Helisoma

DNA-BASED WELLNESS REPORT

Genome Report

Full overview - all matched categories - your DNA in plain language, narrowed to what actually matters for you.

235	83	5	2026-08-24
FINDINGS	TRAITS	DOMAINS	GENERATED

You have carried this your whole life. It is written into every cell, quietly shaping how you sleep, what fuels you, and how you handle a hard week. We just turn it into plain language so you can finally read it for yourself. Nothing here is a diagnosis. Each finding is a small leaning, the kind that suddenly explains an old hunch, like why a late coffee can cost you the whole night. On its own it asks nothing of you, and that is the freedom in it: no stakes, no alarm, just a clearer sense of how you are built. It is yours to test, to live with, and to share with a coach or a doctor whenever it helps.

IMPORTANT

A wellness and educational report, not a medical test. Read each finding as a gentle lean, and take anything health-related to your doctor. The full method is on the next page.

How to read this report

SNP | Your genotype

rs... is the genetic marker. Your genotype is the two DNA letters you carry on the GRCh38 forward (+) strand - the interpretation text is written for exactly this genotype.

Strand

GRCh38 forward. We normalize your file to this strand so the letters always match the text, even if your raw file reported the opposite strand.

Multi-allelic positions

Some positions list a third base in certain databases. When that base does not occur in the population (0% frequency), we show only the two alleles that actually vary - so Ref/Alt reflects what real people carry.

ClinVar review

A few markers here have their own entry in ClinVar, the public database of clinically reviewed variants. Those cards carry ClinVar's review status verbatim and its 0-4 star rating, which measures how much expert review stands behind the classification - "reviewed by expert panel" at the top, "no assertion criteria provided" at the bottom, "conflicting classifications" when submitters disagree. It rates the review, not the size of the effect, and a low rating is a reason to hold the finding loosely rather than a reason to ignore it.

Dosage (0/0 | 0/1 | 1/1)

How many copies of the alternate (ALT) base you carry. ALT just means "non-reference" - it is NOT automatically the "bad" allele.

Ref / Alt | Studied allele

Ref/Alt are the reference vs alternate base. The reference base is the letter GRCh38 carries at that position, not necessarily the more common allele, so a common genotype can read as Dosage 1/1. The "Studied allele (you carry N/2)" is the allele the research measured the effect for, and how many copies you carry. Whether that allele raises or lowers the trait is stated in each card's own sentence - so read the sentence, not Ref/Alt, to see what it means for you. For some markers the research doesn't single out one allele; those cards show "Read per genotype" instead, and the reading for your exact genotype (0/0, 0/1, 1/1) is given directly in the card text, with the source linked.

Marker coverage (3/15)

When a topic could not use every marker it reads, its header shows the ratio, e.g. "3/15 markers". The rest were missing from your file, or present but not readable reliably (some chip formats cannot pin down certain markers); either way they are absent from the reading, not negative. A topic read from a small fraction of its markers rests on thinner evidence, so weigh it more loosely than one where everything arrived. Topics where every marker could be used show a plain count instead.

Evidence & sources

Each finding links to the original PubMed studies or ClinVar records so you can read the source. Strength reflects study size and consistency - a tendency seen across several large studies is firmer than a small or single-study signal.

Why some findings repeat

Markers that sit close together on a chromosome are usually inherited together as a block (linkage disequilibrium), so several cards near the same gene often describe the same underlying signal seen across different studies. We list them individually so each statement can be traced to its own source - read such a cluster as one finding reinforced by several reports, not as separate, independent evidence. The same marker can also appear under more than one topic where the research links it to several traits; that is expected, and does not mean the effects add up.

Evidence types

Three kinds, shown on each card. Population study: an average tendency seen across many people, not a prediction about you. Substance response: how your body processes caffeine, alcohol, or nicotine, and how strongly it affects you. Cataloged variant: a spot in your DNA with its own reviewed entry in a medical reference database.

What we leave out

A marker rarely points at one trait alone. From all the associations reported for it, we keep the wellness-relevant ones that replicate best and set the rest aside - disease links, and the faintest signals. Each card is a selection of a marker's clearest tendencies, not a list of everything ever found; its best-known role may even be a disease we leave out.

Reading it safely

These are tendencies, not predictions or diagnoses. The signals are population-level, and most traits also depend on lifestyle, environment, and many other genes - so treat each card as a gentle leaning, not a verdict about you.

METHOD

Methods & limits

How this report was made, and what it cannot tell you.

What we did	We read the variants in your consumer DNA file (the kind from 23andMe, AncestryDNA and similar) and matched them to published research, then summarized each one in plain language. We focus on small, everyday wellness tendencies, not disease.
How current this is	The knowledge base behind this report was last updated in August 2026, and each card links the study or database record it rests on so you can check its own date. Research gets revised, so a tendency described here can read differently in a few years. Your report is generated fresh every time you download it, so re-downloading gives you the current reading.
Did we read your DNA right?	Almost certainly yes. Home DNA chips are very good at the common variants used here: they agree with full lab sequencing about 99% of the time. You may have read that home DNA tests are unreliable; that is about rare disease variants, like cancer-risk genes, which chips genuinely struggle with, so this is a wellness reading, not a clinical test, and we never report those as findings. We take the file as given, so we cannot catch a mis-read marker inside it.
Do we know what it means for you?	Only roughly. And this, not the reading of the letters, is the soft part. Each variant is a gentle nudge measured across large groups of people, not a fact about you. Once in a while a published study even links a variant to the wrong trait, which is why every card links its source so you can check. For anything medical, see a doctor or genetic counselor and get a clinical test (this is also the CDC's guidance).
Why you will not see many numbers	We describe each effect as "small / typical / leans a little" rather than printing odds ratios and p-values. Those come mostly from large European-ancestry studies and do not transfer cleanly to every person, so showing them would imply a precision we honestly do not have.

Why it is still worth knowing	This is the part you actually take with you. Out of everything that could shape your body, the report points to the specific few things worth your attention, so instead of generic advice you get a shortlist that actually fits you. Think of it as a map of where you tend to lean: it shows what to notice first, what is worth experimenting with, and which questions to bring to a doctor. Each lean is gentle, but you carry it for life, so a small adjustment repeated over years quietly adds up. And much of the value arrives at the right moment: you learn that you clear caffeine slowly, and nothing changes, until the night you lie awake after a late coffee and it finally makes sense. A loose hunch that coffee hits you hard becomes a named tendency you can test on yourself, and your coach can work from how you are actually built, not from an average stranger. And it is simply yours to know: this is your own biology, the research already exists, and curiosity is reason enough. Because the stakes are deliberately low, you are free to explore.
Ancestry	Most of the effect sizes behind this research were measured in European-ancestry groups. Allele frequencies differ between populations, and the marker on a consumer chip is often a stand-in sitting near the causal variant rather than the variant itself, at a distance that also differs by population. So an effect measured in one group can be smaller, larger, or absent in another. We do not recalibrate for ancestry and do not claim to. If your ancestry is not primarily European, read every lean here as looser still.
Bottom line	Nothing here should change a medication, a diagnosis or a big health decision on its own.

01

Body & fitness

21 traits
67 findings

Muscle composition, endurance ceiling, body weight and recovery.

01.01 Blood pressure tendency

8/10 markers

Blood pressure tendency ALDH2

POPULATION STUDY

rs671 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

Two G copies, no A allele. In East Asian populations the A allele tracks with slightly lower blood pressure, partly because A causes the alcohol-flush reaction so carriers drink less, though a more direct vascular effect is debated; G/G carries none of that flush-linked effect. A small, population-level pattern.

EVIDENCE

In population studies, the A allele at this ALDH2 marker is associated with slightly lower blood pressure, and part of this likely reflects reduced drinking: the A allele (common in East Asian populations, rare elsewhere) causes the alcohol-flush reaction, so carriers tend to drink less, and lower alcohol intake lowers blood pressure. Whether the variant also acts more directly on blood vessels is debated, so the mechanism is not settled. A population-level tendency, not a medical prediction; diet, salt and potassium balance, activity, weight and sleep influence blood pressure far more.

SOURCES

PubMed 36233190 | PubMed 39048560 | PubMed 41431988

Blood pressure tendency

HFE

POPULATION STUDY

rs1799945 | GT C/G | DOSE 0/1 | REF/ALT C/G | STUDIED G (1 of 2) | GRCh38 +

INTERPRETATION

One G variant and one C baseline allele - a heterozygous C/G genotype. In research cohorts this genotype is linked to slightly higher measured blood pressure than the C/C baseline. This is a small, population-level shift.

EVIDENCE

In population studies, people who carry the G allele at this HFE marker tend to have slightly higher blood pressure. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - sodium and potassium in the diet, regular movement, body weight, and how well stress is managed - influence blood pressure far more.

SOURCES

PubMed 27618447 | PubMed 27841878 | PubMed 21909115

Blood pressure tendency

NT5C2 region

POPULATION STUDY

rs11191580 | GT T/T | DOSE 0/0 | REF/ALT T/C | STUDIED C (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the T allele - a T/T genotype. In population studies the T allele is associated with modestly higher blood pressure, so a T/T reading sits slightly toward the higher-BP end of this single-marker range. This is a small leaning, not a diagnosis; the tiny push from one common variant is dwarfed by everyday factors like salt, weight, activity and the rest of your genome.

EVIDENCE

In population studies, people who carry the C allele in the NT5C2 region tend to have slightly lower blood pressure, while the T allele leans slightly higher. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - physical activity, sodium and potassium intake, healthy weight, and limiting alcohol - influence blood pressure far more.

SOURCES

PubMed 38459180 | PubMed 38689001 | PubMed 30487518

Blood pressure tendency

AGT M235T

POPULATION STUDY

rs699 | GT A/A | DOSE 0/0 | REF/ALT A/G | STUDIED G (0 of 2) | GRCh38 +

INTERPRETATION

Both copies here carry the A allele - an A/A genotype, with no copies of the blood-pressure-raising G allele. In population studies measured blood pressure at this site sits toward the lower end of a very small range; each G copy is associated with only a tiny upward nudge.

EVIDENCE

In population studies, people who carry the G allele at rs699 (AGT M235T) tend to have very slightly higher blood pressure. It's a tiny, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - salt intake, body weight, activity, and stress - influence blood pressure far more.

SOURCES

PubMed 30578418 | PubMed 38689001 | PubMed 39548300

Blood pressure tendency

CNNM2

POPULATION STUDY

rs12413409 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED G (2 of 2) | GRCh38 +

INTERPRETATION

With the G/G genotype you carry two copies of the G allele. At this 10q24 (CYP17A1-CNNM2) locus the G allele carries a weak population-level association with higher blood pressure; on its own it does not meaningfully predict your individual blood pressure, which a cuff reading captures directly.

EVIDENCE

In population studies, people who carry the G allele at this 10q24 (CYP17A1-CNNM2) variant tend to have slightly higher blood pressure, while the A allele is the lower-leaning version. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - staying active, managing salt and alcohol, sleep, and keeping a steady weight - influence blood pressure far more.

SOURCES

Source

Blood pressure tendency

MTHFR

POPULATION STUDY

rs1801133 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the G allele - a G/G genotype, with no copies of the A allele. In population studies, each A copy is associated with a modestly higher blood-pressure measurement, so G/G sits at the lower end of this weak, population-level association.

EVIDENCE

In population studies, people who carry the A allele at this MTHFR marker tend to have slightly higher blood pressure. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - sodium and potassium in the diet, regular movement, body weight, and how well stress is managed - influence blood pressure far more.

SOURCES

PubMed 39048560

Blood pressure tendency

NOS3

POPULATION STUDY

rs3918226 | GT C/C | DOSE 0/0 | REF/ALT C/T | STUDIED T (0 of 2) | GRCh38 +

INTERPRETATION

Both copies carry the C baseline variant - a C/C genotype, with no copies of the T variant. In population studies this sits at the lower end of the blood-pressure association relative to T-carriers. Each T copy is linked to a small leaning toward higher blood pressure - a population-level shift, not a personal prediction.

EVIDENCE

In population studies, people who carry the T allele in NOS3 tend to have slightly higher blood pressure. NOS3 sits in a pathway that helps regulate blood-vessel tone and fluid balance. It's a small, common genetic leaning, not a prediction about any one person - many other genes and everyday habits, such as salt intake, body weight, regular activity, and alcohol, influence blood pressure far more.

SOURCES

PubMed 38689001 PubMed 38965376

Blood pressure tendency

SH2B3

POPULATION STUDY

rs3184504 | GT C/C | DOSE 1/1 | REF/ALT T/C | STUDIED C (2 of 2) | GRCh38 +

INTERPRETATION

Both copies carry the C allele - a C/C genotype, with no T copies. In research, blood pressure tends to sit at the lower end relative to T-carriers; each T copy would nudge the measurement up slightly. This is a small population-level shift, not a medical prediction.

EVIDENCE

In population studies, people who carry the C allele in SH2B3 tend to have slightly lower blood pressure. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - salt intake, body weight, regular activity, and alcohol - influence blood pressure far more.

SOURCES

PubMed 29455858 | PubMed 39537608

01.02 Body weight & BMI tendency

35/38 markers

BMI & body-weight tendency

MC4R

POPULATION STUDY

rs12970134 | GT G/A | DOSE 0/1 | REF/ALT G/A | STUDIED A (1 of 2) | GRCh38 +

INTERPRETATION

G/A genotype - one A copy and one G copy. In population studies this is linked to a modestly higher body mass index and weight than G/G, with a small per-copy effect.

EVIDENCE

The A allele near MC4R is associated in population studies with higher body mass index and body weight. The per-allele effect is modest and well established - a small per-copy effect on BMI and body weight. MC4R is one of the best-known common body-weight loci, so the direction is solid, though the per-copy effect is small relative to overall lifestyle factors. Note: this is one of 5 correlated variants in/near MC4R reported for this trait. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal - not 5 independent confirmations. Many other genetic and lifestyle factors also influence body weight. This variant is reported for more than one trait; the per-trait effects come from different studies and are not directly comparable.

SOURCES

PubMed 19079260 | PubMed 18454146

Body weight & BMI tendency

FAIM2

POPULATION STUDY

rs7138803 | GT A/A | DOSE 1/1 | REF/ALT G/A | STUDIED A (2 of 2) | GRCh38 +

INTERPRETATION

Both copies here carry the A allele - an A/A genotype. In research cohorts this is linked to a slightly higher body-weight / BMI measurement than G/G. The per-copy effect is small - a relative, population-level shift, not a prediction of any individual's weight. Many other factors also influence body weight.

EVIDENCE

In population studies, each A allele at rs7138803 in FAIM2 is associated with a slightly higher body-weight / BMI measurement, a small per-copy effect. Body weight is a measurement rather than a medical condition, and the per-copy effect is small - a relative, population-level shift that does not on its own meaningfully predict any individual's weight. Many other genetic and lifestyle factors also influence body weight. Well-replicated.

SOURCES

PubMed 33045005 | PubMed 26604143 | PubMed 19079260

Body weight & BMI tendency

FTO region

POPULATION STUDY

rs9941349 | GT C/T | DOSE 0/1 | REF/ALT C/T | STUDIED T (1 of 2) | GRCh38 +

INTERPRETATION

You have the C/T genotype - one T and one C allele, one from each parent. In research cohorts the T allele is associated with a higher body weight. This is a population-level association, not a medical prediction, and lifestyle matters too.

EVIDENCE

In population studies, each T allele at rs9941349 in the FTO region is associated with a higher body weight, with a per-copy effect. This is a measurement, not a medical prediction, and FTO's effect is strongly modified by physical activity and diet. The evidence is strong. Note: this is one of several correlated variants in/near FTO reported for this trait. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal - not several independent confirmations.

SOURCES

PubMed 19553259

Body weight & BMI tendency

FTO

POPULATION STUDY

rs8050136 | GT C/A | DOSE 0/1 | REF/ALT C/A | STUDIED A (1 of 2) | GRCh38 +

INTERPRETATION

You have the C/A genotype - one A and one C allele. In research cohorts, people with one A copy tend toward modestly higher body weight than C/C carriers, with a small per-copy effect. Diet and activity matter more than this single marker.

EVIDENCE

In population studies, each A allele at rs8050136 in FTO is associated with a higher body weight, with a small per-copy effect. This is a measurement, not a medical prediction, and FTO's effect is strongly modified by physical activity and diet. The evidence is strong and well-replicated. Note: this is one of several correlated variants in/near FTO reported for this trait. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal - not several independent confirmations.

SOURCES

PubMed 19079260 | PubMed 24348519

Body weight & BMI tendency

FTO

POPULATION STUDY

rs9930506 | GT A/G | DOSE 0/1 | REF/ALT A/G | STUDIED G (1 of 2) | GRCh38 +

INTERPRETATION

You have the A/G genotype - one G and one A allele, one from each parent. In research cohorts the G allele is associated with a modestly higher body weight. This is a population-level association, not a medical prediction.

EVIDENCE

In population studies, each G allele at rs9930506 in FTO is associated with a modestly higher body weight, with a small per-copy effect. This is a measurement, not a medical prediction, and lifestyle matters more than the genotype. The evidence is moderate. Note: this is one of several correlated variants in/near FTO reported for this trait. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal - not several independent confirmations.

SOURCES

PubMed 17658951

Body weight & BMI tendency

FTO

POPULATION STUDY

rs9939609 | GT T/A | DOSE 0/1 | REF/ALT T/A | STUDIED A (1 of 2) | GRCh38 +

INTERPRETATION

You inherited one A allele and one T allele at rs9939609 - a heterozygous T/A genotype. The A allele is associated with a modestly higher body weight. The effect is small per person and strongly modified by lifestyle, so on its own it does not meaningfully predict your weight.

EVIDENCE

In population studies, each A allele at rs9939609 in FTO is associated with a modestly higher body weight on average. This is a population-level association, not a direct medical prediction (strong evidence). It is one of several correlated variants in/near FTO reported for this trait - neighboring variants are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal rather than many independent confirmations. The per-person effect is small and strongly modified by diet and physical activity, and many other genetic and lifestyle factors also play a role.

SOURCES

PubMed 17434869 | PubMed 19079261

Body weight & BMI tendency

TFAP2B

POPULATION STUDY

rs987237 | GT A/G | DOSE 0/1 | REF/ALT A/G | STUDIED G (1 of 2) | GRCh38 +

INTERPRETATION

You have the A/G genotype - one G and one A allele, one from each parent. In research cohorts the G allele is associated with a modestly higher body weight. This is a population-level association, not a medical prediction, and lifestyle matters more than the genotype.

EVIDENCE

In population studies, each G allele at rs987237 (TFAP2B) is associated with a modestly higher body weight, with a small per-copy effect. This is a measurement, not a medical prediction, and lifestyle matters more than the genotype. The evidence is strong.

SOURCES

PubMed 23563607 | PubMed 20935630 | PubMed 26604143

Body-weight & BMI tendency

MC4R

POPULATION STUDY

rs2229616 | GT C/C | DOSE 0/0 | REF/ALT C/T | STUDIED T (0 of 2) | GRCh38 +

INTERPRETATION

C/C genotype - no copies of the BMI-lowering T allele. In population studies this MC4R marker sits slightly above T-carriers on the body-weight measure. The per-allele T effect is small, so this single marker does not meaningfully predict individual weight on its own.

EVIDENCE

At this MC4R variant, each T allele is associated with a slightly lower body weight / BMI in population studies, with a small per-copy effect. This is a population-level nudge, not a medical prediction - a weak population-level shift that, on its own, does not meaningfully predict your individual weight, which depends far more on diet and activity. Note: this is one of 5 correlated variants in/near MC4R reported for this trait - neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal, not 5 independent confirmations.

SOURCES

PubMed 36581621

Body-weight & BMI tendency

NEGR1

POPULATION STUDY

rs2815752 | GT A/A | DOSE 1/1 | REF/ALT G/A | STUDIED A (2 of 2) | GRCh38 +

INTERPRETATION

A/A genotype - two copies of the A allele. In research cohorts the A allele is associated with a modestly higher body weight / BMI, with a small per-copy effect. Even with two copies this is a weak population-level association; on its own it does not meaningfully predict individual weight, which depends far more on diet and activity.

EVIDENCE

At this NEGR1 marker, each A allele is associated with a modestly higher body weight / BMI in population studies, with a small per-copy effect. This is a population-level nudge, not a medical prediction - a weak population-level shift that, on its own, does not meaningfully predict your individual weight, which depends far more on diet and activity. Limited replication so far.

SOURCES

PubMed 20935630 | PubMed 19079261

Body-weight & BMI-associated variant MC4R

POPULATION STUDY

rs17782313 | GT T/C | DOSE 0/1 | REF/ALT T/C | STUDIED C (1 of 2) | GRCh38 +

INTERPRETATION

One C variant and one T baseline allele - a heterozygous T/C genotype. In research cohorts this genotype is linked to a slightly higher average body weight than the T/T baseline. This is a small, population-level shift, not a personal prediction.

EVIDENCE

rs17782313 sits near MC4R, a gene that helps regulate appetite. In population studies, each copy of the C allele is associated with a modestly higher body weight - a small per-copy effect (a relative, population-level shift, not your absolute chance). This is one of a few correlated variants in or near MC4R reported for this trait; because neighboring variants are usually inherited together, treat them as largely one shared signal rather than several independent confirmations. Many other genetic and lifestyle factors also influence body weight.

SOURCES

PubMed 19151714 | PubMed 19079261 | PubMed 18454148

Body-weight-associated variant FTO

POPULATION STUDY

rs1558902 | GT T/A | DOSE 0/1 | REF/ALT T/A | STUDIED A (1 of 2) | GRCh38 +

INTERPRETATION

T/A genotype - one A allele and one T allele. In population studies the A allele is associated with a modestly higher body weight / BMI, with a small per-copy effect. This is a measurement nudge and a weak population-level association; on its own it does not meaningfully predict individual weight, which depends heavily on diet, activity and many other genes.

EVIDENCE

In population studies, each A allele is associated with modestly higher body weight / BMI, with a small per-copy effect. This is a relative, population-level shift - not your absolute chance. Note: this is one of 11 correlated variants in/near FTO reported for this trait. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal - not 11 independent confirmations. FTO's effect is strongly modified by physical activity and diet, and many other factors also influence body weight. This variant is reported for more than one trait; the per-trait effects come from different studies and are not directly comparable.

SOURCES

PubMed 23563607 | PubMed 21544081 | PubMed 25673413

Higher BMI & body weight tendency

TMEM18

POPULATION STUDY

rs7561317 | GT A/G | DOSE 0/1 | REF/ALT A/G | STUDIED G (1 of 2) | GRCh38 +

INTERPRETATION

One G allele and one A baseline allele - a heterozygous A/G genotype. In research cohorts the tendency toward higher BMI and body weight is modestly above the A/A baseline - a small per-copy increase.

EVIDENCE

In population studies, each G allele at rs7561317 (near TMEM18) is associated with higher BMI and body weight, a small per-copy effect. This is a relative, population-level shift, not a medical prediction - not an absolute chance. It is one of two correlated variants near TMEM18, usually inherited together (linkage disequilibrium), so treat them as largely one shared signal rather than two independent confirmations. Many other genetic and lifestyle factors also influence body weight. Replicated evidence.

SOURCES

PubMed 19079260

Higher-BMI-associated variant

FTO

POPULATION STUDY

rs1421085 | GT T/C | DOSE 0/1 | REF/ALT T/C | STUDIED C (1 of 2) | GRCh38 +

INTERPRETATION

T/C genotype - one C variant and one T allele. In population studies the C allele is associated with a modestly higher BMI, with a small per-copy effect; on its own it does not meaningfully predict your individual outcome.

EVIDENCE

In population studies, each C allele is associated with modestly higher BMI, with a small per-copy effect. This is a relative, population-level shift - not your absolute chance. Note: this is one of 11 correlated variants in/near FTO reported for this trait. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal - not 11 independent confirmations. FTO's effect is strongly modified by physical activity and diet, and many other factors also influence body weight. This variant is reported for more than one trait; the per-trait effects come from different studies and are not directly comparable.

SOURCES

PubMed 26287746 | GWAS rs1421085

Body mass index tendency

MTCH2

POPULATION STUDY

rs10838738 | GT A/G | DOSE 0/1 | REF/ALT A/G | STUDIED G (1 of 2) | GRCh38 +

INTERPRETATION

You carry one G allele and one A allele - a heterozygous A/G genotype, one allele from each parent. In research cohorts measured BMI tends to sit marginally above the A/A group, with a small per-copy effect of the G allele. This is a tiny nudge in a measurement, easily outweighed by diet and activity.

EVIDENCE

In population studies, the G allele at this MTCH2 variant (rs10838738) is associated with a marginally higher measured body mass index, with a small per-copy effect. MTCH2 is an established common-variant BMI locus, but the per-copy effect on this measurement is very small and the association in our evidence rests on a single study - treat this as a minor informational tendency in a routine measurement, not a meaningful personal prediction. Body weight is strongly shaped by diet and activity. Note: this is one of 2 correlated variants in/near MTCH2 reported for this trait - neighboring variants are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal, not 2 independent confirmations.

SOURCES

PubMed 19079261

Body mass index tendency

PPARG

POPULATION STUDY

rs1801282 | GT C/G | DOSE 0/1 | REF/ALT C/G | STUDIED G (1 of 2) | GRCh38 +

INTERPRETATION

You inherited one G allele and one C allele - a heterozygous C/G genotype, one from each parent. In population studies the G allele is associated with a very slightly higher BMI measurement. The effect is tiny and, on its own, does not meaningfully predict your BMI.

EVIDENCE

In population studies, each G allele at rs1801282 (PPARG Pro12Ala) is associated with a very slightly higher body-mass-index measurement. This is a relative, population-level shift, not your absolute chance, and the per-copy effect is tiny, so on its own it does not meaningfully predict your individual BMI. BMI is shaped mostly by diet, activity and many other factors.

SOURCES

PubMed 36581621 | PubMed 29273807 | PubMed 30108127

Body weight & BMI tendency

ADCY3

POPULATION STUDY

rs713586 | GT C/C | DOSE 1/1 | REF/ALT T/C | STUDIED C (2 of 2) | GRCh38 +

INTERPRETATION

Both copies here carry the C allele - a C/C genotype. In research cohorts this is linked to a slightly higher BMI / body weight than T/T. The per-copy effect is small - a relative, population-level shift, not a prediction of any individual's weight. Many other factors also influence body weight.

EVIDENCE

In population studies, each C allele at rs713586 (near ADCY3) is associated with a slightly higher BMI / body-weight measurement, a small per-copy effect. This is a relative, population-level shift, not a medical diagnosis, and the per-copy effect is small - it does not on its own meaningfully predict any individual's weight. It is one of two correlated variants near ADCY3, usually inherited together (linkage disequilibrium), so treat them as largely one shared signal rather than two independent confirmations. Many other genetic and lifestyle factors also influence body weight. Well-replicated.

SOURCES

PubMed 28892062 | PubMed 20935630

Body weight & BMI tendency

BDNF

POPULATION STUDY

rs11030104 | GT A/A | DOSE 0/0 | REF/ALT A/G | STUDIED G (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the A allele - an A/A genotype. In population studies the A allele is associated with a modestly higher body weight, so an A/A reading sits slightly toward the higher end of this single-marker range. This is a small leaning, not a medical condition finding; one common variant has only a tiny effect, and actual body weight is shaped far more by overall genetics, diet and activity.

EVIDENCE

In population studies each G allele at rs11030104 in BDNF is associated with slightly lower body weight (a small per-copy effect - a relative, population-level shift, not your absolute chance). This is a population-level leaning, not a medical prediction. Strong evidence. Note: this is one of 2 correlated variants in/near BDNF reported for this trait; neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal - not 2 independent confirmations. Many other genetic and lifestyle factors also influence body weight. This variant is reported for more than one trait; the per-trait findings come from different studies and are not directly comparable.

SOURCES

PubMed 25673413 | PubMed 24861553

Body weight & BMI tendency

ETV5

POPULATION STUDY

rs7647305 | GT C/C | DOSE 1/1 | REF/ALT T/C | STUDIED C (2 of 2) | GRCh38 +

INTERPRETATION

Both copies here carry the C allele - a C/C genotype. In research cohorts this is linked to a slightly higher body-weight measurement than T/T. The effect is small - a relative, population-level shift, not a prediction of any individual's weight. Many other factors also influence body weight.

EVIDENCE

In population studies, each C allele at rs7647305 (ETV5 / SFRS10 region) is associated with a slightly higher body-weight measurement, with a small per-copy effect. This is a measurement, not a medical diagnosis, and the effect is small - a relative, population-level shift that does not on its own meaningfully predict any individual's weight. Many other genetic and lifestyle factors also influence body weight. This is well-replicated.

SOURCES

PubMed 19079260

Body weight & BMI tendency

FTO

POPULATION STUDY

rs1121980 | GT G/A | DOSE 0/1 | REF/ALT G/A | STUDIED A (1 of 2) | GRCh38 +

INTERPRETATION

You inherited one A variant and one G allele - a heterozygous G/A genotype. In research cohorts the A allele is associated with a modestly higher body weight and BMI, with a small per-copy effect - a weak population-level shift that on its own does not meaningfully predict your individual weight.

EVIDENCE

In population studies, each A allele is associated with a modestly higher body weight and BMI, with a small per-copy effect. These are measurements, not a medical condition; this is a weak, relative population-level shift and not a prediction of your individual weight. Note: this is one of 11 correlated variants in/near FTO reported for this trait. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal - not 11 independent confirmations. Many other genetic and lifestyle factors also influence body weight. This variant is reported for more than one trait; the per-trait effects come from different studies and are not directly comparable.

SOURCES

PubMed 20824172

Body weight & BMI tendency

KCTD15

POPULATION STUDY

rs29941 | GT G/G | DOSE 1/1 | REF/ALT A/G | STUDIED G (2 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the G allele - a G/G genotype. In population studies people with G/G sit at the slightly higher end of the BMI range for this site, but the per-copy effect is very small and not individually meaningful.

EVIDENCE

At this KCTD15 marker, each G allele is associated with very slightly higher body weight / BMI in population studies, a small per-copy effect. The per-allele effect is tiny and is not a meaningful personal signal. Many other genetic and lifestyle factors also influence body weight.

SOURCES

PubMed 19079260 | PubMed 28892062 | PubMed 25673413

Body weight & BMI tendency

MC4R

POPULATION STUDY

rs571312 | GT C/A | DOSE 0/1 | REF/ALT C/A | STUDIED A (1 of 2) | GRCh38 +

INTERPRETATION

One A variant and one C reference - a heterozygous C/A genotype, one from each parent. In population studies the A allele is associated with a slightly higher BMI - too small to be a meaningful personal signal on its own.

EVIDENCE

In population studies, each copy of the A allele near MC4R is associated with a slightly higher body-weight / BMI on average, a small per-copy effect. This is a relative, population-level shift, not a medical prediction - the per-copy shift is small. Note: this is one of several correlated variants in/near MC4R reported for this trait; neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal, not many independent confirmations. Many other genetic and lifestyle factors also influence body weight. This variant is reported for more than one trait; the per-trait effects come from different studies and scales and are not directly comparable.

SOURCES

PubMed 25953783 | PubMed 20935630

Body weight & BMI tendency

MC4R

POPULATION STUDY

rs6567160 | GT T/C | DOSE 0/1 | REF/ALT T/C | STUDIED C (1 of 2) | GRCh38 +

INTERPRETATION

One C allele and one T allele - a heterozygous T/C genotype, one from each parent. In population studies this is associated on average with a slightly higher BMI/body weight than T/T. This is a weak population-level association and does not on its own meaningfully predict individual weight.

EVIDENCE

In population studies, each copy of the C allele near MC4R is associated with a slightly higher body-weight / BMI on average, a small per-copy effect. This is a relative, population-level shift, not a medical prediction - the per-copy shift is small. Strong evidence. Note: this is one of several correlated variants in/near MC4R reported for this trait; neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal, not many independent confirmations. Many other genetic and lifestyle factors also influence body weight. This variant is reported for more than one trait; the per-trait effects come from different studies and scales and are not directly comparable.

SOURCES

PubMed 36581621 | PubMed 29381148 | PubMed 31504550

Body weight & BMI tendency

MTCH2

POPULATION STUDY

rs3817334 | GT C/T | DOSE 0/1 | REF/ALT C/T | STUDIED T (1 of 2) | GRCh38 +

INTERPRETATION

You inherited one T allele and one C reference - a heterozygous C/T genotype, one from each parent. In population studies the T allele is associated with a slightly higher BMI - too small to be a meaningful personal signal on its own.

EVIDENCE

At this MTCH2 marker, each T allele is associated with a slightly higher body weight / BMI in population studies, a small per-copy effect. BMI is a measurement, not a medical condition; the per-copy effect is very small - a relative, population-level shift that, on its own, does not meaningfully predict an individual's weight. Note: this is one of a couple of correlated variants in/near MTCH2 reported for this trait. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal - not independent confirmations. Many other genetic and lifestyle factors also influence body weight.

SOURCES

PubMed 25673413 | PubMed 20935630

Body weight & BMI tendency

NRXN3

POPULATION STUDY

rs10146997 | GT A/A | DOSE 0/0 | REF/ALT A/G | STUDIED G (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the A allele - an A/A genotype, with no copies of the G allele. At this NRXN3 marker A tracks with the slightly lower end of the body-weight measurement. Each G copy is associated with a modestly higher body weight - a small nudge, not a medical prediction.

EVIDENCE

In population studies, each G allele in NRXN3 is associated with a modestly higher body weight on average. This is a population-level nudge, not a medical prediction. Evidence is moderate; note this is one of a couple of correlated variants in/near NRXN3 reported for this trait - neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal rather than independent confirmations. Many other genetic and lifestyle factors also influence body weight. This variant is reported for more than one trait.

SOURCES

PubMed 36581621 | PubMed 19557197

Body weight & BMI tendency

SEC16B region

POPULATION STUDY

rs10913469 | GT T/T | DOSE 0/0 | REF/ALT T/C | STUDIED C (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the T allele - a T/T genotype, with no copies of the BMI-associated C allele. In population studies this sits at the lower end of this very small range; each C copy nudges measured body weight and BMI up only slightly.

EVIDENCE

In population studies, the C allele at rs10913469 (SEC16B region) is associated with marginally higher measured body weight and BMI, with a tiny per-copy effect. This is a weak population-level signal, not a medical prediction; body weight is strongly shaped by diet and activity, and many other genetic and lifestyle factors are involved. Note: this is one of 2 correlated variants in/near SEC16B reported for this trait - neighboring variants are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal, not 2 independent confirmations. This variant is reported for more than one trait; the per-trait effects come from different studies and are not directly comparable.

SOURCES

PubMed 19079260

Body weight & BMI tendency

TMEM18

POPULATION STUDY

rs2867125 | GT T/C | DOSE 0/1 | REF/ALT T/C | STUDIED C (1 of 2) | GRCh38 +

INTERPRETATION

You have the T/C genotype - one C allele and one T allele. In population studies the C allele is associated with a modestly higher body weight / BMI, with a small per-copy effect. This is a measurement nudge and a weak, relative population-level shift; on its own it does not meaningfully predict an individual's weight.

EVIDENCE

At this TMEM18 marker, each C allele is associated with a modestly higher body weight / BMI in population studies, with a small per-copy effect. This is a relative, population-level shift in a measurement - not your absolute chance and not a medical prediction. It is a weak association that, on its own, does not meaningfully predict your individual weight. Well-replicated. Note: this is one of 2 correlated variants in/near TMEM18 reported for this trait. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal - not 2 independent confirmations. Many other genetic and lifestyle factors also influence body weight. This variant is reported for more than one trait; the per-trait effects come from different studies and are not directly comparable.

SOURCES

PubMed 20935630 | PubMed 26426971

Body weight tendency

LINGO2/LRRN6C region

POPULATION STUDY

rs10968576 | GT A/A | DOSE 0/0 | REF/ALT A/G | STUDIED G (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the A allele - an A/A genotype, with no copies of the G variant. Relative to G-carriers this sits at the lower end for body weight in population studies, but the per-allele effect is small.

EVIDENCE

In population studies, each G allele is associated with a modestly higher body weight, with a small per-copy effect. Body weight is a measurement, not a medical condition; this is a weak, relative population-level shift and on its own does not meaningfully predict your individual weight. Many other genetic and lifestyle factors also influence body weight.

SOURCES

PubMed 23563607 | PubMed 20935630 | PubMed 25673413

Body-weight & BMI tendency

BDNF

POPULATION STUDY

rs6265 | GT C/C | DOSE 0/0 | REF/ALT C/T | STUDIED T (0 of 2) | GRCh38 +

INTERPRETATION

Both copies here carry the C allele - a C/C genotype, with no copies of the T allele. The T allele is weakly associated at the population level with a slightly lower body weight, so relative to T-carriers this genotype sits marginally higher. The effect is tiny; it is a small leaning, not a medical condition, and on its own it does not meaningfully predict an individual's weight.

EVIDENCE

Each T allele is associated at the population level with a slightly lower body weight. This is a relative, population-level shift, not a direct medical prediction. Strong evidence. Many other genetic and lifestyle factors also influence body weight. Note: this is one of 2 correlated variants in/near BDNF reported for this trait; treat them as largely a single shared signal (linkage disequilibrium) - not 2 independent confirmations. This variant is reported for more than one trait; the per-trait findings come from different studies and are not directly comparable.

SOURCES

PubMed 28892062 | PubMed 19079260

Body-weight & BMI tendency

BMI region

POPULATION STUDY

rs2112347 | GT T/G | DOSE 0/1 | REF/ALT T/G | STUDIED G (1 of 2) | GRCh38 +

INTERPRETATION

T/G genotype - one G allele and one T allele. In research cohorts the G allele is associated with a slightly lower body weight / BMI, with a small per-copy effect. This is a measurement nudge and a weak population-level association; on its own it does not meaningfully predict individual weight.

EVIDENCE

At this marker, each G allele is associated with a slightly lower body weight / BMI in population studies, with a small per-copy effect. This is a population-level nudge, not a medical prediction - a weak population-level shift that, on its own, does not meaningfully predict your individual weight, which depends far more on diet and activity. Well-replicated across research cohorts.

SOURCES

PubMed 25673413 | PubMed 23563607 | PubMed 36581621

Body-weight & BMI tendency

FTO

POPULATION STUDY

rs17817449 | GT T/G | DOSE 0/1 | REF/ALT T/G | STUDIED G (1 of 2) | GRCh38 +

INTERPRETATION

T/G genotype - one G allele and one T allele. In population studies the G allele is associated with a slightly higher body weight / BMI, with a small per-copy effect. This is a measurement nudge and a weak population-level association; on its own it does not meaningfully predict individual weight.

EVIDENCE

In population studies, each G allele is associated with a higher body weight / BMI measurement, with a small per-copy effect. Note: this is one of 11 correlated variants in/near FTO reported for this trait. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal - not 11 independent confirmations. FTO's effect is strongly modified by physical activity and diet, and many other factors also influence body weight. This variant is reported for more than one trait; the per-trait effects come from different studies and are not directly comparable.

SOURCES

PubMed 33045005 | PubMed 21552555

Body-weight & BMI tendency

MAP2K5

POPULATION STUDY

rs2241423 | GT A/A | DOSE 1/1 | REF/ALT G/A | STUDIED A (2 of 2) | GRCh38 +

INTERPRETATION

A/A genotype - two copies of the BMI-lowering A allele. In research cohorts the A allele is associated with a slightly lower body weight / BMI, with a small per-copy effect. Even with two copies this is a weak population-level association; on its own it does not meaningfully predict individual weight.

EVIDENCE

At this MAP2K5 marker, each A allele is associated with a slightly lower body weight / BMI in population studies, with a small per-copy effect. This is a population-level nudge, not a medical prediction - a weak population-level shift that, on its own, does not meaningfully predict your individual weight, which depends far more on diet and activity. Well-replicated across research cohorts.

SOURCES

PubMed 20935630

Higher BMI & body fat tendency

SEC16B

POPULATION STUDY

rs543874 | GT A/A | DOSE 0/0 | REF/ALT A/G | STUDIED G (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the A reference allele - an A/A genotype, with no copies of the G variant. In research the G allele is associated with a slightly higher BMI / body fat on average; you carry none, so this variant sits at the lower end of its small range for you.

EVIDENCE

In population studies, each G allele is associated with a slightly higher BMI / body fat (SEC16B), a small per-copy effect. BMI and body fat are measurements, not a medical condition; the per-copy effect is small - a relative, population-level shift that, on its own, does not meaningfully predict your individual body composition. Many other genetic and lifestyle factors also influence body composition. Strong evidence. This variant is reported for more than one trait; the per-trait effects come from different studies and scales and are not directly comparable.

SOURCES

PubMed 40216759 | PubMed 38538606 | PubMed 36581621

Higher BMI & body weight tendency

NRXN3

POPULATION STUDY

rs10150332 | GT T/T | DOSE 0/0 | REF/ALT T/C | STUDIED C (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the T allele - a T/T genotype, with no copies of the BMI-associated C allele. In population studies this sits at the lower end of this very small range; each C copy nudges measured BMI up only slightly.

EVIDENCE

In population studies, the C allele at rs10150332 (NRXN3) is associated with marginally higher measured BMI and body weight. This is a small leaning with a tiny effect - a weak population-level signal, not a medical prediction. Body weight is strongly shaped by diet and activity, and many other genetic and lifestyle factors are involved. Strong evidence. Note: this is one of 2 correlated variants in/near NRXN3 reported for this trait - neighboring variants are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal, not 2 independent confirmations. This variant is reported for more than one trait, and the trait-by-trait tendencies come from different studies and are not directly comparable.

SOURCES

PubMed 36581621 | PubMed 20935630

Higher BMI & obesity-associated variant GNPDA2

POPULATION STUDY

rs10938397 | GT A/A | DOSE 0/0 | REF/ALT A/G | STUDIED G (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the A allele - an A/A genotype, with no copies of the G allele. In population studies this sits at the lower end for BMI relative to G-carriers, but the per-allele effect is small.

EVIDENCE

In population studies, the G allele at rs10938397 (GNPDA2) is associated with a modestly higher BMI, with a small per-copy effect. BMI is a measurement, not a medical condition; this is a weak population-level association and on its own it does not meaningfully predict your individual weight, which is strongly shaped by diet and activity. Many other genetic and lifestyle factors are also involved. This variant is reported for more than one trait; the per-trait effects come from different studies and are not directly comparable.

SOURCES

PubMed 36581621 | PubMed 28892062 | PubMed 26426971

Higher BMI tendency FTO

POPULATION STUDY

rs3751812 | GT G/T | DOSE 0/1 | REF/ALT G/T | STUDIED T (1 of 2) | GRCh38 +

INTERPRETATION

You inherited one T allele and one G reference - a heterozygous G/T genotype. In population studies the T allele is associated with a modestly higher BMI, a small per-copy effect. This is a weak, relative population-level shift and, on its own, does not meaningfully predict an individual's weight.

EVIDENCE

At this FTO marker, each T allele is associated with a modestly higher BMI in population studies, a small per-copy effect. BMI is a measurement, not a medical condition; this is a relative, population-level shift that, on its own, does not meaningfully predict an individual's weight. Moderate evidence. Note: this is one of several correlated variants in/near FTO reported for this trait. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal - not many independent confirmations. Many other genetic and lifestyle factors also influence body weight.

SOURCES

PubMed 28448500

01.03 Tendon and ligament strain tendency (COL5A1) 1x

Tendon and ligament strain leaning

POPULATION STUDY

rs12722 | GT T/T | DOSE 1/1 | REF/ALT C/T | STUDIED T (2 of 2) | GRCh38 +

INTERPRETATION

Two T copies carry the small population leaning toward more tendon and ligament strain. Building training load gradually and allowing recovery days can support connective tissue.

EVIDENCE

In population studies, people who carry the T allele tend to have a slightly greater leaning toward soft-tissue strain in tendons and ligaments, while two-C carriers tend to show more connective-tissue resilience. It's a small, common genetic leaning, not a prediction about any one person. The leaning shows up mainly when someone carries two copies. Many genes and everyday habits - gradual training build-up, warm-ups, recovery days, and good hydration - matter more.

SOURCES

PubMed 35241120 | Source

01.04 ACE enzyme activity tendency (ACE) 1x

ACE enzyme activity tendency

ACE

POPULATION STUDY

rs4343 | GT G/A | DOSE 0/1 | REF/ALT G/A | STUDIED G (1 of 2) | GRCh38 +

INTERPRETATION

The G/A genotype is associated in population data with intermediate serum ACE activity - one G and one A copy. This is a neutral biomarker level, not good or bad in itself.

EVIDENCE

rs4343 in the ACE gene tracks the classic ACE insertion/deletion variant, which is the main genetic influence on how much ACE enzyme circulates in the blood. In population studies the G allele is linked to higher serum ACE activity and the A allele to lower activity (a large effect for a single variant). This is a biomarker level - higher or lower ACE activity is not in itself good or bad - and it reflects the same biology that makes the A allele relevant to several ACE-related drug responses elsewhere in this report. This variant is reported for more than one trait; the per-trait effects come from different studies and scales and are not directly comparable.

SOURCES

PubMed 20066004

01.05 Adult height tendency 2x

Adult height tendency GDF5

POPULATION STUDY

rs143384 | GT G/A (file: AG) | DOSE 0/1 | REF/ALT G/A | STUDIED A (1 of 2) | GRCh38 +

INTERPRETATION

G/A genotype - one G variant and one A variant, placing this in the middle of the adult-height range between the shorter A/A and taller G/G. A tiny contribution to overall height.

EVIDENCE

This GDF5 variant is one of the strongest common-variant influences on adult height. In population studies the A allele is associated with shorter stature - a small, relative, population-level shift; A/A toward the lower end, G/G toward the taller end. Height is shaped by thousands of variants plus nutrition, so this single marker nudges the average only a little. Normal everyday tendency, no good or bad meaning. This variant is reported for more than one trait; the per-trait effects come from different studies and scales and are not directly comparable.

SOURCES

PubMed 36224396

Adult height tendency HMGA2

POPULATION STUDY

rs1042725 | GT C/T | DOSE 0/1 | REF/ALT C/T | STUDIED T (1 of 2) | GRCh38 +

INTERPRETATION

The C/T genotype (one C, one T) is associated with an intermediate effect, with adult height nudged toward the middle relative to C/C and T/T. This single variant contributes only a tiny amount to overall height.

EVIDENCE

This common variant sits near HMGA2, one of the best-known genes affecting human height. In population studies the C (reference) allele is associated with slightly taller adult stature and the T allele with slightly shorter stature. The effect is small - only very slightly per copy - and height is shaped by hundreds of genes plus nutrition, so this single variant nudges the population average only a little. The same allele also tracks with very small differences in birth weight and birth length. This is a normal, everyday tendency with no good/bad meaning. This variant is reported for more than one trait; the per-trait effects come from different studies and scales and are not directly comparable.

SOURCES

PubMed 18391950

01.06 Anti-Mullerian hormone & ovarian reserve tendency (MCM8) 1x

Anti-Mullerian hormone & ovarian reserve tendency

MCM8

POPULATION STUDY

rs16991615 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the G variant - a G/G genotype, with no copies of the AMH-raising A variant. At the population level this sits at the lower-AMH / lower-ovarian-reserve end at this site relative to A-carriers.

EVIDENCE

rs16991615 (MCM8) is a strong marker of ovarian reserve. In population studies each minor A allele is associated with higher anti-Mullerian hormone (AMH) levels and with later menopause and a longer reproductive window - a favorable direction for ovarian reserve - while the reference G allele is the lower-AMH side. This is a biomarker association, not a diagnosis, and many other genetic and lifestyle factors also influence ovarian reserve. (Applies to people who menstruate.) This variant is reported for more than one trait, and the findings come from different studies and are not directly comparable.

SOURCES

PubMed 30649302 | PubMed 40229599

01.07 Dry earwax and reduced body-odor tendency (ABCC11) 1x

Dry earwax and lower body-odor leaning

POPULATION STUDY

rs17822931 | GT C/C | DOSE 0/0 | REF/ALT C/T | STUDIED T (0 of 2) | GRCh38 +

INTERPRETATION

Two C copies lean toward wet earwax and a more typical body-odor tendency. Breathable fabrics are a simple everyday comfort lever.

EVIDENCE

In population studies, people who carry the T allele tend to lean toward dry earwax and a reduced body-odor tendency, while C-carriers lean toward wet earwax and more odor. The leaning toward dry earwax and lower odor mainly shows up when someone carries two T copies. It's a small, common genetic leaning, not a prediction about any one person. Many genes and everyday habits - washing routines, clothing fabric choices, and diet - matter more.

SOURCES

PubMed 16444273

01.08 Endurance and VO2max training-response tendency (GABPB1) 1x

Endurance status and VO2max training response

POPULATION STUDY

rs7181866 | GT A/A | DOSE 0/0 | REF/ALT A/G | STUDIED G (0 of 2) | GRCh38 +

INTERPRETATION

No copies of the G allele here, so the leaning toward the endurance and VO2max-response tendency is on the lower end, and this is exploratory and low-confidence anyway. Steady, consistent endurance training still builds capacity over time.

EVIDENCE

In population studies, people who carry the G allele tend to show a stronger leaning toward endurance status and a favorable VO2max training response. It's a small, common genetic leaning, not a prediction about any one person, and everyday habits like consistent endurance training, sleep, fuelling and recovery matter far more. This is an early, low-confidence finding, so treat it as a weak signal rather than a settled result.

SOURCES

PubMed 23486860 | Source

01.09 Estrogen level tendency (CYP19A1) 1x

Estrogen level tendency CYP19A1

POPULATION STUDY

rs727479 | GT C/A | DOSE 0/1 | REF/ALT C/A | STUDIED A (1 of 2) | GRCh38 +

INTERPRETATION

You carry one A variant and one C variant - a heterozygous C/A genotype, one from each parent. In population studies this places you mid-range, between the lower-end C/C and the higher-end A/A. A relative, population-level shift - not your absolute level.

EVIDENCE

rs727479 sits in CYP19A1, the gene for aromatase (the enzyme that makes estrogen). In population studies, each A allele is associated with higher measured estrogen levels, a population-level tendency, not a medical prediction. This signal is reported in women. It is a genome-wide-significant association for both estradiol and estrone in a large hormone GWAS. Many other genetic and lifestyle factors also influence hormone levels. This variant is reported for more than one trait; the effects for different traits come from different studies and scales and are not directly comparable.

SOURCES

PubMed 29325096

01.10 Fat-oxidation and endurance energy-metabolism tendency (PPARD) 1x

Fat-burning and endurance energy-metabolism leaning

POPULATION STUDY

rs2016520 | GT T/T | DOSE 0/0 | REF/ALT T/C | STUDIED C (0 of 2) | GRCh38 +

INTERPRETATION

Two T copies carry less of this exploratory endurance-metabolism leaning. Regular aerobic activity supports fat-burning capacity regardless of genetics.

EVIDENCE

In population studies, people who carry the C allele tend to lean toward greater fat-oxidation and an endurance-type energy metabolism, an association seen more often among elite endurance athletes. This is exploratory, low-confidence wording - the link is modest and some studies have not found it. It's a small, common genetic leaning, not a prediction about any one person. Many genes and everyday habits - endurance-style training, overall activity level, sleep, and diet - matter more.

SOURCES

PubMed 31881714 | Source

01.11 Muscle fiber type: power vs endurance tendency (ACTN3) 1x

Muscle fiber type: power vs endurance leaning

POPULATION STUDY

rs1815739 | GT C/T (file: TC) | DOSE 0/1 | REF/ALT C/T | STUDIED T (1 of 2) | GRCh38 +

INTERPRETATION

One C and one T - a heterozygous C/T genotype, one allele from each parent. In population studies this sits in the middle of the sprint-power range, between T/T and C/C (a relative, population-level shift, not a personal prediction).

EVIDENCE

rs1815739 in ACTN3 is a common ACTN3 variant linked to muscle fiber type. In population studies each T allele is associated with a lower sprint-power muscle tendency, a small per-copy effect - a relative, population-level shift, not a verdict on your training. Many other genetic and lifestyle factors, training above all, influence power and sprint performance.

SOURCES

PubMed 38609671

01.12 Photic (sun) sneeze reflex tendency (ZEB2) 1x

Sun-sneeze (photic sneeze / ACHOO) reflex

POPULATION STUDY

rs10427255 | GT C/C | DOSE 0/0 | REF/ALT C/T | STUDIED per genotype | GRCh38 +

INTERPRETATION

Two C copies. A large European study linked the C allele to a bit more of the sun-sneeze reflex, while a Chinese study pointed to the other allele, so the direction here is genuinely uncertain. Harmless either way; sunglasses soften the change in light if bright sun ever makes you sneeze.

EVIDENCE

Some people sneeze when they step from shade into bright sunlight, a harmless reflex sometimes called the photic sneeze or ACHOO reflex. A common variant in this region is linked to how likely it is, but studies disagree on which allele leans which way: a large European study pointed to the C allele, a smaller Chinese study to the T allele. Either way it is a small, everyday sensory quirk, not anything that affects health.

SOURCES

PubMed 20585627 | PubMed 30899065

01.13 Systolic blood pressure tendency — 2x

Systolic blood pressure tendency

POPULATION STUDY

rs12478601 | GT C/T | DOSE 0/1 | REF/ALT C/T | STUDIED T (1 of 2) | GRCh38 +

INTERPRETATION

You have one C and one T allele - a heterozygous C/T genotype, one from each parent. At the population level the T allele is associated with a modestly lower systolic blood pressure reading. This is an association, not a medical prediction.

EVIDENCE

In population studies, people who carry the T allele at this marker tend to have a slightly lower systolic blood pressure reading. It's a small, common genetic leaning, not a prediction about any one person. Blood pressure is a cuff measurement, and many other genes and everyday habits - salt intake, exercise, body weight, alcohol and stress - influence it far more.

SOURCES

PubMed 33230300

Systolic blood pressure tendency

CYP11B2

POPULATION STUDY

rs1799998 | GT G/G | DOSE 1/1 | REF/ALT A/G | STUDIED G (2 of 2) | GRCh38 +

INTERPRETATION

Both copies carry the G allele - a G/G genotype. At this CYP11B2 marker, each G copy is associated with a slightly lower systolic blood pressure in research cohorts, a small per-copy effect. This is a very small, relative population-level nudge, not a medical prediction.

EVIDENCE

In population studies, people who carry the G allele at CYP11B2 tend to have slightly lower systolic blood pressure. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - dietary salt, physical activity, sleep, and body weight - influence blood pressure far more.

SOURCES

PubMed 39024449

01.14 Testosterone & SHBG level tendency

3/5 markers

Testosterone & SHBG-level tendency

SHBG

POPULATION STUDY

rs6258 | GT C/C | DOSE 0/0 | REF/ALT C/T | STUDIED T (0 of 2) | GRCh38 +

INTERPRETATION

Two copies of the common C allele, the typical genotype here, with no copy of the SHBG-lowering T variant. SHBG and testosterone track the usual population range. A quantitative biomarker level, not a medical prediction.

EVIDENCE

rs6258 is a coding change (Pro185Leu) in the SHBG protein. In population studies the less-common T allele is associated with lower sex hormone-binding globulin (SHBG) in both men and women. Its effect on total testosterone is sex-specific and runs in opposite directions: in men the T allele tracks with lower total testosterone, while in women it tracks with higher total testosterone. So the testosterone direction depends on sex. A small, population-level association, not a medical prediction.

SOURCES

PubMed 32042192 | PubMed 34226706

Testosterone level tendency

SHBG

POPULATION STUDY

rs727428 | GT T/C | DOSE 0/1 | REF/ALT T/C | STUDIED C (1 of 2) | GRCh38 +

INTERPRETATION

One C and one T, a middling level between the two ends. The linked rise in total testosterone is a male-pattern finding; in women it differs. A small, relative shift.

EVIDENCE

In population studies, each C allele at rs727428 in the SHBG region is associated with higher sex hormone-binding globulin (SHBG) and, mainly in men, higher total testosterone. The SHBG effect is seen broadly, but the testosterone direction was characterized mainly in men and differs in women, so treat it as a male-pattern tendency. rs727428 is one of several correlated SHBG variants usually inherited together, so treat them as one shared signal. A small, population-level association, not a medical prediction.

SOURCES

PubMed 32042192

Testosterone-level tendency

FAM9B

POPULATION STUDY

rs5934505 | GT T/T | DOSE 0/0 | REF/ALT T/C | STUDIED C (0 of 2) | GRCh38 +

INTERPRETATION

Carrying the T variant at this X-linked FAM9B marker, with no C. The T side tracks with a small leaning toward lower testosterone, so this sits toward the lower-testosterone end of the range. A relative, population-level shift, not your absolute level.

EVIDENCE

In population studies, people who carry the C variant at this X-chromosome marker near FAM9B tend to have slightly higher circulating testosterone, while the T variant leans the other way. It's a small, common genetic leaning, not a prediction about any one person. Because the marker is on the X chromosome, men carry a single copy and women carry two. Age, body weight, sleep, exercise and stress influence testosterone far more.

SOURCES

PubMed 34337532

01.15 Thyroid-stimulating hormone (TSH) tendency (PDE10A) 1x

Thyroid-stimulating hormone (TSH) tendency

PDE10A

POPULATION STUDY

rs753760 | GT C/C | DOSE 0/0 | REF/ALT C/G | STUDIED C (2 of 2) | GRCh38 +

INTERPRETATION

Both copies here carry the C variant - a C/C genotype. In population studies, people with C/C tend to sit toward the higher end of the measured TSH range, each C copy adding a small upward nudge. A relative, population-level shift - not your absolute level.

EVIDENCE

In population studies, each C allele at rs753760 is associated with a higher measured level of thyroid-stimulating hormone (TSH). TSH is the pituitary's signal to the thyroid rather than a thyroid hormone itself, and it tends to rise when thyroid output runs low. This is a population-level tendency, not a medical prediction; many other genetic and lifestyle factors also influence it.

SOURCES

PubMed 23408906

01.16 Waist-to-hip ratio tendency

2x

Waist-to-hip ratio tendency

body-fat distribution

POPULATION STUDY

rs727428 | GT T/C | DOSE 0/1 | REF/ALT T/C | STUDIED C (1 of 2) | GRCh38 +

INTERPRETATION

You carry one C and one T allele - a heterozygous T/C genotype, one from each parent. The C allele is weakly linked to a slightly lower waist-to-hip ratio at the population level. This is a very small, relative population-level shift, and on its own it does not meaningfully predict your individual body-fat distribution.

EVIDENCE

In population studies, each C allele at rs727428 is weakly associated with a slightly lower waist-to-hip ratio - a population-level tendency, not a medical prediction. The effect is tiny and on its own does not meaningfully predict your individual body-fat distribution. Many other genetic and lifestyle factors also influence where the body stores fat. This variant is reported for more than one trait; the effects for different traits come from different studies and scales and are not directly comparable.

SOURCES

PubMed 30575882

Waist-to-hip ratio tendency FTO

POPULATION STUDY

rs1121980 GT G/A DOSE 0/1 REF/ALT G/A STUDIED A (1 of 2) GRCh38 +

INTERPRETATION

You inherited one A variant and one G allele - a heterozygous G/A genotype. In research cohorts the A allele is associated with a modestly higher waist-to-hip ratio, with a small per-copy effect - a weak population-level shift that on its own does not meaningfully predict your individual body shape.

EVIDENCE

In population studies, each A allele is associated with a modestly higher waist-to-hip ratio, with a small per-copy effect. Waist-to-hip ratio is a measurement, not a medical condition; this is a weak, relative population-level shift and not a prediction of your individual body shape. Many other genetic and lifestyle factors also influence body shape. This variant is reported for more than one trait; the per-trait effects come from different studies and are not directly comparable.

SOURCES

PubMed 25673412

01.17 Aerobic-capacity (VO2max) tendency (NFIA-AS2) 1x

Maximal oxygen uptake (VO2max) tendency

POPULATION STUDY

rs1572312 GT G/G DOSE 0/0 REF/ALT G/T STUDIED G (2 of 2) GRCh38 +

INTERPRETATION

Two copies of the G allele line up with a slightly stronger leaning toward higher aerobic capacity, though this is exploratory and low-confidence. Regular aerobic base-building plays to this tendency.

EVIDENCE

In population studies, people who carry the G allele tend to show a slightly stronger leaning toward higher maximal oxygen uptake (aerobic capacity). It's a small, common genetic leaning, not a prediction about any one person, and everyday habits like aerobic base-building, interval training and recovery matter far more. This is an early, low-confidence finding, so treat it as a weak signal rather than a settled result.

SOURCES

Source PubMed 35622109

01.18 Breathing & lung-capacity tendency (CHRNA3) 1x

Breathing & lung-capacity tendency

CHRNA3

POPULATION STUDY

rs1051730 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the G allele - a G/G genotype. In research cohorts each G copy is associated with slightly higher lung function compared with the A/A version, acting mainly through lighter smoking. A population-level association, not a diagnosis.

EVIDENCE

In population studies, people who carry the A allele near CHRNA3 tend to have slightly lower breathing capacity. This leaning works mainly through smoking - the allele tracks heavier smoking, which lowers lung capacity - so in people who have never smoked the effect is essentially absent. It's a small, common genetic leaning, not a prediction about any one person, and habits like not smoking, regular aerobic activity, and clean air influence breathing far more. When this applies: The CHRNA3/5 effect on lung function (FEV1) is mediated through smoking: the A allele drives heavier smoking, which in turn lowers FEV1. In people who have never smoked this protective/at-risk signal is essentially absent.

SOURCES

PubMed 26634245

01.19 Grip strength & lean mass tendency (GDF5) 1x

Grip strength & lean mass tendency

GDF5

POPULATION STUDY

rs143384 | GT G/A (file: AG) | DOSE 0/1 | REF/ALT G/A | STUDIED G (1 of 2) | GRCh38 +

INTERPRETATION

G/A genotype - one G variant and one A variant. The G allele is associated with slightly higher grip strength and lean mass (the favorable direction) relative to A/A; a small but positive contribution.

EVIDENCE

At this GDF5 variant the G allele is associated in population studies with greater hand-grip strength and lean muscle mass, while the A allele tends toward slightly lower values. Greater strength and lean mass are the favorable direction here, so the G allele points the helpful way. The per-copy effect is small, and regular strength work matters far more. A common genetic leaning, not a prediction about any one person.

SOURCES

PubMed 33097823 PubMed 33510174

01.20 Lean body mass tendency (TRHR) 1x

Lean body mass

POPULATION STUDY

rs7832552 | GT T/T | DOSE 1/1 | REF/ALT C/T | STUDIED T (2 of 2) | GRCh38 +

INTERPRETATION

Two T copies, the form most associated in population studies with a higher leaning toward lean body mass. It's still a small, common tendency rather than a guarantee. Consistent strength work is a reliable habit that helps turn that leaning into results.

EVIDENCE

In population studies, people who carry the T allele tend to have somewhat higher lean body mass. It's a small, common genetic leaning, not a prediction about any one person. Many genes and everyday habits - resistance training, total protein intake, sleep quality, and overall daily movement - matter more.

SOURCES

PubMed 19268274

01.21 Muscle power and strength tendency (ACVR1B) 1x

Muscle power and strength leaning

POPULATION STUDY

rs2854464 | GT A/A | DOSE 0/0 | REF/ALT A/G | STUDIED G (0 of 2) | GRCh38 +

INTERPRETATION

Two A copies lean toward greater muscle power and strength in population studies. Regular resistance training builds on that leaning.

EVIDENCE

In population studies, people who carry the G allele tend to lean toward slightly lower muscle power and strength, while A-carriers lean toward more, an association noted among sprint and power athletes. It's a small, common genetic leaning, not a prediction about any one person. Many genes and everyday habits - resistance training, adequate protein, recovery, and overall activity - matter more.

SOURCES

PubMed 27253421

02

Nutrition

30 traits
110 findings

Food, vitamins, minerals, alcohol metabolism and blood sugar response.

02.01 Alcohol response & consumption tendency

7/8 markers

Alcohol-response variant

ADH1B

CATALOGED VARIANT

rs1229984 | GT C/C | DOSE 1/1 | REF/ALT T/C | STUDIED C (2 of 2) | GRCh38 +

INTERPRETATION

C/C genotype - two copies of the slower-metabolizing ADH1B (Arg48) form. Without the fast His48 (T) allele, acetaldehyde builds up more slowly, so drinking is less self-limited by an aversive response; in population studies this is linked to the higher-consumption end. This is a small, population-level shift only. Many other genetic and lifestyle factors also influence alcohol use.

EVIDENCE

In population studies, people who carry the C allele in ADH1B tend to break down alcohol more slowly and miss the faster acetaldehyde build-up the T allele drives (the strong flush itself is an ALDH2 effect, rs671), and on average tend toward higher alcohol consumption. It's a common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - drinking patterns, social environment, mood and personal choice - influence drinking behavior far more. When this applies: Relevant only if you drink alcohol.

CLINVAR REVIEW

0 of 4 review stars - no assertion criteria provided. Classified: protective. This says how much expert review stands behind the classification, not how strong the effect is.

SOURCES

ClinVar | PubMed 35094024 | PubMed 32451486 | PubMed 37282553

Alcohol consumption tendency

FUT2

SUBSTANCE RESPONSE

rs492602 | GT A/G | DOSE 0/1 | REF/ALT A/G | STUDIED G (1 of 2) | GRCh38 +

INTERPRETATION

You inherited one G variant and one A baseline variant - a heterozygous A/G genotype, one allele from each parent. In population studies the G allele is linked to a modestly higher alcohol-consumption pattern relative to the A/A baseline. This is research context, not a personal prediction; many other factors also influence drinking behavior.

EVIDENCE

In population studies, people who carry the G allele at this FUT2 marker tend to fall on the higher side for alcohol consumption on average. It's a small, common genetic leaning, not a prediction about any one person, and it only shows up at all when alcohol is consumed. Many other genes and everyday habits - social setting, stress, routines and personal choices around drinking - influence alcohol patterns far more. When this applies: Relevant only if you drink alcohol - it reflects a small population leaning in how much people tend to drink, and has no bearing on you if you don't. Early-stage finding - read it as a small, common tendency, not a firm result.

SOURCES

PubMed 30336701 | PubMed 32451486

Alcohol-response variant

ADH1B

POPULATION STUDY

rs2066702 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

Both copies here carry the G allele, a G/G genotype with no copies of the protective A allele. The A allele is linked to a small, well-replicated reduction in heavier-drinking patterns, so relative to A-carriers this genotype carries none of that protection. This is most relevant for people who drink, and many other factors matter more.

EVIDENCE

In population studies, each A allele is associated with a small reduction in the odds of heavier-drinking patterns, with a small per-copy effect - a relative, population-level shift, not your absolute chance. The evidence is strong. Note: this is one of a few correlated variants in/near ADH1B reported for this trait. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal - not several independent confirmations. Many other genetic and lifestyle factors also strongly influence drinking behavior.

SOURCES

PubMed 24166409 | PubMed 30940813

Alcohol consumption preference

POPULATION STUDY

rs11940694 | GT G/G | DOSE 1/1 | REF/ALT A/G | STUDIED G (2 of 2) | GRCh38 +

INTERPRETATION

Two copies of the G allele, the strongest leaning here toward higher alcohol consumption preference. It's still a small tendency; planning alcohol-free days and alternating with water supports staying in a comfortable everyday range.

EVIDENCE

In population studies, people who carry the G allele tend to consume more alcohol on average within the normal range of everyday preference. It's a small, common genetic leaning, not a prediction about any one person. Many genes and everyday habits - social setting, daily routine, hydration, and personal limits - matter more.

SOURCES

PubMed 36477530 | Source

Alcohol-response tendency

CHRNA3

CATALOGED VARIANT

rs578776 | GT G/A | DOSE 0/1 | REF/ALT G/A | STUDIED G (1 of 2) | GRCh38 +

INTERPRETATION

You have the G/A genotype (one G, one A). In population research the tendency is intermediate - one protective A copy is linked to a slightly lower-tendency direction compared with G/G. This is relevant only to people who drink.

EVIDENCE

This CHRNA3 nicotinic-receptor variant also shows a weak population-level association with alcohol-consumption patterns, in the same direction as its nicotine effect. The G allele (the reference, major form here) is associated with the higher-tendency direction and the A allele with the protective direction: in a multi-substance study, rs578776 showed a protective association for the protective allele - a relative, population-level shift, not an individual outcome. The effect on alcohol is smaller and less consistent than for nicotine, so treat it as a minor tendency, and it is relevant only to people who drink. Many other genetic and lifestyle factors strongly influence drinking behavior. Note: this is one of 2 correlated variants in/near CHRNA3 reported for this trait; treat them as largely a single shared signal (linkage disequilibrium) - not 2 independent confirmations. This variant is reported for more than one trait; the per-trait findings come from different studies and are not directly comparable. When this applies: Applies only if you drink alcohol; in people who do not drink it has no effect.

SOURCES

PubMed 20485328

Alcohol-response variant ADH1B

POPULATION STUDY

rs1789891 | GT C/C | DOSE 0/0 | REF/ALT C/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the C baseline variant - a C/C genotype, with no copies of the A variant. In population studies, people with C/C sit at the lower-signal end of this alcohol-use association relative to A-carriers; each A copy would shift the population odds higher. This is a relative, population-level shift, not your absolute chance, and many other factors also influence drinking behavior.

EVIDENCE

In population studies, each A allele at this ADH1B variant is associated with higher odds of alcohol dependence (a problematic, addictive drinking pattern), a relative population-level shift, not your absolute chance. The evidence is moderate. This is only meaningful for people who drink. Note: this is one of a few correlated variants in or near ADH1B reported for this trait; neighboring variants are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal. Many other genetic and lifestyle factors also influence drinking behavior. When this applies: This heavier-drinking leaning only applies to people who drink; it has no effect for people who don't.

SOURCES

PubMed 28714907 | PubMed 22004471

Alcohol-response variant CHRNA3

SUBSTANCE RESPONSE

rs1051730 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED G (2 of 2) | GRCh38 +

INTERPRETATION

You have the G/G genotype at this CHRNA3 marker. A single low-evidence annotation links this version to a somewhat higher tendency toward heavier alcohol consumption if you drink, compared with A/A - but this is weak and direction-uncertain. If you do not drink alcohol, it does not apply to you.

EVIDENCE

Relevant only for people who drink alcohol. This marker sits in the CHRNA3 gene cluster, whose best-replicated effect is on smoking behavior - in population studies the A allele tends to track with heavier smoking. A much weaker single annotation links the locus to alcohol-response, pointing to the G allele as the version associated with somewhat higher overall alcohol consumption (G/G higher, A/A lower). This alcohol signal is low-evidence and direction-uncertain - a tentative research note, not a personal finding. It is one of two neighboring variants usually inherited together, so treat them as a single shared signal. Many genes and everyday habits shape drinking behavior far more. When this applies: Applies only if you drink alcohol, and even then the evidence is weak. This marker's best-replicated link is to smoking behavior, not alcohol. If you do not drink, this trait has no effect on you. Early-stage finding - read it as a small, common tendency, not a firm result.

SOURCES

PubMed 19132693

02.02 LDL & total cholesterol tendency

28/36 markers

LDL & total cholesterol levels tendency

POPULATION STUDY

rs660240 | GT C/C | DOSE 1/1 | REF/ALT T/C | STUDIED C (2 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the C allele - a C/C genotype. In research cohorts, C/C is associated with modestly higher LDL & total cholesterol than T/T. This is a small leaning, not an individual prediction.

EVIDENCE

In population studies, people who carry the C allele at this variant tend to have slightly higher LDL and total cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, soluble fiber, oily fish, and regular activity - influence cholesterol far more.

SOURCES

PubMed 38116116 | PubMed 40841581

LDL & total cholesterol tendency APOB

POPULATION STUDY

rs1367117 | GT A/A | DOSE 1/1 | REF/ALT G/A | STUDIED A (2 of 2) | GRCh38 +

INTERPRETATION

Both copies here carry the A allele - an A/A genotype. In population data the A allele is associated with modestly higher average LDL & total cholesterol than the G allele. This is a small population-level leaning, not a medical prediction - it nudges an average lipid reading, and a blood test reflects your real LDL.

EVIDENCE

In population studies, people who carry the A allele near APOB tend to have slightly higher LDL and total cholesterol, while the G allele leans slightly lower. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, fiber and saturated-fat intake, physical activity, and body weight - influence cholesterol far more. Note: APOB carries more than one cholesterol signal that are only partly correlated, so this variant and the other APOB marker in this report are largely separate signals and can point in different directions.

SOURCES

PubMed 24097068

LDL & total cholesterol tendency

CELSR2

POPULATION STUDY

rs646776 | GT T/T | DOSE 1/1 | REF/ALT C/T | STUDIED T (2 of 2) | GRCh38 +

INTERPRETATION

Both copies carry the T allele - a T/T genotype. In research cohorts this is associated on average with a modestly higher LDL/total cholesterol than C/C. The per-copy effect is small - a weak population-level signal, not a prediction of any individual cholesterol.

EVIDENCE

In population studies, people who carry the T allele near CELSR2 tend to have slightly higher LDL and total cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, swapping saturated for unsaturated fats, fiber intake, and regular movement - influence cholesterol far more.

SOURCES

PubMed 30911093 | PubMed 38116116

LDL cholesterol level tendency

SORT1

POPULATION STUDY

rs12740374 | GT G/G | DOSE 0/0 | REF/ALT G/T | STUDIED T (0 of 2) | GRCh38 +

INTERPRETATION

Both copies here carry the G allele - a G/G genotype. In population data the G allele is associated with modestly higher average LDL cholesterol than the T allele. This is a small population-level leaning, not a medical prediction - it nudges an average lipid reading, and a blood test reflects your real LDL.

EVIDENCE

In population studies, people who carry the T allele near SORT1 tend to have slightly lower LDL cholesterol, while the G allele leans slightly higher. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, fiber and saturated-fat intake, physical activity, and body weight - influence cholesterol far more. This is one of two correlated variants at the 1p13 SORT1/CELSR2 locus reported for LDL cholesterol. They are coded on opposite alleles, so although each names a different allele they tag the same shared signal. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal, not independent confirmations.

SOURCES

PubMed 20686566 | PubMed 21347282

LDL cholesterol tendency

APOE

POPULATION STUDY

rs7412 | GT C/C | DOSE 0/0 | REF/ALT C/T | STUDIED T (0 of 2) | GRCh38 +

INTERPRETATION

You have the C/C genotype at rs7412 - no APOE e2 (T) copy here. In population studies C/C is associated with higher LDL cholesterol than T-carriers (each T copy is associated with modestly lower LDL). This is the less favorable direction at this site, though your overall picture depends on rs429358 and lifestyle.

EVIDENCE

In population studies, people who carry the T allele at this APOE marker (the e2-defining variant) tend to have lower LDL cholesterol on average. It's a common genetic leaning, not a prediction about any one person, and the overall lipid picture also depends on the nearby APOE variant and on daily habits. Many other genes and everyday habits - fiber intake, the balance of fats in the diet, physical activity, and body weight - influence cholesterol far more.

SOURCES

PubMed 39909172 | PubMed 38116116 | PubMed 33339817

LDL cholesterol tendency

PSRC1/CELSR2 region

POPULATION STUDY

rs4970834 | GT C/C | DOSE 0/0 | REF/ALT C/T | STUDIED T (0 of 2) | GRCh38 +

INTERPRETATION

C/C genotype: in research cohorts this form is associated with modestly higher LDL cholesterol than the T/T type, with each C copy linked to a small upward shift. This is a small, population-level nudge - not a medical prediction. Many other factors also influence cholesterol.

EVIDENCE

In population studies, people who carry the T allele in the PSRC1/CELSR2 region tend to have slightly lower LDL cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, soluble fiber, oily fish, and regular activity - influence cholesterol far more.

SOURCES

PubMed 32203549

LDL cholesterol tendency

SORT1

POPULATION STUDY

rs629301 | GT T/T | DOSE 1/1 | REF/ALT G/T | STUDIED T (2 of 2) | GRCh38 +

INTERPRETATION

Both copies carry the T allele - a T/T genotype. In research cohorts this is associated on average with a modestly higher LDL cholesterol than G/G. The per-copy effect is small - a weak population-level signal, not a prediction of any individual LDL.

EVIDENCE

In population studies, people who carry the T allele near SORT1 tend to have slightly higher LDL cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - a fiber-rich diet, more unsaturated fats, regular activity, and keeping a steady weight - influence LDL far more. This is one of two correlated variants at the 1p13 SORT1/CELSR2 locus reported for LDL cholesterol. They are coded on opposite alleles, so although each names a different allele they tag the same shared signal. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal, not independent confirmations.

SOURCES

PubMed 31566214 | PubMed 26690388 | PubMed 30926973

Total & LDL cholesterol tendency

HNF4A

POPULATION STUDY

rs1800961 | GT C/C | DOSE 0/0 | REF/ALT C/T | STUDIED T (0 of 2) | GRCh38 +

INTERPRETATION

At this position you carry the C/C genotype, with no copies of the T allele. In population studies the T allele is the one weakly linked to a lower total/LDL cholesterol reading, so on average C/C sits at the upper end. The shift is a small per-copy leaning and, on its own, does not meaningfully predict your actual cholesterol level - a lipid panel is the real measure.

EVIDENCE

In population studies, people who carry the T allele at this HNF4A site tend to have slightly lower total and LDL cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, fiber and saturated-fat intake, physical activity, and body weight - influence cholesterol far more.

SOURCES

PubMed 32203549 | PubMed 24097068

LDL ('bad') cholesterol tendency

APOA1

POPULATION STUDY

rs964184 | GT C/C | DOSE 1/1 | REF/ALT G/C | STUDIED C (2 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the C allele - a C/C genotype. In population studies C/C is associated with a modestly lower LDL cholesterol measurement than G/G. This is an association, not a medical prediction.

EVIDENCE

In population studies, people who carry the C allele at this APOA1/APOA5 marker tend to have slightly lower LDL ('bad') cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, saturated-fat intake, fiber, exercise and body weight - influence cholesterol far more.

SOURCES

PubMed 25961943

LDL & total cholesterol tendency

ABO

POPULATION STUDY

rs579459 | GT T/T | DOSE 1/1 | REF/ALT C/T | STUDIED T (2 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the T allele - a T/T genotype. In research cohorts T/T is associated with modestly lower LDL & total cholesterol than C/C. This is a small leaning, not a medical prediction.

EVIDENCE

In population studies, people who carry the T allele in ABO tend to have slightly lower LDL and total cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, soluble fiber, oily fish, and regular activity - influence cholesterol far more.

SOURCES

PubMed 30926973 PubMed 26582766

LDL & total cholesterol tendency

APOA5

POPULATION STUDY

rs2266788 | GT A/A | DOSE 1/1 | REF/ALT G/A | STUDIED A (2 of 2) | GRCh38 +

INTERPRETATION

Both copies are the A allele - an A/A genotype. In population studies the A allele is weakly associated with a slightly lower LDL/total cholesterol reading, so A/A sits at the lower end of this small average shift. The per-copy effect is tiny and reflects a population-level average - a lipid panel confirms the actual number.

EVIDENCE

In population studies, people who carry the A allele near APOA5 tend to have slightly lower LDL and total cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - fiber-rich foods, the balance of fats in the diet, and staying active - influence cholesterol far more.

SOURCES

PubMed 29507422

LDL & total cholesterol tendency

APOE

POPULATION STUDY

rs769449 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

G/G genotype, no copies of the A variant. In population studies this is the genotype associated with the lowest measured LDL and total cholesterol at this position; each A copy is associated with a slightly higher measure. This is a tendency, not a prediction; many other factors also influence cholesterol.

EVIDENCE

In population studies, people who carry the A allele at this APOE-region marker tend to have higher LDL and total cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, saturated-fat intake, oily fish, exercise and body weight - influence cholesterol far more.

SOURCES

PubMed 31719535 PubMed 35945198

LDL & total cholesterol tendency

FADS1

POPULATION STUDY

rs174550 | GT C/C | DOSE 1/1 | REF/ALT T/C | STUDIED C (2 of 2) | GRCh38 +

INTERPRETATION

Both copies carry the C allele - a C/C genotype. At this FADS1 marker each C copy is associated with a modestly lower LDL / total-cholesterol measurement. This is a measurement nudge, not a medical prediction.

EVIDENCE

In population studies, people who carry the C allele at this FADS1 marker tend to have slightly lower LDL and total cholesterol. FADS1 helps convert dietary omega-3 and omega-6 fats. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - fiber intake, the balance of fats in the diet, physical activity, and body weight - influence cholesterol far more.

SOURCES

PubMed 39414775

LDL & total cholesterol tendency

FADS2

POPULATION STUDY

rs174583 | GT T/T | DOSE 1/1 | REF/ALT C/T | STUDIED T (2 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the T variant - a T/T genotype. The T allele is associated with lower LDL/total cholesterol, so people with T/T tend to sit at the lower-cholesterol end at this site. The effect is small.

EVIDENCE

In population studies, people who carry the T allele near FADS2 (the less common one here) tend to have slightly lower LDL and total cholesterol, while C-carriers tend to run a touch higher. FADS2 helps convert dietary omega-3 and omega-6 fats, so this is a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, dietary fiber, physical activity, and body weight - influence cholesterol far more. When this applies: FADS is pleiotropic; this entry covers the LDL/total-cholesterol arm, where the minor T allele lowers cholesterol.

SOURCES

PubMed 29507422

LDL & total cholesterol tendency

HFE

POPULATION STUDY

rs1800562 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

Both copies here carry the G allele - a G/G genotype, with no copy of the cholesterol-lowering A variant. In population studies this places people toward the higher end of the LDL and total-cholesterol range. Each A copy is associated with a small downward shift.

EVIDENCE

In population studies, people who carry the A allele at this HFE marker (C282Y) tend to have slightly lower LDL and total cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - fiber intake, the balance of fats in the diet, physical activity, and body weight - influence cholesterol far more.

SOURCES

PubMed 24097068 | PubMed 20686565 | PubMed 32154731

LDL & total cholesterol tendency

HNF1A

POPULATION STUDY

rs1169288 | GT A/A | DOSE 0/0 | REF/ALT A/C | STUDIED C (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the A allele - an A/A genotype. In population studies the A allele is associated with slightly lower average LDL and total cholesterol than the C allele. This is a small population-level leaning, not a medical prediction, and on its own it does not determine your actual cholesterol - a blood test does.

EVIDENCE

HNF1A is a gene that helps the liver regulate cholesterol-related proteins. In population studies, people who carry the C allele here tend to have very slightly higher LDL and total cholesterol. It's a tiny, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, fiber and oily fish, physical activity, and body weight - influence cholesterol far more.

SOURCES

PubMed 30275531 | PubMed 24097068

LDL & total cholesterol tendency

LDLR region

POPULATION STUDY

rs1122608 | GT G/T | DOSE 0/1 | REF/ALT G/T | STUDIED T (1 of 2) | GRCh38 +

INTERPRETATION

One G allele and one T allele - a heterozygous G/T genotype, one from each parent. In population studies each G copy is associated with a slightly higher average LDL and total cholesterol than the T/T pattern. This is a small measurement nudge, not a medical prediction; a lipid panel reflects your real values.

EVIDENCE

The LDLR region sits near the LDL-receptor gene that clears cholesterol from the blood. In population studies, people who carry the T allele here tend to have slightly lower LDL and total cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, fiber and oily fish, physical activity, and body weight - influence cholesterol far more.

SOURCES

PubMed 31566214

LDL & total cholesterol tendency

LDLR

POPULATION STUDY

rs688 | GT C/T | DOSE 0/1 | REF/ALT C/T | STUDIED T (1 of 2) | GRCh38 +

INTERPRETATION

C/T genotype, one allele from each parent. In population studies the T allele is associated with modestly higher measured LDL and total cholesterol. This is a small nudge, not a prediction; many other factors also influence cholesterol.

EVIDENCE

In population studies, people who carry the T allele in the LDLR gene tend to have slightly higher LDL and total cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, saturated-fat intake, fiber, exercise and body weight - influence cholesterol far more.

SOURCES

PubMed 30926973

LDL & total cholesterol tendency

LPA

POPULATION STUDY

rs10455872 | GT A/A | DOSE 0/0 | REF/ALT A/G | STUDIED G (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the A allele - an A/A genotype, with no copies of the G allele. In population studies the G allele is tied to a modestly higher LDL and total cholesterol reading, so A/A sits at the lower end of this range. Informational, not a concern.

EVIDENCE

In population studies, people who carry the G allele at this LPA-region variant tend to have slightly higher LDL and total cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, oily fish and fiber, physical activity, and body weight - influence cholesterol far more.

SOURCES

PubMed 30275531 | PubMed 32226016

LDL & total cholesterol tendency

PCSK9

POPULATION STUDY

rs2479409 | GT A/A | DOSE 1/1 | REF/ALT G/A | STUDIED A (2 of 2) | GRCh38 +

INTERPRETATION

Both copies are the A allele - an A/A genotype. The A allele is weakly associated in research with a slightly lower LDL/total cholesterol reading, so A/A sits at the lower end of this small average shift. The per-copy effect is tiny and, on its own, does not meaningfully predict your individual cholesterol level.

EVIDENCE

In population studies, people who carry the A allele in PCSK9 tend to have slightly lower LDL and total cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - fiber intake, the mix of dietary fats, and regular activity - influence cholesterol far more.

SOURCES

PubMed 24097068 | PubMed 29748315

LDL & total cholesterol tendency

TRIB1 region

POPULATION STUDY

rs2954029 | GT A/T | DOSE 0/1 | REF/ALT A/T | STUDIED T (1 of 2) | GRCh38 +

INTERPRETATION

One A allele and one T allele - a heterozygous A/T genotype, one from each parent. In population studies LDL and total cholesterol tend to run slightly higher than for the T/T type, with each A copy adding a small leaning upward. This is a small population-level shift, not a medical prediction.

EVIDENCE

In population studies, people who carry the T allele in the TRIB1 region tend to have slightly lower LDL and total cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - fiber intake, the balance of dietary fats, and staying active - influence cholesterol far more.

SOURCES

PubMed 24097068

LDL cholesterol tendency

APOE region

POPULATION STUDY

rs157580 | GT G/A | DOSE 0/1 | REF/ALT G/A | STUDIED A (1 of 2) | GRCh38 +

INTERPRETATION

At this position you carry the heterozygous G/A genotype, one allele from each parent. In population studies the A allele is associated with a slightly higher LDL reading on average. This is a weak population-level leaning and, on its own, does not meaningfully predict your individual LDL level; a blood lipid panel is what tells you your actual number.

EVIDENCE

In population studies, people who carry the A allele in the APOE region tend to have slightly higher LDL cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, fiber and saturated-fat intake, physical activity, and body weight - influence cholesterol far more.

SOURCES

PubMed 19060911

LDL cholesterol tendency

FADS1

POPULATION STUDY

rs174547 | GT C/C | DOSE 1/1 | REF/ALT T/C | STUDIED C (2 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the C variant - a C/C genotype. The C allele is associated with lower LDL/non-HDL cholesterol but with higher triglycerides at this pleiotropic FADS1 locus. The shift is small.

EVIDENCE

In population studies, people who carry the C allele at this FADS1 marker tend to have slightly lower LDL and non-HDL cholesterol. FADS1 helps convert dietary omega-3 and omega-6 fats, and the same allele tends to nudge triglycerides upward, so this leaning covers the LDL side only. It's a small, common genetic tendency, not a prediction about any one person. Many other genes and everyday habits - fiber intake, the balance of fats in the diet, physical activity, and body weight - influence cholesterol far more. When this applies: FADS1 is pleiotropic: the C allele lowers LDL/non-HDL cholesterol but raises triglycerides. Directions differ by lipid fraction.

SOURCES

PubMed 31566214 | PubMed 29615537 | PubMed 19060906

LDL cholesterol tendency

PCSK9

POPULATION STUDY

rs11206510 | GT C/C | DOSE 1/1 | REF/ALT T/C | STUDIED C (2 of 2) | GRCh38 +

INTERPRETATION

Both copies carry the C allele - a C/C genotype. In population studies the C allele is associated with a modestly lower LDL cholesterol reading, so C/C sits at the lower end of this range. Informational, not a concern; a blood test reflects your real values.

EVIDENCE

PCSK9 is a gene that helps the body clear LDL cholesterol. In population studies, people who carry the C allele near PCSK9 tend to have slightly lower LDL cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, fiber and oily fish, physical activity, and body weight - influence cholesterol far more.

SOURCES

PubMed 18193043

Total & LDL cholesterol tendency

FGF21 region

POPULATION STUDY

rs838145 | GT A/A | DOSE 1/1 | REF/ALT G/A | STUDIED A (2 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the A allele - an A/A genotype. In population studies the A allele is associated with marginally lower measured total/LDL cholesterol - a small, favorable nudge in a routine blood measurement relative to G-carriers.

EVIDENCE

In population studies, people who carry the A allele at rs838145 (near FGF21) tend to have very slightly lower total and LDL cholesterol. It's a tiny, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, fiber and saturated-fat intake, and activity - influence cholesterol far more.

SOURCES

PubMed 41044249 | PubMed 34594039

Total & LDL cholesterol tendency

LIPG

POPULATION STUDY

rs4939883 | GT C/C | DOSE 1/1 | REF/ALT T/C | STUDIED C (2 of 2) | GRCh38 +

INTERPRETATION

Both copies carry the C allele - a C/C genotype. In population studies this genotype is linked to modestly higher total and LDL cholesterol than T/T, with each C copy adding a small upward shift. This is a small leaning, not a medical prediction.

EVIDENCE

In population studies, people who carry the C allele in LIPG tend to have slightly higher total and LDL cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, soluble fiber, oily fish, and regular activity - influence cholesterol far more.

SOURCES

PubMed 19060911 | PubMed 19936222

Total cholesterol level tendency APOB

POPULATION STUDY

rs679899 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the G allele - a G/G genotype, with no copies of the A allele present. In research cohorts, each A copy tracks with modestly lower total cholesterol, so G/G sits at the higher end of this small leaning.

EVIDENCE

In population studies, people who carry the A allele in APOB tend to have slightly lower total cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, soluble fiber, oily fish, and regular activity - influence cholesterol far more. Note: APOB carries more than one cholesterol signal that are only partly correlated, so this variant and the other APOB marker in this report are largely separate signals and can point in different directions.

SOURCES

GWAS rs679899 | PubMed 36499290

Total cholesterol tendency LIPC

POPULATION STUDY

rs2070895 | GT G/A | DOSE 0/1 | REF/ALT G/A | STUDIED A (1 of 2) | GRCh38 +

INTERPRETATION

One A allele and one G allele - a heterozygous G/A genotype, one from each parent. In research cohorts the A allele is associated with a slightly higher total cholesterol reading on average. This is a weak, relative population-level shift - a lipid panel is the real measure.

EVIDENCE

In population studies, people who carry the A allele at this LIPC site tend to have slightly higher total cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, fiber and saturated-fat intake, physical activity, and body weight - influence cholesterol far more.

SOURCES

GWAS rs2070895 | PubMed 38506378

02.03 LDL cholesterol & apolipoprotein B tendency (APOB) 1x

LDL cholesterol & apolipoprotein B tendency

APOB

POPULATION STUDY

rs693 | GT A/A | DOSE 1/1 | REF/ALT G/A | STUDIED A (2 of 2) | GRCh38 +

INTERPRETATION

Both copies here carry the A allele - an A/A genotype. In research cohorts this is linked to a slightly higher LDL / apoB than G/G. The per-copy effect is small - a relative, population-level shift, not a prediction of any individual's LDL or apoB. Many other factors also influence cholesterol.

EVIDENCE

In population studies, people who carry the A allele in APOB tend to have slightly higher LDL cholesterol and apolipoprotein B. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, unsaturated fats, fiber, and regular activity - influence these markers far more.

SOURCES

PubMed 41044249

02.04 Alcohol flush tendency (ALDH2) 1x

Alcohol flush tendency

ALDH2

POPULATION STUDY

rs671 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the G variant - a G/G genotype, with no copy of the A variant present. In population studies, people with G/G tend to sit toward the lower end of the alcohol-flush range; the A variant that shifts the trait upward is absent here. Each A copy is associated with a small upward shift.

EVIDENCE

rs671 is the ALDH2 variant linked to the alcohol-flush response. In population studies each A allele is associated with a stronger flush tendency, a small per-copy effect, with strong evidence - a relative, population-level shift, not your absolute outcome. Many other genetic and lifestyle factors also influence how a person responds to alcohol.

SOURCES

PubMed 24277619

02.05 Androstenone smell perception tendency (OR7D4 R88W) 1x

How androstenone smells - sweaty vs. sweet/vanilla

POPULATION STUDY

rs61729907 | GT G/A | DOSE 0/1 | REF/ALT G/A | STUDIED A (1 of 2) | GRCh38 +

INTERPRETATION

In population studies, people who carry the A allele tend to find androstenone weaker or less sharp. One copy here gives a mild leaning toward sensing it less. If certain meats ever smell off, choosing well-cooked, fresh cuts keeps the experience pleasant.

EVIDENCE

Androstenone is a compound found in sweat and in some foods like pork; to some noses it smells sweaty or sharp, to others sweet or vanilla-like, and to many it barely registers. A well-studied change in the OR7D4 smell-receptor gene helps set which way it lands. It is a small, common difference in how the nose reads this single scent.

SOURCES

PubMed 17873857

02.06 Asparagus-urine odor detection tendency (OR2M7) 1x

Detection of the asparagus-urine odor

POPULATION STUDY

rs4481887 | GT G/G | DOSE 1/1 | REF/ALT A/G | STUDIED G (2 of 2) | GRCh38 +

INTERPRETATION

Two G copies carry the leaning toward not detecting the asparagus-urine odor. This is just a harmless difference in scent perception.

EVIDENCE

In population studies, people who carry the G allele tend to lean toward a reduced ability to detect the asparagus-urine odor, while A-carriers lean toward being able to smell it. It's a small, common genetic leaning, not a prediction about any one person. Many genes and everyday habits - how much asparagus is eaten, overall sense of smell, and surrounding scents - matter more.

SOURCES

PubMed 20585627

02.07 Bilirubin level tendency (UGT1A1) 3/4 markers

Bilirubin level tendency

UGT1A1

POPULATION STUDY

rs887829 | GT C/T | DOSE 0/1 | REF/ALT C/T | STUDIED T (1 of 2) | GRCh38 +

INTERPRETATION

You inherited one T variant and one C variant - a heterozygous C/T genotype. In population studies the T variant is associated with somewhat higher serum bilirubin (a higher-bilirubin pattern). Mildly raised unconjugated bilirubin is benign, so this is an informational tendency rather than a concern.

EVIDENCE

In population studies, people who carry the T allele in UGT1A1 tend to have higher bilirubin - the common higher-bilirubin pattern, which is benign and harmless, and at these mild levels is generally regarded as neutral or even favorable. It's a small, common genetic leaning, not a prediction about any one person. Everyday factors like hydration, fasting, and normal day-to-day variation also nudge bilirubin around.

SOURCES

PubMed 38826804 | PubMed 36580335 | PubMed 35050183

Bilirubin level tendency

UGT1A1

POPULATION STUDY

rs4148323 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the G variant - a G/G genotype, with no copies of the A variant present. In population studies, people with G/G tend to sit toward the lower end of the bilirubin range; the A variant that would shift the trait upward is absent here. This is a small per-copy effect - a population-level shift, not your absolute outcome.

EVIDENCE

In population studies, people who carry the A allele in UGT1A1 tend to have slightly higher bilirubin - a common, benign higher-bilirubin pattern, which is harmless. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday factors - hydration, fasting, and normal day-to-day variation - also nudge bilirubin around.

SOURCES

PubMed 20639394 | PubMed 23371916

Bilirubin tendency UGT1A1

POPULATION STUDY

rs6742078 | GT G/T | DOSE 0/1 | REF/ALT G/T | STUDIED T (1 of 2) | GRCh38 +

INTERPRETATION

You have one T allele and one G allele - a heterozygous G/T genotype. In research, the T allele is associated with somewhat higher serum bilirubin (a higher-bilirubin pattern). Mildly raised unconjugated bilirubin in this range is benign, so this is an informational tendency, not a concern.

EVIDENCE

In population studies, people who carry the T allele in UGT1A1 tend to have slightly higher bilirubin - the common higher-bilirubin pattern, which is benign and harmless, and at these mild levels is generally regarded as neutral or even favorable. It's a small, common genetic leaning, not a prediction about any one person. Everyday factors like hydration, fasting, and normal day-to-day variation also nudge bilirubin around.

SOURCES

PubMed 23371916 | PubMed 19414484

02.08 Bitter-glucoside (salicin) taste sensitivity tendency (TAS2R16) 1x

Sensitivity to bitter glucoside (salicin) tastes

POPULATION STUDY

rs846664 | GT A/A | DOSE 0/0 | REF/ALT A/C | STUDIED C (0 of 2) | GRCh38 +

INTERPRETATION

Two A copies carry the leaning toward higher sensitivity to bitter glucoside tastes. Gentle, repeated exposure can help bitter foods feel more familiar.

EVIDENCE

In population studies, people who carry the C allele tend to lean toward lower sensitivity to certain bitter glucoside tastes such as salicin. It's a small, common genetic leaning, not a prediction about any one person. Many genes and everyday habits - overall diet variety, repeated exposure to bitter foods, and how foods are prepared - matter more.

SOURCES

PubMed 24177185 | Source

02.09 Bitter-taste perception tendency (TAS2R38) 3x

Bitter taste perception tendency

TAS2R38

POPULATION STUDY

rs1726866 | GT A/A | DOSE 1/1 | REF/ALT G/A | STUDIED G (0 of 2) | GRCh38 +

INTERPRETATION

You have the A/A genotype at this single TAS2R38 position. The A (Val262) allele tends to occur in the AVI haplotype, but a single SNP does not determine taster status - that depends on the full TAS2R38 haplotype (all three positions together). This is normal taste variation.

EVIDENCE

rs1726866 is one of the three positions (here Ala262Val) that make up the TAS2R38 PAV/AVI bitter-taste haplotype. A single SNP does not determine taster status - that depends on the full combination of all three TAS2R38 positions read together (rs713598, rs1726866, rs10246939). The G (Ala262) allele tends to occur in the PAV haplotype and the A (Val262) allele in the AVI haplotype, but on their own they do not fix how strongly you taste bitter compounds. This is normal taste-perception variation. Note: this is one of three correlated variants in/near TAS2R38 reported for this trait. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal - not independent confirmations.

SOURCES

PubMed 31005965 PubMed 30223776

Bitter-taste perception tendency

TAS2R38

POPULATION STUDY

rs713598 | GT C/C | DOSE 0/0 | REF/ALT C/G | STUDIED C (2 of 2) | GRCh38 +

INTERPRETATION

You have the C/C genotype at this single TAS2R38 position. The C (Pro49) allele tends to occur in the more-tasting (PAV) haplotype, but this is just one of three positions - taster status cannot be read from this SNP alone and requires all three TAS2R38 positions together. A normal taste difference, not a health concern.

EVIDENCE

rs713598 (Pro49Ala) is one of three positions that make up the TAS2R38 PAV/AVI bitter-taste haplotype, which helps set how strongly someone tastes bitter compounds such as PTC and PROP (found in foods like broccoli, kale and coffee). The C (Pro49) allele tends to occur in the more-tasting (PAV) haplotype and the G (Ala49) allele in the less-tasting (AVI) haplotype. A single SNP does not determine taster status - that depends on the full combination of all three TAS2R38 positions read together (rs713598, rs1726866, rs10246939). This is a normal taste variation, not a health concern. Note: this is one of 3 correlated variants in/near TAS2R38 reported for this trait. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal - not 3 independent confirmations.

SOURCES

PubMed 20675712

Bitter-taste perception tendency

TAS2R38

POPULATION STUDY

rs10246939 | GT T/T | DOSE 0/0 | REF/ALT T/C | STUDIED C (0 of 2) | GRCh38 +

INTERPRETATION

You have the T/T genotype at this single TAS2R38 position. This is one of three positions in the PAV/AVI bitter-taste haplotype; T tends to occur in the less-tasting (AVI) haplotype, but your actual taster status cannot be read from this SNP alone - it depends on the full TAS2R38 haplotype (all three positions together). This is normal taste variation, not a health concern.

EVIDENCE

rs10246939 is one of the three positions (here Ile296Val) that make up the TAS2R38 PAV/AVI bitter-taste haplotype. A single SNP does not determine taster status - that depends on the full combination of all three TAS2R38 positions read together (rs713598, rs1726866, rs10246939). The C and T alleles here are each part of, respectively, the more-tasting and less-tasting haplotype, but on their own they do not fix how strongly you taste bitter compounds like PROP/PTC. This is normal taste-perception variation, not a medical concern. Note this is one of three correlated variants in/near TAS2R38 reported for this trait - neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal rather than independent confirmations.

SOURCES

PubMed 30223776 | PubMed 23966204

02.10 Coffee & caffeine clearance tendency (CYP1A2) 1x

Coffee & caffeine clearance tendency

CYP1A2

SUBSTANCE RESPONSE

rs762551 | GT A/A | DOSE 1/1 | REF/ALT C/A | STUDIED A (2 of 2) | GRCh38 +

INTERPRETATION

The A/A genotype is the fast caffeine-metaboliser pattern - caffeine is cleared quickly. Relevant only for people who consume caffeine; how caffeine feels day to day depends more on dose, timing and sleep.

EVIDENCE

In population studies, people who carry the C allele at this CYP1A2 marker tend to clear caffeine more slowly, so caffeine lingers longer than it does for fast metabolizers carrying the A allele. It's a small, common genetic leaning, not a prediction about any one person, and it reflects how caffeine is handled rather than coffee's other ingredients. Many other genes and everyday habits - coffee and tea intake, timing of the last cup, sleep and overall diet - shape the picture far more. When this applies: Applies only if you drink coffee or other caffeinated drinks; if you avoid caffeine this variant has no practical effect on you. It reflects how quickly caffeine is cleared, nothing more. Early-stage finding - read it as a small, common tendency, not a firm result.

SOURCES

PubMed 16522833 | PubMed 29514871

02.11 Fasting blood sugar tendency

3x

Fasting blood sugar tendency

GCK

POPULATION STUDY

rs4607517 | GT G/A | DOSE 0/1 | REF/ALT G/A | STUDIED A (1 of 2) | GRCh38 +

INTERPRETATION

You inherited one A allele and one G reference - a heterozygous G/A genotype, one from each parent. In population studies the A allele is associated with slightly higher fasting blood sugar, a small per-copy effect. This is a weak, relative population-level shift and, on its own, does not meaningfully predict an individual's blood sugar.

EVIDENCE

In population studies, people who carry the A allele at rs4607517 (GCK) tend to have slightly higher fasting blood sugar. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, carbohydrate quality, body weight, and activity - influence blood sugar far more.

SOURCES

PubMed 25631608 | PubMed 20081858 | PubMed 28869590

Fasting glucose & HbA1c tendency

MTNR1B

POPULATION STUDY

rs10830963 | GT C/C | DOSE 0/0 | REF/ALT C/G | STUDIED G (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the C allele - a C/C genotype, with no copies of the glucose-associated G allele. In population studies measured fasting glucose and HbA1c sit at the lower end of the range; each G copy would nudge them modestly upward.

EVIDENCE

In population studies, people who carry the G allele at rs10830963 (MTNR1B) tend to have modestly higher fasting blood sugar and HbA1c. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, carbohydrate quality, body weight, and activity - influence blood sugar far more.

SOURCES

PubMed 25631608 | PubMed 20081858 | PubMed 19060907

Fasting glucose tendency FADS1

POPULATION STUDY

rs174550 | GT C/C | DOSE 1/1 | REF/ALT T/C | STUDIED T (0 of 2) | GRCh38 +

INTERPRETATION

You have the C/C genotype at FADS1, associated on average with slightly lower fasting blood glucose in population studies (a small effect).

EVIDENCE

In population studies, people who carry the T allele at FADS1 tend to have slightly higher fasting blood glucose, while C-carriers tend to run a touch lower. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, physical activity, sleep, and body weight - influence blood glucose far more.

SOURCES

GWAS rs174550

02.12 Folate metabolism & homocysteine tendency (MTHFR) 1x

Folate metabolism & homocysteine tendency MTHFR

POPULATION STUDY

rs1801133 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

Both copies carry the G allele, a G/G genotype with no A copies. Without the less-active A variant, homocysteine sits toward the lower end and folate toward the higher end. A small, population-level pattern.

EVIDENCE

In population studies, the A allele at rs1801133 (the MTHFR C677T variant) codes for a less active MTHFR enzyme. Each A copy is associated with higher homocysteine and lower circulating folate, two measures that move in opposite directions, each a small per-copy effect. This is a relative population-level shift, not your absolute chance, and on its own it does not diagnose anything. Many other genetic and lifestyle factors, including dietary folate, also influence these levels.

SOURCES

PubMed 23754956 | PubMed 30339177

02.13 HbA1c tendency (HFE) 1x

HbA1c tendency HFE

POPULATION STUDY

rs1800562 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the G reference allele - a G/G genotype, with no copies of the A allele. In population studies, people with G/G tend to sit toward the higher end of the HbA1c range for this site, since the A allele linked to modestly lower HbA1c is absent. Each A allele is associated with slightly lower HbA1c - a small, population-level shift.

EVIDENCE

In population studies, people who carry the A allele at this HFE marker tend to have a slightly lower HbA1c reading. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - overall carbohydrate intake, physical activity, body weight, and sleep - influence blood-sugar measures far more.

SOURCES

PubMed 39280063

02.14 Higher salt and sodium intake preference tendency (SCNN1B) 1x

Salt and sodium intake preference

POPULATION STUDY

rs239345 | GT T/A | DOSE 0/1 | REF/ALT T/A | STUDIED A (1 of 2) | GRCh38 +

INTERPRETATION

One copy of the A allele, a common intermediate leaning toward a stronger salt preference in population studies. The signal is small and low-confidence; tasting before adding salt and leaning on spices helps moderate intake.

EVIDENCE

In population studies, people who carry the A allele tend to prefer somewhat saltier food and higher sodium intake within the normal everyday range. It's a small, common genetic leaning, not a prediction about any one person, and everyday habits like cooking choices, taste habituation, herbs and spices used in place of salt, and overall diet matter more. This is an early finding from a single study, so treat it as a weak signal rather than a settled result.

SOURCES

PubMed 36899055 | Source

02.15 Iron level tendency 7x

Iron level tendency

TFR2

POPULATION STUDY

rs7385804 | GT C/C | DOSE 0/0 | REF/ALT C/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

Both copies carry the C variant, a C/C genotype with no copy of the A variant. At this TFR2 site serum iron and transferrin saturation lean toward the lower end of the range. A small, population-level leaning, not your absolute level.

EVIDENCE

At this TFR2 variant, each A allele is associated in population studies with slightly higher serum iron and transferrin saturation (how much of the blood's iron-carrying capacity is filled). Its effect on red blood cells is mixed rather than uniform: cells tend to run a little larger and more hemoglobin-rich, but slightly fewer in number, so it is not a simple more-red-cells effect. A small leaning, a normal biomarker variation, not a medical prediction. It is one of a few correlated variants in or near TFR2 usually inherited together (linkage disequilibrium), so treat them as one shared signal.

SOURCES

PubMed 33536631 | PubMed 25352340

Iron level tendency

TMPRSS6

POPULATION STUDY

rs4820268 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the G variant - a G/G genotype, with no copies of the A allele linked to higher iron. People with G/G tend toward the lower end of the measured serum-iron range associated with this variant. Iron status is also strongly shaped by diet, blood loss, and other genes.

EVIDENCE

In population studies, the A allele at this TMPRSS6 marker is associated with higher measured serum iron, while the G allele tracks with lower iron. This is a quantitative biomarker tendency - a relative, population-level shift, not a medical diagnosis. This is one of several correlated variants in/near TMPRSS6 for this trait; neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal - not several independent confirmations.

SOURCES

GWAS rs4820268

Iron levels & ferritin

HFE C282Y

POPULATION STUDY

rs1800562 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

Both copies here carry the G allele, a G/G genotype, with no copy of the C282Y (A) variant. Nothing at this marker leans your iron levels or ferritin in either direction.

EVIDENCE

rs1800562 is the HFE C282Y variant (the A allele), the strongest common iron-raising variant in HFE. Each A copy is associated with higher serum iron, transferrin saturation and ferritin. About 1 in 10 people of European ancestry carry one copy, where the effect on iron stores is usually minor. Two copies (A/A) sit at the high end of the iron range and are the genotype most linked to iron building up over time, though many people with A/A still stay in the normal range. Iron status is measured directly with simple blood tests (serum iron, transferrin saturation and ferritin).

CLINVAR REVIEW

1 of 4 review stars - criteria provided, conflicting classifications. Classified: Conflicting classifications of pathogenicity; other; risk factor. This says how much expert review stands behind the classification, not how strong the effect is.

SOURCES

ClinVar | PubMed 21483845 | PubMed 25352340

Iron levels & transferrin saturation

HFE H63D

POPULATION STUDY

rs1799945 | GT C/G | DOSE 0/1 | REF/ALT C/G | STUDIED G (1 of 2) | GRCh38 +

INTERPRETATION

You carry one H63D (G) variant and one C allele, a heterozygous C/G genotype. This is a common single-copy H63D carrier result. On its own it nudges iron and transferrin saturation only slightly toward the higher end, and rarely affects iron stores in any meaningful way. Higher iron from HFE is mostly seen when H63D is paired with the HFE C282Y variant (rs1800562).

EVIDENCE

rs1799945 is the HFE H63D variant (the G allele), one of the most common gene variants in people of European ancestry (about 1 in 5 carry a copy). The G allele leans iron markers such as serum iron and transferrin saturation slightly higher. The effect is small, and on its own H63D, even in two copies, rarely changes iron stores in a way that matters. Higher iron from HFE is mostly seen when H63D is paired with the HFE C282Y variant (rs1800562). Iron status is shaped far more by diet, blood loss, and other genes than by this single marker.

CLINVAR REVIEW

1 of 4 review stars - criteria provided, conflicting classifications. Classified: Conflicting classifications of pathogenicity; other. This says how much expert review stands behind the classification, not how strong the effect is.

SOURCES

ClinVar | PubMed 25352340

Iron levels & transferrin saturation tendency

TMPRSS6

POPULATION STUDY

rs855791 | GT A/A | DOSE 0/0 | REF/ALT A/G | STUDIED G (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the A allele - an A/A genotype, with no copies of the G allele present. In population studies people with A/A tend to sit toward the lower end of the iron-levels and transferrin-saturation range; the G allele (which shifts the trait upward) is absent here. A small, relative, population-level leaning.

EVIDENCE

At this TMPRSS6 variant, each G allele is associated in population studies with higher measured iron levels and transferrin saturation. This is a small leaning - a normal biomarker variation, not a medical prediction. Note: this is one of 3 correlated variants in/near TMPRSS6 reported for this trait. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal - not 3 independent confirmations.

SOURCES

PubMed 33536631 | PubMed 25352340 | PubMed 38789058

Iron levels tendency

TF / transferrin

POPULATION STUDY

rs8177240 | GT T/G | DOSE 0/1 | REF/ALT T/G | STUDIED T (1 of 2) | GRCh38 +

INTERPRETATION

You have the T/G genotype - one T and one G copy. This places you in the middle, with iron biomarkers nudged higher than T/T but lower than G/G. The effect is small.

EVIDENCE

This variant is in TF, the gene for transferrin - the protein that transports iron in the blood. In population studies the T allele is linked to lower iron and lower transferrin levels (with higher transferrin saturation), while the G allele tends toward higher measured iron. The shifts are small and this is a normal biomarker variation, not a medical condition; many other factors set your actual iron status. Note: this is one of 2 correlated variants in/near TF reported for this trait. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal - not 2 independent confirmations.

SOURCES

GWAS rs8177240

Transferrin / iron-binding capacity tendency

TF

POPULATION STUDY

rs3811647 | GT G/A | DOSE 0/1 | REF/ALT G/A | STUDIED A (1 of 2) | GRCh38 +

INTERPRETATION

One A and one G, an intermediate level of transferrin and iron-binding capacity, between G/G and A/A. A small per-copy leaning.

EVIDENCE

In population studies, each A allele at this TF (transferrin) marker is associated with higher transferrin and total iron-binding capacity, and with somewhat lower transferrin saturation (the share of transferrin actually carrying iron). So the A allele tracks with more iron-transport protein, not more circulating iron. A small per-copy effect with strong evidence, not a medical prediction; iron intake and many other factors shape iron status far more.

SOURCES

PubMed 21483845 | PubMed 21208937 | PubMed 19084217

02.16 Lactose tolerance tendency (LCT) 1x

Lactose tolerance tendency LCT

POPULATION STUDY

rs4988235 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the G variant - a G/G genotype, with no copies of the A variant present. In population studies, people with G/G tend to sit toward the lower end of the lactose tolerance range; the A variant (which would shift the trait upward) is absent here. Each A copy is associated with a small upward shift.

EVIDENCE

Each A allele is associated with higher lactose tolerance, a small per-copy effect - a relative, population-level shift, not your absolute chance. This is not a direct medical prediction. The evidence is strong. Lactase persistence is only relevant for people who consume dairy/lactose. Many other genetic and lifestyle factors also influence how dairy is tolerated. When this applies: Lactase persistence is only phenotypically relevant for people who consume dairy/lactose; it has no effect for those who don't.

CLINVAR REVIEW

0 of 4 review stars - no assertion criteria provided. Classified: association. This says how much expert review stands behind the classification, not how strong the effect is.

SOURCES

ClinVar

02.17 Omega-3 fatty acid levels tendency (FADS1/FADS2) 1x

Omega-3 fatty acid levels tendency FADS1/FADS2

POPULATION STUDY

rs174583 | GT T/T | DOSE 1/1 | REF/ALT C/T | STUDIED T (2 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the T variant - a T/T genotype, with no copies of the omega-3-tracking C variant. In population studies the T allele is associated with lower long-chain omega-3, so people with T/T tend to sit toward the lower end (each C copy is associated with a small leaning toward higher omega-3). Many other factors also influence omega-3 status.

EVIDENCE

At the FADS1/FADS2 locus, the T allele is associated with lower long-chain omega-3 levels (DHA/EPA/DPA) in population studies, while the C allele tracks with higher omega-3. Because higher omega-3 is generally favorable, people carrying the C allele tend to sit further toward that end. This is a relative, population-level shift, not your absolute level. Many other genetic and lifestyle factors also influence omega-3 status. This variant is reported for more than one trait. When this applies: Higher omega-3 is generally favorable, so the C allele (which preserves it) is treated as the helpful allele here.

SOURCES

PubMed 18936223 | PubMed 35120996 | PubMed 35050183

02.18 Omega-3/omega-6 fatty acid metabolism tendency (FADS1) 1x

Omega-3/omega-6 fatty acid metabolism tendency FADS1 POPULATION STUDY

rs174550 | GT C/C | DOSE 1/1 | REF/ALT T/C | STUDIED C (2 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the C variant - a C/C genotype, with no copies of the T variant present. People with C/C tend to sit toward the lower end of the omega-3/omega-6 fatty acid metabolism range; the T variant (which would shift the trait upward) is absent here.

EVIDENCE

At this FADS1 marker, each C allele is associated with lower omega-3/omega-6 fatty acid metabolism in population studies. This is a population-level association, not a medical prediction, and many other genetic and dietary factors also influence fatty-acid metabolism. This variant is reported for more than one trait.

SOURCES

PubMed 24823311

02.19 Omega-6 fatty acid metabolism tendency (FADS1) 1x

Omega-6 fatty acid metabolism tendency FADS1 POPULATION STUDY

rs174547 | GT C/C | DOSE 1/1 | REF/ALT T/C | STUDIED C (2 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the C variant - a C/C genotype, with no copies of the T variant present. People with C/C tend to sit toward the lower end of the omega-6 fatty acid metabolism range; the T variant (which would shift the trait upward) is absent here.

EVIDENCE

At this FADS1 marker, each C allele is associated with lower omega-6 fatty acid metabolism in population studies. This is a population-level association, not a medical prediction, and many other genetic and dietary factors also influence fatty-acid metabolism. Moderate evidence. This variant is reported for more than one trait.

SOURCES

PubMed 26584805 PubMed 24823311

02.20 Perception of white-musk (Galaxolide) smell intensity tendency (OR4D6) 1x

Perceived intensity of white-musk (Galaxolide) scent

POPULATION STUDY

rs1453542 | GT G/G | DOSE 0/0 | REF/ALT G/C | STUDIED C (0 of 2) | GRCh38 +

INTERPRETATION

Two G copies lean toward perceiving white-musk scents at fuller intensity. Trying scents on skin before choosing is a practical everyday lever.

EVIDENCE

In population studies, people who carry the C allele tend to lean toward perceiving white-musk (Galaxolide) scents as less intense. It's a small, common genetic leaning, not a prediction about any one person. Many genes and everyday habits - overall scent exposure, surrounding smells, and individual preference - matter more.

SOURCES

PubMed 35113854

02.21 Sucrose-rich food liking tendency (sucrase-isomaltase) 1x

Sucrose-rich food liking / intake

POPULATION STUDY

rs9290264 | GT C/A | DOSE 0/1 | REF/ALT C/A | STUDIED A (1 of 2) | GRCh38 +

INTERPRETATION

One A copy, a small lean toward liking slightly fewer sucrose-rich foods.

EVIDENCE

rs9290264 is a coding change in SI (sucrase-isomaltase), the gut enzyme that digests table sugar. The A allele leans toward a less active form, and carriers tend to like and eat slightly fewer sucrose-rich foods. A small, common lean, not a prediction; overall diet and how much sugar is around matter more.

SOURCES

PubMed 39542403

02.22 Sweet and sugary food intake drive tendency (SLC2A2) 1x

Drive toward sweet and sugary foods

POPULATION STUDY

rs5400 | GT G/A | DOSE 0/1 | REF/ALT G/A | STUDIED A (1 of 2) | GRCh38 +

INTERPRETATION

One A copy carries a partial leaning toward higher sugary-food intake. Keeping satisfying snack alternatives on hand is a simple everyday lever.

EVIDENCE

In population studies, people who carry the A allele tend to lean toward a higher intake of sweet and sugary foods. It's a small, common genetic leaning, not a prediction about any one person. Many genes and everyday habits - meal regularity, keeping satisfying alternatives on hand, sleep, and mindful eating - matter more.

SOURCES

PubMed 18349384

02.23 Sweet taste preference & sugar intake tendency (FGF21)

1/2 markers

Carbohydrate & sugar (sweet) preference tendency

FGF21

POPULATION STUDY

rs838145 | GT A/A | DOSE 1/1 | REF/ALT G/A | STUDIED G (0 of 2) | GRCh38 +

INTERPRETATION

You have the A/A genotype - two copies of the allele linked in population studies to a weaker preference for sweet foods and a lower share of carbohydrate/sugar in the diet, with relatively more fat and protein. The effect is small; your actual diet still depends mostly on habits and what's available.

EVIDENCE

This variant sits in the FGF21 region. FGF21 is a liver hormone that helps regulate appetite for sugar and sweet foods. The best-replicated signal here is on diet preference: in population studies the G allele is linked to higher carbohydrate and sugar (sweet) intake and somewhat lower fat intake, while the A allele leans the other way (less sugar, relatively more fat/protein). The per-copy effect is very small, so this is a mild dietary leaning, not a strong driver of what anyone eats.

SOURCES

GWAS rs838145

02.24 Triglyceride tendency (FADS1)

1x

Triglyceride tendency

FADS1

POPULATION STUDY

rs174546 | GT T/T | DOSE 1/1 | REF/ALT C/T | STUDIED T (2 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the T variant - a T/T genotype. The T allele is associated with higher triglycerides but also with lower LDL and total cholesterol at this pleiotropic FADS1 locus. The triglyceride shift is small.

EVIDENCE

In population studies, people who carry the T allele at this FADS1 marker tend to have slightly higher triglycerides. FADS1 helps convert dietary omega-3 and omega-6 fats, and the same allele tends to nudge LDL and total cholesterol downward, so it isn't a uniformly 'high cholesterol' leaning. It's a small, common genetic tendency, not a prediction about any one person. Many other genes and everyday habits - sugar and refined-carbohydrate intake, oily fish and omega-3 in the diet, alcohol, physical activity, and body weight - influence triglycerides far more. When this applies: FADS1 is pleiotropic: the T allele raises triglycerides but lowers LDL/total/HDL cholesterol. Directions differ by lipid fraction.

SOURCES

PubMed 24097068 | PubMed 20686565

02.25 Violet and floral (beta-ionone) smell sensitivity tendency (OR5A1) 1x

Sensitivity to violet/floral (beta-ionone) scent

POPULATION STUDY

rs6591536 | GT A/A | DOSE 1/1 | REF/ALT G/A | STUDIED G (0 of 2) | GRCh38 +

INTERPRETATION

In population studies, people who carry the G allele tend to be more sensitive to the violet/floral beta-ionone scent. With no copies here, the leaning points the other way, so this floral note may read as faint. Smelling scents in fresh air rather than a crowded counter helps subtle notes stand out.

EVIDENCE

Beta-ionone is the molecule behind the soft, violet-like floral note found in many flowers, fruits, and perfumes. A well-studied variant in the OR5A1 smell-receptor gene strongly shapes whether this scent reads as a pleasant floral aroma or barely registers. It is a small, common difference in how the nose is tuned.

SOURCES

PubMed 23910657

02.26 Vitamin E level tendency (CYP4F2) 1x

Vitamin E level tendency

CYP4F2

SUBSTANCE RESPONSE

rs2108622 | GT C/T | DOSE 0/1 | REF/ALT C/T | STUDIED T (1 of 2) | GRCh38 +

INTERPRETATION

C/T genotype at rs2108622 (CYP4F2) - one copy of each allele. The T (V433M) allele partly reduces CYP4F2's ability to break down vitamin E, placing this pattern in the middle of the vitamin E range with supplementation, between C/C and T/T. Many other genetic and dietary factors also influence vitamin E levels.

EVIDENCE

rs2108622 is the CYP4F2 V433M variant (the alternate allele is T). In research, each T allele is associated with slightly higher vitamin E (tocopherol) levels when taking vitamin E supplements. The T variant reduces CYP4F2's tocopherol-degrading activity, so less vitamin E is broken down and blood levels rise. This is a small, informational tendency that only matters with supplementation - without it the effect is negligible. Many other genetic and dietary factors also influence vitamin E levels. This variant is reported for more than one trait; the per-trait effects come from different studies and scales and are not directly comparable. When this applies: This only matters if you take vitamin E (tocopherol) supplements. The T allele slows the enzyme (CYP4F2) that breaks vitamin E down, so supplement-takers with T copies tend to reach slightly higher blood vitamin E levels. Without supplementation the effect is negligible. It is a small, informational tendency - not a health risk. Early-stage finding - read it as a small, common tendency, not a firm result.

SOURCES

PubMed 22437554 | PubMed 17341693

02.27 HDL cholesterol tendency

18/20 markers

HDL ('good') cholesterol tendency

APOB

POPULATION STUDY

rs676210 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the G variant - a G/G genotype, with no copies of the HDL-raising A variant present. In research cohorts this pattern misses the modest HDL nudge A-carriers show at this site - slightly lower for each missing A copy relative to A-carriers (a relative, population-level shift).

EVIDENCE

In population studies, people who carry the A allele in the APOB region tend to have slightly higher HDL ('good') cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - regular activity, diet, oily fish, and weight - influence HDL far more.

SOURCES

PubMed 32203549

HDL cholesterol tendency

ALDH2

POPULATION STUDY

rs671 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

You have the G/G genotype at ALDH2, associated with typical HDL ('good') cholesterol levels at this gene in population studies.

EVIDENCE

In population studies, people who carry the A allele at this ALDH2 marker tend to have slightly lower HDL cholesterol. Much of this is indirect: carriers often experience an alcohol flushing response and tend to drink less, and since moderate alcohol intake nudges HDL upward, less drinking leaves HDL a touch lower - a harmless leaning, not a problem in itself. It's a small, common genetic tendency, not a prediction about any one person. Many other genes and everyday habits - aerobic activity, the types of fat in the diet, body weight, and not smoking - influence HDL far more.

SOURCES

PubMed 30334266

HDL cholesterol tendency

ANGPTL4

POPULATION STUDY

rs116843064 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the G allele, a G/G genotype, with no copies of the A (lower-activity) allele. In population studies people with G/G tend to sit toward the lower end of the HDL cholesterol range, since each A allele is associated with a small upward shift.

EVIDENCE

ANGPTL4 is part of how the body handles blood fats. In population studies, people who carry the A allele at this ANGPTL4 variant tend to have slightly higher HDL cholesterol, alongside a generally favorable lean toward lower triglycerides. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits, diet, oily fish, physical activity, and body weight, influence blood fats far more.

SOURCES

PubMed 32203549 | PubMed 29084231 | PubMed 26933753

HDL cholesterol tendency

FADS1

POPULATION STUDY

rs174550 | GT C/C | DOSE 1/1 | REF/ALT T/C | STUDIED C (2 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the C variant - a C/C genotype, with no copies of the higher-HDL T variant. People with C/C tend to sit at the lower-HDL end here; each T copy is associated with slightly higher HDL.

EVIDENCE

In population studies, people who carry the T allele at FADS1 tend to have slightly higher HDL cholesterol, while C-carriers tend to run a touch lower. FADS1 helps convert dietary omega-3 and omega-6 fats, so this is a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, oily fish and other fat sources, physical activity, and body weight - shape HDL cholesterol far more. When this applies: Higher HDL is generally favorable, so the T allele (which raises it) is treated as the helpful allele here.

SOURCES

PubMed 29507422

HDL ('good') cholesterol tendency

APOA1

POPULATION STUDY

rs964184 | GT C/C | DOSE 1/1 | REF/ALT G/C | STUDIED C (2 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the C variant - a C/C genotype. In population studies each C copy tracks with higher HDL cholesterol compared with the G/G baseline. This is an association, not a medical prediction.

EVIDENCE

In population studies, people who carry the C allele at this APOA1/APOA5 marker tend to have slightly higher HDL ('good') cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - exercise, body weight, diet and not smoking - influence HDL far more.

SOURCES

PubMed 32203549

HDL ('good') cholesterol tendency

APOA5

POPULATION STUDY

rs651821 | GT T/T | DOSE 1/1 | REF/ALT C/T | STUDIED T (2 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the T variant - a T/T genotype. In research cohorts, each T copy tracks with higher HDL, so T/T sits toward the higher end of the HDL range. This is a small, population-level leaning.

EVIDENCE

In population studies, people who carry the T allele in APOA5 tend to have slightly higher HDL ('good') cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - regular activity, diet, oily fish, and weight - influence HDL far more.

SOURCES

PubMed 38116116

HDL ('good') cholesterol tendency

CETP

POPULATION STUDY

rs1800777 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

At this position you carry the G/G genotype, with no copies of the A allele. In population studies the A allele is the one linked to lower HDL ('good') cholesterol, so G/G sits at the higher end relative to the A/A baseline. This is a relative, population-level shift, and HDL is also shaped by many other factors.

EVIDENCE

In population studies, people who carry the A allele at this CETP site tend to have slightly lower HDL ('good') cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, physical activity, body weight, and not smoking - influence HDL far more.

SOURCES

PubMed 31719535

HDL ('good') cholesterol tendency HNF4A POPULATION STUDY

rs1800961 GT C/C DOSE 0/0 REF/ALT C/T STUDIED T (0 of 2) GRCh38 +

INTERPRETATION

At this position you carry the C/C genotype, with no copies of the T allele. In population studies the T allele is the one linked to slightly lower HDL ('good') cholesterol, so C/C sits at the higher end relative to the T/T baseline. The effect is a very small per-copy leaning, and HDL is also shaped by many other factors.

EVIDENCE

In population studies, people who carry the T allele at this HNF4A site tend to have slightly lower HDL ('good') cholesterol. It's a very small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, physical activity, body weight, and not smoking - influence HDL far more.

SOURCES

PubMed 32203549

HDL ('good') cholesterol tendency LIPC POPULATION STUDY

rs2070895 GT G/A DOSE 0/1 REF/ALT G/A STUDIED A (1 of 2) GRCh38 +

INTERPRETATION

One A variant and one G variant - a heterozygous G/A genotype, one allele from each parent. In population studies people with one A copy tend toward a slightly higher HDL reading relative to the G/G baseline. A lipid panel is the real measure.

EVIDENCE

In population studies, people who carry the A allele at this LIPC site tend to have slightly higher HDL ('good') cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, physical activity, body weight, and not smoking - influence HDL far more. This is one of two correlated variants in/near LIPC reported for HDL cholesterol. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal, not independent confirmations.

SOURCES

PubMed 32196671

HDL cholesterol tendency

APOA2

POPULATION STUDY

rs5082 | GT G/A | DOSE 0/1 | REF/ALT G/A | STUDIED A (1 of 2) | GRCh38 +

INTERPRETATION

You inherited one G variant and one A variant - a heterozygous G/A genotype, one allele from each parent. In population studies the single G copy tracks with modestly higher HDL than A/A - a small favorable nudge, most relevant on a high-saturated-fat diet.

EVIDENCE

In population studies, people who carry the A allele in the APOA2 promoter region tend to have slightly lower HDL cholesterol, while G-carriers tend to run a touch higher. These APOA2 leanings are diet-modulated and show up most on a diet rich in saturated fat, so this is a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - overall diet, type of dietary fat, physical activity, and body weight - influence HDL cholesterol far more. When this applies: This dietary-fat leaning is strongest for people who eat a lot of saturated fat and fades on a lower-saturated-fat diet.

SOURCES

PubMed 38448586 | PubMed 19901143

HDL cholesterol tendency

APOA5

POPULATION STUDY

rs2266788 | GT A/A | DOSE 1/1 | REF/ALT G/A | STUDIED A (2 of 2) | GRCh38 +

INTERPRETATION

Both copies carry the A variant - an A/A genotype, the combination most associated with the higher-HDL shift at this site. In research cohorts each A copy is linked to a higher HDL reading compared with the G/G baseline. A lipid panel confirms the actual number.

EVIDENCE

In population studies, people who carry the A allele near APOA5 tend to have slightly higher HDL ("good") cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - regular movement, oily fish and other healthy fats, and overall diet - influence cholesterol far more.

SOURCES

PubMed 21386085

HDL cholesterol tendency

CETP

POPULATION STUDY

rs3764261 | GT C/A | DOSE 0/1 | REF/ALT C/A | STUDIED A (1 of 2) | GRCh38 +

INTERPRETATION

You inherited one A variant and one C variant - a heterozygous C/A genotype, one allele from each parent. In research cohorts this places measured HDL partway above the C/C baseline, a small per-copy leaning higher from the A variant. Many other genetic and lifestyle factors also influence cholesterol.

EVIDENCE

In population studies, people who carry the A allele at CETP tend to have slightly higher HDL cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, physical activity, alcohol, and body weight - influence HDL cholesterol far more.

SOURCES

PubMed 31211820 | PubMed 30911093

HDL cholesterol tendency

CETP

POPULATION STUDY

rs5882 | GT G/A | DOSE 0/1 | REF/ALT G/A | STUDIED A (1 of 2) | GRCh38 +

INTERPRETATION

You inherited one G (Val) variant and one A (Ile) variant - a heterozygous G/A genotype, one allele from each parent. In population studies the single G copy tracks with modestly higher HDL than A/A - a small favorable nudge.

EVIDENCE

In population studies, people who carry the A allele at CETP (the I405V variant) tend to have slightly lower HDL cholesterol, while the G allele, which lowers CETP activity, leans toward higher HDL. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, physical activity, alcohol, and body weight - influence HDL cholesterol far more.

SOURCES

PubMed 23497168

HDL cholesterol tendency

CETP

POPULATION STUDY

rs708272 | GT G/A | DOSE 0/1 | REF/ALT G/A | STUDIED A (1 of 2) | GRCh38 +

INTERPRETATION

One A variant and one G variant - a heterozygous G/A genotype, one allele from each parent. In population studies this sits between the G/G and A/A ends of the measured HDL-cholesterol range, leaning slightly higher with the A variant. A small leaning, not a personal prediction.

EVIDENCE

In population studies, people who carry the A allele at rs708272 (CETP) tend to have slightly higher HDL ('good') cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - physical activity, diet quality, and body weight - influence HDL far more.

SOURCES

PubMed 40236292

HDL cholesterol tendency

IRS1

POPULATION STUDY

rs2943641 | GT T/C | DOSE 0/1 | REF/ALT T/C | STUDIED C (1 of 2) | GRCh38 +

INTERPRETATION

You have the T/C genotype - one T allele and one C allele. In population studies the T allele is associated with higher HDL and lower triglycerides, so this genotype carries part of the favorable HDL shift relative to C/C.

EVIDENCE

This IRS1-region variant nudges insulin signalling and the lipid profile. In population studies, people who carry the C allele at rs2943641 tend to have slightly lower HDL ('good') cholesterol, while the T allele leans toward higher HDL and lower triglycerides. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, physical activity, and body weight - influence cholesterol far more.

SOURCES

PubMed 29507422 | PubMed 21353221

HDL cholesterol tendency

LIPC

POPULATION STUDY

rs1800588 | GT C/T | DOSE 0/1 | REF/ALT C/T | STUDIED T (1 of 2) | GRCh38 +

INTERPRETATION

You inherited one T variant and one C variant - a heterozygous C/T genotype, one allele from each parent. The T allele is associated with higher HDL ("good") cholesterol in research cohorts, so people with this genotype tend to sit between the two homozygous groups, with a small per-copy leaning relative to C/C.

EVIDENCE

In population studies, people who carry the T allele at LIPC tend to have slightly higher HDL cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, physical activity, alcohol, and body weight - influence HDL cholesterol far more. This is one of two correlated variants in/near LIPC reported for HDL cholesterol. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal, not independent confirmations.

SOURCES

PubMed 40841581 | GWAS rs1800588 | PubMed 29062379

HDL cholesterol tendency

LIPG

POPULATION STUDY

rs4939883 | GT C/C | DOSE 1/1 | REF/ALT T/C | STUDIED C (2 of 2) | GRCh38 +

INTERPRETATION

Both copies carry the C variant - a C/C genotype, the combination most linked to higher HDL cholesterol at this site. In population studies each C copy adds a small upward shift compared with T/T. This is a small leaning, not a medical prediction.

EVIDENCE

In population studies, people who carry the C allele in LIPG tend to have slightly higher HDL ("good") cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - regular movement, oily fish and other healthy fats, and overall diet - influence HDL cholesterol far more.

SOURCES

PubMed 19060906 | PubMed 19060911

HDL cholesterol tendency

TRIB1 region

POPULATION STUDY

rs2954029 | GT A/T | DOSE 0/1 | REF/ALT A/T | STUDIED T (1 of 2) | GRCh38 +

INTERPRETATION

One T allele and one A allele - a heterozygous A/T genotype, one from each parent. In population studies this genotype shows a partial average uplift in HDL ('good') cholesterol relative to the A/A baseline - a small leaning per T copy. A small population-level shift, not your absolute reading.

EVIDENCE

In population studies, people who carry the T allele in the TRIB1 region tend to have slightly higher HDL ("good") cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - regular movement, oily fish and other healthy fats, and overall diet - influence cholesterol far more.

SOURCES

PubMed 24097068

02.28 Triglyceride tendency

16/17 markers

Triglyceride levels tendency

APOB

POPULATION STUDY

rs676210 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the G allele - a G/G genotype, with no copies of the A allele present. In research cohorts, each A copy tracks with modestly lower triglycerides, so G/G sits at the higher end of this small leaning.

EVIDENCE

In population studies, people who carry the A allele in APOB tend to have slightly lower triglycerides. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, added sugar and alcohol intake, weight, and regular activity - influence triglycerides far more.

SOURCES

PubMed 40210677 | PubMed 32203549

Total cholesterol & triglyceride tendency

APOA2

POPULATION STUDY

rs5082 | GT G/A | DOSE 0/1 | REF/ALT G/A | STUDIED A (1 of 2) | GRCh38 +

INTERPRETATION

You inherited one G allele and one A allele - a heterozygous G/A genotype, one from each parent. In research cohorts the A allele is associated with a modestly lower triglyceride / total-cholesterol measurement, a small per-copy effect. This is a small nudge, not a medical prediction.

EVIDENCE

In population studies, people who carry the A allele at APOA2 tend to have slightly lower total cholesterol and triglyceride levels. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits, including diet, type of dietary fat, physical activity and body weight, influence these levels far more.

SOURCES

PubMed 38448586

Triglyceride & cholesterol tendency

APOA5

POPULATION STUDY

rs3135506 | GT G/G | DOSE 0/0 | REF/ALT G/C | STUDIED C (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the G allele - a G/G genotype, with no C copies. In research, triglyceride and cholesterol levels tend to sit at the lower end relative to C-carriers; each C copy would nudge the measurement up modestly. This is a population-level shift, not a medical prediction.

EVIDENCE

In population studies, people who carry the C allele of the APOA5 S19W variant tend to have slightly higher triglycerides and cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - sugar and refined-carb intake, alcohol, body weight, and regular activity - influence these levels far more.

SOURCES

PubMed 32226016 | PubMed 36220816

Triglyceride & HDL cholesterol tendency

APOC1

POPULATION STUDY

rs439401 | GT T/C | DOSE 0/1 | REF/ALT T/C | STUDIED C (1 of 2) | GRCh38 +

INTERPRETATION

One C variant and one T variant - a heterozygous T/C genotype, one allele from each parent. This tends to place people in the middle of the triglycerides and HDL cholesterol range, between the lower-end T/T and higher-end C/C genotypes. This is a small leaning, not a medical prediction.

EVIDENCE

In population studies, people who carry the C allele in the APOC1/APOE region tend to have slightly higher triglycerides and HDL cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - the balance of dietary fats, sugar and alcohol intake, body weight, and regular activity - influence blood lipids far more.

SOURCES

PubMed 30926973

Triglyceride & HDL cholesterol tendency

GCKR

POPULATION STUDY

rs780094 | GT C/C | DOSE 1/1 | REF/ALT T/C | STUDIED C (2 of 2) | GRCh38 +

INTERPRETATION

You have the C/C genotype - two copies of the C allele. In population studies this version is linked, on average, to lower fasting triglycerides, a lower triglyceride-to-HDL ratio and lower C-reactive protein, but slightly higher fasting blood glucose - a metabolic trade-off rather than a clearly favorable or unfavorable result. Many other factors also shape these markers.

EVIDENCE

GCKR is a common variant that nudges metabolism in two directions at once, so neither version is simply good or bad. In population studies, people who carry the C allele tend to have slightly lower triglycerides but slightly higher fasting glucose, while the T allele leans the opposite way. It's a small, common genetic leaning, not a prediction about any one person - and many other genes and everyday habits, like diet, sugar and refined-carb intake, alcohol, and regular movement, influence these markers far more.

SOURCES

GWAS rs780094

Triglyceride & LDL cholesterol tendency

GCKR

POPULATION STUDY

rs1260326 | GT C/C | DOSE 1/1 | REF/ALT T/C | STUDIED T (0 of 2) | GRCh38 +

INTERPRETATION

C/C genotype at GCKR. In population studies this version is associated with lower triglyceride levels (and, via the same variant, higher fasting glucose).

EVIDENCE

In population studies, people who carry the T allele at rs1260326 (GCKR P446L) tend to have somewhat higher triglyceride and LDL cholesterol levels, while the C allele leans the other way. The same C allele also leans toward slightly higher fasting glucose, so it is shown for general information. It's a small, common genetic leaning, not a prediction about any one person - diet, alcohol, refined-carb intake, and activity influence lipids far more.

SOURCES

GWAS rs1260326

Triglyceride & lipid levels tendency

APOC3

POPULATION STUDY

rs5128 | GT C/C | DOSE 1/1 | REF/ALT G/C | STUDIED G (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the C allele - a C/C genotype, with no copies of the G allele present. In population studies the G allele is linked to modestly higher triglycerides, so C/C sits at the lower end of this small leaning.

EVIDENCE

In population studies, people who carry the G allele in APOC3 tend to have slightly higher triglycerides. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, added sugar and alcohol intake, weight, and regular activity - influence triglycerides far more.

SOURCES

PubMed 42069741

Triglyceride & VLDL tendency

APOA1

POPULATION STUDY

rs964184 | GT C/C | DOSE 1/1 | REF/ALT G/C | STUDIED C (2 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the C allele - a C/C genotype. In population studies C/C is associated with a modestly lower triglyceride and VLDL measurement than G/G. This is an association, not a medical prediction.

EVIDENCE

In population studies, people who carry the C allele at this APOA1/APOA5-cluster marker tend to have slightly lower triglycerides and VLDL. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, sugar and refined-carb intake, alcohol, oily fish and exercise - influence blood-fat levels far more.

SOURCES

PubMed 23505323 | PubMed 27005778

Triglyceride level tendency

APOA5

POPULATION STUDY

rs2266788 | GT A/A | DOSE 1/1 | REF/ALT G/A | STUDIED A (2 of 2) | GRCh38 +

INTERPRETATION

Both copies are the A allele - an A/A genotype. In population studies the A allele is associated with a lower triglyceride reading, so A/A sits at the lower end for this measurement. This is an average shift across people; a lipid panel confirms the actual number.

EVIDENCE

In population studies, people who carry the A allele at this APOA5 site tend to have slightly lower triglycerides. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, refined-carbohydrate and alcohol intake, physical activity, and body weight - influence triglycerides far more.

SOURCES

PubMed 21386085

Triglyceride level tendency

LIPC

POPULATION STUDY

rs2070895 | GT G/A | DOSE 0/1 | REF/ALT G/A | STUDIED A (1 of 2) | GRCh38 +

INTERPRETATION

One A allele and one G allele - a heterozygous G/A genotype, one from each parent. In research cohorts the A allele is associated with a slightly higher triglyceride reading on average. This is a weak, relative population-level shift - a lipid panel is the real measure.

EVIDENCE

In population studies, people who carry the A allele at this LIPC site tend to have slightly higher triglycerides. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, refined-carbohydrate and alcohol intake, physical activity, and body weight - influence triglycerides far more.

SOURCES

PubMed 38116116

Triglyceride tendency

FADS1

POPULATION STUDY

rs174550 | GT C/C | DOSE 1/1 | REF/ALT T/C | STUDIED T (0 of 2) | GRCh38 +

INTERPRETATION

You have the C/C genotype at FADS1 - no copies of the triglyceride-lowering T allele, so people with C/C tend to sit toward the higher end of the triglyceride range.

EVIDENCE

In population studies, people who carry the T allele at FADS1 tend to have slightly lower triglycerides. FADS1 plays a role in processing dietary omega-3 and omega-6 fats, so this is a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, alcohol, physical activity, and body weight - influence triglyceride levels far more.

SOURCES

GWAS rs174550

Triglyceride tendency

TRIB1 region

POPULATION STUDY

rs2954029 | GT A/T | DOSE 0/1 | REF/ALT A/T | STUDIED T (1 of 2) | GRCh38 +

INTERPRETATION

One A allele and one T allele - a heterozygous A/T genotype, one from each parent. In population studies triglyceride levels tend to run slightly higher than for the T/T type, with each A copy adding a small leaning upward. This is a small population-level shift, not a medical prediction.

EVIDENCE

In population studies, people who carry the T allele in the TRIB1 region tend to have slightly lower triglycerides. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - sugar and refined-carb intake, alcohol, body weight, and regular movement - influence triglycerides far more.

SOURCES

PubMed 38116116 | PubMed 24097068

Triglyceride/HDL ratio tendency

LPL

POPULATION STUDY

rs268 | GT A/A | DOSE 0/0 | REF/ALT A/G | STUDIED G (0 of 2) | GRCh38 +

INTERPRETATION

A/A genotype - both copies carry the A allele, no copies of the G (N291S) allele. In population studies, people with A/A tend toward the lower (more favorable) end of the triglyceride/HDL ratio range. The per-copy effect is a small, relative population-level shift.

EVIDENCE

In population studies, people who carry the G allele at rs268 (LPL N291S) tend to have a higher triglyceride-to-HDL ratio - somewhat higher triglycerides and lower HDL cholesterol. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, alcohol, refined-carb intake, and physical activity - influence these lipids far more.

SOURCES

PubMed 38200128

Triglyceride levels tendency

APOA5

POPULATION STUDY

rs651821 | GT T/T | DOSE 1/1 | REF/ALT C/T | STUDIED C (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the T variant - a T/T genotype, with no copies of the triglyceride-raising C variant. In research cohorts this pattern sits toward the lower, more favorable end of the triglyceride range at this site.

EVIDENCE

In population studies, people who carry the minor C allele in APOA5 tend to have higher triglycerides, while the common T allele tracks with lower levels. It's a common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, added sugar and alcohol intake, weight, and regular activity - influence triglycerides far more.

SOURCES

PubMed 38116116

Triglyceride tendency

APOA5

POPULATION STUDY

rs662799 | GT A/A | DOSE 1/1 | REF/ALT G/A | STUDIED G (0 of 2) | GRCh38 +

INTERPRETATION

Both copies here carry the A allele - an A/A genotype, with no copies of the triglyceride-raising G allele. In population studies measured triglycerides at this site tend to sit toward the lower end of the range - a favorable position relative to G-carriers.

EVIDENCE

In population studies, people who carry the G allele at rs662799 (APOA5) tend to have slightly higher triglyceride levels, while the more common A allele leans the other way. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, alcohol, refined-carb intake, and physical activity - influence triglycerides far more.

SOURCES

PubMed 24023260 | PubMed 32335043 | PubMed 29212154

Triglyceride tendency LPL

POPULATION STUDY

rs328 | GT C/G | DOSE 0/1 | REF/ALT C/G | STUDIED G (1 of 2) | GRCh38 +

INTERPRETATION

You inherited one G variant and one C variant - a heterozygous C/G genotype, one allele from each parent. The single G copy (the LPL S447X form) is associated in research cohorts with modestly lower triglycerides relative to C/C - a small favorable shift.

EVIDENCE

In population studies, people who carry the G allele at LPL tend to have slightly lower triglycerides; this variant makes the lipoprotein lipase enzyme a little more active. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - diet, alcohol, physical activity, and body weight - influence triglyceride levels far more.

SOURCES

PubMed 35945198 PubMed 22171074

02.29 Vitamin B12 level tendency — 2/3 markers

Vitamin B12 level tendency FUT2

POPULATION STUDY

rs492602 | GT A/G | DOSE 0/1 | REF/ALT A/G | STUDIED G (1 of 2) | GRCh38 +

INTERPRETATION

You inherited one G variant and one A variant - a heterozygous A/G genotype, one allele from each parent. In research cohorts this places measured B12 partway above the A/A baseline, a small per-copy leaning higher from the G variant. Many other genetic and lifestyle factors also influence B12 status.

EVIDENCE

In population studies, each G allele at this FUT2 marker is associated with higher measured vitamin B12 levels, a small per-copy effect. This is a relative, population-level shift, not a medical prediction. This marker reflects FUT2 secretor status: the non-secretor version tends to show higher measured vitamin B12, and at this marker that is the G allele. The two FUT2 B12 markers tag one shared secretor signal, so read them together rather than as separate confirmations. This variant is reported for more than one trait.

SOURCES

PubMed 18776911

Vitamin B12 level tendency

TCN1

POPULATION STUDY

rs526934 | GT A/A | DOSE 1/1 | REF/ALT G/A | STUDIED G (0 of 2) | GRCh38 +

INTERPRETATION

You have the A/A genotype - no copies of the lower-B12 G allele. Across several populations the A allele tracks with somewhat higher circulating vitamin B12, so two copies sit at the favorable end of this small genetic tendency. It is only a modest nudge; your actual level still depends mostly on diet and absorption.

EVIDENCE

This variant sits in TCN1 (transcobalamin-1 / haptocorrin, a B12 carrier protein). Across several populations (including Chinese, Icelandic, Italian and US cohorts), the G allele tracks with somewhat lower circulating vitamin B12 and the A allele with somewhat higher. The effect is small, so on its own it does not meaningfully predict your individual level - your B12 status depends mostly on diet and absorption. REF=G (lower-B12 allele), ALT=A (higher-B12 allele).

SOURCES

GWAS rs526934

02.30 Vitamin D level tendency

3/4 markers

Vitamin D level tendency

CYP2R1

POPULATION STUDY

rs10741657 | GT A/G | DOSE 0/1 | REF/ALT A/G | STUDIED A (1 of 2) | GRCh38 +

INTERPRETATION

You inherited one A variant and one G variant - a heterozygous A/G genotype, one from each parent. In population studies the A allele is associated with higher vitamin D (generally favorable), so you get part of that shift relative to G/G - a small per-copy effect.

EVIDENCE

In population studies, the A allele in CYP2R1 is associated with higher vitamin D levels, while the G allele is associated with lower vitamin D. Because higher vitamin D is generally favorable, A-carriers come out ahead at this site. This is a population-level association, not a medical diagnosis, and a relative, population-level shift rather than your absolute level - many other factors also influence vitamin D. Strong evidence. When this applies: Higher vitamin D is generally favorable, so the A allele (which raises it) is treated as the helpful allele here.

SOURCES

PubMed 33382404 | PubMed 29325163 | PubMed 20541252

Vitamin D level tendency

GC

POPULATION STUDY

rs7041 | GT A/C | DOSE 0/1 | REF/ALT A/C | STUDIED C (1 of 2) | GRCh38 +

INTERPRETATION

One C variant and one A variant - a heterozygous A/C genotype, one allele from each parent. In population studies this sits between the A/A and C/C ends of the measured vitamin-D range, leaning higher with the C variant. A small leaning, not a personal prediction.

EVIDENCE

In population studies, each C allele at rs7041 (GC, vitamin D-binding protein) is associated with higher measured vitamin D. This is a small leaning, not a medical prediction. Strong evidence. Note: this is one of 3 correlated variants in/near GC reported for this trait. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal - not 3 independent confirmations. Many other genetic and lifestyle factors also influence vitamin D levels.

SOURCES

PubMed 36811574 | PubMed 36792583 | PubMed 24740207

Vitamin D levels tendency

GC

POPULATION STUDY

rs2282679 | GT T/T | DOSE 0/0 | REF/ALT T/G | STUDIED G (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the T variant - a T/T genotype, the combination linked in population studies to the higher end of vitamin D levels at this site. People with T/T tend to sit furthest from the low-vitamin-D (deficiency) end relative to G-carriers. The effect is modest; your actual level still depends mainly on sun and diet.

EVIDENCE

This variant in GC (the vitamin-D-binding protein gene) relates to how much vitamin D circulates in the blood. In population studies, each G allele is associated with lower vitamin D levels and the T allele with higher levels, so people carrying the T allele tend to sit further from the deficiency end. This is a relative, population-level shift, not a medical diagnosis; your actual vitamin D still depends mostly on sun exposure and intake. Note: this is one of several correlated variants in/near GC reported for this trait - neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal, not several independent confirmations.

SOURCES

PubMed 29325163 | PubMed 22673963 | PubMed 20418485

03

Sleep & energy

7 traits
15 findings

Chronotype, sleep quality, caffeine sensitivity and circadian tendencies.

03.01 Caffeine & sleep tendency (CYP1A2) — 1x

Caffeine & sleep tendency

CYP1A2

SUBSTANCE RESPONSE

rs762551 | GT A/A | DOSE 1/1 | REF/ALT C/A | STUDIED A (2 of 2) | GRCh38 +

INTERPRETATION

The A/A fast-metaboliser genotype: you clear caffeine quickly, so it is less likely to linger into the evening.

EVIDENCE

rs762551 sets how fast your liver clears caffeine (CYP1A2). Slow metabolisers (C-carriers) keep caffeine in the blood longer, so an afternoon coffee can still be active near bedtime; fast metabolisers (A/A) clear it quickly. How much that actually disturbs sleep varies a lot with dose, timing and personal sensitivity. When this applies: Applies only if you consume caffeine; if you avoid it this variant won't affect your sleep. The variant changes how fast you clear caffeine, not sleep on its own. Early-stage finding - read it as a small, common tendency, not a firm result.

SOURCES

PubMed 36833216 | PubMed 29514871

03.02 Caffeine jitteriness sensitivity tendency (ADORA2A) — 2/3 markers

Caffeine-induced anxiety tendency

ADORA2A

SUBSTANCE RESPONSE

rs5751876 | GT T/C | DOSE 0/1 | REF/ALT T/C | STUDIED T (1 of 2) | GRCh38 +

INTERPRETATION

You have the T/C genotype at rs5751876, carrying one copy of the C allele. In population studies, people with this genotype show a smaller tendency toward caffeine-related jitteriness or anxiety than those with the T/T genotype. Many other genetic and lifestyle factors also influence how a person responds to caffeine.

EVIDENCE

rs5751876 sits in ADORA2A, the gene for the adenosine A2A receptor, the same receptor caffeine blocks to make you feel alert. In population studies the T allele (the reference allele here) is linked to feeling more jittery after caffeine. In small studies, people with the T/T genotype reported more anxiety after a moderate caffeine dose (roughly 1 to 2 cups of coffee), while C-allele carriers (T/C and C/C) reported little or no increase. The effect is strongest in people who don't normally drink much caffeine, and it only matters if you consume caffeine. Note: this is one of a few correlated variants in/near ADORA2A reported for this trait; neighboring variants at one locus are usually inherited together, so treat them as largely a single shared signal, not several independent confirmations. This is an early, tentative signal, so read it as a small, common tendency rather than a certainty. When this applies: Applies only if you consume caffeine (coffee, tea, energy drinks, pre-workout); if you avoid caffeine, this variant has no effect on you. Early-stage finding - read it as a small, common tendency, not a firm result.

SOURCES

PubMed 18305461

Caffeine-sensitivity tendency

ADORA2A

SUBSTANCE RESPONSE

rs3761422 | GT T/C | DOSE 0/1 | REF/ALT T/C | STUDIED T (1 of 2) | GRCh38 +

INTERPRETATION

You have the T/C genotype (one T, one C). If you consume caffeine, your response is likely intermediate - between the more-sensitive T/T type and the calmer C/C type. If you do not use caffeine, this does not apply to you. Many other factors also shape caffeine sensitivity.

EVIDENCE

This variant in the ADORA2A adenosine-receptor gene affects how strongly caffeine tends to make people feel jittery or on-edge. The T allele (the reference form here) is linked to a stronger response to caffeine; the C allele is linked to a calmer one. In a controlled caffeine-challenge study, people with two T copies reported the largest jump in jitteriness after caffeine. This is about sensitivity to caffeine, not a medical condition, and only matters if you actually consume caffeine. No size of effect is reported in the source. Note: this is one of 3 correlated variants in/near ADORA2A reported for this trait; neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal - not 3 independent confirmations. Many other genetic and lifestyle factors also shape caffeine sensitivity. This variant is reported for more than one trait; the per-trait findings come from different studies and are not directly comparable. When this applies: Applies only if you consume caffeine (coffee, tea, energy drinks). If you avoid caffeine, this variant has no effect on you. Early-stage finding - read it as a small, common tendency, not a firm result.

SOURCES

PubMed 20520601

03.03 Caffeine-metabolism tendency (CYP1A2) 1x

Caffeine-metabolism tendency

CYP1A2

SUBSTANCE RESPONSE

rs762551 | GT A/A | DOSE 1/1 | REF/ALT C/A | STUDIED A (2 of 2) | GRCh38 +

INTERPRETATION

You have the A/A genotype at rs762551, associated with faster caffeine metabolism through CYP1A2. In population studies, fast metabolizers tend to break caffeine down quickly, so coffee's effects often wear off sooner.

EVIDENCE

rs762551 marks the CYP1A2 *1F variant, in the gene for the main liver enzyme that breaks down caffeine. The A allele (ALT) is the 'fast' allele: more A copies are associated with quicker caffeine clearance. In population studies, A/A people tend to be fast metabolisers (caffeine wears off sooner), C/C tend to be slow metabolisers (caffeine lingers, effects feel stronger and last longer), and C/A is in between. This shapes how long coffee keeps you alert or affects sleep, and it only matters if you consume caffeine; many other genetic and lifestyle factors also play a role. The day-to-day caffeine feel is the most reliable part, so read this as a common tendency rather than a fixed rule. When this applies: Applies only if you consume caffeine (coffee, tea, energy drinks); if you avoid caffeine, this variant has no practical effect on you. Early-stage finding - read it as a small, common tendency, not a firm result.

SOURCES

PubMed 29514871

03.04 Chronotype (morning/evening) tendency

3/15 markers

Chronotype (morning vs. evening preference) tendency

POPULATION STUDY

rs12140153 | GT G/G | DOSE 0/0 | REF/ALT G/T | STUDIED T (0 of 2) | GRCh38 +

INTERPRETATION

Both copies carry the G variant - a G/G genotype. In population studies people with G/G tend to sit toward the more morning-leaning end of the chronotype range, with each G copy contributing a small upward shift.

EVIDENCE

This is a common variant studied for chronotype - whether people lean toward being a morning or an evening type. In population studies each T allele is associated with a small shift toward the evening end of the range - a relative, population-level shift. The effect of one variant is small and informational; sleep timing is shaped by many genes plus light exposure, schedule and habits. This variant is reported for more than one trait; the per-trait effects come from different studies and scales and are not directly comparable.

SOURCES

PubMed 30696823

Chronotype (morningness) / daytime rest tendency

POPULATION STUDY

rs2653349 | GT G/G | DOSE 1/1 | REF/ALT A/G | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

No copies of the A allele, so this morningness leaning is at the lower end here. Limiting bright evening light helps an unhurried wind-down.

EVIDENCE

In population studies, people who carry the A allele tend to lean toward morningness and a slightly greater tendency for daytime rest. It's a small, common genetic leaning, not a prediction about any one person. Many genes and everyday habits - morning daylight exposure, consistent wake times, evening screen light, and meal timing - matter more.

SOURCES

Source

Chronotype (morningness/eveningness) tendency

POPULATION STUDY

rs11545787 | GT A/A | DOSE 1/1 | REF/ALT G/A | STUDIED G (0 of 2) | GRCh38 +

INTERPRETATION

No copies of the G allele here, so no nudge toward the morning-leaning tendency from this marker. A steady wake time each day helps anchor the body clock regardless.

EVIDENCE

In population studies, people who carry the G allele tend to lean a little more toward a morning-oriented body clock. It's a small, common genetic leaning, not a prediction about any one person. Many genes and everyday habits - consistent wake times, morning light exposure, and limiting late-evening screens - matter more.

SOURCES

PubMed 30696823 | Source

03.05 Coffee & tea consumption 6x

Coffee & tea consumption tendency

CYP1A1/CYP1A2 | POPULATION STUDY

rs2472297 | GT C/C | DOSE 0/0 | REF/ALT C/T | STUDIED T (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the C variant - a C/C genotype, with no copies of the T variant. In population studies, people with C/C tend to sit toward the lower end of the coffee & tea consumption range; the T variant (which tracks with the upper end) is absent here. The effect is a small per-copy leaning.

EVIDENCE

At this CYP1A1 marker, each T allele is associated with higher coffee & tea consumption in population studies, a small per-copy effect. This is a relative, population-level shift, not a direct medical prediction. Strong evidence. Note: this is one of several correlated variants in/near CYP1A1 reported for this trait - neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal, not several independent confirmations.

SOURCES

PubMed 32193382 | PubMed 35653391 | PubMed 21357676

Coffee consumption

POPULATION STUDY

rs2470893 | GT C/T | DOSE 0/1 | REF/ALT C/T | STUDIED T (1 of 2) | GRCh38 +

INTERPRETATION

One copy of the T allele, a small middling leaning toward higher coffee consumption.

EVIDENCE

In population studies, the T allele at this CYP1A1/CYP1A2 marker is associated with higher coffee consumption, each copy a small nudge. A common behavioral leaning, not a prediction; how much someone drinks is shaped far more by habit and culture. This is one of several correlated variants in/near CYP1A1/CYP1A2 (15q24) reported for coffee consumption. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal, not independent confirmations.

SOURCES

PubMed 21876539

Coffee consumption tendency

AHR

POPULATION STUDY

rs4410790 | GT C/C | DOSE 1/1 | REF/ALT T/C | STUDIED C (2 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the C variant - a C/C genotype. People with C/C tend to sit toward the higher end of the coffee-consumption range, with each C copy contributing a small upward shift - a relative, population-level shift.

EVIDENCE

In population studies, each C allele at this AHR marker is associated with higher coffee consumption, a small per-copy effect. This is a relative, population-level behavioral shift, not a medical prediction. Strong evidence. This is one of two correlated variants in/near AHR (7p) reported for coffee consumption. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal, not independent confirmations.

SOURCES

PubMed 25288136

Coffee-consumption tendency

AHR region

POPULATION STUDY

rs6968865 | GT T/T | DOSE 1/1 | REF/ALT A/T | STUDIED T (2 of 2) | GRCh38 +

INTERPRETATION

Both copies here carry the T variant - a T/T genotype. In population studies people with T/T tend to sit toward the higher end of the coffee-consumption range, with each T copy adding a small upward shift. A relative, population-level leaning.

EVIDENCE

In population studies, each T allele at rs6968865 (AHR region) is associated with slightly higher self-reported coffee consumption. This is a small behavioral leaning, not a medical prediction. Moderate evidence. Note: this is one of 2 correlated variants in/near AHR reported for this trait. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal - not 2 independent confirmations. Many other genetic and lifestyle factors also influence how much coffee someone drinks.

SOURCES

PubMed 21357676

Habitual coffee consumption

POPULATION STUDY

rs17685 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

No copies of the A allele, so on average a slightly lower leaning toward heavy coffee drinking in population studies. This is a faint tendency only; a regular sleep routine keeps daytime energy even on its own.

EVIDENCE

In population studies, people who carry the A allele tend to drink more coffee on average. It's a small, common genetic leaning, not a prediction about any one person. Many genes and everyday habits - sleep quality, daily routine, stress levels, and the timing of the last cup - matter more.

SOURCES

PubMed 25288136 | Source

Habitual coffee consumption

POPULATION STUDY

rs2472304 | GT A/A | DOSE 1/1 | REF/ALT G/A | STUDIED A (2 of 2) | GRCh38 +

INTERPRETATION

Two copies of the A allele, the strongest leaning in this exploratory finding toward higher habitual coffee consumption. It's only a faint tendency; shifting the last cup earlier in the day can protect evening wind-down.

EVIDENCE

In population studies, people who carry the A allele tend to drink more coffee on average. It's a small, common genetic leaning, not a prediction about any one person. This is an exploratory, low-confidence finding from a single study. Many genes and everyday habits - sleep timing, caffeine sensitivity, daily routine, and how late in the day coffee is consumed - matter more. This is one of several correlated variants in/near CYP1A1/CYP1A2 (15q24) reported for coffee consumption. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal, not independent confirmations.

SOURCES

PubMed 21490707

03.06 Daytime rest tendency (PNOC) 1x

Daytime rest / daytime-nap tendency

POPULATION STUDY

rs351776 | GT A/A | DOSE 0/0 | REF/ALT A/C | STUDIED C (0 of 2) | GRCh38 +

INTERPRETATION

No copies of the C allele, so the leaning toward daytime rest is at the lower end here. A steady, regular bedtime helps keep daytime energy even.

EVIDENCE

In population studies, people who carry the C allele tend to show a somewhat greater leaning toward daytime rest and napping. It's a small, common genetic leaning, not a prediction about any one person. Many genes and everyday habits - nighttime sleep length, consistent sleep and wake times, daylight exposure, and afternoon caffeine - matter more.

SOURCES

Source

03.07 Sleep-quality tendency (VEGFA) 1x

Sleep-quality tendency VEGFA

POPULATION STUDY

rs833061 | GT C/C | DOSE 0/0 | REF/ALT C/T | STUDIED T (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the C allele - a C/C genotype, with no copies of the T allele. In population studies the T allele carries a very small association with lighter sleep, so C/C sits at the calm end of this tiny range. Informational, not a concern.

EVIDENCE

At VEGFA rs833061, the T allele shows a very small association with lighter, more restless sleep in population studies. The effect is tiny - a weak population-level leaning that on its own does not meaningfully predict your sleep. Moderate evidence. Many other genes and everyday habits influence sleep far more.

SOURCES

PubMed 35835914

04

Brain & mind

7 traits
14 findings

Focus, memory, mood, cognitive aging and reward-seeking tendencies.

04.01 APOE status (Alzheimer's research gene)

1/2 markers

APOE e2 variant

APOE

POPULATION STUDY

rs7412 | GT C/C | DOSE 0/0 | REF/ALT C/T | STUDIED T (0 of 2) | GRCh38 +

INTERPRETATION

No epsilon 2 copies at rs7412 (C/C genotype): the common situation. This entry describes a protection you do not carry, which is itself the norm; it says nothing adverse.

EVIDENCE

APOE is the best-studied gene in late-life memory research, and its epsilon 2 form (tagged by the T allele here) is the protective end: epsilon 2/epsilon 3 carriers show roughly 40 percent lower odds of late-onset Alzheimer's disease than the epsilon 3/epsilon 3 majority, and two copies looked more protective still in a 5,000-brain autopsy study. A favorable finding, not a guarantee; the usual brain-healthy habits still earn their keep. For health decisions, talk to a professional - this is general wellness information.

SOURCES

GWAS rs7412 | PubMed 9343467 | PubMed 32015339

04.02 Cannabis (THC) response tendency (FAAH)

1x

Cannabis (THC) response tendency FAAH

SUBSTANCE RESPONSE

rs324420 | GT C/C | DOSE 0/0 | REF/ALT C/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

You have the C/C genotype at rs324420 (two ref C's). In the cited study of cannabis users, people without the A allele tended toward a slightly higher chance of heavy, dependence-style THC use than A/A carriers, because the A allele is the one linked to easier-going endocannabinoid signaling. This only matters if you choose to use cannabis, the evidence is mixed across studies, and many other genetic and lifestyle factors shape how cannabis affects you. Treat it as a small nudge, not a verdict.

EVIDENCE

rs324420 (Pro129Thr) is a missense variant in FAAH, the enzyme that clears the body's own cannabis-like molecules (endocannabinoids). REF=C, ALT=A; the A allele lowers FAAH activity and raises endocannabinoid tone. In the cited study of cannabis users, A/A carriers were less likely to show heavy, dependence-style THC use, while carriers of the C allele tended toward a higher chance of it. The literature is mixed (some studies instead link the A allele to heavier drug or alcohol use), and the source reports no size of effect, so treat this as a weak population-level tendency that is only relevant if you use cannabis. Many other genetic and lifestyle factors also shape cannabis use patterns. This variant is reported for more than one trait; the per-trait findings come from different studies and are not directly comparable. When this applies: Applies only if you use cannabis/THC; if you don't, this variant has no effect on you. Early-stage finding - read it as a small, common tendency, not a firm result.

SOURCES

PubMed 17290447

04.03 Cocaine-response variant (CHRNA5) 1x

Cocaine-response variant CHRNA5

CATALOGED VARIANT

rs684513 | GT C/C | DOSE 0/0 | REF/ALT C/G | STUDIED per genotype | GRCh38 +

INTERPRETATION

You have the C/C genotype at this CHRNA5 position. This variant has been linked to cocaine use in population studies, but which allele points in which direction isn't established on a comparable basis here - the reported risk allele isn't pinned down for this trait - so this is shown for information only, with no higher-or-lower direction implied. It would only be relevant for people who use cocaine, and many other genetic and lifestyle factors also matter.

EVIDENCE

This is a common variant in the CHRNA5 nicotinic-receptor gene cluster (forward strand REF=C, ALT=G). It has been associated with cocaine use in population studies, but which allele drives the tendency is not established on the forward strand from a verifiable primary source - the cited study reports an association without a confirmed risk-allele letter, and the GWAS Catalog assigns this variant a risk allele only for unrelated lung-function traits, not cocaine. So no direction is implied here and it is shown for information only. It is one of several correlated variants in/near CHRNA5 (linkage disequilibrium), so treat them as one shared signal. It would only be relevant for people who use cocaine. Many other genetic and lifestyle factors also influence consumption patterns; this is a behavioral tendency, not a diagnosis. When this applies: Applies only if you use cocaine - it relates to heavier-use patterns. If you do not use cocaine, this has no effect on you.

SOURCES

PubMed 20485328

04.04 Memory & learning style (BDNF Val66Met) 1x

Memory & learning style BDNF

POPULATION STUDY

rs6265 | GT C/C | DOSE 0/0 | REF/ALT C/T | STUDIED T (0 of 2) | GRCh38 +

INTERPRETATION

Carries two C forms (no T). In population studies this is the more common pattern linked to steadier activity-driven support for memory and learning circuits. A small, average leaning only, not a prediction for any individual.

EVIDENCE

In population studies, the C form at this spot is linked to somewhat steadier activity-driven support for memory circuits, while the T form leans toward slightly less of that support. It is a small, common tendency, not a prediction about any one person, and many genes and everyday habits like sleep, exercise and practice matter far more. The signal is exploratory: it comes mainly from candidate-gene and brain-imaging work, and larger follow-up studies have given mixed results.

SOURCES

PubMed 12553913

04.05 **Nicotine-response tendency** 8/9 markers

Nicotine response tendency

CHRNA3 SUBSTANCE RESPONSE

rs3743078 | GT G/G | DOSE 1/1 | REF/ALT C/G | STUDIED G (2 of 2) | GRCh38 +

INTERPRETATION

You have the G/G genotype at rs3743078 in CHRNA3. You carry two copies of the form of this variant that has been associated with stronger nicotine cravings and heavier smoking, so this nicotinic-receptor signal may nudge you somewhat more firmly in that direction. This is only relevant if you use nicotine. Many other genetic and lifestyle factors also shape smoking behavior.

EVIDENCE

rs3743078 sits in CHRNA3, a nicotinic acetylcholine-receptor gene in the CHRNA5/A3/B4 cluster that population studies have repeatedly linked to how heavily people smoke. The G form of this variant has been associated with stronger nicotine cravings and heavier smoking, while the C form is not. The effect for this single variant is modest and it is only relevant if you use nicotine. Note: this is one of 3 correlated variants in/near CHRNA3 reported for this trait; neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal - not 3 independent confirmations. Many other genetic and lifestyle factors also shape smoking behavior. When this applies: Only relevant if you smoke or use nicotine; it has no effect on people who never use nicotine. The reported link is weak and studies disagree on which allele matters, so treat it as informational only. Early-stage finding - read it as a small, common tendency, not a firm result.

SOURCES

PubMed 20886544

Nicotine response tendency

CHRNA3/5

POPULATION STUDY

rs8034191 | GT T/T | DOSE 0/0 | REF/ALT T/C | STUDIED C (0 of 2) | GRCh38 +

INTERPRETATION

Both copies here carry the T allele - a T/T genotype, with no copies of the C allele. Among people who smoke, each C copy is weakly associated with a slightly higher number of cigarettes per day; with no C copies this sits at the lower end. The effect is small and on its own does not meaningfully predict individual behavior; it is a small leaning among smokers, not a medical condition.

EVIDENCE

rs8034191 sits in the chromosome-15 CHRNA3/CHRNA5 nicotine-receptor cluster. In population studies each C allele is associated with a slightly higher number of cigarettes per day among people who use tobacco (a small per-copy effect - a relative, population-level shift, not your absolute chance). This is a population-level leaning, not a medical prediction, and it only manifests in people who use tobacco; it has no consequence for people who have never smoked. Limited evidence. Many other genetic and lifestyle factors also influence this trait. This variant is reported for more than one trait; the per-trait findings come from different studies and are not directly comparable. When this applies: The cigarettes-per-day effect of the C allele only applies to people who use tobacco; it has no effect for never-smokers.

SOURCES

PubMed 33144568

Nicotine-response tendency CHRNA3

CATALOGED VARIANT

rs578776 | GT G/A | DOSE 0/1 | REF/ALT G/A | STUDIED G (1 of 2) | GRCh38 +

INTERPRETATION

You have the G/A genotype (one G, one A). In population research the nicotine-response tendency for this group is intermediate - the single A copy is linked to partial protection compared with G/G. This is relevant only to people who use tobacco.

EVIDENCE

This variant is in CHRNA3, part of the nicotinic-receptor gene cluster strongly tied to smoking behavior. The G allele (the reference, major form here) is associated with stronger nicotine response; the A allele (the minor form) is associated with the opposite, protective direction. In population research, smokers carrying two A copies showed a strong protective association - a relative, population-level shift, not an individual outcome. It describes how strongly the body tends to respond to nicotine once a person smokes, so it is relevant only to people who use tobacco - it does not make anyone start smoking. Many other genetic and lifestyle factors also influence smoking behavior. Note: this is one of 3 correlated variants in/near CHRNA3 reported for this trait; treat them as largely a single shared signal (linkage disequilibrium) - not 3 independent confirmations. This variant is reported for more than one trait; the per-trait findings come from different studies and are not directly comparable. When this applies: Applies only if you smoke or use nicotine; in people who have never used tobacco it has no effect.

SOURCES

PubMed 20886544 | PubMed 18618000

Nicotine-response tendency CHRNA3

POPULATION STUDY

rs1051730 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

Both copies here carry the G allele - a G/G genotype, with no copies of the A allele. Among people who smoke, each A copy is associated with a modestly higher number of cigarettes per day; with no A copies this measurement sits at the lower end. This describes smoking intensity once a person smokes, not whether they start, and it is a small nudge rather than a medical condition.

EVIDENCE

In population studies, each A allele is associated with smoking more cigarettes per day, with a small per-copy effect (CHRNA3). This applies only to people who smoke - it tracks smoking intensity once a person smokes, not whether they start. The evidence is strong and replicated. Note: this is one of a few correlated variants in/near CHRNA3 reported for this trait. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal - not several independent confirmations. Many other genetic and lifestyle factors also influence smoking behavior. When this applies: This effect on how much someone smokes only applies to people who use tobacco; for never-smokers it has no effect.

SOURCES

PubMed 18385739 | PubMed 25891233 | PubMed 19628476

Nicotine-response tendency CHRNA5

POPULATION STUDY

rs16969968 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

You have the G/G genotype - no copies of the A allele at this CHRNA5 marker. Among people who smoke, each A copy is associated with a modestly higher nicotine-response / smoking-intensity measurement; with no A copies this sits at the lower end. This is a population-level nudge among smokers, not a medical condition; if you don't smoke it has no effect.

EVIDENCE

rs16969968 (CHRNA5 D398N) is a variant in a nicotinic-receptor gene. Among people who use tobacco, each A allele is associated with a higher measure of nicotine response and smoking intensity, with a small per-copy effect - a relative, population-level shift, not your absolute chance. This is an association among smokers, not a medical prediction; in people who never smoke the variant has no measurable effect (a gene-by-smoking interaction). Note: this is one of a couple of correlated variants in/near CHRNA5 reported here, and neighboring variants are usually inherited together (linkage disequilibrium), so they act as largely one shared signal rather than independent confirmations. Many other genetic and lifestyle factors also influence tobacco use. When this applies: Relates to how much someone smokes and only applies to people who use tobacco - a gene-by-smoking interaction with no effect in never-smokers.

SOURCES

PubMed 31294817 | PubMed 24733007 | PubMed 18618000

Nicotine-response variant CHRNA5

CATALOGED VARIANT

rs684513 | GT C/C | DOSE 0/0 | REF/ALT C/G | STUDIED per genotype | GRCh38 +

INTERPRETATION

You have the C/C genotype. Which allele drives heavier nicotine use isn't established here - this variant is a marker in linkage and population studies disagree - so this is shown for information only, with no higher-or-lower direction implied. It would only be relevant if you use tobacco, which carries major health risks for everyone.

EVIDENCE

rs684513 is a common variant in the CHRNA5 nicotinic-receptor gene cluster (forward-strand REF=C, ALT=G) on chromosome 15q25. It has been studied as a marker near the well-known nicotine signal, but for nicotine-use quantity specifically the direction is not settled - it is a tag in linkage rather than a proven causal change, and population studies disagree. Which allele drives heavier nicotine use isn't established here, so this is shown for information only, with no higher-or-lower direction implied. It would only be relevant if you use tobacco - and tobacco carries major health risks for everyone regardless of genotype. Note: this is one of several correlated variants in/near CHRNA5 inherited together (linkage disequilibrium); treat them as one shared signal, not independent confirmations. Many other genetic and lifestyle factors also influence tobacco-use patterns. When this applies: Applies only if you smoke or use tobacco - it relates to how strongly the body responds to nicotine. If you do not use tobacco, this has no effect on you.

SOURCES

PubMed 20886544

Nicotine-response variant DRD1

CATALOGED VARIANT

rs4532 | GT C/T | DOSE 0/1 | REF/ALT C/T | STUDIED T (1 of 2) | GRCh38 +

INTERPRETATION

Among people who use nicotine, the C/T genotype sits at an intermediate population-level tendency. The evidence is weak, so this is only a faint signal; how much someone uses and their environment matter far more.

EVIDENCE

This is a variant in DRD1, the gene for the dopamine D1 receptor, part of the brain's reward system (here REF=C, ALT=T; on the forward strand of GRCh38 this site is C/T, the same variant older reports describe on the opposite strand). In population studies of smokers, this position has been linked to nicotine consumption patterns, with the T allele reported as the associated allele, though the reports are limited and partly conflicting. This is a weak behavioral tendency that matters only for people who use nicotine, and no size of effect is established. How much someone uses nicotine and their environment shape this far more than one variant. This variant is reported for more than one trait; the per-trait findings come from different studies and are not directly comparable. When this applies: Applies only if you smoke or use nicotine - it relates to how strongly the body responds to nicotine; if you do not use nicotine it has no effect on you.

CLINVAR REVIEW

1 of 4 review stars - criteria provided, single submitter. Classified: Benign. This says how much expert review stands behind the classification, not how strong the effect is.

SOURCES

ClinVar | PubMed 18092181

Nicotine-response variant DRD1

CATALOGED VARIANT

rs686 | GT G/A | DOSE 0/1 | REF/ALT G/A | STUDIED A (1 of 2) | GRCh38 +

INTERPRETATION

With the G/A genotype, population studies place this genotype between the two homozygous groups for the tendency toward a nicotine-use habit pattern - stronger than G/G, weaker than A/A. This applies only to people who use nicotine, and many other genetic and lifestyle factors also shape how readily such a pattern forms.

EVIDENCE

rs686 sits in the 3' untranslated region of the DRD1 gene, which makes the dopamine D1 receptor (part of the brain's reward system). Studies have reported that, among people who use nicotine, the A allele is associated with a stronger tendency toward a nicotine-use habit pattern, while the G allele is associated with a weaker one. The effect is modest and no effect size is given. This matters only for people who use nicotine, and it's one of many genetic and environmental factors that shape how readily such a habit pattern forms. This is one of 2 correlated variants in or near DRD1 reported for this trait; neighboring variants are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal, not 2 independent confirmations. When this applies: Applies only if you smoke or use nicotine - it relates to how strongly the body responds to nicotine; if you don't use nicotine, it has no effect on you.

SOURCES

PubMed 18092181

04.06 Stress & working-memory style (COMT) 1x

Stress-processing & working-memory style worrier/warrior

POPULATION STUDY

rs4680 | GT G/A | DOSE 0/1 | REF/ALT G/A | STUDIED A (1 of 2) | GRCh38 +

INTERPRETATION

One G and one A form. In population studies this sits in between the two styles, with traits shading gently in either direction. A small, common tendency only - not a prediction for any individual, and one this exploratory finding (mixed replication) can't pin down.

EVIDENCE

In population studies, people who carry the A form at this spot tend to lean toward the "worrier" style - a touch more sensitive to stress alongside slightly steadier working memory at rest - while those without it lean toward the steadier-under-pressure "warrior" style. This is a small, common, two-sided leaning, not a prediction about any one person, and many other genes and everyday habits matter far more. Treat it as exploratory: this is candidate-gene work with mixed replication, so the effect is small and not consistently seen.

SOURCES

PubMed 11381111 | PubMed 18339359

04.07 Memory performance & cognitive aging

1/4 markers

Memory & cognitive-decline tendency

APOE

POPULATION STUDY

rs769449 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED G (2 of 2) | GRCh38 +

INTERPRETATION

G/G genotype, the combination most associated with the favorable direction at this site. In population studies each G copy is linked to a small upward shift in measured memory performance during cognitive aging relative to the A/A baseline. This is a tendency, not a prediction; many other factors also influence memory.

EVIDENCE

rs769449 sits in the APOE gene region. In population studies the G allele is associated with a small shift toward better measured memory performance during cognitive aging; equivalently, the A allele tracks a slightly lower measure. This is an association, not a medical prediction, and it is a relative population-level shift rather than your individual outcome. Many other genetic and lifestyle factors also influence memory and cognitive aging. Note: this is one of a few correlated variants in/near APOE reported for this association, usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal rather than independent confirmations. This variant is reported for more than one trait, and the findings come from different studies and are not directly comparable.

SOURCES

PubMed 28800603

05

Health & longevity

18 traits
29 findings

Disease risks, cardiometabolic profile, hormones and longevity markers.

05.01 Bone density tendency

4/5 markers

Bone density tendency

TNFSF11/RANKL region

POPULATION STUDY

rs9594738 | GT C/T | DOSE 0/1 | REF/ALT C/T | STUDIED T (1 of 2) | GRCh38 +

INTERPRETATION

You carry one T allele and one C allele - a heterozygous C/T genotype, one from each parent. In population studies the T allele is associated with modestly lower bone mineral density than C/C. This is a small population-level leaning, not an individual diagnosis.

EVIDENCE

In population studies, people who carry the T allele at this variant tend to have slightly lower bone density, while the C allele leans toward higher bone density. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - weight-bearing exercise, dietary calcium and protein, and vitamin D - influence bone strength far more. This is one of two correlated variants in/near TNFSF11 (RANKL) at 13q14 reported for bone mineral density. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal, not independent confirmations.

SOURCES

PubMed 29304378 | PubMed 30598549 | PubMed 31015401

Bone density tendency

TNFSF11/RANKL region

POPULATION STUDY

rs9533090 | GT C/T | DOSE 0/1 | REF/ALT C/T | STUDIED T (1 of 2) | GRCh38 +

INTERPRETATION

You carry one T allele and one C baseline allele - a heterozygous C/T genotype, one from each parent. In population studies bone mineral density tends to be modestly lower than for people with the C/C baseline. This is a relative, population-level shift, not your absolute outcome.

EVIDENCE

In population studies, people who carry the T allele at this variant tend to have slightly lower bone density, while the C allele is the more common baseline. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - weight-bearing exercise, dietary calcium and protein, and vitamin D - influence bone strength far more. This is one of two correlated variants in/near TNFSF11 (RANKL) at 13q14 reported for bone mineral density. Neighboring variants at one locus are usually inherited together (linkage disequilibrium), so treat them as largely a single shared signal, not independent confirmations.

SOURCES

PubMed 22504420

Bone density tendency

LRP5

POPULATION STUDY

rs3736228 | GT C/C | DOSE 0/0 | REF/ALT C/T | STUDIED T (0 of 2) | GRCh38 +

INTERPRETATION

The C/C genotype carries two copies of the C baseline allele and no T copies. In population data this sits at the favorable end of the bone-mineral-density range relative to T-carriers - a small, relative shift, not a prediction about your bones.

EVIDENCE

In population studies, people who carry the T allele in LRP5 tend to have slightly lower bone density, while the C allele is the more common baseline. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - weight-bearing exercise, dietary calcium and protein, vitamin D, and hormones - influence bone strength far more.

SOURCES

PubMed 22504420 PubMed 30158200

Bone mineral density tendency

ESR1

POPULATION STUDY

rs2234693 | GT T/T | DOSE 0/0 | REF/ALT T/C | STUDIED C (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the T variant - a T/T genotype, with no copies of the C variant present. In population studies this genotype sits toward the lower end for bone mineral density relative to C-carriers; each C copy is linked to a small upward shift.

EVIDENCE

In population studies, people who carry the C allele at this ESR1 marker tend to have slightly higher bone mineral density. It's a small, common genetic leaning, not a prediction about any one person. Many other genes and everyday habits - weight-bearing exercise, calcium and vitamin D intake, and not smoking - influence bone density far more.

SOURCES

PubMed 30048462

05.02 Type 2 diabetes (TCF7L2) 1x

Type 2 diabetes-associated variant

TCF7L2

POPULATION STUDY

rs7903146 | GT C/T | DOSE 0/1 | REF/ALT C/T | STUDIED T (1 of 2) | GRCh38 +

INTERPRETATION

One T copy at rs7903146 (C/T genotype): in population studies type 2 diabetes affects roughly 13 in 100 people with this genotype versus 10 in 100 without the T variant. Structured diet and activity programs cut progression to diabetes roughly in half in prevention trials, so this lean is very answerable.

EVIDENCE

TCF7L2 helps insulin-producing cells respond to rising blood sugar, and its T variant is the strongest common type 2 diabetes signal known. Roughly 10 in 100 adults live with type 2 diabetes; people with one T copy sit nearer 13 in 100, and people with two T copies nearer 18 in 100 (OR 1.39 per copy). Weight, diet, and activity move this number more than the gene does: in prevention trials, structured lifestyle change cut progression to diabetes roughly in half, and it worked in T carriers. For health decisions, talk to a professional - this is general wellness information.

SOURCES

PubMed 38374256 | PubMed 32541925 | PubMed 30297969 | PubMed 11832527 | PubMed 16855264

05.03 APOC3 centenarian variant 1x

Centenarian lipid profile

APOC3 -641

POPULATION STUDY

rs2542052 | GT A/C | DOSE 0/1 | REF/ALT A/C | STUDIED A (1 of 2) | GRCh38 +

INTERPRETATION

One A copy at rs2542052 (A/C genotype): partway between the homozygous groups; the centenarian enrichment was clearest for two copies. Typical with a mild favorable lean.

EVIDENCE

APOC3 slows the clearance of triglyceride-rich particles, and less of it is better for the heart. The A version of this promoter variant dials APOC3 down: in studies of Ashkenazi Jewish centenarians and their children, people with two A copies were clearly overrepresented and showed larger lipid particles, better insulin sensitivity, and less hypertension. Roughly 1 in 6 people of European descent carries two A copies. Candidate-level evidence from one research group's centenarian families; broader replication is limited, so hold it lightly.

SOURCES

PubMed 16602826

05.04 C-reactive protein (inflammation marker) tendency

1/2 markers

C-reactive protein (inflammation marker) tendency

HNF4A

POPULATION STUDY

rs1800961 | GT C/C | DOSE 0/0 | REF/ALT C/T | STUDIED T (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position are the C allele, a C/C genotype, with no T alleles. The T allele is the one weakly linked to a lower CRP reading, so on average C/C sits at the upper end for this inflammation measurement. The per-copy shift is small (beta ~ 0.10) and reflects a population-level average, a blood test is the actual measure.

EVIDENCE

In population studies, each T allele at rs1800961 (HNF4A) is associated with a modestly lower C-reactive protein (CRP) measurement (about 0.10 per copy on the study scale). CRP is a blood inflammation marker, so this is a population-level shift, not a medical prediction. A blood test is the real measure. Strong evidence, 11 publications. This variant is reported for more than one trait; the per-trait effect sizes come from different studies and scales and are not directly comparable. For health decisions, talk to a professional - this is general wellness information.

SOURCES

PubMed 30388399 PubMed 34594039

05.05 CETP and longevity

1x

Exceptional longevity lipid profile

CETP I405V

POPULATION STUDY

rs5882 | GT G/A | DOSE 0/1 | REF/ALT G/A | STUDIED G (1 of 2) | GRCh38 +

INTERPRETATION

One V405 copy at rs5882 (G/A genotype): between the two homozygous groups; the centenarian findings concentrated in people with two V copies, so this reads as typical with a mild lean.

EVIDENCE

CETP shuffles cholesterol between particles; the V405 version (G allele here) runs at lower activity, which leaves larger, buoyant lipoprotein particles. In a study of Ashkenazi Jewish centenarians, people with two V copies were markedly overrepresented among those reaching 95 to 105, and a later study tied the same genotype to slower memory decline. About 1 in 9 people of European descent carries two V copies. Candidate-level evidence: the double-V genotype was nearly three times as common among centenarians as in controls, but genome-scale studies have not confirmed a longevity effect, so hold it lightly.

SOURCES

PubMed 14559957 | PubMed 20068209

05.06 Coronary artery disease (Lp(a))

1/2 markers

Coronary artery disease-associated variant

LPA

POPULATION STUDY

rs10455872 | GT A/A | DOSE 0/0 | REF/ALT A/G | STUDIED G (0 of 2) | GRCh38 +

INTERPRETATION

No copies of this Lp(a)-raising variant at rs10455872 (A/A genotype), like about 6 in 7 people of European descent: coronary odds from this marker sit at baseline. A one-time Lp(a) test remains reasonable if early heart disease runs in your family, since other variants can raise it.

EVIDENCE

Lipoprotein(a) is a cholesterol-like particle that statins barely touch and lifestyle does not move; its level is set almost entirely by genes. Each G copy of this LPA variant is associated with about 1.3 times the odds of coronary artery disease and heart attack versus the no-copy group (about 6 in 7 people of European descent carry none). The step that converts this into something useful is a one-time Lp(a) blood test: guidelines now recommend measuring it once in adulthood, and a high result brings prevention forward by years. For health decisions, talk to a professional - this is general wellness information.

SOURCES

PubMed 36474045 | PubMed 38072966 | PubMed 28714975 | PubMed 36036785

05.07 Heart valve narrowing (aortic stenosis, Lp(a))

1x

Aortic valve stenosis-associated variant

LPA

POPULATION STUDY

rs10455872 | GT A/A | DOSE 0/0 | REF/ALT A/G | STUDIED G (0 of 2) | GRCh38 +

INTERPRETATION

No copies of this variant at rs10455872 (A/A genotype): the aortic valve signal from this marker sits at baseline.

EVIDENCE

The same LPA variant that raises lipoprotein(a) also shows up in the aortic valve: each G copy is associated with about 1.45 times the odds of aortic valve stenosis, a slow stiffening of the heart's outflow valve that typically surfaces after 65 in a small minority of people. There is no pill for Lp(a) yet, but knowing it is high means valve-relevant symptoms (breathlessness on exertion, fainting) get taken seriously and echoed early rather than dismissed. For health decisions, talk to a professional - this is general wellness information.

SOURCES

PubMed 41419685 | PubMed 37038246

05.08 Iron overload (hereditary hemochromatosis) 1x

Hereditary hemochromatosis

HFE C282Y

CATALOGED VARIANT

rs1800562 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

No C282Y copies at rs1800562 (G/G genotype): typical iron regulation from this gene. Nothing to act on here.

EVIDENCE

HFE controls how much iron the gut absorbs; with two changed C282Y copies, iron can build up slowly over decades (hereditary hemochromatosis). In UK Biobank, 22 in 100 men and 10 in 100 women with two copies had a hemochromatosis diagnosis by the end of a study that followed people aged 40 to 70, versus fewer than 1 in 100 people without the variant. One copy alone almost never causes iron overload. Iron status is easy to check with two blood tests (ferritin and transferrin saturation), and early treatment, essentially donating blood on a schedule, prevents the complications. For health decisions, talk to a professional - this is general wellness information.

CLINVAR REVIEW

1 of 4 review stars - criteria provided, conflicting classifications. Classified: Conflicting classifications of pathogenicity; other; risk factor. This says how much expert review stands behind the classification, not how strong the effect is.

SOURCES

ClinVar | PubMed 30651232

05.09 LDL & heart protection (PCSK9 R46L)

1x

Lower LDL cholesterol & coronary-artery-associated variant

PCSK9 | POPULATION STUDY

rs11591147 | GT G/G | DOSE 0/0 | REF/ALT G/T | STUDIED T (0 of 2) | GRCh38 +

INTERPRETATION

No R46L copies at rs11591147 (G/G genotype): typical PCSK9 activity, like about 29 in 30 people. Nothing lost; LDL is highly manageable by other means.

EVIDENCE

Your liver clears LDL cholesterol partly under the control of PCSK9; the R46L variant dials PCSK9 down, which keeps LDL roughly 15 mg/dL lower for life and is associated with about 20 to 30 percent lower odds of coronary artery disease (an effect medicine now imitates with PCSK9-inhibitor drugs). About 1 in 30 people of European descent carries it. A tailwind rather than a shield: normal heart care still applies. For health decisions, talk to a professional - this is general wellness information.

SOURCES

PubMed 29748315 | PubMed 33339817 | GWAS rs11591147

05.10 Menopause-timing tendency

6/8 markers

Age at menopause tendency

TMEM150B | POPULATION STUDY

rs11668344 | GT A/G | DOSE 0/1 | REF/ALT A/G | STUDIED G (1 of 2) | GRCh38 +

INTERPRETATION

One A and one G variant - a heterozygous A/G genotype, one from each parent. This places the trait in the middle of the age-at-menopause range, between the lower-end G/G and the higher-end A/A genotypes.

EVIDENCE

In population studies each G allele at this variant is associated with a slightly lower age at natural menopause, while the A allele tends toward a slightly higher age. This is an association, not a medical prediction - a small population-level shift, and many other genetic and lifestyle factors also influence menopause timing.

SOURCES

PubMed 34349265

Age at menopause tendency

UIMC1

POPULATION STUDY

rs365132 | GT G/G | DOSE 0/0 | REF/ALT G/T | STUDIED T (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the G variant - a G/G genotype, with no copies of the T variant. In population studies people with G/G tend to sit toward the lower end of the age-at-menopause range; the T variant, which would shift the trait upward, is absent here. A relative, population-level shift - not your absolute outcome.

EVIDENCE

In population studies of women, each T allele at rs365132 in UIMC1 is associated with a slightly higher age at natural menopause. This is a population-level tendency across research cohorts - a relative, population-level shift, not a personal prediction - and many other genetic and lifestyle factors also influence menopausal timing.

SOURCES

PubMed 26414677

Age at natural menopause tendency

BRSK1

POPULATION STUDY

rs1172822 | GT C/T | DOSE 0/1 | REF/ALT C/T | STUDIED T (1 of 2) | GRCh38 +

INTERPRETATION

You inherited one C variant and one T variant - a heterozygous C/T genotype, one allele from each parent. At the population level this places you in the middle of the age-at-natural-menopause range, between the lower-end T/T and the higher-end C/C genotypes.

EVIDENCE

In population studies, each T allele at rs1172822 (BRSK1) is associated with a slightly earlier age at natural menopause. This is an association, not a medical prediction, and many other genetic and lifestyle factors also influence reproductive timing. (Applies to people who menstruate.)

SOURCES

PubMed 19448621

Later age at menopause tendency

MCM8

POPULATION STUDY

rs16991615 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the G variant - a G/G genotype, with no copies of the A variant present. In population studies this genotype sits toward the earlier end for age at natural menopause relative to A-carriers; each A copy is linked to a later-menopause shift.

EVIDENCE

In population studies, each A allele at rs16991615 (MCM8) is associated with a later age at natural menopause - the favorable direction for a longer reproductive window. This is an association, not a medical prediction, and many other genetic and lifestyle factors also influence reproductive timing. (Applies to people who menstruate.) This variant is reported for more than one trait, and the findings come from different studies and are not directly comparable.

SOURCES

PubMed 34349265

Menopause-timing tendency

NPIPB2 region

POPULATION STUDY

rs10852344 | GT C/C | DOSE 0/0 | REF/ALT C/T | STUDIED T (0 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the C variant - a C/C genotype. In population studies people with C/C tend to sit toward the later end of the menopause-age range, each C copy contributing a small upward shift. Relative, population-level shift - not your absolute timing.

EVIDENCE

In population studies, each T allele at this marker is associated with a slightly earlier age at menopause. This is an association, not a medical prediction. Many other genetic and lifestyle factors also influence menopause timing.

SOURCES

PubMed 26414677

Menopause-timing tendency PRRC2A

POPULATION STUDY

rs1046089 | GT G/A | DOSE 0/1 | REF/ALT G/A | STUDIED A (1 of 2) | GRCh38 +

INTERPRETATION

You have the G/A genotype at this menopause-timing locus. The two strongest studies disagree on the direction of the A allele's effect, so no reliable earlier-or-later tendency can be read from your genotype; menopause timing is shaped by many genes and lifestyle factors.

EVIDENCE

This marker (rs1046089, near PRRC2A in the MHC region) is an established age-at-natural-menopause locus, but the direction of the A allele's effect is genuinely inconsistent between the two strongest studies (one reports the A allele shifts menopause later, the other earlier - a likely strand/allele-coding discrepancy). Because the direction can't be resolved, this is shown as informational only, with no earlier-or-later signal. Many other genetic and lifestyle factors also influence menopause timing.

SOURCES

PubMed 22267201

05.11 Oxidative stress handling

2/3 markers

Antioxidant enzyme stability NQO1 P187S

POPULATION STUDY

rs1800566 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

Two stable copies at rs1800566 (G/G genotype, about 6 in 10 people): full NQO1 activity, the baseline setting for this quinone-clearing enzyme.

EVIDENCE

NQO1 neutralizes quinones, a reactive chemical class from smoke, exhaust, and some foods, and helps recycle antioxidants like CoQ10. The S187 version (A here) makes the enzyme unstable: one copy lowers activity and two copies leave almost none, which blood protein measurements confirm directly. About 1 in 25 people of European descent has the low-activity genotype. The enzyme effect is directly confirmed in blood protein studies (about two thirds of a standard deviation less enzyme per copy); health links beyond that are far less settled.

SOURCES

PubMed 22209713 | GWAS rs1800566

Mitochondrial antioxidant transport

SOD2 Ala16Val

POPULATION STUDY

rs4880 | GT A/A | DOSE 0/0 | REF/ALT A/G | STUDIED G (0 of 2) | GRCh38 +

INTERPRETATION

Two Val16 copies at rs4880 (A/A genotype, about 3 in 10 people): the version with less efficient delivery of this antioxidant enzyme into mitochondria. Outcome studies are mixed; antioxidant-rich food patterns and regular exercise support this system regardless.

EVIDENCE

SOD2 is the mitochondria's main defense against the reactive oxygen they generate making energy. This variant changes the shipping label on the enzyme: the Ala version (G here) gets imported into mitochondria more efficiently than the Val version (A). Whether more import is better appears to depend on context, so this reads as a setting, not a score. The import difference is solid biochemistry; links from there to health outcomes are mixed across studies, which is why this reads as a setting rather than a risk.

SOURCES

PubMed 12618592

05.12 Protective heart variants (APOE e2, ANGPTL4)

2x

Coronary artery disease-associated variant

ANGPTL4

POPULATION STUDY

rs116843064 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

No E40K copies at rs116843064 (G/G genotype), like about 24 in 25 people: typical ANGPTL4 braking. Triglycerides respond well to the usual levers: activity, alcohol moderation, and refined-carb control.

EVIDENCE

ANGPTL4 acts as a brake on lipoprotein lipase, the enzyme that clears triglyceride-rich fats from the blood; the E40K variant weakens that brake, so fats clear faster. Carriers (about 1 in 25 people of European descent) run lower triglycerides and show roughly 15 to 20 percent lower odds of coronary artery disease (protective OR 1.19). Drug developers are actively imitating this effect, which says something about how good it is to have naturally. For health decisions, talk to a professional - this is general wellness information.

SOURCES

PubMed 36474045 | PubMed 26933753

Coronary artery disease-associated variant APOE

POPULATION STUDY

rs7412 | GT C/C | DOSE 0/0 | REF/ALT C/T | STUDIED T (0 of 2) | GRCh38 +

INTERPRETATION

No epsilon 2 copies at rs7412 (C/C genotype): the common situation; your APOE heart signal is neutral here.

EVIDENCE

APOE ferries cholesterol between tissues, and its epsilon 2 form (tagged by the T allele here) is the favorable end for the heart: carriers run lower LDL and about 20 percent lower odds of coronary artery disease than the epsilon 3/epsilon 3 majority (protective OR 1.25). One caveat lives at two copies: by classic estimates roughly 1 in 10 to 1 in 20 people with the rare epsilon 2/epsilon 2 genotype develop a very treatable lipid condition (type III hyperlipoproteinemia), which a standard lipid panel picks up. For health decisions, talk to a professional - this is general wellness information.

SOURCES

GWAS rs7412 | PubMed 17878422

05.13 Telomere length tendency

2/3 markers

Telomere length tendency OBFC1/STN1

POPULATION STUDY

rs9420907 | GT A/A | DOSE 1/1 | REF/ALT C/A | STUDIED A (2 of 2) | GRCh38 +

INTERPRETATION

Both copies at this position carry the A variant, an A/A genotype, associated in research cohorts with shorter telomeres at this site. A neutral measurement, not a health benefit or signal on its own.

EVIDENCE

At this OBFC1 marker, the A allele is associated in population studies with shorter telomere length and the C allele with longer telomeres. Telomere length is a neutral biological measurement with no simple good-or-bad meaning, shown for information only. Evidence is limited. Many genes and everyday habits also influence it.

SOURCES

PubMed 35530816 | PubMed 23535734

Telomere length tendency

TERC region

POPULATION STUDY

rs1317082 | GT A/A | DOSE 0/0 | REF/ALT A/G | STUDIED G (0 of 2) | GRCh38 +

INTERPRETATION

An A/A genotype at this TERC variant, associated in population studies with slightly longer telomere length. This is a neutral measurement, not a good or bad health signal.

EVIDENCE

At the TERC locus, the G allele is associated in population studies with slightly shorter telomere length and the other allele with longer. Telomere length is a neutral biological measurement with no simple good-or-bad meaning, so this is shown for information only. Evidence is limited (a single record), and it is one of two neighboring variants usually inherited together, treat them as a single shared signal. Many genes and everyday habits also influence this measurement.

SOURCES

PubMed 35530816 PubMed 23535734

05.14 TP53 survival trade-off

1x

Survival trade-off variant

TP53 Pro72Arg

POPULATION STUDY

rs1042522 | GT C/C | DOSE 1/1 | REF/ALT G/C | STUDIED G (0 of 2) | GRCh38 +

INTERPRETATION

Two Arg72 copies at rs1042522 (C/C genotype, about half of Europeans): the more vigilant damage-control setting; the Leiden survival advantage was seen in the Pro/Pro group instead. Typical, with the trade-off running the other way.

EVIDENCE

TP53 is the genome's damage-control chief: it decides whether a stressed cell repairs itself or shuts down. Its two common versions strike slightly different bargains. In a Dutch cohort followed from age 85, people with two Pro versions showed about 40 percent better survival than Arg carriers, while other studies link Pro to a somewhat softer touch against cancer earlier in life. A genuine trade-off, not a score. The evidence comes mainly from one well-run Dutch cohort (about 40 percent better survival after 85) with partial replication elsewhere; a documented lean, not a rule.

SOURCES

PubMed 15732191

05.15 Age-related vision loss (macular degeneration)

1/2 markers

Age-related vision loss variant

AMD, ARMS2

POPULATION STUDY

rs10490924 | GT G/G | DOSE 0/0 | REF/ALT G/T | STUDIED T (0 of 2) | GRCh38 +

INTERPRETATION

No risk copies at rs10490924 (G/G genotype), the situation of roughly 2 in 3 people of European descent: this AMD signal sits at baseline. Routine eye care after 50 still applies, as AMD has other drivers, including the separate CFH signal.

EVIDENCE

ARMS2 sits in retinal support tissue, and its A69S variant is the other of the two main genetic signals for age-related macular degeneration (AMD), a gradual loss of central vision late in life, alongside CFH. Advanced AMD affects roughly 3 in 100 people over 75; each risk copy here nearly triples the odds (OR 2.9), yet most carriers still never develop it. The same two levers apply: not smoking, and a routine dilated eye exam after 50 so change is caught while treatment protects vision. For health decisions, talk to a professional - this is general wellness information.

SOURCES

PubMed 23455636 | PubMed 37696869 | PubMed 22705344 | GWAS rs10490924 | PubMed 28712657

05.16 Factor V Leiden (clotting) 1x

Factor V Leiden variant

F5

CATALOGED VARIANT

rs6025 | GT C/C | DOSE 0/0 | REF/ALT C/T | STUDIED T (0 of 2) | GRCh38 +

INTERPRETATION

No Leiden copies at rs6025 (C/C genotype): typical clotting from this gene, the situation of roughly 19 in 20 people of European descent.

EVIDENCE

Factor V is a clotting protein; the Leiden variant makes it harder to switch off once activated, which raises the chance of blood clots in veins (legs, lungs). About 1 to 2 in 1000 adults per year develop such a clot; for people with one Leiden copy the rate is roughly 3 to 5 in 1000 per year, and most carriers never have one. The risk concentrates around surgery, long immobility, pregnancy, and estrogen-containing contraception, which is exactly when telling your doctor about this variant pays off. For health decisions, talk to a professional - this is general wellness information.

CLINVAR REVIEW

1 of 4 review stars - criteria provided, conflicting classifications. Classified: Conflicting classifications of pathogenicity. This says how much expert review stands behind the classification, not how strong the effect is.

SOURCES

ClinVar | PubMed 28373160 | PubMed 33592630 | PubMed 19652888

05.17 Prothrombin clotting variant (F2) 1x

Prothrombin G20210A variant F2

CATALOGED VARIANT

rs1799963 | GT G/G | DOSE 0/0 | REF/ALT G/A | STUDIED A (0 of 2) | GRCh38 +

INTERPRETATION

No G20210A copies at rs1799963 (G/G genotype): typical prothrombin level from this gene, the situation of roughly 49 in 50 people of European descent.

EVIDENCE

Prothrombin is the raw material for the main clotting enzyme; the G20210A variant nudges its level up, which raises venous clot risk (legs, lungs) about 2 to 3 times. In absolute terms that is roughly 3 to 5 clots per 1000 carriers per year versus 1 to 2 per 1000 without it, so most carriers never have one. As with Factor V Leiden, the value is situational: mention it before surgery, during pregnancy planning, and when choosing contraception. For health decisions, talk to a professional - this is general wellness information.

CLINVAR REVIEW

2 of 4 review stars - criteria provided, multiple submitters, no conflicts. Classified: Pathogenic/Likely pathogenic/Pathogenic, low penetrance/Established risk allele; risk factor. This says how much expert review stands behind the classification, not how strong the effect is.

SOURCES

ClinVar | PubMed 12069454 | PubMed 19652888

05.18 Survival to age 90+ 1x

Survival to age 90+ variant 5q33.3 region

POPULATION STUDY

rs2149954 | GT C/T | DOSE 0/1 | REF/ALT C/T | STUDIED T (1 of 2) | GRCh38 +

INTERPRETATION

One T copy at rs2149954 (C/T genotype): about 10 percent higher odds of reaching age 90+ versus the no-copy group in a genome-wide longevity study, which also reported lower cardiovascular mortality among carriers. A small, well-grounded tailwind.

EVIDENCE

One of the very few longevity signals to clear the strictest statistical bar in a dedicated genome-wide study: each T copy here was associated with about 10 percent higher odds of living past 90, with lower cardiovascular mortality also reported among carriers. A small but well-grounded tailwind; about 6 in 10 Europeans carry at least one T copy.

SOURCES

PubMed 24688116 | GWAS rs2149954