWER ACTIVITY INSIG2 (Weight) Predisposed to a lower INSIG2 activity	 HIGHER ACTIVITY MTNR1B (Diet & Blood Sugar) Predisposed to higher MTNR1B activity
 HIGHER ACTIVITY FOXO1 (Blood Sugar) Likely higher FOXO1 activity	 LOWER ACTIVITY MADD (Blood Sugar & Insulin) Predisposed to lower MADD activity	 LOWER ACTIVITY ADCY5 (Blood Sugar) Likely lower ADCY5 activity
 LOWER ACTIVITY CDKN2B (Blood Sugar) Likely lower CDKN2B activity	 LOWER ACTIVITY PPM1K (Blood Sugar/Diet) Likely lower PPM1K activity	 TYPICAL ACTIVITY LEPR (Weight/Leptin Resistance) Likely typical LEPR activity
 TYPICAL ACTIVITY HHEX (Blood Sugar) Predisposed to a typical HHEX activity	 TYPICAL ACTIVITY KCN (Blood Sugar) Predisposed to a typical KCN activity	 TYPICAL SLC2A2 (Sugar Intake) Predisposed to typical sugar intake
 TYPICAL ACTIVITY PPARGC1A (Fitness/Blood Sugar) Likely typical PPARGC1A activity	 TYPICAL ACTIVITY GIPR (Blood Sugar) Likely typical GIPR activity	 TYPICAL GENETICS CRY2 (Blood Sugar) Likely typical CRY2 genetics
 TYPICAL ACTIVITY ADRA2A (Blood Sugar) Predisposed to typical ADRA2A activity	 TYPICAL SLC2A2 (Sugar Intake) Predisposed to typical sugar intake	 TYPICAL ACTIVITY CLTCL1 (Blood Sugar/Diet) Predisposed to typical CLTCL1 activity

 TYPICAL ACTIVITY GCKR (Blood Sugar) Likely typical GCKR activity	 BALANCED PREFERENCES FGF21 (Carbs vs Fats) Likely balanced macronutrient preferences	 HIGHER ACTIVITY IRS1 (Metabolic Health) Predisposed to higher IRS1 activity
 HIGHER ACTIVITY AMY1A (Starch Digestion) Predisposed to higher AMY1A activity	 HIGHER ACTIVITY SUOX (PCOS/ Blood Sugar) Likely higher SUOX activity	

 Fat Metabolism & Obesity

 WORSE GENETICS ADRB2 (Weight) Likely worse ADRB2 genetics	 LOWER ACTIVITY PPARD (Exercise Response) Predisposed to lower PPARD activity	 LOWER ACTIVITY UCP1 (Weight) Likely lower UCP1 activity
 TYPICAL ACTIVITY PPARG (Metabolism) Likely typical PPARG activity	 TYPICAL GENETICS ADIPOQ (Weight/ Blood Sugar) Likely typical ADIPOQ genetics	 TYPICAL ACTIVITY ADRB3 (Weight) Likely typical ADRB3 activity
 TYPICAL ACTIVITY UCP2 (Weight) Likely typical UCP2 activity	 TYPICAL ACTIVITY UCP3 (Weight) Likely typical UCP3 activity	 TYPICAL ACTIVITY TFAP2B (Weight/Diet) Likely typical TFAP2B activity
 TYPICAL ACTIVITY MC3R (Weight) Likely typical MC3R activity	 TYPICAL ACTIVITY CD36 (Fat Preference/Weight) Likely typical CD36 activity	 BETTER FTO (Weight) Likely better FTO genetics



HIGHER ACTIVITY

FADS1/2 (Fatty Acid Metabolism)

Likely higher FADS1/2 activity



HIGHER ACTIVITY

MC4R (Weight/ Blood Sugar)

Likely higher MC4R activity



LOWER ACTIVITY

PLIN (Weight, Blood Sugar)

Likely lower PLIN activity



BETTER GENETICS

TMEM18 (Weight)

Likely better TMEM18 genetics

Inflammatory Disorders



HIGHER ACTIVITY

NF-kB (Inflammation)

Likely higher NF-kB activity



HIGHER ACTIVITY

IL1A (Inflammation)

Predisposed to a higher IL1A activity



LOWER ACTIVITY

HRH1 (Allergies)

Likely lower HRH1 activity



HIGHER ACTIVITY

HLA (Inflammation)

Likely higher HLA activity



TYPICAL ACTIVITY

IL6 Gene (Inflammation)

Likely typical IL6 activity



TYPICAL ACTIVITY

TNF Gene (Inflammation)

Likely typical TNF activity



TYPICAL ACTIVITY

CRP Gene

Predisposed to typical CRP gene activity



TYPICAL ACTIVITY

IL10 Gene (Autoimmunity)

Predisposed to typical IL10 activity



TYPICAL ACTIVITY

IL4 (Allergies, Autoimmunity)

Likely typical IL4 activity



TYPICAL ACTIVITY

IL13 (Allergies, Lung Function)

Predisposed to typical IL13 activity



TYPICAL ACTIVITY

HRH4 (Allergies, Inflammation)

Predisposed to typical HRH4 activity



TYPICAL ACTIVITY

DAO (Histamine)

Likely typical DAO activity

TYPICAL ACTIVITY HDC (Histamine) Predisposed to a typical HDC activity	TYPICAL ACTIVITY HMGB1 (Inflammation) Predisposed to typical HMGB1 activity	TYPICAL ACTIVITY SMAD3 (Allergies) Predisposed to typical SMAD3 activity
TYPICAL ACTIVITY GATA3 (Allergies) Likely typical GATA3 activity	LOWER ACTIVITY IL1B (Inflammation/ Fatigue) Likely lower IL1B activity	HIGHER ACTIVITY IL2 (Allergies) Predisposed to a higher IL2 activity
LOWER ACTIVITY IL21 (Autoimmunity & Allergies) Predisposed to lower IL21 activity	LOWER ACTIVITY IL8 (Inflammation) Likely lower IL8 activity	BETTER GENETICS HRH2 (Gut Health) Likely better HRH2 genetics
HIGHER ACTIVITY HNMT (Histamine) Likely higher HNMT activity		

Food Responses

HIGHER ACTIVITY ADH1B (Alcohol Sensitivity) Predisposed to higher ADHB1 activity	LOWER ACTIVITY MCM6 (Lactose Intolerance) Likely lower MCM6 activity	LOWER ACTIVITY SI (Digestive Issues) Predisposed to lower SI activity
HIGHER Dairy Sensitivity (Casein) Likely higher sensitivity to dairy	LOWER ACTIVITY ALDH2 (Alcohol Sensitivity) Likely typical ALDH2 activity	TYPICAL GENETICS ALDH1B1 (Alcohol Sensitivity) Predisposed to typical ALDH1B1 genetics

 TYPICAL GENETICS HLA-DQ (Gluten) Likely typical HLA-DQ genetics	 TYPICAL ACTIVITY ALDOB (Fructose Intolerance) Predisposed to typical ALDOB activity	 INTERMEDIATE ACTIVITY TAS2R38 (Bitter Taste Perception) Likely intermediate TAS2R38 activity
 TYPICAL ACTIVITY TAS2R16 (Bitter Taste/ Longevity) Likely typical TAS2R16 activity	 HIGHER ACTIVITY CYP1A2 (Detox) Likely higher CYP1A2 activity	 LIKELY TOLERANT Lactose Intolerance Likely lactose tolerant

Gut Inflammation & Microbiome

 LOWER ACTIVITY IBD5 (Gut Inflammation) Predisposed to lower IBD5 activity	 HIGHER ACTIVITY HTR3A (Gut Health) Predisposed to a higher HTR3A activity	 HIGHER ACTIVITY IL23R (Gut Inflammation) Predisposed to higher IL23R activity
 HIGHER ACTIVITY NLRP3 (Gut Inflammation) Likely higher NLRP3 activity	 LOWER ACTIVITY HNF4A (Gut Inflammation) Likely lower HNF4A activity	 SECRETOR FUT2 (Gut/ Vitamin B12) Likely secretor
 TYPICAL ACTIVITY TLR4 (Inflammation) Likely typical TLR4 activity	 TYPICAL ACTIVITY TNFSF15 (Gut Inflammation) Predisposed to a typical TNFSF15 activity	 TYPICAL GENETICS MUC1 (Immunity, Gut, Minerals) Likely typical MUC1 genetics



TYPICAL GENETICS

SLC2A14 (Gut Inflammation)

Likely typical SLC2A14 genetics



LOWER ACTIVITY

MRGPRX2 (Gut Inflammation)

Predisposed to lower MRGPRX2 activity



LOWER ACTIVITY

JAK2 (Gut Inflammation)

Likely lower JAK2 activity



Vitamins & Minerals



HIGHER ACTIVITY

TFR2 (Iron)

Predisposed to a higher TFR2 activity



LOWER ACTIVITY

BCO1 (Vitamin A)

Predisposed to a lower BCO1 activity



LOWER ACTIVITY

TCN2 (Vitamin B12)

Likely lower TCN2 activity



TYPICAL ACTIVITY

TMPRSS6 (Iron)

Predisposed to typical TMPRSS6 activity



TYPICAL ACTIVITY

TRPM6 (Magnesium & Calcium)

Predisposed to typical TRPM6 activity



TYPICAL ACTIVITY

TF (Iron & Cognition)

Predisposed to typical TF activity



TYPICAL ACTIVITY

SLC23A1 (Vitamin C)

Predisposed to typical SLC23A1 activity



TYPICAL ACTIVITY

VDR (Vitamin D)

Likely typical VDR activity



TYPICAL ACTIVITY

CYP24A1 (Vitamin D)

Likely typical CYP24A1 activity



TYPICAL ACTIVITY

PDXK (Vitamin B6)

Likely typical PDXK activity



HIGHER ACTIVITY

GC (Vitamin D)

Likely higher GC activity



HIGHER ACTIVITY

CUBN (Vitamin B12)

Likely higher CUBN activity

 Detox

 HIGHER ACTIVITY CYP1B1 (Detox/ Skin Health) Predisposed to higher CYP1B1 activity	 LOWER ACTIVITY PON1 (Detox) Likely lower PON1 activity	 LOWER ACTIVITY CYP2D6 (Mental Health, Detox) Predisposed to a lower CYP2D6 activity
 TYPICAL ACTIVITY CYP1A1 (Detox) Likely typical CYP1A1 activity	 TYPICAL ACTIVITY CYP2A6 (Detox, Nicotine) Likely typical CYP2A6 activity	 EXTENSIVE METABOLIZER CYP2C19 (Detox) Likely an extensive metabolizer
 TYPICAL ACTIVITY CYP3A4 (Detox) Likely typical CYP3A4 activity	 TYPICAL ACTIVITY CYP3A5 (Detox/Blood Pressure) Predisposed to a typical CYP3A5 activity	 HIGHER ACTIVITY SULT1A1 (Detox) Likely higher SULT1A1 activity
 INTERMEDIATE ACETYLATOR NAT1 (Detox) Likely an intermediate acetylator	 HIGHER ACTIVITY UGT (Detox) Likely higher UGT activity	 INTERMEDIATE NAT2 (Detox) Likely an intermediate acetylator

 Oxidative Stress

 LOWER ACTIVITY SOD2 (Oxidative Stress) Likely lower SOD2 activity	 LOWER ACTIVITY GCLC (Glutathione & Detox) Predisposed to lower GCLC activity	 LOWER ACTIVITY GSTA1 (Detox/ Glutathione) Predisposed to lower GSTA1 activity

 TYPICAL ACTIVITY GCLM (Glutathione & Detox) Predisposed to a typical GCLM activity	 TYPICAL ACTIVITY GPX4 (Selenium & Glutathione) Predisposed to typical GPX4 activity	 TYPICAL ACTIVITY GSR (Oxidative Stress) Predisposed to a typical GSR activity
 TYPICAL ACTIVITY CAT (Oxidative Stress) Predisposed to a typical CAT activity	 HIGHER ACTIVITY GSTP1 (Detox) Likely higher GSTP1 activity	 HIGHER ACTIVITY SOD1 (Oxidative Stress) Predisposed to higher SOD1 activity
 HIGHER ACTIVITY GPX1 (Glutathione/Detox) Likely higher GPX1 activity	 HIGHER ACTIVITY GSTM3 (Oxidative Stress) Predisposed to a higher GSTM3 activity	 HIGHER ACTIVITY HMOX1 (Oxidative Stress) Predisposed to a higher HMOX1 activity
 HIGHER ACTIVITY NQO1 (Detox) Likely higher NQO1 activity		

 Methylation

 LOWER ACTIVITY MTHFR Likely lower MTHFR activity	 LOWER ACTIVITY MTR (Methylation) Likely typical MTR activity	 LOWER ACTIVITY MTHFD1 (Folate/ Methylation) Likely lower MTHFD1 activity
 WORSE GENETICS MTHFS (Folate/ Methylation) Likely worse MTHFS genetics	 WORSE GENETICS SHMT1 (Folate/ Methylation) Likely worse SHMT1 genetics	 HIGHER ACTIVITY GNMT (Methylation) Likely higher GNMT activity



TYPICAL ACTIVITY

MTRR (Methylation)

Likely typical MTRR activity



TYPICAL ACTIVITY

PEMT (Choline)

Likely typical PEMT activity



TYPICAL ACTIVITY

MTHFD1L (Folate/
Methylation)

Likely typical MTHFD1L activity



TYPICAL GENETICS

CHDH (Choline)

Likely typical CHDH genetics



TYPICAL

BHMT (Methylation)

Likely typical BHMT genetics



HIGHER ACTIVITY

DHFR (Folate/
Methylation)

Likely higher DHFR activity



TYPICAL GENETICS

FOLH1 (Folate/
Methylation)

Likely typical FOLH1 genetics



TYPICAL GENETICS

DNMT3B (Methylation)

Likely typical DNMT3B genetics



TYPICAL ACTIVITY

MAT1A (Methylation)

Likely typical MAT1A activity



TYPICAL ACTIVITY

AHCY (Methylation)

Likely typical AHCY activity



TYPICAL GENETICS

TYMS (Methylation)

Likely typical TYMS genetics



HIGHER ACTIVITY

CBS (Transsulfuration)

Likely higher CBS activity

Hormones



LOWER ACTIVITY

SHBG Gene (Sex
Hormones)

Predisposed to a lower SHBG activity



HIGHER ACTIVITY

CYP17A1 (Sex
Hormones)

Predisposed to a higher CYP17A1 activity



HIGHER ACTIVITY

HSD3B1 (Sex
Hormones)

Predisposed to a higher HSD3B1 activity



TYPICAL ACTIVITY

UGT2B15 (Sex
Hormones, Detox)

Predisposed to a typical UGT2B15 activity



TYPICAL

TPO (Thyroid)

Likely typical TPO genetics



TYPICAL ACTIVITY

PGR (Sex Hormones)

Predisposed to a typical PGR activity



TYPICAL GENETICS

LHCGR (Sex Hormones)

Predisposed to typical LHCGR genetics



TYPICAL ACTIVITY

UGT2B15 (Sex Hormones, Detox)

Predisposed to a typical UGT2B15 activity



TYPICAL ACTIVITY

CYP11A1 (Sex Hormones)

Predisposed to typical CYP11A1 genetics



TYPICAL ACTIVITY

ESR1 (Estrogen)

Likely typical ESR1 activity



HIGHER ACTIVITY

DIO1 (Thyroid)

Likely higher DIO1 activity



HIGHER ACTIVITY

DIO2 (Thyroid)

Likely higher DIO2 activity



LOWER ACTIVITY

FOXE1 (Thyroid)

Predisposed to a lower FOXE1 activity

 Neurotransmitters



HIGHER ACTIVITY

MAOA (Dopamine/Serotonin)

Likely higher MAOA activity



HIGHER ACTIVITY

MAOB (Dopamine/ Norepinephrine)

Likely higher MAOB activity



LOWER ACTIVITY

SLC18A1 (Mental Health)

Predisposed to a lower SLC18A1 activity



LOWER ACTIVITY

DRD2 (Dopamine)

Likely lower DRD2 activity



HIGHER ACTIVITY

CHRNA4 (Smoking, Cognition)

Predisposed to a higher CHRNA4 activity



WORSE

GAD1 (Glutamate/GABA)

Likely worse GAD1 genetics



LIKELY S/S

5-HTTLPR (Serotonin)

Likely S/S genotype at 5HTTLPR



LOWER ACTIVITY

SLC6A4 (Fatigue, Mental Health)

Likely lower SLC6A4 activity



LOWER ACTIVITY

HTR2C (Weight, Mental Health)

Likely lower HTR2C activity



HIGHER ACTIVITY

HTR3A (Gut Health)

Predisposed to a higher HTR3A activity



LOWER ACTIVITY

SLC18A1 (Mental Health)

Predisposed to a lower SLC18A1 activity



TYPICAL ACTIVITY

COMT

Likely typical COMT activity



TYPICAL ACTIVITY

TH (Mental Health, Blood Pressure)

Predisposed to a typical TH activity



TYPICAL ACTIVITY

DBH (Mental Health)

Predisposed to typical DBH activity



TYPICAL ACTIVITY

SLC18A2 (Mental Health)

Predisposed to typical SLC18A2 activity



TYPICAL ACTIVITY

SLC6A3 (Mental Health)

Predisposed to a typical SLC6A3 activity



TYPICAL ACTIVITY

CHRNA5 (Smoking, Cognition)

Predisposed to typical CHRNA5 activity



TYPICAL LEVELS

IDO (Kynurenine)

Predisposed to typical IDO levels



TYPICAL ACTIVITY

KMO (Mental Health)

Predisposed to a typical KMO activity



TYPICAL ACTIVITY

TPH2 (Serotonin)

Predisposed to typical TPH2 activity



TYPICAL ACTIVITY

HTR2A (Serotonin)

Likely typical HTR2A activity



HIGHER ACTIVITY

HTR1A (Serotonin)

Likely higher HTR1A activity



TYPICAL ACTIVITY

CNR1 (Cannabinoids)

Likely typical CNR1 activity



TYPICAL ACTIVITY

CNR2 (Cannabinoids)

Predisposed to a typical CNR2 activity



LOWER ACTIVITY

FAAH (Cannabinoids)

Likely lower FAAH activity

Neuronal Plasticity & Stress Response

 SLIGHTLY LOWER LEVELS BDNF Relatively lower BDNF levels	 LOWER ACTIVITY ENPP6 (Cognition) Likely lower ENPP6 activity	 LOWER ACTIVITY ULK4 (Cognition) Likely lower ULK4 activity
 LOWER ACTIVITY CRHR2 (Stress/HPA Axis) Likely lower CRHR2 activity	 TYPICAL ACTIVITY LMX1A (Cognition) Likely typical LMX1A activity	 INTERMEDIATE ACTIVITY NR3C1 (Fatigue) Likely intermediate NR3C1 activity
 LOWER ACTIVITY CRHR1 (Stress/HPA Axis) Likely lower CRHR1 activity	 LOWER ACTIVITY FKBP5 (Stress/ HPA Axis) Likely lower FKBP5 activity	 HIGHER ACTIVITY KIBRA/WWC1 (Cognition) Likely higher KIBRA/WWC1 activity
 HIGHER ACTIVITY TUSC3 (Cognition) Likely higher TUSC3 activity		

Sleep & Circadian Rhythm

 WORSE GENETICS NPAS2 (Mood/ Sleep Schedule) Likely worse NPAS2 genetics	 WORSE GENETICS NPAS2 (Mood/ Sleep Schedule) Likely worse NPAS2 genetics	 TYPICAL ACTIVITY CLOCK (Sleep & Weight) Likely typical CLOCK activity



TYPICAL GENETICS

AANAT (Circadian Rhythm)

Predisposed to typical AANAT genetics



TYPICAL GENETICS

ARNTL (Circadian Rhythm)

Predisposed to typical ARNTL genetics



TYPICAL ACTIVITY

TPH2 (Serotonin)

Predisposed to typical TPH2 activity



TYPICAL ACTIVITY

GRIA3 (Sleep/Mood)

Likely typical GRIA3 activity



TYPICAL ACTIVITY

BHLHE41 (Sleep)

Predisposed to typical BHLHE41 activity



TYPICAL ACTIVITY

ASMT (Mood, Neurodivergence)

Predisposed to a typical ASMT activity



HIGHER ACTIVITY

MTNR1A (Melatonin)

Likely higher MTNR1A activity

Sports & Exercise



TYPICAL ACTIVITY

ACTN3 (Power)

Likely typical ACTN3 activity



TYPICAL ACTIVITY

COL5A1 (Collagen)

Likely typical COL5A1 activity



TYPICAL ACTIVITY

AMPD1 (Muscle Performance)

Predisposed to typical AMPD1 activity



HIGHER ACTIVITY

COL1A1 (Collagen)

Predisposed to higher COL1A1 activity



HIGHER ACTIVITY

SLC16A1 (Athletic Performance)

Likely higher SLC16A1 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		DOSAGE	
1	Dietary Folate	2	Methylfolate400 mcg
3	Leafy Green Vegetables	4	Betaine (TMG)500 mg
5	Zinc15 mg	6	Avoid High-Dose Niacin Supplements35 mg
7	Dietary Riboflavin (Vitamin B2)	8	Riboflavin (Vitamin B2)25 mg
9	Yoga30 minutes	10	Green Tea400 mg
11	Limit Calorie Intake	12	Coffee
13	Limit Dairy Intake	14	Mindfulness Meditation30 minutes
15	Berberine1000 mg	16	Cocoa
17	Green Tea Extract250 mg	18	Lactobacillus Plantarum10 billion CFU
19	L-Tyrosine500 mg	20	Phosphatidylserine300 mg
21	Resistant Starch40 g Hi-Maize	22	Spend Time in Nature2 hours
23	Tai Chi1 hour	24	Walnuts
25	Vitamin B12100 mcg	26	Pyridoxine (Vitamin B6)50 mg
27	Cognitive-Behavioral Therapy (CBT)	28	Tryptophan500 mg
29	Aromatherapy30 minutes	30	Ginger500 mg

31	Blackcurrants		32	Manganese supplements	1 mg
33	Limit Saturated Fat		34	Maintain Optimal Vitamin D Levels	1000 iu
35	Mediterranean Diet		36	Sleep for 7+ Hours	
37	Avoid Air Pollution		38	Cognitive Activity	15 minutes
39	Carnosine And Anserine	1 g	40	Dietary B Vitamins	
41	Dietary Nitrates		42	Power Training	1 hour
43	Avoid Cannabis		44	Mindfulness	30 minutes
45	SAM-e	400 mg	46	Core-Strengthening Exercises	20 minutes
47	N-acetylcysteine (NAC)	1200 mg	48	Sufficient Calorie Intake	
49	Tennis		50	Whole-Body Vibration	15 minutes

Your Results in Details



Cardiovascular Health

Cardiovascular disease is a leading cause of death globally, and many genetic factors contribute to your risk. Genetics can influence various aspects of cardiovascular health, from blood pressure regulation to blood vessel function and cholesterol metabolism. Understanding your genetic predisposition can help you take preventive steps to reduce your overall risk.

Genes like *ACE*, *NOS3*, and *AGT* are involved in blood pressure regulation, while *CDKN2B-AS* and *PITX2* are linked to heart development and rhythm. Additionally, variants in *EDN1* and *ITGB3* can influence blood vessel constriction and clot formation, which are crucial in the development of cardiovascular diseases. By identifying these genetic factors, you can work with healthcare professionals to tailor lifestyle changes and treatments to better manage your heart health.

 HIGHER ACTIVITY AGT (Cardiovascular & Fitness) Likely higher AGT activity	 HIGHER ACTIVITY AGTR1 (Blood Pressure) Predisposed to higher AGTR1 activity	 HIGHER ACTIVITY ITGB3 (Cardiovascular) Predisposed to higher ITGB3 activity
 WORSE GENETICS PHACTR1 (Cardiovascular) Predisposed to a worse PHACTR1 genetics	 LOWER ACTIVITY CDKN2B-AS (Cardiovascular) Predisposed to a lower CDKN2B-AS activity	 LOWER ACTIVITY PITX2 (Cardiovascular) Predisposed to a lower PITX2 activity
 HIGHER ACTIVITY ADRB1 (Cardiovascular) Predisposed to higher ADRB1 activity	 LOWER ACTIVITY CPS1 (Cardiovascular, Kidney Health) Likely lower CPS1 activity	 HIGHER ACTIVITY CELSR2-PSRC1-SORT1 (Cardiovascular) Likely higher CELSR2-PSRC1-SORT1 activity



TYPICAL ACTIVITY

NOS3 (Cardiovascular)

Likely typical NOS3 activity



TYPICAL ACTIVITY

EDN1 (Blood Pressure)

Predisposed to a typical EDN1 activity



TYPICAL ACTIVITY

ACYP2 (Cardiovascular, Cancer)

Likely typical ACYP2 activity



TYPICAL ACTIVITY

ACMSD (Cardiovascular)

Predisposed to typical ACMSD activity



TYPICAL ACTIVITY

BPIFB4 (Cardiovascular & Longevity)

Predisposed to typical BPIFB4 activity



TYPICAL GENETICS

SCARB1 (Cardiovascular)

Predisposed to a typical SCARB1 genetics



TYPICAL ACTIVITY

FN1 (Neurodegeneration, Cardiovascular)

Predisposed to typical FN1 activity



TYPICAL GENETICS

CORIN (Cardiovascular)

Predisposed to a typical CORIN genetics



LOWER ACTIVITY

ACE (Fitness/ Cardiovascular)

Likely lower ACE activity



HIGHER ACTIVITY

GCH1 (Cardiovascular)

Likely higher GCH1 activity



HIGHER ACTIVITY

GLUL (Cardiovascular)

Predisposed to a higher GLUL activity

AGT (Cardiovascular & Fitness)

The main *AGT* polymorphism is [rs699](#), commonly called M235T in the literature. Its ‘G’ allele may increase angiotensinogen production, thereby promoting the production of angiotensin II and aldosterone [\[R\]](#).

This variant has been associated with an increased risk of:

- Hypertension [\[R, R, R\]](#)
- Preeclampsia [\[R, R, R\]](#)
- Ischemic stroke [\[R, R, R\]](#)
- Coronary heart disease [\[R, R, R, R, R\]](#)
- Heart failure [\[R, R\]](#)
- Heart attack [\[R, R\]](#)
- Diabetic nephropathy [\[R\]](#)
- End-stage kidney disease [\[R\]](#)

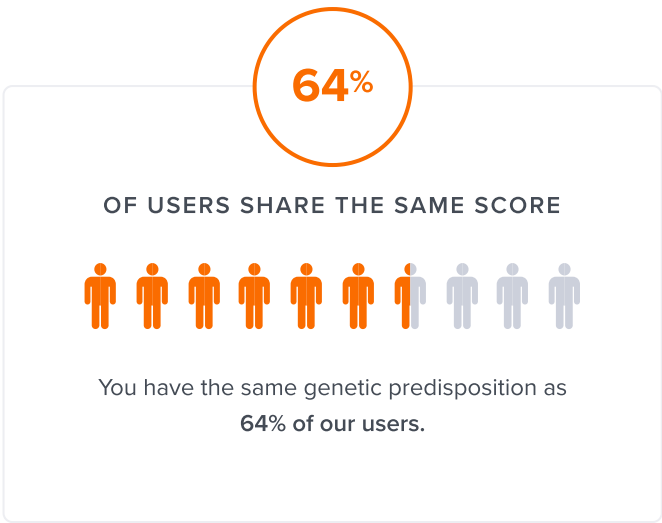
Moreover, carriers of this variant may see a weaker reduction of their blood pressure in response to exercise [\[R\]](#).

On the bright side, this variant may confer greater athletic performance in power sports [\[R, R\]](#).



HIGHER ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
AGT	rs699	GG

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

AGTR1 (Blood Pressure)

The main *AGTR1* polymorphism is [rs5186](#), commonly called A1166C in the literature. Its minor ‘C’ allele may increase the expression of this gene, potentially increasing RAS signaling [\[R\]](#).

In line with this, this variant has been associated with an increased risk of:

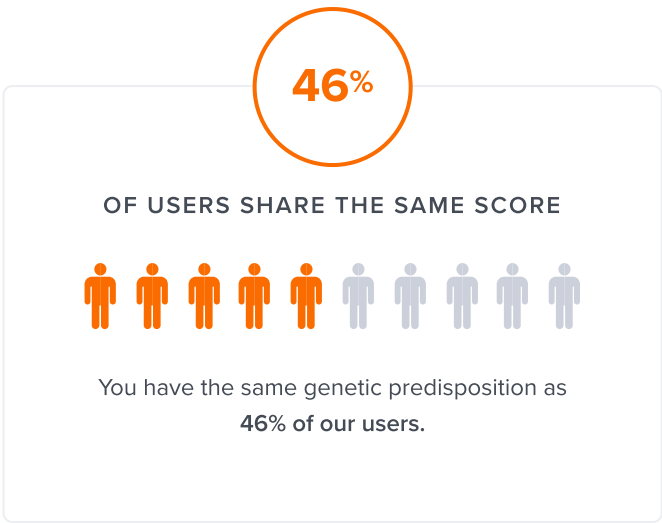
- Hypertension [\[R, R, R, R, R, R\]](#)
- Hypertensive disorders of pregnancy [\[R, R, R, R\]](#)
- Chronic kidney disease [\[R\]](#)
- End-stage kidney disease [\[R\]](#)
- Coronary artery disease [\[R, R, R\]](#)
- Myocardial infarction [\[R\]](#)
- Metabolic syndrome [\[R\]](#)
- Aortic aneurysm [\[R\]](#)

Interestingly, this variant has been associated with higher triglycerides and cholesterol but lower adiponectin levels in response to dietary fats, predicting an increased risk of NAFLD, insulin resistance, and impaired blood vessel function in people eating a high-fat diet [\[R\]](#).



HIGHER ACTIVITY

Predisposed to higher AGTR1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CPA3	rs5186	CA

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

ITGB3 (Cardiovascular)

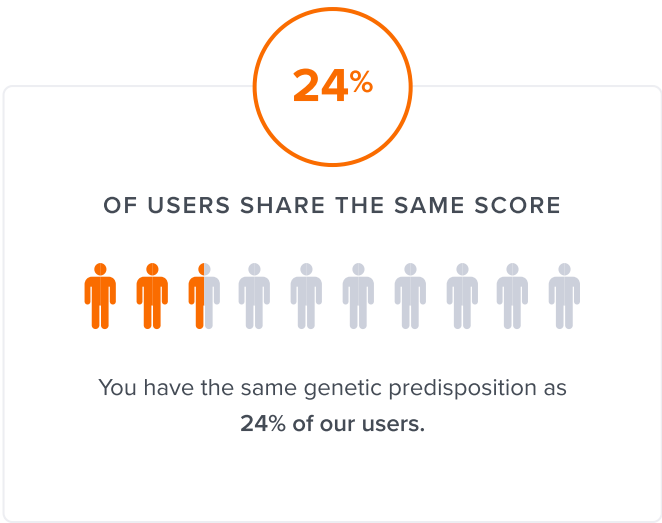
The *ITGB3* polymorphism most strongly associated with cardiovascular outcome is [rs5918](#) (Leu59Pro), commonly referred to as *PIA1/A2*. Its minor ‘C’ allele (*PIA2*) encodes an αIIbβ3 protein that makes platelets more likely to clump together. This variant has been associated with an increased risk of [\[R\]](#):

- Coronary artery disease [\[R\]](#), [\[R\]](#)
- Myocardial infarction [\[R\]](#), [\[R\]](#)
- Sudden cardiac death and coronary thrombosis [\[R\]](#)
- Congenital heart defects [\[R\]](#)
- Heart failure [\[R\]](#)
- Metabolic syndrome [\[R\]](#)
- Atherosclerotic plaque rupture [\[R\]](#)



HIGHER ACTIVITY

Predisposed to higher ITGB3 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ITGB3	rs5918	TC

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

PHACTR1 (Cardiovascular)

The best-characterized *PHACTR1* variant is [rs9349379](#). Its minor ‘G’ allele may lower *PHACTR1* expression in vascular cells. This variant has been associated with an increased risk of [\[R\]](#), [\[R\]](#):

- Coronary artery disease [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Coronary artery calcification, especially in smokers [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Coronary atherosclerosis [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Coronary microvascular dysfunction [\[R\]](#)
- Myocardial infarction [\[R\]](#), [\[R\]](#)
- Unstable angina [\[R\]](#)
- Restenosis after stent placement or coronary artery bypass grafting [\[R\]](#)

However, this variant has also been associated with a decreased risk of:

- Hypertension [\[R\]](#), [\[R\]](#)
- Migraines [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Fibromuscular dysplasia [\[R\]](#)
- Spontaneous cervical and coronary artery dissection [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

Another well-researched variant is [rs12526453](#). Its major ‘C’ allele has been associated with an increased risk of:

- Myocardial infarction [\[R\]](#), [\[R\]](#)
- Coronary artery disease [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Coronary artery calcification [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Coronary artery stenosis [\[R\]](#)

This variant has also been linked to higher LDL cholesterol and ApoB levels, which may in part explain its association with heart disease [\[R\]](#).

However, a study of almost 1000 patients with ST-elevation myocardial infarction associated this variant with better long-term survival [\[R\]](#)



Predisposed to a worse PHACTR1 genetics based on 2 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PHACTR1	rs9349379	GG
TBC1D7	rs12526453	CC

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

CDKN2B-AS (Cardiovascular)

The most widely investigated *CDKN2B-AS* variant is [rs10757278](#). Its minor ‘G’ allele has been associated with an increased risk of:

- Coronary artery disease [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Ischemic stroke [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Myocardial infarction [\[R\]](#)
- Intracranial aneurysm [\[R\]](#), [\[R\]](#)
- Cancer (especially, breast and prostate) [\[R\]](#)

Another well-researched variant, the ‘G’ allele of [rs10757274](#), has been associated with an increased risk of:

- Coronary artery disease [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Ischemic stroke [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Sudden cardiac death [\[R\]](#)
- Angina pectoris [\[R\]](#)

The ‘G’ allele of [rs2383206](#) also increases *CDKN2B-AS* expression. This variant has been associated with an increased risk of:

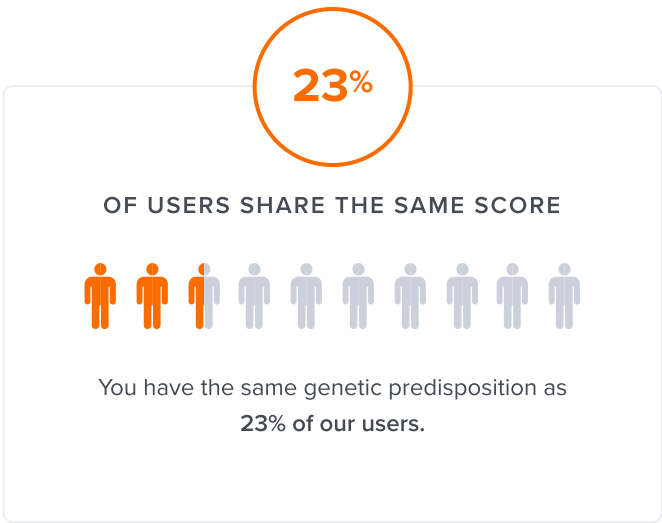
- Coronary artery disease [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Ischemic stroke [\[R\]](#), [\[R\]](#), [\[R\]](#)

Finally, the ‘G’ allele of [rs2383207](#) has been associated with an increased risk of coronary artery disease [\[R\]](#), [\[R\]](#), [\[R\]](#).

These variants are associated with lower expression of a region in this gene and are usually inherited together, meaning you will most likely have all or none of them [\[R\]](#).



Predisposed to a lower CDKN2B-AS activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CDKN2B	rs10757278	GG
CDKN2A	rs10757274	GG
CDKN2B	rs2383206	GG
CDKN2B	rs2383207	GG

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

PITX2 (Cardiovascular)

Several *PITX2* variants have been associated with an increased risk and recurrence of atrial fibrillation, as well as an increased risk of ischemic stroke. These include:

- ‘T’ of [rs2200733](#) [R, R, R, R, R, R, R, R]
- ‘T’ of [rs10033464](#) [R, R, R, R, R, R]
- ‘A’ of [rs1906591](#) [R, R, R]

Although their effects on PITX2 activity are poorly understood, reduced *PITX2* expression in the heart has been associated with an abnormal development of the atrial chambers. Based on this mechanism, these variants may reduce PITX2 activity [R].



Predisposed to a lower PITX2 activity based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PITX2	rs2200733	CT
PITX2	rs1906591	GA
PITX2	rs10033464	GG

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

ADRB1 (Cardiovascular)

The best-researched *ADRB1* polymorphism is [rs1801253](#) (Arg389). Its minor ‘C’ allele may increase the activation of the beta-1 adrenergic receptors and has been associated with [\[R\]](#):

- Higher blood pressure [\[R, R, R, R, R\]](#)
- Increased risk of cardiovascular disease [\[R, R, R, R\]](#)
- Increased risk of sudden cardiac death [\[R\]](#)
- Higher LDL cholesterol levels [\[R\]](#)
- Lower training-induced exercise tolerance [\[R\]](#)
- Increased risk of postoperative pain [\[R\]](#)

On the bright side, carriers may have a decreased risk of adverse effects in response to blood pressure medication (beta blockers) [\[R, R\]](#).

Another variant, ‘A’ of [rs1801252](#) (Ser49Gly), may increase ADRB1 stability and has been associated with [\[R\]](#):

- Higher blood pressure [\[R\]](#)
- Increased risk of cardiovascular disease [\[R\]](#)
- Increased risk of sudden cardiac death [\[R\]](#)
- Lower odds of LVEF recovery in heart failure patients [\[R\]](#)
- Lower renin levels [\[R\]](#)

However, this variant has also been associated with a better response to beta blockers. Moreover, people with resistant hypertension are less likely to carry this variant [\[R, R\]](#).

Finally, the ‘T’ allele of [rs10787516](#) has been associated with relatively higher blood pressure. This variant may also increase ADRB1 activity, leading to increased heart rate and contraction pressure [\[R, R\]](#).



Predisposed to higher ADRB1 activity based on 3 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ADRB1	rs1801253	CC
ADRB1	rs1801252	AA
ADRB1	rs10787516	TT

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

CPS1 (Cardiovascular, Kidney Health)

The best-characterized *CPS1* polymorphism is [rs1047891](#) (T1405N), formerly known as rs7422339. Its minor ‘A’ allele has been associated with lower urea levels in the blood and ammonia levels in the liver, suggesting lower enzyme activity. This variant has also been associated with higher LDL cholesterol, homocysteine, and glycine levels but lower HDL cholesterol and apoA1 levels, as well as with [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#):

- Hyperammonemia in epilepsy patients taking valproic acid [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Increased risk of coronary artery disease [\[R\]](#)
- Increased risk of migraine and CKD [\[R\]](#), [\[R\]](#)
- Lower platelet count [\[R\]](#)
- Lower eGFR [\[R\]](#)

However, this variant has been associated with a decreased risk of liver scarring in people with NAFLD and lower odds of necrotizing enterocolitis [\[R\]](#), [\[R\]](#), [\[R\]](#).

Another variant believed to decrease *CPS1* expression, ‘C’ of [rs715](#), has been associated with:

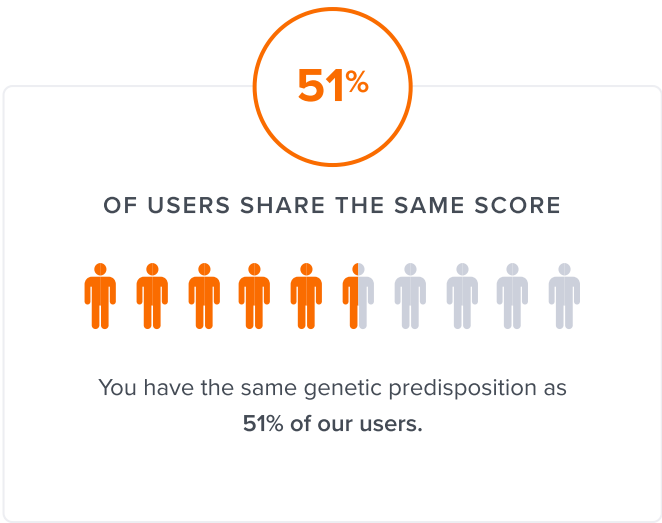
- Higher glycine and lower citrulline and TMAO levels [\[R\]](#), [\[R\]](#)
- Lower eGFR [\[R\]](#)
- Higher BMI [\[R\]](#)
- Decreased risk of macular telangiectasia type 2 [\[R\]](#)
- Decreased risk of severe coronary artery disease [\[R\]](#)

This variant is usually inherited together with rs1047891, meaning you will most likely have both or neither of them.



LOWER ACTIVITY

Likely lower CPS1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CPS1	rs1047891	AC
CPS1	rs715	CT

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

CELSR2-PSRC1-SORT1

(Cardiovascular)

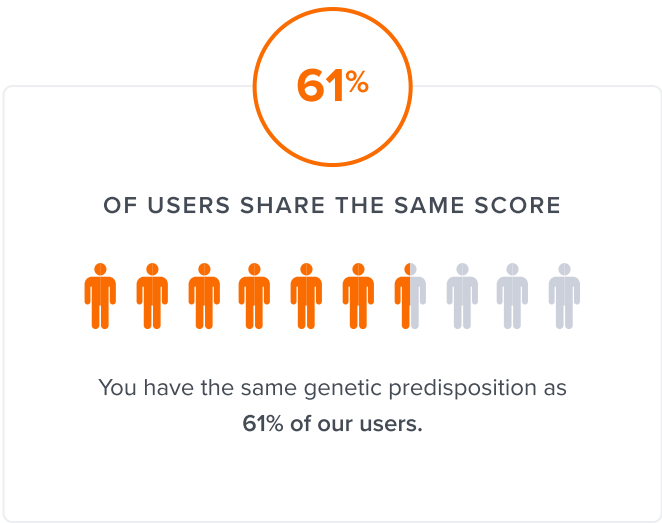
The best-characterized polymorphism within this cluster is [rs599839](#). Its minor ‘A’ allele may increase *PSRC1* and *SORT1* expression. This variant has been associated with higher total cholesterol, LDL cholesterol, ApoB, CRP, IL-1beta, and TNF-alpha levels, as well as with an increased risk of coronary artery disease and myocardial infarction [[R](#), [R](#), [R](#), [R](#), [R](#), [R](#), [R](#), [R](#), [R](#)].

Another well-researched polymorphism is [rs646776](#). Its minor ‘T’ allele may increase *PSRC1* and *SORT1* expression. This variant has been associated with higher total cholesterol, LDL cholesterol, and ApoB levels, as well as an increased risk of cardiovascular disease, including coronary artery disease, peripheral artery disease, and myocardial infarction [[R](#), [R](#), [R](#), [R](#), [R](#)].

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



Likely higher CELSR2-PSRC1-SORT1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SORT1	rs599839	AA
SORT1	rs646776	TT

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

NOS3 (Cardiovascular)

Among the different *NOS3* polymorphisms, [rs1799983](#) has been most widely studied. The ‘T’ variant produces a protein that can’t reach its activation sites in cell membranes, ultimately decreasing NO production [\[R\]](#).

In line with the beneficial cardiovascular effects of NO, the ‘T’ variant has been associated with an increased risk of coronary heart disease and heart attack in several studies. It’s also more frequent in children with congenital heart disease and predicts a faster progression of heart damage in people with diabetes [\[R, R, R, R\]](#).

However, overproducing ‘GG’ genotype may also have negative effects on the heart. It’s associated with reduced heart function in people with kidney disease, increased risk of death in those with high blood pressure, and heart failure in African-Brazilians [\[R, R, R\]](#).

The ‘T’ allele of [rs1549758](#) has also been associated with an increased risk of coronary heart disease and hypertension but is usually inherited with the ‘T’ allele of [rs1799983](#), meaning you will most likely have both or neither of them [\[R\]](#).

Another SNP, [rs2070744](#), is also linked to an increased risk of coronary heart disease. The ‘C’ allele can be bound by a protein that blocks *NOS3* production. However, the ‘T’ variant at this polymorphism is the one associated with myocardial infarction [\[R, R, R\]](#).

These variants may exert their harmful effects through their associations with:

- Higher blood pressure [\[R, R, R, R, R, R, R, R, R, R\]](#)
- Higher vessel stiffness and blood cholesterol [\[R, R, R, R\]](#)
- Increased risk of complications after heart surgery [\[R, R, R\]](#)
- Reduced effectiveness of conventional and alternative therapies [\[R, R\]](#)

The minor variants of [rs179983](#) and [rs2070744](#) have also been associated with:

- Worse [athletic performance](#) in power sports [\[R, R, R, R, R, R\]](#)
- Longer and more frequent [migraines](#) [\[R, R, R, R\]](#)



Likely typical NOS3 activity based on 2 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NOS3	rs2070744	TC
NOS3	rs1549758	CT
NOS3	rs3918226	CT

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

On the bright side, they are also linked to:

- Improved performance in aerobic sports and soccer [R, R, R]
- Greater decreases in triglycerides, cholesterol, and blood pressure in [response to unsaturated fats](#) such as [omega-3 fatty acids](#) and [extra virgin olive oil](#) [R, R, R, R, R]

Finally, the ‘T’ allele of [rs3918226](#) also results in lower NOS3 levels and is associated with a higher risk and severity of heart and coronary events. Fortunately, this allele is extremely rare and most people (81-99%) have the ‘CC’ genotype [R, R, R].

EDN1 (Blood Pressure)

The main *EDN1* gene variant is [rs5370](#), also known as Lys198Asn. Its minor ‘T’ allele may increase endothelin 1 levels. This variant has been associated with an increased risk of [\[R\]](#):

- Hypertension [\[R, R, R\]](#)
- Pulmonary hypertension [\[R\]](#)
- Ischemic stroke [\[R, R, R, R\]](#)
- Preeclampsia [\[R\]](#)
- Coronary artery disease [\[R\]](#)
- Coronary atherosclerosis [\[R, R\]](#)
- Lupus [\[R\]](#)
- Age-related hearing impairment [\[R\]](#)

The risk of hypertension may be higher in low-fit subjects. Moreover, carriers of the ‘T’ allele may respond less to endurance exercise training [\[R\]](#).

This variant has also been associated with lower HDL cholesterol levels [\[R\]](#).

Another well-researched polymorphism is [rs1800541](#). Its major ‘T’ allele may increase endothelin-1 levels and has been associated with an increased risk of [\[R\]](#):

- Subarachnoid hemorrhage [\[R\]](#)
- Diabetic kidney disease [\[R\]](#)
- Chemoresistant or metastatic osteosarcoma [\[R, R\]](#)
- Hormone-refractory prostate cancer [\[R\]](#)
- Sudden infant death [\[R\]](#)

On the bright side, this variant has been associated with a decreased risk of fibromyalgia [\[R\]](#).

The ‘T’ allele of [rs2070699](#) may also increase endothelin-1 levels and has been associated with an increased risk of [\[R\]](#):

- Pulmonary hypertension [\[R\]](#)
- Aneurysm rebleeding after subarachnoid hemorrhage [\[R\]](#)
- Lupus [\[R\]](#)
- Acute mountain sickness [\[R\]](#)
- Colorectal cancer [\[R, R\]](#)

In contrast, this variant has been associated with a decreased risk of ischemic stroke, chemoresistant osteosarcoma, and hormone-refractory prostate cancer [\[R, R, R\]](#).



Predisposed to a typical EDN1 activity based on 4 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
EDN1	rs5370	GT
EDN1	rs1800541	TG
EDN1	rs2070699	TG
EDN1	rs3087459	AC

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

Finally, the ‘A’ allele of [rs3087459](#) has been associated with higher endothelin-1 levels and an increased risk of [\[R\]](#):

- Coronary artery disease [\[R\]](#)
- Acute coronary syndrome [\[R\]](#)

However, this variant has been associated with a decreased risk of lupus [\[R\]](#).

ACYP2 (Cardiovascular, Cancer)

Several minor alleles of *ACYP2* polymorphisms have been associated with an increased risk of ischemic stroke. These include [\[R\]](#):

- ‘A’ of [rs11125529](#) (also associated with coronary heart disease) [\[R\]](#)
- ‘A’ of [rs12615793](#) (especially in smokers) [\[R\]](#)
- ‘T’ of [rs843711](#)
- ‘A’ of [rs843706](#)
- ‘C’ of [rs17045754](#)

These and other variants have also been associated with an increased risk of the following cancer types:

- Stomach (rs11125529, rs12615793, rs84371, rs17045754, rs843706, ‘G’ of [rs6713088](#)) [\[R\]](#)
- Oropharyngeal (rs11125529) [\[R\]](#)
- Laryngeal (rs11125529, rs12615793, rs84371, rs17045754) [\[R\]](#)
- Esophageal (rs11125529, rs17045754) [\[R\]](#)
- Kidney (rs843711, rs6713088) [\[R\]](#)
- Liver (rs84371, rs843706, rs6713088, ‘G’ of [rs843645](#)) [\[R\]](#), [\[R\]](#)
- Colorectal (rs84371, rs843706, rs6713088, rs843645) [\[R\]](#)

However, they have been associated with a decreased risk of breast cancer (rs11125529, rs84371, rs17045754, rs843706) [\[R\]](#).

The ‘G’ allele of rs6713088 has also been associated with increased telomerase levels, while the ‘A’ allele of rs11125529 has been associated with longer telomere length. This suggests these variants may decrease ACYP2 activity and contribute to cancer through enhanced immortalization of malignant cells [\[R\]](#), [\[R\]](#).



Likely typical ACYP2 activity based on 7 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TSPYL6	rs11125529	CC
TSPYL6	rs17045754	GG
ACYP2	rs12615793	GG
TSPYL6	rs843711	CC
TSPYL6	rs843706	CC
ACYP2	rs843645	TT
ACYP2	rs6713088	CC

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

ACMSD (Cardiovascular)

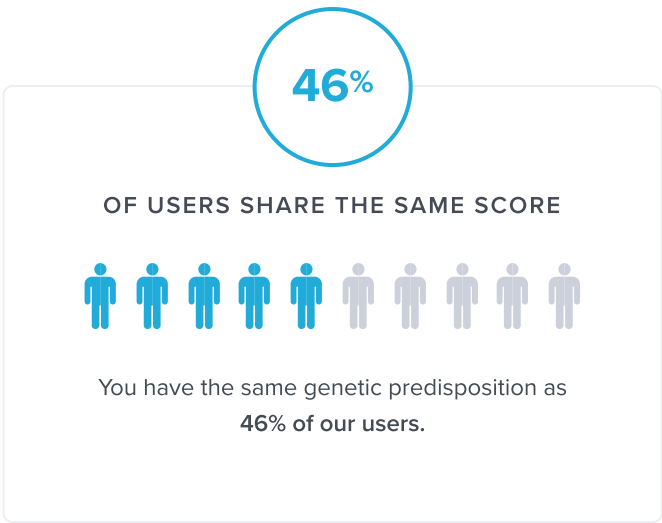
A study of 1,162 participants associated the ‘T’ allele of an *ACMSD* polymorphism ([rs10496731](#)) with higher 4PY and VCAM-1 levels, as well as with an increased risk of cardiovascular events. The risk may be higher in people with high niacin intake [\[R\]](#).

The same study also associated the ‘C’ allele of [rs6430553](#) and the ‘G’ allele of [rs6729702](#) with higher 4PY levels, suggesting similar negative effects on cardiovascular risk [\[R\]](#).

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



Predisposed to typical ACMSD activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CCNT2	rs10496731	TG
CCNT2	rs6430553	CT
CCNT2	rs6729702	GA

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

BPIFB4 (Cardiovascular & Longevity)

The main BPIFB4 variant is [rs2070325](#). People with two copies of its minor allele (GG) may have [\[R, R, R, R\]](#):

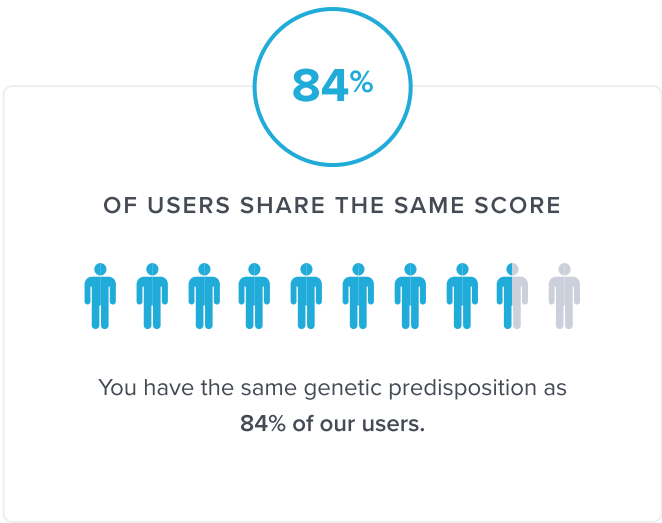
- Longer lifespan
- Reduced frailty in older age
- Lower blood pressure

This variant likely increases BPIFB4 activity and boosts blood vessel function through nitric oxide [\[R, R\]](#).



TYPICAL ACTIVITY

Predisposed to typical BPIFB4 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
BPIFB4	rs2070325	AA

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

SCARB1 (Cardiovascular)

The main *SCARB1* polymorphism is [rs5888](#). Its minor ‘A’ allele has been associated with lower SCARB1 levels. This variant has been associated with a decreased risk of coronary heart disease and myocardial infarction, lower triglycerides, and higher HDL cholesterol levels [\[R, R, R, R, R\]](#).

People with this variant may also respond better to vitamin A supplementation in terms of decreased adiposity and cardiometabolic risk. Moreover, women with acute coronary syndrome may lower their total cholesterol, LDL cholesterol, and ApoB levels after atorvastatin treatment more if they carry this variant [\[R, R, R\]](#).

Another well-researched variant is [rs4238001](#). Its ‘T’ allele has also been associated with lower levels of the SCARB1 protein and an increased risk of coronary heart disease and myocardial infarction [\[R, R, R\]](#).

This variant has also been linked to higher LDL cholesterol levels, especially in people eating a diet high in saturated fats. In contrast, people with myocardial infarction taking rosuvastatin may raise their HDL cholesterol levels more if they have the ‘CC’ genotype [\[R, R\]](#).

Finally, the minor ‘C’ allele of [rs10846744](#) may not affect *SCARB1* expression but has been suggested to affect the expression of genes involved in cell death, inflammation, and endothelial injury repair. This variant has been associated with an increased risk of atherosclerosis and coronary artery disease, as well as with higher total cholesterol, LDL cholesterol, triglycerides, and Lp-PLA2 activity [\[R, R, R, R, R, R, R, R\]](#).



Predisposed to a typical SCARB1 genetics based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SCARB1	rs5888	AG
SCARB1	rs10846744	GC
SCARB1	rs4238001	CC

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

FN1 (Neurodegeneration, Cardiovascular)

A study of 3,578 individuals identified two rare *FN1* variants that decrease the risk of Alzheimer’s disease in carriers of the *APOE* ε4 allele: ‘T’ of [rs140926439](#) and ‘A’ of [rs116558455](#). Based on the potential contribution of fibronectin to neurodegeneration, the authors speculated that these variants reduce the levels of this protein. A more recent study confirmed the protective role of the rs140926439 variant [\[R, R\]](#).

Another variant decreasing fibronectin levels, the minor ‘T’ allele of [rs1250229](#), wasn’t identified in the previously-mentioned studies but has been associated with a reduced risk of coronary artery disease and lower total and LDL cholesterol levels [\[R, R, R, R, R, R\]](#).



Predisposed to typical FN1 activity based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
FN1	rs140926439	CC
FN1	rs116558455	GG
ATIC	rs1250229	CC

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

CORIN (Cardiovascular)

A study of 6814 Black Americans identified two minor *CORIN* alleles, ‘T’ of [rs111253292](#) (Q568P) and ‘A’ of [rs75770792](#) (T555I) with higher blood pressure and increased risk of hypertension. These variants, encoding versions of the protein with impaired activation, are found in 12% of Black Americans and may, in part, explain the higher risk of hypertension observed in this ethnicity [\[R, R\]](#).

These variants have also been associated with an increased risk of death or hospitalization in patients with heart failure (rs111253292) and left ventricular hypertrophy in patients with untreated hypertension (rs75770792) [\[R, R\]](#).

However, the most recent study failed to associate these variants with changes in blood pressure or ANP levels [\[R\]](#).

In Chinese populations, the following *CORIN* variants have been associated with higher blood pressure and an increased risk of hypertension:

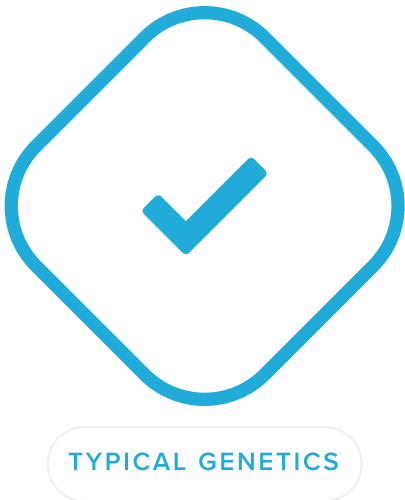
- ‘T’ of [rs2271037](#) [\[R\]](#)
- ‘G’ of [rs3749585](#) [\[R\]](#)
- ‘G’ of [rs73814824](#) [\[R\]](#)

A study of 124 families from 7 Chinese villages associated the ‘G’ variant of [rs3749584](#) with a greater reduction of DBP and MAP in response to a low-salt diet, while ‘T’ of [rs4695253](#) and ‘A’ of [rs17654278](#) were associated with a greater reduction of PP with this diet [\[R\]](#).

Moreover, the same study associated the following variants with a lower increment of SBP, DBP, and MAP in response to a high-salt diet [\[R\]](#):

- ‘T’ of rs2271037
- ‘T’ of [rs2271036](#)
- ‘A’ of [rs12641823](#)
- ‘T’ of [rs2351783](#)

The rs2271036 and rs2271037 alleles have also been associated with a decreased risk of preeclampsia in Caucasian women [\[R\]](#).



Predisposed to a typical CORIN genetics based on 11 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CORIN	rs111253292	TT
CORIN	rs73814824	GG
NFXL1	rs2271037	TT
NFXL1	rs3749584	CC
CORIN	rs17654278	GG
ATP10D	rs4695253	CT
CORIN	rs2351783	CT
CORIN	rs75770792	GG
CORIN	rs3749585	AA
NFXL1	rs2271036	TT
CORIN	rs12641823	AA

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

ACE (Fitness/ Cardiovascular)

The two main ACE gene variants, [rs4341](#) and [rs4343](#), influence gene and enzyme activity. Their “G” alleles may increase ACE activity and levels. In line with this, they are linked to **high blood pressure and heart disease** [\[R, R, R, R\]](#).

Regarding athletic performance, higher ACE activity may favor short, high-intensity bursts of activity. This makes it advantageous for **power-based sports** [\[R, R, R\]](#).

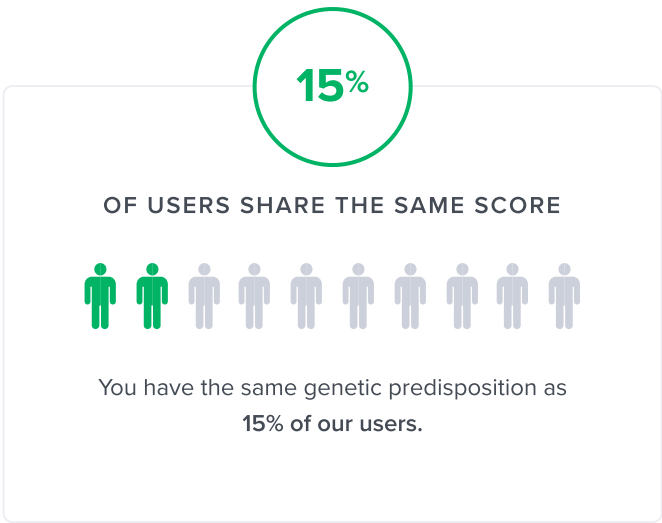
Conversely, **rs4341-C and rs4343-A** are linked to lower ACE activity, which may offer some protection against hypertension and cardiovascular conditions. Lower ACE activity can enhance **endurance** by improving blood flow and oxygen delivery to muscles during prolonged physical activity [\[R, R, R, R\]](#).

Please note: Some people's genetic files don't contain the rs4341 variant, so we didn't include it in the model. However, this variant is almost always inherited with rs4343, so one of them is sufficient to estimate your ACE genetics.



LOWER ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ACE	rs4343	AA

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

GCH1 (Cardiovascular)

The following three variants may **reduce GCH1 activity**. They have been linked to **lower BH4 levels**, especially in people with both copies [\[R\]](#):

- [rs10483639](#)-C
- [rs3783641](#)-A
- [rs8007267](#)-T

People with these variants may be prone to **blood vessel issues**, especially if they have diabetes or heart disease. Lower BH4 levels mean less nitric oxide, a substance that widens the blood vessels and keeps them healthy [\[R\]](#), [\[R\]](#).

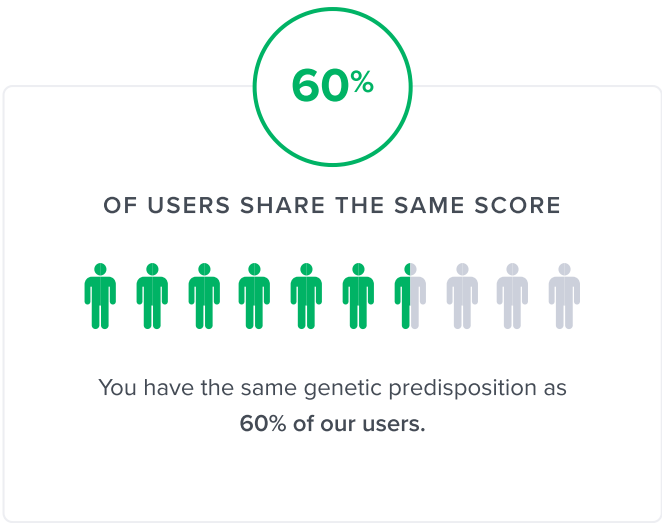
On the other hand, BH4 is involved in pain signalling, and these variants are linked to **reduced pain sensitivity** [\[R\]](#), [\[R\]](#), [\[R\]](#).

Note that these variants are often inherited together, meaning you will likely have either all or none.



HIGHER ACTIVITY

Likely higher GCH1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GCH1	rs10483639	GG
GCH1	rs8007267	CC
GCH1	rs3783641	TT

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

GLUL (Cardiovascular)

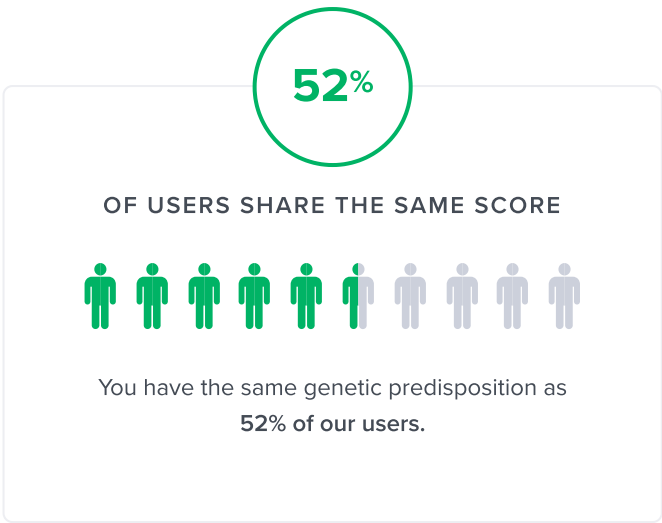
The main *GLUL* variant is [rs10911021](#). Its minor ‘T’ allele has been associated with a decreased risk and mortality of coronary heart disease in patients with type 2 diabetes. Moreover, this variant has been associated with a better blood lipid profile (lower LDL and lipoprotein(a) but higher HDL cholesterol levels) and better antioxidant status (lower MDA and GSSG and higher GSH levels) [[R](#), [R](#), [R](#), [R](#), [R](#), [R](#)].

This variant has been associated with higher *GLUL* expression, resulting in enhanced ammonia and glutamate detoxification. The authors of the study suggested that supplementation with glutamine may help prevent coronary heart disease in carriers of the ‘C’ allele [[R](#)].



HIGHER ACTIVITY

Predisposed to a higher **GLUL** activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ZNF648	rs10911021	TC

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



Lipid Metabolism

Cholesterol and triglycerides (blood lipids) are key lab markers when it comes to heart health! Too much can increase the risk of health issues like heart disease.

How your body handles fats and cholesterol depends on a variety of factors, including your genetics. Variants of genes involved in the metabolism and transport of blood lipids, such as *APOB*, *CETP*, *LDLR*, or *FABP2*, may cause their buildup in the blood vessels, ultimately increasing the risk of heart disease. Knowing your genetic risks can help you implement lifestyle changes to counteract the effects of these variants.

HIGHER ACTIVITY PCSK9 (Cholesterol) Likely higher PCSK9 activity	LOWER ACTIVITY DOCK7 (Blood Lipids) Likely lower DOCK7 activity	TYPICAL ACTIVITY APOC3 (Blood Lipids/Longevity) Likely typical APOC3 activity
E3/E3 APOE You carry two APOE ε3 variants	TYPICAL ACTIVITY HMGCR (Cholesterol) Likely typical HMGCR activity	TYPICAL ACTIVITY APOA2 (Weight, Blood Lipids) Likely typical APOA2 activity
TYPICAL ACTIVITY ABCG8 (Cholesterol & Gallstones) Predisposed to typical ABCG8 activity	TYPICAL ACTIVITY APOB Gene (Cardiovascular) Likely typical APOB activity	TYPICAL GENETICS ABCA1 (Cholesterol) Likely typical ABCA1 genetics
TYPICAL GENETICS ABCA6 (Cholesterol) Likely typical ABCA6 genetics	TYPICAL ACTIVITY APOA5 (Cardiovascular) Predisposed to typical APOA5 activity	TYPICAL ACTIVITY LPA (Blood Lipids & Heart Health) Predisposed to typical LPA activity



TYPICAL ACTIVITY

**LDLR (Cholesterol,
Cardiovascular)**

Predisposed to typical LDLR activity



LOWER ACTIVITY

**FABP2 (Blood Sugar/
Cardiovascular)**

Likely lower FABP2 activity



TYPICAL GENETICS

**SCARB1
(Cardiovascular)**

Predisposed to a typical SCARB1 genetics



TYPICAL ACTIVITY

DHCR7 (Cholesterol)

Predisposed to typical DHCR7 activity



TYPICAL ACTIVITY

APOA1 (Blood Lipids)

Predisposed to a typical APOA1 activity



LOWER ACTIVITY

**CETP (Cholesterol/
Longevity)**

Likely lower CETP activity



HIGHER ACTIVITY

LIPC (Cardiovascular)

Likely higher LIPC activity



LOWER ACTIVITY

**MLXIPL (Triglycerides,
Cardiovascular)**

Likely lower MLXIPL activity



LOWER ACTIVITY

**SOAT1 (Cholesterol/
Cognition)**

Likely lower SOAT1 activity

PCSK9 (Cholesterol)

One PCSK9 variant, [rs562556-G](#), is linked to **lower LDL cholesterol levels and lower odds of heart disease** [\[R\]](#), [\[R\]](#).

In a large study, two rare PCSK9 variants have shown similar protective associations [\[R\]](#):

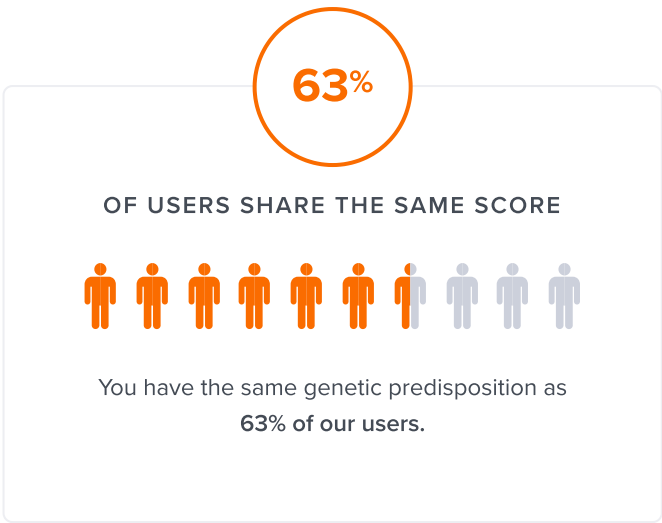
- [rs11591147-T \(R46L\)](#): 15% lower LDL and 2 times lower odds of heart disease in people of European ancestry.
- [rs28362286-A \(C679X\)](#): 28% lower LDL and 9 times lower odds of heart disease in people of African ancestry.

The second variant is extremely rare and exists only in people of African ancestry.

These variants reduce PCSK9 activity, which then increases the number of LDL receptors and enhances cholesterol metabolism.



Likely higher PCSK9 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PCSK9	rs562556	AA
PCSK9	rs11591147	GG
PCSK9	rs28362286	CC

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

DOCK7 (Blood Lipids)

One of the most widely researched *DOCK7* polymorphism is [rs10889353](#). Its minor ‘C’ allele has been associated with higher *DOCK7* expression, lower total cholesterol, LDL cholesterol, and triglyceride levels, and a decreased risk of coronary heart disease [\[R, R, R, R, R\]](#).

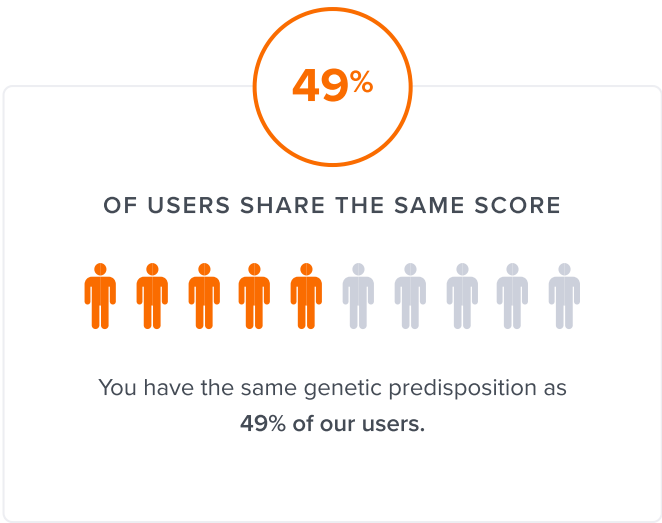
Another well-characterized polymorphism is [rs2131925](#). Its minor ‘G’ allele has been associated with lower total cholesterol, LDL cholesterol, and triglyceride levels, higher vitamin D levels, and an a decreased risk of hypertension [\[R, R, R, R, R, R, R, R\]](#).

These variants are usually inherited together in people of Europea ancestry, meaning you will most likely have both or neither of them.



LOWER ACTIVITY

Likely lower DOCK7 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
DOCK7	rs2131925	TT
DOCK7	rs10889353	AA

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

APOC3 (Blood Lipids/Longevity)

The main *APOC3* variants, [rs5128](#) and [rs2542052](#), may influence the metabolism of blood lipids. Their “**C**” alleles are linked to [\[R\]](#), [\[R\]](#), [\[R\]](#):

- Lower triglycerides
- Lower LDL or “bad” cholesterol levels
- Higher HDL or “good” cholesterol levels

However, their impact on cardiovascular health is less clear. A meta-analysis of 79 studies looked at the link between stroke and APOC3, along with many other related genes. According to the results, rs5128 and other *APOC3* SNPs have no association with stroke [\[R\]](#).

When it comes to direct links with longevity, a study looked at rs2542052 in Ashkenazi Jews. The results suggest that the “**CC**” **genotype** may be more common in people who live past 100 years old. They also had better cardiovascular health and glucose metabolism [\[R\]](#).

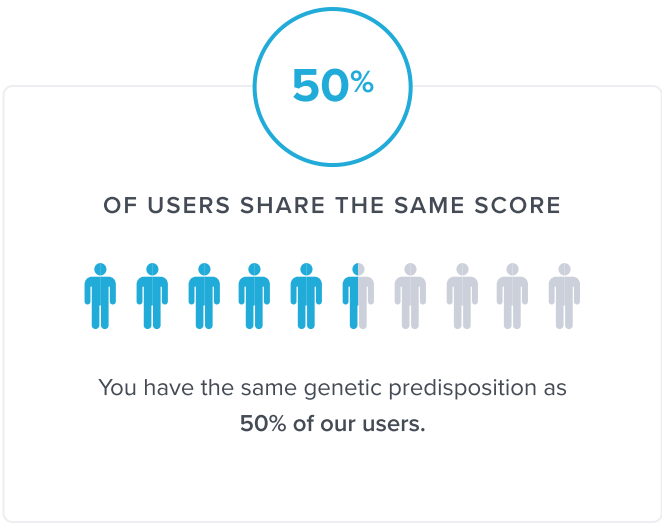
However, one study looked at 749 American Caucasians who have exceptionally long lifespans and found no association between rs2542052 and longevity [\[R\]](#).

Finally, the rare “**A**” allele of [rs138326449](#) encodes a version of the protein with impaired function. This variant has been associated with decreased triglyceride and VLDL cholesterol but increased HDL cholesterol levels [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).



TYPICAL ACTIVITY

Likely typical APOC3 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SIDT2	rs2542052	AC
PCSK7	rs5128	CC

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

APOE

Key Takeaways:

- If you carry one or both **ε4** variants, your risk for Alzheimer's disease may be higher.
- The risk is greatest for late onset (after age 65) Alzheimer's disease.
- Even if your risk is higher due to the **ε4** variants, numerous other factors from your environment to lifestyle to other genetic variants impact overall risk.
- People with both variants may never get Alzheimer's, and some who have neither variant can get the disease.

There are three major forms (variants) of the *APOE* gene. These are called ε2, ε3, and ε4. You can have two copies of the same variant or two different variants [R, R].

ε2, ε3, and ε4 change the shape of the ApoE protein. This can impact how well ApoE functions [R, R].

ε3 is the most common variant. It makes a protein that is good at clearing plaque from the brain and fats from the blood. Most people have two ε3 variants and a typical risk of Alzheimer's disease [R].

ε4 is less common. It makes a protein that is not as good at clearing plaque from the brain and fats from the blood. ε4 has been linked to a higher risk of Alzheimer's disease and artery hardening [R, R].

ε2 is another less common variant. It makes a protein that is better than ε3 at removing plaque from the brain, but not as good at removing fats from the blood. ε2 has been linked to a lower risk of Alzheimer's disease [R, R, R].

However, it has also been linked to a higher risk of artery hardening in people with two ε2 variants and an underlying chronic health condition, such as obesity or diabetes [R, R, R].

Did you know? The **ε4** variant was much more common among ancient hunter-gatherers. Scientists suggest this variant might have improved their [R]:



E3/E3

You carry two APOE ε3 variants based on the genetic variants we looked at

62%

OF USERS SHARE THE SAME SCORE



You have the same genetic predisposition as 62% of our users.

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
APOE	rs7412	CC
APOE	rs429358	TT

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

- Inflammatory response to germs in the wilderness
- Vitamin D status in less sunny European areas
- Aerobic endurance, crucial for a hunter-gatherer lifestyle

As humans largely switched to farming, some effects of this variant became useless or even harmful. For this reason, evolution strongly favored the **ε3** variant in ancient farmers and their modern descendants [\[R\]](#).

HMGCR (Cholesterol)

The most well-researched *HMGCR* polymorphism is [rs3846662](#). Its ‘G’ allele results in an increased production of an inactive HMGCR version and has been associated with higher LDL apoB, and total cholesterol but lower HDL cholesterol levels. On the bright side, carriers of this variant may lower their LDL cholesterol more in response to statins [\[R, R, R, R, R, R, R, R, R, R, R, R, R\]](#).

This allele has also been associated with an increased risk of:

- Alzheimer’s disease [\[R, R, R, R, R\]](#)
- Mild cognitive impairment [\[R\]](#)
- Myocardial infarction [\[R\]](#)
- Overweight, especially in those with low soy intake [\[R\]](#)

Another well-researched polymorphism is [rs12916](#). Its ‘C’ allele, linked to lower *HMGCR* expression in the liver, has been associated with higher total and LDL cholesterol levels and increased risk of ovarian cancer, but also with an increased effectiveness of statin treatment [\[R, R, R, R, R\]](#).

This variant has also been associated with:

- Insomnia [\[R\]](#)
- Prostate cancer [\[R\]](#)
- Aortic aneurysm [\[R\]](#)
- Premature triple-vessel coronary disease and residual cholesterol risk [\[R, R\]](#)

The ‘T’ allele of [rs17244841](#) has been associated with a reduced effectiveness of statin therapy at lowering LDL cholesterol. Interestingly, a study of 306 patients with Parkinson’s disease found that those carrying this allele had higher HDL cholesterol levels [\[R, R, R, R, R\]](#).

Finally, the ‘G’ allele at [rs17238540](#) has been associated with a reduced effectiveness of statin therapy at lowering cholesterol and triglycerides, as well as with [\[R, R, R\]](#):

- Greater triglyceride decrease in response to a low-fat, high-fiber diet [\[R\]](#)
- Higher blood pressure and increased stroke risk [\[R\]](#)

Because this variant and rs17244841 are usually inherited together, you will most likely carry either both or neither of them.



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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HMGCR	rs3846662	GA
HMGCR	rs12916	CT
ANKRD31	rs17244841	AA
HMGCR	rs17238540	TT

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

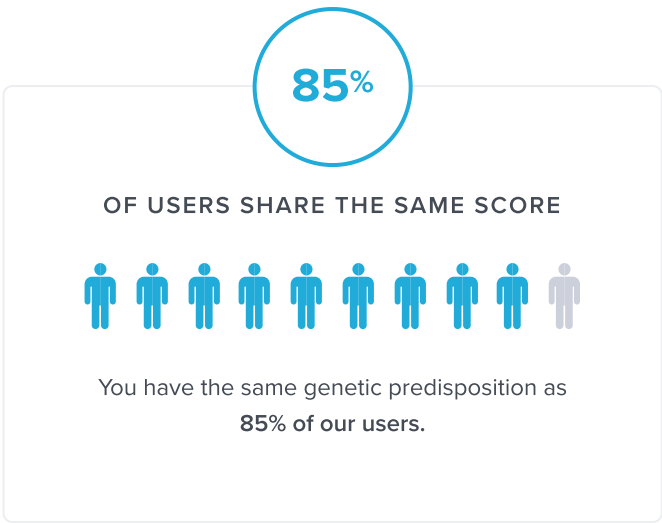
This *APOA2* variant may increase the levels of [ghrelin](#) or the “hunger hormone.” A study of 1,225 obese subjects found that people with the “GG” genotype who consume more SF have higher ghrelin levels. In other words, SF fails to satiate their hunger [\[R\]](#).

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



TYPICAL ACTIVITY

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



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.

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

ABCG8 (Cholesterol & Gallstones)

[ABCG8](#) promotes the removal of excess cholesterol through bile. This effect is good in moderation but may contribute to gallstones if too much cholesterol is processed [\[R\]](#).

In line with this, variants that increase ABCG8 activity are linked to **lower total and LDL cholesterol but higher odds of gallstones**. They include:

- [rs11887534](#)-C
- [rs6544713](#)-C
- [rs4148217](#)-A



Predisposed to typical ABCG8 activity based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ABCG8	rs4148217	AC
ABCG8	rs11887534	GG
ABCG8	rs6544713	CC

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

APOB Gene (Cardiovascular)

The best-studied *APOB* polymorphism is [rs693](#), also known as Xbal. Its minor ‘A’ allele may increase APOB activity, leading to higher total cholesterol, LDL cholesterol, VLDL cholesterol, apoB, and triglycerides but lower levels of HDL cholesterol. This variant has been associated with an increased risk of [\[R, R, R, R, R, R, R, R\]](#):

- Coronary artery disease [\[R\]](#)
- Metabolic syndrome [\[R\]](#)
- Calcific aortic stenosis [\[R\]](#)
- Ischemic stroke [\[R, R, R\]](#)
- Gallstone disease [\[R, R\]](#)

Another well-characterized *APOB* variant is [rs515135](#). Its minor ‘T’ allele has been associated with higher total and LDL cholesterol levels, as well as with an increased risk of coronary artery disease [\[R, R, R, R, R, R\]](#).

Another variant linked to increased total cholesterol, LDL cholesterol, and ApoB levels, ‘T’ at [rs1801701](#), has been associated with an increased risk of [\[R\]](#):

- Myocardial infarction [\[R\]](#)
- Coronary artery disease [\[R\]](#)
- Ischemic stroke [\[R\]](#)

Finally, the minor ‘A’ allele of [rs934197](#) increases *APOB* expression and has been associated with higher total and LDL cholesterol levels [\[R, R, R, R, R\]](#).



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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
APOB	rs693	AG
APOB	rs934197	AG
APOB	rs515135	CC
APOB	rs1801701	CC

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

ABCA1 (Cholesterol)

A variant in the ABCA1 gene, [rs1883025-T](#), is linked to **lower levels of cholesterol (all subtypes) and lower heart disease rates** [\[R\]](#).

This variant is also linked to a lower risk of **age-related macular degeneration** (AMD), an eye condition that can cause vision loss. It may help remove cholesterol from cells in the macula, a critical part of the retina [\[R\]](#).

Another notable ABCA1 variant is [rs2230808](#), with the “T” allele linked to lower cholesterol levels and lower inflammation [\[R\]](#).

ABCA1 variants have robust links with HDL or “good” cholesterol. **Despite the reduction in HDL, the net effect on heart health is protective**, likely due to LDL reduction [\[R\]](#), [\[R\]](#).

A rare variant, [rs9282541-A](#) (R230C), was linked to lower HDL levels and poor metabolic profile in Mexican women who consumed a high-carb diet. Women with this variant on a high-fat diet had higher LDL and better metabolic profiles [\[R\]](#).



TYPICAL GENETICS

Likely typical ABCA1 genetics based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ABCA1	rs1883025	CC
ABCA1	rs2230808	CC
ABCA1	rs9282541	GG

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

ABCA6 (Cholesterol)

A rare ABCA6 variant, [rs77542162-G](#), is linked to **higher levels** of [\[R\]](#):

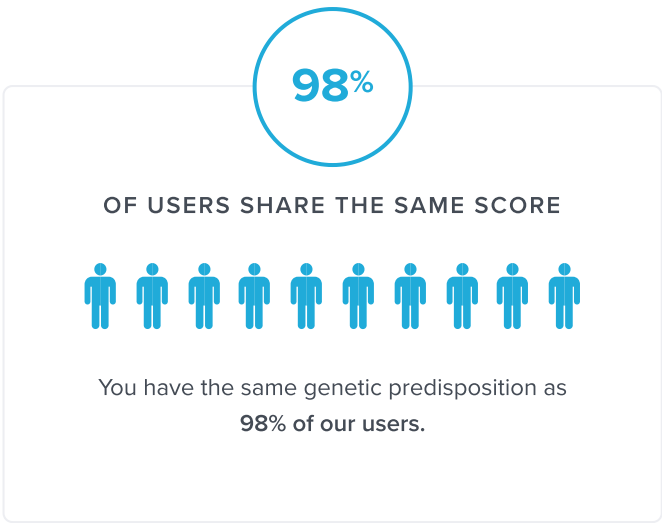
- Total cholesterol
- LDL cholesterol
- ApoB

Interestingly, people with this variant may see a greater **LDL reduction from fish oil supplements** [\[R\]](#).

While scientists haven't fully mapped out all of ABCA6's functions, we know it's particularly active in the liver, the body's main cholesterol processing center. ABCA6 controls how cells handle cholesterol and other fatty molecules, potentially influencing how much cholesterol moves in and out of cells.



Likely typical ABCA6 genetics based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ABCA6	rs77542162	AA

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

APOA5 (Cardiovascular)

Different *APOA5* variants are linked to impaired metabolic and cardiovascular health, more precisely [\[R, R, R, R, R, R, R, R\]](#):

- Higher triglycerides, LDL, and apoB levels
- Lower HDL and vitamin D levels
- Heart problems

They may reduce **APOA5 activity**, impairing the removal of excess fat and cholesterol from the blood [\[R\]](#).



Predisposed to typical APOA5 activity based on 6 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
APOA5	rs2075291	CC
PCSK7	rs662799	AA
SIDT2	rs651821	TT
SIDT2	rs2266788	AA
SIDT2	rs964184	CC
APOA5	rs3135506	GG

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

LPA (Blood Lipids & Heart Health)

In line with the *LPA* gene roles, its variants are linked to [\[R\]](#), [\[R\]](#):

- Lipoprotein(a) levels
- Total and LDL cholesterol levels
- Heart disease (CAD)

These variants likely **increase LPA activity**, resulting in higher Lp(a) levels and negative downstream effects.

In one large study, low-dose aspirin canceled out the cardiovascular risk from the *LPA* gene variant [rs3798220](#)-C [\[R\]](#).



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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
LPA	rs41272114	CC
LPA	rs6415084	TT
LPA	rs1853021	GG
SLC22A3	rs10755578	GC
SLC22A3	rs9364559	AA
LPA	rs10455872	AA
LPA	rs3798220	TT

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

LDLR (Cholesterol, Cardiovascular)

The best-researched *LDLR* polymorphism is [rs688](#). Its minor ‘T’ allele may decrease LDLR production and has been associated with higher total and LDL cholesterol levels, as well as with an increased risk of [\[R, R, R, R, R, R, R\]](#):

- Coronary artery disease [\[R, R\]](#)
- Ischemic stroke [\[R, R, R, R, R\]](#)
- Cardiovascular disease in end-stage kidney disease patients [\[R\]](#)

Similarly, the minor ‘C’ allele of [rs5925](#) has been associated with a decreased *LDLR* expression, higher total cholesterol, LDL cholesterol, and triglyceride levels, and an increased risk of ischemic stroke and coronary artery disease [\[R, R, R, R, R, R\]](#).

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

Another well-characterized polymorphism is [rs6511720](#). Its minor ‘T’ allele may increase *LDLR* expression by creating binding sites for proteins that enhance it in the gene sequence. Multiple studies have associated this allele with lower LDL cholesterol levels, as well as with a decreased risk of [\[R, R, R, R, R, R, R, R, R, R, R\]](#):

- Coronary heart disease [\[R, R, R\]](#)
- Ischemic stroke [\[R\]](#)
- Myocardial infarction [\[R\]](#)
- Atherosclerosis [\[R\]](#)

Another variant, the minor ‘T’ allele of [rs1122608](#), has been associated with lower total cholesterol and LDL cholesterol levels, as well as with a decreased risk of [\[R\]](#):

- Coronary artery disease [\[R, R\]](#)
- Myocardial infarction, especially in people who smoke or drink alcohol [\[R, R\]](#)
- Ischemic stroke [\[R\]](#)

Interestingly, this variant may decrease the expression of a nearby gene ([SRSF3](#)), resulting in lower levels of the IL-1β cytokine [\[R\]](#).



Predisposed to typical LDLR activity based on 5 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
LDLR	rs6511720	GG
LDLR	rs2228671	CC
LDLR	rs688	TC
LDLR	rs5925	CT
SMARCA4	rs1122608	TG

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

Finally, the minor ‘T’ allele of [rs2228671](#), has also been associated with lower LDL cholesterol levels and a decreased risk of coronary artery disease [[R](#), [R](#), [R](#)].

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

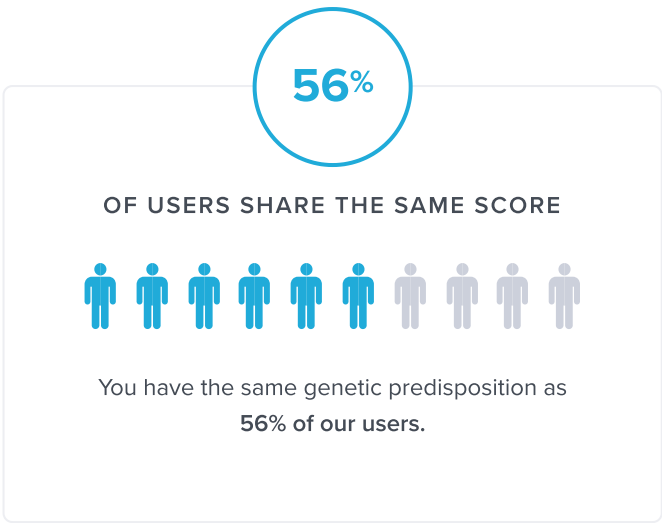
Moreover, women with the ‘C’ variant lowered their total cholesterol, triglycerides, fasting glucose, and HbA1c more after following a low-glycemic-index diet for 4-5 weeks in a study on 165 patients with type 2 diabetes. However, this variant didn’t modify the effectiveness of the intervention in men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

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



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.

SCARB1 (Cardiovascular)

The main *SCARB1* polymorphism is [rs5888](#). Its minor ‘A’ allele has been associated with lower SCARB1 levels. This variant has been associated with a decreased risk of coronary heart disease and myocardial infarction, lower triglycerides, and higher HDL cholesterol levels [\[R, R, R, R, R\]](#).

People with this variant may also respond better to vitamin A supplementation in terms of decreased adiposity and cardiometabolic risk. Moreover, women with acute coronary syndrome may lower their total cholesterol, LDL cholesterol, and ApoB levels after atorvastatin treatment more if they carry this variant [\[R, R, R\]](#).

Another well-researched variant is [rs4238001](#). Its ‘T’ allele has also been associated with lower levels of the SCARB1 protein and an increased risk of coronary heart disease and myocardial infarction [\[R, R, R\]](#).

This variant has also been linked to higher LDL cholesterol levels, especially in people eating a diet high in saturated fats. In contrast, people with myocardial infarction taking rosuvastatin may raise their HDL cholesterol levels more if they have the ‘CC’ genotype [\[R, R\]](#).

Finally, the minor ‘C’ allele of [rs10846744](#) may not affect *SCARB1* expression but has been suggested to affect the expression of genes involved in cell death, inflammation, and endothelial injury repair. This variant has been associated with an increased risk of atherosclerosis and coronary artery disease, as well as with higher total cholesterol, LDL cholesterol, triglycerides, and Lp-PLA2 activity [\[R, R, R, R, R, R, R, R\]](#).



Predisposed to a typical SCARB1 genetics based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SCARB1	rs5888	AG
SCARB1	rs10846744	GC
SCARB1	rs4238001	CC

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

DHCR7 (Cholesterol)

Smith-Lemli-Opitz syndrome (SLOS) is caused by DHCR7 variants that impair 7-dehydrocholesterol reductase function. This leads to both cholesterol deficiency and toxic accumulation of 7DHC, resulting in developmental abnormalities [R].

The main variant linked to this condition is [rs138659167](#)-G. It is rare, and a person needs to carry two risk alleles for the condition to occur. **People with one risk allele are just carriers.** They may not have any signs or symptoms and may have slightly lower cholesterol levels [R, R, R].

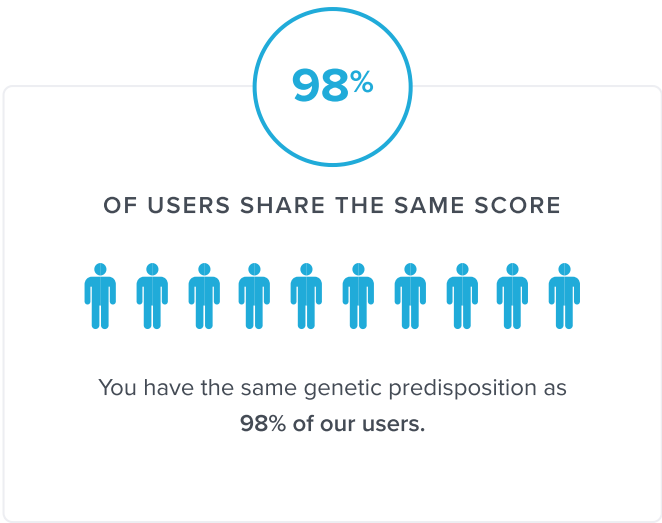
Other variants, [rs11555217](#)-T and [rs80338853](#)-A, are linked to SLOS. However, most people don't have them in their genetic files, so we couldn't include them in the model [R].

Please note: This report is not diagnostic of SLOS. Other genetic variants not analyzed in this report may cause the condition. Commercial genetic testing may not detect very rare mutations with high accuracy.



TYPICAL ACTIVITY

Predisposed to typical DHCR7 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
DHCR7	rs138659167	CC

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

APOA1 (Blood Lipids)

By far, the best-characterized *APOA1* variant is [rs670](#) (75G>A). Its minor ‘T’ allele may increase *APOA1* expression, resulting in higher ApoA-I levels. This variant has been associated with [\[R, R, R, R, R, R, R, R, R\]](#):

- Higher HDL cholesterol
- Lower BMI
- Decreased risk of dyslipidemia
- Decreased risk of diabetes and hyperglycemia
- Decreased risk of metabolic syndrome
- Decreased risk of arterial stiffness

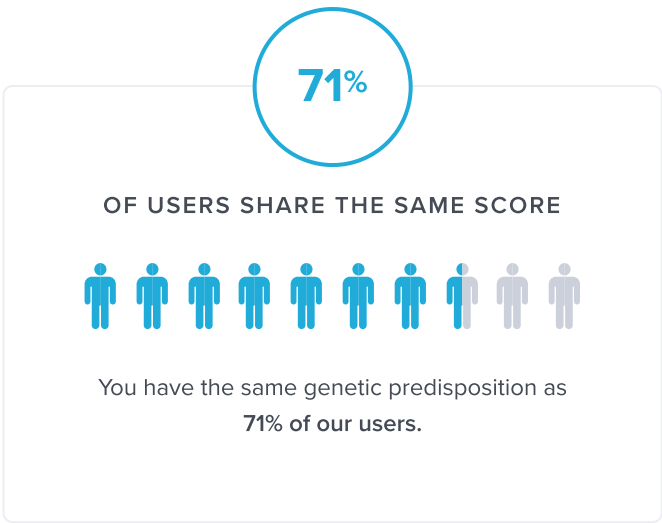
Moreover, dietary interventions with low calorie or high PUFA intake may improve HDL cholesterol, total and LDL cholesterol, fat mass, insulin, and insulin resistance more in people with this variant [\[R, R, R, R, R\]](#).

However, this allele has also been associated with an increased risk of gout [\[R\]](#).



TYPICAL ACTIVITY

Predisposed to a typical APOA1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
APOA1	rs670	CC

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

CETP (Cholesterol/ Longevity)

Certain variants in *CETP* are associated with longevity in some studies. According to researchers, this life-extending effect may be due to improved cholesterol levels, which may help prevent a number of heart conditions [R].

Longevity research has focused on two particular variants. The ‘GG’ genotype in [rs5882](#) (also known as the “I405V” polymorphism) and the ‘AA’ genotype in [rs708272](#) (also called the “TaqIB” polymorphism) have each been associated with lower CETP activity and longer lifespan [R].

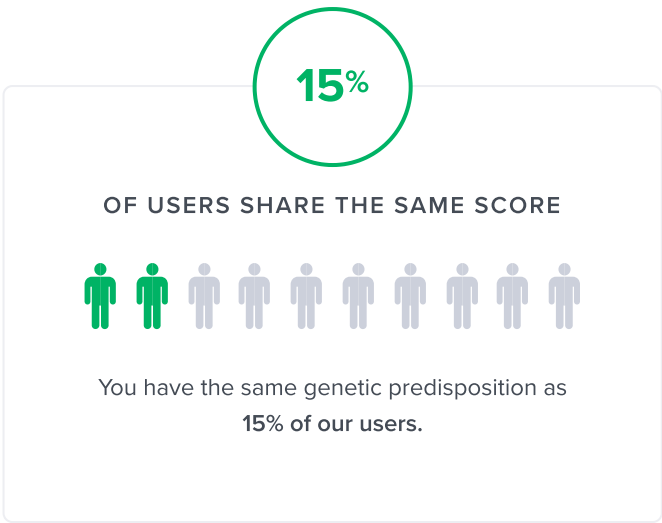
These variants have also been associated with a more favorable blood lipid profile characterized by [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#):

- Higher HDL
- Larger HDL and LDL particle size
- Higher apolipoprotein A-I
- Lower triglycerides



LOWER ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CETP	rs5882	AG
CETP	rs708272	AA

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

LIPC (Cardiovascular)

The most well-researched LIPC polymorphism is [rs1800588](#). Its ‘T’ allele encodes an enzyme with lower activity and has been associated with higher total cholesterol, HDL cholesterol, apolipoprotein AI, and triglyceride levels. The HDL-cholesterol and triglyceride profile may be more unfavorable in ‘T’ carriers who eat a diet high in saturated fats [\[R\]](#), [\[R\]](#), [\[R\]](#).

This variant has also been associated with an increased risk of [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#):

- Type 2 diabetes
- Hypertriglyceridemia
- Coronary artery disease
- Coronary artery calcification
- Vitamin D insufficiency

On the bright side, aerobic exercise may improve HDL-cholesterol more in carriers of this allele. In contrast, people with two copies of the ‘C’ allele may benefit more from a high-carbohydrate diet for decreasing their apoB100/apoAI ratio [\[R\]](#), [\[R\]](#).

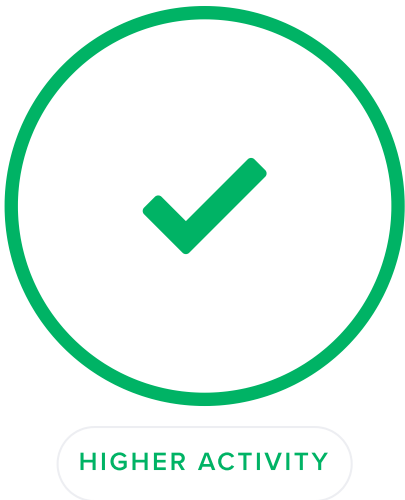
The minor ‘A’ allele of another polymorphism, [rs2070895](#) (250A/G), has been associated with higher LDL cholesterol and lower HDL cholesterol, as well as with an increased risk of [\[R\]](#):

- Coronary artery disease [\[R\]](#)
- Artery hardening [\[R\]](#)
- Diabeic dyslipidemia [\[R\]](#)
- Hypertension, especially in alcohol drinkers [\[R\]](#), [\[R\]](#)
- Peripheral artery disease [\[R\]](#)

However, this variant may be protective for stroke and type 2 diabetes [\[R\]](#), [\[R\]](#).

Interestingly, carriers of this variant may lower their total and LDL cholesterol levels more if they adopt a low-fat diet and raise their HDL cholesterol level more if they exercise [\[R\]](#), [\[R\]](#).

Another well-researched *LIPC* variant is [rs10468017](#). Its minor ‘T’ allele may increase enzyme activity in the liver and has been linked to higher HDL and triglycerides, and lower LDL cholesterol levels. This variant has also been associated with a



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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ADAM10	rs1800588	CC
ADAM10	rs2070895	GG
ALDH1A2	rs10468017	TT
ALDH1A2	rs493258	TT

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

decreased risk of age-related macular degeneration and choroidal neovascularization [R, R, R, R, R, R].

Finally, the ‘T’ allele of rs493258 may also be linked to increased LPC activity in the liver and has been associated with a decreased risk of age-related macular degeneration and higher plasma zeaxanthin levels [R, R, R].

MLXIPL (Triglycerides, Cardiovascular)

The best-researched *MLXIPL* polymorphism is [rs3812316](#) (G771C). The rare ‘G’ allele has been associated with lower triglyceride levels due to the production of an unstable version of the protein. This allele has also been associated with a decreased risk of [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#):

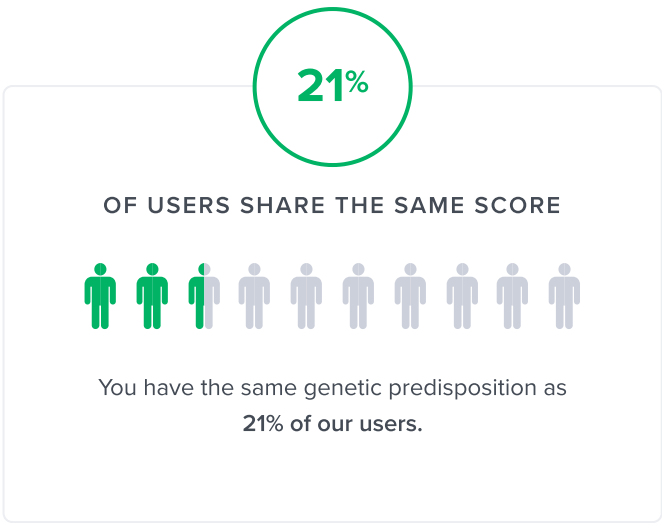
- Coronary artery disease [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Myocardial infarction [\[R\]](#)
- NAFLD [\[R\]](#)
- Obesity [\[R\]](#)
- Metabolic syndrome [\[R\]](#)
- Elevated uric acid levels [\[R\]](#)

Interestingly, the beneficial effects of this variant may be enhanced by the Mediterranean diet [\[R\]](#).



LOWER ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MLXIPL	rs3812316	CG

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

SOAT1 (Cholesterol/ Cognition)

The main *SOAT1* (ACAT-1) variant is [rs1044925](#). Its “C” allele is **generally considered “bad”** due to its link with higher SOAT1 activity, which may imply increased cholesterol buildup.

Studies have linked this variant to [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#):

- Higher cholesterol levels in men
- Higher blood pressure
- Alzheimer’s disease
- Chagas disease (tropical disease that may involve heart problems)

However, some studies didn’t find a link between this variant and blood lipids or Alzheimer’s. One study even found a protective effect on heart health due to higher HDL cholesterol levels [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

Other SOAT1 variants include:

- [rs11545566](#)-G: A study linked it to heart disease and artery hardening [\[R\]](#)
- [rs2247071](#)-C: May be linked to Alzheimer’s disease [\[R\]](#)
- [rs13306731](#)-G: Studies have linked it to heart problems [\[R\]](#)

Lower SOAT1 (ACAT-1) activity means less conversion of free cholesterol to cholesterol esters. This reduces cholesterol storage in cells and may help prevent foam cell formation in arteries. It may also reduce the formation of amyloid plaques in the brain [\[R\]](#), [\[R\]](#).



LOWER ACTIVITY

Likely lower SOAT1 activity based on 4 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
AXDND1	rs11545566	GG
TOR3A	rs2247071	CC
AXDND1	rs1044925	AA
SOAT1	rs13306731	AA

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



Metabolic Syndrome & Diabetes

Metabolic syndrome and diabetes are closely related conditions that can significantly impact overall health. **Genetics plays a key role in the development of these conditions by influencing insulin sensitivity, glucose metabolism, and fat storage.** Understanding how your genes affect these processes can help you manage or even prevent metabolic syndrome and type 2 diabetes.

Genes like *TCF7L2* and *IRS1* are known for their impact on insulin signaling and glucose regulation. *LEPR* and *PPARGC1A* play a role in fat metabolism and energy balance, while *KCNJ11* and *SLC30A8* are linked to insulin secretion and beta-cell function. These genetic variants can increase your susceptibility to insulin resistance, obesity, and type 2 diabetes, which are major contributors to cardiovascular disease. Knowing your genetic risks allows you to implement preventive strategies and lifestyle changes to improve your metabolic health.



LOWER ACTIVITY

WFS1 (Blood Sugar)

Predisposed to a lower WFS1 activity



WORSE GENETICS

TCF7L2 (Blood Sugar/Diet)

Likely worse TCF7L2 genetics



LOWER ACTIVITY

ACSL1 (Blood Sugar)

Predisposed to a lower ACSL1 activity



LOWER ACTIVITY

SLC30A8 (Blood Sugar)

Predisposed to lower SLC30A8 activity



LOWER ACTIVITY

INSIG2 (Weight)

Predisposed to a lower INSIG2 activity



HIGHER ACTIVITY

MTNR1B (Diet & Blood Sugar)

Predisposed to higher MTNR1B activity



HIGHER ACTIVITY

FOXO1 (Blood Sugar)

Likely higher FOXO1 activity



LOWER ACTIVITY

MADD (Blood Sugar & Insulin)

Predisposed to lower MADD activity



LOWER ACTIVITY

ADCY5 (Blood Sugar)

Likely lower ADCY5 activity

LOWER ACTIVITY

CDKN2B (Blood Sugar)

Likely lower CDKN2B activity

LOWER ACTIVITY

PPM1K (Blood Sugar/Diet)

Likely lower PPM1K activity

TYPICAL ACTIVITY

LEPR (Weight/Leptin Resistance)

Likely typical LEPR activity

TYPICAL ACTIVITY

HHEX (Blood Sugar)

Predisposed to a typical HHEX activity

TYPICAL ACTIVITY

KCN (Blood Sugar)

Predisposed to a typical KCN activity

TYPICAL

SLC2A2 (Sugar Intake)

Predisposed to typical sugar intake

TYPICAL ACTIVITY

PPARGC1A (Fitness/Blood Sugar)

Likely typical PPARGC1A activity

TYPICAL ACTIVITY

GIPR (Blood Sugar)

Likely typical GIPR activity

TYPICAL GENETICS

CRY2 (Blood Sugar)

Likely typical CRY2 genetics

TYPICAL ACTIVITY

ADRA2A (Blood Sugar)

Predisposed to typical ADRA2A activity

TYPICAL

SLC2A2 (Sugar Intake)

Predisposed to typical sugar intake

TYPICAL ACTIVITY

CLTCL1 (Blood Sugar/Diet)

Predisposed to typical CLTCL1 activity

TYPICAL ACTIVITY

GCKR (Blood Sugar)

Likely typical GCKR activity

BALANCED PREFERENCES

FGF21 (Carbs vs Fats)

Likely balanced macronutrient preferences

HIGHER ACTIVITY

IRS1 (Metabolic Health)

Predisposed to higher IRS1 activity

HIGHER ACTIVITY

AMY1A (Starch Digestion)

Predisposed to higher AMY1A activity

HIGHER ACTIVITY

SUOX (PCOS/ Blood Sugar)

Likely higher SUOX activity

WFS1 (Blood Sugar)

The main *WFS1* gene variant is [rs10010131](#). Its minor ‘A’ allele has been associated with a decreased risk of type 2 diabetes, metabolic syndrome, gestational diabetes, and diabetic retinopathy [\[R, R, R, R, R, R, R, R\]](#).

Greater GLP-1-induced insulin secretion and insulin sensitivity in ‘A’ carriers may explain its protective effects. Because the WFS1 protein is required for insulin maturation, this variant may increase *WFS1* activity [\[R, R\]](#).

Moreover, a study of 50 adults with obesity and without diabetes found that those with the ‘A’ allele lost more weight after receiving oral dapagliflozin (10 mg/day) and subcutaneous long-acting exenatide (2 mg) for 24 weeks [\[R\]](#).

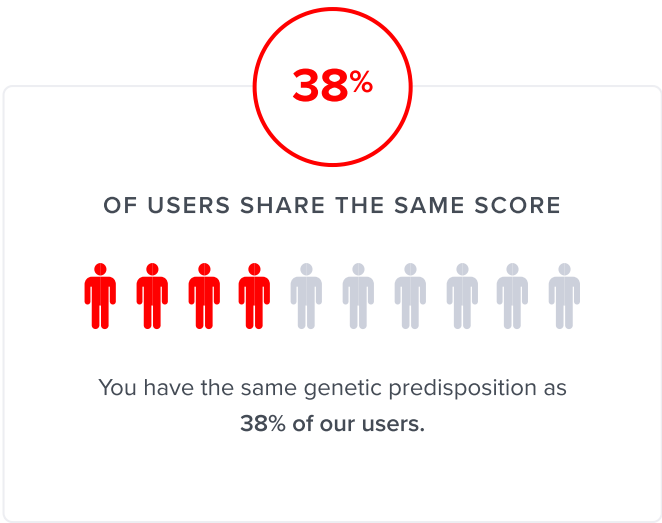
However, this allele has been associated with lower eGFR and a higher urinary albumin-to-creatinine ratio in patients with type 2 diabetes [\[R\]](#).

Another variant associated with a reduced risk of diabetes is the ‘G’ allele of [rs1046320](#). This variant is usually inherited together with the minor allele of rs10010131, meaning you will most likely have both or neither of them [\[R\]](#).



LOWER ACTIVITY

Predisposed to a lower WFS1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
WFS1	rs10010131	GG
WFS1	rs1046320	AA

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

TCF7L2 (Blood Sugar/Diet)

The two main *TCF7L2* variants are [rs7903146](#) and [rs12255372](#). They are almost always inherited together, which means they act as a single genetic factor.

Their "T" alleles have one of the strongest known [links with type 2 diabetes](#). From studies on [TCF7L2, diabetes & carbs](#), we learned that this link is much stronger in people who eat more carbs, especially starchy and refined cabs [\[R, R\]](#).

According to studies on [TCF7L2 & dietary fat](#), people with these variants may want to [\[R, R\]](#):

- Limit the intake of fat, especially saturated fat (fatty meat, butter, palm oil, coconut oil).
- Limit the intake of omega-6 fatty acids, mainly from sweets, pastries, and fried foods.
- Replace the above with fish, olive oil, nuts, and seeds in moderation.

It’s hard to fit all of the above associations into a dietary pattern, but the [interaction between these variants and the Mediterranean diet](#) appears to be beneficial.

In two large studies, the Mediterranean diet reversed the harmful metabolic effects of these variants (higher blood sugar, [LDL](#), and [triglycerides](#)) and promoted weight loss [\[R, R\]](#).

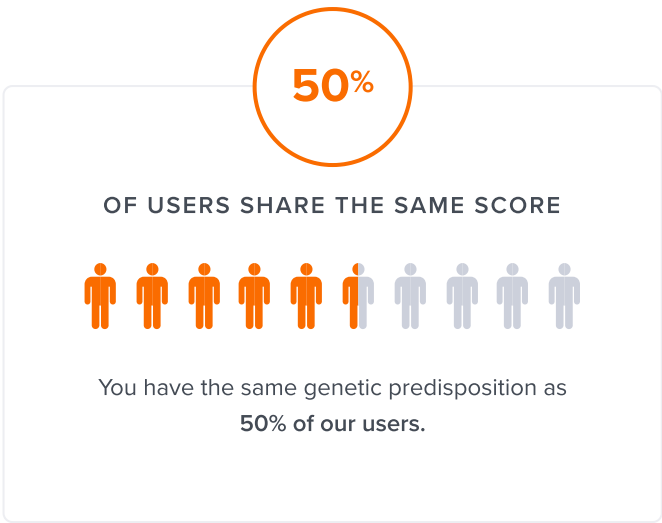
Certain people with these *TCF7L2* variants showed metabolic improvements on a low-fat diet. Still, it may not be the best choice, given the high carb content, which can hinder their blood glucose control. It may even correlate with higher stroke rates in people with these variants, unlike the Mediterranean diet [\[R, R, R\]](#).

According to the research, the quality of dietary fat and other nutrients in the Mediterranean diet may be more important than their quantity [\[R, R, R\]](#).



WORSE GENETICS

Likely worse *TCF7L2* genetics based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TCF7L2	rs7903146	CT
TCF7L2	rs12255372	GT

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

ACSL1 (Blood Sugar)

A study of ~26,000 participants associated the following variants with lower *ACSL1* expression, lower beta-cell function, and increased risk of type 2 diabetes [R]:

- ‘T’ of [rs58730668](#)
- ‘T’ of [rs4862423](#)
- ‘T’ of [rs72695654](#)

Another variant, the ‘G’ allele of [rs9997745](#), has been associated with an increased risk of metabolic syndrome, higher fasting glucose and insulin levels, and lower insulin sensitivity (especially in people eating a diet low in PUFAs). However, a study associated its ‘A’ allele with an increased risk of prediabetes, especially in obese participants [R, R].



Predisposed to a lower ACSL1 activity based on 4 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ACSL1	rs58730668	TT
ACSL1	rs4862423	TT
ACSL1	rs72695654	TT
ACSL1	rs9997745	GG

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

SLC30A8 (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].

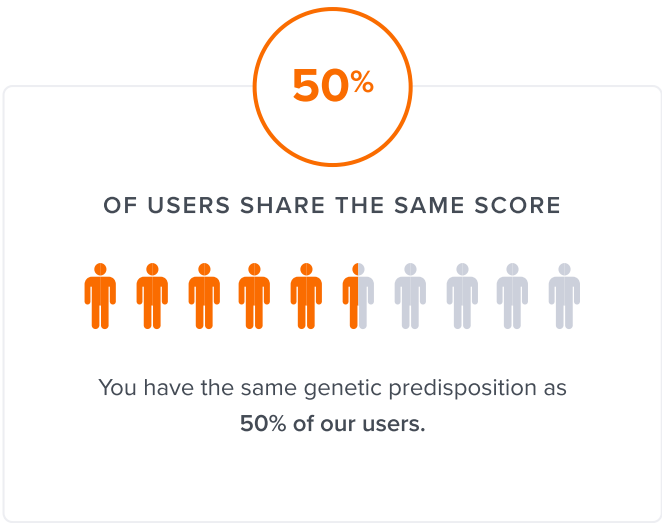
The three variants are usually inherited together, so you will typically carry all risk variants or none of them.

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.

INSIG2 (Weight)

The minor ‘C’ variant of the [rs7566605](#) SNP has been associated with obesity and high BMI in multiple populations [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

Moreover, it was linked to increased fat and weight gain during pregnancy in American women and waist-to-hip ratio in Norwegian men [\[R\]](#), [\[R\]](#), [\[R\]](#).

Similarly, the ‘C’ variant was associated with childhood obesity in multiple studies [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

A meta-analysis investigating these discrepancies found the variant is overall unrelated to obesity, possibly due to different ethnicities between studies. However, it did find an association between the ‘C’ variant and extreme obesity (BMI> 37.5 kg/m²) [\[R\]](#).

The rs7566605 polymorphism is located near the *INSIG2* gene, in a region believed to affect its expression. Although its contribution to *INSIG2* expression and activity hasn’t been investigated, the variant might reduce INSIG2 based on its effects on body weight [\[R\]](#).

In a trial on American and Irish Caucasian men, carriers of the ‘C’ variant lost less body fat from resistance training [\[R\]](#).

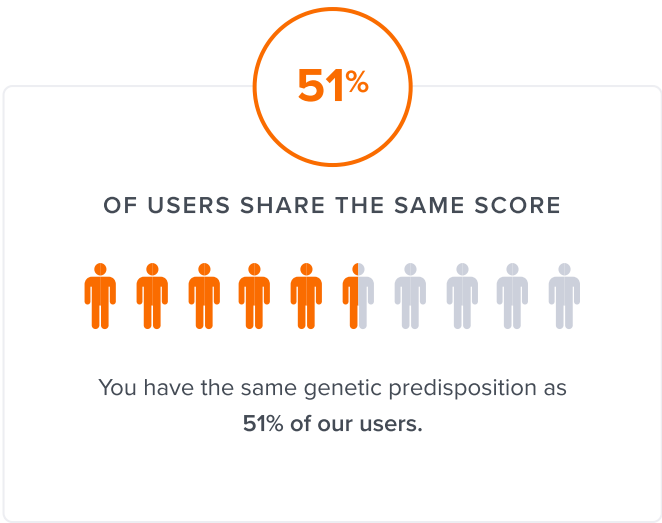
Similarly, obese children and adolescents with the ‘C’ variant lost less weight from an intervention combining exercise and dietary education in two German studies [\[R\]](#), [\[R\]](#).

A Danish study failed to associate this variant with obesity but found that carriers gained more weight during periods of physical inactivity [\[R\]](#).

However, this variant increased the positive effects of a lifestyle intervention in a multi-ethnic American study, and carriers of the ‘G’ allele re-gained more intramuscular fat in the arm one year after stopping physical training [\[R\]](#), [\[R\]](#).



Predisposed to a lower INSIG2 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CCDC93	rs7566605	CG

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

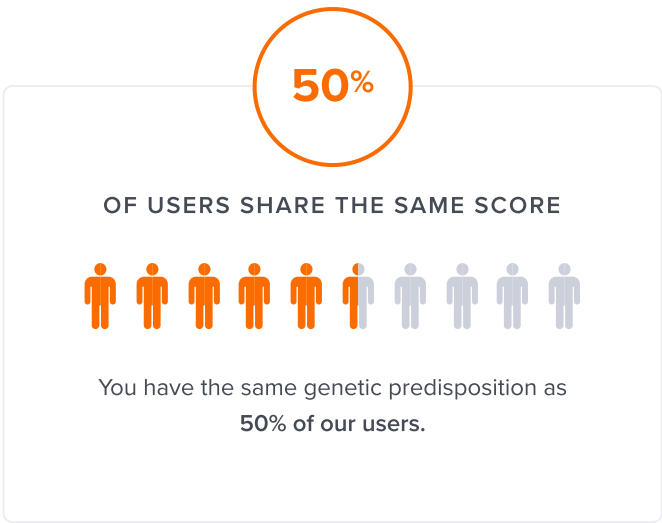
New research has uncovered something unexpected: carriers of this variant produce melatonin for about 41 minutes longer than non-carriers, and their melatonin offset (when melatonin levels drop in the morning) is **delayed by about 80 minutes** [\[R\]](#).

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 [\[R\]](#).



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



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.

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.

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 [\[R\]](#).

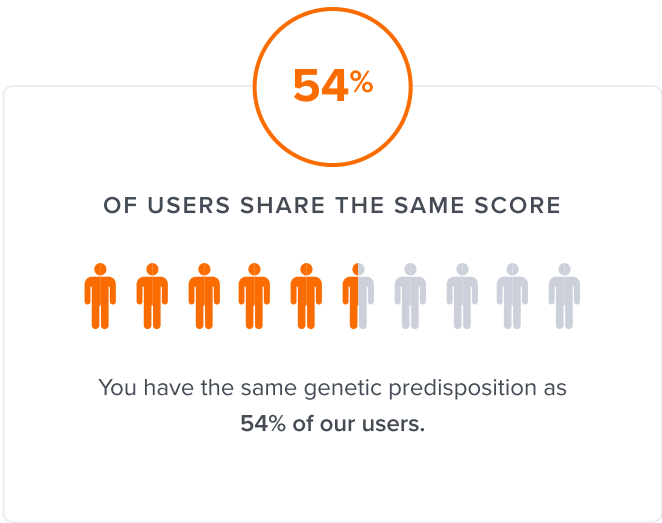
The mechanism behind these contradictory findings isn’t yet clear. The MADD gene has tissue-specific roles. In pancreatic beta cells, this variant may impair insulin production. However, the same variant might have detrimental effects in vascular or neural tissues, such as increasing blood pressure or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



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.

ADCY5 (Blood Sugar)

Two variants of *ADCY5* have been associated with [elevated blood sugar during periods of fasting](#) (i.e., no consumption of food or drinks). Not surprisingly, these variants have also been associated with an increased risk of [type 2 diabetes](#). These variants are [\[R, R\]](#):

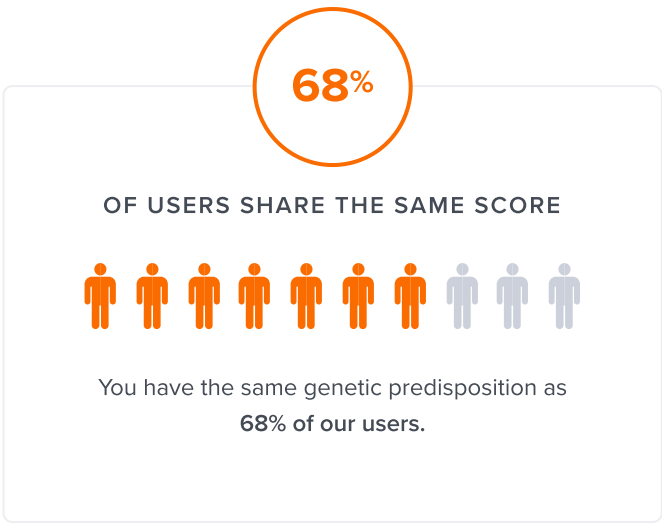
- ‘A’ of [rs11708067](#)
- ‘T’ of [rs11717195](#)

Studies have shown that these variants may lower production of adenylate cyclase 5, leading to decreased insulin release and elevated blood sugar levels while fasting [\[R, R\]](#).

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



Likely lower ADCY5 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ADCY5	rs11708067	AA
ADCY5	rs11717195	TT

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 [\[R\]](#).

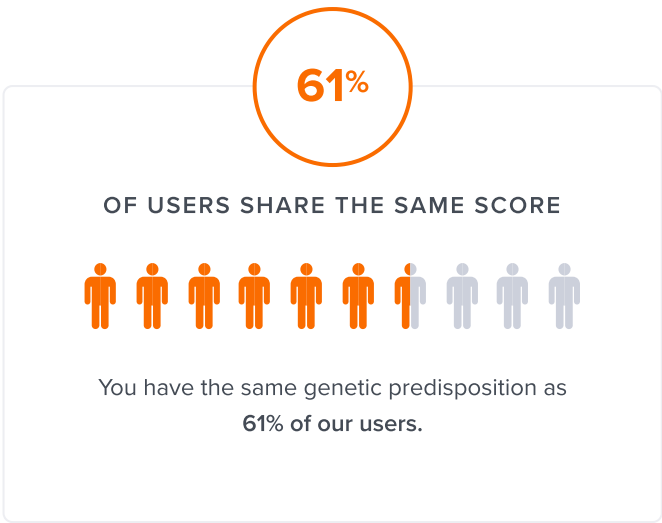
This variant has also been linked to a reduced risk of:

- Glaucoma [\[R\]](#)
- Myocardial infarction [\[R\]](#)

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



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



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 [\[R\]](#).

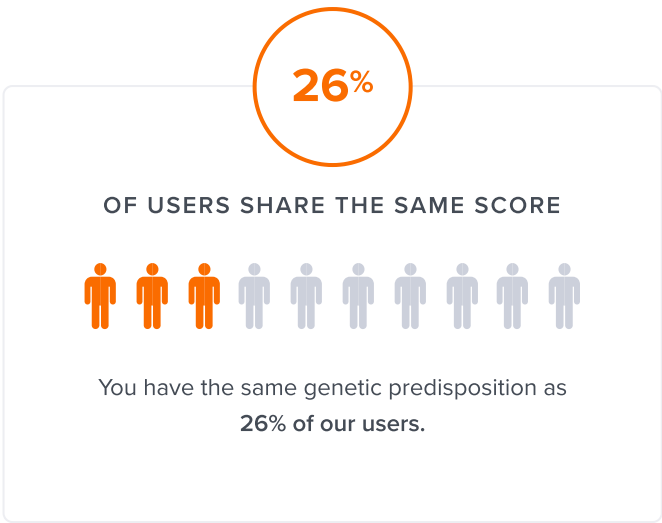
Similarly, those carrying the ‘C’ allele experienced *worse* improvements in weight and insulin resistance while on a calorie-restricted high-fat diet in the NUGENOB trial [\[R\]](#).

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



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.

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

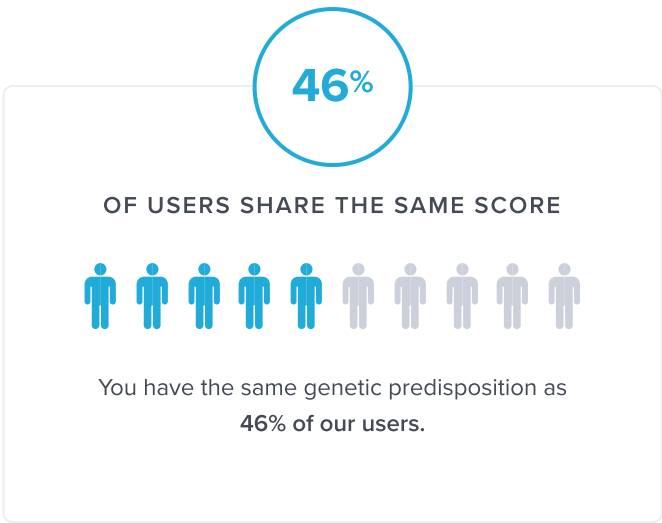
Studies have linked another variant, [rs12405556-T](#), to higher weight and blood lipid levels. This variant is often inherited together with rs1137101-G, meaning many people carry either both or none of them [\[R\]](#).

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



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.

HHEX (Blood Sugar)

The best-characterized *HHEX* polymorphism is [rs1111875](#). Its ‘C’ allele has been associated with an increased risk of:

- Type 2 diabetes [[R](#), [R](#), [R](#), [R](#)]
- Gestational diabetes [[R](#)]
- Lower birth weight [[R](#)]
- Colorectal cancer [[R](#)]

On the bright side, this variant has been associated with a decreased risk of breast cancer [[R](#)].

Another variant associated with an increased risk of type 2 diabetes is the ‘G’ allele of [rs7923837](#). This variant has also been associated with an increased risk of [[R](#), [R](#)]:

- Gestational diabetes [[R](#)]
- Diabetes in kidney transplant patients [[R](#)]
- Cancer in patients with diabetes [[R](#)]
- Multiple sclerosis [[R](#), [R](#)]
- High triglycerides [[R](#)]
- Colorectal cancer [[R](#)]

Finally, the ‘C’ allele of [rs5015480](#) has been associated with an increased risk of type 2 diabetes and gestational diabetes. The association with diabetes may be stronger in heavy-smoking men [[R](#), [R](#), [R](#), [R](#)].

These variants have been shown to reduce beta-cell function, resulting in decreased insulin sensitivity. Because the *HHEX* gene is involved in the development of pancreatic beta-cells, these variants may reduce HHEX levels or function [[R](#), [R](#)].



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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HHEX	rs1111875	CT
HHEX	rs7923837	GA
HHEX	rs5015480	CT

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

KCN (Blood Sugar)

The following variants of *KCNJ11* and *KCNQ1* have been associated with low blood sugar levels [R, R, R]:

- ‘C’ of [rs5215](#)
- ‘T’ of [rs5219](#)
- ‘C’ of [rs2237892](#)
- ‘C’ of [rs2237897](#)

These variants may decrease the production or activity of potassium channels, leading to overactivity of pancreatic cells. This can result in excess release of insulin and low blood sugar levels. In the long run, high insulin levels can result in decreased insulin receptor sensitivity. This may increase the risk of [type 2 diabetes](#) [R].



TYPICAL ACTIVITY

Predisposed to a typical KCN activity based on 4 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CDKN1C	rs2237897	CC
CDKN1C	rs2237892	CC
KCNJ11	rs5219	CC
KCNJ11	rs5215	TT

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 [\[R\]](#).

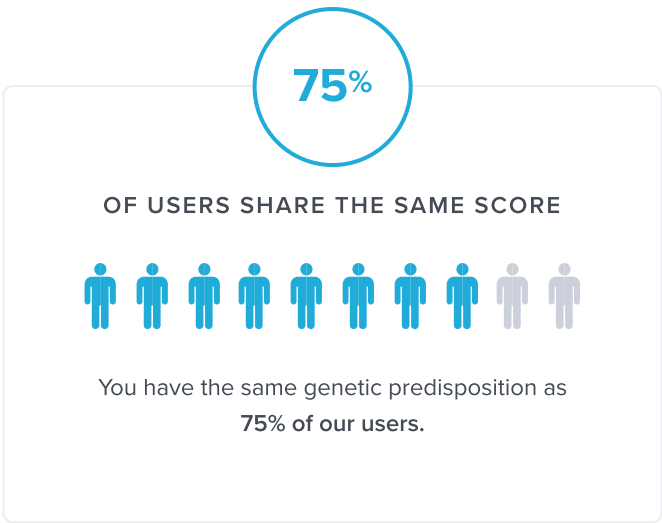
On the other hand, the variant might increase GLUT2 activity in the liver and pancreas, leading to enhanced glucose uptake and metabolism. This could explain lower blood sugar but higher blood lipids, liver enzymes, and body weight.

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



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.

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

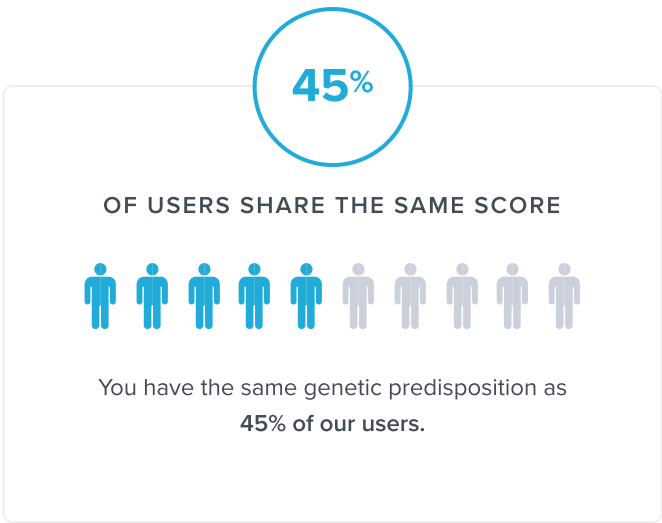
It may also be linked to **worse cold adaptation** due to reduced PGC-1α levels and impaired mitochondrial function.

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PPARGC1A	rs8192678	TC

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

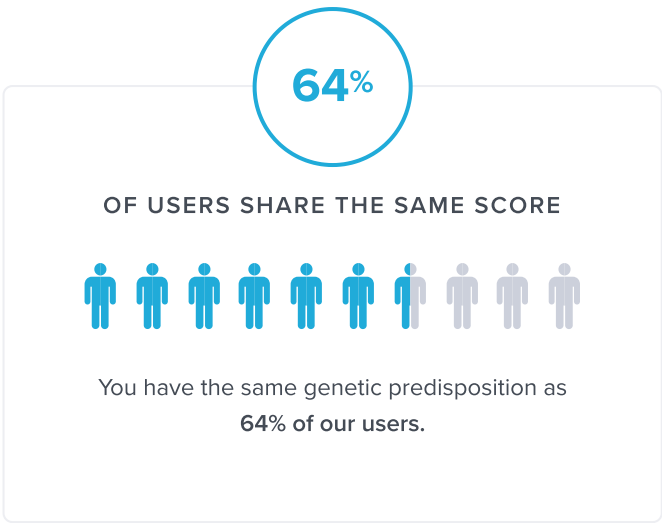
These variants may **reduce insulin secretion** after a meal. This may impair blood sugar control and contribute to diabetes but also promote fat burning [\[R\]](#), [\[R\]](#).

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



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.

CRY2 (Blood Sugar)

The main CRY2 variant is [rs11605924](#). Its “**A**” **allele** is associated with **higher blood sugar and diabetes** [\[R\]](#), [\[R\]](#), [\[R\]](#).

On the bright side, this variant may be linked to:

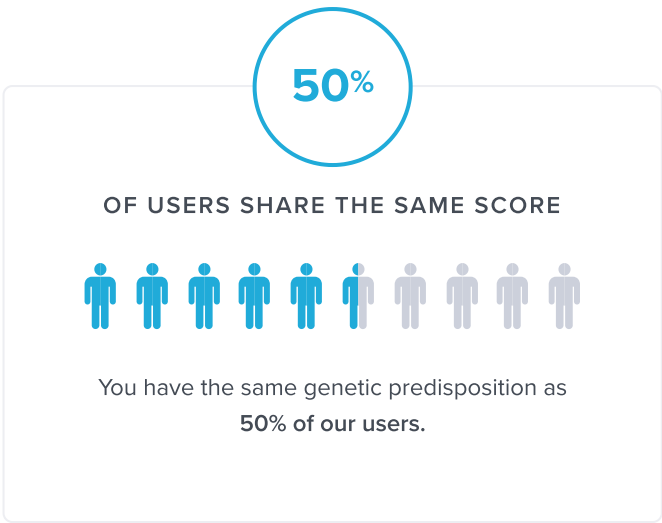
- Enhanced fat burning on a high-fat diet [\[R\]](#)
- Higher HDL or “good” cholesterol levels [\[R\]](#), [\[R\]](#)

Circadian rhythm is well-established in diet, metabolism, and blood sugar control. However, researchers are unsure how this variant affects sugar and fat metabolism [\[R\]](#).



TYPICAL GENETICS

Likely typical CRY2 genetics based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CRY2	rs11605924	AC

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

ADRA2A (Blood Sugar)

The best-characterized *ADRA2A* variant is [rs553668](#). Its minor ‘A’ allele may increase the number of ADRA2A receptors. This allele has been associated with higher fasting glucose and increased risk of type 2 diabetes (especially in Europeans) in multiple studies. Myocardial infarction patients had a 13% higher glucose in one of them [[R](#), [R](#), [R](#), [R](#), [R](#), [R](#), [R](#), [R](#)].

The major ‘G’ allele of [rs10885122](#) has also been associated with higher fasting glucose and worse beta-cell function in both healthy subjects and people recovering from a heart attack. However, the results are mixed and a meta-analysis failed to confirm this effect of the ‘G’ allele. Moreover, the variant associated with gestational diabetes was the minor ‘T’ allele [[R](#), [R](#), [R](#), [R](#), [R](#), [R](#), [R](#), [R](#)].



TYPICAL ACTIVITY

Predisposed to typical ADRA2A activity based on 2 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ADRA2A	rs10885122	GG
PDCD4	rs553668	GG

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 [\[R\]](#).

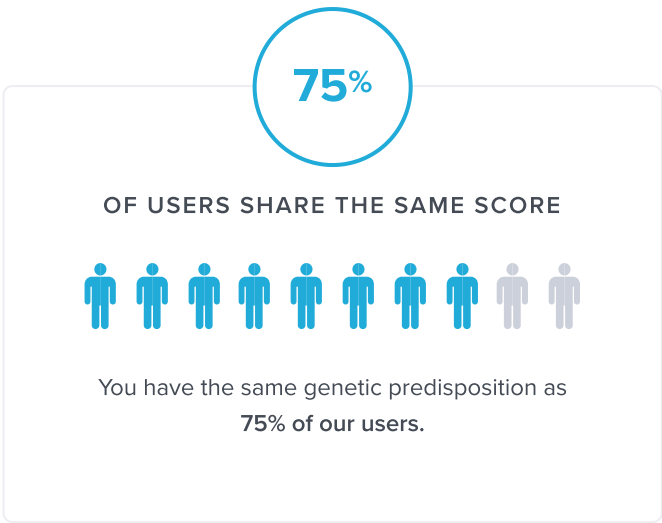
On the other hand, the variant might increase GLUT2 activity in the liver and pancreas, leading to enhanced glucose uptake and metabolism. This could explain lower blood sugar but higher blood lipids, liver enzymes, and body weight.

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



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.

CLTCL1 (Blood Sugar/Diet)

A *CLTCL1* variant, the ‘C’ allele of [rs1061325](#), first became prevalent in the genomes of ancient Neolithic farmers and is more frequent in farming populations than in hunter-gatherers [\[R\]](#).

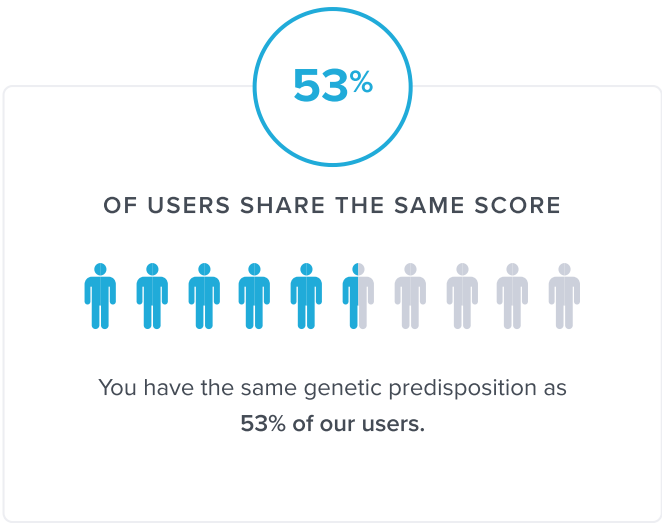
This variant encodes a version of the CHC22 protein that is less effective at retaining the GLUT4 transporter inside cells. Because this causes more GLUT4 to be found on the cell surface, carriers of this variant lower their blood sugar more in response to insulin and are better adapted to eating high-carbohydrate diets [\[R\]](#).

Note that the evidence linking this variant to carbohydrate tolerance is scarce and low-quality. Other variants such as [rs7903146](#) at [TCF7L2](#) may have a much stronger impact on type 2 diabetes risk and tolerance to dietary carbohydrates [\[R\]](#), [\[R\]](#).



TYPICAL ACTIVITY

Predisposed to typical CLTCL1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CLTCL1	rs1061325	CT

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

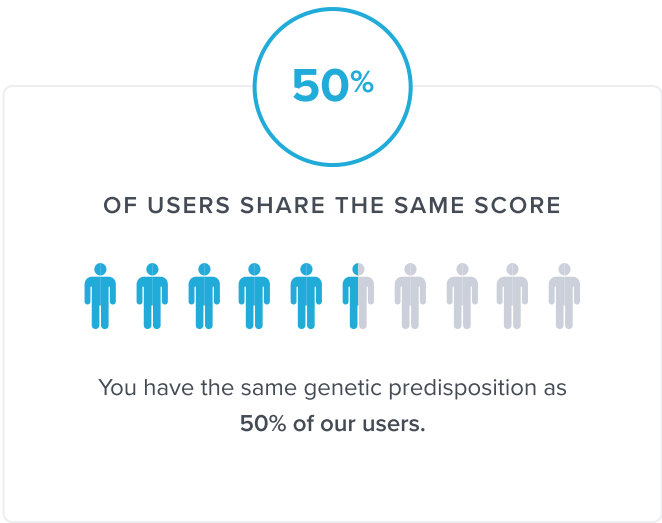
On the positive side, these variants are linked to lower levels of cholesterol and other blood lipids [\[R\]](#).

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



Your top variants that most likely impact your genetic predisposition:

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

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 [\[R\]](#).

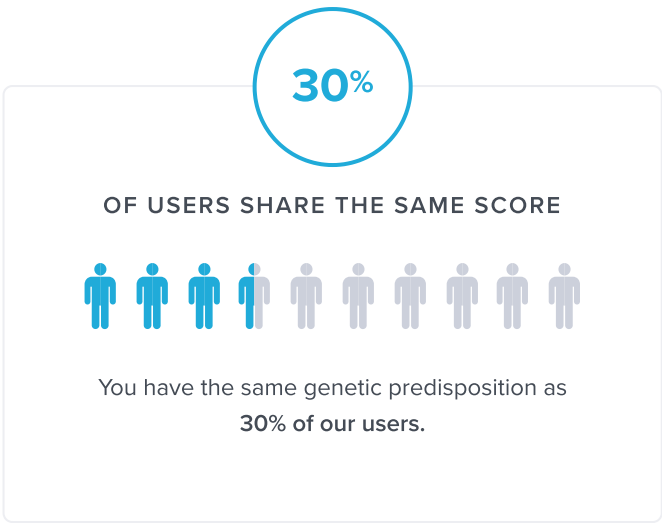
Another trial of 38,000 participants confirmed the link between rs838133 and lower fat/higher carb preference [\[R\]](#).

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
FGF21	rs838133	AG
FGF21	rs838145	GA

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

IRS1 (Metabolic Health)

One of the most studied IRS1 variants is [rs2943641](#). People with the “**T**” **allele** may have [\[R\]](#):

- Lower odds of heart disease and diabetes
- Lower levels of fasting glucose, insulin, and triglycerides
- Higher levels of HDL or “good” cholesterol

Interestingly, a study linked this variant to improved insulin resistance and weight loss on a high-carb, low-fat diet. However, another study reached the opposite conclusion [\[R, R\]](#).

Another IRS1 variant, [rs7578326-G](#), showed similar associations as rs2943641-T. These two variants are sometimes inherited together, so some people will carry either none or both of them [\[R, R\]](#).

One rare variant, [rs1801278](#) (G972R), changes the structure of the IRS1 protein. Its “**T**” **allele** is linked to:

- Type 2 diabetes [\[R\]](#)
- Gestational diabetes (diabetes in pregnancy) [\[R\]](#)
- Higher cholesterol [\[R\]](#)

According to limited evidence, this variant also seems to favor high-carb diets [\[R\]](#).

Variants linked to better metabolic outcomes may boost IRS1 activity, improving insulin receptor activation. This stimulates the downstream signaling pathways essential for glucose uptake and lipid metabolism, directly impacting blood sugar and fat storage [\[R\]](#).



HIGHER ACTIVITY

Predisposed to higher IRS1 activity based on 3 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
IRS1	rs2943641	TC
IRS1	rs7578326	GA
IRS1	rs1801278	CC

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

AMY1A (Starch Digestion)

Individuals carrying more [AMY1A](#) gene copies tend to have increased amylase enzyme activity, and an improved ability to **break down starch and complex carbs** into simple sugars [\[R\]](#).

Some studies linked lower numbers of *AMY1A* copies to obesity and diabetes, while other studies found opposite results [\[R, R, R, R, R\]](#).

Variants (SNPs) of the *AMY1A* gene are thought to be associated with an individual's *AMY1A* gene copy number [\[R\]](#).

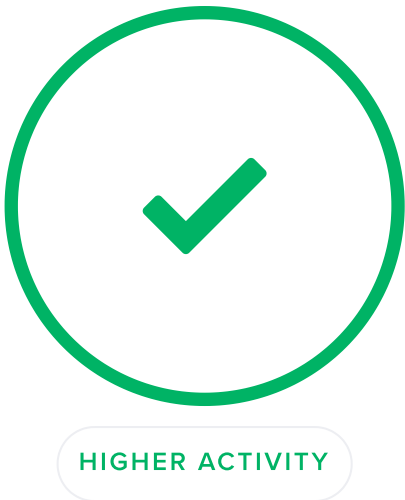
There are several *AMY1A* gene variant alleles that have been associated with significantly lower gene copy numbers. Each variant carried is thought to cause a difference of up to 2 gene copies [\[R, R\]](#).

One such variant is [rs11185098](#). When put on a diet, people with the **higher-amylase ‘A’ variant** tended to **lose more weight**. The study included over 800 obese participants monitored for 2 years. However, other studies have had mixed results [\[R, R\]](#).

The link between AMY1A activity and obesity may depend on diet type. Some studies found that **lower-amylase variants may offer protection against obesity on a high-carb diet**, likely because a portion of those carbs goes undigested. A study observed this effect **only in women** [\[R\]](#).

Simply put, if you have higher AMY1A activity, you may metabolize starches more efficiently. This is good for your metabolic health, but if you eat a lot of starches, it may contribute to weight gain.

Note that some of these variants also affect the activity of the [AMY2A](#) gene, which helps make **pancreatic** amylase.



Predisposed to higher AMY1A activity based on 9 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
AMY2B	rs4244372	AT
AMY1C	rs1566154	AA
AMY2B	rs11185098	GA
AMY2A	rs1330403	GA
AMY2A	rs11577390	CT
AMY1B	rs6696797	AA
AMY1B	rs2132957	AA
AMY2B	rs1999478	CC
AMY1C	rs1930212	AA

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

SUOX (PCOS/ Blood Sugar)

The most well-researched *SUOX polymorphism* is [rs705702](#). Its minor ‘G’ allele has been associated with an increased risk of PCOS in several studies. In one of them, the variant was also linked to higher insulin and glucose levels after an oral glucose test. Increased oxidative stress due to reduced SUOX activity and homocysteine buildup may be responsible for these negative effects [\[R, R, R, R, R\]](#).

Another variant, ‘G’ at [rs10876864](#), has been associated with vitiligo and allergic diseases, and showed trends for an association with type 1 diabetes [\[R, R, R\]](#).

Finally, the minor ‘G’ allele of [rs1131017](#) has also been associated with an increased risk of type 1 diabetes, worse disease progression, and higher insulin auto-antibody levels. Another study associated this variant with alopecia areata [\[R, R, R, R\]](#).



HIGHER ACTIVITY

Likely higher SUOX activity based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
IKZF4	rs10876864	GA
SUOX	rs1131017	CG
RPS26	rs705702	AA

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



Fat Metabolism & Obesity

Fat metabolism and obesity are critical factors that influence health and overall well-being. **Genetics plays a significant role in how your body processes fat, stores energy, and regulates appetite.** Variants in certain genes can increase the risk of obesity by affecting fat metabolism, energy expenditure, and appetite regulation.

Genes like *FTO*, *MC4R*, and *LEPR* are closely linked to body weight regulation and appetite control. *PPARG* and *ADIPOQ* play roles in fat storage and insulin sensitivity, while *FADS1* and *FADS2* influence fatty acid metabolism, impacting lipid profiles and inflammation. Understanding your genetic predisposition to obesity and fat metabolism can help you adopt personalized lifestyle changes to manage weight and reduce cardiovascular risk.

 WORSE GENETICS ADRB2 (Weight) Likely worse ADRB2 genetics	 LOWER ACTIVITY PPARD (Exercise Response) Predisposed to lower PPARD activity	 LOWER ACTIVITY UCP1 (Weight) Likely lower UCP1 activity
 TYPICAL ACTIVITY PPARG (Metabolism) Likely typical PPARG activity	 TYPICAL GENETICS ADIPOQ (Weight/ Blood Sugar) Likely typical ADIPOQ genetics	 TYPICAL ACTIVITY ADRB3 (Weight) Likely typical ADRB3 activity
 TYPICAL ACTIVITY UCP2 (Weight) Likely typical UCP2 activity	 TYPICAL ACTIVITY UCP3 (Weight) Likely typical UCP3 activity	 TYPICAL ACTIVITY TFAP2B (Weight/Diet) Likely typical TFAP2B activity
 TYPICAL ACTIVITY MC3R (Weight) Likely typical MC3R activity	 TYPICAL ACTIVITY CD36 (Fat Preference/Weight) Likely typical CD36 activity	 BETTER FTO (Weight) Likely better FTO genetics



HIGHER ACTIVITY

FADS1/2 (Fatty Acid Metabolism)

Likely higher FADS1/2 activity



HIGHER ACTIVITY

MC4R (Weight/ Blood Sugar)

Likely higher MC4R activity



LOWER ACTIVITY

PLIN (Weight, Blood Sugar)

Likely lower PLIN activity



BETTER GENETICS

TMEM18 (Weight)

Likely better TMEM18 genetics

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	GC

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

PPARD (Exercise Response)

The best-characterized *PPARD* polymorphism when it comes to exercise response is [rs2016520](#) polymorphism (also known as *PPARD* +294T/C). Its minor ‘C’ allele may increase *PPARD* expression and has been associated with [\[R\]](#):

- Higher odds of reaching elite endurance athletic status [\[R, R\]](#)
- Greater HDL cholesterol and Apo-A1 increase from aerobic training [\[R\]](#)
- Greater total cholesterol and triglyceride decrease from aerobic training [\[R\]](#)
- Greater weight loss from an intervention including diet and exercise [\[R\]](#)
- Better dynamic balance performance [\[R\]](#)

However, those with the ‘CC’ genotype may increase their aerobic capacity (VO2 max) and maximal power output less from aerobic training [\[R\]](#).

Another well-researched polymorphism is [rs2267668](#). Its minor ‘G’ allele, presumably reducing *PPARD* activity, has been associated with:

- Higher odds of being overweight in physically active men [\[R\]](#)
- Lower muscle mass gain and fat mass loss from an intervention including exercise [\[R\]](#)
- Decreased aerobic fitness and insulin sensitivity increase from aerobic training [\[R\]](#)

However, this variant has been associated with greater improvements in metabolic parameters such as total cholesterol, triglycerides, HbA1c, ALT, and GGT from lifestyle interventions including exercise [\[R, R\]](#).

This variant has also been associated with better dynamic balance performance [\[R\]](#).

Finally, the major ‘T’ allele of [rs1053049](#) has been associated with:

- Higher odds of reaching elite athletic endurance status [\[R\]](#)
- Greater improvement in insulin sensitivity and fasting glucose from aerobic training [\[R\]](#)
- Lower odds of being overweight in physically active men [\[R\]](#)



Predisposed to lower PPARD activity based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
DEF6	rs1053049	CC
DEF6	rs2016520	CT
DEF6	rs2267668	GA

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

- Higher muscle mass gain and fat mass loss from an intervention including exercise [\[R\]](#), [\[R\]](#).

Based on these phenotypes, this variant may show higher *PPARD* activity than the minor ‘C’ allele.

However, those with the ‘TT’ genotype may have higher triglyceride levels after an aerobic training intervention [\[R\]](#).

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 [\[R\]](#).

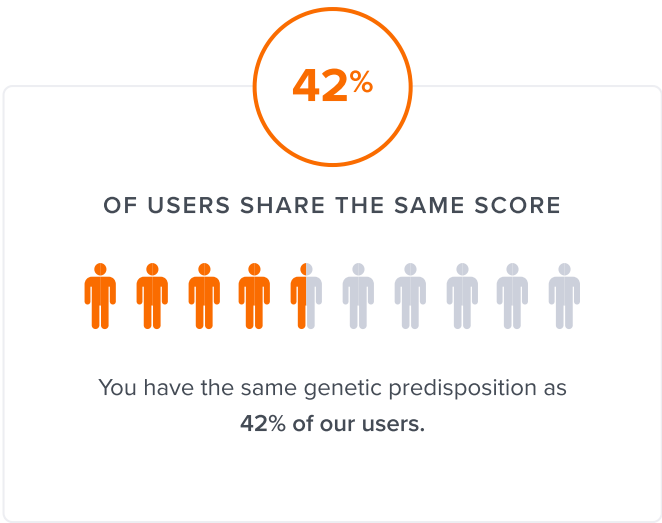
Several studies link the 'C' allele and the 'CC' genotype to metabolic disturbances commonly associated with being overweight. In various studies, the ‘C’ allele has been associated with elevated blood pressure, greater [insulin resistance](#), and higher [LDL cholesterol](#) and [triglycerides](#) [\[R,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

Fun fact: worldwide, about 30% of people have the 'TT' genotype, which is associated with higher resting metabolism



LOWER ACTIVITY

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



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.

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 [\[R\]](#).

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

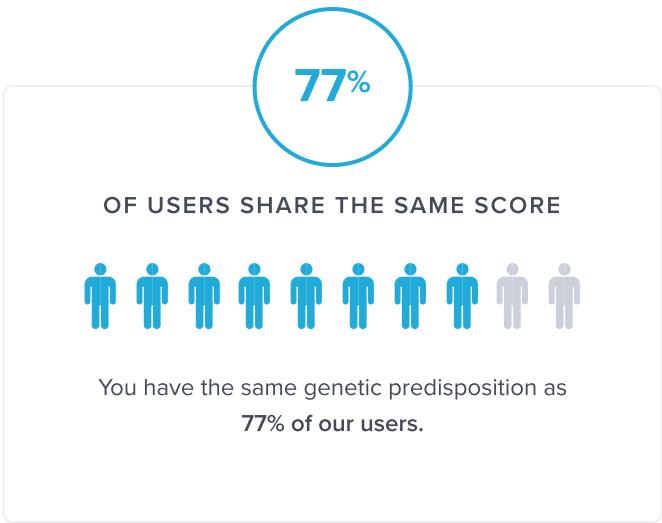
PPAR-γ can be a double-edged sword when it comes to regulating [blood sugar levels](#). While its activity in the liver and pancreas enhances glucose metabolism and insulin sensitivity, excess PPAR-γ activity in fat cells can accumulate fatty acids and other factors that contribute to [insulin resistance](#)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



TYPICAL ACTIVITY

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



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.

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](#)].

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](#)].

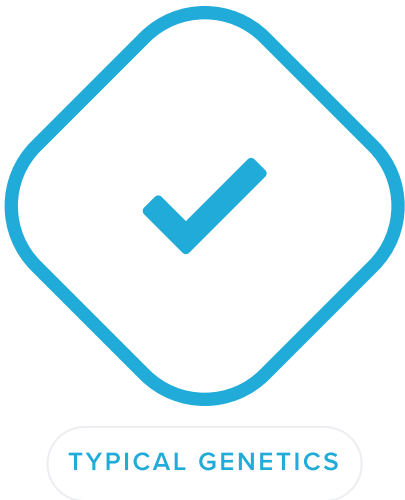
The minor ‘**G**’ variant of the [rs2241766](#) polymorphism is linked to:

- 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](#)].



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.

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:

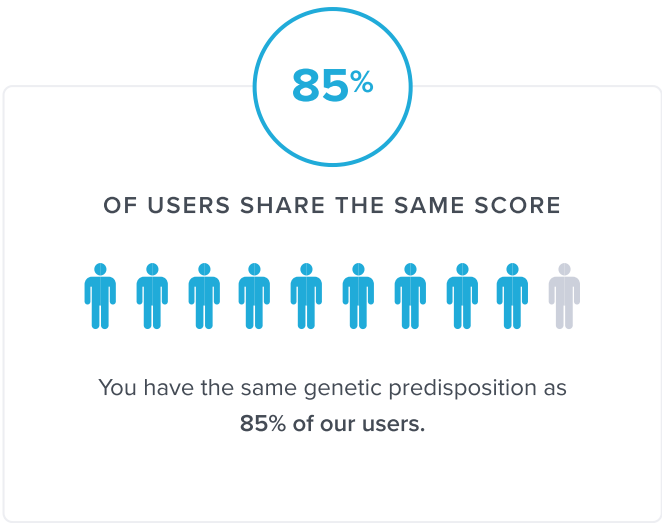
- Diabetes [\[R\]](#), [\[R\]](#)
- High blood pressure [\[R\]](#)
- Heart disease [\[R\]](#)

Those genetic effects were also more pronounced in East Asians.



TYPICAL ACTIVITY

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



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.

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

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

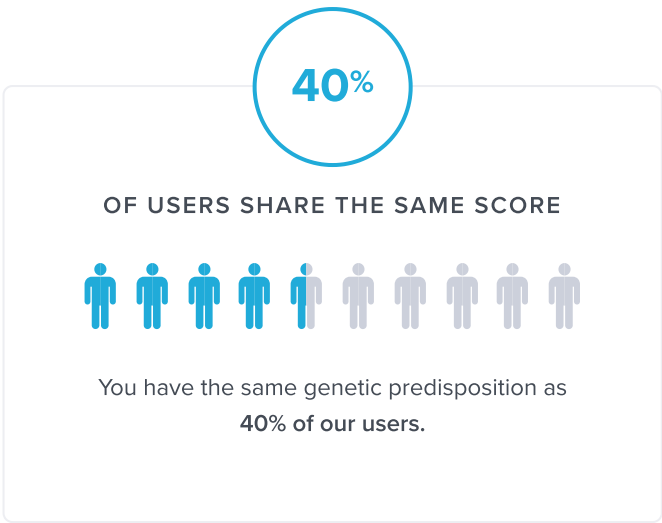
However, this variant may have negative effects on blood lipids, glucose control, and diabetic outcomes [\[R, R, R, R, R, R, R, R, R\]](#).

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



TYPICAL ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
UCP2	rs659366	CT

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	AG
UCP2	rs2075577	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 [\[R\]](#).

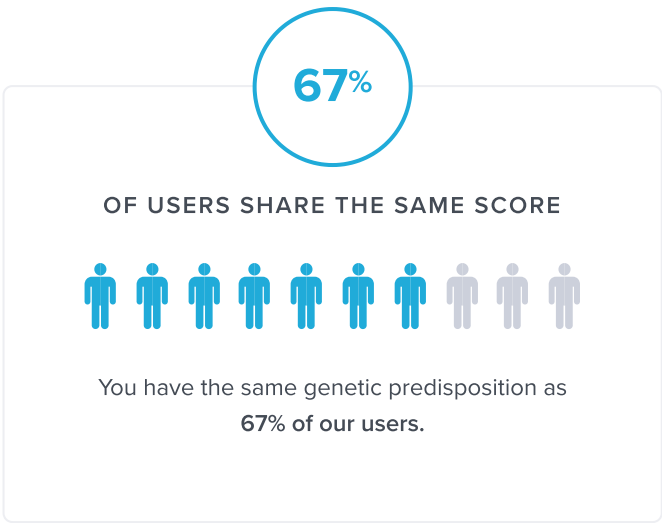
One trial analyzed weight maintenance in 468 European people who completed a weight loss program. Those following a high-protein diet regained 1.84 kg more per each “G” allele. On the other hand, the “AA” carriers did better on a high vs. low-protein diet [\[R\]](#).

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



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.

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

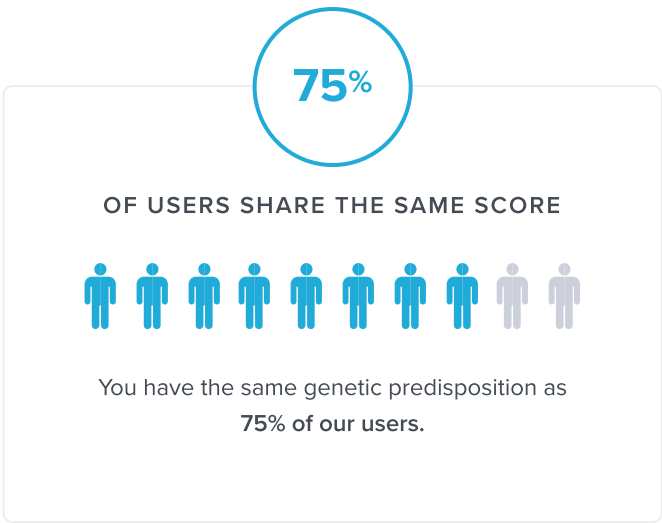
Clinical studies have confirmed the link between the “A” alleles, **excess leptin, and suppressed fat oxidation** [\[R, R, R, R\]](#).

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



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.

CD36 (Fat Preference/Weight)

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

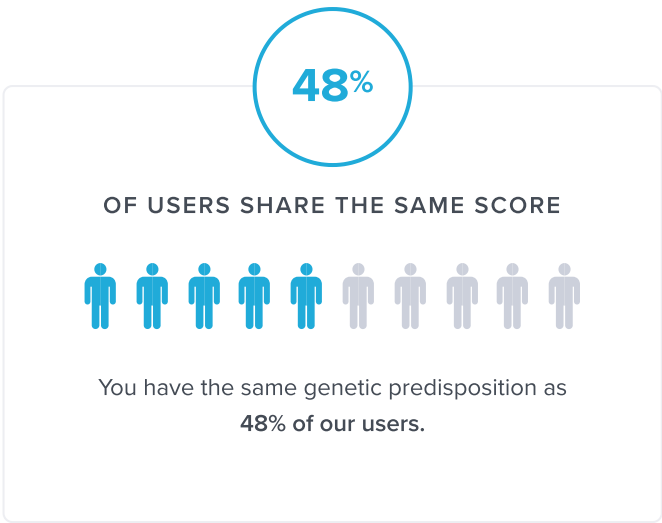
The ‘G’ allele at [rs1761667](#) is associated with a number of metabolic changes compared to the ‘AA’ genotype. For example, people with at least one copy have increased appetite and food intake. However, the ‘G’ allele is also associated with lower BMI [\[R\]](#), [\[R\]](#), [\[R\]](#).

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CD36	rs1761667	AG

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

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

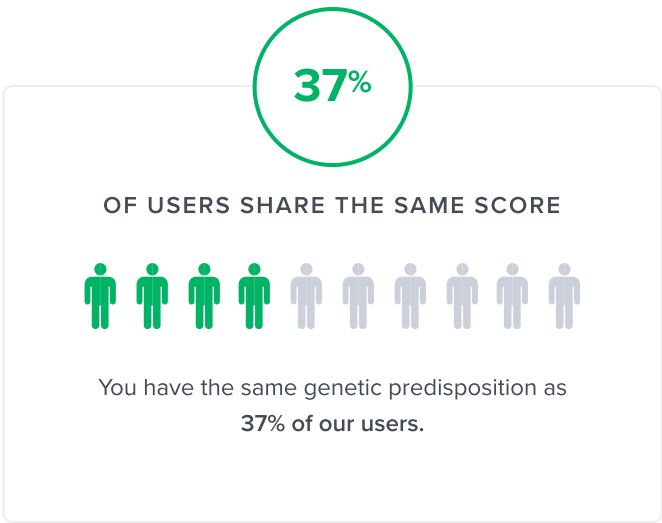
The good news is that **healthy dietary choices may reduce and even cancel out the harmful effects of the ‘A’ variant!**

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



BETTER

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



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.

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

FADS1/2 (Fatty Acid Metabolism)

A number of studies suggest that specific variants in *FADS1/2* may cause [fat metabolism issues](#).

Using data from 426 individuals from Spain, one genome-wide study found that the ‘T’ allele of [rs174546](#) is associated with lower levels of PUFAs, such as the omega-6 fatty acid [arachidonic acid](#) (AA) and the omega-3s EPA and DHA. This may be because this variant impairs the conversion of ALA from plant sources into EPA and DHA [\[R\]](#).

Another study of 224 people from an isolated island population in the U.S. found that the ‘T’ allele is also associated with reduced levels of omega-6 fatty acids [\[R\]](#).

This allele has also been associated with higher triglyceride levels, but lower [LDL](#) and [total cholesterol](#), based on research in European, Canadian, and Mexican populations [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

Similar results have been found for other SNPs, such as [rs174547](#), [rs174548](#), [rs174550](#), and [rs1535](#). In all these cases, the minor allele was associated with lower D5D activity and decreased levels of omega-3 and omega-6 fatty acids. These alleles are usually inherited together, so you will typically carry all the minor variants or none of them [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

A different study looked at the SNP **rs174547** in over 21,000 Japanese and over 1,200 Mongolian individuals. Researchers also found that the ‘C’ allele was associated with higher triglycerides and lower [HDL cholesterol](#) in Japanese individuals. In the Mongolian population, the ‘C’ allele was only associated with lower LDL cholesterol but was unrelated to HDL or triglyceride levels [\[R\]](#).

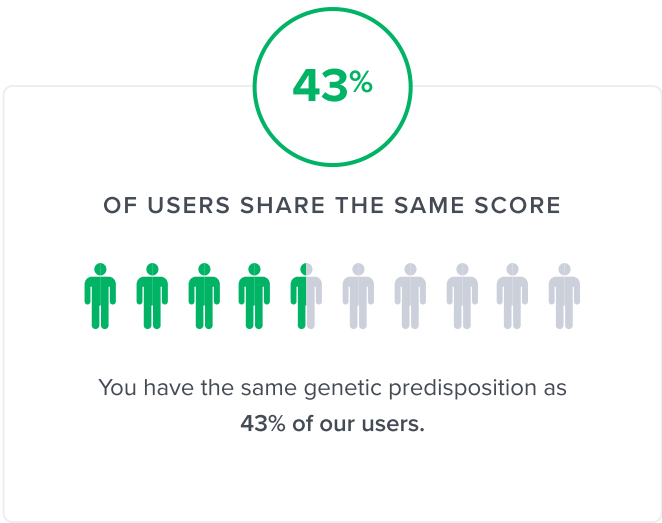
Another study of **rs174547** suggests that the ‘C’ allele is only associated with lower LDL in people who have a low intake of omega-3 fatty acids [\[R\]](#).

In addition, the ‘C’ allele of **rs174550** may also be associated with low HDL and higher triglyceride levels, according to research in Chinese populations [\[R\]](#).



HIGHER ACTIVITY

Likely higher FADS1/2 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
FADS2	rs174546	CC
FADS2	rs174550	TT
FADS2	rs174548	CC
FADS2	rs174547	TT

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

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:

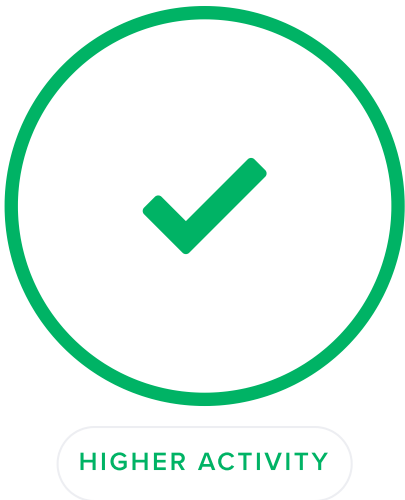
- Obesity, higher BMI, and waist circumference [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Food cravings and increased beverage consumption [\[R\]](#)
- High blood sugar and insulin resistance [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)

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

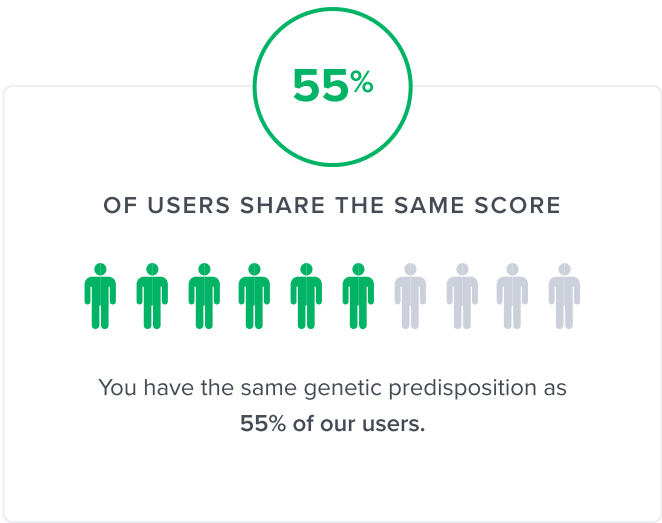
“Bad” *MC4R* variants likely **reduce gene expression or receptor activity**, thus increasing food intake and hindering glucose and fat metabolism.

[Learn more about the link between MC4R variants, weight, and food intake.](#)

[Learn more about the link between MC4R variants and blood sugar.](#)



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



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.

PLIN (Weight, Blood Sugar)

The most well-characterized *PLIN1* polymorphism is [rs894160](#) (11482 G>A). Its minor ‘T’ allele has been associated with enhanced lower perilipin levels, enhanced fat breakdown, and decreased BMI and obesity risk in white women [\[R, R, R, R\]](#).

However, women with this variant may reduce their waist circumference less in response to energy restriction. This may be related to the composition of the diet, since two studies found that the ‘A’ allele is associated with smaller waist and hip circumferences when complex carbohydrate intake is higher than 144 g/day but with larger waist circumference when it’s lower [\[R, R, R\]](#).

In contrast, this allele has been associated with an increased risk of type 2 diabetes, metabolic syndrome, and PCOS [\[R, R, R, R\]](#).

Moreover, two studies found that increasing complex carbohydrates improved insulin sensitivity while dietary fat had the opposite effect in people with the ‘A’ allele. Interestingly, this variant has also been associated with lower postprandial triglycerides [\[R, R, R\]](#).

Another allele usually inherited together with the previous one, ‘C’ at [rs2289487](#) (PLIN1 6209T>C), has been associated with:

- Decreased BMI and obesity risk in white women [\[R\]](#)
- Lower postprandial triglycerides [\[R\]](#)
- Higher risk of type 2 diabetes [\[R\]](#)
- Greater weight loss in response to dietary interventions [\[R, R\]](#)

Another well-researched variant is [rs1052700](#) (14995A>T), usually inherited together with rs894160 in Asians. Its minor ‘T’ allele has been associated with lower body weight, body fat, and waist circumference in whites, especially in women [\[R, R\]](#).

This variant has also been associated with greater weight loss in response to calorie restriction and intense exercise (HIIT) [\[R, R\]](#).

In contrast, this allele has been associated with an increased risk of type 2 diabetes and glucose intolerance. As was the case for rs894160, carriers of the minor allele may benefit from



LOWER ACTIVITY

Likely lower PLIN activity based on 7 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PEX11A	rs894160	CT
KIF7	rs1052700	AT
PLIN1	rs2289487	TC
PLIN1	rs2304795	AG
PLIN1	rs6496589	CC
UBXN6	rs8887	TT
PLIN1	rs2304796	GA

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

increasing their dietary ratio of complex carbohydrates to fat when it comes to reducing insulin resistance [R, R, R, R].

Another variant, ‘G’ at [rs2304795](#) has been associated with:

- Lower obesity risk, body fat percentage, and waist circumference [R]
- Greater weight loss in response to calorie restriction [R]
- Worse bone health [R]
- Lower fat mass loss in malnourished head and neck cancer patients [R]

This variant is usually inherited together with [rs2304796](#), whose minor ‘A’ allele has been associated with a reduced risk of central obesity and diabetes [R].

The minor ‘C’ allele of [rs6496589](#) has also been associated with a decreased risk of central obesity and type 2 diabetes [R, R].

Finally, the minor ‘T’ allele of *PLIN4* [rs8887](#) may reduce gene expression by creating a binding site of an inhibitor. This allele has been associated with a lower waist-to-hip ratio and decreased weight in response to dietary omega-3 fatty acids [R, R].

TMEM18 (Weight)

There are 4 SNPs in the *TMEM18* gene that are often inherited together and that have all been linked to obesity in many different studies. In all cases, the variant associated with obesity is the major allele.

The 'C' allele in the [rs6548238](#) SNP near the *TMEM18* gene was first linked to BMI and obesity in 2009, in a meta-analysis of 15 genome-wide association studies with over 91k people [\[R\]](#).

Since then, the link has been confirmed in many different studies, large and small. These studies involved children, adolescents, and/or adults from all over the world -- from Japan, China and New Zealand, over Europe and Africa, to North and South America [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

In a German study with over 12k people, each copy of the 'T' allele was linked to a 14% lower chance of being obese (BMI ≥ 30 kg/m²) [\[R\]](#).

In a study with over 700 brazilian children, those with 'CC' genotypes had a higher BMI already at 3.5 years old [\[R\]](#).

It's important to stress here, however, that this SNP seems to have different impacts in different populations. For example, the 'C' allele of rs6548238 seems to have a smaller impact on obesity in Africans compared to Europeans [\[R\]](#).

The 'C' allele of [rs2867125](#) has been linked to higher BMI in Europe, Korea, Japan, Singapore (including Chinese, Malay, and Indian ethnicities) and US (including Whites, African-Americans, and Pima Indians) [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

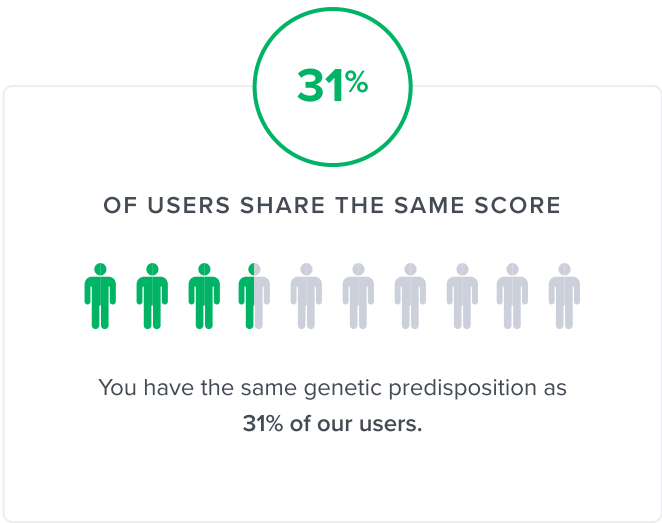
Interestingly, a study with over 5.4k Pima Indians found that the rs2867125 'C' allele corresponded to around a 3.0% increase in BMI per copy, which was a 2-fold larger effect compared to Europeans [\[R\]](#).

The 'A' allele of [rs7561317](#) has been associated with lower and the 'G' allele with higher body weight. The link to BMI and obesity was found in Europe, Emirates, Japan, Mexico, Pakistan, and US [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).



BETTER GENETICS

Likely better **TMEM18** genetics based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SH3YL1	rs6548238	TC
SH3YL1	rs2867125	TC
SH3YL1	rs7561317	AG
SH3YL1	rs4854344	GT

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

In a study with over 18k middle-aged Danes, each copy of the 'G' allele was associated with a 1.2 times higher risk of obesity and 1.5 times higher risk of morbid obesity [\[R\]](#).

Interestingly, in a study with over 1.4k Czech adolescents, the 'A' allele increased the risk of being underweight [\[R\]](#).

Finally, the 'T' allele of [rs4854344](#) has been associated with higher body weight, BMI, greater waist circumference and an increased risk of obesity in Greek, Japanese, Spanish, and US studies [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).



Inflammatory Disorders

Inflammation plays a key role in many chronic diseases, including cardiovascular disease. Genetic variations can influence how your immune system responds to infections, tissue damage, and other stressors, making some individuals more susceptible to inflammatory disorders. **Chronic inflammation has been linked to conditions like heart disease and autoimmune disorders.**

Genes such as *IL6*, *TNF*, and *CRP* are central to inflammation regulation and have been linked to cardiovascular disease risk. *IL1A* and *IL1B* are involved in early inflammatory responses, while *NF-kB* is a major regulator of inflammation at the cellular level. Additionally, *IL10* and *IL4* are anti-inflammatory cytokines that help modulate immune responses. Understanding how your genes affect inflammation can guide lifestyle adjustments and interventions to reduce your risk of inflammatory-related heart issues.

HIGHER ACTIVITY NF-kB (Inflammation) Likely higher NF-kB activity	HIGHER ACTIVITY IL1A (Inflammation) Predisposed to a higher IL1A activity	LOWER ACTIVITY HRH1 (Allergies) Likely lower HRH1 activity
HIGHER ACTIVITY HLA (Inflammation) Likely higher HLA activity	TYPICAL ACTIVITY IL6 Gene (Inflammation) Likely typical IL6 activity	TYPICAL ACTIVITY TNF Gene (Inflammation) Likely typical TNF activity
TYPICAL ACTIVITY CRP Gene Predisposed to typical CRP gene activity	TYPICAL ACTIVITY IL10 Gene (Autoimmunity) Predisposed to typical IL10 activity	TYPICAL ACTIVITY IL4 (Allergies, Autoimmunity) Likely typical IL4 activity
TYPICAL ACTIVITY IL13 (Allergies, Lung Function) Predisposed to typical IL13 activity	TYPICAL ACTIVITY HRH4 (Allergies, Inflammation) Predisposed to typical HRH4 activity	TYPICAL ACTIVITY DAO (Histamine) Likely typical DAO activity



TYPICAL ACTIVITY

HDC (Histamine)

Predisposed to a typical HDC activity



TYPICAL ACTIVITY

HMGB1 (Inflammation)

Predisposed to typical HMGB1 activity



TYPICAL ACTIVITY

SMAD3 (Allergies)

Predisposed to typical SMAD3 activity



TYPICAL ACTIVITY

GATA3 (Allergies)

Likely typical GATA3 activity



LOWER ACTIVITY

**IL1B (Inflammation/
Fatigue)**

Likely lower IL1B activity



HIGHER ACTIVITY

IL2 (Allergies)

Predisposed to a higher IL2 activity



LOWER ACTIVITY

**IL21 (Autoimmunity &
Allergies)**

Predisposed to lower IL21 activity



LOWER ACTIVITY

IL8 (Inflammation)

Likely lower IL8 activity



BETTER GENETICS

HRH2 (Gut Health)

Likely better HRH2 genetics



HIGHER ACTIVITY

HNMT (Histamine)

Likely higher HNMT activity

NF-KB (Inflammation)

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

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

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

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



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

Your top variants that most likely impact your genetic predisposition:

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

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

IL1A (Inflammation)

The best-characterized *IL1A* polymorphism is [rs1800587](#), also known as C889T. Its ‘A’ allele may increase IL1A expression and has been associated with an increased risk of [\[R\]](#):

- Acne [\[R\]](#)
- Intervertebral disc disease [\[R\]](#)
- Chronic rhinosinusitis [\[R\]](#)
- Graves’ disease [\[R\]](#)
- Ankylosing spondylitis [\[R\]](#)
- Hearing loss and Ménière’s disease [\[R\]](#)

Another well-researched variant is [rs17561](#), also known as +4845 G>T. Its minor ‘A’ allele may reduce IL-1α release from blood cells and retain more inside them. This variant has been associated with an increased risk of [\[R\]](#):

- Acne [\[R\]](#)
- Chronic rhinosinusitis and nasal polyposis [\[R\]](#)
- Ankylosing spondylitis [\[R\]](#)
- Gum disease [\[R\]](#)
- Endometriosis [\[R\]](#)



HIGHER ACTIVITY

Predisposed to a higher IL1A activity based on 2 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SLC20A1	rs1800587	GG
IL1A	rs17561	CC

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

HRH1 (Allergies)

The most widely-investigated *HRH1* polymorphism is [rs901865](#) (17C>T). Its minor ‘T’ allele decreases HRH1 production. This variant has been associated with:

- Increased prevalence of allergic asthma [\[R\]](#)
- Increased prevalence of allergic rhinitis and worse response to H1 antihistamines [\[R\]](#)

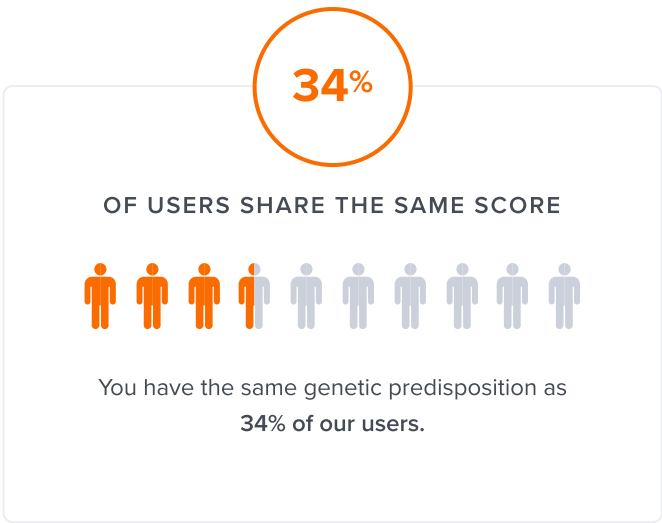
In turn, the major ‘C’ allele has been associated with:

- Severe sedation from desloratadine treatment for hives [\[R\]](#)
- Lower heel BMD [\[R\]](#)



LOWER ACTIVITY

Likely lower HRH1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
VGLL4	rs901865	CC

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

HLA (Inflammation)

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

- ‘A’ at [rs4349859](#)
- ‘G’ at [rs13202464](#)
- ‘T’ at [rs116488202](#)

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

- Celiac disease [\[R\]](#), [\[R\]](#)
- Allergies to latex and fruits [\[R\]](#)
- Grave’s disease [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)

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

- Peanut allergy [\[R\]](#), [\[R\]](#)
- Shrimp allergy [\[R\]](#)
- Multiple sclerosis [\[R\]](#)
- Primary sclerosing cholangitis [\[R\]](#)

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

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

The ‘C’ allele of [rs3763313](#) has also been associated with higher rates of Crohn’s disease and ulcerative colitis. This variant has



HIGHER ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

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

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

also been associated with an increased risk of tuberculosis but may be protective against dilated cardiomyopathy [R, R, R, R].

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

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

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

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

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

- Epstein-Barr virus infection
- Asthma
- Ulcerative colitis

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

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

IL6 Gene (Inflammation)

Since IL-6 can have both pro-inflammatory and anti-inflammatory properties, variants with decreased activity may increase the risk of infections while reducing chronic inflammation [R, R, R].

By far, the most well-researched *IL6* polymorphism is [rs1800795](#) (also known as the “-174G>C” polymorphism). The major 'G' allele of this SNP has been linked with higher levels of IL-6, while the 'C' allele has been associated with lower IL-6 levels [R].

The ‘G’ variant has been associated with some health benefits such as:

- [Faster muscle recovery and increased power performance](#) [R, R]
- [Increased longevity](#) [R]
- [Reduced risk of obesity](#) [R, R, R]
- [Reduced risk and severity of PTSD](#) [R]
- Reduced incidence of respiratory infections [R]
- Reduced risk of type 2 diabetes and insulin resistance [R, R]
- Reduced risk of blood clots [R]
- Decreased risk of coronary artery disease (especially in smokers or drinkers) [R, R]
- Decreased risk of cervical cancer [R, R, R]
- Decreased risk of prostate cancer [R]
- Decreased risk of PCOS [R]

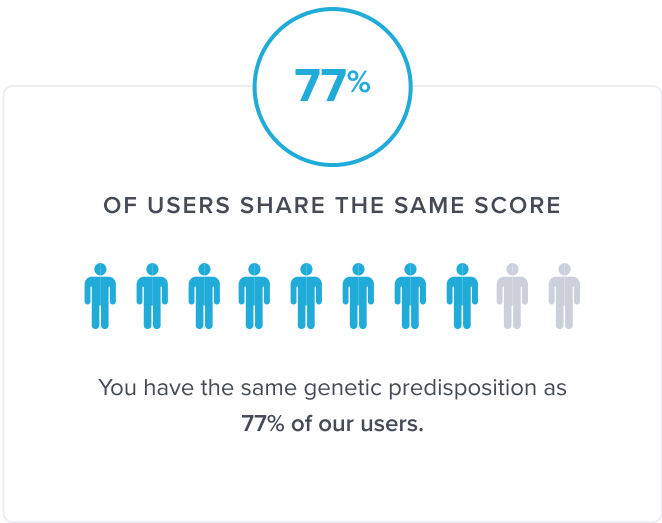
However, it may also increase the risk of some conditions such as:

- Gum disease [R, R]
- Rheumatoid arthritis [R]
- Coronary artery disease [R]
- Liver cancer [R]



TYPICAL ACTIVITY

Likely typical IL6 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
IL6	rs1800795	GG

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

TNF Gene (Inflammation)

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

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

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

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

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

- Crohn’s disease [\[R, R\]](#)
- Hashimoto’s disease [\[R\]](#)

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

- IBD [\[R, R, R\]](#)
- Acne [\[R\]](#)

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



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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PHACTR4	rs188618085	CC
YTHDF2	rs185682149	CC
TMEM71	rs72725197	CC
CRISPLD2	rs118135095	GG
TRIB2	rs116434579	GG
TNF	rs1799724	TC
NCR3	rs1800610	AG
ROBO1	rs149420276	AG
GPX3	rs111332265	GA
/	rs7256693	CC
ATP10B	rs149126334	GG
ANO3	rs10834997	GG
TNF	rs1800629	GG
SPRY1	rs115669577	GG
ERICH1	rs138808635	CC
PSD3	rs79105320	GG
MICB	rs361525	GG
TNF	rs1799964	TT
/	rs72841564	TT
GTF2E1	rs10511404	GG
/	rs8121916	CC
RBM11	rs72490194	CC
GBA2	rs10814274	TT

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

CRP Gene

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

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

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

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

- Higher CRP levels
- Obesity
- Pneumonia
- PTSD

Other notable CRP variants include:

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



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

Your top variants that most likely impact your genetic predisposition:

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

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

IL10 Gene (Autoimmunity)

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

- [IBD](#)
- Type 1 diabetes
- [Lupus](#)

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



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

Your top variants that most likely impact your genetic predisposition:

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

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

IL4 (Allergies, Autoimmunity)

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

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

- Hay fever [\[R\]](#)
- Eczema [\[R\]](#)
- Asthma [\[R\]](#), [\[R\]](#), [\[R\]](#)

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

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

- Rheumatoid arthritis [\[R\]](#)
- Multiple sclerosis [\[R\]](#), [\[R\]](#)
- Autoimmune thyroid disease [\[R\]](#)

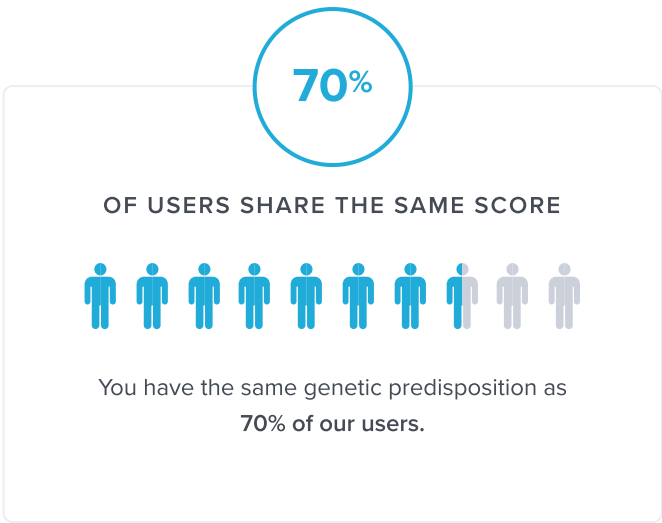
The ‘T’ allele may also increase the risk of infections such as tuberculosis and hepatitis B and C. However, it has also been associated with a decreased risk of severe symptoms in people with this and other infectious diseases such as HIV and malaria and with a better response to the hepatitis B vaccine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

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



TYPICAL ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SLC22A5	rs2243250	CC

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

IL13 (Allergies, Lung Function)

The most widely investigated IL13 variant is [rs20541](#). Its minor ‘A’ allele has been linked to elevated IL-13 and IgE levels. The ‘A’ allele has also been associated with an increased risk of:

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

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

The ‘A’ allele of [rs1295685](#) has also been associated with elevated IgE levels, as well as with an increased risk of asthma, psoriasis, eczema, and COPD [\[R, R, R, R, R, R, R\]](#).

The ‘T’ allele of [rs847](#) has been associated with an increased risk of COPD and eczema, as well as an increased severity of rosacea symptoms [\[R, R, R\]](#).

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

Another well-researched allele, ‘T’ at [rs1800925](#), may increase IL-13 levels. This variant has been associated with an increased risk of [\[R\]](#):

- Asthma [\[R, R\]](#)
- COPD [\[R, R, R\]](#)

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

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

Another variant usually inherited with the previous one, the ‘A’ allele of [rs848](#), has been associated with elevated IgE levels



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

Your top variants that most likely impact your genetic predisposition:

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

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

and an increased risk and severity of asthma, COPD, eczema, and alopecia areata [R, R, R, R, R, R].

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

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

HRH4 (Allergies, Inflammation)

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

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



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



Your top variants that most likely impact your genetic predisposition:

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

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

DAO (Histamine)

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

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

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

Two other minor variants, ‘T’ at [rs2268999](#) and ‘A’ at [rs2071514](#), have also been linked to lower DAO activity [R].

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



TYPICAL ACTIVITY

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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
AOC1	rs1049793	GC
AOC1	rs1049742	CC
AOC1	rs10156191	CC
AOC1	rs2268999	AA
AOC1	rs2052129	GG
AOC1	rs35070995	AA

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

HDC (Histamine)

One of the main *HDC* polymorphisms is [rs2073440](#), also known as Asp644Glu. Its minor ‘G’ allele may decrease histamine production and has been associated with a reduced risk of allergic rhinitis [\[R\]](#).

Another important *HDC* polymorphism is [rs17740607](#), also known as Thr31Met. Its minor ‘A’ allele may reduce HDC activity and has been associated with a decreased risk of chronic heart failure [\[R\]](#).



TYPICAL ACTIVITY

Predisposed to a typical HDC activity based on 2 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HDC	rs2073440	TT
HDC	rs17740607	GG

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

HMGB1 (Inflammation)

The best-researched *HMGB1* variant is [rs2249825](#). Its minor ‘C’ allele may increase *HMGB1* expression and has been associated with a decreased risk of [\[R\]](#):

- Colorectal cancer [\[R\]](#)
- Cervical cancer [\[R\]](#)
- Sepsis [\[R\]](#)
- Pneumonia [\[R\]](#)
- Hypertension [\[R\]](#)
- Delayed cerebral ischemia after subarachnoid hemorrhage [\[R\]](#)
- Atrial fibrillation after coronary artery bypass surgery [\[R\]](#)
- Delayed cerebral ischemia [\[R\]](#)
- Breast cancer progression [\[R\]](#)

However, this variant has also been associated with a decreased risk of rheumatoid arthritis and ischemic stroke [\[R\]](#), [\[R\]](#).

Another variant linked to higher HMGB1 levels is the ‘T’ allele of [rs1045411](#). This variant has been associated with an increased risk of:

- Cancer (particularly, breast and liver cancer) [\[R\]](#), [\[R\]](#)
- Atrial fibrillation [\[R\]](#)
- Sepsis [\[R\]](#), [\[R\]](#)
- Ischemic stroke [\[R\]](#)
- Lupus nephritis [\[R\]](#)
- Death in patients with advanced lung cancer [\[R\]](#)
- Prostate cancer progression [\[R\]](#)

However, this variant has also been associated with a decreased risk of COVID-19 infection, a better prognosis in patients with aggressive stomach cancer, and a reduced risk and invasiveness of urothelial cell carcinoma [\[R\]](#), [\[R\]](#), [\[R\]](#).

Another well-researched variant is [rs1412125](#). Its minor ‘T’ allele reduces *HMGB1* expression and has been associated with a decreased risk of [\[R\]](#):

- Cancer (overall and cervical) [\[R\]](#), [\[R\]](#)
- Kawasaki disease and complications in people with this condition [\[R\]](#), [\[R\]](#)
- Pneumonia [\[R\]](#)
- Ischemic stroke [\[R\]](#)
- Prostate cancer progression [\[R\]](#)



Predisposed to typical HMGB1 activity based on 4 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HMGB1	rs1412125	CC
HMGB1	rs1360485	TT
HMGB1	rs2249825	GG
HMGB1	rs1045411	CC

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

- Breast cancer progression [\[R\]](#)

However, this allele has been associated with an increased risk of lung cancer and distant metastasis in patients with liver cancer [\[R\]](#), [\[R\]](#).

Finally, the minor ‘T’ allele of [rs1360485](#) may increase *HMGB1* expression and has been associated with a decreased risk of:

- Pneumoconiosis in coal workers [\[R\]](#)
- Breast cancer progression [\[R\]](#)
- Poorly differentiated, fast-spreading lung carcinoma [\[R\]](#)

However, this variant has been associated with an increased risk of:

- Lung cancer [\[R\]](#)
- Prostate cancer progression [\[R\]](#)
- Rheumatoid arthritis [\[R\]](#)

SMAD3 (Allergies)

The following *SMAD3* variants have been associated with allergic diseases such as asthma, hay fever, and eczema:

- ‘G’ of [rs17228058](#) [R]
- ‘A’ of [rs72743461](#) [R]
- ‘T’ of [rs56062135](#) [R]
- ‘G’ of [rs17294280](#) [R]
- ‘T’ of [rs17293632](#) [R]

These variants may reduce SMAD3 activity, thereby blocking the anti-inflammatory action of TGF-beta [R].



TYPICAL ACTIVITY

Predisposed to typical SMAD3 activity based on 4 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SMAD3	rs72743461	CC
AAGAB	rs56062135	CC
SMAD3	rs17294280	AA
SMAD3	rs17293632	CC
SMAD3	rs17228058	AA

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

GATA3 (Allergies)

The following *GATA3* variants have been associated with allergic diseases such as asthma, hay fever, and eczema [R, R, R]:

- ‘C’ of [rs962993](#)
- ‘T’ of [rs12785018](#)
- ‘A’ of [rs10795656](#)
- ‘C’ of [rs12413578](#)
- ‘G’ of [rs72782676](#)
- ‘T’ of [rs11255753](#)
- ‘C’ of [rs7894791](#)

These variants may increase production or activity of GATA3, which may lead to elevated production of Th2-related cytokines, enhanced airway responsiveness to allergens, and excess release of IgE antibodies.



Likely typical GATA3 activity based on 7 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GATA3	rs7894791	CC
GATA3	rs962993	TC
GATA3	rs10795656	GA
GATA3	rs12413578	TC
GATA3	rs11255753	GT
/	rs72782676	CC
GATA3	rs12785018	CC

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

IL1B (Inflammation/ Fatigue)

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

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

In addition, the variant has been linked to:

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

Other variants that may increase *IL1B* activity include:

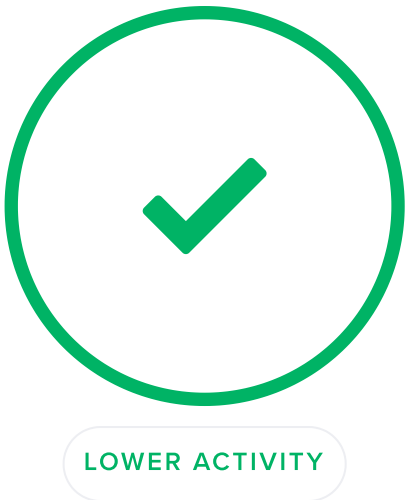
- ‘C’ at [rs1143633](#) [R](#)
- ‘T’ at [rs1143643](#) [R](#)
- ‘A’ at [rs1143627](#) [R](#)
- ‘A’ at [rs1143634](#) [R](#), [R](#)
- ‘C’ at [rs1143623](#) [R](#)

These variants have been associated with

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

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

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



LOWER ACTIVITY

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

Your top variants that most likely impact your genetic predisposition:

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

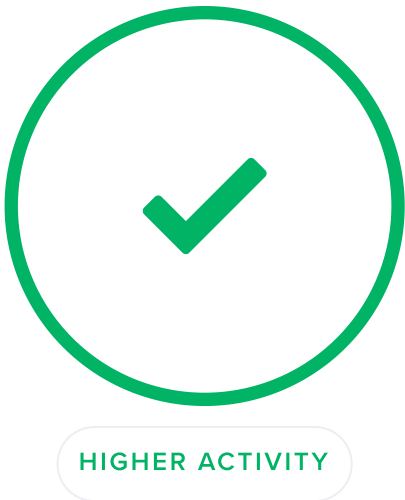
The number of "risk" variants in this table doesn't necessarily reflect your overall result.

IL2 (Allergies)

The following variants in the *IL2* gene have been associated with allergic diseases including asthma, eczema, and hay fever [\[R\]](#):

- ‘A’ of [rs17388568](#)
- ‘G’ of [rs17454584](#)
- ‘G’ of [rs148505069](#)
- ‘C’ of [rs2069772](#)
- ‘A’ of [rs2069763](#)
- ‘C’ of [rs2069762](#)

IL-2 is considered a [Th1 cytokine](#), while allergic reactions are considered part of the Th2 response. Therefore, it’s possible that these genetic variants that decrease IL-2 production could increase susceptibility to allergies [\[R\]](#).



Predisposed to a higher IL2 activity based on 6 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
IL2	rs2069762	AC
KIAA1109	rs2069772	TT
KIAA1109	rs17454584	AA
IL2	rs2069763	CC
KIAA1109	rs17388568	GG
KIAA1109	rs148505069	AA

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

IL21 (Autoimmunity & Allergies)

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

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

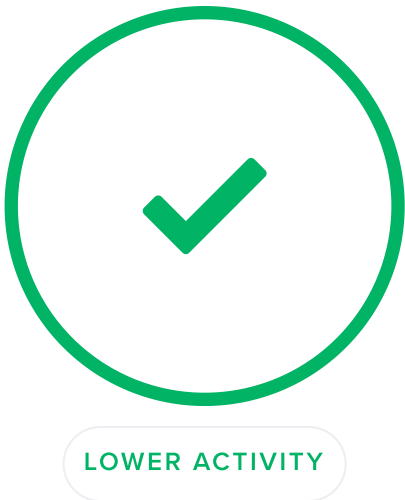
This variant is almost always inherited with [rs13119723-G](#), meaning that most people will either have none or both of them [\[R\]](#).

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

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

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

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



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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
IL21	rs6822844	TG
IL21	rs907715	TC
KIAA1109	rs13119723	GA
IL2	rs6848139	AA
KIAA1109	rs2221903	TT

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

IL8 (Inflammation)

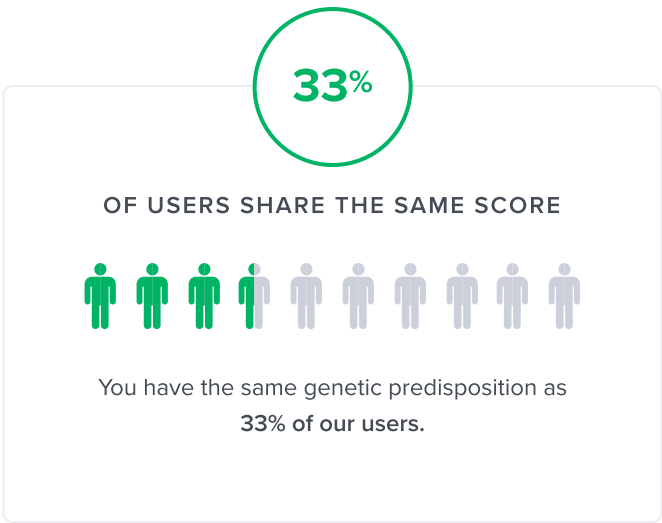
The main IL8 polymorphism is [rs4073](#). Its ‘A’ allele increases *IL8* expression in white blood cells, which may contribute to inflammatory conditions. In line with this, this variant has been associated with an increased risk of [\[R\]](#), [\[R\]](#), [\[R\]](#):

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



LOWER ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PF4V1	rs4073	TT

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

HRH2 (Gut Health)

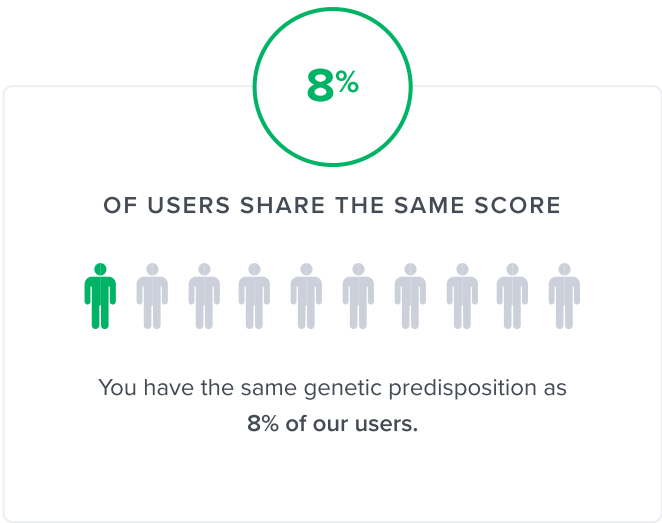
The most widely-investigated *HRH2* polymorphism is [rs2067474](#) (-1018 G>A). Its minor ‘A’ allele may affect gene expression based on its position within the gene sequence but its exact contribution to HRH2 function hasn’t been established. This variant has been associated with:

- Decreased risk of stomach lining atrophy [\[R\]](#), [\[R\]](#)
- Decreased risk of stomach cancer [\[R\]](#)



BETTER GENETICS

Likely better HRH2 genetics based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HRH2	rs2067474	GG

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

HNMT (Histamine)

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

- Allergies
- Asthma
- Eczema
- Migraines
- ADHD

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

Another variant, the ‘T’ allele at [rs11558538](#), is associated with decreased HNMT activity and increased risk of schizophrenia, ADHD, and migraine [\[R\]](#).



HIGHER ACTIVITY

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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HNMT	rs1050891	GA
HNMT	rs11558538	CC

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



Food Responses

Food sensitivities can significantly impact overall health, and understanding your genetic predisposition to certain food reactions can help you make more informed dietary choices. **Sensitivities to ingredients like gluten, dairy, and certain flavors or chemicals may contribute to digestive discomfort and inflammation.**

Genes like *CYP1A2* and *ALDH2* influence how your body metabolizes caffeine and alcohol, respectively. *HLA-DQ2* is associated with gluten sensitivity, while *MCM6* plays a role in lactose digestion. Additionally, the gene encoding the bitter taste receptor *TAS2R38* can affect how you perceive certain foods. By understanding your genetic risks for food sensitivities, you can better manage your health and make dietary choices that support your well-being.

 HIGHER ACTIVITY ADH1B (Alcohol Sensitivity) Predisposed to higher ADHB1 activity	 LOWER ACTIVITY MCM6 (Lactose Intolerance) Likely lower MCM6 activity	 LOWER ACTIVITY SI (Digestive Issues) Predisposed to lower SI activity
 HIGHER Dairy Sensitivity (Casein) Likely higher sensitivity to dairy	 LOWER ACTIVITY ALDH2 (Alcohol Sensitivity) Likely typical ALDH2 activity	 TYPICAL GENETICS ALDH1B1 (Alcohol Sensitivity) Predisposed to typical ALDH1B1 genetics
 TYPICAL GENETICS HLA-DQ (Gluten) Likely typical HLA-DQ genetics	 TYPICAL ACTIVITY ALDOB (Fructose Intolerance) Predisposed to typical ALDOB activity	 INTERMEDIATE ACTIVITY TAS2R38 (Bitter Taste Perception) Likely intermediate TAS2R38 activity
 TYPICAL ACTIVITY TAS2R16 (Bitter Taste/ Longevity) Likely typical TAS2R16 activity	 HIGHER ACTIVITY CYP1A2 (Detox) Likely higher CYP1A2 activity	 LIKELY TOLERANT Lactose Intolerance Likely lactose tolerant

ADH1B (Alcohol Sensitivity)

Carrying a ‘T’ allele of [rs1229984](#), a SNP in the *ADH1B* gene, can result in a 70- to 80-fold increase in the rate of conversion of alcohol to acetaldehyde following alcohol consumption. The ‘T’ allele of rs1229984 can also cause side effects, including [migraines](#), flushing, and breathlessness [\[R, R, R\]](#).

The ‘T’ variant of rs1229984 is particularly common in East Asians, where it is also associated with lower alcohol consumption and negative reactions to drinking alcohol, such as flushing [\[R, R, R\]](#).

On the other hand, several studies have associated carrying a ‘C’ allele of rs1229984, a SNP in the *ADH1B* gene, with an overall increase in alcohol drinking. This is relative to carriers of the ‘T’ allele, which has been linked to a reduced risk of alcohol dependence [\[R, R, R\]](#).

A study found that carriers of the ‘A’ allele of [rs2066702](#) felt more tired and had an accelerated heart rate after drinking alcohol [\[R\]](#).

Two genome-wide studies, of more than 69,000 people combined, found a significant protective effect of carrying an ‘A’ allele of the SNP rs2066702 on alcohol dependence in African Americans. There are additional studies also confirming the link [\[R, R, R\]](#).

Finally, two studies (one of almost 3000 people in China and another of around 4600 Australian twins) found a significant relationship between carrying a ‘C’ allele of [rs1042026](#) and reduced alcohol consumption [\[R, R\]](#).

It's important to mention that there are also conflicting studies that have found no association between *ADH1B* variant carriers and alcohol dependence or breakdown. Other genetic variants and environmental factors such as peer pressure and social drinking may play a greater role in drinking habits [\[R, R\]](#).



Predisposed to higher ADHB1 activity based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ADH1B	rs1229984	TC
ADH1C	rs1042026	TC
ADH1B	rs2066702	GG

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

MCM6 (Lactose Intolerance)

Humans became capable of digesting lactose into adulthood recently on an evolutionary scale: about 5,000 to 8,000 years ago. It’s likely that all humans who walked this Earth over 10,000 years ago were lactose-intolerant hunter-gatherers. But by 4000 BC, a shift occurred in Europe: farming became the norm. Hunter-gatherer societies were pushed out of the picture [R].

This overlaps with the origins of animal domestication and the practice of dairying. Scientists proposed that lactose digestion emerged as an evolutionary advantage. It likely helped some of our ancestors survive during times of famine. **People who can digest lactose are thought to be descendants of those early dairy farmers.** This might explain why the lactase-persistence *MCM6* variants are more common among people of European descent [R].

The following *MCM6* variants have been associated with lactase persistence and a decreased risk of lactose intolerance [R, R]:

- ‘A’ at [rs4988235](#)
- ‘T’ at [rs182549](#)
- ‘C’ at [rs41380347](#)
- ‘G’ at [rs145946881](#)



Likely lower MCM6 activity based on 4 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
LCT	rs4988235	AG
MCM6	rs41380347	AA
MCM6	rs145946881	CC
LCT	rs182549	TC

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

SI (Digestive Issues)

As far as we know, CSID is an autosomal recessive disorder, meaning that both copies of a person’s *SI* gene must be defective for them to suffer from CSID. There are currently four known variants that can cause CSID: Val577Gly (‘C’ of [rs121912615](#)), Gly1073Asp (‘T’ of [rs121912616](#)), Phe1745Cys (‘C’ of [rs79717168](#)), and Arg1124X (‘A’ of [rs200451408](#)). Any combination of these four mutations, as long as they are present on both copies of the *SI* gene, causes CSID [\[R\]](#), [\[R\]](#), [\[R\]](#).

While defective *SI* is best known for causing CSID, smaller reductions in *SI* activity could be responsible for some cases of IBS. In fact, one study of 1887 people found that *SI* variants were significantly more common in IBS patients than in matched controls [\[R\]](#).

The most significant mutation was ‘A’ at [rs9290264](#), which may reduce enzymatic activity by up to 35% compared to the *SI* enzyme without detrimental variants. This variant has been rated in the top 1% of most deleterious changes in the human genome. Carriers of this variant may be at increased risk of IBS-D and IBS-M. Moreover, following a low-FODMAP diet may be less effective at improving IBS symptoms in those with two copies [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

Two other *SI* variants with decreased activity and associated with reduced effectiveness of a low-FODMAP diet are ‘C’ at [rs147207752](#) and ‘G’ at [rs146785675](#) [\[R\]](#).



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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SI	rs9290264	AA
SI	rs79717168	AA
/	rs200451408	GG
SI	rs121912615	AA
SI	rs147207752	TT
SI	rs146785675	AA

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

Dairy Sensitivity (Casein)

As mentioned, IgG antibodies to casein may be a measure of dairy sensitivity. Up to **45%** of differences in these antibodies may be due to genetics [R].

However, the impact of individual gene variants is questionable. Scientists found one gene variant, [rs11635085](#)-G, that might be linked to lower levels of antibodies to casein. It’s still not clear how this variant might affect the immune response to casein [R].

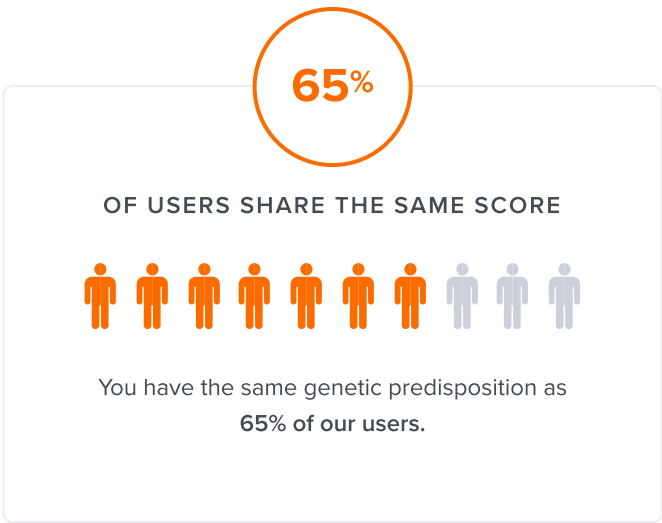
Importantly, we all produce IgG antibodies to casein. They peak in infancy when cow’s milk is introduced and then steadily drop as we age. Hence, **high levels of these antibodies don’t necessarily mean a person is sensitive to dairy** [R, R].

Please note: *The number of available gene variants for this report is low, so take your results with a grain of salt. We will update the report as soon as more research is available. Also, keep in mind that your diet, age, and environment can also influence dairy sensitivity. Consult a healthcare professional before making any major dietary changes.*



HIGHER

Likely higher sensitivity to dairy based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ST8SIA2	rs11635085	TT

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

ALDH2 (Alcohol Sensitivity)

A study of 251 Japanese people found that carriers of an ‘A’ allele at the *ALDH2* [rs671](#) variant were more likely to experience a hangover. As a result, carriers drink less and are less likely to be alcohol-dependent than non-carriers [\[R\]](#).

Carriers of the ‘A’ allele have been reported to get drunk faster and are more likely to experience a hangover, especially if they have [vitamin B12](#) deficiency. Vitamin B12 can assist in acetaldehyde breakdown. Variant carriers who drink alcohol with this deficiency may be more at risk of the negative health effects associated with acetaldehyde buildup [\[R\]](#), [\[R\]](#).

The rs671 SNP is most prevalent in Asian populations and is almost non-existent in other populations. 31% of individuals of East Asian descent carry at least one copy of the ‘A’ allele, while less than 1% of any other ethnicity carry a copy [\[R\]](#).

Enzyme activity is completely reduced in those who carry two copies of the ‘A’ allele, and by 50-70% for those who carry one [\[R\]](#).

Acetaldehyde is toxic and has also been shown to damage DNA. This means that the acetaldehyde buildup caused by drinking alcohol whilst carrying the rs671 variant may inflict nerve pain. This may also put the carrier at increased risk of cancer, such as throat, stomach, liver, bowel, and mouth [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

In fact, one study noted that carriers of the rs671 variant were at 20% higher relative risk of developing cancers than non-carriers, although this was not related to alcohol intake [\[R\]](#).

Individuals with 'AA' or 'AG' genotypes of rs671 have a higher heart disease risk if they don’t **exercise regularly** or if they eat fried food regularly [\[R\]](#).

The rs671 variant has also been linked to:

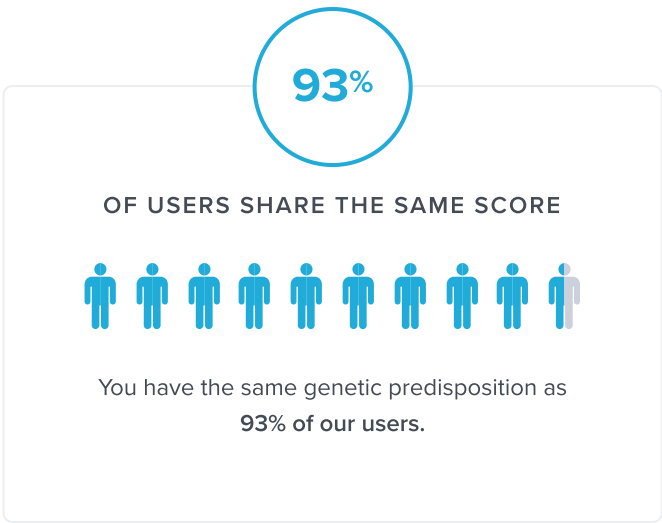
- Nonalcoholic fatty liver disease [\[R\]](#)
- Alzheimer's disease [\[R\]](#)
- Parkinson’s disease [\[R\]](#)

ALDH enzymes (particularly ALDH2) metabolize imidazole acetaldehyde, the intermediate compound created when



LOWER ACTIVITY

Likely typical ALDH2 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ALDH2	rs671	GG

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

histamine is broken down by diamine oxidase (DAO). This step is essential for converting histamine byproducts into forms safely eliminated from the body.

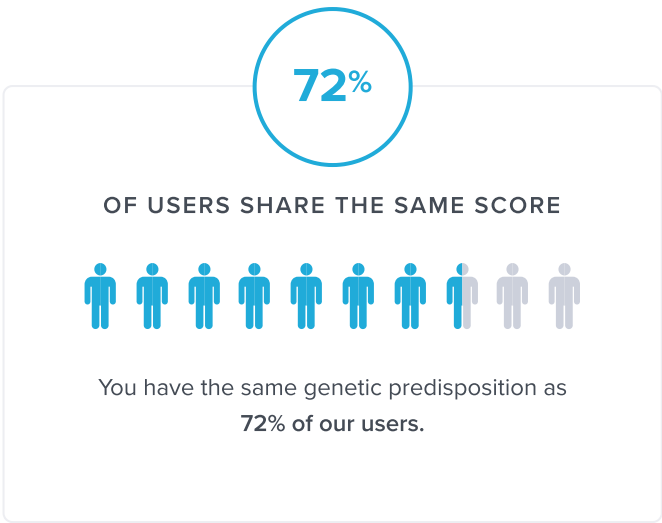
ALDH1B1 (Alcohol Sensitivity)

A study of two Danish populations (1216 and 6784 subjects) associated the minor ‘T’ allele of [rs2228093](#) with an increased risk of alcohol-induced hypersensitivity. In line with this, the variant has been associated with lower alcohol intake [\[R\]](#), [\[R\]](#).



TYPICAL GENETICS

Predisposed to typical ALDH1B1 genetics based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ALDH1B1	rs2228093	CC

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

HLA-DQ (Gluten)

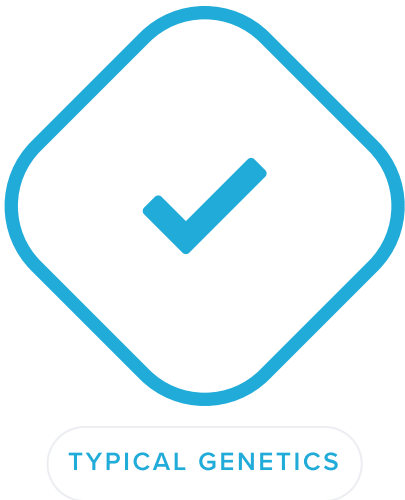
Different alleles in these genes can produce different types of HLA-DQ structures. The **DQ2** type (especially the DQ2.5 subtype) is present in up to 98% of celiac disease patients, depending on the population. That is among the strongest known links to autoimmunity in the entire HLA system [R, R].

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

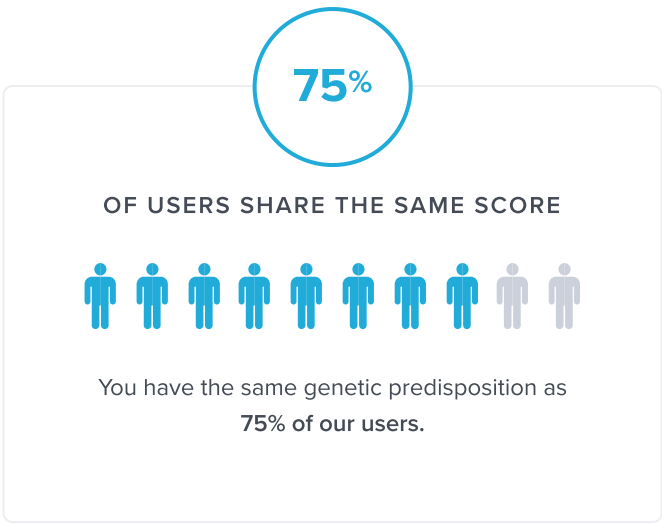
The ‘T’ variant of [rs2187668](#) serves as a genetic marker — it tags the DQ2.5 haplotype with high precision. In other words, the vast majority of people with this allele will have this haplotype. A study of over 27,000 subjects identified this SNP as the primary genetic factor for celiac disease.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

The 'C' variant of [rs74541084](#) tags the DQ8 haplotype, an additional marker for gluten sensitivity in people who don't carry DQ2.5.

Please note: this report only analyzes the HLA-DQ gene. Variants in many other genes have shown associations with gluten sensitivity.



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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HLA-DQB1	rs2858331	AA
HLA-DQA1	rs2187668	CC
HLA-DQA2	rs7454108	TT

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

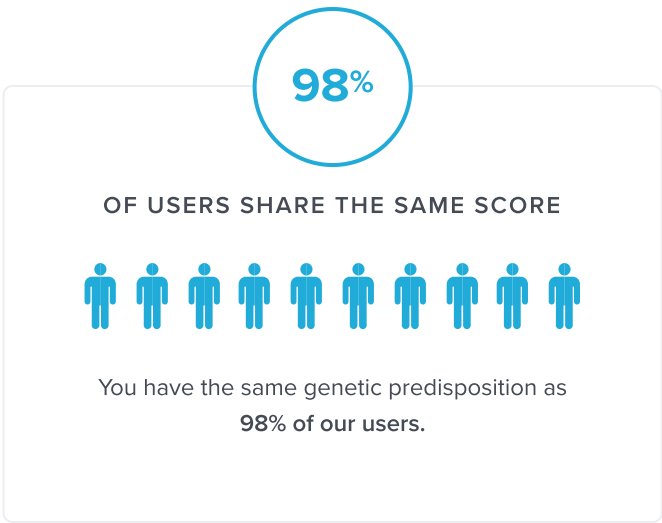
ALDOB (Fructose Intolerance)

Several rare mutations encoding less active or stable forms of the ALDOB protein have been associated with hereditary fructose intolerance. These include the ‘G’ allele of [rs1800546](#) (A149P) and the ‘T’ allele of [rs76917243](#) (A174D) [[R](#), [R](#), [R](#)].

Note that these variants are very rare, so you will most likely not carry any of them.



Predisposed to typical ALDOB activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
/	rs76917243	GG
ALDOB	rs1800546	CC

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

TAS2R38 (Bitter Taste Perception)

There are three main variants in the *TAS2R38* gene that are "linked", which means they are often inherited together:

- [rs713598](#): where 'G' is the "taster" and 'C' is the "non-taster"
- [rs1726866](#): where 'G' is the "taster" and 'A' is the "non-taster"
- [rs10246939](#): where 'C' is the "taster" and 'T' is the "non-taster"

These variants account for up to 85% of individual differences in the perception of bitterness [\[R\]](#)!

People who have two copies of the "taster" variant in each of these SNPs ('GG' for rs713598 + 'GG' for rs1726866 + 'CC' for rs10246939) are considered "super-tasters" [\[R, R, R\]](#).

On the other hand, people with two copies of the "non-taster" variant for each SNP ('CC' for rs713598 + 'AA' for rs1726866 + 'TT' for rs10246939) are considered "non-tasters" and are about 80% likely to not taste the bitterness of specific foods [\[R, R, R\]](#). This doesn't mean they don't taste bitterness at all; it means they have a lower sensitivity to it.

You may fall somewhere in between, in which case you'd have intermediate sensitivity to bitterness [\[R\]](#).

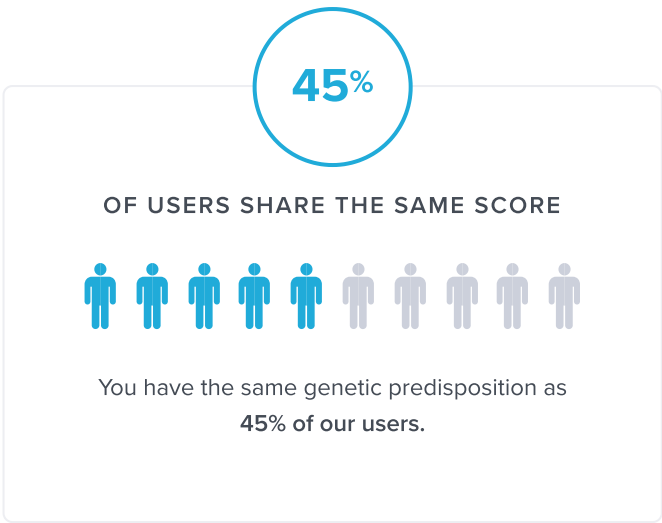
In addition to lower sensitivity to bitterness, non-tasters may have:

- Higher [tendency to overeat](#) or eat calorie-dense foods [\[R, R, R\]](#)
- Increased [body weight](#) and risk of obesity [\[R, R, R\]](#)



INTERMEDIATE ACTIVITY

Likely intermediate TAS2R38 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TAS2R38	rs713598	GC
TAS2R38	rs1726866	GA
TAS2R38	rs10246939	CT

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

TAS2R16 (Bitter Taste/Longevity)

Some researchers have suggested that people with highly sensitive bitter taste receptors may not consume as many bitter foods—including healthy vegetables like broccoli [\[R\]](#), [\[R\]](#), [\[R\]](#).

Some people think broccoli and Brussels sprouts taste good, while others can’t see past their bitter qualities. But avoiding vegetables may have consequences: people who regularly eat cruciferous vegetables live, on average, longer lives than those who don’t [\[R\]](#).

Certain variants of *TAS2R16* may improve the subjective taste of cruciferous vegetables and other bitter foods. People with these alleles may be naturally inclined to seek out and eat more of these foods and, thus, pursue a healthier diet. These include [\[R\]](#):

- ‘C’ at [rs6466849](#)
- ‘C’ at [rs860170](#)
- ‘T’ at [rs978739](#)

One especially strange possible link between *TAS2R16*, taste, and longevity crops up at rs6466849. People with the ‘C’ allele here are both more likely to live longer and less likely to think red wine tastes sour (using vinegar as a point of comparison) [\[R\]](#), [\[R\]](#).



Likely typical TAS2R16 activity based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TAS2R16	rs860170	TT
TAS2R16	rs6466849	CT
TAS2R16	rs978739	TC

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

CYP1A2 (Detox)

CYP1A2 is an enzyme that helps break down caffeine, drugs, and certain toxins like mold. Variants in the *CYP1A2* gene affect how fast people break down those substances [R, R, R].

The "slow metabolizer" variants make a less efficient enzyme. People who carry these variants may be more **sensitive to caffeine**. Accordingly, they may be more likely to experience negative effects when drinking coffee [R, R, R].

In terms of detox, they may be more susceptible to the adverse effects of certain drugs and toxins. However, the link between CYP1A2 variants and environmental toxins is more complex and requires further investigation [R, R].

The "fast metabolizer" variant makes a protein that breaks down caffeine. People with these variants may be less sensitive to its effects [R, R, R, R].

Nevertheless, "fast metabolizers" may experience the benefits of caffeine supplementation on athletic performance after a short time while "slow metabolizers" may need a longer ingestion period [R, R].

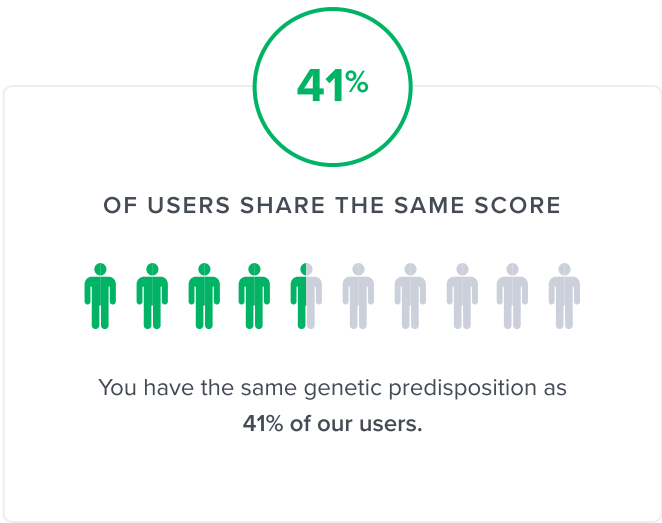
The following factors and substances may **increase** CYP1A2 activity:

- Cigarette smoke: 1.72-fold for >20 cigarettes per day [R, R]
- Coffee consumption: 1.45-fold per liter of coffee drunk daily [R, R]
- Meat pan-fried at high temperatures: 1.4-fold [R]
- Chargrilled meat: 1.89-fold [R]
- Cruciferous vegetables [R, R, R]
- Green and black tea [R]
- Insulin [R]
- Being female: 0.90-fold [R]
- Heavy exercise [R]
- Omeprazole [R]
- Evodia
- Reishi
- Andrographis,
- Modafinil
- Glycyrrhizin (liquorice)



HIGHER ACTIVITY

Likely higher CYP1A2 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CYP1A2	rs762551	AA
LMAN1L	rs2069514	GG

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

The following factors and substances may **decrease** CYP1A2 activity:

- Apiaceous vegetables (carrots, parsnips, celery, and parsley) [\[R\]](#)
- Curcumin [\[R\]](#)
- Grapefruit juice and its component naringenin [\[R\]](#)
- Echinacea [\[R\]](#)
- Quercetin [\[R\]](#)
- Antibiotic fluoroquinolones [\[R\]](#)
- Fluvoxamine, an antidepressant [\[R\]](#)
- Peppermint, [chamomile](#), and [dandelion](#) tea [\[R\]](#)
- Garlic
- Berberine
- Chamomile
- Lactoferrin
- Hops
- Galangin (galangal root)
- Scutellaria baicalensis,
- Tangeritin
- Trans-resveratrol

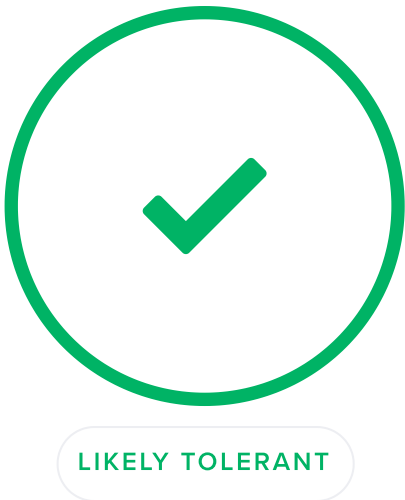
Lactose Intolerance

Lactose intolerance means a person cannot digest lactose, a sugar found in dairy. To be able to digest lactose, you need an enzyme called *lactase*. People with lactose intolerance may experience symptoms such as diarrhea, stomach cramps, nausea, bloating, and gas after eating dairy [R, R].

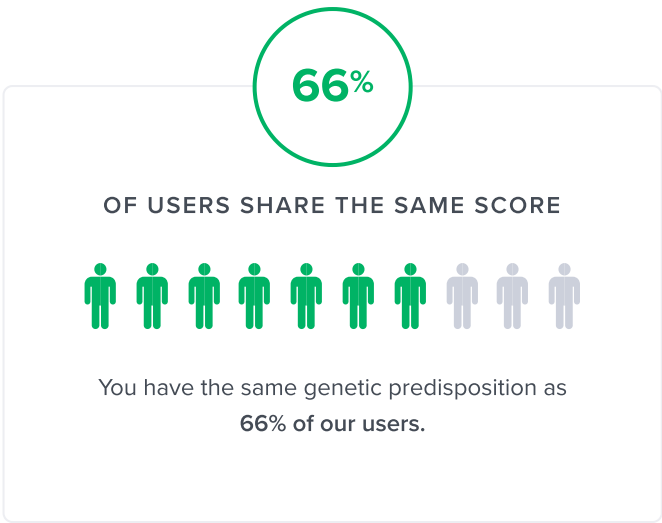
In people who are lactose intolerant, the gene that makes the enzyme lactase—*LCT*—gets "turned off" in adulthood. Without this enzyme, people may have trouble digesting dairy as adults [R, R].

A common variant near the *LCT* gene (rs4988235 'A') is responsible for keeping the lactase enzyme “turned on.” This variant is responsible for lactose tolerance in most people who are able to digest milk as adults. The 'T' allele of rs182549 has the same effect. Because these variants are usually inherited together, you will most likely have both variants or neither of them [R, R].

It's important to note that there are also other less common variants linked to lactose tolerance that we are not including in this report [R, R]. In addition, the way people respond to dairy may also depend on factors like diet, gut bacteria, and certain health conditions.



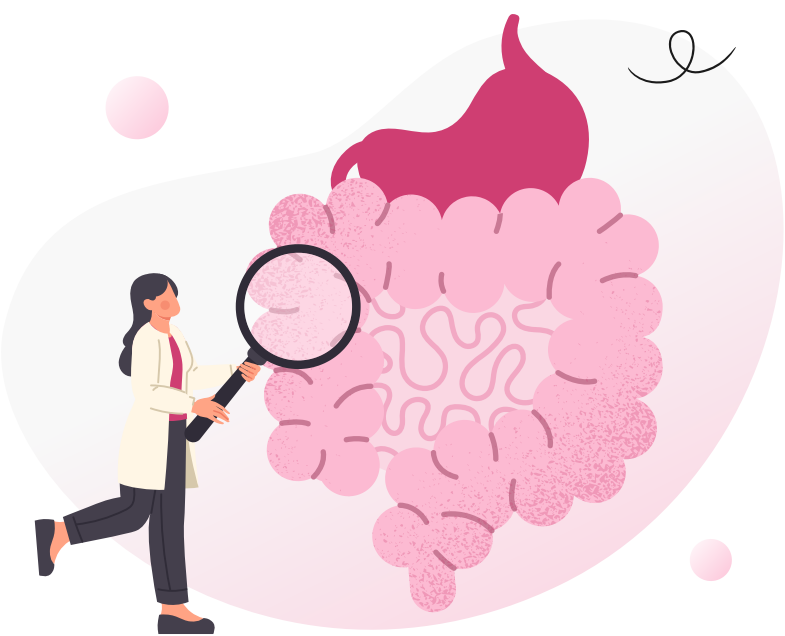
Likely lactose tolerant based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
LCT	rs4988235	AG

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





Gut Inflammation & Microbiome

The health of your gut is closely linked to overall well-being, including immune function, metabolism, and heart health. **Gut inflammation and an imbalanced microbiome can contribute to chronic diseases by influencing systemic inflammation and nutrient absorption.**

Genes like FUT2, TLR4, and NOD2 play a key role in the immune response and gut inflammation, while IL23R is associated with inflammatory bowel diseases like Crohn’s disease. MUC1 and IBD5 are involved in maintaining the integrity of the gut lining, which helps prevent inflammation. Additionally, NLRP3 and JAK2 influence inflammatory pathways that affect both gut health and cardiovascular disease risk. Understanding these genetic factors can help guide interventions to optimize gut health and reduce inflammation, ultimately benefiting overall heart health.

 LOWER ACTIVITY IBD5 (Gut Inflammation) Predisposed to lower IBD5 activity	 HIGHER ACTIVITY HTR3A (Gut Health) Predisposed to a higher HTR3A activity	 HIGHER ACTIVITY IL23R (Gut Inflammation) Predisposed to higher IL23R activity
 HIGHER ACTIVITY NLRP3 (Gut Inflammation) Likely higher NLRP3 activity	 LOWER ACTIVITY HNF4A (Gut Inflammation) Likely lower HNF4A activity	 SECRETOR FUT2 (Gut/ Vitamin B12) Likely secretor
 TYPICAL ACTIVITY TLR4 (Inflammation) Likely typical TLR4 activity	 TYPICAL ACTIVITY TNFSF15 (Gut Inflammation) Predisposed to a typical TNFSF15 activity	 TYPICAL GENETICS MUC1 (Immunity, Gut, Minerals) Likely typical MUC1 genetics

 TYPICAL GENETICS SLC2A14 (Gut Inflammation) Likely typical SLC2A14 genetics	 LOWER ACTIVITY MRGPRX2 (Gut Inflammation) Predisposed to lower MRGPRX2 activity	 LOWER ACTIVITY JAK2 (Gut Inflammation) Likely lower JAK2 activity

IBD5 (Gut Inflammation)

A large meta-analysis of over 75,000 subjects found 16% higher disease rates per “T” allele of the [rs2188962](#) polymorphism. In another meta-analysis of over 15,000 participants, this variant was associated with 25% higher odds of Crohn’s disease. However, a review with nearly 10,000 people didn’t find a link between rs2188962-T and ulcerative colitis. This variant belongs to a gene called [C5ORF56](#). Interestingly, the study on Crohn’s disease patients found that all *IBD5* variants were associated with decreased expression of another gene, [SLC22A5](#) [\[R\]](#), [\[R\]](#), [\[R\]](#).

A study of 158 families with Crohn's disease-affected members also suggested *SLC22A5* as a causal genetic factor. More precisely, the authors suggested two SNPs that may play a role in IBD development [\[R\]](#):

- [rs1050152](#)-T in the [SLC22A4](#) (OCTN1) gene
- [rs2631367](#)-C in the *SLC22A5* (OCTN2) gene

These variants are almost always inherited together, along with other ones from the *IBD5* region. *SLC22* genes enable the uptake of the amino acid [carnitine](#) in the gut lining.

A meta-analysis of 26 studies confirmed the link between the above two variants and IBD. They correlated with 20-23% higher rates of both Crohn's disease and ulcerative colitis. This paper identified another significant *IBD5* SNP, [rs12521868](#), associated with 36% higher IBD rates [\[R\]](#).

The *SLC22A4* and *SLC22A5* genes encode transport proteins (OCTN1 and OCTN2) that supply the gut lining with carnitine and other essential components. Mice lacking OCTN2 develop gut inflammation, ulcers, and perforations due to carnitine deficiency [\[R\]](#), [\[R\]](#).

A study of 944 participants found that people with *SLC22A4* and *SLC22A5* variants have reduced gene expression in the blood and gut tissue, making them prone to inflammation. This confirms the mentioned link between *IBD5* SNPs and lower *SLC22A5* expression [\[R\]](#).



LOWER ACTIVITY

Predisposed to lower IBD5 activity based on 4 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SLC22A5	rs12521868	TT
SLC22A5	rs2188962	TT
SLC22A4	rs1050152	TC
SLC22A4	rs2631367	CG

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

HTR3A (Gut Health)

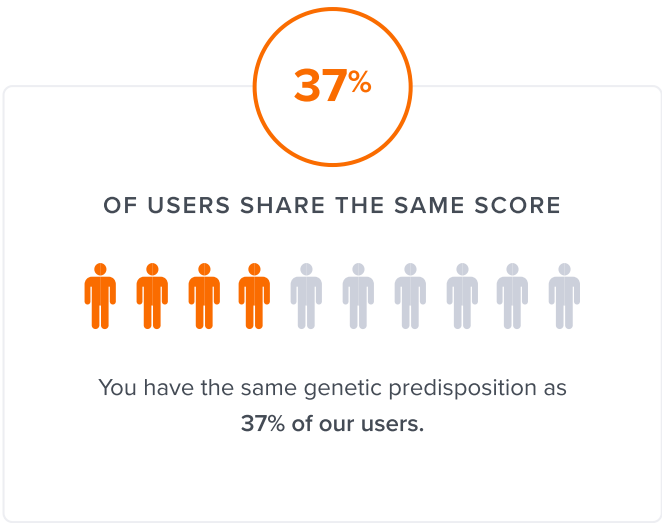
In theory, variants in the 5-HT3 receptor that increase its activity could increase movement in the gut and increase susceptibility to IBS.

One variant in the *HTR3A* gene has been directly linked to IBS. At [rs1062613](#), the minor ‘T’ allele was associated with increased rates of IBS and increased receptor activation [[R](#), [R](#)].



HIGHER ACTIVITY

Predisposed to a higher HTR3A activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NNMT	rs1062613	CT

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

IL23R (Gut Inflammation)

Scientists have identified different SNPs in the *IL23R* gene associated with both Crohn’s disease and ulcerative colitis. Given their effects vary between populations, we will first outline the results for Europeans (Caucasians) and then mention the key differences in Asians.

A comprehensive review of 41 studies and over 47,000 participants has found a robust link between two SNPs and IBD [\[R\]](#):

- [rs11209026](#): the “A” allele was associated with lower rates of both CD and UC
- [rs10889677](#): the “A” allele was associated with higher rates of both CD and UC

Combined, people with rs11209026-A had two times lower rates, and those with rs10889677-A had 37% higher rates of IBD.

In two meta-analyses of over 38,000 subjects, another IL23R variant correlated with ulcerative colitis. People with the “G” allele at [rs7517847](#) had significantly lower UC rates. This allele was also protective against Crohn’s disease in a meta-analysis of 60 trials and 50K subjects [\[R, R, R\]](#).

When it comes to Asian populations [\[R, R, R, R\]](#):

- rs11209026 is protective against CD but not against UC (non-existent in East Asia)
- rs10889677 is weakly associated with higher rates of UC but not CD
- rs7517847 is associated with neither CD nor UC



Predisposed to higher IL23R activity based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
IL23R	rs11209026	GG
IL23R	rs7517847	TT
IL23R	rs10889677	CA

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

NLRP3 (Gut Inflammation)

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

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

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

- [rs3806265](#)-C
- [rs4925648](#)-T
- [rs3738447](#)-G

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



HIGHER ACTIVITY

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

Your top variants that most likely impact your genetic predisposition:

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

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

HNF4A (Gut Inflammation)

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

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

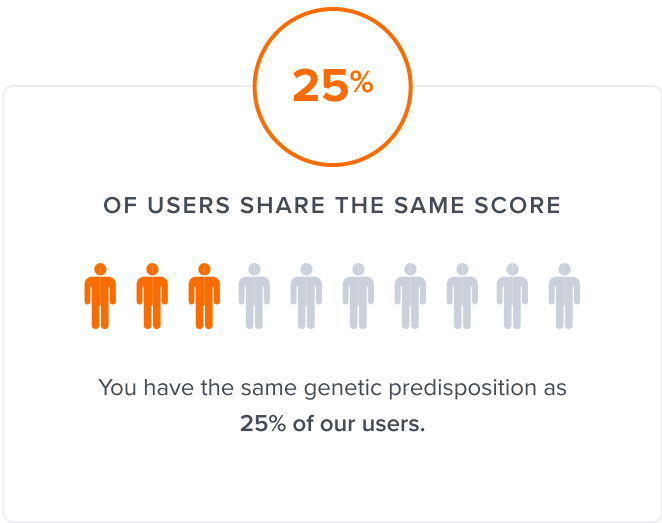
Likewise, two more studies of over 2,000 Dutch and 1,900 Korean patients also found that those carrying a ‘C’ allele were significantly more likely to have UC [R, R].

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



LOWER ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HNF4A	rs6017342	CC

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

FUT2 (Gut/ Vitamin B12)

The ‘AA’ (non-secretor) genotype of [rs601338](#) has been associated with higher [vitamin B12](#) absorption in comparison to secretor genotypes in European and West African populations. In European populations, this variant has been associated with a greater risk of developing celiac disease but may protect against the development of Crohn’s disease and ulcerative colitis [\[R\]](#).

The same goes for [rs602662](#)-A and [rs492602](#)-G, which are almost always inherited with rs601338-A [\[R\]](#), [\[R\]](#).

Non-secretors (AA) lack histo-blood group antigens in the gut lining, preventing certain pathogens like norovirus from attaching and causing disease. However, this also limits beneficial bacteria (e.g., bifidobacteria), potentially reducing their immune-supporting and protective effects against harmful species [\[R\]](#), [\[R\]](#).

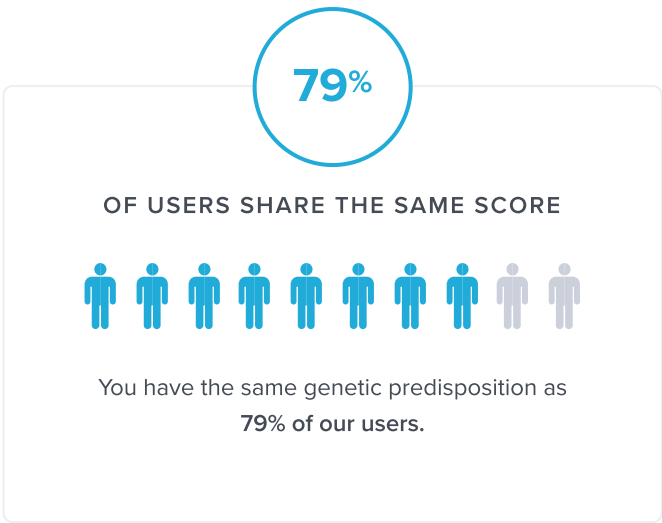
The minor ‘T’ allele of another variant, [rs1047781](#), has been associated with higher vitamin B12 absorption but a greater risk of developing Crohn’s disease. This allele has also been associated with a reduced amount and diversity of gut bacteria [\[R\]](#), [\[R\]](#), [\[R\]](#).

The rs1047781 is closely associated with rs601338 in all individuals but those of East Asian (Japanese) descent. In individuals of Japanese descent, only rs1047781 determines secretor status. People with the ‘TT’ genotype are non-secretors [\[R\]](#).

FUT2 status is significant for **methylation** because FUT2 influences B12 absorption and availability, which is crucial for the methylation cycle. "Secretors" may have reduced B12 absorption, which can impact the methylation cycle since B12 is required as a cofactor for methionine synthase, a key enzyme in the methylation pathway that helps convert homocysteine to methionine.



Likely secretor based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
FUT2	rs601338	AG
FUT2	rs1047781	AA

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

TLR4 (Inflammation)

The most well-researched TLR4 variant is [rs4986791](#). Its minor ‘T’ allele is believed to increase TLR4 activation based on its pro-inflammatory effects [\[R\]](#).

This variant has been associated with an increased risk of:

- Crohn’s disease and severe childhood [IBD](#) [\[R\]](#), [\[R\]](#)
- Rheumatoid arthritis [\[R\]](#)
- Alzheimer’s disease [\[R\]](#)
- Septic shock [\[R\]](#)
- Asthma [\[R\]](#)
- Tuberculosis [\[R\]](#)
- Gum disease [\[R\]](#)
- Glaucoma [\[R\]](#)
- Cancer [\[R\]](#), [\[R\]](#)

However, this variant has also been linked to:

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

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

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

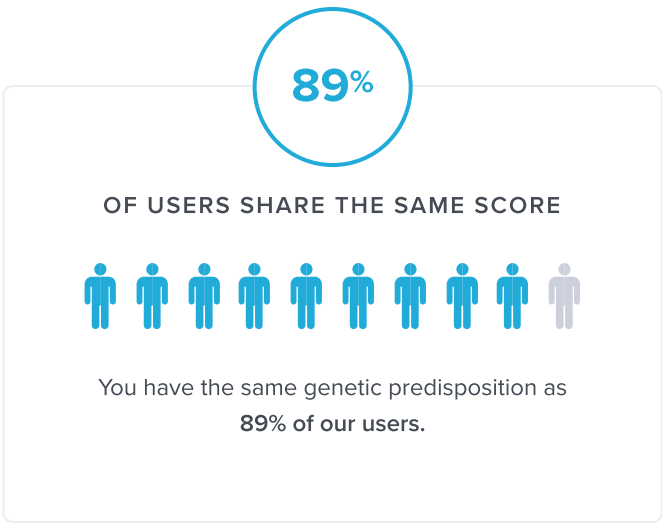
However, this has also been associated with a reduced acne severity [\[R\]](#).

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



TYPICAL ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TLR4	rs4986791	CC
TLR4	rs4986790	AA

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

TNFSF15 (Gut Inflammation)

Four *TNFSF15* polymorphisms have been most widely studied:

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

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

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

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

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

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

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

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



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

Your top variants that most likely impact your genetic predisposition:

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

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

MUC1 (Immunity, Gut, Minerals)

The main MUC1 variant is [rs4072037](#). Its “C” allele is linked to [\[R, R\]](#):

- Stomach cancer
- Lung conditions
- IBD
- Weaker bones and lower calcium levels
- Lower magnesium levels

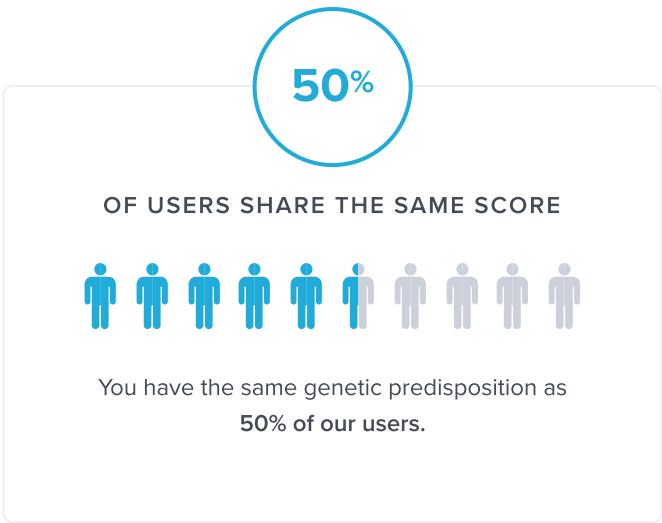
Scientists are unsure about the mechanisms behind these associations. They may reflect the protective role of MUC1 in mucous membranes, but on the other hand, some studies also linked higher MUC1 activity in cancer tissue [\[R\]](#).

Its link to weaker bones stems from calcium and magnesium loss. On the bright side, the variant is also linked to **lower uric acid levels** and thus lower odds of gout [\[R, R\]](#).



TYPICAL GENETICS

Likely typical MUC1 genetics based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
THBS3	rs4072037	TC

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

SLC2A14 (Gut Inflammation)

A study of 388 participants identified the following SLC2A14 variants as increasing the risk of both Crohn’s disease and ulcerative colitis by 2- to 5-fold [R]:

- ‘A’ of [rs2889504](#)
- ‘G’ of [rs10846086](#)
- ‘T’ of [rs12815313](#)

The authors speculated that vitamin C imbalances caused by these variants may increase oxidative damage to the gut lining and reduce its barrier function.



TYPICAL GENETICS

Likely typical SLC2A14 genetics based on 3 genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SLC2A14	rs2889504	CC
SLC2A14	rs12815313	CC
SLC2A3	rs10846086	AA

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

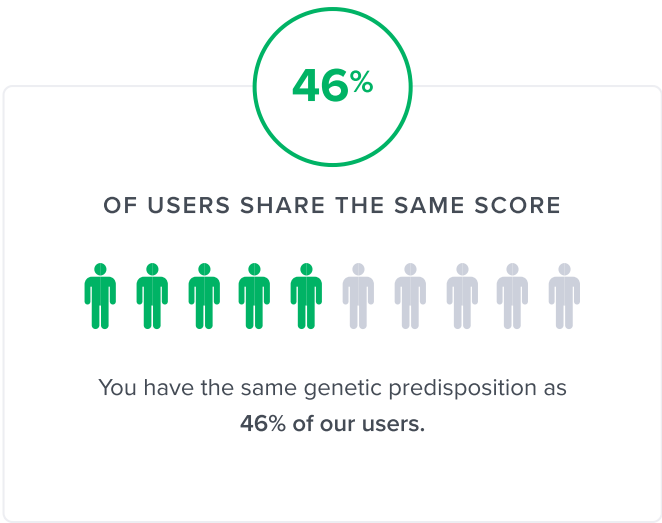
MRGPRX2 (Gut Inflammation)

The minor ‘C’ allele of [rs10833049](#) (N62S) encodes a version of the protein that cannot be activated by triggers such as the neuropeptide substance P or the antineoplastic agent daunorubicin hydrochloride, leading to impaired mast cell activation. In line with this, this variant has been associated with a decreased risk of ulcerative colitis [\[R, R\]](#).



LOWER ACTIVITY

Predisposed to lower MRGPRX2 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MRGPRX2	rs10833049	CC

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

JAK2 (Gut Inflammation)

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

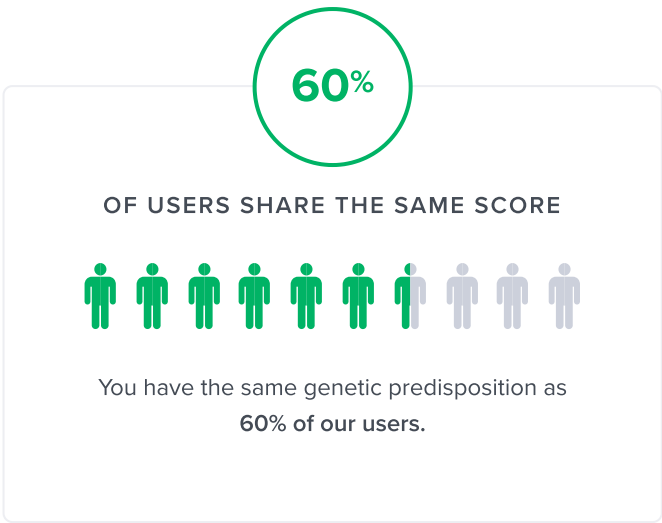
As a result, it’s linked to higher odds of [IBD — both Crohn’s disease and ulcerative colitis](#). The link may be stronger for Crohn’s disease, especially in people of European ancestry [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

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



LOWER ACTIVITY

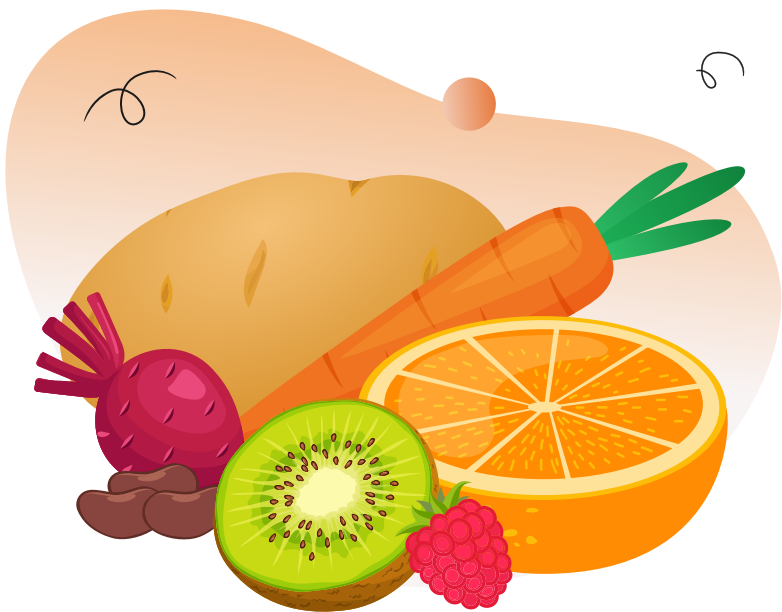
Likely lower JAK2 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
JAK2	rs10758669	CA
JAK2	rs12340895	CC

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



Vitamins & Minerals

Vitamins and minerals are essential for numerous bodily functions, including immune support, energy production, and bone health. Your body’s ability to absorb, process, and utilize these nutrients can be influenced by genetic factors. **Deficiencies or imbalances in key vitamins and minerals can impact your health and overall well-being.**

Genes like *TMPRSS6* and *TRPM6* are involved in iron metabolism, influencing iron absorption and storage. *VDR* and *CYP2R1* regulate vitamin D levels, which are crucial for bone health and inflammation control. *BCMO1* plays a role in converting carotenoids to vitamin A, while *CUBN* and *TCN2* are involved in vitamin B12 and folate absorption. Understanding these genetic variants can help identify potential nutrient deficiencies, allowing you to make targeted dietary or supplementation choices to support overall wellness.

HIGHER ACTIVITY TFR2 (Iron) Predisposed to a higher TFR2 activity	LOWER ACTIVITY BCO1 (Vitamin A) Predisposed to a lower BCO1 activity	LOWER ACTIVITY TCN2 (Vitamin B12) Likely lower TCN2 activity
TYPICAL ACTIVITY TMPRSS6 (Iron) Predisposed to typical TMPRSS6 activity	TYPICAL ACTIVITY TRPM6 (Magnesium & Calcium) Predisposed to typical TRPM6 activity	TYPICAL ACTIVITY TF (Iron & Cognition) Predisposed to typical TF activity
TYPICAL ACTIVITY SLC23A1 (Vitamin C) Predisposed to typical SLC23A1 activity	TYPICAL ACTIVITY VDR (Vitamin D) Likely typical VDR activity	TYPICAL ACTIVITY CYP24A1 (Vitamin D) Likely typical CYP24A1 activity
TYPICAL ACTIVITY PDXK (Vitamin B6) Likely typical PDXK activity	HIGHER ACTIVITY GC (Vitamin D) Likely higher GC activity	HIGHER ACTIVITY CUBN (Vitamin B12) Likely higher CUBN activity

TFR2 (Iron)

The main *TFR2* polymorphism is [rs7385804](#). Its ‘C’ allele has been associated with higher soluble transferrin receptor levels and a lower iron status, resulting in [\[R, R, R, R, R, R, R, R, R\]](#):

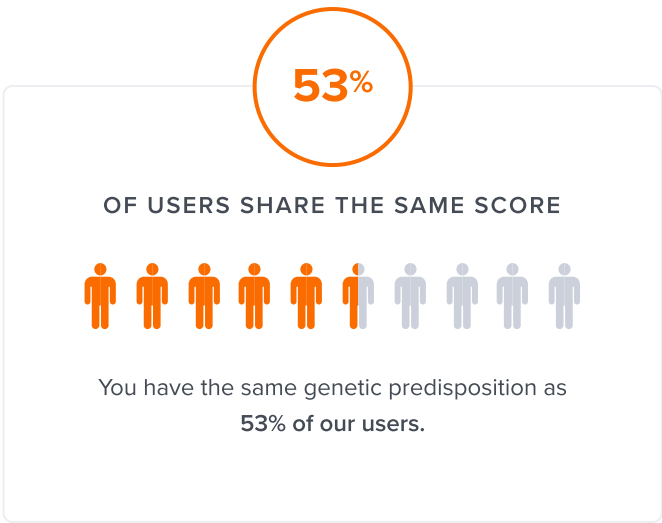
- Lower blood iron levels
- Lower serum transferrin levels
- Lower transferrin saturation measurement
- Lower hematocrit
- Lower MCV
- Lower MCHC
- Higher red blood cell count

However, a study failed to associate this variant with iron-deficiency anemia. Interestingly, a study of 680 pregnant women found that supplementation with folic acid plus iron increased neonatal birth weight more if the woman had the ‘AA’ genotype [\[R, R\]](#).



HIGHER ACTIVITY

Predisposed to a higher TFR2 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MOSPD3	rs7385804	AC

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

BCO1 (Vitamin A)

The main *BCO1* gene variant is [rs7501331](#). Its minor ‘T’ allele encodes a protein with a 32-57% reduced ability to convert beta-carotene to retinol [\[R\]](#), [\[R\]](#).

In contrast, this allele has been associated with lower beta-carotene levels in the prostate, suggesting increased conversion [\[R\]](#).

This variant has also been associated with higher lutein levels in the retina (higher macular pigment optical density) of adults, but lower levels in children [\[R\]](#), [\[R\]](#).

Another relatively well-characterized variant is [rs6564851](#). Its major ‘G’ allele may reduce BCO1 activity by 48%, resulting in higher blood beta-carotene levels [\[R\]](#), [\[R\]](#), [\[R\]](#).

This variant has also been associated with lower levels of the carotenoids lutein and zeaxanthin in the blood, as well as with a lower increase of blood lycopene levels in response to tomato or watermelon juice. In contrast, consuming dietary beta-carotene may increase its blood levels more in people with the ‘GG’ genotype [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

The ‘G’ allele of [rs6420424](#) encodes a BCO1 protein with 59% lower activity, resulting in higher blood beta-carotene levels [\[R\]](#).

This variant has also been associated with higher levels of the carotenoids lutein and zeaxanthin in the blood but lower levels in the retina [\[R\]](#), [\[R\]](#).

Finally, the ‘G’ allele of [rs11645428](#) may also reduce the conversion of beta-carotene to retinol, specifically by 51%. This variant has also been associated with lower lutein levels in the retina in some, but not all, studies [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).



Predisposed to a lower BCO1 activity based on 4 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
BCO1	rs6420424	GG
C16ORF46	rs11645428	GG
BCO1	rs7501331	CT
C16ORF46	rs6564851	TT

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

TCN2 (Vitamin B12)

The main TCN2 variant is [rs1801198](#) or **C776G**. According to a large review of 34 studies, the “GG” genotype is linked to [\[R\]](#):

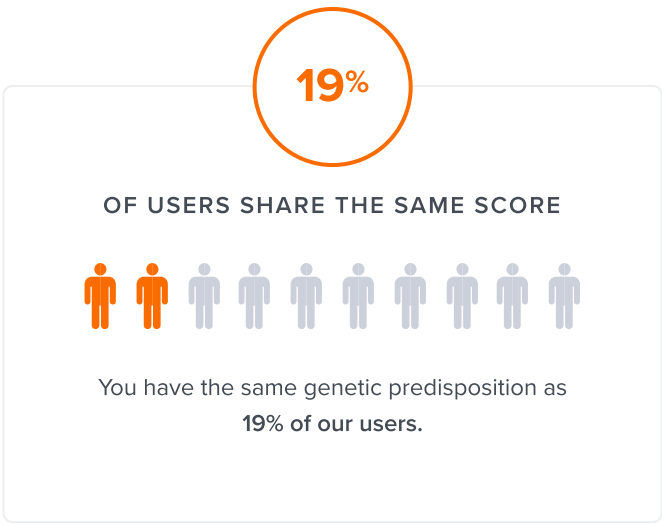
- Lower levels of active vitamin B12, holotranscobalamin
- Higher homocysteine levels (in European descendants)

On the other hand, the review found **no link** between this variant and health conditions like birth defects, cancer, or Alzheimer’s disease.

The “G” allele most likely reduces TCN2 activity, leading to impaired vitamin B12 uptake into tissues [\[R\]](#).



Likely lower TCN2 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TCN2	rs1801198	GG

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

TMPRSS6 (Iron)

At least three *TMPRSS6* variants have been associated with blood iron levels and incidence of iron deficiency: [rs4820268](#), [rs855791](#), and [rs228916](#) [R, R].

At rs4820268, the less common ‘G’ allele is associated with lower blood iron levels and possible iron deficiency. Similarly, the less common ‘A’ allele at rs855791 is associated with relatively lower blood iron levels in the majority of studies, though its effect may vary with population [R, R, R].

At rs228916, the rare ‘C’ allele is associated with slightly higher blood iron than the common ‘T’ allele. Here, the ‘C’ allele may be somewhat protective against iron deficiency, but more common in those prone to iron overload [R].



TYPICAL ACTIVITY

Predisposed to typical *TMPRSS6* activity based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TMPRSS6	rs4820268	GA
TMPRSS6	rs855791	AG
TMPRSS6	rs228916	TT

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

TRPM6 (Magnesium & Calcium)

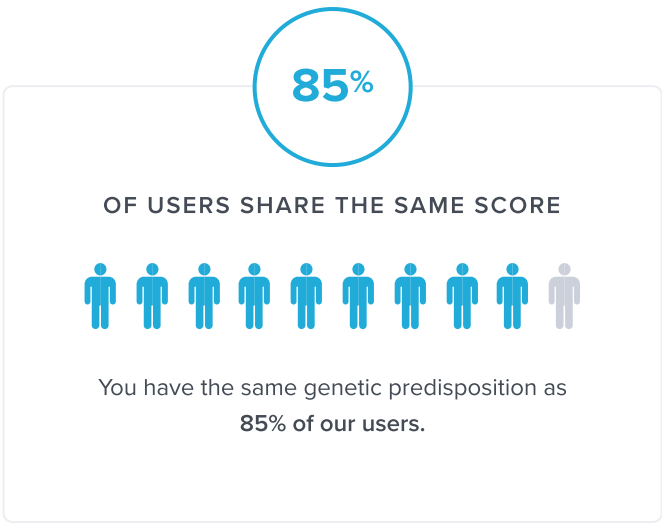
The main TRPM6 variant is [rs11144134](#). Its minor “**C**” **allele** is linked to **higher magnesium** levels. This variant may increase TRPM6 activity, boosting magnesium reabsorption in the kidneys [\[R\]](#).

However, this variant is also linked to **higher blood pressure, kidney stones, and weaker bones**. It may disrupt the balance of other minerals, more precisely **reduce calcium** and increase phosphate levels [\[R\]](#).



TYPICAL ACTIVITY

Predisposed to typical TRPM6 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TRPM6	rs11144134	TT

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

TF (Iron & Cognition)

The TF variant [rs1049296](#) (Pro570Ser; C1/C2) changes the transferrin protein. This small change can have significant effects on iron metabolism. The “**T**” **allele**, also called **C2**, may impair transferrin’s iron binding capacity. This may lead to either increased or decreased iron levels in different contexts [\[R\]](#).

Studies have linked this variant to [\[R\]](#), [\[R\]](#), [\[R\]](#):

- Lower hemoglobin and hematocrit levels
- Higher odds of Alzheimer’s disease

When transferrin function is impaired:

1. **Less efficient iron delivery** to bone marrow for hemoglobin synthesis, potentially leading to lower hemoglobin levels
2. Paradoxically, while overall iron transport is reduced, there can be inappropriate **iron accumulation in the brain** due to disrupted iron homeostasis and trafficking

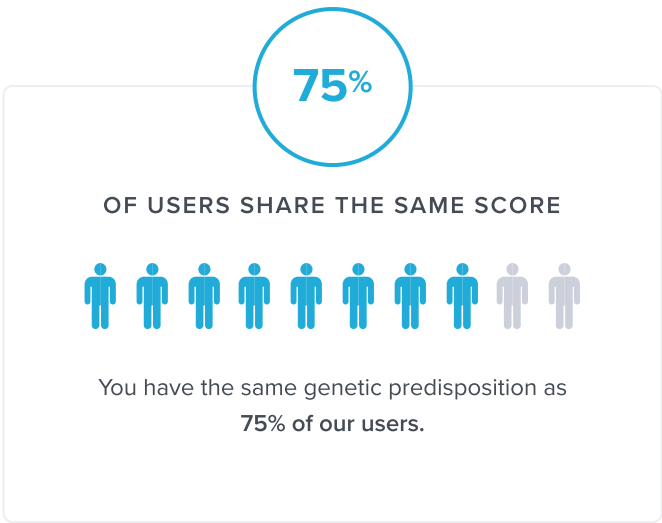
Think of it like a traffic system where the trucks (transferrin) aren't working properly—you get less delivery to the factory (bone marrow) and traffic jams in sensitive areas (brain).

Another TF variant, [rs3811647](#), may affect transferrin function, but its links to iron parameters are mixed and don’t point to a clear conclusion [\[R\]](#).



TYPICAL ACTIVITY

Predisposed to typical TF activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TF	rs1049296	CC

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

SLC23A1 (Vitamin C)

One variant in the [SLC23A1](#) gene, [rs33972313](#), is linked to reduced vitamin C levels. On average, the levels tend to be **5.98μmol/L lower per each “T” allele** [\[R, R\]](#).

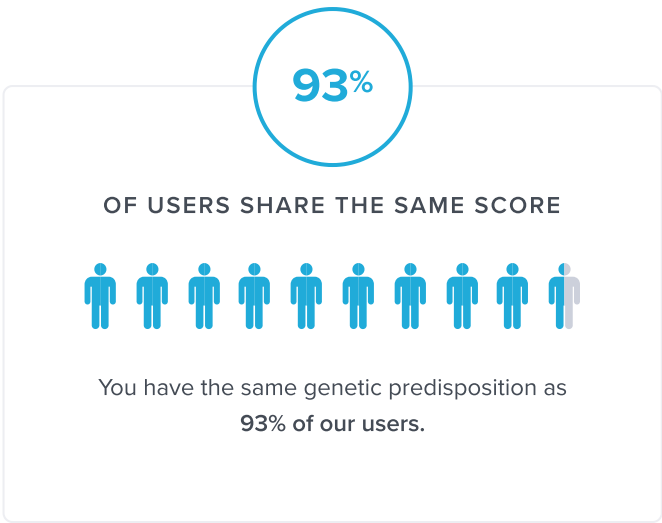
The variant may **reduce** SLC23A1 activity, impairing vitamin C absorption [\[R, R\]](#).

Researchers studied another SLC23A1 variant, [rs10063949](#), but the results are **mixed and inconclusive**. The “C” allele may be linked to higher vitamin C levels but also higher odds of Crohn’s disease. This variant showed no link with glaucoma or macular degeneration [\[R, R, R, R\]](#).



TYPICAL ACTIVITY

Predisposed to typical SLC23A1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SLC23A1	rs33972313	CC

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

VDR (Vitamin D)

Among the different *VDR* polymorphisms, [rs2228570](#), also known as FokI, has been most widely researched. Its minor ‘A’ variant encodes a slightly longer version of the protein associated with decreased VDR levels and activity. In line with this, the major ‘G’ variant has been associated with [\[R\]](#), [\[R\]](#):

- Better response to vitamin D supplementation
- Better calcium absorption
- Higher bone mineral density
- Reduced risk of fractures

The major variant has also been associated with:

- [Increased fat mass](#) [\[R\]](#), [\[R\]](#)
- [Increased longevity](#) (with mixed results) and better cognition in the elderly [\[R\]](#), [\[R\]](#), [\[R\]](#)

Another important *VDR* polymorphism is [rs1544410](#), also known as BsmI. Its minor ‘T’ allele may have decreased VDR activity and has been associated with:

- Lower BMD and increased risk of osteoporosis [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Increased risk of scoliosis [\[R\]](#)
- Decreased risk of breast cancer [\[R\]](#)
- Decreased risk of metabolic syndrome [\[R\]](#)

Other *VDR* variants include [rs7975232](#) (ApaI) and [rs731236](#) (TaqI).

Potential effects of *VDR* variants on dopamine:

- Reduced VDR activity can lead to lower dopamine production because vitamin D helps activate genes that make dopamine
- This may affect dopamine-related functions like mood, focus, and motivation
- The body might have trouble maintaining stable dopamine levels

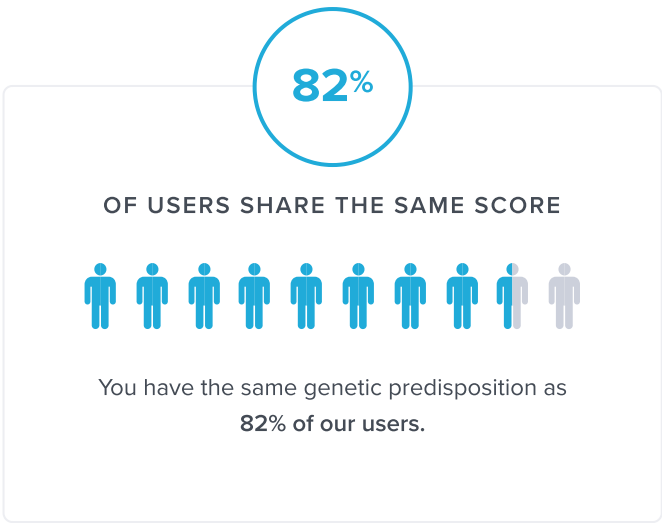
Potential effects of *VDR* variants on methylation:

- When VDR activity is reduced, it can affect methylation cycles
- This might impact how well your body processes certain chemicals and nutrients



TYPICAL ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
VDR	rs2228570	GA
VDR	rs1544410	CT
VDR	rs7975232	AA
VDR	rs731236	AG

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

- The methylation of genes involved in dopamine production and function may be affected

CYP24A1 (Vitamin D)

The best-characterized *CYP24A1* polymorphism is [rs6013897](#). Its minor ‘A’ allele has been associated with lower vitamin D levels and reduced effectiveness of vitamin D supplementation, suggesting increased CYP24A1 activity. This variant has also been associated with [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#):

- Lower BMD [\[R\]](#)
- Higher fasting glucose in pregnant women [\[R\]](#)
- Increased risk of colorectal cancer [\[R\]](#)
- Increased risk of breast cancer [\[R\]](#)
- Increased risk of attempted suicide [\[R\]](#)
- Increased risk of schizophrenia [\[R\]](#)
- Increased risk of cirrhosis and liver cancer [\[R\]](#)
- Decreased risk of aggressive prostate cancer [\[R\]](#), [\[R\]](#)
- Decreased risk of tuberculosis [\[R\]](#)
- Better survival in glioma patients [\[R\]](#)

Another well-researched polymorphism is [rs2248359](#). Its major ‘C’ allele may increase *CYP24A1* expression in the prefrontal cortex, leading to lower baseline vitamin D levels. This allele has also been associated with [\[R\]](#), [\[R\]](#), [\[R\]](#):

- Increased risk of multiple sclerosis [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Increased risk of eczema [\[R\]](#)
- Higher leucocyte counts [\[R\]](#)
- Decreased risk of type 2 diabetes [\[R\]](#)
- Decreased risk of NAFLD [\[R\]](#)
- Decreased risk of CKD in people with hypertension [\[R\]](#)
- Decreased risk of chronic hepatitis C infection [\[R\]](#)

Finally, the major ‘A’ allele of [rs4809957](#) has been associated with lower vitamin D levels, as well as with [\[R\]](#):

- Increased risk of breast cancer [\[R\]](#), [\[R\]](#)
- Increased risk of pancreatic cancer [\[R\]](#)
- Increased risk of colorectal cancer [\[R\]](#)
- Increased risk of ulcerative colitis [\[R\]](#)
- Increased risk of lung cancer and worse survival [\[R\]](#), [\[R\]](#)
- Increased risk of NSAID-induced gastrointestinal bleeding [\[R\]](#), [\[R\]](#)
- Decreased risk of vitamin D deficiency in type 2 diabetes patients [\[R\]](#)



Likely typical CYP24A1 activity based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
BCAS1	rs6013897	TA
CYP24A1	rs2248359	CT
CYP24A1	rs4809957	AA

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

PDXK (Vitamin B6)

One rare variant close to the [PDXK](#) gene, [rs147242481-A](#), is linked to higher homocysteine levels. This variant is also close to the [CSTB](#) gene, so the mechanism behind this association is not entirely clear [\[R\]](#).

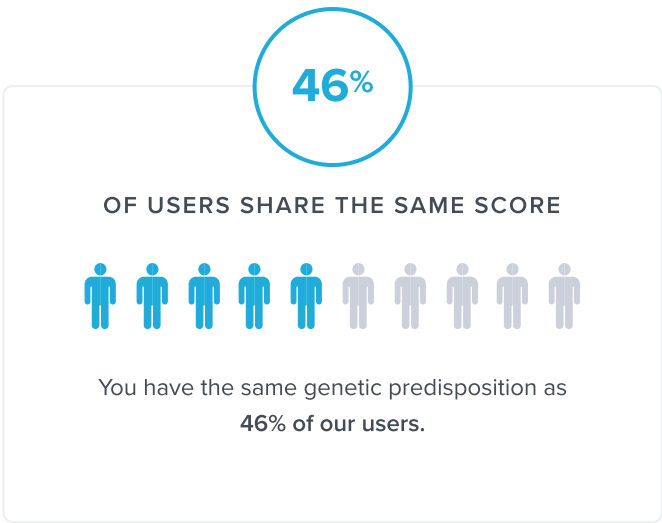
This variant likely reduces PDXK activity, leading to lower PLP levels. This may compromise key enzymes involved in homocysteine clearance: SHMT and CBS.

Another PDXK variant, [rs2010795-A](#), may be linked to higher odds of Parkinson’s disease. PLP supports the production of dopamine, which is depleted in people with Parkinson’s disease. However, two smaller studies didn’t find this association [\[R, R, R\]](#).



TYPICAL ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PDXK	rs2010795	GA
PDXK	rs147242481	GG

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

GC (Vitamin D)

In a meta-analysis of 15 clinical trials and over 30,000 European descendants, [rs2282679](#) showed the strongest association with vitamin D status. The minor “G” allele correlated with 63% and 49% higher odds of vitamin D insufficiency and deficiency, respectively [\[R\]](#).

The link of this variant with vitamin D deficiency may be independent of age, weight, and other risk factors [\[R\]](#).

This variant may also influence the outcomes of vitamin D supplementation during pregnancy; in 682 white women, each “T” allele correlated with 4.2 nmol/L higher vitamin D levels upon delivery [\[R\]](#).

Two studies of nearly 4,000 participants associated the ‘G’ allele with lower DBP levels [\[R\]](#), [\[R\]](#).

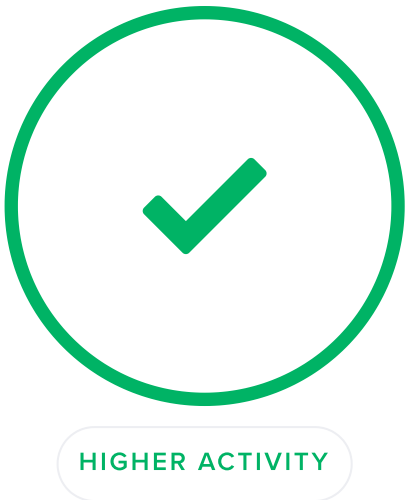
Another SNP, [rs4588](#), showed comparable effects due to a nearly perfect overlap with rs2282679, so it wasn’t an independent genetic factor [\[R\]](#).

Another SNP with a meaningful impact on vitamin D status is [rs7041](#). The above meta-analysis and another review of 17 studies confirmed the association between the “A” allele and lower vitamin D levels [\[R\]](#), [\[R\]](#).

This variant may reduce the efficacy of vitamin D supplementation [\[R\]](#), [\[R\]](#).

Other GC variants have shown links with vitamin D levels, but the supporting evidence is weak:

- [rs17467825](#)
- [rs1155563](#)
- [rs222020](#)
- [rs2298849](#)



Likely higher GC activity based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GC	rs2282679	GT
GC	rs7041	AC
GC	rs4588	TG

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

CUBN (Vitamin B12)

The main CUBN gene variant is [rs1801222](#). This variant seems to reduce CUBN activity, affecting vitamin B12 absorption.

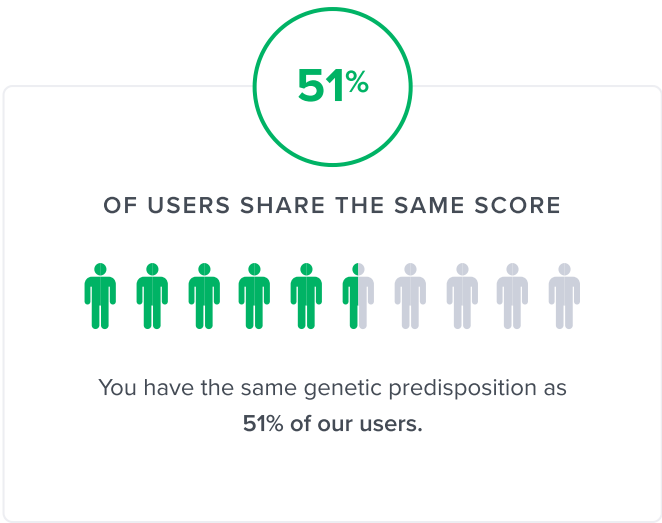
Its “A” allele is linked to [\[R, R, R, R, R\]](#):

- Lower vitamin B12 levels
- Increased homocysteine levels
- Pernicious anemia (B12 deficiency anemia)
- Megaloblastic (large cell) anemia



HIGHER ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CUBN	rs1801222	GG

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



 Detox

Detoxification is a vital process that helps the body eliminate harmful substances, maintain balance, and support overall health. Genetic variations in detoxification genes can influence how effectively your body processes and clears toxins, impacting your risk for conditions like neurodegenerative diseases, liver damage, and even cancer.

Genes like *CYP1A1*, *CYP1B1*, *CYP2A6*, *CYP2C19*, *CYP2D6*, and *CYP3A4* are involved in the metabolism of environmental toxins, drugs, and other chemicals, affecting how your body responds to these substances. Understanding how these genetic variations affect your detox pathways can guide lifestyle and dietary adjustments to optimize your detoxification processes and reduce the impact of toxins on your health.

 HIGHER ACTIVITY CYP1B1 (Detox/ Skin Health) Predisposed to higher CYP1B1 activity	 LOWER ACTIVITY PON1 (Detox) Likely lower PON1 activity	 LOWER ACTIVITY CYP2D6 (Mental Health, Detox) Predisposed to a lower CYP2D6 activity
 TYPICAL ACTIVITY CYP1A1 (Detox) Likely typical CYP1A1 activity	 TYPICAL ACTIVITY CYP2A6 (Detox, Nicotine) Likely typical CYP2A6 activity	 EXTENSIVE METABOLIZER CYP2C19 (Detox) Likely an extensive metabolizer
 TYPICAL ACTIVITY CYP3A4 (Detox) Likely typical CYP3A4 activity	 TYPICAL ACTIVITY CYP3A5 (Detox/Blood Pressure) Predisposed to a typical CYP3A5 activity	 HIGHER ACTIVITY SULT1A1 (Detox) Likely higher SULT1A1 activity
 INTERMEDIATE ACETYLATOR NAT1 (Detox) Likely an intermediate acetylator	 HIGHER ACTIVITY UGT (Detox) Likely higher UGT activity	 INTERMEDIATE NAT2 (Detox) Likely an intermediate acetylator

CYP1B1 (Detox/ Skin Health)

The main *CYP1B1* variant is [rs1056836](#) (Leu432Val). The **G (Val) allele** seems to **increase** the enzyme's activity. This may lead to increased production of toxic metabolites of hormones and toxins.

In line with this, studies have found a link between this allele and higher odds of multiple myeloma and lung cancer [\[R, R\]](#).

On the other hand, increased CYP1B1 activity may protect the skin against excessive UV radiation. The high-activity G allele may be linked to lower odds of skin cancer [\[R\]](#).

Other important *CYP1B1* variants include:

- [rs1800440](#): The “C” allele may increase enzyme activity. It’s linked to lower odds of skin cancer but also lower vitamin D levels and higher odds of asthma [\[R\]](#)
- [rs10012](#): The “C” allele may increase enzyme activity. It’s linked to lower odds of skin cancer but higher odds of bladder cancer [\[R, R\]](#)



HIGHER ACTIVITY

Predisposed to higher CYP1B1 activity based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CYP1B1	rs1056836	GG
CYP1B1	rs1800440	CT
CYP1B1	rs10012	GC

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

PON1 (Detox)

The [rs662](#) variant influences how active the PON1 enzyme is. The risk allele “T” reduces the activity of the protein, resulting in less breakdown (and more potential buildup) of toxins [\[R\]](#).

Another *PON1* variant, [rs854560](#), controls the levels of this protein in the blood. Carriers of the risk allele “A” have lower blood levels of PON1. This means they have a reduced ability to detox [\[R\]](#).

In line with this, a large analysis of 9 studies associated both variants with an [increased risk of organophosphate toxicity](#). While carriers of the rs662 variant had 74% higher risk of organophosphate toxicity, the rs854560 increased the risk by 82%. In both cases, the risk was even higher in Caucasians [\[R\]](#).

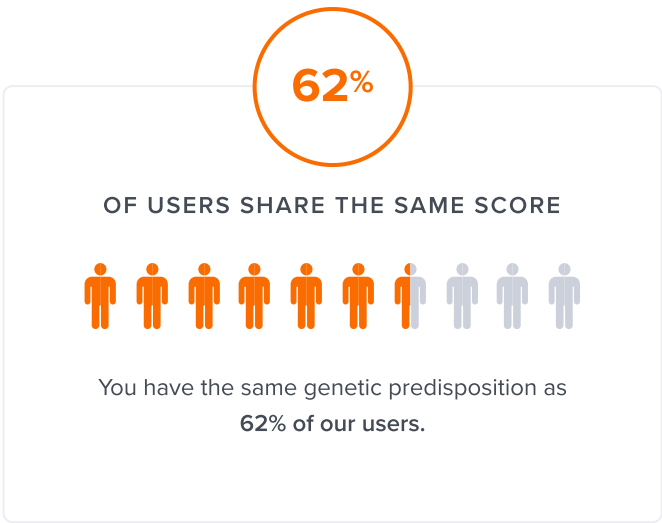
Possibly due to their decreased breakdown of oxidized lipids, harmful bacteria, and organophosphate pesticides, these variants have also been associated with:

- [Increased risk of heart disease](#)
- [Decreased longevity](#)



LOWER ACTIVITY

Likely lower PON1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PON1	rs662	TT
PON1	rs854560	AT

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

CYP2D6 (Mental Health, Detox)

The *CYP2D6* gene has more than 100 known variants. These include [\[R, R\]](#):

- Normal enzyme function variants (e.g., *1 and *2) [\[R, R\]](#)
- Reduced function variants (e.g., *9, *10, *17, and *41) [\[R, R\]](#)
- Non-functional variants (e.g., *3, *4, *5, *6, and *7) [\[R, R\]](#)

It gets more complicated, however, because this gene can also be missing or duplicated. This means that you can have 0, 1, 2, or more than 2 copies of the gene. Therefore, most clinical laboratories also report the copy number [\[R\]](#).

*CYP2D6**1 is the wild-type variant. The change from an ‘A’ to a ‘G’ at [rs16947](#) encodes *CYP2D6**2. Although its activity is indistinguishable from that of *CYP2D6**1, having several copies of this variant can result in an ultrafast phenotype. This variant has been associated with tyramine intolerance, suggesting a slightly lower metabolic efficiency for this substrate [\[R, R\]](#).

The ‘T’ allele of [rs3892097](#) corresponds to *CYP2D6**4. This is the most frequent non-functional variant in Europeans and North Americans (18.0%), accounting for 70-90% of cases [\[R\]](#).

Another non-functional variant is *CYP2D6**6, consisting of the deletion of an ‘A’ at [rs5030655](#). This variant is generally rare, but may be slightly more common in Europeans (around 1%) [\[R, R, R\]](#).

Finally, the ‘G’ allele of [rs5030867](#) encodes the non-functional *CYP2D6**7 variant. This allele is very rare but may be slightly more common in South East Asians (around 1%) [\[R\]](#).

The ‘A’ allele of [rs1065852](#) encodes the intermediate-activity *CYP2D6**10 variant. This variant is especially common in Thai (50%) and East Asians (42%) [\[R, R\]](#).

Another intermediate-activity variant is *CYP2D6**17, encoded by the ‘A’ allele of [rs28371706](#). This variant is most common in Africans (20%-35%) [\[R, R\]](#).

Studies suggest that people with low CYP2D6 enzyme activity may be more prone to anxiety and less successful at socializing



Predisposed to a lower CYP2D6 activity based on 5 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CYP2D6	rs5030867	TT
CYP2D6	rs5030655	CC
CYP2D6	rs3892097	CC
CYP2D6	rs28371706	GG
CYP2D6	rs1065852	GG
CYP2D6	rs16947	GA

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

than extensive metabolizers. Reduced activity has also been associated with impulsivity or novelty seeking [\[R\]](#), [\[R\]](#), [\[R\]](#).

Alternatively, poor metabolizers may perform better in cognitive tasks that demand sustained attention or vigilance and spatial working memory. They may also have higher conscientiousness, responsibility, orderliness, and perseverance [\[R\]](#), [\[R\]](#), [\[R\]](#).

Low-activity variants have been associated with an increased risk of developing the following conditions:

- Alzheimer’s disease [\[R\]](#)
- Parkinson’s disease [\[R\]](#)
- Systemic sclerosis [\[R\]](#), [\[R\]](#)
- Lupus [\[R\]](#)
- Ankylosing spondylitis [\[R\]](#)
- Pesticide toxicity [\[R\]](#)
- Adverse effects from antidepressants [\[R\]](#)

In contrast, they have been linked to a reduced risk of:

- Schizophrenia [\[R\]](#)
- Bulimia [\[R\]](#)
- Heavy smoking [\[R\]](#)
- Adverse effects from codeine and tramadol [\[R\]](#)

CYP1A1 (Detox)

The ‘G’ allele of [rs4646903](#), linked to higher CYP1A1 levels, has been associated with an increased risk of leukemia, cervical, hepatocellular, lung, prostate, colorectal, breast and head and neck cancers, possibly by turning PAHs into cancer-causing chemicals. This variant may also increase the risk of PCOS, recurrent pregnancy loss, and male infertility through its role in estrogen metabolism [\[R, R, R, R, R, R, R, R, R\]](#).

Similarly, the ‘C’ variant of [rs1048943](#) increases CYP1A1 activity and has been associated with an increased risk of lung cancer, laryngeal cancer, oral cancer, colorectal cancer, cervical cancer, hepatocellular carcinoma, prostate cancer, and leukemia. In the case of lung cancer, the risk may be even higher in smokers carrying this variant [\[R, R, R, R, R, R, R, R, R, R\]](#).

The ‘A’ allele of [rs2606345](#) may decrease CYP1A1 levels. This variant has been associated with an increased risk of lung conditions such as pneumonia or ARDS, possibly by increasing inflammation and oxidative stress in the lungs. In contrast, the ‘C’ allele has been associated with esophageal and testicular cancer [\[R, R, R, R, R, R, R, R\]](#).

The ‘T’ allele of [rs2472297](#) may increase CYP1A1 activity. This variant has been associated with greater coffee consumption, as well as with a decreased increment in blood glucose after consuming caffeine [\[R, R, R, R\]](#).

Finally, the ‘A’ allele of [rs4646421](#) has been associated with hepatocellular carcinoma, endometrial cancer (especially in women with abdominal obesity), esophageal cancer (in hot tea drinkers), laryngeal cancer, decreased lung function in elderly people exposed to PAHs, and chronic hepatitis B infection [\[R, R, R, R, R, R\]](#).



Likely typical CYP1A1 activity based on 4 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CSK	rs2606345	AC
ULK3	rs2472297	CC
CYP1A1	rs4646903	AA
CYP1A1	rs1048943	TT
ARID3B	rs4646421	GG

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

CYP2A6 (Detox, Nicotine)

The *CYP2A6* gene is highly polymorphic, with more than 40 known alleles affecting its metabolic activity compared to the wild-type *CYP2A6*1A* allele [R].

In general, variants with reduced activity (slow metabolism) are associated with reduced smoking behaviors. For instance, a variant consisting of the total absence of the protein (*CYP2A6*4*) has been associated with decreased odds of being a smoker, reduced number of cigarettes smoked, decreased nicotine dependence, higher success of smoking cessation, and reduced risk of lung cancer and emphysema [R, R, R, R, R, R, R, R].

The most well-known *CYP2A6* SNP is [rs1801272](#), commonly referred to as *CYP2A6*2* in the literature. Its ‘T’ allele encodes an enzyme with reduced activity and has been associated with [R, R]:

- Decreased odds of being a smoker [R, R]
- Lower number of cigarettes smoked [R, R]
- Decreased risk of COPD [R]
- Decreased risk of lung cancer [R]

Other variants with decreased enzyme activity include:

- ‘A’ of [rs5031017](#) (*CYP2A6*5*): associated with fewer cigarettes smoked per day, smoking initiation at a later age, and shorter smoking time before quitting [R].
- ‘G’ of [rs5031016](#) (*CYP2A6*7*): associated with fewer cigarettes smoked per day, smoking initiation at a later age, and shorter smoking time before quitting [R].
- ‘C’ of [rs56113850](#) (*CYP2A6*9*): associated with reduced likelihood of being a smoker, fewer cigarettes smoked per day, smoking initiation at a later age, shorter smoking time before quitting, reduced nicotine dependence, and increased risk of lung cancer but lower smoking cessation rates [R, R, R, R, R, R, R].
- ‘C’ of [rs8102683](#) (*CYP2A6*12*): associated with fewer cigarettes smoked per day [R].

In contrast, variants with faster nicotine metabolism such as ‘A’ of [rs28399433](#) (*CYP2A6*1B*) have been associated with more cigarettes smoked per day and increased risk of smoking initiation [R, R].



TYPICAL ACTIVITY

Likely typical CYP2A6 activity based on 5 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CYP2A6	rs1801272	AA
/	rs5031016	AA
EGLN2	rs56113850	TT
C19ORF54	rs8102683	TT
EGLN2	rs28399433	AA

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

CYP2C19 (Detox)

There are more than 30 known *CYP2C19* variants. Based on the variants they carry, individuals can be categorized as [R](#), [R](#):

- Ultrarapid metabolizers (*1 /*17 or *17 /*17)
- Extensive metabolizers (*1/*1)
- Intermediate metabolizers (*1 /*2, *1 /*3, or *2 /*17)
- Poor metabolizers (*2 /*2 or *2 /*3)

The normal version of the gene is called CYP2C19*1.

The two most common polymorphisms associated with reduced CYP2C19 activity and clopidogrel resistance are CYP2C19*2 ([rs4244285](#)) and CYP2C19*3 ([rs4986893](#)). Carriers of one copy of the minor ‘A’ alleles have a reduced ability to break down drugs, including CBD. Those with two copies can metabolize very little or none of the drug and are classified as poor metabolizers [R](#), [R](#), [R](#).

The [rs12248560](#) polymorphism, also known as CYP2C19*17, is located in the region that controls gene expression (the *promoter*). Its minor allele ‘T’ is associated with increased gene expression and protein activity. People with this variant are classified as ultrarapid metabolizers [R](#), [R](#).

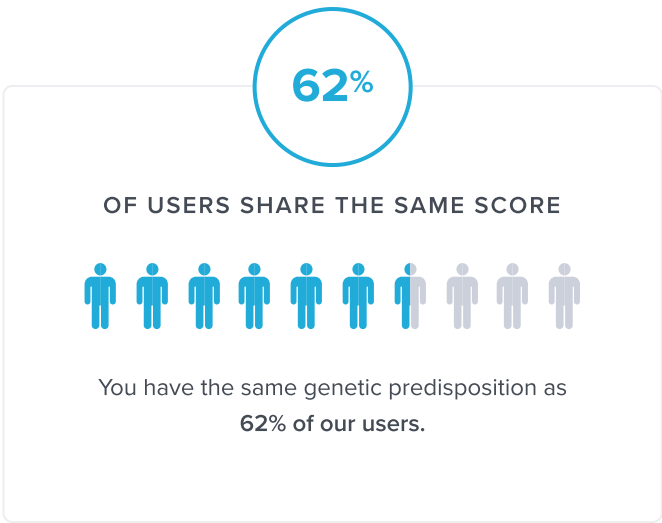
The following supplements and chemicals may decrease CYP2C19 activity [R](#), [R](#), [R](#), [R](#), [R](#), [R](#), [R](#), [R](#), [R](#), [R](#), [R](#):

- Propolis
- Caffeic acid
- Quercetin
- Ginger
- Kale
- African lettuce
- Saint John's wort
- Astaxanthin
- Canthaxanthin
- Hops
- Licorice
- Berberine
- Capsaicin
- Bulbocapnine, canadine, and protopine
- Fluconazole



EXTENSIVE METABOLIZER

Likely an extensive metabolizer based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NOC3L	rs12248560	CC
CYP2C19	rs4986893	GG
CYP2C19	rs4244285	GG

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

CYP3A4 (Detox)

The ‘A’ allele of [rs35599367](#), also known as CYP3A4*22, reduces CYP3A4 levels and activity by approximately half, resulting in slower drug metabolism [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

Similarly, the minor alleles ‘C’ of [rs12721627](#) (CYP3A4*16) and ‘G’ of [rs55785340](#) (CYP3A4*2) have been associated with lower enzyme activity [\[R\]](#), [\[R\]](#).

Two other variants with decreased enzyme activity are ‘C’ at [rs2740574](#) (CYP3A4*1B) and ‘T’ at [rs2242480](#) (CYP3A4*1G) [\[R\]](#), [\[R\]](#).

The minor ‘G’ allele of [rs680055](#) (CYP3A4*3) encodes a protein with an amino acid change believed to reduce enzyme activity [\[R\]](#).

In carriers of these variants, the breakdown of cannabinoids such as THC and CBD may be slower. Moreover, people with these variants shouldn’t use cannabis if they are being treated with ketoconazole, fluconazole, or diltiazem, which are CYP3A inhibitors [\[R\]](#), [\[R\]](#).

In contrast, the ‘G’ allele of [rs28371759](#) (CYP3A4*18) increases enzyme activity [\[R\]](#), [\[R\]](#).



TYPICAL ACTIVITY

Likely typical CYP3A4 activity based on 7 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CYP3A4	rs28371759	AA
CYP3A43	rs680055	CC
CYP3A4	rs55785340	AA
TRIM4	rs35599367	GG
TRIM4	rs2740574	TT
CYP3A5	rs2242480	CC
/	rs12721627	GG

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

CYP3A5 (Detox/Blood Pressure)

There are over 25 variants of this enzyme, but many of them are non-functional. The fully functional variant is known as the *CYP3A5*1*. Only individuals with at least one *CYP3A5*1* have large amounts of the enzyme. They metabolize some CYP3A substrates more rapidly than do people without this enzyme [R].

The frequency of the functional variant is substantially different across ethnic groups. The functional enzyme is found in 45–94% of subjects of African descent, 8–15% of Whites, and 23–40% of Asians [R, R].

The main non-functional variant is the ‘C’ allele of [rs776746](#), commonly designated as *CYP3A5*3*. Its frequency ranges from 14% among sub-Saharan Africans to over 95% in European populations [R, R].

This variant has been mainly studied regarding its ability to metabolize immunosuppressive drugs such as tacrolimus and sirolimus. Compared to the expresser *CYP3A5*1* allele, this variant has been associated with lower dose requirements and a decreased risk of kidney graft rejection (in Asian patients) [R, R, R, R, R, R, R, R, R, R, R].

Similarly, the ‘C’ allele has been associated with a better response to and lower required dose of:

- Anticoagulants [R]
- Antiseizure medications [R]
- Anticancer drugs [R]

The *CYP3A5*3* allele has also been associated with higher blood pressure in Whites, but not in Africans. However, this variant may protect against salt-sensitive hypertension. This may be because it reduces sodium reabsorption in the kidneys [R, R, R, R].

However, this variant has also been associated with an increased risk of the following cancer types:

- Leukemia [R, R]
- Prostate cancer [R]
- Colorectal cancer [R]



Predisposed to a typical CYP3A5 activity based on 2 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CYP3A5	rs10264272	CC
CYP3A5	rs41303343	TT
CYP3A5	rs776746	CC

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

The ‘T’ allele of [rs10264272](#) encodes another non-functional variant, commonly referred to as *CYP3A5**6. It is frequent in Africans (7-17%), but rare or absent in Europeans and Asians [\[R\]](#), [\[R\]](#).

This variant has been associated with higher levels of immunosuppressive drugs such as tacrolimus and cyclosporine due to slower clearance [\[R\]](#), [\[R\]](#), [\[R\]](#).

Finally, the ‘TA’ allele of [rs41303343](#) encodes another non-functional variant (*CYP3A5**7) that has also been associated with higher levels of immunosuppressive drugs. This variant is most common in Africans (12%) and usually absent in other ethnicities such as Europeans and Asians [\[R\]](#), [\[R\]](#), [\[R\]](#).

SULT1A1 (Detox)

A variant in this gene known as **SULT1A1*2** may reduce its activity. People with the “**T**” allele at [rs1042028](#) (previously named rs9282861) carry this variant [\[R\]](#).

As mentioned, **SULT1A1 is a double-edged sword when it comes to detox.**

In theory, **lower** SULT1A1 activity may increase the toxicity of some compounds present in smoke, like polycyclic aromatic hydrocarbons (PAHs). On the other hand, it should be protective against some other toxins, like heterocyclic amines (HAs) [\[R\]](#), [\[R\]](#).

In line with this, some studies have linked the lower-activity variant, **SULT1A1*2 ([rs1042028-T](#))**, to:

- Higher odds of stomach, lung, and colon cancers in smokers [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Higher odds of breast cancer in those who eat more smoked meat [\[R\]](#)

However, other studies have linked this variant to **lower odds** of prostate, bladder, colon, and oral cancers in smokers [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

Some studies failed to confirm any link between this variant and detox [\[R\]](#), [\[R\]](#).

Researchers have found other SULT1A1 variants that influence the activity of this gene [\[R\]](#), [\[R\]](#), [\[R\]](#):

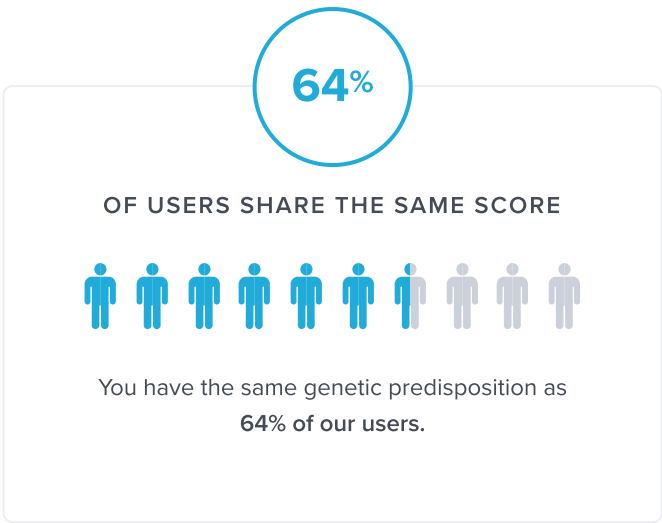
- [rs6839](#)
- [rs1042157](#)
- [rs1801030](#)

However, the impact of these variants is much lower compared with the main one, and their relevance for detox is unclear.



HIGHER ACTIVITY

Likely higher SULT1A1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SULT1A1	rs1042028	CC

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

NAT1 (Detox)

The ‘A’ allele of [rs4986782](#) is commonly referred to as *NAT1*14B* and encodes a slow-acetylator version of the protein. This variant has been associated with an increased risk of [\[R\]](#):

- Lung cancer in smokers [\[R\]](#)
- Bladder cancer, but lower in those exposed to combustion fumes [\[R\]](#)

A study identified two *NAT1* variants as decreasing *NAT1* gene expression and NAT1 activity: ‘A’ at [rs1057126](#) and ‘A’ at [rs15561](#). While the combination of both major variants encodes the *NAT1*4* allele, the minor allele of rs1057126 is referred to as *NAT1*3* and the combination of both minor variants produces the *NAT1*10* allele [\[R\]](#).

The *NAT1*3* allele has been associated with an increased risk of bladder cancer. In contrast, this variant has been associated with a decreased risk of colorectal cancer [\[R\]](#), [\[R\]](#).

The ‘A’ allele has been associated with an increased risk of non-invasive bladder cancer. However, carriers may benefit more from the preventive effects of dietary cruciferous vegetables. As was the case for the *NAT1*3* allele, this variant has been associated with an increased risk of colorectal cancer [\[R\]](#), [\[R\]](#).

Finally, the ‘G’ allele of [rs6586714](#) may increase NAT1 activity and has been associated with an increased risk of colorectal cancer in people with a high intake of cooked meat [\[R\]](#).



INTERMEDIATE ACETYLATOR

Likely an intermediate acetylator based on 4 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MTUS1	rs6586714	GG
ASAH1	rs1057126	AT
ASAH1	rs15561	AC
NAT1	rs4986782	GG

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

UGT (Detox)

Certain genetic variations in [UGT1A1](#) reduce the enzymatic activity of UGT, which [impairs the body’s ability to detox](#) and results in a buildup of toxic substances in the body. The normal version of *UGT1A1* (the one with normal detox activity) is called *UGT1A1*1*, or the “wild” type allele [\[R\]](#).

One of the most important and well-studied genetic variants is ***UGT1A1*28*** ([rs34983651](#)). This variant causes an “insertion” mutation, which means it adds an extra ‘TA’ into the gene where there should be none. According to some reports, ***UGT1A1*28* causes a 70% reduction in enzyme activity** [\[R\]](#).

Other variants include ***UGT1A1*6*** ([rs4148323](#)) and ***UGT1A1*27*** ([rs6742078](#)), which also lower enzyme activity, leading to a reduced ability to detox. All of these variations in *UGT1A1* can lead to toxicity disorders. The minor variants of *UGT1A1*27* and *UGT1A1*28* are usually inherited together [\[R\]](#).

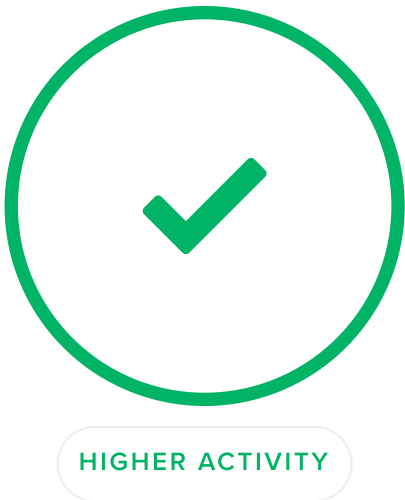
Other, less well-researched *UGT1A1* variants with reduced enzyme activity include:

- ‘A’ at [rs10929302](#) (*UGT1A1*93*) [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)
- ‘T’ at [rs887829](#) (*UGT1A1*80*) [\[R\]](#), [\[R\]](#), [\[R\]](#)
- ‘G’ at [rs4124874](#) (*UGT1A1*60*) [\[R\]](#), [\[R\]](#), [\[R\]](#)
- ‘T’ at [rs4148325](#) [\[R\]](#), [\[R\]](#)
- ‘T’ at [rs199539868](#) [\[R\]](#)
- ‘T’ at [rs114982090](#) [\[R\]](#)

A [UGT2A1](#) variant, ‘G’ at [rs10518065](#), has been associated with poorer [cognitive performance](#). The negative effects of this allele are likely due to decreased production of glutathione, leading to increased oxidative stress in the brain [\[R\]](#), [\[R\]](#), [\[R\]](#).

The low-activity [UGT1A6](#) variants ‘G’ at [rs2070959](#) and ‘C’ at [rs1105879](#) have been associated with higher bilirubin levels and increased risk of toxicity from aspirin, benzene, and vinyl chloride [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

Finally, two [UGT2B7](#) variants, ‘G’ at [rs7438135](#) and ‘T’ at [rs7439366](#), have been associated with increased enzyme activity and decreased effectiveness of opioids [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).



Likely higher UGT activity based on 11 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
UGT2A1	rs10518065	GA
UGT2B7	rs7439366	TC
USP40	rs4124874	GT
UGT1A6	rs34983651	CC
UGT1A6	rs887829	CC
UGT1A6	rs6742078	GG
UGT1A6	rs4148325	CC
UGT1A1	rs4148323	GG
UGT2B7	rs7438135	GG
UGT1A6	rs2070959	AA
UGT1A6	rs1105879	AA
UGT1A6	rs10929302	GG
UGT1A1	rs199539868	CC
UGT1A1	rs114982090	CC

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

NAT2 (Detox)

NAT2 activity or status varies widely among individuals due to **genetic differences**. This status influences how one's body processes a range of environmental toxins.

Research has linked a lot of *NAT2* gene variants to changes in enzyme activity, but most resources use 7 main variants to determine the acetylator status. The frequency of this status varies widely between different ethnicities. Roughly **45%** of the overall population and **60%** of European descendants are **slow acetylators** [R].

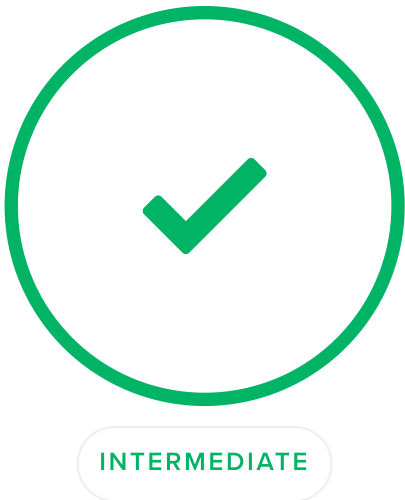
Being a slow NAT2 acetylator may be linked to higher odds of different types of cancer, especially bladder cancer. This link tends to be stronger in people exposed to **cigarette smoke and chemical dyes** [R, R, R, R, R].

The link between the slow NAT2 status and other types of cancer and other chronic conditions like asthma and diabetes is weaker [R, R, R].

Slow acetylators may also have a harder time detoxing certain drugs, which may put them at higher odds of side effects. For example, they tend to have higher rates of liver injury from the anti-tuberculosis drug isoniazid [R].

On the other hand, fast acetylators may be more prone to colon cancer, especially if they consume **well-done meat** frequently. This may be due to activation (O-acetylation) of certain toxins by NAT2 in the colon [R, R].

NAT2 also indirectly affects **histamine metabolism** by acetylating compounds that influence histamine levels. "Slow acetylators" with reduced NAT2 activity may experience more pronounced histamine intolerance symptoms due to difficulty processing dietary histamine or histamine-releasing substances.



Likely an intermediate acetylator based on 7 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NAT2	rs1041983	CT
NAT2	rs1799930	GA
ASAH1	rs4271002	CG
NAT2	rs1801280	TT
NAT2	rs1801279	GG
NAT2	rs1799931	GG
NAT2	rs1799929	CC
NAT2	rs1208	AA

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



Oxidative Stress

Oxidative stress occurs when there’s an imbalance between free radicals and antioxidants in the body, leading to cell damage and contributing to various chronic diseases. **Your body’s ability to manage oxidative stress is partly determined by your genetics, influencing how effectively you neutralize harmful free radicals.**

Genes like *GSTP1*, *SOD2*, *GXP1*, *CAT*, or *NQO1* are critical in protecting cells from oxidative damage by encoding antioxidant enzymes that help detoxify free radicals. Understanding your genetic makeup can help you manage oxidative stress and reduce your risk of related health issues.

 LOWER ACTIVITY SOD2 (Oxidative Stress) Likely lower SOD2 activity	 LOWER ACTIVITY GCLC (Glutathione & Detox) Predisposed to lower GCLC activity	 LOWER ACTIVITY GSTA1 (Detox/ Glutathione) Predisposed to lower GSTA1 activity
 TYPICAL ACTIVITY GCLM (Glutathione & Detox) Predisposed to a typical GCLM activity	 TYPICAL ACTIVITY GPX4 (Selenium & Glutathione) Predisposed to typical GPX4 activity	 TYPICAL ACTIVITY GSR (Oxidative Stress) Predisposed to a typical GSR activity
 TYPICAL ACTIVITY CAT (Oxidative Stress) Predisposed to a typical CAT activity	 HIGHER ACTIVITY GSTP1 (Detox) Likely higher GSTP1 activity	 HIGHER ACTIVITY SOD1 (Oxidative Stress) Predisposed to higher SOD1 activity
 HIGHER ACTIVITY GPX1 (Glutathione/Detox) Likely higher GPX1 activity	 HIGHER ACTIVITY GSTM3 (Oxidative Stress) Predisposed to a higher GSTM3 activity	 HIGHER ACTIVITY HMOX1 (Oxidative Stress) Predisposed to a higher HMOX1 activity



HIGHER ACTIVITY

NQO1 (Detox)

Likely higher NQO1 activity

SOD2 (Oxidative Stress)

The [SOD2](#) gene has many described polymorphisms. Among them, [rs4880](#) has got most of the spotlight in SOD2 research. Its minor allele ‘G’ is associated with decreased activity and worse protection against oxidative stress. However, some cell research suggests that this variant can cross the mitochondrial membrane more easily [\[R\]](#).

Owing to its decreased antioxidant activity, this variant has been associated with diseases such as [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#):

- Breast, prostate, and colorectal cancer
- Hypertension
- Sporadic motor neuron disease
- Alzheimer’s disease
- Parkinson’s disease
- Noise-induced hearing loss
- Cisplatin-induced ear toxicity
- Infertility
- Phthalate-induced lung damage

In contrast, the major ‘A’ variant is more common among people with [\[R\]](#):

- Cardiomyopathy
- Atherosclerosis
- Lung cancer

The association of this variant with [longevity](#) isn’t straightforward either. While a study found the ‘G’ variant was more common among very elderly Danish people, the ‘A’ variant was prevalent among very elderly Ashkenazi Jewish men in another study. Nevertheless, **the growing consensus is that ‘G’ is the risk allele of rs4880** [\[R\]](#), [\[R\]](#).

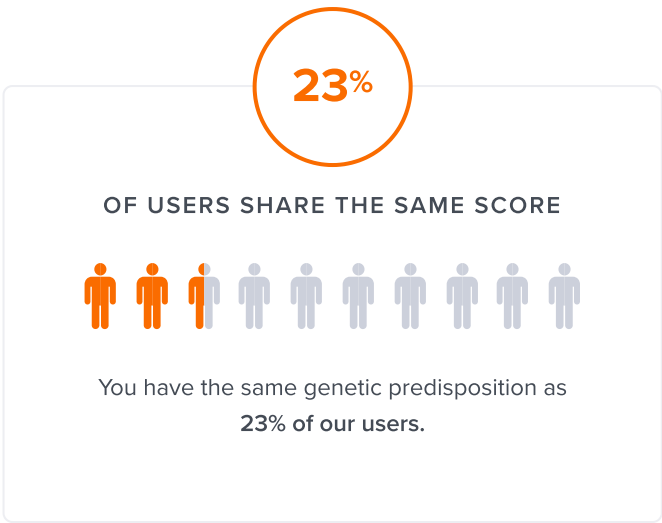
A second SNP, [rs2758331](#), has also been associated with lifespan. In one study of exceptionally long-lived people in New England, the ‘C’ allele of rs2758331 was significantly more common in the oldest old than in the general population [\[R\]](#).

This SNP is nowhere near as well-studied as rs4880, and only a single study has investigated its effect on lifespan so far. Furthermore, while the ‘A’ allele of rs2758331 has been associated with prostate cancer (thereby supporting the idea of



LOWER ACTIVITY

Likely lower SOD2 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SOD2	rs4880	GG
TCP1	rs2758331	AA

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

a beneficial ‘C’ allele), the ‘C’ allele has been associated with liver damage after bisphenol A (BPA) exposure [\[R\]](#), [\[R\]](#).

GCLC (Glutathione & Detox)

One GCLC variant, [rs17883901-A](#), showed a link with higher levels of mercury in hair and blood in several studies. The effects were particularly big for people with two copies (AA). However, one small study found the opposite effect [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

This variant may also be linked to:

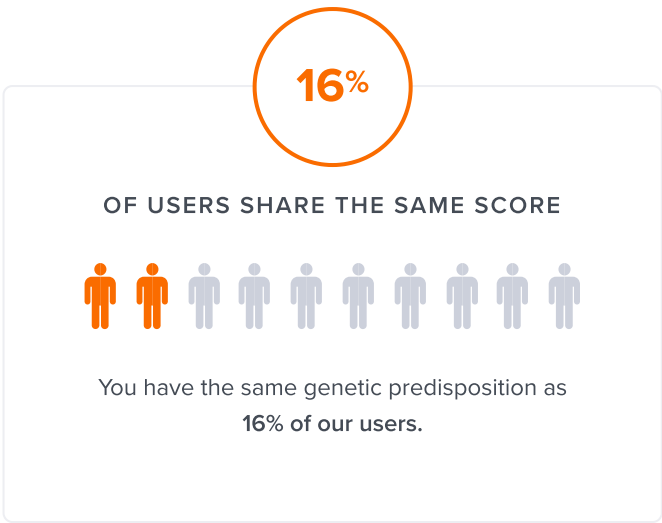
- Diabetic eye disease (retinopathy) [\[R\]](#)
- Reduced kidney function in people with diabetes [\[R\]](#)
- Fatty liver (NASH) [\[R\]](#)
- Reduced lung function [\[R\]](#)
- High blood pressure in pregnancy (preeclampsia) [\[R\]](#)

Another less-studied GCLC gene variant, [rs761142-A](#), may affect mercury levels and toxicity. In one study, mothers with two copies of this variant (AA) had higher hair mercury levels. Also, increased blood mercury levels during pregnancy were linked to developmental issues in their children [\[R\]](#).



LOWER ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GCLC	rs17883901	AG
GCLC	rs761142	CA

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

GSTA1 (Detox/ Glutathione)

The most studied GSTA1 variant is [rs3957357](#). The **A allele** is associated with reduced enzyme activity compared to the G allele. Individuals carrying the A allele may have decreased detoxification capacity, particularly for specific environmental toxins and medications [\[R\]](#).

One study investigated the link between this SNP and Balkan Endemic Nephropathy (BEN) - a mysterious kidney disease that occurs almost exclusively in certain rural areas along the Danube River in southeastern Europe. People with the A allele were 60% more likely to have BEN. The study also suggested that GSTA1 is involved in **fungal toxin** (ochratoxin) metabolism [\[R\]](#).

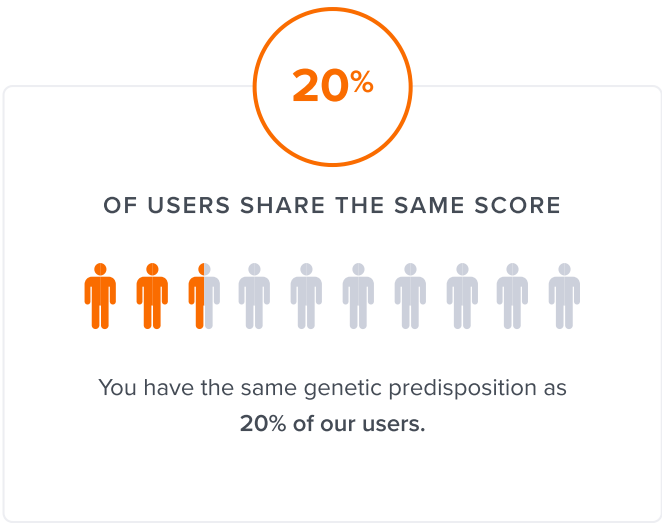
This variant may also be linked to [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#):

- Higher odds of liver cancer
- Stronger adverse effects of chemo
- Higher odds of asthma and allergies
- Lower hemoglobin levels
- Lower free testosterone levels

Another GSTA1 variant, [rs3957356](#), has shown similar associations. These two variants are almost always inherited together, meaning that you likely have either none or both of them.



Predisposed to lower GSTA1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GSTA1	rs3957357	AA

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

GCLM (Glutathione & Detox)

The main GCLM variant is [rs41303970](#). Studies have linked its “**A**” **allele** to:

- Improved urinary clearance of mercury [\[R\]](#)
- Lower hair mercury levels (“AA”) [\[R\]](#)
- Better lead detox and lower blood levels [\[R\]](#)
- Higher glutathione levels in people with diabetes [\[R\]](#)

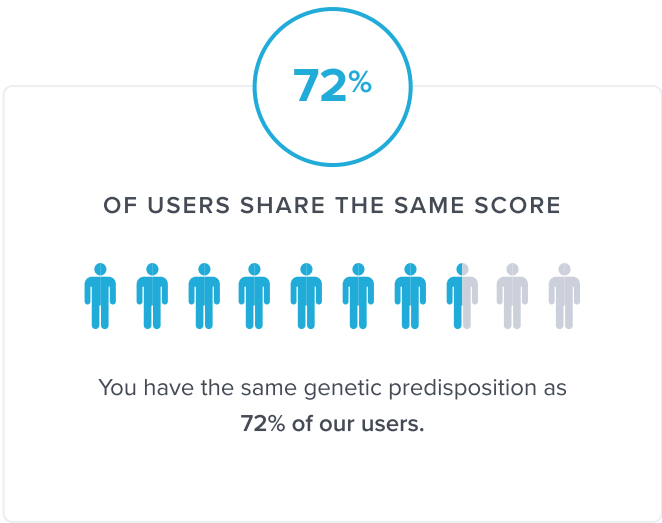
However, other studies have linked it to:

- Higher odds of diabetes and heart disease [\[R\]](#)
- Thyroid inflammation [\[R\]](#)
- Higher blood mercury levels infrequent fish consumers [\[R\]](#)
- Higher odds of liver disease due to hepatitis infection [\[R\]](#)



TYPICAL ACTIVITY

Predisposed to a typical GCLM activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GCLM	rs41303970	GG

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

GPX4 (Selenium & Glutathione)

The main GPX4 variant is [rs713041](#). Its “T” allele may be linked to:

- Colorectal cancer [\[R\]](#)
- Stroke [\[R\]](#)
- Poor prognosis of breast cancer [\[R\]](#)
- Pancreas inflammation [\[R\]](#)
- Vitamin B deficiencies [\[R\]](#)

However, some studies did not confirm the above findings [\[R\]](#), [\[R\]](#).

Some studies even found **protective associations** between this variant and:

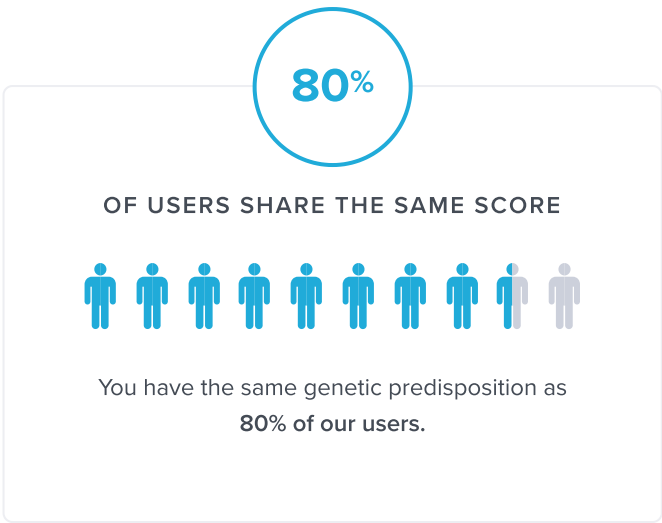
- High blood pressure in pregnancy [\[R\]](#)
- DNA damage [\[R\]](#)
- Heart problems in people with diabetes [\[R\]](#)
- Endometriosis [\[R\]](#)
- Alzheimer’s disease [\[R\]](#)

Scientists are unsure about the reason behind these conflicting findings. This variant seems to be **beneficial when enough dietary selenium is available**, so people carrying it should pay special attention to selenium intake [\[R\]](#).



TYPICAL ACTIVITY

Predisposed to typical GPX4 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GPX4	rs713041	CT

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

GSR (Oxidative Stress)

The main *GSR* gene variant is [rs1002149](#). Its minor ‘T’ allele has been associated with an increased risk of noise-induced hearing loss and COPD [\[R\]](#), [\[R\]](#).

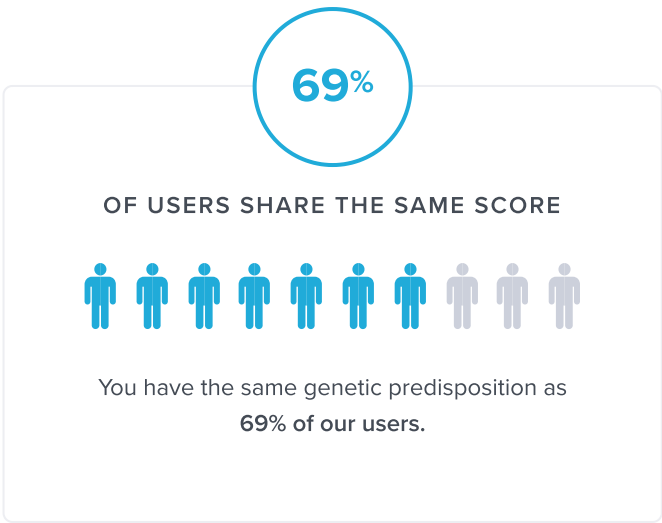
Moreover, carriers of this variant may further increase their risk of breast cancer if they drink alcohol [\[R\]](#).

This variant may increase enzyme activity [\[R\]](#).



TYPICAL ACTIVITY

Predisposed to a typical GSR activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GSR	rs1002149	GG

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

CAT (Oxidative Stress)

The best-characterized *CAT* polymorphism is [rs1001179](#), commonly referred to as -262G>A. Its minor ‘T’ allele may increase *CAT* expression but decrease it in people with conditions such as type 2 diabetes or chronic hepatitis C. This variant has been associated with an increased risk of [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#):

- Type 2 diabetes [\[R\]](#)
- Prostate cancer [\[R\]](#), [\[R\]](#)
- Breast cancer, especially in women using HRT or eating low fish amounts [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Aggressive chronic lymphocytic leukemia [\[R\]](#)
- Cerebral palsy [\[R\]](#)
- NAFLD and NASH [\[R\]](#), [\[R\]](#)
- COVID-19 hospitalization [\[R\]](#)
- Liver scarring in hepatitis C patients [\[R\]](#)
- Malaria [\[R\]](#)

However, this variant has also been associated with:

- Lower oxidative stress [\[R\]](#)
- Higher folate and lower homocysteine levels [\[R\]](#)
- Reduced risk of glaucoma [\[R\]](#)
- Reduced risk of male infertility [\[R\]](#)
- Reduced risk of Crohn’s disease [\[R\]](#)
- Reduced risk of acoustic neuroma [\[R\]](#)
- Reduced risk of bronchiolitis [\[R\]](#)
- Later onset of Wilson’s disease [\[R\]](#)

Another well-researched polymorphism is [rs769217](#), commonly referred to as -1167C>T. Its minor ‘T’ allele may increase *CAT* expression. This variant has been associated with [\[R\]](#):

- Lower oxidative stress [\[R\]](#)
- Reduced risk of steroid-dependent nephrotic syndrome [\[R\]](#)
- Reduced risk of AST elevation [\[R\]](#)

However, the variant has also been associated with an increased risk of

- Chronic hepatitis B, liver cirrhosis, and hepatocellular carcinoma [\[R\]](#)
- Noise-induced hearing loss [\[R\]](#)
- ICU admission, sepsis, and mortality in AKI patients [\[R\]](#)



TYPICAL ACTIVITY

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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CAT	rs769217	CC
CAT	rs1001179	TC
CAT	rs7943316	TA

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

Finally, the minor ‘T’ allele of [rs7943316](#) (-21A>T) may lower *CAT* expression and has been associated with [R](#), [R](#):

- Increased risk of vitiligo [R](#)
- Increased risk of keratoconus [R](#)
- Increased risk of depression [R](#)
- Increased risk of breast cancer [R](#)
- Increased risk of male infertility [R](#)
- Increased risk of noise-induced hearing loss [R](#), [R](#)
- Higher systolic blood pressure [R](#)
- Reduced risk of stroke [R](#)

GSTP1 (Detox)

The main GSTP1 gene variant is [rs1695](#) or **Ile105Val**. The “**G**” allele of this variant changes the GSTP1 structure and reduces its activity. As a result, it may impact the body's ability to detoxify various substrates, including carcinogens, drugs, and products of oxidative stress.

Studies have linked it to:

- Increased drug toxicity (chemotherapy) [\[R\]](#)
- Increased mercury toxicity [\[R\]](#)
- Higher odds of asthma due to smoke exposure (“GG” genotype) [\[R\]](#)
- Higher odds of breast cancer [\[R\]](#)
- Allergic reactions in people exposed to air pollution [\[R\]](#)

However, some studies **failed to confirm** the link between this variant and asthma, mercury toxicity, or cancer [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

The effects of rs1695-G on breast cancer may be more pronounced in women who eat less **cruciferous vegetables**. This finding makes sense given that cruciferous vegetables are rich in glutathione and other antioxidants [\[R\]](#).

Another important GSTP1 variant is [rs1138272](#) or **Ala114Val**. Its minor “**T**” allele may be linked to:

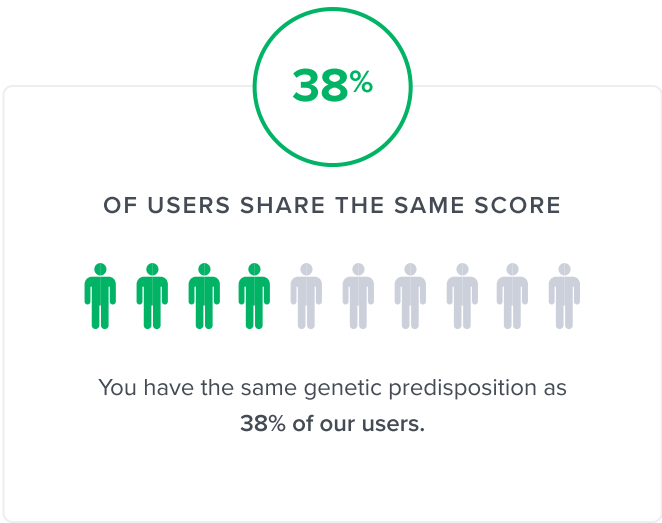
- Stronger effects of smoking on Parkinson’s disease [\[R\]](#)
- Increased mercury toxicity [\[R\]](#)
- Nerve problems [\[R\]](#)

However, many studies **didn’t find the negative effects** of this variant on detox ability and cancer [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).



HIGHER ACTIVITY

Likely higher GSTP1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GSTP1	rs1695	AA
GSTP1	rs1138272	CC

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

SOD1 (Oxidative Stress)

The best-characterized *SOD1* polymorphism is [rs2070424](#). Its minor ‘G’ allele is believed to increase the expression of the gene, resulting in lower oxidative stress. This variant has been associated with a decreased risk of:

- Alzheimer’s disease [\[R\]](#)
- Stomach cancer [\[R\]](#)
- Ulcerative colitis [\[R\]](#)
- PCOS [\[R\]](#)
- Type 2 diabetes [\[R\]](#)
- Myelomeningocele [\[R\]](#)

However, this variant has been linked to an increased risk of

- Breast cancer [\[R\]](#)
- Noise-induced hearing loss [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Parkinson’s disease [\[R\]](#)
- Hepatitis from antituberculosis drugs [\[R\]](#)
- Cataracts in patients with type 2 diabetes [\[R\]](#)

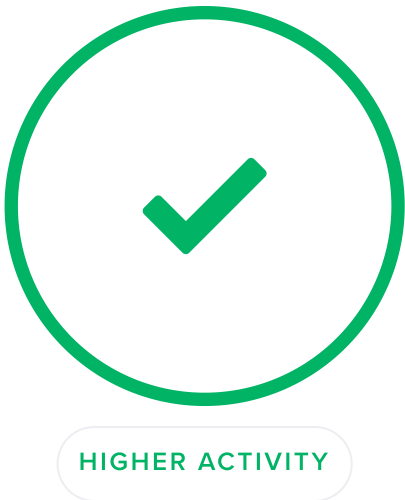
A variant possibly reducing SOD1 activity, ‘T’ at [rs1041740](#), has been associated with an increased risk of:

- Breast cancer [\[R\]](#)
- Diabetic nephropathy [\[R\]](#)
- Microalbuminuria [\[R\]](#)
- Cardiovascular disease and mortality [\[R\]](#), [\[R\]](#)
- Peritonitis [\[R\]](#)

However, this variant has been associated with a decreased risk of type 1 diabetes and lower total and LDL cholesterol levels [\[R\]](#), [\[R\]](#).

Finally, the ‘G’ allele of [rs10432782](#) has been associated with decreased prostate cancer risk and severity, suggesting increased SOD1 activity [\[R\]](#), [\[R\]](#).

However, this variant has been linked to an increased risk of noise-induced hearing loss and cardiovascular death in type 2 diabetes patients [\[R\]](#), [\[R\]](#), [\[R\]](#).



Predisposed to higher SOD1 activity based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SCAF4	rs2070424	AA
SOD1	rs10432782	TT
SOD1	rs1041740	CC

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

GPX1 (Glutathione/Detox)

One study found a direct link between a common [GPX1](#) variant and human [longevity](#). According to a cohort of elderly Danish people born in 1905, the heterozygous **genotype ‘AG’ at [rs1050450](#)** was significantly more common in the very elderly than in the general population [\[R\]](#).

The authors of the study suggested there could be some kind of survival benefit for the ‘AG’ genotype, but they did not speculate as to why the heterozygote might have an advantage over ‘AA’ and ‘GG’ [\[R\]](#).

That said, other studies have strongly suggested that the **‘G’ allele at rs1050450 confers higher GPx activity**, which is linked to better health outcomes [\[R\]](#), [\[R\]](#).

Along with other variants, like [rs1800668](#) and [rs3811699](#), this variant has also been linked to [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#):

- Rheumatoid arthritis
- Kashin-Beck disease
- Heart disease
- Some types of cancer

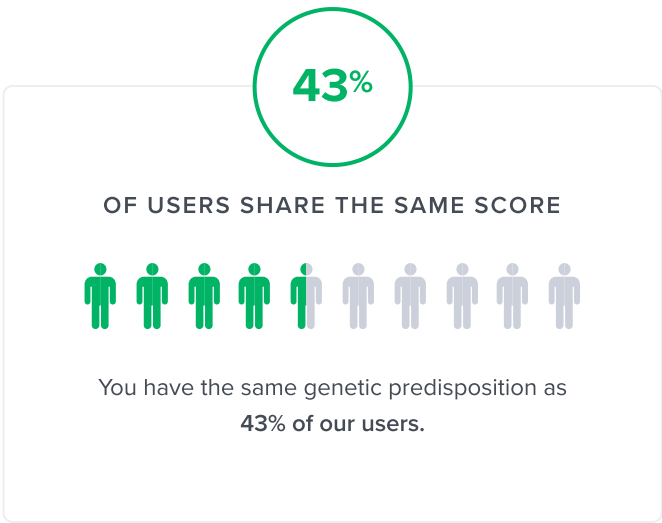
Kashin-Beck disease (KBD) is a bone disease that causes arthritis-like joint pain, enlarged joints, and decreased range of motion. People with KBD tend to have significantly higher oxidative stress and significantly lower selenium, suggesting that the disease could be caused (at least in part) by poor GPx activity [\[R\]](#), [\[R\]](#).

Please note: These three variants are closely linked, so if you have a “bad” allele at one, you will likely also have “bad” alleles at others.



HIGHER ACTIVITY

Likely higher GPX1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GPX1	rs1050450	GG

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

GSTM3 (Oxidative Stress)

The main *GSTM3* gene variant is [rs7483](#). Its minor ‘T’ allele may reduce the levels of the GSTM3 protein, potentially increasing oxidative stress in the body. This variant has been associated with an increased risk of [\[R\]](#):

- Alzheimer’s disease (especially in APOE4-allele non-carriers) [\[R\]](#)
- Prostate cancer progression to castration-resistant cancer [\[R\]](#)

However, this variant may also protect from peptic ulcers caused by *H. pylori* by increasing pepsinogen I levels [\[R\]](#).

Another variant is [rs1332018](#), whose ‘G’ allele is also linked to lower GSTM3 levels. This variant has been associated with an increased risk of [\[R\]](#), [\[R\]](#):

- Kidney cancer [\[R\]](#)
- Advanced testicular cancer [\[R\]](#)
- Brain cancer in children born to mothers with high cured meat intake [\[R\]](#)



HIGHER ACTIVITY

Predisposed to a higher GSTM3 activity based on 2 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GSTM3	rs1332018	GT
GSTM3	rs7483	CC

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

HMOX1 (Oxidative Stress)

The main *HMOX1* polymorphism is [rs2071746](#). Its minor ‘A’ allele may increase HO-1 production. This variant has been associated with a decreased risk of [\[R\]](#):

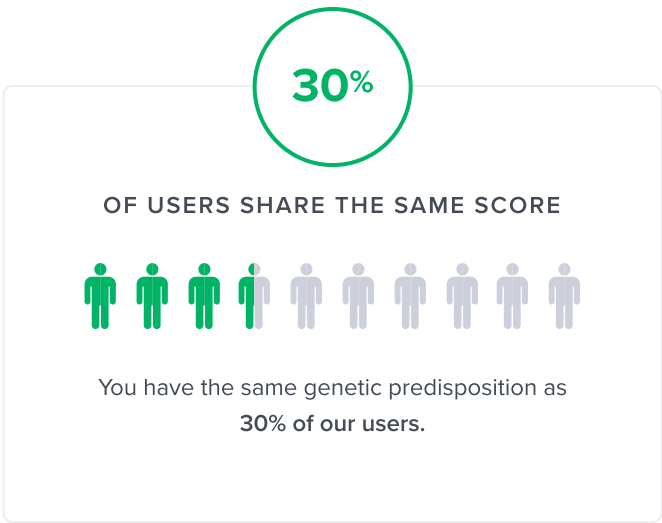
- Atherosclerotic stroke [\[R\]](#)
- Alzheimer’s disease [\[R\]](#), [\[R\]](#)
- Essential tremor [\[R\]](#)
- Restless legs syndrome [\[R\]](#)
- Sickle cell disease [\[R\]](#)
- Kidney complications [\[R\]](#)

However, this variant has been associated with an increased risk of Parkinson’s disease. This may be because increased HO-1 activity may contribute to iron buildup [\[R\]](#).



HIGHER ACTIVITY

Predisposed to a higher HMOX1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TOM1	rs2071746	AA

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

NQO1 (Detox)

Certain [NQO1](#) gene variants may impair the body’s [ability to detox](#) by reducing the activity of this enzyme [\[R\]](#), [\[R\]](#).

One of the main culprits is a variant called **NQO1*2**, which corresponds to [rs1800566-A](#). People with this variant have reduced NQO1 activity [\[R\]](#), [\[R\]](#).

Another major one is **NQO1*3**, which corresponds to [rs1131341-A](#). It leads to a slower conversion of quinone into its safer metabolites [\[R\]](#).

NQO1 gene variants may play a role in **DNA damage**. Research has found their associations with different types of cancer [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

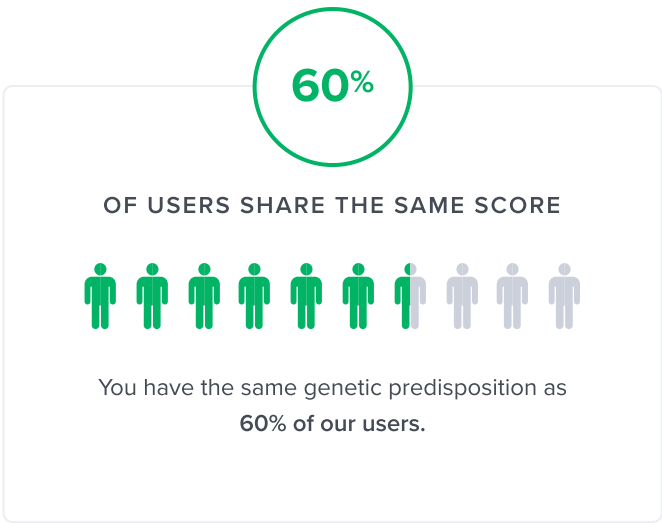
Research shows that people with the NQO1*2 variant (rs1800566-A) are up to 7.6 times more likely to experience **benzene toxicity**, even from low-level exposure [\[R\]](#), [\[R\]](#), [\[R\]](#).

This variant may also be linked to Alzheimer’s disease, but the evidence is mixed [\[R\]](#).



HIGHER ACTIVITY

Likely higher NQO1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NQO1	rs1800566	GG
NQO1	rs1131341	GG

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



Methylation

Methylation is a crucial biochemical process that regulates gene expression, DNA repair, and detoxification. This process plays a significant role in overall health, influencing inflammation, oxidative stress, and homocysteine levels. Genetic variations in methylation-related genes can affect how efficiently these processes occur, potentially increasing disease risk.

Genes like *MTHFR*, *MTR*, *MTRR*, and *CBS* are involved in the regulation of folate metabolism and homocysteine levels, while *PEMT* and *SHMT1* are involved in choline metabolism and methylation reactions. Additionally, *DNMT3B* and *MAT1A* are key players in maintaining DNA integrity and cellular function. Understanding how these genes impact methylation can help you take targeted steps to reduce your risk for chronic conditions.

LOWER ACTIVITY

MTHFR

Likely lower MTHFR activity

LOWER ACTIVITY

MTR (Methylation)

Likely typical MTR activity

LOWER ACTIVITY

MTHFD1 (Folate/
Methylation)

Likely lower MTHFD1 activity

WORSE GENETICS

MTHFS (Folate/
Methylation)

Likely worse MTHFS genetics

WORSE GENETICS

SHMT1 (Folate/
Methylation)

Likely worse SHMT1 genetics

HIGHER ACTIVITY

GNMT (Methylation)

Likely higher GNMT activity

TYPICAL ACTIVITY

MTRR (Methylation)

Likely typical MTRR activity

TYPICAL ACTIVITY

PEMT (Choline)

Likely typical PEMT activity

TYPICAL ACTIVITY

MTHFD1L (Folate/
Methylation)

Likely typical MTHFD1L activity

TYPICAL GENETICS

CHDH (Choline)

Likely typical CHDH genetics

TYPICAL

BHMT (Methylation)

Likely typical BHMT genetics

HIGHER ACTIVITY

DHFR (Folate/
Methylation)

Likely higher DHFR activity



TYPICAL GENETICS

FOLH1 (Folate/
Methylation)

Likely typical FOLH1 genetics



TYPICAL GENETICS

DNMT3B (Methylation)

Likely typical DNMT3B genetics



TYPICAL ACTIVITY

MAT1A (Methylation)

Likely typical MAT1A activity



TYPICAL ACTIVITY

AHCY (Methylation)

Likely typical AHCY activity



TYPICAL GENETICS

TYMS (Methylation)

Likely typical TYMS genetics



HIGHER ACTIVITY

CBS (Transsulfuration)

Likely higher CBS activity

MTHFR

Key Takeaways:

- MTHFR is an enzyme that helps your body process folate, an important nutrient for many body functions and processes.
- If you have lower MTHFR activity due to genetics, make sure you include folate-rich foods in your diet, like fruits and vegetables or other fortified foods. This is even more important with pregnancy.

The most common *MTHFR* SNP is **rs1801133** (C677T). The '**A**' **variant** of this SNP decreases the activity of the MTHFR enzyme. People with two 'A' variants may have about 16% lower blood folate levels ('A' equals 'T' on the opposite DNA strand) [R].

The '**G**' **variant*** of another SNP, **rs1801131** (A1298C), also decreases MTHFR enzyme activity, but less so than rs1801133. The effects of this variant may only be meaningful in people who also have the other low-activity variant, rs1801133-AA ('G' equals 'C' on the opposite DNA strand) [R, R, R, R, R].

Read [this blog post](#) for more details about MTHFR variants and potential ways to reduce their impact.

If you carry a lower-activity variant, make sure your diet is healthy, well-balanced, and contains plenty of folate-rich food sources. These include [R, R, R]:

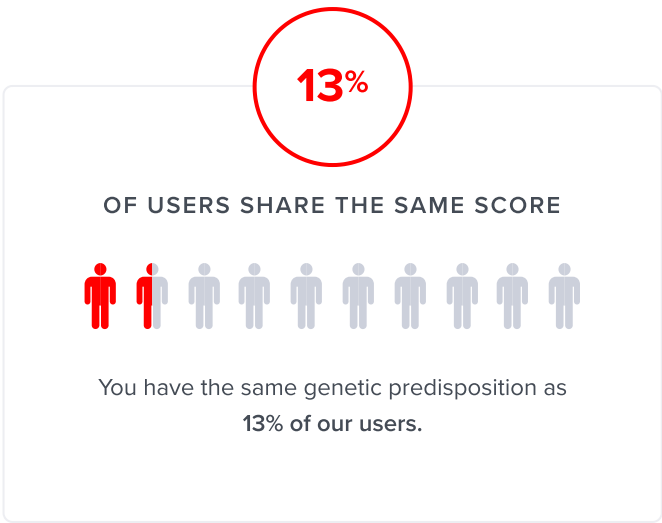
- Spinach
- Black-eyed and green peas
- Asparagus
- Lettuce
- Avocado
- Broccoli
- Citrus fruits
- Fortified rice, bread, and pasta

Some sources recommend methylfolate supplements instead of folic acid. Methylfolate supplements would in theory bypass the MTHFR enzyme, which converts folic acid to methylfolate. However, even if you have lower-activity *MTHFR* variants, experts say you can still process folic acid without any issues [R].



LOWER ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MTHFR	rs1801133	AA
MTHFR	rs1801131	TT

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

Importantly, CDC notes that folic acid is the only folate supplement proven to reduce neural tube defects. Methylfolate supplements have not been properly studied [R].

In addition to folate, there is some evidence that people with *MTHFR* variants may do better if they get more [riboflavin](#) (vitamin B2). This vitamin helps MTHFR work properly [R, R, R, R, R, R, R, R].

Good sources of riboflavin include [R, R]:

- Eggs
- Dairy (milk, cheese, yogurt)
- Lean and organ meats
- Green vegetables
- Fortified cereals
- Mushrooms
- Almonds

MTR (Methylation)

The main *MTR* gene variant is [rs1805087](#) or **A2756G**. The “G” allele changes the enzyme structure and appears to **increase** MTR activity, judging by its link with **lower homocysteine levels** [\[R, R, R, R, R\]](#).

However, studies have also linked this allele to:

- Fertility problems [\[R\]](#)
- Autism [\[R\]](#)
- Depression and stress [\[R, R\]](#)
- Cognitive impairment [\[R\]](#)

Assuming a higher activity, the “G” allele should increase methylation, and some studies have confirmed this. The mechanism behind the negative associations of this allele is not clear, but it may involve **excessive or altered DNA methylation** [\[R, R\]](#).

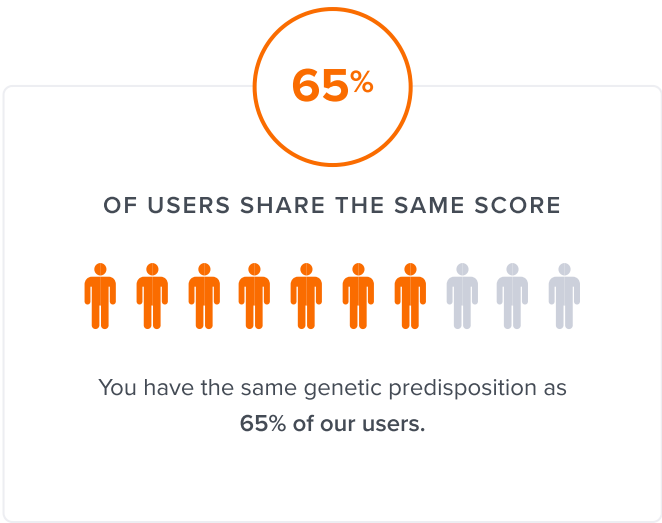
Finally, studies have found negative or mixed results for the link between rs1805087 and:

- Cancer [\[R, R\]](#)
- Neural tube defects [\[R\]](#)
- Congenital heart disease [\[R\]](#)



LOWER ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MTR	rs1805087	AA

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

MTHFD1 (Folate/ Methylation)

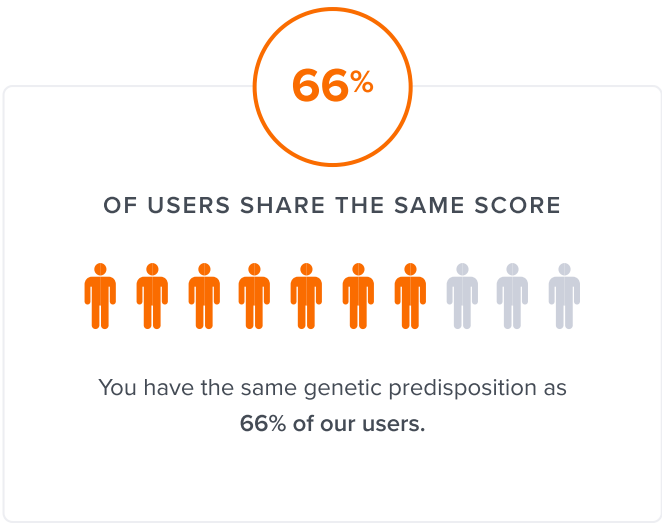
A study of 54 participants studied the impact of *MTHFD1* gene variations on [choline dietary requirements](#). When placed on a low-choline diet, people with the “A” allele at [rs2236225](#) were seven times more likely to develop the signs of choline deficiency, such as fatty liver [\[R\]](#).

In another trial of 43 young women, this variant worsened the effects of folate deficiency. Women with the “AA” genotype saw a higher increase in homocysteine levels during a low-folate diet. This variant had no impact during folate treatment (400 and 800 µg daily) [\[R\]](#).

The presence of the “A” allele at rs2236225 changes one amino acid in the MTHFD1 enzyme, making it less stable and more temperature-sensitive. Reduced MTHFD1 activity means less methyl-THF, which forces the body to use more choline for homocysteine methylation. When the intake of choline and folate is low, this effect may become significant and contribute to the signs of deficiency [\[R\]](#).



Likely lower MTHFD1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MTHFD1	rs2236225	GA

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

MTHFS (Folate/ Methylation)

The main MTHFS variant is [rs6495446](#). A large study has found a link between the “C” allele of this variant and higher odds of kidney disease [\[R\]](#).

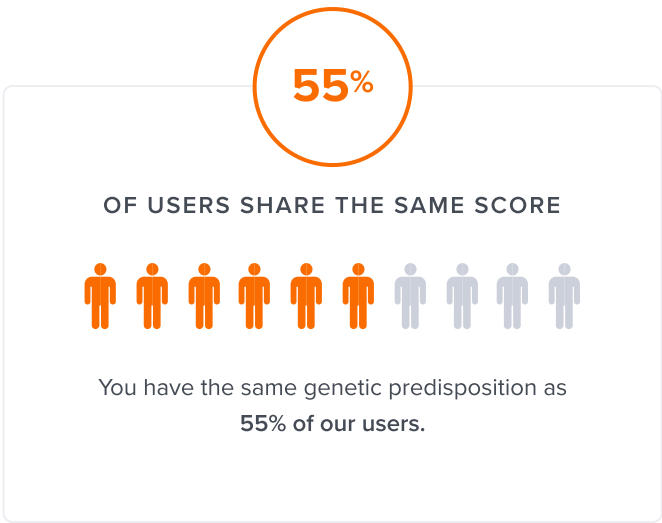
This finding is not surprising, given the various benefits of folate for kidney function. However, scientists are not sure if this variant has a causal effect and what the underlying mechanism is. Interestingly, they found that the “C” allele may increase MTHFS activity [\[R\]](#).

A study on people with type 2 diabetes found no link between this variant and diabetic kidney disease [\[R\]](#).



WORSE GENETICS

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MTHFS	rs6495446	CC

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

SHMT1 (Folate/ Methylation)

The main SHMT1 variant is **rs1979277**. Its “**A**” **allele** seems to reduce the ability of SHMT to produce 5,10-MTHF, leading to **lower levels of active folate**. This may have a detrimental effect on methylation. In other words, this variant may favor the folate cycle over the methylation cycle [\[R\]](#).

This variant may be linked to [\[R\]](#), [\[R\]](#):

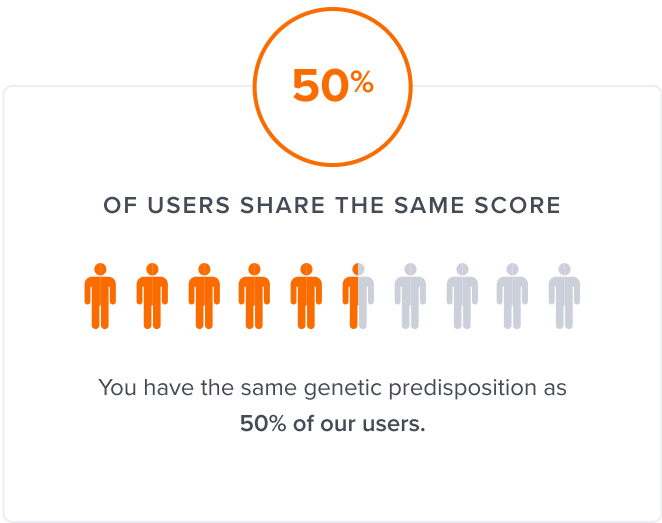
- Liver cirrhosis
- Congenital problems with blood vessels
- Down’s syndrome

In one study, the link between the MTHFR gene variant and heart disease was stronger in people who also carried this SHMT1 variant [\[R\]](#).



WORSE GENETICS

Likely worse SHMT1 genetics based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SHMT1	rs1979277	AA

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

GNMT (Methylation)

Even though the GNMT enzyme plays a key role in the methylation cycle, the available research on GNMT variants is scarce.

One GNMT variant, **rs9296404-T**, may be linked to higher homocysteine levels in response to dietary methionine [R].

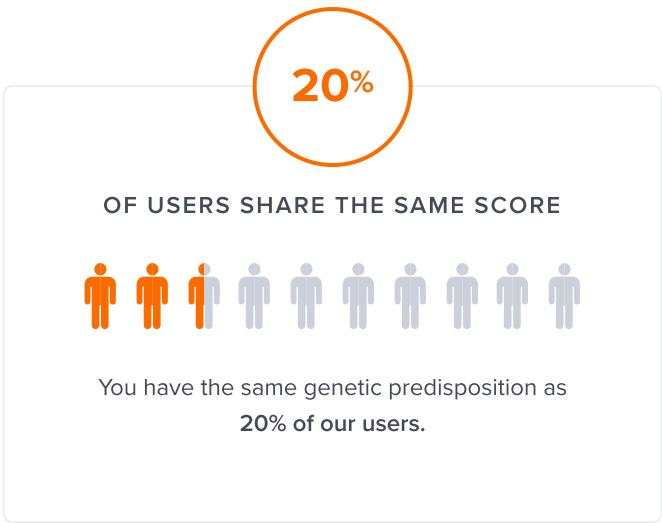
Another closely related GNMT variant, **rs10948059-C**, showed a link with liver damage and higher cholesterol levels. However, this variant may also be linked to better methotrexate (folate-targeting drug) response and its lower liver toxicity. The ‘C’ allele at another polymorphism (**rs11752813**) is usually inherited together with this variant, so you will most likely carry both or neither of them [R, R].

The above variants likely **increase GNMT activity**, shifting the SAM-e to SAH ratio towards the latter. As a result, the **methylation potential is reduced**. These variants are usually inherited together, meaning you probably carry either none or both [R].



HIGHER ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GNMT	rs9296404	TT
GNMT	rs10948059	CC

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

MTRR (Methylation)

The most studied SNP in the *MTRR* gene is [rs1801394](#) or **A66G**. The “**G**” **allele** changes one amino acid in the MTRR structure, **reducing** its ability to bind and activate MTR [\[R\]](#).

This variant has shown mixed results when it comes to homocysteine levels. Most studies observed its link with [elevated homocysteine](#) but some found no link. People with rs1801394-G may have an impaired response to folic acid for homocysteine reduction [\[R, R, R, R, R\]](#).

Studies have observed a potential link between this variant and:

- Colorectal and other types of cancer [\[R, R\]](#)
- Male fertility issues (mostly in Asians) [\[R, R\]](#)
- ADHD in children [\[R\]](#)
- Congenital heart disease (only in Asians) [\[R, R\]](#)
- Down syndrome [\[R, R\]](#)
- Increased choline needs [\[R\]](#)

A large meta-analysis failed to confirm the link between this variant and neural tube defects. In one study, the “GG” genotype was linked to spina bifida only in a subgroup of mothers **deficient in vitamin B12** [\[R, R\]](#).

Another well-researched SNP in this gene is [rs1532268](#). The “**T**” **allele** changes the enzyme structure and **reduces its activity** [\[R\]](#).

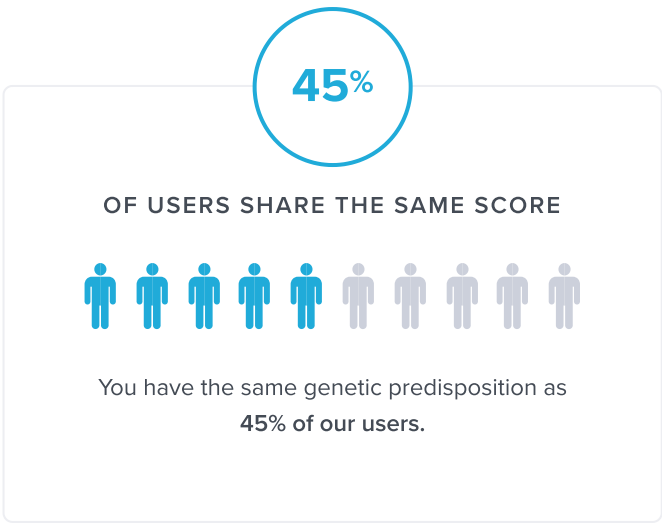
The effects of this variant may **also depend on vitamin B12 status**. In one study, it was associated with increased homocysteine when B12 status was low. Other studies have linked it to [\[R\]](#):

- Gastric cancer [\[R\]](#)
- Congenital heart disease [\[R\]](#)
- Neural tube defects (mixed evidence) [\[R, R\]](#)



TYPICAL ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MTRR	rs1801394	AG
MTRR	rs1532268	CT

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

PEMT (Choline)

Certain *PEMT* variant make the gene less responsive to estrogen stimulation. This prevents estrogen from binding to this gene and boosting its expression. As a result, PEMT activity drops and the liver doesn’t make enough choline to prevent deficiency [R, R].

A study identified one *PEMT* variation, [rs12325817](#), strongly associated with choline deficiency. Women with a ‘G’ allele were 25 times more likely to experience organ damage on a low-choline diet (<50 mg/70 kg daily) [R].

The research team added 33 women to the study and identified four additional variants with a weaker effect: [rs4646343](#)-T, [rs3760188](#)-T, [rs1531100](#)-A, and [rs4646365](#)-T. The last two were significant only in postmenopausal women and their impact was marginal [R].

The first three variants —rs12325817, rs4646343, rs3760188—are almost always inherited together, which means you will either have all risk alleles or none of them. Similarly, the last two —rs1531100 and rs4646365—are always inherited together.

Another polymorphism, [rs7946](#), has been associated with reduced PEMT function. Carriers of the minor ‘T’ variant can’t produce enough PC. In one lab test, the “TT” genotype resulted in a 30% loss of PEMT function [R].

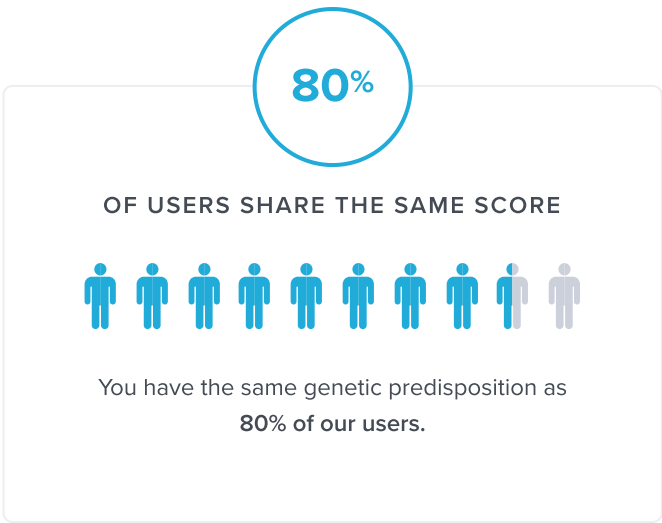
In addition to [choline deficiency](#), these variants have been associated with an increased risk of:

- [Heart disease](#)
- [Fatty liver](#)

However, they have also been linked to [lower obesity rates](#).



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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PEMT	rs1531100	AA
PEMT	rs4646365	TT
PEMT	rs4646343	GT
PEMT	rs7946	CT
PEMT	rs3760188	CT
PEMT	rs12325817	CC

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

MTHFD1L (Folate/Methylation)

The main MTHFD1L variant is [rs11754661](#). Studies have linked its “A” allele to [\[R, R, R, R\]](#):

- Alzheimer’s disease
- Parkinson’s disease
- Depression

Another important variant is [rs6922269](#). Its “A” allele has shown links to **heart disease** in different studies. However, some studies did not confirm this result [\[R, R, R, R\]](#).

Folate is necessary for homocysteine clearance. Folate deficiency and elevated homocysteine are related to both heart and brain health issues [\[R, R, R\]](#).

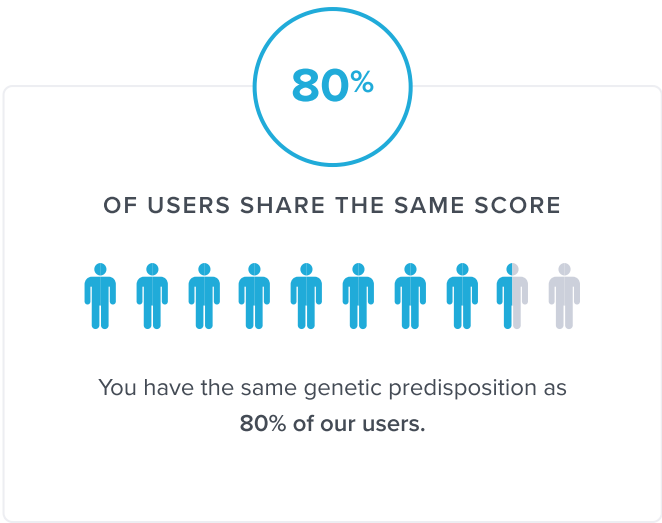
These variants likely reduce MTFD1 activity, impairing methylation and homocysteine removal. However, the available evidence is limited [\[R\]](#).

On the other hand, increased MTHFD1 activity may play a role in cancer development [\[R, R\]](#).



TYPICAL ACTIVITY

Likely typical MTHFD1L activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MTHFD1L	rs11754661	GG
MTHFD1L	rs6922269	GG

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

CHDH (Choline)

A study identified two [CHDH](#) variants associated with liver and muscle damage when dietary choline was restricted to <50 mg/day [\[R\]](#):

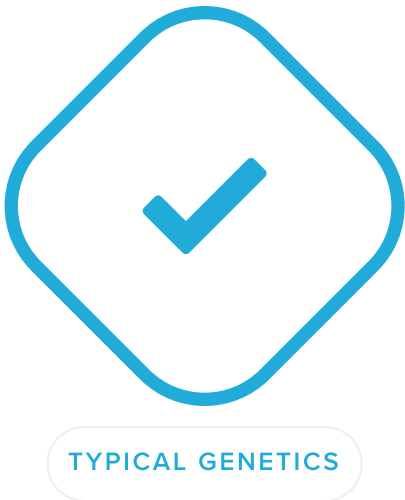
- [rs9001](#): those with the rare 'G' allele were 5x less likely to develop signs of choline deficiency
- [rs12676](#): those with the 'A' allele were 20x more likely to develop signs of choline deficiency

That said, the above study included only 57 subjects [\[R\]](#).

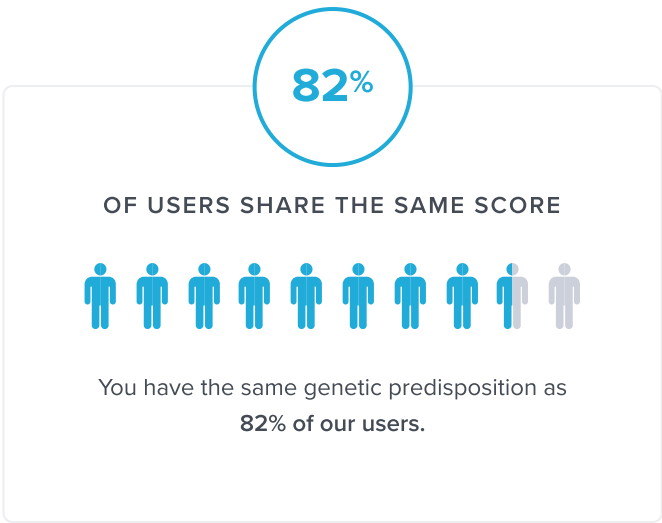
Other authors have noticed that people with the protective SNP, rs9001-G, use more choline for phosphatidylcholine production, and less for betaine production. On the contrary, rs12676-A favors the transformation of choline to betaine [\[R\]](#), [\[R\]](#).

Variants that favor betaine production may deplete choline stores and impair phosphatidylcholine synthesis. In the absence of dietary choline, this effect can contribute to organ damage due to choline deficiency [\[R\]](#).

The rs12676-A variant may also be linked to breast cancer [\[R\]](#).



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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CHDH	rs9001	TT
CHDH	rs12676	CC

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

BHMT (Methylation)

By far, the most well-researched *BHMT* polymorphism is [rs3733890](#). Its minor ‘A’ allele encodes an enzyme with increased affinity for homocysteine. Its effects on homocysteine levels are unclear but a study in pregnant women found lower transformation of betaine to DMG in ‘A’-carriers with high folate levels [\[R\]](#), [\[R\]](#).

This variant may increase the risk of:

- Folate therapy failure [\[R\]](#), [\[R\]](#)
- Head and neck cancer [\[R\]](#)
- Pulmonary thromboembolism [\[R\]](#)
- Placental abruption [\[R\]](#)
- Having a child with congenital heart disease [\[R\]](#)
- Having a child with Down’s syndrome [\[R\]](#)
- Neural tube defects [\[R\]](#), [\[R\]](#)
- Omphalocele [\[R\]](#)
- Spina bifida [\[R\]](#)
- Short telomeres [\[R\]](#)
- Liver toxicity from methotrexate [\[R\]](#)

However, it has also been associated with a decreased risk of:

- Cervical cancer [\[R\]](#), [\[R\]](#)
- Breast cancer and death from this condition [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Colorectal cancer [\[R\]](#)

In any case, the results are mixed in some cases and the effects may depend on other, less well-researched variants of the same gene, variants of other methylation pathway genes such as [MTHFR](#), [MTR](#), or [MTRR](#), and folate status.

Other variants of this gene include:

- ‘C’ at [rs651852](#): associated with lower folate levels [\[R\]](#)
- ‘C’ at [rs3797546](#): associated with cleft palate [\[R\]](#), [\[R\]](#)
- ‘C’ at [rs625879](#): associated with cleft palate and congenital heart defects, [\[R\]](#), [\[R\]](#)



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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
BHMT	rs3733890	AG
BHMT	rs651852	CT
JMY	rs3797546	TC
BHMT2	rs625879	AC

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

DHFR (Folate/ Methylation)

One of the most well-researched *DHFR* polymorphisms is [rs408626](#), also known as 317A>G. Its major ‘T’ allele may increase gene expression. This variant has been associated with [\[R\]](#):

- Worse response to methotrexate in patients with rheumatoid arthritis [\[R\]](#)
- Lower event-free survival in children with acute lymphoblastic leukemia [\[R\]](#)
- Increased risk of methotrexate-induced leucopenia [\[R\]](#)

However, this allele has also been associated with:

- Decreased risk of acute lymphoblastic leukemia relapse [\[R\]](#), [\[R\]](#)
- Better response to methotrexate in Crohn’s disease [\[R\]](#)

Another common polymorphism is [rs1650697](#), also known as 473T>C. Its minor ‘A’ allele may be linked to increased DHFR levels. This variant has been associated with [\[R\]](#):

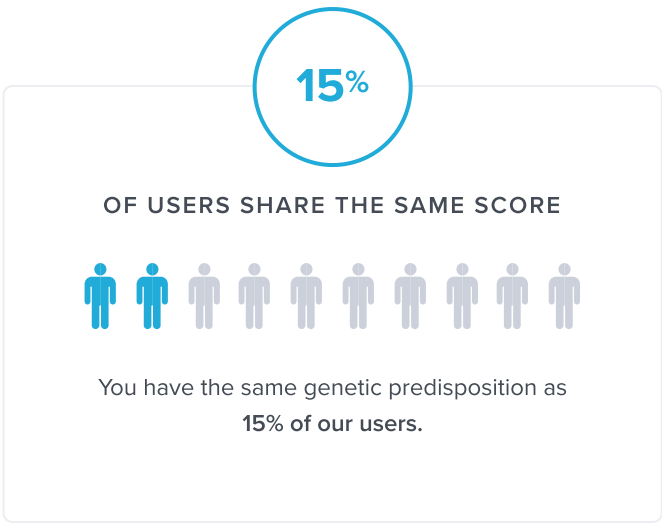
- Decreased risk of methotrexate hepatotoxicity [\[R\]](#)
- Increased survival in non-small cell lung cancer patients treated with methotrexate [\[R\]](#)

However, this variant may also increase the risk of pemetrexed toxicity [\[R\]](#).



HIGHER ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MSH3	rs408626	TT
MSH3	rs1650697	AA

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

FOLH1 (Folate/ Methylation)

The most well-researched *FOLH1* polymorphism is [rs202676](#). Its minor ‘G’ allele alters FOLH1 activity and negatively affects methylation [\[R\]](#).

It has been associated with an increased risk of neural tube defects such as anencephaly [\[R\]](#), [\[R\]](#).

People with this variant may also have:

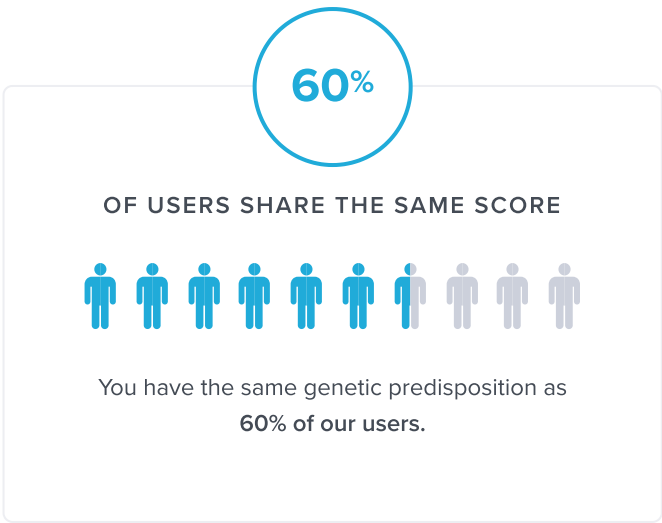
- Lower IQ and visual memory performance [\[R\]](#)
- Impaired arsenic metabolism through methylation [\[R\]](#)

Moreover, a study on patients with schizophrenia found that carriers of the ‘G’ allele failed to improve their negative symptoms in response to folate plus vitamin B12 supplementation [\[R\]](#).



TYPICAL GENETICS

Likely typical FOLH1 genetics based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
FOLH1	rs202676	AA

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

DNMT3B (Methylation)

One DNMT3B variant, **rs2424913-T**, has shown links with several traits related to methylation [R, R]:

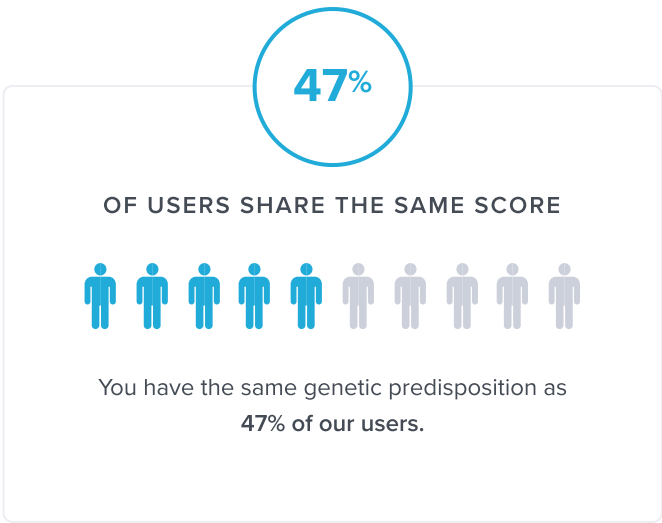
- Folate deficiency anemia
- Increased toxicity and reduced effectiveness of methotrexate
- Liver disease
- High blood sugar

The mechanism behind these findings is unclear. One possibility is that the variant increases DNMT3B activity, thus spending too much SAM-e for DNA methylation and depleting it from other pathways. Another possibility is that the variant reduces DNMT3B activity, thus impairing DNA methylation [R].



TYPICAL GENETICS

Likely typical DNMT3B genetics based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
DNMT3B	rs2424913	TC

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

MAT1A (Methylation)

A study has linked one MAT1A variant, **rs3851059** or d18777A, with methylation issues. People with the “**A**” **allele** had higher homocysteine levels if their folate status was low. They also had over 4x higher odds of stroke [\[R\]](#).

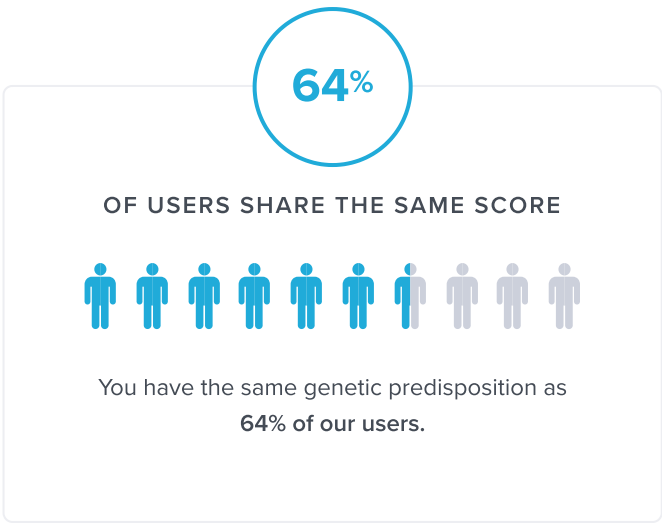
In the same study, people with another MAT1A variant, **rs7087728-A**, were less likely to have high blood pressure and stroke. They also had lower rates of DNA damage, especially if their vitamin B6 levels were high [\[R\]](#).

Interestingly, another study found a link between rs3851059-A and high homocysteine only in people with **higher fat intake** [\[R\]](#).



TYPICAL ACTIVITY

Likely typical MAT1A activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MAT1A	rs3851059	AG
MAT1A	rs7087728	GA

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

AHCY (Methylation)

Researchers have identified two rare *AHCY* gene variants that may change the enzyme structure and reduce its activity: [rs41301825-T](#) and [rs13043752-A](#) [R].

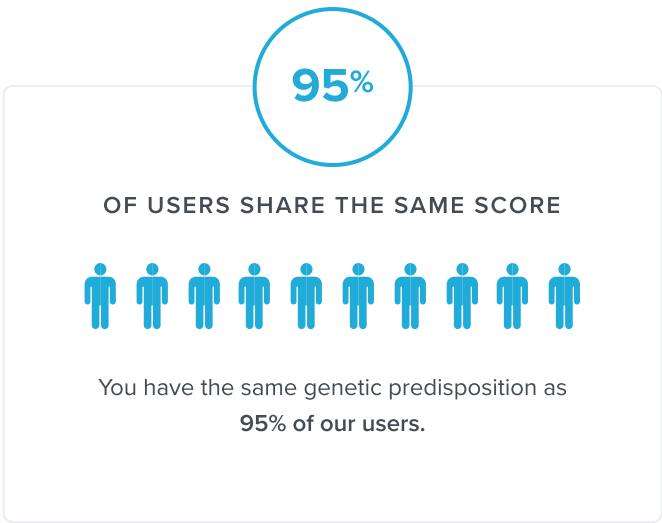
However, the research on AHCY variants is scarce, so we can't be sure about their potential effects on methylation and human health. One paper has linked [rs13043752](#) to venous thrombosis, independent of homocysteine levels [R].

People with rare AHCY mutations may have severe SAHH deficiency. **This report is not looking at such mutations and is not diagnostic of any health condition.**



TYPICAL ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
AHCY	rs13043752	GG
AHCY	rs41301825	CC

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

TYMS (Methylation)

One TYMS gene variant, **rs2853533-C**, seems to alter its activity. It may be linked to [R, R, R]:

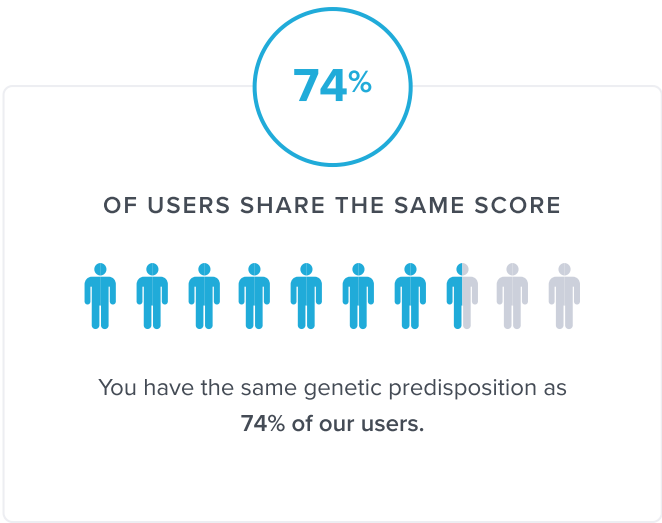
- Colorectal cancer
- Chemotherapy (5-FU) toxicity
- Spina bifida (unclear)

The available information about this variant is scarce, so we can't be sure about its associations and underlying mechanisms. It most likely involves depletion of active folate or impaired DNA production.



TYPICAL GENETICS

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TYMS	rs2853533	GG

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

CBS (Transsulfuration)

The main *CBS* variant is [rs234706](#). Its minor ‘A’ allele has been associated with lower homocysteine. In pregnant women, this variant was also found to reduce the risk of preeclampsia [\[R\]](#), [\[R\]](#).

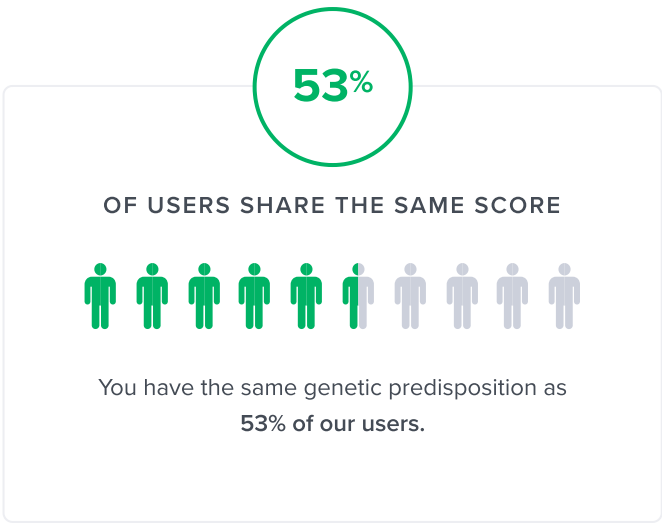
Other variants associated with lower homocysteine levels include:

- 'T' at [rs234709](#) [\[R\]](#)
- 'A' at [rs234713](#) [\[R\]](#), [\[R\]](#)
- ‘C’ at [rs2851391](#) [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)



HIGHER ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CBS	rs234706	AG

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



Hormones

Hormones play a central role in regulating many of the body’s functions, including metabolism and reproduction. **Imbalances in hormone levels can contribute to various health issues, including obesity, diabetes, and reproductive issues.** Genetics can influence how your body produces, metabolizes, and responds to hormones, affecting your overall health.

Genes like *DIO1* and *DIO2* are involved in thyroid hormone metabolism. *TPO* and *FOXE1* play roles in thyroid gland function, while *PGR* and *LHCGR* are linked to reproductive hormones. Additionally, *CYP11A1* and *CYP17A1* are involved in steroid hormone synthesis. Understanding how these genetic factors influence hormone balance can help you manage health risks related to hormonal imbalances.

LOWER ACTIVITY SHBG Gene (Sex Hormones) Predisposed to a lower SHBG activity	HIGHER ACTIVITY CYP17A1 (Sex Hormones) Predisposed to a higher CYP17A1 activity	HIGHER ACTIVITY HSD3B1 (Sex Hormones) Predisposed to a higher HSD3B1 activity
TYPICAL ACTIVITY UGT2B15 (Sex Hormones, Detox) Predisposed to a typical UGT2B15 activity	TYPICAL TPO (Thyroid) Likely typical TPO genetics	TYPICAL ACTIVITY PGR (Sex Hormones) Predisposed to a typical PGR activity
TYPICAL GENETICS LHCGR (Sex Hormones) Predisposed to typical LHCGR genetics	TYPICAL ACTIVITY UGT2B15 (Sex Hormones, Detox) Predisposed to a typical UGT2B15 activity	TYPICAL ACTIVITY CYP11A1 (Sex Hormones) Predisposed to typical CYP11A1 genetics
TYPICAL ACTIVITY ESR1 (Estrogen) Likely typical ESR1 activity	HIGHER ACTIVITY DIO1 (Thyroid) Likely higher DIO1 activity	HIGHER ACTIVITY DIO2 (Thyroid) Likely higher DIO2 activity



LOWER ACTIVITY

FOX E1 (Thyroid)

Predisposed to a lower FOX E1 activity

SHBG Gene (Sex Hormones)

One of the best-characterized *SHBG* polymorphisms is [rs6259](#), commonly referred to as Asp327Asn. Its minor ‘A’ allele encodes an alternative version of the protein with an additional carbohydrate chain that reduces the clearance of the protein, resulting in higher SHBG levels [\[R\]](#), [\[R\]](#), [\[R\]](#).

This variant has been associated with an increased risk of:

- Male infertility [\[R\]](#), [\[R\]](#)
- Low BMD [\[R\]](#)
- Poor prognosis in prostate cancer [\[R\]](#)
- Biliary tract cancer and stones [\[R\]](#)

However, this variant has been associated with a decreased risk of:

- Breast cancer in postmenopausal Asian women (but lower protective effects of soy isoflavones) [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Type 2 diabetes (especially in those consuming at least 2 cups of coffee per day) [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Metabolic syndrome [\[R\]](#)
- Liver cancer [\[R\]](#)
- Endometrial cancer (but lower protective effect of tea and soy isoflavones) [\[R\]](#)

Another well-researched polymorphism is [rs1799941](#). Its minor ‘A’ allele has been associated with higher SHBG and testosterone levels, as well as with [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#):

- Lower BMD [\[R\]](#)
- Increased risk of male hypogonadism [\[R\]](#)
- Decreased risk of metabolic syndrome [\[R\]](#)
- Decreased risk of type 2 diabetes [\[R\]](#), [\[R\]](#)

The minor ‘T’ allele of [rs727428](#) may decrease SHBG levels and has been associated with [\[R\]](#), [\[R\]](#), [\[R\]](#):

- Increased risk of PCOS [\[R\]](#), [\[R\]](#)
- Decreased risk of male infertility [\[R\]](#)
- Higher testosterone levels [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Lower estradiol levels [\[R\]](#), [\[R\]](#)
- Lower HbA1c levels [\[R\]](#)

Finally, the rare ‘C’ allele of [rs6257](#) has been associated with lower SHBG levels, as well as with [\[R\]](#), [\[R\]](#):



Predisposed to a lower SHBG activity based on 4 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TNFSF12	rs6259	GG
TNFSF12	rs1799941	GG
TNFSF12	rs727428	TT
SAT2	rs6257	TT

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

- Increased risk of gestational diabetes [\[R\]](#)
- Increased risk of type 2 diabetes and worse protective effects of coffee [\[R\]](#), [\[R\]](#)
- Decreased risk of breast cancer [\[R\]](#)
- Lower waist circumference [\[R\]](#)
- Lower triglyceride levels [\[R\]](#)

CYP17A1 (Sex Hormones)

The main *CYP17A1* polymorphism is [rs743572](#). Its minor ‘G’ allele increases enzyme activity, resulting in higher sex hormone levels. This variant has been associated with an increased risk of [\[R\]](#):

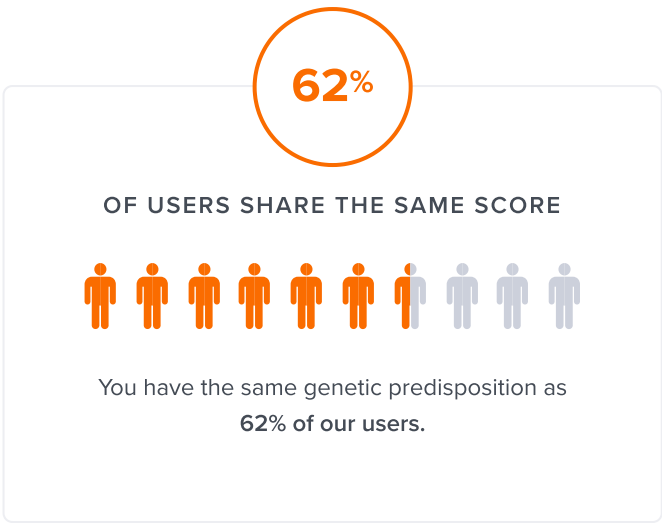
- Acne [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)
- PCOS [\[R\]](#)
- Breast cancer (in premenopausal but not postmenopausal women) [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Prostate cancer (in Black but not Caucasian or Asian populations) [\[R\]](#), [\[R\]](#)
- Ovarian cancer [\[R\]](#)
- Uterine fibroids [\[R\]](#)
- Endometriosis [\[R\]](#)

In contrast, it has been associated with a decreased risk of endometrial and pancreatic cancer [\[R\]](#), [\[R\]](#).



HIGHER ACTIVITY

Predisposed to a higher CYP17A1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
BORCS7	rs743572	AG

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

HSD3B1 (Sex Hormones)

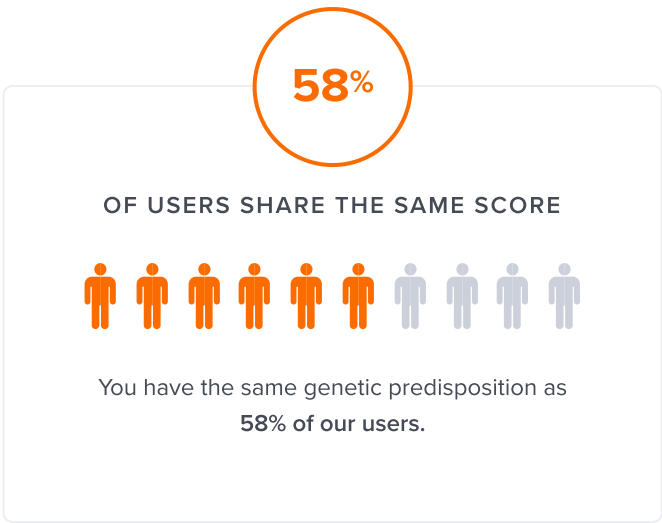
The main *HSD3B1* polymorphism is [rs1047303](#), commonly referred to as 1245C. Its minor ‘C’ allele encodes a version of the protein that is resistant to breakdown, resulting in its buildup. This variant has mostly been investigated in the context of prostate cancer and associated with several negative outcomes, such as [\[R\]](#):

- Worse response to androgen deprivation therapy [\[R, R, R\]](#)
- Increased risk of progression [\[R\]](#)
- Greater mortality [\[R, R\]](#)

However, a study associated this variant with an decreased risk and better prognosis of endometrial and breast cancer [\[R\]](#).



Predisposed to a higher HSD3B1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HSD3B1	rs1047303	CC

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

UGT2B15 (Sex Hormones, Detox)

The best-characterized *UGT2B15* variant is ***UGT2B15*2*** ([rs1902023](#)), commonly referred to as Y85D or G253T. Its minor ‘A’ allele encodes an enzyme with decreased activity that has been associated with slower metabolism of chemicals such as bisphenol A, S-oxazepam, parabens, sipoglitazar, and norandrosterone [\[R, R, R, R, R, R, R\]](#).

In contrast, the enzyme encoded by the minor allele may be more efficient at breaking down sex hormones such as androstenediol and DHT [\[R, R\]](#).

In line with the association of DHT with prostate cancer, the ‘A’ allele has been associated with a decreased risk of this disease [\[R\]](#).

Another well-researched variant is ***UGT2B15*5*** ([rs4148269](#)), commonly referred to as K523T. Its minor ‘G’ allele has been associated with lower *UGT2B15* expression, slower bisphenol A metabolism, and a decreased risk of prostate cancer. Moreover, a haplotype containing the major allele of this polymorphism was associated with an increased risk of colorectal cancer [\[R, R, R, R\]](#).



Predisposed to a typical UGT2B15 activity based on 2 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
UGT2B15	rs1902023	AC
UGT2B15	rs4148269	GT

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].

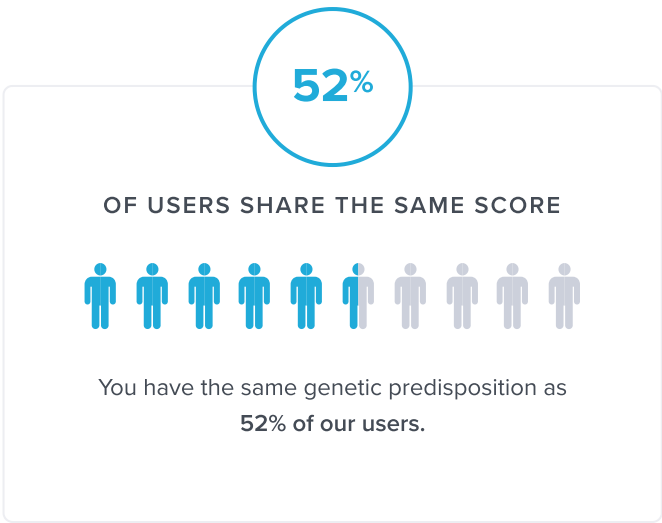
Doctors from Iran came to a similar conclusion after conducting a small study of 150 participants. The ‘C’ allele correlated with subclinical or “hidden” hypothyroidism [R].

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.

PGR (Sex Hormones)

The main *PGR* gene variant is [rs10895068](#), commonly referred to as +331G/A. Its minor ‘T’ allele selectively increases the expression of the PR-B isoform. This variant has been associated with an increased risk of [\[R\]](#):

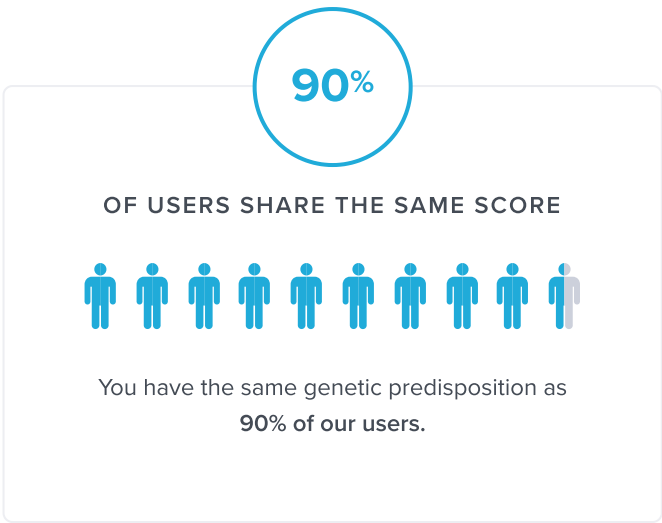
- Ovarian cancer [\[R\]](#)
- Endometrial cancer [\[R\]](#)
- Uterine fibroids [\[R\]](#)
- Implantation failure during in vitro fertilization [\[R\]](#)
- Preeclampsia [\[R\]](#)

On the bright side, this allele has been associated with a reduced risk of ovarian hyperstimulation syndrome and endometriosis [\[R\]](#), [\[R\]](#).



TYPICAL ACTIVITY

Predisposed to a typical PGR activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PGR	rs10895068	CC

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

LHCGR (Sex Hormones)

The main *LHCGR* gene variant is [rs13405728](#). Multiple studies have associated its major ‘A’ allele with an increased risk of PCOS. Women with this condition may have higher levels of testosterone, triglycerides, and LDL cholesterol if they carry this allele [\[R, R, R, R, R\]](#).

This variant has also been associated with an increased risk of endometroid adenocarcinoma and worse ovarian response to assisted reproductive technology [\[R, R, R\]](#).

Another well-researched polymorphism is [rs2293275](#). Its minor ‘T’ allele has been associated with an increased risk of PCOS and disease progression, and higher testosterone but lower LH and LH/FSH values in women with this condition [\[R, R, R, R, R, R\]](#).

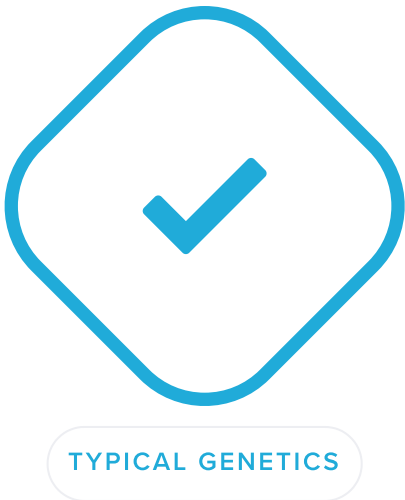
This variant has also been associated with accelerated ovarian aging and ovarian hyperstimulation syndrome and worse pregnancy outcomes after assisted reproductive technologies, but a higher number of oocytes retrieved [\[R, R, R, R\]](#).

In men, this variant has been associated with a lower risk of infertility, maldescended testicles, and aggressive prostate cancer [\[R, R, R\]](#).

The following variants have also been associated with an increased risk of PCOS:

- ‘T’ of [rs4953616](#) [\[R, R, R\]](#)
- ‘T’ of [rs7371084](#) [\[R, R\]](#)
- ‘C’ of [rs12470652](#) (only in Punjab) [\[R\]](#)

In men, the ‘C’ allele of rs7371084 has been associated with an increased risk of testicular cancer metastasis, as well as with higher LH levels and androgen sensitivity index [\[R\]](#).



Predisposed to typical LHCGR genetics based on 5 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
LHCGR	rs13405728	AA
LHCGR	rs7371084	TT
LHCGR	rs2293275	TC
STON1-GTF2A1L	rs4953616	CT
LHCGR	rs12470652	TT

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

UGT2B15 (Sex Hormones, Detox)

The best-characterized *UGT2B15* variant is ***UGT2B15*2*** ([rs1902023](#)), commonly referred to as Y85D or G253T. Its minor ‘A’ allele encodes an enzyme with decreased activity that has been associated with slower metabolism of chemicals such as bisphenol A, S-oxazepam, parabens, sipoglitazar, and norandrosterone [\[R, R, R, R, R, R, R\]](#).

In contrast, the enzyme encoded by the minor allele may be more efficient at breaking down sex hormones such as androstenediol and DHT [\[R, R\]](#).

In line with the association of DHT with prostate cancer, the ‘A’ allele has been associated with a decreased risk of this disease [\[R\]](#).

Another well-researched variant is ***UGT2B15*5*** ([rs4148269](#)), commonly referred to as K523T. Its minor ‘G’ allele has been associated with lower *UGT2B15* expression, slower bisphenol A metabolism, and a decreased risk of prostate cancer. Moreover, a haplotype containing the major allele of this polymorphism was associated with an increased risk of colorectal cancer [\[R, R, R, R\]](#).



Predisposed to a typical UGT2B15 activity based on 2 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
UGT2B15	rs1902023	AC
UGT2B15	rs4148269	GT

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

CYP11A1 (Sex Hormones)

The main *CYP11A1* polymorphism is [rs11632698](#). Its ‘G’ allele has been associated with an increased risk of:

- Recurrent pregnancy loss [\[R\]](#)
- PCOS [\[R, R\]](#)
- Bone loss in breast cancer patients treated with aromatase inhibitors [\[R\]](#)

Another variant, the ‘T’ allele of [rs4077582](#) has also been associated with an increased risk of:
recurrent pregnancy loss and PCOS [\[R, R, R, R, R, R\]](#).

Other variants associated with an increased risk of PCOS include:

- ‘A’ of [rs4887139](#) [\[R, R\]](#)
- ‘C’ of [rs4886595](#) [\[R\]](#)
- ‘G’ of [rs6495096](#) [\[R\]](#)

The minor ‘T’ allele of [rs2279357](#) has been associated with an increased risk of breast cancer in women. However, this variant has been associated with a decreased risk of late-onset hypogonadism in men with benign prostate hyperplasia [\[R, R\]](#).

Finally, the following variants have been associated with an increased risk of endometrial cancer [\[R\]](#):

- ‘A’ of [rs4555110](#)
- ‘A’ allele of [rs3825944](#)
- ‘C’ allele of [rs7173655](#)



TYPICAL ACTIVITY

Predisposed to typical CYP11A1 genetics based on 9 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CYP11A1	rs11632698	GG
ULK3	rs4077582	TT
CYP11A1	rs4887139	AA
CYP11A1	rs7173655	TT
CYP11A1	rs6495096	CC
CYP11A1	rs4886595	AA
CSK	rs4555110	GG
CSK	rs3825944	GG
CYP11A1	rs2279357	CC

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

ESR1 (Estrogen)

The two main *ESR1* variants are [rs2234693](#) (-397T>C or PvuII) and [rs9340799](#) (-351A>G or XbaI). They are often inherited together, meaning you will likely carry either none or both.

Their “C” and “G” alleles, respectively, may be linked to the following **positive health outcomes**:

- Stronger bones (higher BMD) [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Lower odds of endometrial cancer [\[R\]](#)
- Slower cognitive decline (only in European ancestry) [\[R\]](#)
- [Lower odds of anxiety_\(phobia\)](#) [\[R\]](#)
- Better cardiovascular health [\[R\]](#)
- Increased fertility in men [\[R\]](#)

On the other hand, they may be linked to the following **negative health outcomes**:

- Endometriosis [\[R\]](#)
- Breast cancer [\[R\]](#), [\[R\]](#)
- Prostate cancer [\[R\]](#), [\[R\]](#)
- Depression [\[R\]](#)

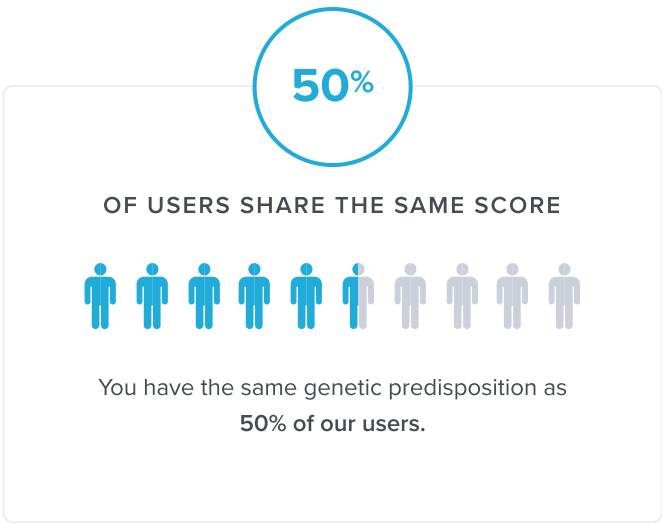
Expectedly, the effects of these variants are more pronounced in women. According to most of the above associations and some lab experiments, **rs2234693-C increases *ESR1* expression**, leading to more pronounced effects of estrogen [\[R\]](#).

However, some studies have found no links – or even opposite links – of these variants with most of the above health outcomes. They may be partly explained by different results in people of Asian vs European ancestry [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).



TYPICAL ACTIVITY

Likely typical ESR1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ESR1	rs2234693	CT
ESR1	rs9340799	GA

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].

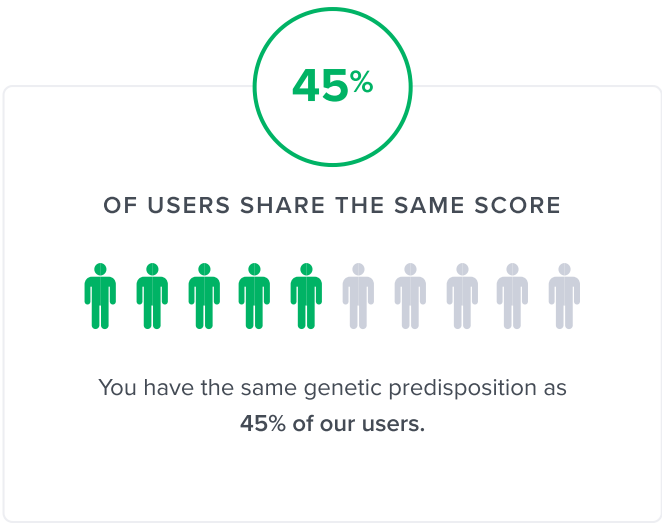
A larger trial came to a similar conclusion, though the effects were significant only for patients who had both copies of the “C” allele [R].

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



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.

FOXE1 (Thyroid)

In a study with 6,300 participants, the minor alleles of the following four *FOXE1* variants were associated with a decreased risk of [hypothyroidism](#) [R]:

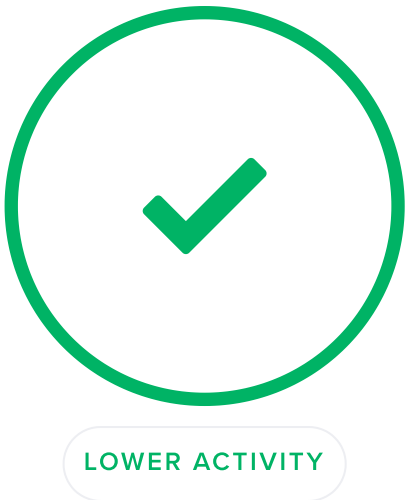
- ‘C’ of [rs925489](#)
- ‘A’ of [rs7850258](#)
- ‘A’ of [rs965513](#)
- ‘A’ of [rs10759944](#)

Of them, rs7850258 showed the strongest connection, with carriers of the ‘A’ allele showing 26% lower rates.

Scientists suggest that the above variants reduce *FOXE1* expression and allow for improved thyroid hormone production (T4) [R, R].

The effect allele carriers also had lower rates of autoimmune thyroiditis or [Hashimoto’s disease](#), which suggests autoimmunity as another potential mechanism [R].

On the other hand, some of these variants are associated with thyroid cancer, indicating a tumor-suppressor role of the *FOXE1* gene [R].

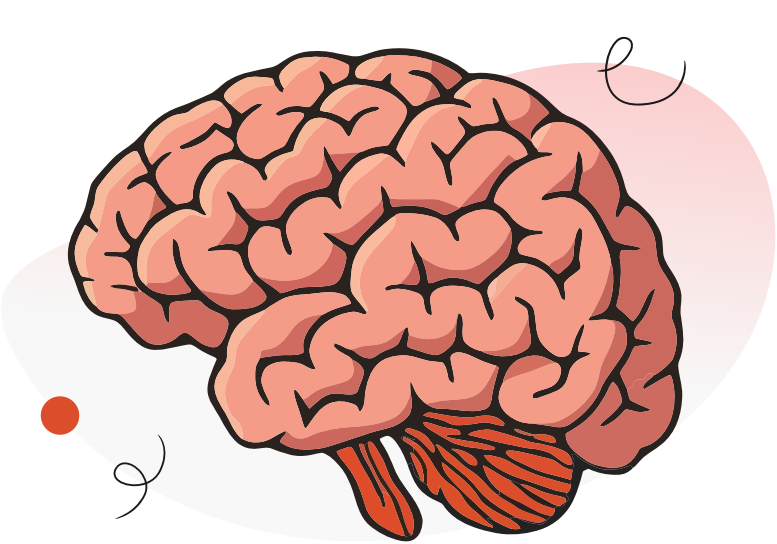


Predisposed to a lower FOXE1 activity based on 4 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TRMO	rs965513	AA
TRMO	rs925489	CC
FOXE1	rs7850258	AA
TRMO	rs10759944	AA

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



Neurotransmitters

Neurotransmitters are chemical messengers that regulate mood, cognition, sleep, and many other brain functions. Genetic variations can influence the production, breakdown, and transport of these molecules, impacting mental health, behavior, and neurological processes.

Genes such as *COMT*, *MAOA*, and *MAOB* affect the breakdown of key neurotransmitters like dopamine and serotonin. *DRD2* and *ANKK1* are involved in dopamine receptor function, influencing reward and motivation pathways. Additionally, *SLC6A3* regulates dopamine reuptake, while *TPH2* and *HTR2A* play important roles in serotonin synthesis and signaling. Understanding your genetic makeup related to neurotransmitters can provide insight into your brain chemistry and help guide lifestyle or treatment choices.

! HIGHER ACTIVITY MAOA (Dopamine/Serotonin) Likely higher MAOA activity	! HIGHER ACTIVITY MAOB (Dopamine/ Norepinephrine) Likely higher MAOB activity	! LOWER ACTIVITY SLC18A1 (Mental Health) Predisposed to a lower SLC18A1 activity
! LOWER ACTIVITY DRD2 (Dopamine) Likely lower DRD2 activity	! HIGHER ACTIVITY CHRNA4 (Smoking, Cognition) Predisposed to a higher CHRNA4 activity	! WORSE GAD1 (Glutamate/GABA) Likely worse GAD1 genetics
! LIKELY S/S 5-HTTLPR (Serotonin) Likely S/S genotype at 5HTTLPR	! LOWER ACTIVITY SLC6A4 (Fatigue, Mental Health) Likely lower SLC6A4 activity	! LOWER ACTIVITY HTR2C (Weight, Mental Health) Likely lower HTR2C activity
! HIGHER ACTIVITY HTR3A (Gut Health) Predisposed to a higher HTR3A activity	! LOWER ACTIVITY SLC18A1 (Mental Health) Predisposed to a lower SLC18A1 activity	✓ TYPICAL ACTIVITY COMT Likely typical COMT activity



TYPICAL ACTIVITY

TH (Mental Health,
Blood Pressure)

Predisposed to a typical TH activity



TYPICAL ACTIVITY

DBH (Mental Health)

Predisposed to typical DBH activity



TYPICAL ACTIVITY

SLC18A2 (Mental
Health)

Predisposed to typical SLC18A2 activity



TYPICAL ACTIVITY

SLC6A3 (Mental Health)

Predisposed to a typical SLC6A3 activity



TYPICAL ACTIVITY

CHRNA5 (Smoking,
Cognition)

Predisposed to typical CHRNA5 activity



TYPICAL LEVELS

IDO (Kynurenine)

Predisposed to typical IDO levels



TYPICAL ACTIVITY

KMO (Mental Health)

Predisposed to a typical KMO activity



TYPICAL ACTIVITY

TPH2 (Serotonin)

Predisposed to typical TPH2 activity



TYPICAL ACTIVITY

HTR2A (Serotonin)

Likely typical HTR2A activity



HIGHER ACTIVITY

HTR1A (Serotonin)

Likely higher HTR1A activity



TYPICAL ACTIVITY

CNR1 (Cannabinoids)

Likely typical CNR1 activity



TYPICAL ACTIVITY

CNR2 (Cannabinoids)

Predisposed to a typical CNR2 activity



LOWER ACTIVITY

FAAH (Cannabinoids)

Likely lower FAAH activity

MAOA (Dopamine/Serotonin)

There are multiple *MAOA* variants affecting enzyme activity. While low-activity variants lead to increased levels of the monoamine neurotransmitters dopamine, serotonin, and norepinephrine, variants with high activity decrease them. Among them, two of the most well-researched ones are [rs6323](#), [rs1137070](#), and [rs909525](#). Their “G”, “T”, and “C” alleles, respectively, encode MAOA proteins with higher activity [\[R\]](#).

The first low-activity variants described were associated with increased aggressiveness, which earned *MAOA* the nickname “the Warrior gene.” This received high media coverage and raised the ethical question of whether carriers of certain variants should be held fully responsible for their actions. In some cases, it even resulted in sentence reductions [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)!

Other conditions associated with lower MAOA activity include:

- Autism [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Schizophrenia [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Suicidal behavior [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Alcoholism [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Substance use disorder [\[R\]](#), [\[R\]](#)
- Obesity [\[R\]](#), [\[R\]](#), [\[R\]](#)

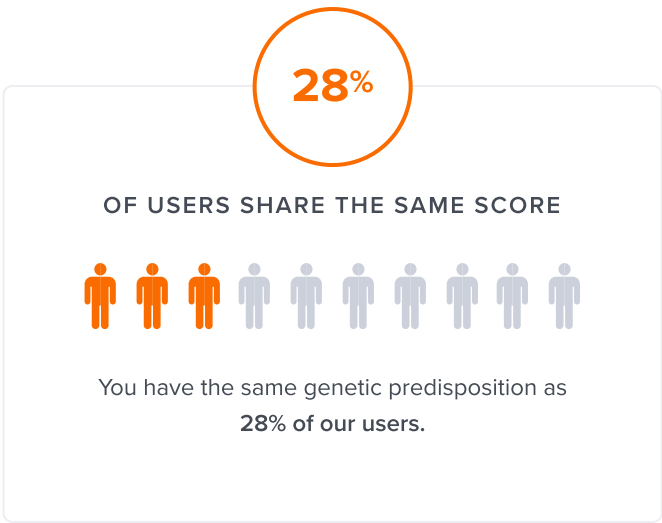
In contrast, variants with high activity lead to reduced dopamine, serotonin, and norepinephrine levels. These variants have been associated with the following conditions:

- Depression [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Panic disorder [\[R\]](#), [\[R\]](#)
- Obsessive-compulsive disorder [\[R\]](#), [\[R\]](#)
- ADHD [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Tourette syndrome [\[R\]](#), [\[R\]](#)
- Heavy smoking [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Parkinson’s disease [\[R\]](#), [\[R\]](#)
- Migraines [\[R\]](#), [\[R\]](#)
- Chronic fatigue syndrome [\[R\]](#)

Drugs that block MAOA can improve several of these conditions and are commonly prescribed for mood disorders. However, inhibitors of another monoamine oxidase version (MAOB) are



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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MAOA	rs6323	G
MAOA	rs1137070	T

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

preferred in the case of Parkinson’s disease because they are more effective for motor symptoms and brain cell death [\[R\]](#), [\[R\]](#).

MAOA also plays a role in **histamine metabolism**. It breaks down histamine by removing an amine group, primarily in brain and gut tissues. When MAOA activity is reduced, histamine remains active longer, potentially contributing to prolonged allergy symptoms or sensitivity reactions.

Nevertheless, keep in mind that the research on the health effects of *MAOA* variants is very complex, and often results in mixed or inconsistent findings. Factors that may modify the associations of specific *MAOA* variants with health conditions include:

- Their combination with other *MAOA* variants
- Many other genes
- Gender and sex hormone levels
- Environmental factors

MAOB (Dopamine/ Norepinephrine)

A higher amount of monoamine oxidase B implies lower monoamine levels (due to increased breakdown), and vice versa. *MAOB* variants with increased activity have been associated with [chronic fatigue syndrome](#) [R, R].

The main variants associated with this condition are [R]:

- ‘G’ at [rs3027452](#)
- ‘G’ at [rs2283729](#)
- ‘T’ at [rs1799836](#)

The ‘T’ allele of rs1799836 is also associated with higher anger [R].

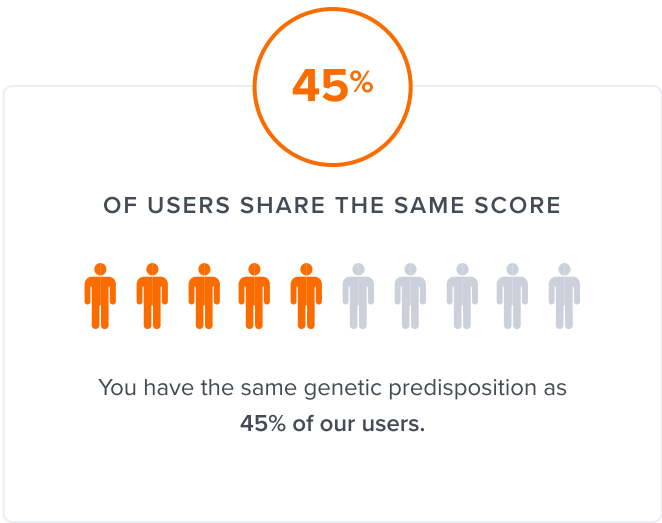
The ‘G’ allele of rs3027452 is linked to lower blood pressure and higher mood improvement in response to tryptophan treatment [R, R].

MAOB also plays a role in **histamine metabolism**. While it primarily targets other compounds, it is a backup system for histamine degradation when MAOA function is insufficient. It's crucial in the liver and certain brain regions where it can compensate for limited MAOA activity in processing excess histamine.



HIGHER ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MAOB	rs3027452	G
MAOB	rs2283729	G
MAOB	rs1799836	T

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

SLC18A1 (Mental Health)

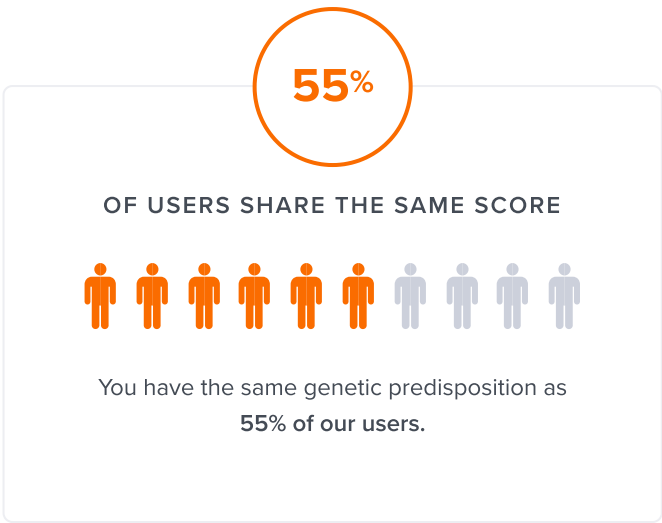
The main *SLC18A1* polymorphism is [rs1390938](#), commonly referred to as Thr136Ile. Its minor ‘A’ allele has been linked to higher VMAT1 and monoamine transport activity. This variant has been associated with a decreased risk of [\[R\]](#):

- Autism spectrum disorder [\[R\]](#)
- Anxiety, affective, and alcohol use disorders [\[R\]](#), [\[R\]](#)
- Severe alcohol withdrawal [\[R\]](#)
- Damaging neurological changes in alcoholics [\[R\]](#)
- Bipolar disorder [\[R\]](#)
- Low sperm motility [\[R\]](#)



LOWER ACTIVITY

Predisposed to a lower SLC18A1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SLC18A1	rs1390938	GG

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

DRD2 (Dopamine)

When it comes to the *DRD2* gene, the most studied variant is [rs1800497](#), also known as Taq1A. Interestingly, this variant is found in another gene, [ANKK1](#), which controls the activity of *DRD2* [\[R\]](#), [\[R\]](#), [\[R\]](#).

The **“A” (“A1”) allele** of this variant is linked to different pleasure-seeking behaviors, such as:

- Alcohol and substance misuse [\[R\]](#), [\[R\]](#), [\[R\]](#)
- [Food cravings and emotional eating](#) [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Other addictive behaviors [\[R\]](#)

This allele is linked to a **30%** lower number of D2 receptors in brain regions responsible for **motivation and reward**. People with this allele may **seek more pleasure** from alcohol, high-calorie foods, drugs, and other addictive substances and behaviors [\[R\]](#), [\[R\]](#), [\[R\]](#).

This variant may also be linked to [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#):

- Depression
- Negative feelings
- [Chronic pain](#)
- [PTSD](#)

On the positive side, people with this variant may be more motivated and have better [cognitive function](#) [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

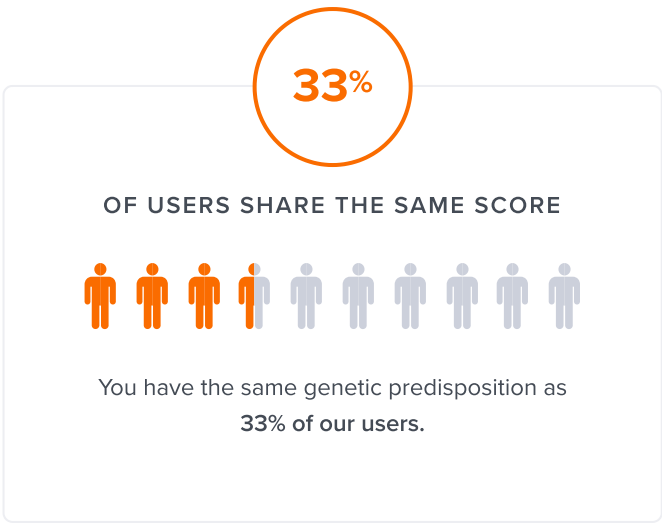
Due to the complex functions of D2 receptors, we can’t simply say if this variant increases or decreases dopamine. **Hence, people with this variant should try to maintain balanced dopamine levels.** Some natural ways to balance dopamine include [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#):

- Exercise [\[R\]](#)
- Relaxation [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Reduced sugar intake [\[R\]](#)



LOWER ACTIVITY

Likely lower DRD2 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ANKK1	rs1800497	GA

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

CHRNA4 (Smoking, Cognition)

The main *CHRNA4* polymorphism is [rs1044396](#). Its minor ‘A’ allele encodes a receptor with reduced binding efficiency [\[R\]](#).

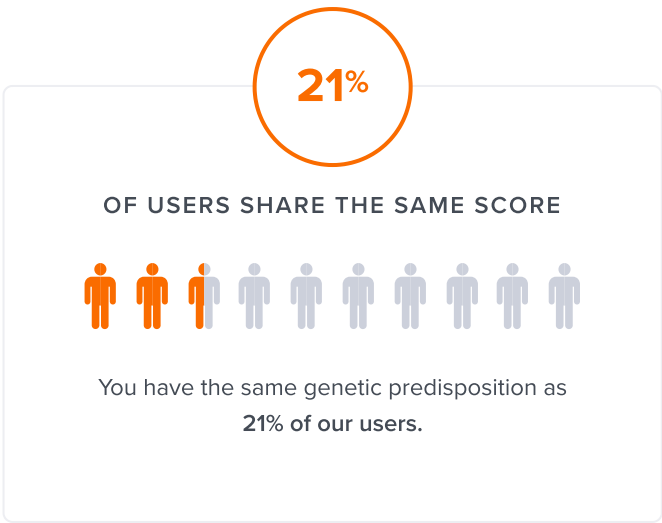
Carriers of this allele may have lower nicotine dependence and higher success rates of smoking cessation therapies. However, this allele has been associated with high odds of being a smoker in Parkinson’s disease patients [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

This variant has also been associated with better cognitive performance in tasks involving attention and processing speed. However, the evidence is mixed [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).



HIGHER ACTIVITY

Predisposed to a higher CHRNA4 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CHRNA4	rs1044396	GG

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

GAD1 (Glutamate/GABA)

The most well-researched *GAD1* polymorphism is [rs3749034](#). Its major ‘G’ allele has been shown to reduce GAD1 activity, resulting in lower GABA levels in the brain. In addition, this variant has been associated with an increased risk of [\[R\]](#):

- Schizophrenia [\[R, R, R\]](#)
- Hyperactive and impulsive symptoms in ADHD [\[R\]](#)

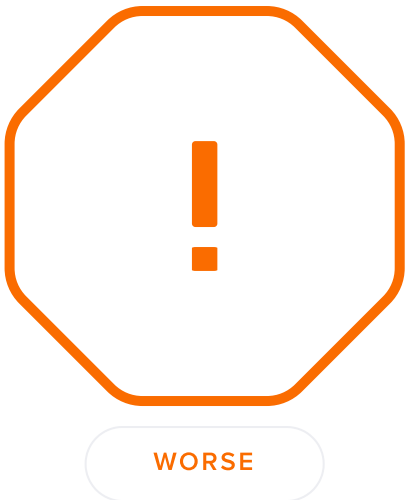
However, this variant has also been linked to lower odds of bipolar disorder and fewer respiratory symptoms in people with panic disorder [\[R, R\]](#).

Another variant associated with schizophrenia is the minor ‘A’ allele at [rs1978340](#). Surprisingly, this variant has been shown to reduce GABA levels. The fact that GABA acts as an inhibitory neurotransmitter in the mature brain but has the opposite activity during brain development may explain the complex effects of GABA levels on conditions such as schizophrenia and ADHD [\[R, R, R, R\]](#).

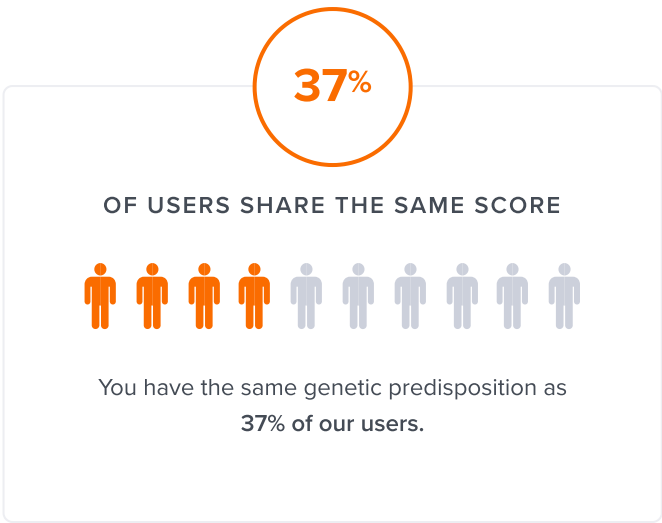
This variant has also been linked to:

- Cocaine dependence [\[R\]](#)
- Panic disorder and respiratory symptoms in this condition [\[R, R\]](#)
- Earlier onset of alcohol dependence [\[R\]](#)

However, this allele was associated with a reduced risk of heroin addiction [\[R\]](#).



Likely worse GAD1 genetics based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GAD1	rs3749034	GG
GAD1	rs1978340	GA

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

5-HTTLPR (Serotonin)

Depending on the presence or absence of a specific sequence, the 5HTTLPR variant can originate two different alleles [\[R\]](#):

- Short (S): lower activity of the gene
- Long (L) allele: 3-fold higher activity of the gene compared to the S allele

The S allele reduces the rate at which serotonin is recycled after a signal, ultimately lowering the circulating levels of this chemical [\[R\]](#).

This polymorphism has been widely investigated in psychiatry. Although the evidence is mixed in some cases, the S allele has been associated with an increased risk of:

- Major depressive disorder [\[R\]](#), [\[R\]](#)
- Postpartum depression [\[R\]](#)
- Depression in Parkinson’s disease [\[R\]](#)
- Depression in coronary heart disease [\[R\]](#)
- Post-stroke depression [\[R\]](#), [\[R\]](#)
- Geriatric depression [\[R\]](#)
- Illness anxiety (hypochondria) [\[R\]](#)
- PTSD [\[R\]](#)
- Bipolar disorder [\[R\]](#), [\[R\]](#)
- Alcohol abuse and dependence [\[R\]](#), [\[R\]](#)
- Anorexia nervosa [\[R\]](#)
- Suicide attempts [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Premature ejaculation [\[R\]](#), [\[R\]](#)
- Migraines [\[R\]](#)
- Canker sores [\[R\]](#)
- Antidepressant-induced mania [\[R\]](#)
- Failure of SSRI treatment [\[R\]](#)

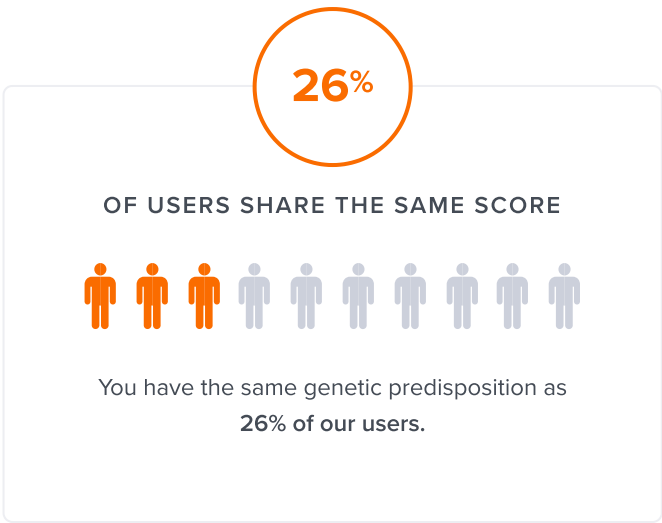
In turn, the L allele has been linked to:

- IBS, especially IBS-C [\[R\]](#), [\[R\]](#)
- Obstructive sleep apnea [\[R\]](#)

Different combinations of the [rs2129785](#) and [rs11867581](#) polymorphisms of this gene are usually inherited together with the S and L alleles and can be used to predict the genotype of this variant. While carrying the ‘T’ variant at rs2129785 and ‘A’ at rs11867581 predicts the S allele in 91% of cases, 96% of people carrying ‘T’ at rs2129785 and ‘G’ at rs11867581 and 100% of



Likely S/S genotype at 5HTTLPR based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
BLMH	rs2129785	TT
BLMH	rs11867581	AA

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

those with ‘C’ at rs2129785 and ‘A’ at rs11867581 have the L allele [\[R\]](#).

Other variants of this gene have been associated with [chronic fatigue syndrome](#) [\[R\]](#), [\[R\]](#).

Note: This report doesn’t directly look into the long and short 5HTTLPR alleles, but estimates your genotype based on two polymorphisms that are usually inherited together with these variants. Hence, your predicted genotype may be inaccurate and depend on the testing company from which you uploaded your DNA results.

SLC6A4 (Fatigue, Mental Health)

Two *SLC6A4* variants, ‘G’ at [rs2066713](#) and ‘T’ at [rs140701](#), have been associated with higher rates of chronic fatigue syndrome [\[R\]](#).

The rs2066713-G variant has also been associated with:

- Increased risk of schizophrenia [\[R\]](#)
- Increased risk of migraines with aura [\[R\]](#)
- Increased risk of nicotine dependence [\[R\]](#)
- Decreased risk of alcohol use disorder [\[R\]](#)
- Decreased risk of autism [\[R\]](#)
- Decreased risk of type 2 diabetes [\[R\]](#)

In turn, the rs140701-T allele has been associated with:

- Increased anxiety and risk of panic disorder and social anxiety disorder [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Increased risk of breast cancer [\[R\]](#)

These variants may reduce SLC6A4 activity. As a result, they may increase the brain’s sensitivity to both good and bad environmental changes [\[R\]](#), [\[R\]](#).



Likely lower SLC6A4 activity based on 2 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
BLMH	rs140701	TT
BLMH	rs2066713	GG

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

HTR2C (Weight, Mental Health)

The best-characterized *HTR2C* polymorphism is [rs3813929](#). Its minor ‘T’ allele may increase gene expression, leading to a higher density of 5-HT2C receptors. This allele has been associated with [\[R\]](#):

- Increased risk of major depressive disorder [\[R\]](#)
- Increased risk of type 2 diabetes, impaired glucose tolerance, and heart disease [\[R\]](#)
- Higher rates of migraines [\[R\]](#)
- Lower weight gain in response to antipsychotics [\[R\]](#), [\[R\]](#)
- Decreased risk of ADHD [\[R\]](#)

Another allele increasing *HTR2C* expression, ‘C’ at [rs518147](#), has been associated with:

- Lower weight gain in response to antipsychotics [\[R\]](#)
- Decreased risk of ADHD [\[R\]](#)
- Lower likelihood of smoking [\[R\]](#)

Several other variants have been found to increase 5-HT2C activity and decrease BMI, appetite, and antipsychotic-induced weight gain:

- ‘T’ at [rs3813928](#) [\[R\]](#)
- ‘G’ at [rs6318](#) [\[R\]](#)
- ‘A’ at [rs498207](#) [\[R\]](#)
- ‘G’ at [rs498177](#) [\[R\]](#)



Likely lower HTR2C activity based on 6 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HTR2C	rs3813929	C
HTR2C	rs518147	G
HTR2C	rs3813928	G
HTR2C	rs6318	G
HTR2C	rs498207	A
HTR2C	rs498177	G

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

HTR3A (Gut Health)

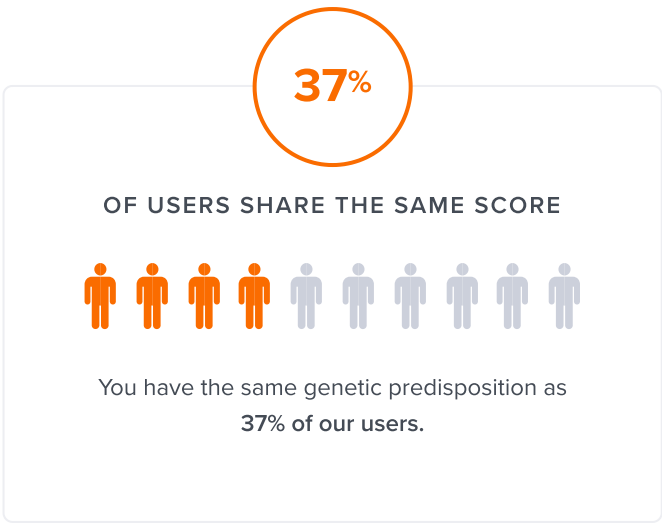
In theory, variants in the 5-HT3 receptor that increase its activity could increase movement in the gut and increase susceptibility to IBS.

One variant in the *HTR3A* gene has been directly linked to IBS. At [rs1062613](#), the minor ‘T’ allele was associated with increased rates of IBS and increased receptor activation [\[R, R\]](#).



HIGHER ACTIVITY

Predisposed to a higher HTR3A activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NNMT	rs1062613	CT

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

SLC18A1 (Mental Health)

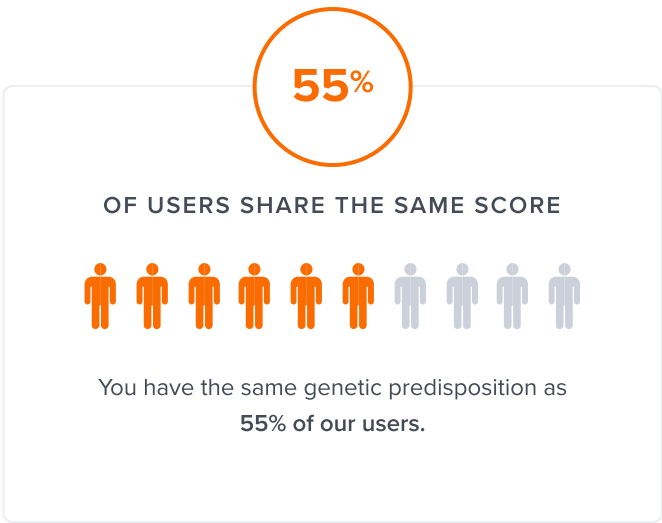
The main *SLC18A1* polymorphism is [rs1390938](#), commonly referred to as Thr136Ile. Its minor ‘A’ allele has been linked to higher VMAT1 and monoamine transport activity. This variant has been associated with a decreased risk of [\[R\]](#):

- Autism spectrum disorder [\[R\]](#)
- Anxiety, affective, and alcohol use disorders [\[R\]](#), [\[R\]](#)
- Severe alcohol withdrawal [\[R\]](#)
- Damaging neurological changes in alcoholics [\[R\]](#)
- Bipolar disorder [\[R\]](#)
- Low sperm motility [\[R\]](#)



LOWER ACTIVITY

Predisposed to a lower SLC18A1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SLC18A1	rs1390938	GG

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

COMT

One common variant of the *COMT* gene, [rs4680](#), may affect COMT enzyme activity. Some people call rs4680 the “worrier or warrior” variant [\[R\]](#), [\[R\]](#).

The “G” allele of this variant is linked to a higher COMT enzyme activity. People with two copies of this allele (GG) have been nicknamed the “warriors.” They break down stress-related chemical messengers more quickly. This may help improve their performance under stress [\[R\]](#).

On the negative side, “warriors” may have lower cognitive performance under relaxed conditions [\[R\]](#), [\[R\]](#), [\[R\]](#).

People with two copies of the “A” allele (AA) may have lower COMT enzyme activity. They have been nicknamed the “worriers.” They break down stress-related chemical messengers more slowly in the brain. For this reason, they may be more vulnerable to stress. This includes an increased susceptibility to heart disease, possibly due to the effects of these chemical messengers on blood pressure and heart rate [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

The good news is that "worriers" may become more emotionally resilient with age. They also tend to have enhanced cognitive performance under relaxed conditions. Interestingly, "worriers" seem to have a more pronounced placebo response due to higher dopamine levels [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

People carrying both alleles (AG) tend to be in between the described extremes [\[R\]](#), [\[R\]](#).

Did you know? People with “warrior” genetics may be more likely to engage in combat sports, justifying the nickname of this variant [\[R\]](#).

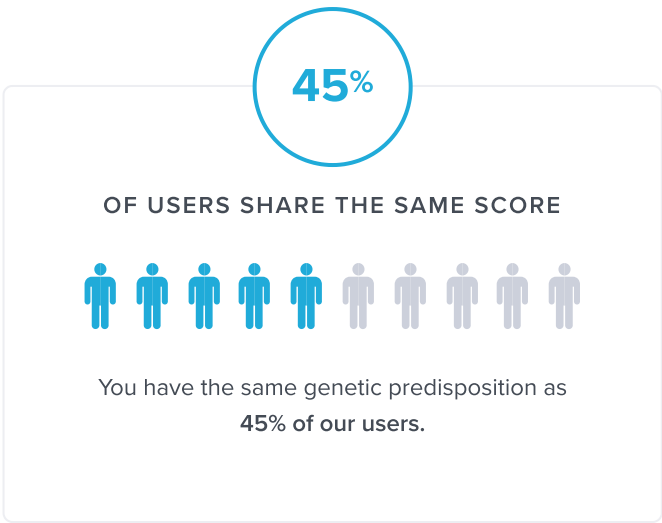
However, keep in mind that your cognitive function and response to stress are also influenced by other factors, such as:

- Other variants in the *COMT* gene
- Many other genes
- Environmental factors



TYPICAL ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
COMT	rs4680	AG

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

TH (Mental Health, Blood Pressure)

The best-characterized *TH* polymorphism is [rs6356](#), whose minor ‘T’ allele may increase enzyme activity. This variant has been associated with:

- Increased risk of late-onset Parkinson’s disease [\[R\]](#)
- Increased risk of schizophrenia [\[R\]](#)
- Increased risk of migraines [\[R\]](#)
- Increased risk of cognitive and attentional impulsivity in maltreated children [\[R\]](#)
- Increased risk of hypertension [\[R\]](#)
- Higher SHGB levels [\[R\]](#)

Another variant, the ‘A’ allele of [rs10770141](#), may increase *TH* expression. This variant has been associated with [\[R\]](#):

- Increased risk of schizophrenia [\[R\]](#)
- Higher IQ in people with schizophrenia [\[R\]](#)
- Better cognitive control in case of consciously perceived conflicts [\[R\]](#)
- Lower novelty seeking [\[R\]](#)
- Lower persistence in patients with chronic fatigue syndrome [\[R\]](#)
- Increased risk of opioid dependence [\[R\]](#)
- Increased sensitivity to both “good” and “bad” effects of cocaine [\[R\]](#)
- Higher blood pressure in response to cold stress [\[R\]](#)
- Decreased risk of retinopathy of prematurity [\[R\]](#)

This allele is usually inherited together with the ‘C’ allele of [rs10770140](#), which also increases *TH* expression and has been associated with an increased risk of opioid dependence [\[R\]](#), [\[R\]](#).

Finally, the ‘G’ allele of [rs2070762](#) may also increase *TH* expression and has been associated with:

- Increased risk of pre-eclampsia [\[R\]](#)
- Increased risk of hypertension [\[R\]](#), [\[R\]](#)
- Increased risk of migraines [\[R\]](#)
- Increased risk of mild arm pain [\[R\]](#)
- Increased risk of cocaine and opioid dependence [\[R\]](#), [\[R\]](#)
- Greater body height [\[R\]](#)



Predisposed to a typical TH activity based on 4 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
C11ORF21	rs2070762	GG
TH	rs10770141	AG
TH	rs6356	CC
TH	rs10770140	TT

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

DBH (Mental Health)

The best-characterized *DBH* polymorphism is [rs1611115](#). Its ‘T’ allele may decrease DBH production, resulting in higher dopamine and lower norepinephrine levels. This allele has been associated with [\[R\]](#):

- Increased neuroticism, novelty seeking, impulsivity, and aggression [\[R\]](#)
- Decreased conscientiousness [\[R\]](#)
- Increased risk of adult ADHD [\[R\]](#)
- Decreased risk of migraines but more frequent migraine-related side effects [\[R\]](#)

Another variant leading to lower DHB levels, ‘A’ at [rs1108580](#), has been associated with [\[R\]](#):

- Increased risk of ADHD [\[R\]](#), [\[R\]](#)
- Increased risk of schizophrenia [\[R\]](#)
- Decreased risk of alcohol dependence [\[R\]](#)

The following, less well-researched variants have also been associated with lower DHB levels and increased risk of ADHD:

- ‘T’ of [rs2519154](#) [\[R\]](#), [\[R\]](#)
- ‘C’ of [rs2873804](#) [\[R\]](#), [\[R\]](#)
- ‘T’ of [rs2519152](#) [\[R\]](#), [\[R\]](#), [\[R\]](#)
- ‘C’ of [rs1076150](#) [\[R\]](#), [\[R\]](#)



TYPICAL ACTIVITY

Predisposed to typical DBH activity based on 6 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
DBH	rs2519154	TT
DBH	rs2873804	CC
DBH	rs1076150	CC
DBH	rs2519152	CT
DBH	rs1611115	CC
DBH	rs1108580	GG

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

SLC18A2 (Mental Health)

Several *SLC18A2* polymorphisms resulting in decreased VMAT2 levels have been associated with different mental health issues. These include:

- The ‘T’ allele of [rs363276](#): associated with an increased risk of PTSD [\[R\]](#), [\[R\]](#).
- The ‘T’ allele of [rs363387](#): associated with an increased risk of alcohol and nicotine dependence, as well as with poorer inhibitory control [\[R\]](#), [\[R\]](#), [\[R\]](#).
- The ‘G’ allele of [rs363324](#): associated with an increased risk of Parkinson’s disease [\[R\]](#), [\[R\]](#).
- The ‘C’ allele of [rs363227](#): associated with an increased risk of methamphetamine use disorder but a decreased risk of psychotic disorders [\[R\]](#), [\[R\]](#).
- The ‘G’ allele of [rs363371](#): associated with an increased risk of Parkinson’s disease and schizophrenia but a decreased risk of ALS [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).



Predisposed to typical SLC18A2 activity based on 5 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SLC18A2	rs363387	TT
PDZD8	rs363227	CC
PDZD8	rs363324	GA
PDZD8	rs363371	AG
SLC18A2	rs363276	CC

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

SLC6A3 (Mental Health)

Several *SLC6A3* variants have been associated with an increased risk of ADHD, as well as increased impulsivity and inattention in individuals with ADHD. These include [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#):

- ‘T’ of [rs6347](#)
- ‘G’ of [rs11564750](#)
- ‘C’ of [rs27072](#)
- ‘T’ of [rs2550948](#)
- ‘C’ of [rs27048](#)

While the mechanism behind this association is not clear, these variants may increase the density of the dopamine transporter in certain regions of the brain. This would decrease the amount of dopamine available in these brain regions, giving rise to ADHD and its associated symptoms [\[R\]](#), [\[R\]](#).



Predisposed to a typical SLC6A3 activity based on 5 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CLPTM1L	rs11564750	GG
LPCAT1	rs27072	CC
SLC6A3	rs6347	TT
SLC6A3	rs2550948	CT
SLC6A3	rs27048	TT

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

CHRNA5 (Smoking, Cognition)

The following [CHRNA5](#) variants are linked to **smoking, nicotine dependence, and different lung conditions** [\[R, R, R, R, R\]](#):

- [rs16969968](#)-A
- [rs588765](#)-C
- [rs637137](#)-T
- [rs1051730](#)-A (located in a nearby [CHRNA3](#) gene)

At the same time, these variants are linked to **reduced cognitive function** [\[R, R\]](#).

These variants may **reduce CHRNA5** (and CHRNA3) activity, impairing receptor function and **lower acetylcholine levels** in brain regions responsible for cognition. Since nicotine activates these receptors and temporarily boosts cognition, people with these variants tend to smoke more and be more addicted to nicotine [\[R, R, R, R, R\]](#).

It’s important to note that nicotine only provides short-term cognitive benefits. In the long run, nicotine, especially smoking, impairs cognitive function [\[R, R\]](#).



TYPICAL ACTIVITY

Predisposed to typical CHRNA5 activity based on 4 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CHRNA5	rs637137	TT
CHRNA5	rs16969968	GA
CHRNA5	rs588765	TC
CHRNA3	rs1051730	GA

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

IDO (Kynurenine)

Factors influencing IDO activity include:

- Inflammatory responses: Certain cytokines released during inflammation can induce IDO production.
- Stress and psychological factors: Stress and other psychological factors can influence the kynurenine pathway, potentially affecting IDO activity.
- Diet and nutrition: The availability of tryptophan, the substrate for IDO, can influence its activity.

A few genetic variants have been associated with IDO levels.
Genetically higher IDO levels may protect against the following conditions [\[R\]](#), [\[R\]](#), [\[R\]](#):

- Ischemic heart disease
- Type 2 diabetes
- Prostate cancer



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

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NLRP12	rs4632248	GG
IDO1	rs7010461	TC
MST1	rs3197999	GG

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

KMO (Mental Health)

One of the main *KMO* polymorphisms is [rs1053230](#). Its ‘T’ allele may increase *KMO* expression and activity, at least in people with psychiatric disorders. This variant has been associated with an increased risk of [\[R\]](#), [\[R\]](#), [\[R\]](#):

- Postpartum depression [\[R\]](#)
- Suicidal attempts [\[R\]](#)

In contrast, the major ‘C’ allele is the one that has been associated with

- Depression [\[R\]](#), [\[R\]](#)
- Schizophrenia [\[R\]](#), [\[R\]](#)
- Bipolar disorder [\[R\]](#)
- Substance (cannabis and cocaine) use disorder [\[R\]](#)

Another variant, the ‘T’ allele of [rs2275163](#), may decrease *KMO* expression and has been associated with an increased risk of schizophrenia but better cognitive performance in people with this condition. This variant has also been associated with an increased risk of bipolar disorder and substance use (cannabis, cocaine, and opioid) use disorder [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).



TYPICAL ACTIVITY

Predisposed to a typical KMO activity based on 2 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
KMO	rs1053230	CT
KMO	rs2275163	CC

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

TPH2 (Serotonin)

The [rs4570625](#) variant has been most widely investigated. Its minor ‘T’ allele is believed to reduce TPH2 production, resulting in lower serotonin levels. Carriers of this variant show higher activity in the part of the brain responsible for stress and fear processing (the *amygdala*) in response to emotional stimuli [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

The ‘T’ variant has also been associated with higher [GABA](#) levels. GABA is the main inhibitory neurotransmitter preventing over-stimulation of neurons. An abnormal GABA decrease can produce mental illness and symptoms such as anxiety, depression, insomnia, convulsions, and epilepsy [\[R\]](#), [\[R\]](#), [\[R\]](#).

The link of this variant with both low serotonin and high GABA levels may explain its complex association with anxiety, mood, and fatigue. This variant has been associated with a decreased risk of:

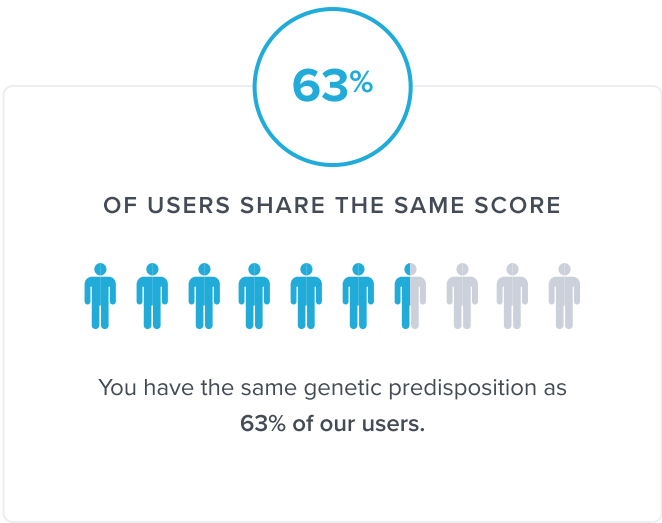
- [Anxiety](#) disorders such as panic disorder [\[R\]](#)
- Severe [ADHD](#) symptoms and poor sustained attention in healthy individuals [\[R\]](#)
- Early-onset OCD [\[R\]](#)
- Depression [\[R\]](#)
- Suicide attempts [\[R\]](#)
- Schizophrenia [\[R\]](#)
- Failure of placebo treatments to improve anxiety [\[R\]](#)

However, it increases [fatigue](#) in women with IBS [\[R\]](#).



TYPICAL ACTIVITY

Predisposed to typical TPH2 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TPH2	rs4570625	GG

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

HTR2A (Serotonin)

The most widely investigated variant is [rs6313](#). Its minor ‘A’ allele increases the number of active receptors. This variant has been associated with an increased risk of [\[R\]](#), [\[R\]](#):

- [Chronic pain](#), including hip and low back pain [\[R\]](#), [\[R\]](#)
- [Panic disorder](#) [\[R\]](#)

Carriers may also require higher doses of anesthetic drugs (propofol) and longer times to induce sedation [\[R\]](#).

However, it may be protective against:

- [Fatigue disorders](#) such as chronic fatigue syndrome and fibromyalgia [\[R\]](#)
- [Headaches](#) [\[R\]](#)
- IBS [\[R\]](#)
- Temporomandibular joint disorders [\[R\]](#)
- Suicide attempts [\[R\]](#)

Another well-researched variant is [rs6311](#). Its minor ‘T’ variant is usually inherited together with the ‘A’ variant at rs6313 and also increases the number of active 5HT2A receptors. This variant has been associated with [\[R\]](#), [\[R\]](#):

- Increased odds of chronic pain, including hip and low back pain [\[R\]](#), [\[R\]](#)
- Greater depressive symptoms [\[R\]](#)
- Increased risk of IBS [\[R\]](#)

However, this variant may be protective against:

- Headaches [\[R\]](#)
- Rheumatoid arthritis [\[R\]](#)

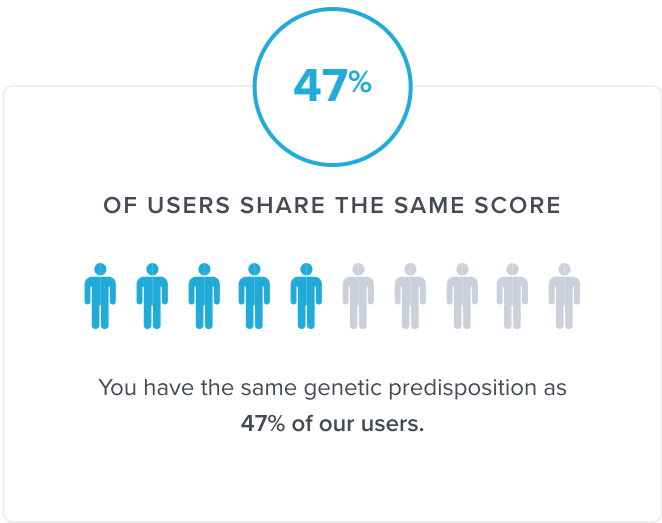
Interestingly, this variant has been associated with decreased aggressiveness and social dominance. This was linked to being a worse [speed dater](#) in men but better in women [\[R\]](#).

Finally, two variants believed to impair serotonin signaling (‘A’ at [rs7322347](#) and ‘C’ at [rs7984966](#)) have been associated with an increased risk of [ADHD](#) [\[R\]](#), [\[R\]](#).



TYPICAL ACTIVITY

Likely typical HTR2A activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HTR2A	rs6313	GA
HTR2A	rs6311	CT

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

HTR1A (Serotonin)

The most widely-investigated *HTR1A* variant is [rs6295](#). Its minor ‘C’ allele leads to higher expression of 5HT1A autoreceptors, a lower expression of postsynaptic receptors, and decreased overall activity in serotonin neurons [\[R\]](#), [\[R\]](#).

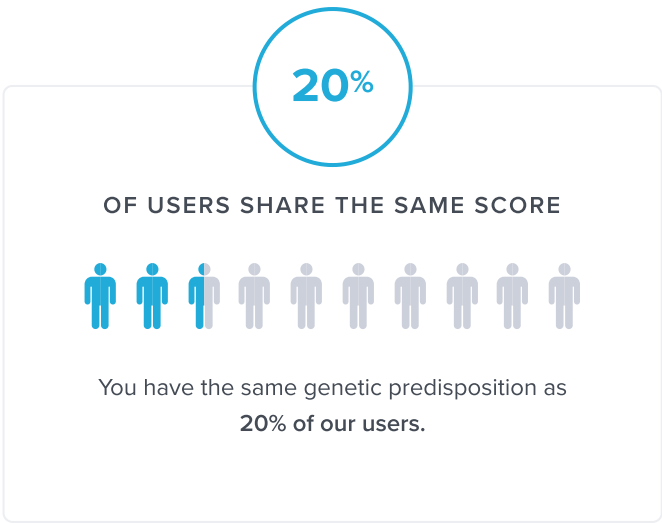
This variant has been associated with:

- Decreased self-awareness or emotional intelligence ([alexithymia](#)), especially in males with early-life stress [\[R\]](#), [\[R\]](#)
- Decreased comfort with [close relationships](#) and lower odds of being in a romantic relationship [\[R\]](#)
- Higher susceptibility to anxiety and depression from stressful events [\[R\]](#)
- Increased impulsiveness and aggression [\[R\]](#)
- Higher odds of ADHD [\[R\]](#)
- Impaired working memory in premenstrual women [\[R\]](#)
- Increased pain perception in response to intense stimuli (but decreased perception of mild pain) [\[R\]](#)



HIGHER ACTIVITY

Likely higher HTR1A activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
RNF180	rs6295	GG

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

CNR1 (Cannabinoids)

The most studied SNP in this gene is [rs1049353](#) or 1359G/A. Its minor ‘T’ allele doesn’t change the CB1 receptor structure, but is believed to impair gene expression and receptor activity [\[R\]](#), [\[R\]](#). This variant has been associated with:

- Lower BMI, belly fat, and [weight gain](#) [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Increased risk of major depression and PTSD but better response to antidepressants [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Increased [risk of brain fog](#)
- Reduced risk of risky gambling, cannabis use, and heroin addiction [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Increased risk of [lectin sensitivity](#)
- Reduced risk of ulcerative colitis and young-onset Crohn’s disease but worse gut inflammation in people with IBD [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Reduced risk of heart disease in people with type 2 diabetes [\[R\]](#), [\[R\]](#)

Another polymorphism, ‘G’ at [rs6454674](#), seems to lower blood CB1 levels. This variant has been linked to [\[R\]](#):

- Increased odds of childhood obesity [\[R\]](#)
- Increased susceptibility to anxiety disorders and, possibly, depression [\[R\]](#)
- Increased dependence to alcohol, cannabis, and cocaine [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)

The minor ‘C’ allele of [rs2023239](#) seems to increase CB1 levels, especially in people using marijuana. This variant has been associated with [\[R\]](#), [\[R\]](#), [\[R\]](#):

- Decreased odds of childhood obesity [\[R\]](#)
- Increased impulsivity, cigarette smoking, and cannabis rewarding, craving, and withdrawal [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Decreased odds of major depression in opiate-dependent outpatients on methadone therapy [\[R\]](#)
- Increased risk of metabolic syndrome [\[R\]](#)

The minor ‘T’ variant of [rs806378](#) is believed to increase CNR1 activity. This allele has been associated with [\[R\]](#), [\[R\]](#):

- Faster colonic transit and gassiness in people with diarrhea-predominant IBS [\[R\]](#), [\[R\]](#)
- Weight gain in people treated with antipsychotics [\[R\]](#)



Likely typical CNR1 activity based on 7 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CNR1	rs1049353	TT
CNR1	rs806378	CC
CNR1	rs2180619	GG
CNR1	rs806380	AA
CNR1	rs806374	TT
CNR1	rs6454674	TT
CNR1	rs2023239	CC

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

Another minor variant believed to decrease CB1 levels is ‘G’ at [rs806380](#). This variant has been associated with:

- Decreased risk of unsafe sex behavior, alcohol use, and cannabis dependence [[R](#), [R](#)]
- Repeated episodes of severe nausea and vomiting [[R](#)]

The minor ‘C’ variant of [rs806374](#), which seems to reduce CB1 levels in the gut and fatty tissues, has been linked to frequent marijuana use around the age of 18 [[R](#), [R](#)].

Finally, a variant that seems to decrease CB1 levels in the brain, ‘G’ at [rs2180619](#), has been linked to substance abuse in European Americans but not in African Americans [[R](#), [R](#)].

CNR2 (Cannabinoids)

The most studied SNP in this gene is [rs2501432](#) or Q63R. Its minor ‘C’ allele encodes a version of the protein that is less responsive to activation by 2-AG [\[R\]](#), [\[R\]](#).

This variant has been associated with an increased risk of [\[R\]](#):

- Depression [\[R\]](#)
- Schizophrenia [\[R\]](#), [\[R\]](#)
- Low bone mineral density [\[R\]](#)

Another variant with decreased binding affinity for 2-AG, the minor ‘A’ allele of [rs2229579](#) (Tyr316His), has been associated with an increased risk of:

- Synthetic cannabinoid use disorder [\[R\]](#)
- Osteoporosis of the lumbar spine/femoral neck [\[R\]](#)
- Schizophrenia [\[R\]](#)

Finally, the ‘T’ allele of [rs41311993](#) (Leu133Ile) may reduce the ability of the receptor to trigger β -arrestin-2 signaling and has been associated with an increased risk of bipolar disorder [\[R\]](#), [\[R\]](#).



TYPICAL ACTIVITY

Predisposed to a typical CNR2 activity based on 3 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CNR2	rs2501432	TC
CNR2	rs41311993	GG
CNR2	rs2229579	GG

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

FAAH (Cannabinoids)

One common variant of the *FAAH* gene, [rs324420](#), is associated with reduced FAAH levels and activity. As a result, carriers of the minor ‘A’ allele have higher levels of endocannabinoids such as anandamide [\[R\]](#).

Carriers of this variant may have:

- [Lower anxiety in response to stressful situations](#) [\[R\]](#)
- [Lower odds of PTSD](#) [\[R\]](#)
- [Decreased pain sensitivity](#) [\[R\]](#)
- Better athletic performance [\[R\]](#), [\[R\]](#)

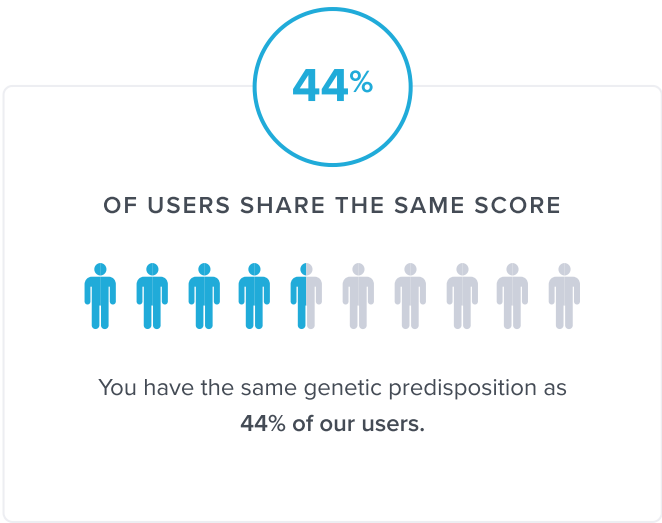
However, this variant is also linked to an increased risk of:

- [ARDS](#) [\[R\]](#)
- Obesity (especially early-onset) [\[R\]](#), [\[R\]](#)
- Antipsychotic-induced weight gain [\[R\]](#)
- Problematic drug and alcohol use [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Generalized epilepsy [\[R\]](#)
- Heart attack [\[R\]](#)



LOWER ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
FAAH	rs324420	CA

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



Neuronal Plasticity & Stress Response

Neuronal plasticity is the brain’s ability to adapt and reorganize itself, which is essential for learning, memory, and recovery from stress. How your body responds to stress is also influenced by genetics, affecting mood, resilience, and overall mental well-being.

Genes like *BDNF* and *KIBRA* are key regulators of brain plasticity and cognitive function. *NR3C1*, *MR*, *CRHR1*, *CRHR2*, and *FKBP5* encode receptors that mediate the effects of stress hormones like cortisol. Understanding these genetic factors can help you better comprehend your stress resilience and cognitive health.

SLIGHTLY LOWER LEVELS BDNF Relatively lower BDNF levels	LOWER ACTIVITY ENPP6 (Cognition) Likely lower ENPP6 activity	LOWER ACTIVITY ULK4 (Cognition) Likely lower ULK4 activity
LOWER ACTIVITY CRHR2 (Stress/HPA Axis) Likely lower CRHR2 activity	TYPICAL ACTIVITY LMX1A (Cognition) Likely typical LMX1A activity	INTERMEDIATE ACTIVITY NR3C1 (Fatigue) Likely intermediate NR3C1 activity
LOWER ACTIVITY CRHR1 (Stress/HPA Axis) Likely lower CRHR1 activity	LOWER ACTIVITY FKBP5 (Stress/ HPA Axis) Likely lower FKBP5 activity	HIGHER ACTIVITY KIBRA/WWC1 (Cognition) Likely higher KIBRA/WWC1 activity
HIGHER ACTIVITY TUSC3 (Cognition) Likely higher TUSC3 activity		

BDNF

The [BDNF](#) gene helps produce BDNF and strongly impacts its levels and activity [\[R\]](#).

A crucial *BDNF* gene variant is [rs6265](#), also known as “[Val66Met](#)”. It may affect BDNF production, storage, and release in brain cells [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

As a result, the “T” (“Met”) allele is linked to reduced cognitive function, including [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#):

- Learning difficulties
- Poor memory
- Dementia

Besides cognitive effects, this variant may also play a role in [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#):

- [Stress and anxiety](#)
- [PTSD](#) and [OCD](#)
- [Weight control](#)
- [Migraines](#)
- [Fatigue](#)

Moreover, the "T" variant may impair response to the antidepressant effects of low-dose ketamine. Nevertheless, this variant may not affect the effectiveness of ketamine for treatment-resistant depression [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

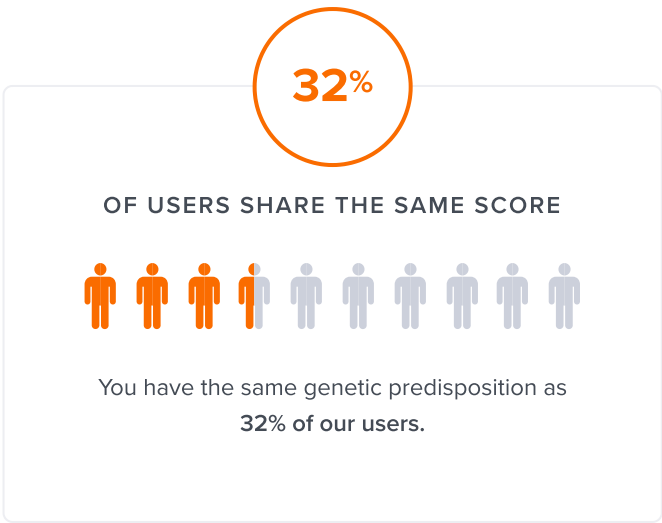
However, you should keep in mind some **important limitations** [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#):

- The effects of this variant on some traits are conflicting.
- Many studies looking into the cognitive effects of this variant are limited to people with mental health problems.
- The link between this variant and some conditions, such as OCD and dementia, may be significant only in women.
- **Your other genetic variants, lifestyle, and environment may also influence your BDNF levels and activity.**



SLIGHTLY LOWER LEVELS

Relatively lower BDNF levels based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
BDNF	rs6265	TC

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

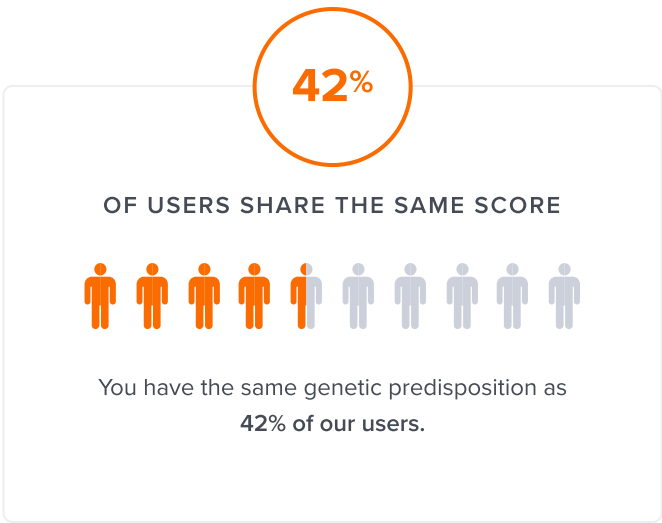
ENPP6 (Cognition)

SNPs in the *ENPP6* gene (such as [rs4241784](#)) have also been associated with more specific individual cognitive functions, such as **fluid intelligence**, **processing speed**, and **learning and memory**. The ‘T’ allele of this polymorphism is associated with enhanced cognitive ability due to increased myelination throughout the brain [[R](#), [R](#), [R](#)].



LOWER ACTIVITY

Likely lower ENPP6 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ENPP6	rs4241784	GG

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

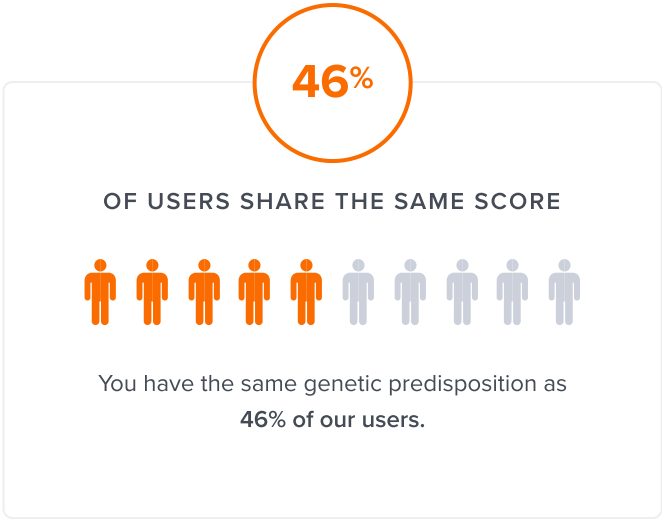
ULK4 (Cognition)

The main *ULK4* polymorphism when it comes to cognitive function is [rs7627367](#). Its major ‘G’ allele has been linked to **a reduced *ULK4* activity**. This, in turn, is associated with **relatively lower fluid intelligence** compared to people who carry the beneficial ‘T’ allele [\[R\]](#).



LOWER ACTIVITY

Likely lower ULK4 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ULK4	rs7627367	GG

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

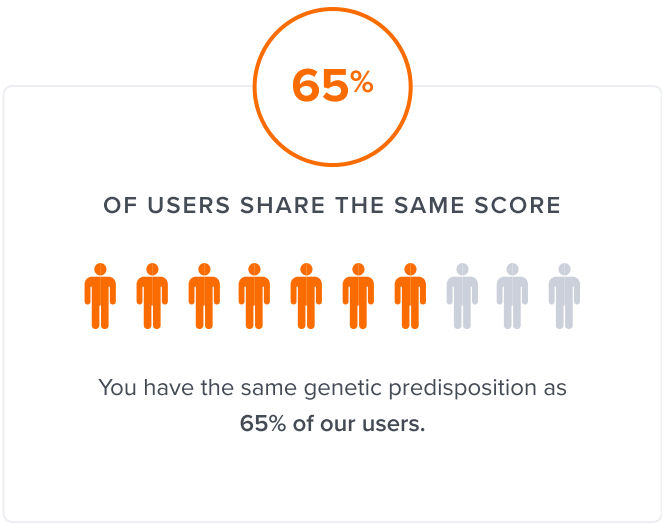
CRHR2 (Stress/HPA Axis)

A study of 491 veterans exposed to trauma and their partners associated the ‘A’ allele of [rs2267715](#) and [rs2190242](#) with an increased risk of [PTSD](#) in women. The study speculated that both variants may decrease *CRHR2* activity, thereby stimulating the stress response [\[R\]](#).

The rs2267715 variant was associated with PTSD symptom severity in a study of 1132 earthquake survivors [\[R\]](#).



Likely lower CRHR2 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CRHR2	rs2267715	AA
CRHR2	rs2190242	AA

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

LMX1A (Cognition)

A gene-targeted study in 218 participants found that the ‘T’ allele of [rs11809911](#) was associated with reduced performance on a wide variety of cognitive tests, including tests of learning and memory, cognitive flexibility (executive function and attention), processing speed, and even general intelligence (IQ) [\[R\]](#).

Although this particular study only reported this effect for bipolar patients, this result also agrees with several other studies linking risk factors in the *LMX1A* gene to differences in cognitive performance among otherwise healthy people.

For example, people with two copies of another variant (‘A’ at [rs4657412](#)) show enhanced performance on tests of working memory, and also achieve higher levels of improvement in their working memory after going through cognitive training programs [\[R, R\]](#)!



Likely typical LMX1A activity based on 2 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
LMX1A	rs11809911	TC
LMX1A	rs4657412	AG

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

NR3C1 (Fatigue)

Variants of *NR3C1* have been associated with chronic fatigue syndrome, an illness characterized by prolonged low energy. These variants may lower the sensitivity or activity of the glucocorticoid receptor, thus resulting in dysregulated HPA axis activity, excess cortisol release, and inflammation. They include [R](#), [R](#), [R](#), [R](#):

- ‘A’ at [rs852977](#) [R](#), [R](#), [R](#)
- ‘A’ at [rs1866388](#) [R](#), [R](#), [R](#)
- ‘C’ at [rs6188](#) [R](#), [R](#), [R](#)
- ‘T’ at [rs2918419](#) [R](#), [R](#), [R](#)
- ‘A’ at [rs6196](#) [R](#), [R](#), [R](#)
- ‘C’ at [rs41423247](#) [R](#), [R](#)



INTERMEDIATE ACTIVITY

Likely intermediate NR3C1 activity based on 5 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NR3C1	rs2918419	TT
NR3C1	rs6196	AA
NR3C1	rs852977	GA
NR3C1	rs1866388	GA
NR3C1	rs6188	AC

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

CRHR1 (Stress/HPA Axis)

Two variants, ‘A’ at [rs12938031](#) and ‘C’ at [rs4792887](#), have been associated with increased odds and severity of [PTSD](#). These variants are believed to increase *CRHR1* expression or activity [\[R, R\]](#).

Another variant believed to increase CRHR1 activity, ‘G’ at [rs12944712](#), has been associated with acute PTSD symptoms in children. However, the ‘A’ allele at this polymorphism is the one associated with [depression](#) [\[R, R\]](#).

The ‘G’ variant of [rs17689882](#), which may also increase CRHR1 activity, has been associated with depression due to childhood trauma in young adulthood [\[R\]](#).

The ‘A’ variant of [rs110402](#) is believed to decrease *CRHR1* expression. This variant has been associated with relatively higher rates of [chronic fatigue](#). However, this variant may protect from depression linked to childhood abuse and [IBS](#). In IBS patients, the ‘A’ variant is associated with predomination of diarrhea in men and constipation or mixed symptoms in women [\[R, R, R, R, R, R\]](#).

Finally, the ‘G’ allele of [rs242924](#), which may increase CRHR1 activity, has been associated with higher IBS rates and associated anxiety. This variant has also been associated with major depression in adults who suffered from childhood abuse. However, this variant may be protective against chronic fatigue [\[R, R, R\]](#).



LOWER ACTIVITY

Likely lower CRHR1 activity based on 6 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ARHGAP27	rs4792887	CC
MAPT	rs12944712	GG
CRHR1	rs17689882	GG
MAPT	rs12938031	GA
MAPT	rs242924	TT
MAPT	rs110402	AA

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

FKBP5 (Stress/ HPA Axis)

The following variants increase *FKBP5* expression and impair stress response. People carrying the risk alleles have a harder time recovering from childhood or early life traumas, leading to increased severity of PTSD [R, R, R]:

- ‘C’ at [rs3800373](#)
- ‘T’ at [rs1360780](#)
- ‘T’ at [rs9470080](#)

In contrast, these variants have also been associated with a reduced susceptibility to chronic pain, such as back pain and neuralgia [R, R, R].

Another variant believed to increase FKBP5 activity, ‘G’ at [rs9394314](#), was found to be protective against chronic neuralgia. However, this allele was associated with overall and neck pain after trauma [R, R].



LOWER ACTIVITY

Likely lower FKBP5 activity based on 4 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TULP1	rs3800373	AA
FKBP5	rs1360780	CC
TULP1	rs9470080	CC
FKBP5	rs9394314	AG

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

KIBRA/WWC1 (Cognition)

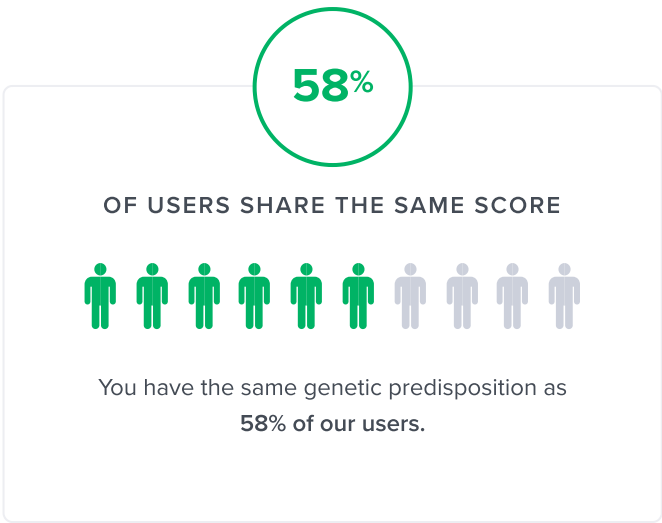
One of the best-studied polymorphisms of this gene is [rs17070145](#). Its minor ‘T’ allele has been linked to larger hippocampal volume and better connectivity between this brain region and the prefrontal cortex. In line with this, this allele has been associated with [\[R\]](#):

- Better episodic and working memory performance [\[R\]](#), [\[R\]](#), [\[R\]](#)
- Slower cognitive decline during aging [\[R\]](#)
- Decreased risk of Alzheimer’s disease [\[R\]](#), [\[R\]](#)



HIGHER ACTIVITY

Likely higher KIBRA/WWC1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
WWC1	rs17070145	TT

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

TUSC3 (Cognition)

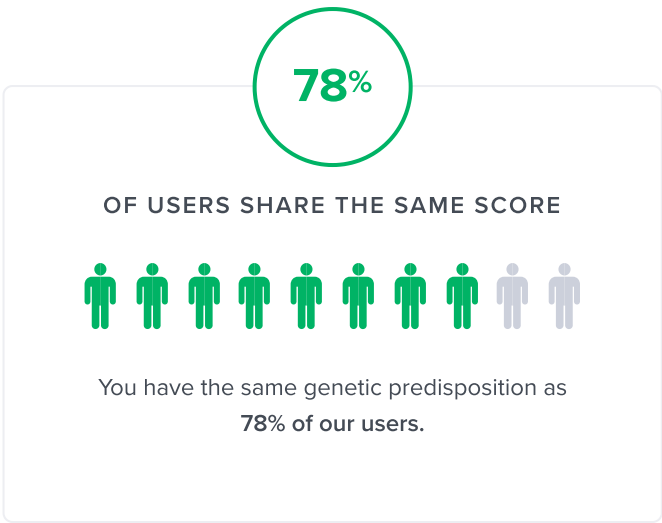
A large-scale GWAS study with ~1,500 participants found that there were significant differences in the IQ scores of people with different genotypes for the [rs240657](#) polymorphism. People with two copies of the major ‘A’ allele came out on top with an above-average mean IQ score of 112. By comparison, people with ‘G’ alleles tended to have IQ scores closer to the general population average of 100 — and the more copies of the ‘G’ allele they had, the lower their relative scores were [\[R\]](#).

This suggests that carriers of the minor ‘G’ allele may have a copy of this gene that is less efficient at absorbing and transporting magnesium.



HIGHER ACTIVITY

Likely higher TUSC3 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TUSC3	rs240657	AA

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



Sleep & Circadian Rhythm

Sleep and circadian rhythms regulate your body’s internal clock, influencing energy levels, mood, and overall health. Genetic variations can affect how well you sleep, how your body adapts to day-night cycles, and your vulnerability to sleep disorders.

Genes such as *CLOCK* and *ARNTL* are core components of the circadian rhythm machinery, helping to regulate sleep-wake cycles. *NPAS2* works alongside these genes to maintain circadian timing, while *AANAT* is involved in melatonin production, the hormone that signals sleep. Additionally, *BHLHE41* influences sleep duration and quality. Understanding your genetic influences on sleep can help you optimize rest and maintain a healthy daily rhythm.

WORSE GENETICS

NPAS2 (Mood/ Sleep Schedule)

Likely worse NPAS2 genetics

WORSE GENETICS

NPAS2 (Mood/ Sleep Schedule)

Likely worse NPAS2 genetics

TYPICAL ACTIVITY

CLOCK (Sleep & Weight)

Likely typical CLOCK activity

TYPICAL GENETICS

AANAT (Circadian Rhythm)

Predisposed to typical AANAT genetics

TYPICAL GENETICS

ARNTL (Circadian Rhythm)

Predisposed to typical ARNTL genetics

TYPICAL ACTIVITY

TPH2 (Serotonin)

Predisposed to typical TPH2 activity

TYPICAL ACTIVITY

GRIA3 (Sleep/Mood)

Likely typical GRIA3 activity

TYPICAL ACTIVITY

BHLHE41 (Sleep)

Predisposed to typical BHLHE41 activity

TYPICAL ACTIVITY

ASMT (Mood, Neurodivergence)

Predisposed to a typical ASMT activity

HIGHER ACTIVITY

MTNR1A (Melatonin)

Likely higher MTNR1A activity

NPAS2 (Mood/ Sleep Schedule)

The main *NPAS2* variant, [rs2305160](#), is often studied in relation to cancer. A 2015 meta-analysis confirmed the link between the **A allele and lower odds of cancer, especially breast cancer** [\[R\]](#).

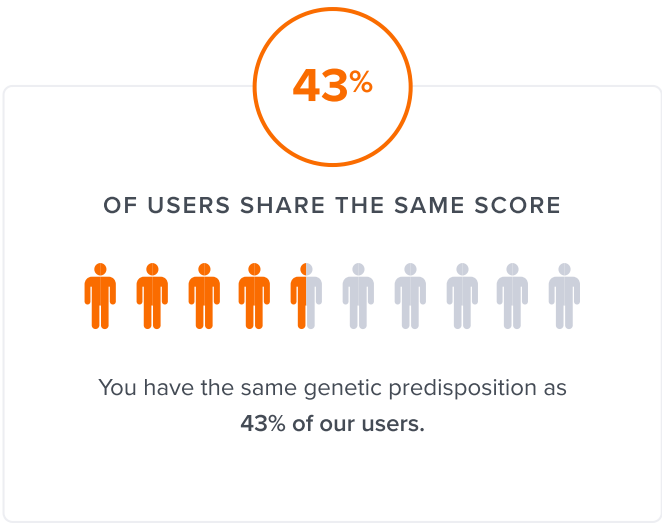
Another *NPAS2* variant, [rs11123857](#), may also be linked to slightly higher odds of breast cancer. It may also correlate with **depression**, according to one study. People with the **G allele** had 1.5x higher odds of depression [\[R\]](#), [\[R\]](#).

NPAS2 helps determine when we sleep and when we eat. The above associations are not surprising, given the key role of our internal clock in mood and cellular health.

More precisely, **eating late and having small fasting windows** may contribute to inflammatory changes linked to breast cancer. Disrupted circadian rhythm also has an established role in depression and other mood disorders [\[R\]](#), [\[R\]](#).



Likely worse NPAS2 genetics based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NPAS2	rs2305160	GG
NPAS2	rs11123857	AA

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

NPAS2 (Mood/ Sleep Schedule)

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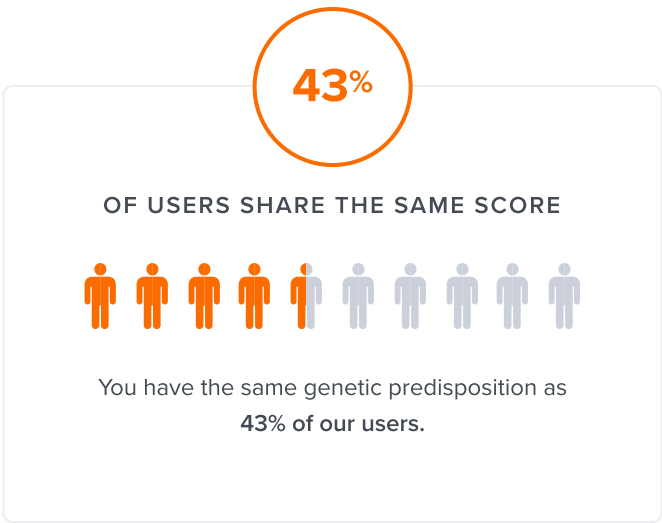
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NPAS2 helps determine when we sleep and when we eat. The above associations are not surprising, given the key role of our internal clock in mood and cellular health.

More precisely, **eating late and having small fasting windows** may contribute to inflammatory changes linked to breast cancer. Disrupted circadian rhythm also has an established role in depression and other mood disorders [\[R\]](#), [\[R\]](#).



Likely worse NPAS2 genetics based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NPAS2	rs2305160	GG
NPAS2	rs11123857	AA

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

CLOCK (Sleep & Weight)

The [rs1801260](#) polymorphism is the most studied SNP in the *CLOCK* gene. Its minor 'G' allele increases CLOCK activity and has been associated with many sleep-related traits, such as [\[R\]](#):

- Abnormal and less stable circadian rhythms [\[R, R\]](#)
- Shorter sleep duration (≤ 6 h per day) [\[R, R, R\]](#)
- Being an evening person [\[R, R, R\]](#)
- Less activity overall, being active later in the day, and being sleepier during the morning [\[R, R\]](#)

This variant has also been linked to:

- Higher prevalence of obesity (mixed findings) [\[R, R\]](#)
- Higher ghrelin (hunger hormone) levels and lower satiety [\[R, R\]](#)
- Higher insulin and insulin resistance [\[R\]](#)
- Low compliance with dietary programs, such as the Mediterranean diet [\[R\]](#)
- More difficulty losing weight from diets or bariatric surgery [\[R, R, R\]](#)

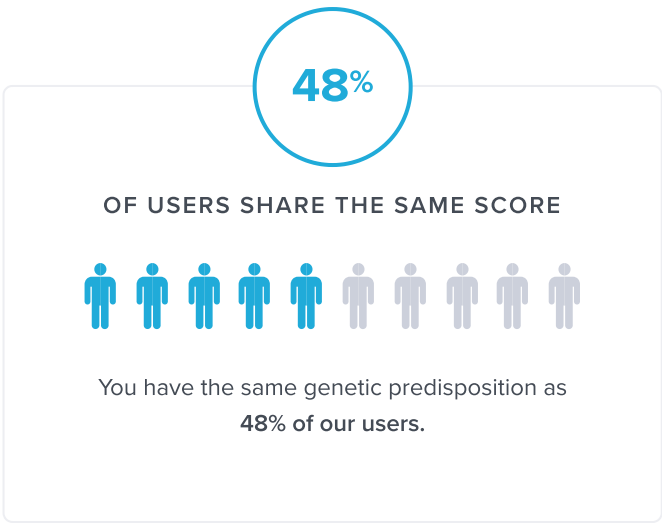
There is a link between how the *CLOCK* gene affects circadian rhythms and sleep on one hand, and metabolic balance and eating behavior on the other [\[R, R\]](#).

Studies have found that sleep deprivation disrupts metabolism by increasing the levels of the stress hormone cortisol and decreasing insulin sensitivity. Furthermore, a lack of sleep increases the levels of the hunger hormone ghrelin, which increases hunger and appetite -- thereby increasing the risk of obesity [\[R, R, R, R, R, R\]](#).



TYPICAL ACTIVITY

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



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
CLOCK	rs1801260	AG

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

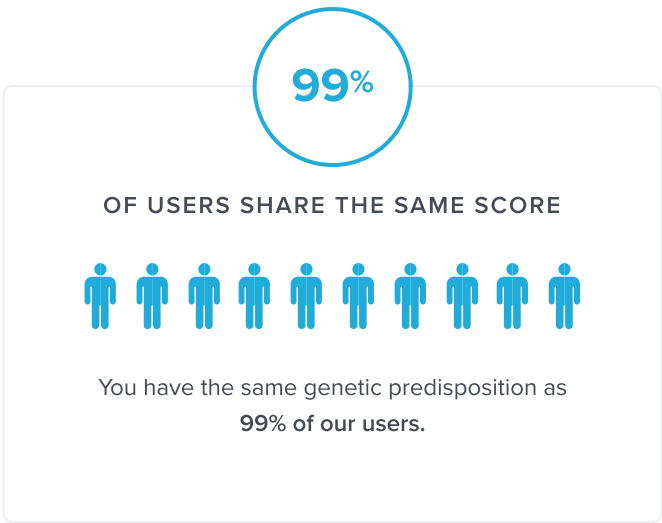
AANAT (Circadian Rhythm)

A study of 211 participants associated the rare ‘A’ allele of [rs28936679](#) (G619A) with an increased risk of delayed sleep phase disorder, a condition in which individuals have a sleep/wake cycle delayed by 2 hours or more from usual sleep patterns [\[R\]](#).

This variant has also been associated with abnormal biological rhythms in patients with bipolar disorder [\[R\]](#).



Predisposed to typical AANAT genetics based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
AANAT	rs28936679	GG

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

ARNTL (Circadian Rhythm)

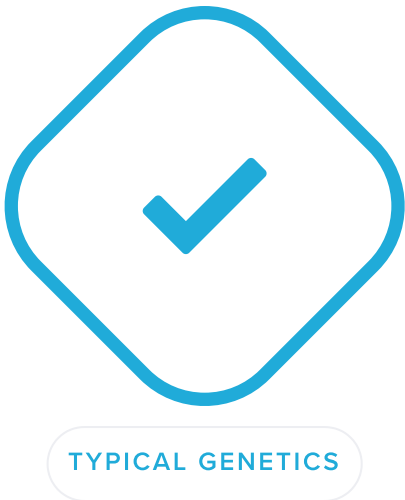
The best-researched *ARNTL* polymorphism is [rs2278749](#). Its minor ‘T’ allele has been associated with:

- Increased risk of disturbing dreams [\[R\]](#)
- Increased risk of miscarriages [\[R\]](#)
- Increased risk of Alzheimer’s disease (in [APOE](#) ε4 non-carriers) [\[R\]](#)
- Decreased seasonal variation of energy levels [\[R\]](#)
- Decreased risk of breast cancer in night shift workers [\[R\]](#)

Another variant, ‘T’ at [rs11022775](#), has been associated with an increased risk of type 2 diabetes but a decreased risk of gestational diabetes, dyslipidemia, and high blood pressure [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

The minor ‘A’ allele of [rs2290035](#) has been associated with an increased risk of lung cancer and night shift-associated breast cancer [\[R\]](#), [\[R\]](#).

Finally, the ‘T’ allele of [rs6486122](#) has been associated with an increased risk of coronary artery disease and type 2 diabetes, as well as with higher levels of CRP, triglycerides, and testosterone [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).



Predisposed to typical ARNTL genetics based on 4 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ARNTL	rs11022775	CC
BTBD10	rs2278749	TC
BTBD10	rs2290035	TA
ARNTL	rs6486122	CC

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

TPH2 (Serotonin)

The [rs4570625](#) variant has been most widely investigated. Its minor ‘T’ allele is believed to reduce TPH2 production, resulting in lower serotonin levels. Carriers of this variant show higher activity in the part of the brain responsible for stress and fear processing (the *amygdala*) in response to emotional stimuli [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

The ‘T’ variant has also been associated with higher [GABA](#) levels. GABA is the main inhibitory neurotransmitter preventing over-stimulation of neurons. An abnormal GABA decrease can produce mental illness and symptoms such as anxiety, depression, insomnia, convulsions, and epilepsy [\[R\]](#), [\[R\]](#), [\[R\]](#).

The link of this variant with both low serotonin and high GABA levels may explain its complex association with anxiety, mood, and fatigue. This variant has been associated with a decreased risk of:

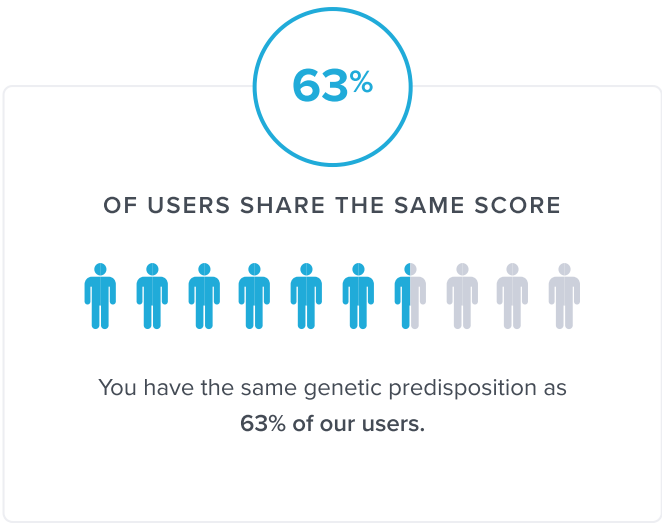
- [Anxiety](#) disorders such as panic disorder [\[R\]](#)
- Severe [ADHD](#) symptoms and poor sustained attention in healthy individuals [\[R\]](#)
- Early-onset OCD [\[R\]](#)
- Depression [\[R\]](#)
- Suicide attempts [\[R\]](#)
- Schizophrenia [\[R\]](#)
- Failure of placebo treatments to improve anxiety [\[R\]](#)

However, it increases [fatigue](#) in women with IBS [\[R\]](#).



TYPICAL ACTIVITY

Predisposed to typical TPH2 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TPH2	rs4570625	GG

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

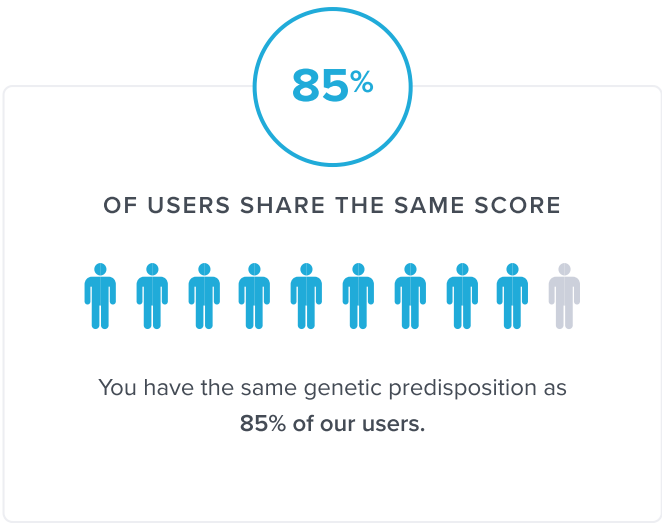
GRIA3 (Sleep/Mood)

The minor ‘A’ allele of the [rs687577](#) polymorphism has been associated with a decreased risk of depression in women and with longer sleep duration. Given the role of glutamate as an excitatory neurotransmitter, this variant may reduce GRIA3 activity by either decreasing the number of active receptors or attenuating the excitatory effects upon glutamate binding [[R](#), [R](#)].



TYPICAL ACTIVITY

Likely typical GRIA3 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GRIA3	rs687577	C

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

BHLHE41 (Sleep)

Several BHLHE41 variants have been associated with a sleep disorder known as ‘familial natural short sleep’ (FNSS), in which individuals can sleep 6 hours or less per night without experiencing any negative symptoms of sleep deprivation. They include:

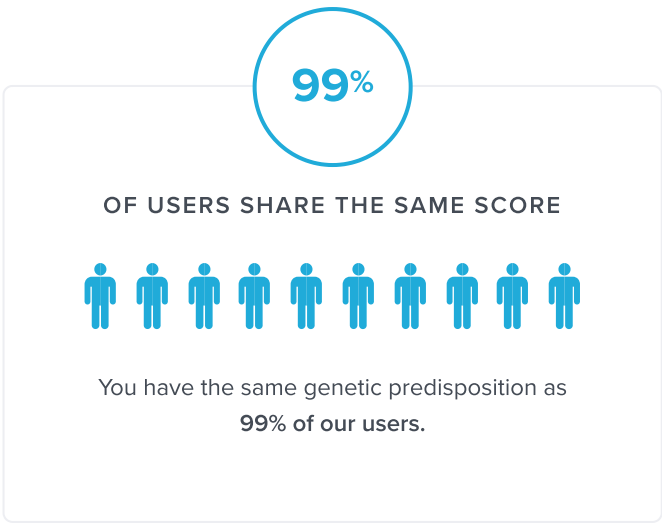
The rare ‘T’ allele of [rs121912617](#) (P384R) has been associated with a sleep disorder known as ‘familial natural short sleep’ (FNSS) in which an individual sleeps 6-6.5 per night without experiencing any negative symptoms of sleep deprivation [\[R\]](#).

This variant weakens the DEC2 protein's ability to suppress the activation of the CLOCK-BMAL1 complex, leading to increased orexin production and longer wakefulness [\[R\]](#).



TYPICAL ACTIVITY

Predisposed to typical BHLHE41 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
BHLHE41	rs121912617	GG

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

ASMT (Mood, Neurodivergence)

The main ASMT polymorphisms are [rs4446909](#) and [rs5989681](#). Their minor alleles, ‘A’ and ‘C’ respectively, may increase ASMT activity, resulting in higher melatonin and 5-HTP production [\[R, R\]](#).

These variants have been associated with a decreased risk of:

- Autism spectrum disorder [\[R\]](#)
- Bipolar disorder [\[R\]](#)

The minor rs4446909 allele has also been associated with a decreased risk of recurrent depressive disorder and milder depressive symptoms in people with delayed sleep disorder. In contrast, the rs5989681 variant associated with a decreased risk of recurrent depressive disorder is the major ‘G’ allele [\[R, R\]](#).

When it comes to sleep and circadian rhythms, only the rs4446909 polymorphism has been investigated. Its minor ‘A’ allele has been associated with shorter sleep duration, lower activity during active periods of sleep, and decreased circadian rhythm stability [\[R\]](#).



Predisposed to a typical ASMT activity based on 2 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
AKAP17A	rs4446909	G
ASMT	rs5989681	G

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

MTNR1A (Melatonin)

The most well-researched *MTNR1A* allele is [rs2119882](#). Its ‘C’ allele may prevent the binding of proteins that activate gene expression, resulting in a lower number of MT1 receptors. This variant has been associated with an increased risk and severity of:

- PCOS [\[R\]](#)
- Gestational diabetes [\[R\]](#)
- Liver cancer metastasis [\[R\]](#)

However, this allele has also been associated with a decreased risk of:

- Breast cancer in shift workers [\[R\]](#)
- Graves’ disease [\[R\]](#)

The ‘A’ allele of [rs12506228](#) reduces the number of MT1 melatonin receptors in the brain. This variant as been associated with:

- Job-related exhaustion in shift workers [\[R\]](#)
- Alzheimer’s disease in old age [\[R\]](#)
- Slower cognitive development in infants [\[R\]](#)

Another allele believed to prevent the binding of proteins that activate *MTNR1A* expression is ‘A’ at [rs13140012](#). This allele has been associated with

- Oral cancer and worse prognostic in betel quid chewers [\[R\]](#)
- Kidney stones [\[R\]](#)

The ‘G’ allele of [rs6553010](#) may also affect *MTNR1A* expression and has been associated with an increased risk of

- Aggressive urothelial cell carcinoma [\[R\]](#)
- Pain after root canal treatment [\[R\]](#)

However, this allele may also reduce the risk of kidney stones [\[R\]](#).

Finally, a study associated the ‘GG’ genotype of [rs7665392](#) with an increased risk of breast cancer in premenopausal women. The authors speculated that this variant impairs the function of the MT1 receptor, resulting in an activation of estradiol-induced genes linked to breast cancer [\[R\]](#).



HIGHER ACTIVITY

Likely higher MTNR1A activity based on 5 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MTNR1A	rs13140012	AA
MTNR1A	rs6553010	GA
MTNR1A	rs2119882	TT
MTNR1A	rs12506228	CC
MTNR1A	rs7665392	TT

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



Sports & Exercise

Your ability to perform and recover during physical activity is influenced by a combination of genetics and lifestyle. **Genetic variations can affect muscle strength, endurance, recovery time, and how your body adapts to exercise.**

Genes like *ACTN3* are well-known for their role in muscle fiber composition and power output, while *COL1A1* and *COL5A1* influence connective tissue strength and injury risk. *AMPD1* plays a role in muscle energy metabolism. Understanding these genetic factors can help you tailor your fitness routine for optimal performance and injury prevention.

TYPICAL ACTIVITY ACTN3 (Power) Likely typical ACTN3 activity	TYPICAL ACTIVITY COL5A1 (Collagen) Likely typical COL5A1 activity	TYPICAL ACTIVITY AMPD1 (Muscle Performance) Predisposed to typical AMPD1 activity
HIGHER ACTIVITY COL1A1 (Collagen) Predisposed to higher COL1A1 activity	HIGHER ACTIVITY SLC16A1 (Athletic Performance) Likely higher SLC16A1 activity	

ACTN3 (Power)

Power is the ability to produce short, intense movements (e.g., sprinting, jumping, throwing). Our muscles are made up of fibers. The type of fibers determines what a muscle is good at. **Fast-twitch fibers support rapid movements and are more common in power athletes** [R, R, R].

Fast-twitch muscle fibers contain higher amounts of a specific protein (alpha-actinin-3) that supports muscle power. The gene that helps make this protein is called [ACTN3](#) [R].

A specific *ACTN3* gene variant determines whether people produce this protein or not. People who produce this protein tend to have increased power performance. They are more likely to be elite power athletes [R, R, R].

On the other hand, people with more slow-twitch muscle fibers may be more suitable for endurance sports. They may also have better cold adaptation.

However, fitness level and other lifestyle factors have a strong effect on your power. The impact of genetics is more pronounced in elite athletes because they have improved all other factors [R].

Also, other gene variants not included in this report may influence power performance.



Likely typical ACTN3 activity based on the genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ACTN3	rs1815739	TC

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

COL5A1 (Collagen)

Variants resulting in reduced type V collagen may increase the risk of injuries. This may be because carriers of these variants may have weaker ligaments, tendons, and muscles. One of such variants is [rs12722](#). Its minor ‘T’ allele has been associated with an increased risk of:

- Achilles tendon injury [\[R\]](#), [\[R\]](#)
- Anterior cruciate ligament injury [\[R\]](#), [\[R\]](#)
- Tennis elbow [\[R\]](#), [\[R\]](#)
- Carpal tunnel syndrome [\[R\]](#)
- Muscle cramps [\[R\]](#)

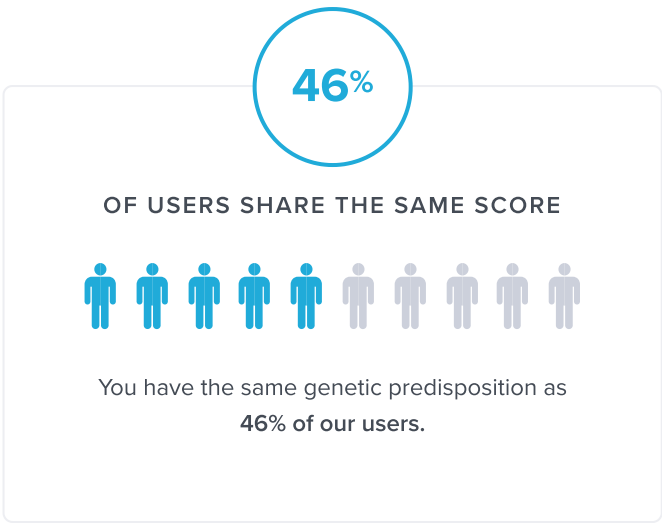
People with the ‘TT’ genotype may also have more severe (but not more frequent) muscle injuries. Probably due to the association of this variant with soft tissue injuries, carriers may have lower odds of elite rugby status [\[R\]](#), [\[R\]](#).

On the bright side, this genotype has also been associated with improved endurance performance in runners. Moreover, the ‘T’ variant may reduce the risk of rotator cuff injury [\[R\]](#), [\[R\]](#).



TYPICAL ACTIVITY

Likely typical COL5A1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
COL5A1	rs12722	TC

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

AMPD1 (Muscle Performance)

A common variant [rs17602729](#)-A reduces AMPD1 function. Some individuals with the A allele get **muscle cramps and pain** when they exert themselves. In line with this, they are less likely to be elite power- or strength-based athletes [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

The effects are much more pronounced in people with two copies (AA) [\[R\]](#), [\[R\]](#), [\[R\]](#).

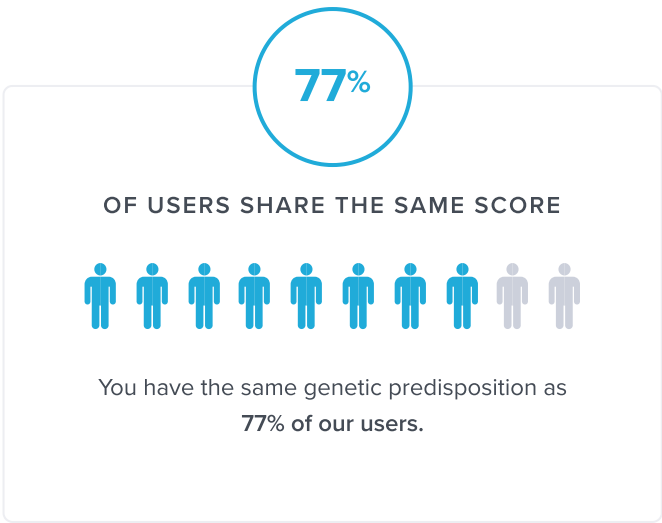
Recent research on Long COVID patients found that those with AMPD1 deficiency had higher post-exercise lactic acid levels compared to those with typical AMPD1. These patients improved more after a 12-week exercise program, though their lactic acid levels remained higher than normal [\[R\]](#).

On the bright side, people with this variant may have a better prognosis of heart disease [\[R\]](#), [\[R\]](#), [\[R\]](#).



TYPICAL ACTIVITY

Predisposed to typical AMPD1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
AMPD1	rs17602729	GG

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

COL1A1 (Collagen)

The most well-researched *COL1A1* polymorphism is [rs1800012](#). Its minor ‘A’ allele promotes the expression of an alternative version of the protein with increased pro-α1 chain levels, potentially increasing its mechanical strength and stability [\[R\]](#).

This variant has been associated with a decreased risk of:

- Sports-related tendon and ligament injuries [\[R\]](#)
- Musculoskeletal soft tissue injuries [\[R\]](#)
- Cervical insufficiency [\[R\]](#)
- Pelvic organ prolapse [\[R\]](#)

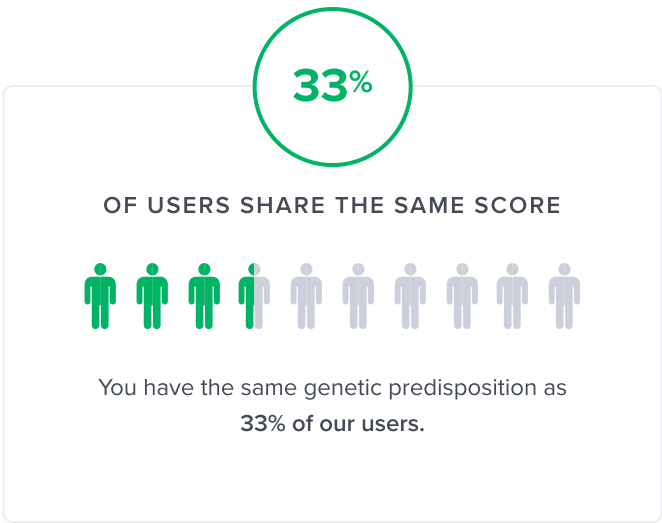
In contrast, it may increase the risk of osteoporosis and bone fractures [\[R\]](#), [\[R\]](#).

The ‘A’ allele may also affect athletic performance and has been found more commonly in competitive runners [\[R\]](#).



HIGHER ACTIVITY

Predisposed to higher COL1A1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
COL1A1	rs1800012	AC

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

SLC16A1 (Athletic Performance)

The most widely investigated *SLC16A1* variant is [rs1049434](#). Its minor ‘A’ allele encodes a protein with a 60-65% reduction in lactate transport, resulting in higher blood lactate levels during high-intensity exercise. Carriers may also have higher oxygen requirements and glycogen use. In line with this, the variant has been associated with a **decreased endurance performance** and lower odds of reaching elite status [\[R, R, R, R, R, R, R, R, R, R, R, R, R, R\]](#).

However, this variant has been associated with better **performance at power- and strength-oriented sports** such as wrestling, a reduced incidence of muscle and bone injuries in athletes, and faster lactate clearance during exercise recovery [\[R, R, R, R\]](#).

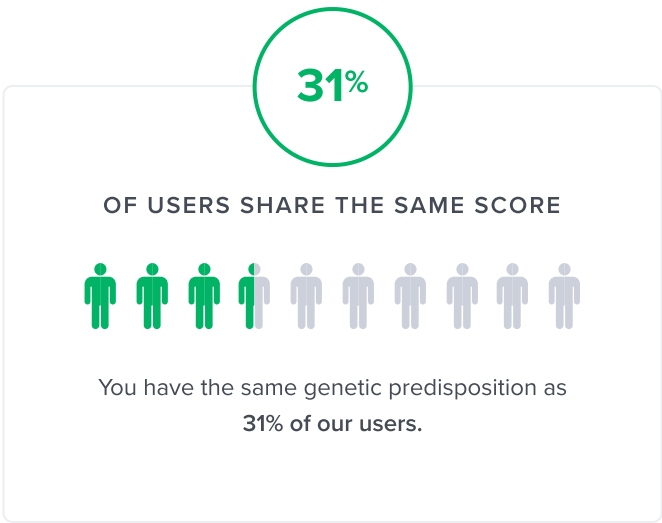
Moreover, acute myeloid leukemia and non-small cell lung cancer patients carrying this variant may show better overall survival [\[R, R\]](#).

A variant usually inherited together with this one, ‘A’ at [rs7169](#), has been associated with increased survival in multiple myeloma patients and better response to valproic acid in children with epilepsy [\[R, R\]](#).



HIGHER ACTIVITY

Likely higher SLC16A1 activity based on the genetic variants we looked at



Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SLC16A1	rs1049434	TT
SLC16A1	rs7169	AA

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

Recommendations Details

1



Dietary Folate

Increase your intake of folate-rich foods such as leafy green vegetables, fruits, nuts, and legumes. Aim to consume these foods daily, incorporating them into various meals throughout the day to meet the recommended dietary allowance of 400 micrograms for adults.

Helps with these Symptoms & Conditions:

Cognitive Decline

Food Allergies

Helps with these Goals:

Energy

Mood

Helps with these DNA Risks:

 MTHFR

 MTHFD1 (Folate/ Methylation)

How it helps

 MTHFR

IMPACT

5 / 5

EVIDENCE

5 / 5

People with lower MTHFR activity may have 16% lower folate levels, and they tend to have increased homocysteine [R].

High dietary intake of folate is associated with lower homocysteine levels [R, R].

It's always a good practice to get plenty of folate by eating a variety of fresh fruits and vegetables. This is especially true for people with lower MTHFR activity. Folate in food sources is natural or "active" form. In theory, this means it is equally beneficial for people with lower MTHFR activity [R, R, R, R, R].

Rich sources of folate include [R, R]:

- Beef liver
- Spinach
- Black-eyed peas
- Asparagus
- Citrus fruits

 MTHFD1 (Folate/ Methylation)

IMPACT

3 / 5

EVIDENCE

2 / 5

[Folate](#) deficiency in people with the MTHFD1 variant contributed to both fatty liver (due to low choline) and homocysteine elevation. To counteract these effects, make sure to get enough folate daily [\[R\]](#), [\[R\]](#).

2

Methylfolate

Take an L-methyl folate supplement (400-800 micrograms daily), ideally with a meal, to improve absorption. This dosage is recommended for adults, including pregnant women, to support overall health, especially to reduce the risk of neural tube defects in developing fetuses. Continue daily use as part of your regular supplement routine.

TYPICAL STARTING DOSE

400 mcg

Helps with these Symptoms & Conditions:

- Artery Hardening
- Cognitive Decline

Helps with these Goals:

- Cognitive Function
- Fat Loss
- Libido
- Mood

Helps with these DNA Risks:

-  MTHFR
-  MTHFD1 (Folate/ Methylation)

How it helps



MTHFR

IMPACT

5 / 5

EVIDENCE

5 / 5

People with lower MTHFR activity may have 16% lower folate levels, and they tend to have increased homocysteine [\[R\]](#).

Supplementation with folate (0.5-1 mg/day) may lower homocysteine levels. It may work in healthy people, those with [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#):

- Heart problems
- Cognitive decline
- High blood sugar

CDC notes that **folic acid** is the only supplement proven to reduce birth defects due to low folate [\[R\]](#).



MTHFD1 (Folate/ Methylation)

IMPACT

2 / 5

EVIDENCE

2 / 5

Folate supplements may help by providing more of this vitamin and enhancing homocysteine methylation, especially in pregnant women and those planning to conceive. Most supplements contain the recommended daily amount of 400 mcg [\[R\]](#), [\[R\]](#).

People with [specific MTHFR](#) variants can’t produce enough methylfolate, which is the active form. They should supplement with [L-methylfolate](#) [\[R\]](#).

3

Leafy Green Vegetables

Incorporate at least one serving of leafy green vegetables, such as spinach, kale, or Swiss chard, into your diet daily. This can be done by adding them to salads, smoothies, or as a side dish to your meals.

Helps with these Symptoms & Conditions:

Artery Hardening

Helps with these Goals:

LongevityMemoryShort Term Memory

Helps with these DNA Risks:

 MTHFR BDNF

How it helps

 MTHFR

IMPACT

3 / 5

EVIDENCE

2 / 5

Research indicates that higher intake of dark green leafy vegetables is associated with a lower risk of cutaneous squamous cell carcinoma (SCC) in people carrying specific MTHFR gene variants [R].

Additionally, a study found that people with the MTHFR TT genotype may experience increased benefits from high green vegetable intake, which may lower their risk of breast cancer compared to those with low intake [R].

Leafy green vegetables may help due to their high folate content.

 BDNF

IMPACT

0 / 5

EVIDENCE

0 / 5

Leafy greens have a variety of nutrients that may support brain health and contribute to increasing BDNF.

4

Betaine (TMG)

To take Betaine (TMG) as a supplement, consume 500-2000 mg daily, preferably with a meal to enhance absorption. It is recommended to start at the lower end of the dosage range and adjust based on personal tolerance and effectiveness. This supplement can be taken indefinitely for ongoing support of heart health and liver function.

TYPICAL STARTING DOSE

500 mg

Helps with these Goals:

- Fat Loss
- Strength

Helps with these DNA Risks:

 MTHFR

How it helps

 MTHFR

IMPACT

3 / 5

EVIDENCE

2 / 5

TMG or betaine helps turn homocysteine into methionine. For this reason, it plays a key role in the methylation cycle.

People with lower MTHFR activity and poor methylation may have reduced betaine production. To make up for this effect, consume a variety of betaine-rich foods such as [\[R\]](#):

- Liver meats
- [Quinoa](#)
- Beets
- Wheat germ
- Spinach

Supplementing with TMG (1.5-4 g/day for 6-24 weeks) may lower homocysteine levels, which tend to be higher in people with impaired MTHFR function [\[R\]](#).

Homocystinuria is a rare genetic disorder that results in elevated homocysteine levels in the urine. In people with this condition, TMG is approved by the FDA to lower urinary homocysteine [\[R\]](#).

A study of 860 mothers observed much lower neural tube defect (NTD) rates for the highest vs. lowest dietary intakes of choline, betaine, and methionine. NTDs are of particular concern for people with reduced MTHFR activity due to impaired methylation [\[R\]](#).

According to preliminary findings, early betaine supplementation may improve outcomes in cases of MTHFR deficiency [\[R\]](#).

Please note: *Doses above 4 g/day may increase LDL and triglyceride levels. TMG supplementation can cause a person’s urine and sweat to smell fishy* [\[R\]](#).

5



Zinc

Take a 15 mg zinc supplement daily, ideally with a meal to enhance absorption.

TYPICAL STARTING DOSE

15 mg

Helps with these Symptoms & Conditions:

- Artery Hardening
- Food Allergies
- Hair Loss
- Underactive Thyroid

Helps with these Goals:

- Cognitive Function
- Focus
- Libido
- Memory
- Mood

Helps with these DNA Risks:

-  MTHFR
-  MTR (Methylation)
-  SLC30A8 (Blood Sugar)

How it helps

 MTHFR

IMPACT2 / 5

EVIDENCE2 / 5

Zinc is important for folate absorption and healthy methylation. Ensure that your zinc levels are optimal [\[R\]](#).

If you are deficient in zinc, your gut enzymes can’t break down folate into the form you can absorb [\[R\]](#), [\[R\]](#).

Zinc also helps folate carry out its role in the body [\[R\]](#).

 MTR (Methylation)

IMPACT0 / 5

EVIDENCE0 / 5

Zinc is crucial for proper methylation because it supports the functioning of enzymes that transfer methyl groups to DNA. You can consider it as fuel for your body's methylation engine, helping it to work efficiently.

 SLC30A8 (Blood Sugar)

IMPACT4 / 5

EVIDENCE3 / 5

In some people, the pancreas produces too much insulin, which can lead to insulin resistance. **Zinc may be able to prevent this and protect the pancreas from damage** [\[R\]](#), [\[R\]](#).

Zinc-deficient people are more likely to have high blood sugar [\[R\]](#).

Supplementing with zinc (25-34 mg/day) may reduce blood sugar. It may also lower the odds of type 2 diabetes [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).



PERSONALIZED TO YOUR GENES

Your [SLC30A8](#) gene variant has been associated with increased blood glucose levels. Supplementation with zinc may be particularly effective at counteracting its detrimental effects [\[R\]](#).

YOUR GENETIC VARIANTS

GENE	SNP	GENOTYPE	EVIDENCE
SLC30A8	rs11558471	AA	

6



Avoid High-Dose Niacin Supplements

Ensure your daily intake of niacin (vitamin B3) from supplements does not exceed 35 mg, which is the upper intake level for adults, to prevent the risk of negative side effects like flushing and liver damage. Always check the label of your supplement to confirm the niacin dosage.

TYPICAL STARTING DOSE

35 mg

Helps with these DNA Risks:



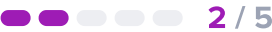
MTHFR

How it helps



MTHFR

IMPACT



EVIDENCE



MTHFR is essential for converting homocysteine to methionine. High doses of niacin can exacerbate the accumulation of homocysteine in individuals with reduced MTHFR function.

This is because niacin in large amounts can deplete methyl donors like SAME (S-adenosylmethionine), which are needed for homocysteine metabolism [\[R\]](#).

7



Dietary Riboflavin (Vitamin B2)

Include riboflavin-rich foods in your daily diet, such as milk, cheese, eggs, lean meats, green leafy vegetables (like spinach), almonds, and fortified cereals. Aim for an intake of 1.1 to 1.3 mg of riboflavin per day for adults, as recommended by dietary guidelines.

Helps with these DNA Risks:



MTHFR

How it helps

 **MTHFR**

IMPACT

1 / 5

EVIDENCE

2 / 5

There is some evidence that people with MTHFR variants may do better if they get more [riboflavin](#) (vitamin B2) [\[R, R, R, R, R, R, R, R\]](#).

This vitamin helps MTHFR work properly [\[R, R, R\]](#).

Good sources of riboflavin include [\[R, R\]](#):

- Eggs
- Dairy
- Lean and organ meats
- Green vegetables
- Fortified cereals

8



Riboflavin (Vitamin B2)

Take a riboflavin (vitamin B2) supplement daily, with a dose ranging from 5mg to 400mg, depending on the specific health concern or advice from a healthcare provider. Swallow the supplement with water, preferably with a meal to enhance absorption. This regimen can be continued long-term or as directed by a healthcare professional.

TYPICAL STARTING DOSE

25 mg

Helps with these Goals:

Mood

Helps with these DNA Risks:

 **MTHFR**

How it helps

 **MTHFR**

IMPACT

1 / 5

EVIDENCE

3 / 5

There is some evidence that people with MTHFR variants may do better if they get more [riboflavin](#) (vitamin B2) [\[R, R, R, R, R, R, R, R\]](#).

For example, supplementing with riboflavin may decrease blood pressure more in people with reduced MTHFR activity [\[R, R, R, R\]](#).

This vitamin helps MTHFR work properly [\[R, R, R\]](#).

9



Yoga

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

TYPICAL STARTING DOSE
30 minutes

Helps with these Symptoms & Conditions:

- Artery Hardening
- Cognitive Decline

Helps with these Goals:

- Cognitive Function
- Creativity
- Energy
- Fat Loss
- Focus
- Libido
- Longevity
- Memory
- Mood
- Short Term Memory

Helps with these DNA Risks:

-  BDNF
-  DRD2 (Dopamine)

How it helps

 BDNF

IMPACT

2 / 5

EVIDENCE

2 / 5

Different yoga programs increased BDNF in several trials of middle-aged volunteers, older adults, and patients with major depressive disorder [\[R, R, R, R\]](#).

 DRD2 (Dopamine)

IMPACT

3 / 5

EVIDENCE

2 / 5

Chronic stress during adolescence may blunt dopamine responses during adulthood according to animal studies. In contrast, acute stress may increase dopamine activity [\[R, R\]](#).

Both a single yoga session and regular yogic practice may increase dopamine levels [\[R, R\]](#).

10



Green Tea

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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 instructions, usually once a day with water.

TYPICAL STARTING DOSE

400 mg

Helps with these Symptoms & Conditions:

- Artery Hardening
- Cognitive Decline

Helps with these Goals:

- Cognitive Function
- Energy
- Fat Loss
- Focus
- Longevity
- Memory
- Mood

Helps with these DNA Risks:

- ! BDNF
- ! FOXO1 (Blood Sugar)

How it helps

! BDNF

IMPACT0 / 5

EVIDENCE0 / 5

Green tea boosts brain-derived neurotrophic factor (BDNF), a protein that supports brain health by facilitating brain cell survival and growth. Through its antioxidants, it also helps prevent any damage to your brain cells, effectively reducing the chances of low BDNF levels.

! FOXO1 (Blood Sugar)

IMPACT3 / 5

EVIDENCE3 / 5

Many plant polyphenols such as [EGCG](#) from green tea activate AMPK; eating foods that contain them may help you balance FOXO1 and maintain healthy insulin sensitivity [\[R\]](#).

A meta-analysis of 7 studies and almost 287k healthy participants associated drinking green tea with an 18% lower incidence of type 2 diabetes [\[R\]](#).

Its potential to improve blood sugar control in people with type 2 diabetes or at risk of developing this condition is less clear. While 2 meta-analyses (the largest one with 22 trials and 1,584 participants) found it effective at lowering fasting blood glucose (one of them found that high doses also lowered fasting insulin), a third meta-analysis (7 trials and 510 participants) found green tea ineffective at lowering type 2 diabetes markers in people at risk [\[R\]](#), [\[R\]](#), [\[R\]](#).

Please note: polyphenols from green tea may bind to iron and form insoluble complexes, which reduces iron absorption in the gut. If you have anemia, consult your healthcare provider before using green tea [\[R\]](#).

11



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 Goals:

Fat Loss

Longevity

Mood

Helps with these DNA Risks:

! FOXO1 (Blood Sugar)

! UCP1 (Weight)

How it helps

! FOXO1 (Blood Sugar)

IMPACT

4 / 5

EVIDENCE

5 / 5

[Intermittent fasting](#) and calorie restriction reduce available energy and activate AMPK [\[R\]](#).

A meta-analysis of 43 studies and 2,094 participants found that calorie restriction reduced fasting insulin and insulin resistance in both healthy and diabetic people [\[R\]](#).

Eating a low-calorie diet is a good way to lose weight. **It may also lower blood sugar, especially in overweight people** [\[R, R, R, R\]](#).

! UCP1 (Weight)

IMPACT

5 / 5

EVIDENCE

5 / 5

To promote weight loss, experts recommend lowering your calorie intake by 30% of your daily requirement [\[R, R\]](#).

Eating fewer calories and intermittent fasting may support short-term weight loss equally. On the other hand, people who eat fewer calories may maintain their weight loss better in the long run [\[R, R, R, R, R, R\]](#).

Types of intermittent fasting that may be especially helpful for weight loss include:

- **Alternate-day fasting:** fasting every other day [\[R\]](#)
- **Time-restricted eating:** eating within a certain time frame each day [\[R, R, R, R, R\]](#)

A good way to limit calorie intake is to eat more low-calorie foods. These include soups and foods rich in protein and fiber. You can also try eating a low-calorie snack before a meal [\[R, R, R\]](#).

These methods can help make you feel more full and thus eat less [\[R, R, R\]](#).

Please note: *Limiting calorie intake too much or fasting for too long can cause malnutrition, anemia, eating disorders, and other health problems. Children, pregnant women, and people with certain chronic conditions or substance abuse disorders shouldn’t do intermittent fasting. Talk to your doctor before implementing any drastic calorie restriction* [\[R\]](#).

12



Coffee

Drink 1 to 3 cups of black coffee daily, preferably in the morning to minimize potential sleep disturbances. Avoid adding sugar or cream to keep it healthy. Continue this habit daily for long-term benefits.

Helps with these Symptoms & Conditions:

- Artery Hardening
- Cognitive Decline

Helps with these Goals:

- Cognitive Function
- Creativity
- Fat Loss
- Focus
- Longevity
- Memory
- Mood
- Short Term Memory

Helps with these DNA Risks:

-  BDNF
-  DRD2 (Dopamine)

How it helps

 BDNF

IMPACT

0 / 5

EVIDENCE

0 / 5

Coffee increases the levels of a protein called BDNF (Brain-Derived Neurotrophic Factor) in the brain. Higher BDNF levels boost brain health, improving memory and cognitive function, thus helping prevent neurological conditions.

 DRD2 (Dopamine)

IMPACT

2 / 5

EVIDENCE

2 / 5

Caffeine may increase dopamine levels and boost dopamine function [\[R\]](#), [\[R\]](#).

Interestingly, both caffeinated and decaffeinated coffee rapidly increased blood dopamine levels in a study [\[R\]](#).

Please note: Too much caffeine may lead to sleep problems, high blood pressure and cholesterol, fast heart rate, and dependence. If you're pregnant, try to limit caffeine to 200 mg per day [\[R\]](#).

Please note: polyphenols and tannins from coffee may bind to iron and form insoluble complexes, which reduces iron absorption in the gut. If you have anemia, consult your healthcare provider before using coffee or coffee supplements.

13



Limit Dairy Intake

Reduce your consumption of dairy products, such as milk, cheese, yogurt, and butter, to no more than one serving per day. A serving is equivalent to 1 cup (240 ml) of milk, 1 cup of yogurt, 1.5 ounces (42 grams) of natural cheese, or 2 ounces (56 grams) of processed cheese.

Helps with these Symptoms & Conditions:

Hashimoto's Disease

Helps with these DNA Risks:

! Dairy Sensitivity (Casein)

How it helps

! Dairy Sensitivity (Casein)

IMPACT

5 / 5

EVIDENCE

5 / 5

People with dairy sensitivity may have symptoms ranging from gut problems to migraines after consuming dairy. Because the immune system attacks casein in this case, **people with dairy sensitivity don’t tolerate lactose-free dairy either** [R, R, R].

People with suspected dairy sensitivity may want to avoid all dairy products. If you're avoiding dairy, make sure you're getting plenty of calcium from other foods [R].

Consult a healthcare professional before making any major dietary changes.

14



Mindfulness Meditation

Practice mindfulness meditation for 10-20 minutes daily. Find a quiet, comfortable place to sit or lie down, then focus on your breath, observing thoughts and sensations without judgment. Consistency is key, so try to incorporate it into your daily routine, perhaps in the morning or before bed.

TYPICAL STARTING DOSE

30 minutes

Helps with these Goals:

- Creativity
- Fat Loss
- Focus
- Libido
- Memory
- Mood
- Short Term Memory

Helps with these DNA Risks:

! BDNF

! DRD2 (Dopamine)

How it helps

! BDNF

IMPACT

0 / 5

EVIDENCE

0 / 5

Practicing mindfulness meditation can increase BDNF production, potentially supporting cognitive functions and mental wellbeing.

**DRD2 (Dopamine)**

IMPACT

0 / 5

EVIDENCE

0 / 5

Mindfulness meditation may increase dopamine production and promote neuroplasticity in the brain.

15



Berberine

Take 500 mg of berberine two times a day before meals. Continue this regimen for up to three months, then evaluate its effects with your healthcare provider.

TYPICAL STARTING DOSE

1000 mg

Helps with these Goals:

Fat Loss

Helps with these DNA Risks:

 **FOXO1 (Blood Sugar)**

 **HNF4A (Gut Inflammation)**

 **TCF7L2 (Blood Sugar/Diet)**

How it helps

**FOXO1 (Blood Sugar)**

IMPACT

4 / 5

EVIDENCE

4 / 5

Berberine (0.5-1.5 g/day for 2-6 months) may help control blood sugar. It likely boosts sensitivity to insulin and lowers inflammation [\[R, R, R\]](#).

The combination of berberine and [milk thistle](#) may provide similar benefits [\[R, R, R, R\]](#).

In cell studies, berberine directly activates AMPK. Berberine can interact with some medications, so make sure to do your research and talk to your doctor before supplementing [\[R, R\]](#).

**HNF4A (Gut Inflammation)**

IMPACT

1 / 5

EVIDENCE

1 / 5

In a placebo-controlled trial of 16 people with ulcerative colitis, using 900 mg/day berberine as an add-on to mesalamine for 3 months reduced gut tissue damage better than mesalamine alone [\[R\]](#).

In studies using rat and mouse cells, berberine increased the expression of *HNF4A* [\[R, R\]](#).

Berberine may help by reinforcing the barrier function of the gut lining and promoting a healthy gut microbiome.

**TCF7L2 (Blood Sugar/Diet)**

IMPACT

4 / 5

EVIDENCE

5 / 5

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Berberine (0.5-1.5 g/day for 2-6 months) may help control blood sugar. It likely boosts sensitivity to insulin and lowers inflammation [R, R, R].

The combination of berberine and [milk thistle](#) may provide similar benefits [R, R, R, R].

Importantly, berberine may boost GLP-1 [R].

16



Cocoa

Incorporate 30-60 grams of high-quality dark chocolate (with at least 70% cocoa content) into your daily diet. Enjoy it as a snack or dessert to reap the potential heart health benefits.

Helps with these Symptoms & Conditions:

- Artery Hardening
- Cognitive Decline

Helps with these Goals:

- Cognitive Function
- Energy
- Focus
- Memory
- Mood
- Short Term Memory

Helps with these DNA Risks:

-  BDNF

How it helps

 BDNF

IMPACT

0 / 5

EVIDENCE

0 / 5

Cocoa is rich in flavonoids, compounds that stimulate the production of Brain-Derived Neurotrophic Factor (BDNF), a protein essential for brain health. Higher levels of BDNF can help prevent neurodegenerative diseases by promoting brain cell growth and survival.

17



Green Tea Extract

Take a green tea extract supplement containing 250-500 mg of EGCG (the active compound in green tea) daily, preferably with a meal to enhance absorption. This dosage is typically split into two separate doses, taken in the morning and later in the day. Continue this regimen for at least three months to observe potential health benefits.

TYPICAL STARTING DOSE

250 mg

Helps with these Symptoms & Conditions:

Artery Hardening

Helps with these Goals:

- Cognitive Function
- Energy
- Fat Loss
- Focus

Helps with these DNA Risks:

! BDNF

How it helps

! BDNF

IMPACT

0 / 5

EVIDENCE

0 / 5

Green tea catechins such as EGCG are antioxidants that might protect the brain by reducing oxidative stress and potentially improving cognitive functions.

18



Lactobacillus Plantarum

Take a probiotic supplement containing *Lactobacillus plantarum* 299V daily. The typical dosage is 10 billion colony-forming units (CFUs). Continue for at least 4 weeks to assess benefits on digestive health.

TYPICAL STARTING DOSE
10 billion CFU

Helps with these Symptoms & Conditions:

- Artery Hardening
- Bloating
- Cognitive Decline
- Food Allergies

Helps with these Goals:

- Fat Loss
- Mood

Helps with these DNA Risks:

! DRD2 (Dopamine)

How it helps

! DRD2 (Dopamine)

IMPACT

0 / 5

EVIDENCE

0 / 5

Lactobacillus Plantarum, a probiotic, can boost dopamine production by influencing your gut microbiome and the brain-gut axis. This could potentially help manage DRD2-associated issues, like dopamine regulation disorders, by increasing dopamine levels.

19

L-Tyrosine

Take 500-2000 mg of L-Tyrosine supplement daily, preferably 30 minutes before meals or snacks. This can be taken in one dose or divided into two doses throughout the day. Continue this supplementation daily for a duration of at least a few weeks to assess its effects on your body.

TYPICAL STARTING DOSE

500 mg

Helps with these Goals:

- Cognitive Function
- Creativity
- Focus
- Memory

Helps with these DNA Risks:

-  DRD2 (Dopamine)

How it helps

 DRD2 (Dopamine)

IMPACT

0 / 5

EVIDENCE

0 / 5

L-Tyrosine is a precursor to dopamine, meaning your body uses it to produce dopamine. So, taking L-Tyrosine may potentially increase dopamine levels in your body, which could be beneficial for conditions involving dopamine, such as DRD2.

Please note: Long-term safety of L-tyrosine supplementation in daily doses above **150 mg/kg** is unclear. That is about **1 g/day** for a 70-kg person.

20

Phosphatidylserine

Take 100 mg of phosphatidylserine three times daily with meals. Continue this regimen for up to 6 months to evaluate its effectiveness.

TYPICAL STARTING DOSE

300 mg

Helps with these Symptoms & Conditions:

- Cognitive Decline

Helps with these Goals:

- Cognitive Function
- Focus
- Memory
- Short Term Memory

Helps with these DNA Risks:

 BDNF

How it helps

 BDNF

IMPACT

0 / 5

EVIDENCE

0 / 5

Phosphatidylserine is a component of the cell membrane that is crucial for maintaining cellular function in the brain and may help support cognitive functions.

21



Resistant Starch

Incorporate 40g of Jo's resistant starch into your daily diet. This can be done by adding it to smoothies, yogurt, or baked goods. Ensure to spread the intake throughout the day for better tolerance.

TYPICAL STARTING DOSE
40 g Hi-Maize

Helps with these Symptoms & Conditions:

Food Allergies

Helps with these Goals:

- Fat Loss
- Mood

Helps with these DNA Risks:

-  BDNF
-  NLRP3 (Gut Inflammation)
-  TCF7L2 (Blood Sugar/Diet)

How it helps

 BDNF

IMPACT

0 / 5

EVIDENCE

0 / 5

Resistant starches act as food for gut bacteria, which then produce short-chain fatty acids that can increase the brain-derived neurotrophic factor (BDNF). Increased BDNF levels can aid in brain health and decrease the risk of neurological conditions.

 NLRP3 (Gut Inflammation)

IMPACT

2 / 5

EVIDENCE

2 / 5

In animal studies, [butyrate](#) protected the gut by blocking NLRP3 activation, which reduced inflammation and preserved gut barrier function [\[R\]](#), [\[R\]](#).

Butyrate is available as a supplement; dietary factors also affect how much butyrate is produced by your gut bacteria. The best foods to increase butyrate production are those with plenty of resistant starches—starch that you can’t digest, but that the gut flora can. Baked potatoes and green bananas are two good sources of resistant starch [\[R\]](#), [\[R\]](#), [\[R\]](#).

Resistant starch (5-20 g/day for up to 12 months) may promote remission in people with IBD. However, the quality of the evidence is low [\[R\]](#).

 TCF7L2 (Blood Sugar/Diet)

IMPACT

4 / 5

EVIDENCE

4 / 5

Resistant starch supplements (10-30 g/day) may help reduce blood sugar and prevent diabetes [\[R\]](#), [\[R\]](#), [\[R\]](#).

Resistant starch **delays the absorption of sugar**, which prevents blood sugar spikes. It may even boost your response to insulin [\[R\]](#).

 PERSONALIZED TO YOUR GENES

You carry the "hunter-gatherer" variant of the [TCF7L2](#) gene. People with your *TCF7L2* gene variant don't seem to handle carbs well. You may need to limit even high-fiber carbs like whole grains, which are protective against diabetes in other people [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#). To reap the benefits of fiber for blood sugar, try supplementing with resistant starch instead.

GENE	SNP	GENOTYPE	EVIDENCE
TCF7L2	rs7903146	CT	

22

Spend Time in Nature

Aim to spend at least 120 minutes per week in natural environments, such as parks, forests, or beaches. This can be divided into short durations throughout the week, for example, 17 minutes per day or longer sessions on weekends.

TYPICAL STARTING DOSE

2 hours

Helps with these Goals:

- Creativity
- Energy
- Longevity
- Mood

Helps with these DNA Risks:

 BDNF

How it helps

 **BDNF**

IMPACT

0 / 5

EVIDENCE

0 / 5

Being in nature can reduce stress and increase BDNF levels, aiding in brain cell growth and resilience.

23



Tai Chi

Practice Tai Chi for 30 to 60 minutes at least twice a week. Choose a quiet, spacious area and follow along with a qualified instructor, either in person at a class or through an online video tutorial, to ensure proper technique and maximum benefit.

TYPICAL STARTING DOSE

1 hour

Helps with these Symptoms & Conditions:

Cognitive Decline

Helps with these Goals:

Cognitive Function

Energy

Fat Loss

Focus

Longevity

Memory

Mood

Helps with these DNA Risks:

 **BDNF**

How it helps

 **BDNF**

IMPACT

0 / 5

EVIDENCE

0 / 5

Tai Chi incorporates physical activity and meditation, both of which have been associated with increased BDNF levels.

24



Walnuts

Incorporate a handful of walnuts (about 1 ounce or 28 grams) into your daily diet. You can eat them as a snack, add them to salads, cereals, yogurts, or use in baking recipes. Do this consistently as part of your daily dietary intake.

Helps with these Symptoms & Conditions:

- Artery Hardening
- Cognitive Decline

Helps with these Goals:

- Mood
- Short Term Memory

Helps with these DNA Risks:

-  BDNF

How it helps

 BDNF

IMPACT

0 / 5

EVIDENCE

0 / 5

Walnuts are a good source of alpha-linolenic acid (ALA), an omega-3 fatty acid that could positively impact BDNF production.

25



Vitamin B12

Take a 50 mcg vitamin B12 supplement daily, preferably with a meal to enhance absorption.

TYPICAL STARTING DOSE

100 mcg

Helps with these Symptoms & Conditions:

- Cognitive Decline
- Underactive Thyroid

Helps with these Goals:

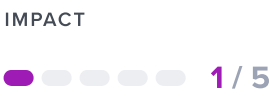
- Mood

Helps with these DNA Risks:

-  MTHFD1 (Folate/ Methylation)
-  MTR (Methylation)
-  TCN2 (Vitamin B12)

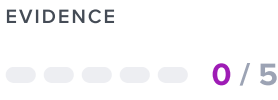
How it helps

MTHFD1 (Folate/ Methylation)



Vitamin B12 is involved in the same homocysteine removal pathway as folate. People with certain MTHFD1 variants may benefit from supplementing with this vitamin [\[R\]](#).

MTR (Methylation)



Vitamin B12 assists with the MTR (MTR being a gene that plays a significant role in methylation) as it helps to convert homocysteine to methionine, a crucial step in methylation. Methylation, a chemical reaction in the body, is important for cellular repair, detoxification, energy production, and immune response.

Vitamin B12 helps the MTR enzyme work properly and turn homocysteine into methionine [\[R\]](#).

People with high vitamin B12 levels may have lower homocysteine. Supplementation with vitamin B12 (1 mg/day) may lower homocysteine levels in older people [\[R, R, R, R\]](#).

Methylcobalamin is the active form of this vitamin used by MTR. In theory, this form may be more effective at increasing MTR activity.

TCN2 (Vitamin B12)



Some people may not eat enough foods rich in vitamin B12. Others may have trouble absorbing this vitamin from the food they eat. This may lead to vitamin B12 deficiency [\[R\]](#).

People more prone to low levels of vitamin B12 include [\[R\]](#):

- Vegetarians and vegans
- Older adults
- People with gut disorders (e.g., Crohn’s disease, celiac disease)

If your diet doesn't include sufficient food sources of vitamin B12 or you are deficient despite a normal intake of this vitamin, discuss vitamin B12 supplements with your doctor. In some cases, your doctor may prescribe vitamin B12 shots [\[R, R\]](#).



PERSONALIZED TO YOUR GENES

Your [TCN2](#) variant has been associated with lower levels of active vitamin B12. Supplementation may help counteract the negative effects of this variant [\[R\]](#).

YOUR GENETIC VARIANTS

GENE	SNP	GENOTYPE	EVIDENCE
TCN2	rs1801198	GG	

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Pyridoxine (Vitamin B6)

Take a pyridoxine (vitamin B6) supplement daily. Requirements range from 1.3 to 1.7 milligrams per day for adults, but supplement doses usually start from 50 mg. Consult with a healthcare provider for higher doses or specific medical conditions that might benefit from increased supplementation.

TYPICAL STARTING DOSE

50 mg

Helps with these DNA Risks:

 MTHFD1 (Folate/ Methylation)

How it helps

 MTHFD1 (Folate/ Methylation)

IMPACT

1 / 5

EVIDENCE

1 / 5

The body has another option for removing homocysteine by converting it to [cysteine](#) (see image). This pathway depends on [vitamin B6](#). People with certain MTHFD1 variants may benefit from supplementing with this vitamin too [\[R, R\]](#).

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Cognitive-Behavioral Therapy (CBT)

Schedule weekly sessions with a certified cognitive-behavioral therapist for a period of 5 to 20 weeks. Engage actively in exercises assigned by your therapist both during sessions and as homework to apply CBT strategies to daily life.

Helps with these Goals:

Energy

Fat Loss

Focus

Libido

Memory

Mood

Helps with these DNA Risks:

 CRHR2 (Stress/HPA Axis)

 MAOA (Dopamine/Serotonin)

How it helps

 CRHR2 (Stress/HPA Axis)

IMPACT

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EVIDENCE

5 / 5

Cognitive-behavioral therapy (CBT) is the best type of talk therapy for stress relief. CBT can help almost anyone under stress, including [\[R, R, R\]](#):

- People with anxiety
- People with depression
- Students
- Caregivers
- Cancer patients

CBT may reduce your levels of the stress hormone [cortisol](#) and balance the HPA axis. After going through CBT, people often feel less anxious and more relaxed [\[R, R, R\]](#).

 MAOA (Dopamine/Serotonin)

IMPACT

5 / 5

EVIDENCE

5 / 5

CBT (8-20h of sessions) is the best type of psychotherapy for anxiety. Health experts worldwide recommend CBT as the “gold standard” treatment for anxiety [R, R, R, R, R, R, R, R].

Two clinical trials of people with OCD associated this condition with reduced methylation of the *MAOA* gene, resulting in increased MAOA activity. In both of them, CBT reduced MAOA activity and improved the condition [R, R].

Moreover, carriers of high-activity variants may be more responsive to this type of therapy. This was the case in 2 clinical trials of people with panic disorder [R, R].

CBT can soothe anxiety by helping you [R, R, R]:

- Cope with stress
- Control negative thoughts and emotions
- Improve social skills
- Build stronger relationships

Studied forms of CBT include [R, R]:

- In-person
- Internet-based
- Self-guided

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Tryptophan

Take 500 mg of tryptophan supplement daily. This dosage can be taken all at once, preferably before bedtime to support sleep, or as directed by a healthcare professional.

TYPICAL STARTING DOSE

500 mg

Helps with these Symptoms & Conditions:

Food Allergies

Helps with these Goals:

Mood

Helps with these DNA Risks:

-  MAOA (Dopamine/Serotonin)
-  MAOB (Dopamine/ Norepinephrine)

How it helps

 MAOA (Dopamine/Serotonin)

IMPACT

2 / 5

EVIDENCE

2 / 5

Low tryptophan levels are associated with depression [R, R, R].

Foods rich in tryptophan—such as oats, bananas, dried prunes, milk, tuna, cheese, bread, chicken, turkey, peanuts, and chocolate—may help restore normal serotonin levels in people with high MAOA activity [R, R, R].

A supplement with tryptophan, [tyrosine](#), and blueberry juice designed to counteract the negative effects of high MAOA activity (increased monoamine breakdown and oxidative stress) helped prevent postpartum depression in a clinical trial. However, this supplement failed to prevent depression caused by cigarette withdrawal in smokers [R, R].

 **MAOB (Dopamine/ Norepinephrine)**

IMPACT

3 / 5

EVIDENCE

3 / 5

Low tryptophan levels are associated with depression [R, R, R].

Consider increasing your intake of tryptophan-rich foods.

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Aromatherapy

Select essential oils like lavender, peppermint, or eucalyptus. Use a diffuser to disperse the scent into your room for 30-60 minutes at a time, up to 3 times a day. Alternatively, apply a few drops diluted in a carrier oil directly to your skin, such as on your temples or wrists, twice a day.

TYPICAL STARTING DOSE

30 minutes

Helps with these Symptoms & Conditions:

Underactive Thyroid

Helps with these Goals:

Energy

Fat Loss

Memory

Mood

Helps with these DNA Risks:

 **CRHR2 (Stress/HPA Axis)**

How it helps

 **CRHR2 (Stress/HPA Axis)**

IMPACT

3 / 5

EVIDENCE

3 / 5

Aromatherapy (for 5-10 min) may help control stress by promoting relaxation [R, R].

Studied essential oils include those of [R, R, R, R, R, R, R, R, R, R, R, R, R, R, R]:

- Lavender

- Rose
- Peppermint

Oil blends may also help e.g., clary sage, lavender, and bergamot.

Some of these essential oils may suppress CRH, leading to lower cortisol levels [R].

However, note that most studies are low-quality and some show that aromatherapy doesn’t reduce stress [R, R].

Please note: Some essential oils may cause skin irritation, allergic reactions, sun sensitivity, or breathing problems. Avoid using them near the eyes, nose, or other sensitive areas. Some oils are not safe for children, pregnant women, or pets. Consult your health provider before using essential oils [R].

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Ginger

Take a 500 mg ginger supplement daily, preferably with a meal to enhance absorption and minimize potential stomach discomfort.

TYPICAL STARTING DOSE
500 mg

Helps with these Goals:

- Fat Loss
- Libido

Helps with these DNA Risks:

-  UCP1 (Weight)

How it helps

 UCP1 (Weight)

IMPACT

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EVIDENCE

3 / 5

A meta-analysis of 26 trials found that supplementation with ginger decreases BMI. Doses above 1500 mg/day taken for over 8 weeks were most effective [R].

Another meta-analysis of 14 trials and 473 participants found beneficial effects of ginger supplementation on body weight, waist-to-hip ratio, and hip ratio [R].

In a non-placebo-controlled trial of 10 overweight men, consuming ginger (2 g) with a meal enhanced thermogenesis and reduced feelings of hunger [R].

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Blackcurrants

Incorporate about 1/2 cup of fresh or frozen blackcurrants into your daily diet. You can add them to your morning yogurt, oatmeal, or smoothies. For those who prefer, blackcurrant juice or preserves can also be consumed, but be mindful of added sugars in processed forms.

Helps with these DNA Risks:

 **MAOB (Dopamine/ Norepinephrine)**

How it helps

 **MAOB (Dopamine/ Norepinephrine)**

IMPACT

1 / 5

EVIDENCE

1 / 5

In two randomized controlled trials, blackcurrant juice was found to temporarily increase monoamine levels, while also improving mood. Although this effect was attributed to inhibition of monoamine oxidase B, it was almost completely reversed 24 hours after consumption of a single (500 mg blackcurrant polyphenol) drink [\[R, R\]](#).

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Manganese supplements

Take 1-5 mg of manganese supplement daily, preferably with a meal to enhance absorption.

TYPICAL STARTING DOSE

1 mg

Helps with these DNA Risks:

 **SOD2 (Oxidative Stress)**

How it helps

 **SOD2 (Oxidative Stress)**

IMPACT

1 / 5

EVIDENCE

1 / 5

SOD2 requires manganese to function, so it’s important to make sure you get enough manganese in your diet. Higher than average manganese levels were found in the hair of people who lived to a hundred years of age. However, both manganese deficiency and excess manganese have been linked with oxidative stress and degenerative disease [\[R, R\]](#).

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Limit Saturated Fat

Reduce your intake of saturated fats by choosing lean cuts of meat, opting for low-fat or fat-free dairy products, and using cooking oils high in unsaturated fats (like olive or canola oil) instead of butter or lard. Aim to keep saturated fat to less than 10% of your total daily calories. For someone consuming 2000 calories a day, this means 20 grams or less of saturated fat per day.

Helps with these Symptoms & Conditions:

Artery Hardening

Helps with these Goals:

Fat Loss

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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:

Artery HardeningCognitive DeclineFood AllergiesHair LossHashimoto's DiseaseUnderactive Thyroid

Helps with these Goals:

Cognitive FunctionEnergyFocusLibidoLongevityMoodStrength

Helps with these DNA Risks:

⚠️ MAOA (Dopamine/Serotonin)

How it helps

⚠️ MAOA (Dopamine/Serotonin)

IMPACT

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EVIDENCE

3 / 5

Active [vitamin D](#) (calcitriol) reduced MAOA levels and increased dopamine and serotonin production in studies on brain cells. Similarly, supplementation with this vitamin reduced MAOA to normal levels in diabetic rats [\[R\]](#), [\[R\]](#), [\[R\]](#).

Vitamin D (400-5,000 IU/day for 6-8 weeks) may improve symptoms of depression. However, more research is necessary to confirm this benefit [\[R\]](#), [\[R\]](#), [\[R\]](#), [\[R\]](#).

Vitamin D may help by increasing the levels of [\[R\]](#), [\[R\]](#):

- [Dopamine](#)

- [Norepinephrine](#) (noradrenaline)
- [Epinephrine](#) (adrenaline)

Please note: Experts recommend getting 600-800 IU of vitamin D per day. Medical bodies recommend against taking more than 4,000 IU per day [\[R\]](#).

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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:

- Artery Hardening
- Cognitive Decline
- Hair Loss
- Hashimoto's Disease

Helps with these Goals:

- Cognitive Function
- Energy
- Fat Loss
- Focus
- Libido
- Longevity
- Memory
- Mood
- Short Term Memory

Helps with these DNA Risks:

- 

TCF7L2 (Blood Sugar/Diet)

How it helps



TCF7L2 (Blood Sugar/Diet)

IMPACT

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EVIDENCE

4 / 5

The Mediterranean diet may lower blood sugar levels in the long run. It’s rich in foods that help reduce inflammation and insulin resistance [\[R, R, R, R, R, R\]](#).

People who stick to the Mediterranean diet may thus be less likely to have diabetes. Studies suggest this type of diet may be the best choice for people at risk of diabetes [\[R, R, R, R\]](#).

The Mediterranean diet is rich in [olive oil](#). **Eating olive oil may lower blood sugar levels and reduce the risk of diabetes** [\[R\]](#).

 PERSONALIZED TO YOUR GENES

Your **TCF7L2** gene variant is linked to higher blood sugar levels. The Mediterranean diet may cancel out the negative effects of this variant [\[R\]](#).

YOUR GENETIC VARIANTS			
GENE	SNP	GENOTYPE	EVIDENCE
TCF7L2	rs7903146	CT	

36

Sleep for 7+ Hours

Ensure you allocate enough time in your schedule to achieve a minimum of 7 hours of sleep each night. This might involve going to bed earlier or adjusting your evening routine to promote relaxation and make it easier to fall asleep.

Helps with these Symptoms & Conditions:

- Artery Hardening
- Cognitive Decline
- Hair Loss
- Hashimoto's Disease
- Underactive Thyroid

Helps with these Goals:

- Cognitive Function
- Creativity
- Energy
- Fat Loss
- Focus
- Libido
- Longevity
- Memory
- Mood
- Short Term Memory

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Avoid Air Pollution

Stay indoors on days when air quality indexes (AQI) indicate high pollution levels, which are often reported by weather services or government environmental agencies. **Install air purifiers** in your home, especially in bedrooms, to reduce indoor pollutants. Limit outdoor exercise when air pollution warnings are issued, opting for indoor activities instead.

Helps with these Symptoms & Conditions:

- Cognitive Decline

Helps with these Goals:

- Cognitive Function
- Longevity
- Mood

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Cognitive Activity

Engage in mentally stimulating activities, such as puzzles, reading, or learning a new skill, for at least 15 minutes daily. Consistency is key, so incorporate these activities into your daily routine for ongoing cognitive health benefits.

TYPICAL STARTING DOSE
15 minutes

Helps with these Symptoms & Conditions:

Cognitive Decline

Helps with these Goals:

- Cognitive FunctionMemoryShort Term Memory

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Carnosine And Anserine

Take a combined dose of 1 to 1.5 grams of carnosine and anserine supplements daily. Divide this total dose into two smaller doses to take in the morning and evening. Continue this supplementation daily for at least 8 to 12 weeks to observe benefits.

TYPICAL STARTING DOSE
1 g

Helps with these Symptoms & Conditions:

Cognitive Decline

40

Dietary B Vitamins

Incorporate foods rich in B vitamins like whole grains, meat, eggs, dairy products, leafy green vegetables, beans, peas, and nuts into your daily meals. For a balanced intake, aim to include at least one serving of a B vitamin-rich food in each meal throughout the day.

Helps with these Symptoms & Conditions:

- Cognitive DeclineHair Loss

Helps with these Goals:

- Energy
- Mood

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Dietary Nitrates

Incorporate foods high in dietary nitrates, such as beets, spinach, arugula, and celery, into your daily meals. Aim for about 300-400 mg of dietary nitrates daily, which equates to roughly 2 cups of nitrate-rich vegetables.

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Power Training

Engage in power training exercises, such as squats, lunges, and bench presses, at least twice per week. Each session should include 3-5 sets of 6-10 repetitions for each exercise, with a focus on explosive, powerful movements. Rest for at least 48 hours between power training sessions to allow for muscle recovery.

TYPICAL STARTING DOSE

1 hour

Helps with these Goals:

- Strength

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Avoid Cannabis

Stop any form of cannabis use, including smoking, vaping, edibles, and topical applications. If you currently use cannabis, it's recommended to cease usage immediately and seek support or counseling if necessary for cessation.

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Mindfulness

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

TYPICAL STARTING DOSE

30 minutes

Helps with these Symptoms & Conditions:

Cognitive Decline

Helps with these Goals:

- Cognitive Function
- Creativity
- Energy
- Fat Loss
- Focus
- Libido
- Longevity
- Memory
- Mood
- Short Term Memory

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SAM-e

Take 400-1600 mg of SAM-e as a supplement daily, preferably on an empty stomach to enhance absorption. It is often recommended to start with low dosage and observe how your body responds over a few weeks, adjusting as necessary under the guidance of a healthcare provider.

TYPICAL STARTING DOSE
400 mg

Helps with these Goals:

- Longevity
- Mood

46



Core-Strengthening Exercises

Incorporate exercises like planks, bridges, abdominal crunches, and leg lifts into your daily routine. Aim to do these exercises for 20-30 minutes, 3 times a week. Each exercise should be performed in sets of 10-15 repetitions.

TYPICAL STARTING DOSE
20 minutes

Helps with these Goals:

- Strength

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N-acetylcysteine (NAC)

Take 600 mg of N-Acetylcysteine (NAC) supplement daily with water. It can be taken at any time of the day, but try to take it at the same time each day for best results.

TYPICAL STARTING DOSE

1200 mg

Helps with these Goals:

- Energy
- Mood

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Sufficient Calorie Intake

Consume a daily amount of calories that meets your body's energy needs based on your age, sex, weight, height, and physical activity level. Use an online calorie calculator or consult a dietitian to determine your specific caloric needs. Aim to distribute your calorie intake evenly across meals throughout the day.

Helps with these Goals:

- Energy

49



Tennis

Helps with these Goals:

- Fat Loss

50



Whole-Body Vibration

Stand, sit, or lie on a machine with a vibrating platform for about 15 minutes a day, three times a week. Make sure to start with a lower intensity and gradually increase as you get more accustomed to the vibrations.

TYPICAL STARTING DOSE

15 minutes

Helps with these Goals:

- Fat Loss
- Strength

