

SAMPLE REPORT

NUTRITION REPORT • 6 SECTIONS

Your nutrition report.

What published genetic studies say about 27 traits (nutrient levels in blood and food habits) and 24 gene highlights, read from your raw DNA file. Generated in your browser.

Fig. 0 – Specimen label		1 of 1
REPORT	SCOPE	
Nutrition genetics report	6 sections • 27 traits • 24 gene highlights	
SOURCES	INPUT	
GWAS Catalog release 2026-06-01 • studies linked to PubMed	Raw DNA file, read locally	
DNA DATA SENT TO OUR SERVERS	USE	
0 bytes	Informational • not dietary advice	

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About this report

WHAT THIS REPORT IS

An informational report on how the genetic variants measured in your raw DNA file compare with published genome-wide association studies of nutrient levels and food-related traits. It is not a medical or diagnostic test, does not assess any disease, and gives no diet, supplement or treatment recommendations.

How to read this report

The report opens with a one-page overview of every result. Each section then starts with a cover page that lists what it contains. Within a section, each trait starts with what it is and your result: how many of its known variants your file measures and, when enough are measured, the tendency they point to. A table of the variants follows, then general information that applies to everyone, whatever their genes. Gene highlights read single, well-studied variants one by one and say what your genotype usually means. Three appendices list the nutrients without reliable genetic studies yet, popular gene claims this report leaves out and why, and the methods and sources.

Variant	A position in the DNA where people commonly differ, named by an rsID such as rs4988235. Your genotype is the two letters your file reports there, one inherited from each parent.
Effect allele	The letter that a study linked with a higher or lower value. Your copies of it, 0, 1 or 2, are compared with the copies the average person of European ancestry carries.
Tendency	When enough of a trait's variants are measured, the copies of alleles linked with higher values are added up and those linked with lower values subtracted, each against the average person. When the total lies clearly above or below the average, the report gives a tendency toward a slightly higher or lower value; otherwise it finds no meaningful difference. About one person in three gets a tendency for a given trait.
Not assessable	Your file measures fewer than a third of the variants known for the trait. The missing ones could point either way, so no tendency is given. This says nothing about your level.
Too few variants for a tendency	Fewer than three variants could be compared. The report then lists the variants your file measures and gives no tendency.
Stand-in	When your file lacks a known variant, another variant can be read in its place. Tables and gene highlights show this as rs1 ≈ rs2: rs1 was read in place of rs2. In a trait's table, the stand-in is a nearby variant, within 50,000 bases, that studies also linked with the same trait; it is scored with its own effect, which can differ from that of the missing variant. A gene highlight uses only stand-ins that are usually inherited with its marker, so its reading is very likely the same.
Gene highlight	A single, well-studied variant, or a few of them, read with a rule of its own and explained one by one. Evidence A means the finding was replicated in large studies; evidence B that it was replicated in smaller or less direct studies. Because a highlight reads one to a few strong variants on their own while a trait counts up to 20, a highlight and the trait it sits under can point different ways.

How to read the variant tables

A trait's table lists every variant your file measures for it, those at which you carry a copy of an effect allele first. Rows in gray are variants at which you carry no copy: they count toward the tendency too, because the average person carries some copies.

Variant	The variant's rsID; rs1 ≈ rs2 marks a stand-in.
Genotype	The two letters your file reports at the variant, as your file writes them; on a man's X chromosome, of which he has one copy, the report shows one letter.
Effect allele	The allele the chosen study linked with the trait, shown as it reads on your file's strand.
Copies	How many copies of the effect allele your genotype holds.

Frequency	How common the effect allele is in people of European ancestry, as a share between 0 and 1. The average person carries twice this number of copies.
Effect per copy	The change per copy in the chosen study, for example +0.12 SD (standard deviations) or a change in the study's unit; 'unit' means the study did not state it. The sign gives the direction. '(not scored)' marks a variant that does not count toward the tendency.
Studies (PubMed IDs)	Every study in the GWAS Catalog that reports the variant for this trait, linked to its PubMed record.

What this report cannot tell you

- Genes are a small part of the picture. For most nutrients, the common variants studied here explain only a small part of the differences in blood levels between people. Diet, sun, body weight, age, health and medicines usually matter more.
- Only a blood test measures a level. A tendency toward a lower level does not mean that your level is low, and a tendency toward a higher level does not mean that it is high or adequate.
- Nutrient needs are set by age, sex, life stage and health, not by these variants. General reference intakes apply to you as to everyone; the figures quoted are EFSA's for the adult population.
- This report gives no diet, supplement or treatment recommendations. Do not start, stop or change a diet, a supplement or a medicine because of it.
- Your variants are compared with the average person of European ancestry, the group most of these studies were done in. For people of other ancestries the comparison is less certain.
- Consumer DNA chips measure only part of the known variants, and research moves on. This report uses the GWAS Catalog release of 2026-06-01.

QUESTIONS

Your genes are only one small influence on your nutrient levels. If you have symptoms or questions about your diet, talk to a doctor or a registered dietitian; a blood test is the only way to know your level.

Your results at a glance

Each line is one trait of this report or one gene highlight. For a trait, the mark shows the tendency your measured variants point to, compared with the average person of European ancestry, and the bar shows how many of its known variants your file measures: the tick marks one third, below which no tendency is given. For a gene highlight, the bar shows how many of its markers your file has. The pages that follow explain each result. A gene highlight reads one to a few strong variants on their own while a trait counts up to 20, so a highlight and its trait can point different ways.

11

traits with a tendency

10

close to average

6

no tendency given

24/24

gene highlights read

▲ higher or more ▼ lower or less — average ○ no tendency ● highlight read █ measured of known

VITAMINS		7 traits · 7 highlights
Vitamin A	— average	4/5
BC01 (BCM01)	● no copy	1/1
Vitamin B6	○ too few variants	1/1
ALPL	● two copies	1/1
Folate (vitamin B9)	○ not assessable	1/7
Vitamin B12	— average	8/12
FUT2	● no copy	1/1
TCN2	● no copy	1/1
Vitamin C	▲ higher	4/10
SLC23A1 (SVCT1)	● no copy	1/1
Vitamin D	▼ lower	11/20
GC / CYP2R1 / DHCR7	● more lowering	3/3
Vitamin E	○ too few variants	2/2
CYP4F2	● no copy	1/1

FATTY ACIDS AND HOMOCYSTEINE		5 traits · 3 highlights
Omega-3 fatty acids	— average	9/20
FADS1 / FADS2	● one copy	1/1
DHA	▼ lower	10/20
ELOVL2	● one copy	1/1
Omega-6 to omega-3 ratio	— average	9/20
Linoleic acid (omega-6)	— average	12/20
Homocysteine	▼ lower	11/12
MTHFR	● one copy	1/1

FOOD TOLERANCE		2 highlights
Lactase persistence	● no copy	1/1
Celiac-linked HLA types	● no DQ2 or DQ8 tag	5/6

MINERALS AND IRON		10 traits · 4 highlights
Calcium	— average	15/20
CASR	● no copy	1/1
Magnesium	— average	3/5
Phosphorus	▼ lower	12/20
Iron	▼ lower	7/17
HFE	● C282Y not in file	2/3
TMPRSS6	● two copies	1/1
Ferritin	▼ lower	7/20
Transferrin	— average	7/7
Transferrin saturation	▼ lower	4/7
Zinc	— average	3/3
Copper	○ too few variants	2/5
Selenium	○ too few variants	1/1
DMGDH / BHMT	● no copy	1/1

FOOD AND DRINK HABITS		5 traits · 4 highlights
Coffee intake	▲ more	9/15
CYP1A1-CYP1A2 / AHR	● fewer	2/2
Alcohol intake	▼ less	10/20
ALDH2	● no copy	1/1
ADH1B Arg48His	● no copy	1/1
ADH1B Arg370Cys	● one copy	1/1
Protein intake	— average	4/4
Fat intake	▲ more	3/7
Carbohydrate intake	○ too few variants	2/5

TASTE AND SMELL		4 highlights
Bitter taste (TAS2R38)	● AVI/AVI	3/3
Floral smell of beta-ionone (OR5A1)	● two copies	1/1
Smelling asparagus in urine	● no copy	1/1
Sweet preference (FGF21)	● no copy	1/1

Tendencies are small: they describe your variants, not your levels, and only a blood test measures a level. Informational only, not medical or dietary advice.

01of 6

Vitamins

Section 01 of 6 • 7 traits • 7 gene highlights

Fig. 01 – In this section		7 traits • 7 gene highlights
01	Vitamin A	
02	Vitamin B6	
03	Folate (vitamin B9)	
04	Vitamin B12	
05	Vitamin C	
06	Vitamin D	
07	Vitamin E	
and 7 gene highlights		

Vitamins

Vitamins are substances the body needs in small amounts but cannot make, or cannot make in sufficient amounts. This section compares the variants in your DNA file with genome-wide studies of vitamin levels in blood. These common variants explain only a small part of the differences between people. Diet, supplements, sunlight, age, health and medicines usually matter more, and only a blood test can measure your level. These variants do not change how much of a vitamin you need. The reference values quoted are EFSA's figures for the adult population, and they apply whatever your genotype.

Vitamin A

Vitamin A is a fat-soluble vitamin. Animal foods supply it ready-made as retinol, and plants supply carotenoids such as beta-carotene, which the body turns into vitamin A as needed. Vitamin A contributes to the maintenance of normal vision, skin and mucous membranes, to normal iron metabolism and to the normal function of the immune system.

YOUR RESULT

— No meaningful difference  4/5

Independent variants known for this trait: 5; with a call in your file: 4; compared with the average person: 4; carrying at least one copy of the effect allele: 2.

Taken together, your scored variants point to no meaningful difference from the average person (net effect-allele copies +0.7; a total more than one standard deviation from the average counts as a tendency).

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs1667255	AC	C	1	0.38	+0.03 ln(µg/L)	21878437
rs645040	GT	T	1	0.77	−0.09 unit	36357675
rs10882272 ≈ rs10882283	TT	C	0	0.38	−0.03 ln(µg/L)	21878437
rs780093	CC	T	0	0.41	+0.10 unit	36357675

rs1 ≈ rs2: your file lacks the known variant rs2, so rs1, a nearby variant within 50,000 bases that studies also linked with this trait, was read in its place and scored with its own effect.

What the studies measured

The studies measured retinol in blood serum or plasma. Most of them measured it along with many other small molecules in the same blood sample. The liver stores vitamin A and keeps the blood level steady over a wide range of intakes, so the level says little about diet. It also falls during infections.

If intake is too low (general information)

A long-term lack of vitamin A first affects vision in dim light and the surface of the eyes; it is rare in Europe. These signs have many causes, and only a blood test and a doctor can tell.

Too much (general information)

Too much preformed vitamin A (retinol), almost always from supplements or from eating liver often, can harm the liver, the bones and the unborn child. EFSA has set a tolerable upper intake level of 3,000 micrograms of preformed vitamin A a day for adults. Beta-carotene from plants does not count toward this limit.

Reference intake

EFSA's population reference intake for adults is 750 micrograms of retinol equivalents a day for men and 650 micrograms for women.

Food sources

Liver, dairy products, eggs and oily fish provide retinol; carrots, sweet potatoes, pumpkin, dark green leafy vegetables, apricots and mangoes provide beta-carotene.

Beta-carotene to vitamin A conversion

YOUR GENOTYPE **rs6564851 • TT**

Your file shows no copy of the G version near BCO1, like about 1 in 4 people of European ancestry and more than 4 in 10 of African ancestry. Studies found that people with this pattern tend to have slightly lower beta-carotene and higher lutein and zeaxanthin in the blood, which fits a more active conversion of beta-carotene into vitamin A.

Background	BCO1 is the enzyme that splits beta-carotene from plant foods into vitamin A. In a genome-wide study of 3,941 people, each G copy near BCO1 went with higher beta-carotene and lower lutein, zeaxanthin and lycopene in the blood, by 0.10 to 0.28 standard deviations per copy, but with no difference in vitamin A (retinol) itself. A study of 2,344 women confirmed the link with higher beta-carotene. This pattern fits a less active conversion of beta-carotene, but conversion itself has only been measured in small studies.
Keep in mind	Vitamin A also comes ready-made from animal foods and its blood level is tightly controlled by the body, so this matters mostly for how plant carotenoids are used; other BCO1 variants not read here also play a part. Only a blood test shows your levels.

SOURCES [19185284](#), [22113863](#), [23134893](#)

Vitamin B6

Vitamin B6 is a water-soluble vitamin. Its active form, pyridoxal phosphate (PLP), helps more than 100 enzymes, most of them in protein metabolism. Vitamin B6 contributes to normal protein and glycogen metabolism, normal red blood cell formation and normal homocysteine metabolism. It also contributes to normal functioning of the nervous system and to the normal function of the immune system.

YOUR RESULT

☐ Too few variants for a tendency  1/1

Independent variants known for this trait: 1; with a call in your file: 1; compared with the average person: 1; carrying at least one copy of the effect allele: 1.

Only one variant is known for the blood level of vitamin B6, and your file measures it. One variant is not enough for a tendency, so this report gives none; the variant is listed below.

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs4654748	CC	C	2	0.53	-1.45 ng/mL	19303062

What the studies measured	This item rests on one gene region. Two studies measured vitamin B6 in blood plasma, one of them in people who had had a stroke, and both found variants near the ALPL gene. ALPL makes an enzyme that breaks down PLP in blood. Besides genes, the level depends on diet, supplements, inflammation and kidney function.
If intake is too low (general information)	A lack of vitamin B6 on its own is uncommon. It usually occurs together with low levels of other B vitamins and can cause cracked lips, a sore tongue and a type of anemia. These signs have many causes, and only a blood test and a doctor can tell.
Too much (general information)	Too much vitamin B6, almost always from supplements, can damage the nerves of the hands and feet. EFSA has set a tolerable upper intake level of 12 mg a day for adults.
Reference intake	EFSA's population reference intake for adults is 1.7 mg a day for men and 1.6 mg a day for women.
Food sources	Poultry, fish, meat, potatoes, pulses, bananas, nuts and whole grains.

Vitamin B6 blood form (ALPL)

YOUR GENOTYPE **rs4654748 • CC**

Your file shows two copies of the C version at ALPL, like about 1 in 4 people of European ancestry and most people of African ancestry. Studies found that each C copy goes with about 1.5 ng/mL lower PLP, the blood form of vitamin B6, so about 3 ng/mL lower than people with no copy. This reflects how the blood form is processed and does not by itself show how much B6 your cells get.

Background ALPL makes alkaline phosphatase, an enzyme that releases vitamin B6 from its main blood form, PLP, so it can pass into cells. In a genome-wide study of 2,934 people, each C copy went with about 1.5 ng/mL (about 6 nmol/L) lower PLP in the blood, and a second study of 4,763 people confirmed the link. In 162,774 Icelanders the same C version went with higher alkaline phosphatase, which fits this explanation.

Keep in mind Lower blood PLP linked to this gene may not mean less vitamin B6 in your cells, because the released form also reaches the tissues. Diet matters more, and only a blood test shows your level.

SOURCES [19303062](#), [19744961](#), [31666285](#)

Folate (vitamin B9)

Folate, vitamin B9, is a water-soluble vitamin; folic acid is the form used in supplements and fortified foods. Folate has a role in the process of cell division. It contributes to normal blood formation, normal amino acid synthesis and normal homocysteine metabolism, and to maternal tissue growth during pregnancy.

YOUR RESULT

☐ Not assessable (1 of 7 variants measured)  1/7

Independent variants known for this trait: 7; with a call in your file: 1; compared with the average person: 1; carrying at least one copy of the effect allele: 1.

Your file measures fewer than a third of these variants (1 of 7), so no overall tendency is given: the variants your chip did not measure could point either way.

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs4846080	GG	G	2	0.55	-0.08 unit	39414775

What the studies measured	The studies measured folate in blood. Most findings come from one study that used routine blood tests in the medical records of British adults of South Asian ancestry. A few come from Chinese patients who had had a stroke. Blood folate reflects recent intake from food, fortified foods and supplements. Besides genes, the level also depends on alcohol, smoking, some medicines and vitamin B12 levels.
If intake is too low (general information)	Low folate over a long time can lead to a type of anemia with large red blood cells and to a sore tongue. These signs have many causes, and only a blood test and a doctor can tell.
Too much (general information)	Folate from food causes no harm, but high intakes of folic acid can hide a lack of vitamin B12. For folic acid and similar forms added to foods or supplements, EFSA has set a tolerable upper intake level of 1,000 micrograms a day for adults. This limit does not include folate naturally present in food.
Reference intake	EFSA's population reference intake for adults is 330 micrograms of dietary folate equivalents a day.
Food sources	Dark green leafy vegetables, pulses, liver, eggs, whole grains, oranges and fortified foods.

Vitamin B12

Vitamin B12, cobalamin, is a water-soluble vitamin found naturally almost only in foods of animal origin. Its absorption needs intrinsic factor, a protein made in the stomach. Vitamin B12 contributes to normal red blood cell formation, normal functioning of the nervous system and normal homocysteine metabolism. It also has a role in the process of cell division.

YOUR RESULT

No meaningful difference 8/12

Independent variants known for this trait: 12; with a call in your file: 8; compared with the average person: 8; carrying at least one copy of the effect allele: 2.

Taken together, your scored variants point to no meaningful difference from the average person (net effect-allele copies -0.8; a total more than one standard deviation from the average counts as a tendency).

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs1047891	AC	A	1	0.30	-0.06 unit	39414775
rs1801222 ≈ rs12243895	AG	A	1	0.29	-0.22 unit	25147783
rs1131603	TT	C	0	0.07	+0.35 unit	39414775
rs2232775	TT	C	0	0.04	+0.23 unit	39414775
rs34324219	CC	A	0	0.12	-0.28 unit	25147783, 28334792, 39414775
rs3760775 ≈ rs12019136	GG	T	0	0.07	+0.25 SD	28334792
rs41281112	CC	T	0	0.03	-0.19 unit	39414775
rs61901623	AA	G	0	0.05	-0.16 unit	39414775

rs1 ≈ rs2: your file lacks the known variant rs2, so rs1, a nearby variant within 50,000 bases that studies also linked with this trait, was read in its place and scored with its own effect.

What the studies measured	The studies measured vitamin B12 in blood serum or plasma. Most findings come from routine blood tests in the medical records of British adults of South Asian ancestry. Besides genes, the level depends on how much animal food people eat and on stomach acid, which declines with age. Medicines such as metformin and long-term acid-reducing drugs also lower it.
If intake is too low (general information)	A lack of vitamin B12 develops slowly, often over years, and can lead to anemia and to nerve problems such as tingling in the hands and feet. These signs have many causes, and only a blood test and a doctor can tell.
Too much (general information)	No harmful effects of high intakes are known, and EFSA lists no tolerable upper intake level for vitamin B12.
Reference intake	EFSA's adequate intake for adults is 4 micrograms a day.
Food sources	Meat, liver, fish, shellfish, eggs and dairy products; plant foods contain it only when fortified.

GENE HIGHLIGHT · FUT2

Evidence A

FUT2 secretor status and vitamin B12 readings

YOUR GENOTYPE rs492602 ≈ rs601338 · AA

Your file shows no copy of the main non-secretor version of FUT2, like about 3 in 10 people of European ancestry, which usually means you are a secretor. Studies found no gene-related shift in total B12 readings for this pattern.

A marker shown as rs1 ≈ rs2 was read from a neighboring marker, rs1, because your file lacks rs2. In people of European ancestry the two almost always travel together, so the reading is very likely the same.

Background	FUT2 decides whether you are a secretor, meaning whether blood-group sugars are made in saliva and the gut lining; it takes two copies of the non-secretor version to be a non-secretor. In a study of about 2,700 women, non-secretors had total vitamin B12 readings about 80 pg/mL (about 60 pmol/L) higher on a standard blood test, while
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people with one copy had the same readings as people with none. Genome-wide studies of up to 45,568 Icelanders and Danes confirmed the link. The extra B12 is bound to haptocorrin, a carrier that only the liver takes up, while the form that cells use, holo-transcobalamin, did not differ.

Keep in mind

A higher total B12 reading linked to this gene does not mean more B12 reaches your cells, and this card says little for people of East Asian ancestry, where a different FUT2 variant not read here is common. Only a blood test shows your B12 level.

SOURCES [18776911](#), [19303062](#), [23754956](#), [29040465](#)

GENE HIGHLIGHT • TCN2

Evidence B

TCN2 and vitamin B12 readingsYOUR GENOTYPE [rs1131603](#) • TT

Your file shows no copy of the less common TCN2 version. This is the usual pattern, found in about 87 in 100 people of European ancestry and almost everyone of African or East Asian ancestry. Studies found no gene-related shift in B12 readings for this pattern.

Background

TCN2 makes transcobalamin, the protein that carries vitamin B12 into cells. A less common version of it (Leu376Ser) went with higher total B12 on blood tests in two large genome-wide studies: about 0.2 to 0.3 standard deviations per copy in 45,575 Icelanders and Danes, and about 0.35 in British South Asians. About 1 in 8 people of European ancestry carries it; it is rare in other groups.

Keep in mind

The studies measured total B12 in blood, not how much reaches the cells, and the effect was measured mostly in European and South Asian ancestry. Only a blood test shows your B12 level.

SOURCES [23754956](#), [39414775](#)

Vitamin C

Vitamin C, ascorbic acid, is a water-soluble vitamin that the human body cannot make. It contributes to normal collagen formation for the normal function of blood vessels, bones, cartilage, gums, skin and teeth. Vitamin C also contributes to the protection of cells from oxidative stress and to the normal function of the immune system, and it increases iron absorption.

YOUR RESULT

▲ Slightly higher level  4/10

Independent variants known for this trait: 10; with a call in your file: 4; compared with the average person: 4; carrying at least one copy of the effect allele: 4.

Your scored variants point to a tendency toward a slightly higher blood level of vitamin C than in the average person of European ancestry (net effect-allele copies +1.7; a total more than one standard deviation from the average counts as a tendency). The effect is small: genes explain only part of the differences between people, and diet, age, health and medicines usually matter more. This report does not measure your level; only a blood test can.

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs174547	CT	C	1	0.35	+0.04 unit	33203707
rs33972313	CC	C	2	0.97	+0.36 unit	33203707
rs6693447	TT	T	2	0.54	+0.04 unit	33203707
rs9895661	TT	T	2	0.81	+0.06 unit	33203707

What the studies measured

The studies measured vitamin C in blood plasma. It mostly follows recent intake of fruit, vegetables and supplements; smokers have lower levels, and levels fall during illness.

If intake is too low (general information)	Very low intake over weeks to months causes scurvy, with bleeding gums, easy bruising and slow wound healing; it is rare in Europe today. These signs have many causes, and only a blood test and a doctor can tell.
Too much (general information)	Large doses from supplements can cause stomach upset and diarrhea. EFSA has not set a tolerable upper intake level for vitamin C.
Reference intake	EFSA's population reference intake for adults is 110 mg a day for men and 95 mg a day for women.
Food sources	Peppers, citrus fruit, kiwi, berries, broccoli, Brussels sprouts, cabbage and potatoes.

GENE HIGHLIGHT · SLC23A1 (SVCT1)Evidence A

Vitamin C transporter

YOUR GENOTYPE rs33972313 · CC

Your file shows no copy of the less common version of the vitamin C transporter. This is the usual pattern, found in about 93 in 100 people of European ancestry and 86 in 100 of African ancestry. Studies found no gene-related shift in blood vitamin C for this pattern.

Background

SLC23A1 makes SVCT1, the transporter that takes up vitamin C in the gut and recovers it in the kidneys. A less common version (Val264Met) went with about 6 micromol/L lower blood vitamin C per copy across five studies of more than 15,000 people, and this gene was the strongest genetic signal in a study of 52,018 people of European ancestry.

Keep in mind

Diet has a much larger effect on blood vitamin C than this gene, and only a blood test shows your level.

SOURCES 20519558, 33203707, 25527764

Vitamin D

Vitamin D is a fat-soluble vitamin that the skin makes in sunlight and that a few foods also provide. Vitamin D contributes to the normal absorption and use of calcium and phosphorus and to the maintenance of normal bones, teeth and muscle function. It also contributes to the normal function of the immune system.

YOUR RESULT

▼ Slightly lower level11/20

Independent variants known for this trait: 20; with a call in your file: 11; compared with the average person: 11; carrying at least one copy of the effect allele: 5.

Your scored variants point to a tendency toward a slightly lower blood level of vitamin D (25-hydroxyvitamin D) than in the average person of European ancestry (net effect-allele copies −2.5; a total more than one standard deviation from the average counts as a tendency). The effect is small: genes explain only part of the differences between people, and diet, age, health and medicines usually matter more. This report does not measure your level; only a blood test can.

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs12130219 ≈ rs12123821	AG	G	1	0.24	−0.02 unit	33462484
rs12785878 ≈ rs12803256	GT	T	1	0.70	+0.04 unit	29343764
rs12794714 ≈ rs117913124	AG	G	1	0.55	+0.09 unit	32242144
rs4588 ≈ rs11723621	GT	T	1	0.25	−0.25 SD	28757204, 29545823, 33382404, 36811574
rs6068816 ≈ rs2762942	CT	T	1	0.12	−0.03 unit	33462484
rs11023156 ≈ rs72632971	AA	G	0	0.17	+0.03 unit	33462484
rs138600084 ≈ rs577185477	CC	T	0	0.01	+0.09 unit	33462484
rs17216525	CC	T	0	0.07	+0.04 unit	33462484

rs1 ≈ rs2: your file lacks the known variant rs2, so rs1, a nearby variant within 50,000 bases that studies also linked with this trait, was read in its place and scored with its own effect.

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs1800588	CC	T	0	0.21	-0.03 unit	32059762
rs58542926	CC	T	0	0.07	+0.04 unit	32059762 , 33462484
rs74669452 ≈ rs2011425	AA	C	0	0.01	-0.06 unit	33462484

rs1 ≈ rs2: your file lacks the known variant rs2, so rs1, a nearby variant within 50,000 bases that studies also linked with this trait, was read in its place and scored with its own effect.

What the studies measured	The studies measured 25-hydroxyvitamin D in blood serum, the form used to judge vitamin D status. Besides genes, sun exposure, season, latitude, skin color, body weight, age, diet and supplements usually matter more.
If intake is too low (general information)	In central and northern Europe the skin makes little vitamin D from about October to March. Low vitamin D over a long time can soften the bones, causing rickets in children and bone pain and muscle weakness in adults. These signs have many causes, and only a blood test and a doctor can tell.
Too much (general information)	Too much vitamin D, almost always from high-dose supplements and never from sunlight, raises blood calcium. EFSA has set a tolerable upper intake level of 100 micrograms a day for adults.
Reference intake	EFSA's adequate intake for adults is 15 micrograms a day, set for people whose skin makes little vitamin D.
Food sources	Oily fish, egg yolks, liver, some mushrooms exposed to UV light and fortified foods; for most people, sunlight on the skin is the main source.

GENE HIGHLIGHT • GC / CYP2R1 / DHCR7

Evidence A

Vitamin D transport and production genes

YOUR GENOTYPE GC • rs4588 • GT CYP2R1 • rs10741657 • GG DHCR7 • rs12785878 • GT

Vitamin D lowering copies: 5 of 8 (GC copies count double)

Your file shows more vitamin D lowering versions than most people across these three genes, like about 1 in 7 people of European ancestry; this pattern is more common in people of East Asian, South Asian or Latin American ancestry. Adding up the per-copy effects from large studies, people with this pattern tend to have vitamin D levels roughly 6 nmol/L below the study average, small next to the usual spread of about 25 nmol/L between people.

Background	Three genes shape the vitamin D blood level that doctors measure (25-hydroxyvitamin D): GC makes the protein that carries vitamin D in the blood, CYP2R1 turns vitamin D into this storage form in the liver, and DHCR7 uses up the substance in the skin that sunlight turns into vitamin D. In a combined analysis of about 38,000 people of European ancestry, each lowering copy went with about 4.7 nmol/L (GC), 1.7 nmol/L (CYP2R1) and 2.1 nmol/L (DHCR7) lower levels. Because the GC effect is about twice as large, GC copies count double in the pattern shown here. Together these genes explain only a few percent of the differences between people.
Keep in mind	The effects were measured mostly in people of European ancestry and may differ in others, and sun exposure, season, skin type, body weight and diet matter much more. Only a blood test shows your vitamin D level.

SOURCES [20541252](#), [23393431](#), [29343764](#), [36811574](#)

Vitamin E

Vitamin E is a group of fat-soluble compounds, of which alpha-tocopherol is the main form in the body. It sits in cell membranes, and vitamin E contributes to the protection of cells from oxidative stress.

YOUR RESULT

○ Too few variants for a tendency  2/2

Independent variants known for this trait: 2; with a call in your file: 2; compared with the average person: 2; carrying at least one copy of the effect allele: 0.

Your file measures both variants known for the blood level of vitamin E. A tendency needs at least three, so this report gives none; the variants measured are listed below.

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs11057830	GG	A	0	0.17	+0.03 unit	21729881
rs2108622	CC	T	0	0.29	+0.03 unit	21729881

What the studies measured	The studies measured alpha-tocopherol in blood serum. Vitamin E travels in blood with cholesterol and other fats, so its level partly follows blood fat levels; diet and supplements also matter.
If intake is too low (general information)	A lack of vitamin E is rare. It occurs mainly in people who cannot absorb fat normally, and it can cause muscle weakness and problems with balance. These signs have many causes, and only a blood test and a doctor can tell.
Too much (general information)	High doses of vitamin E, almost always from supplements, can interfere with blood clotting. EFSA has set a tolerable upper intake level of 300 mg of alpha-tocopherol a day for adults. EFSA did not set this limit for people on medicines that reduce blood clotting. High doses of vitamin E can add to the effect of these medicines. Questions about supplements and medicines belong with a doctor or pharmacist.
Reference intake	EFSA's adequate intake for adults is 13 mg of alpha-tocopherol a day for men and 11 mg a day for women.
Food sources	Vegetable oils such as sunflower, rapeseed and olive oil, nuts, seeds, whole grains and green leafy vegetables.

GENE HIGHLIGHT • CYP4F2

Evidence B

Vitamin E breakdown (CYP4F2)

YOUR GENOTYPE **rs2108622 • CC**

Your file shows no copy of the Met433 version of CYP4F2, like about half of people of European ancestry and most people of African ancestry. Studies found no gene-related shift in vitamin E for this pattern.

Background	CYP4F2 is a liver enzyme that starts the breakdown of vitamin E and vitamin K1. Liver samples with the Met433 version contained less of this enzyme and broke down less vitamin K1. In studies of several thousand people, each copy went with about 0.3 standard deviations lower levels of a vitamin E breakdown product and slightly higher vitamin E in the blood; together with two other genes, it explained less than 2 percent of the differences in vitamin E between people.
Keep in mind	The effect on blood vitamin E is small, and a link with vitamin K levels was only suggestive. Diet matters more, and only a blood test shows your levels.

SOURCES [19297519](#), [21729881](#), [36635386](#), [25411281](#)

02 of 6

Minerals and iron

Section 02 of 6 • 10 traits • 4 gene highlights

Fig. 02 – In this section

10 traits • 4 gene highlights

- 01 Calcium
- 02 Magnesium
- 03 Phosphorus
- 04 Iron
- 05 Ferritin
- 06 Transferrin
- 07 Transferrin saturation

and 3 more • 4 gene highlights

Minerals and iron

Minerals are elements the body cannot make. Calcium, magnesium and phosphorus are needed in larger amounts; iron, zinc, copper and selenium are trace elements needed in small amounts. For calcium, magnesium and phosphorus the body keeps the blood level within a narrow range. A genetic tendency describes a small shift within that range, not your intake. Iron appears as four related blood markers: serum iron, ferritin for iron stores, transferrin for iron transport, and transferrin saturation. Only a blood test can measure any of these levels.

Calcium

Calcium is the most abundant mineral in the body, and almost all of it is in the bones and teeth. Calcium is needed for the maintenance of normal bones and teeth and contributes to normal muscle function, normal neurotransmission and normal blood clotting.

YOUR RESULT

— No meaningful difference  15/20

Independent variants known for this trait: 20; with a call in your file: 15; compared with the average person: 15; carrying at least one copy of the effect allele: 7.

Taken together, your scored variants point to no meaningful difference from the average person (net effect-allele copies +1.4; a total more than one standard deviation from the average counts as a tendency).

NOTE

The body keeps this blood level within a narrow range whatever you eat. A genetic tendency toward a slightly higher or lower level within the usual range says nothing about whether you eat enough calcium.

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs10491003	CT	T	1	0.10	+0.07 unit	24068962, 33462484, 33887147
rs10739679 ~ rs10819178	GG	G	2	0.65	+0.03 unit	34594039
rs11671010 ~ rs1672991	CT	C	1	0.17	+0.05 unit	33462484
rs1260326	CC	C	2	0.59	-0.05 unit	33887147, 34594039, 39024449, 39789286
rs2498323 ~ rs13108218	AG	A	1	0.08	+0.04 unit	33462484
rs4240624	AG	A	1	0.93	+0.05 unit	33887147, 39789286
rs838705 ~ rs838717	AA	A	2	0.59	-0.04 unit	34594039
rs11078597	TT	C	0	0.17	+0.05 unit	33887147, 34594039, 39789286
rs12597024 ~ rs12918968	TT	C	0	0.33	-0.02 unit	33462484
rs13107325	CC	T	0	0.08	-0.0062 mmol/L	33887147, 34594039, 39024449, 39789286
rs1801725 ~ rs73186030	GG	T	0	0.15	+0.19 unit	24068962, 33462484, 33887147, 39414775
rs2762934 ~ rs17216707	GG	A	0	0.22	-0.03 unit	33462484
rs34331440 ~ rs6909201	TT	C	0	0.12	-0.03 unit	33462484
rs77678215 ~ rs2518134	CC	T	0	0.02	-0.04 unit	33462484
rs77732258	GG	T	0	0.01	+0.16 unit	33462484

rs1 ~ rs2: your file lacks the known variant rs2, so rs1, a nearby variant within 50,000 bases that studies also linked with this trait, was read in its place and scored with its own effect.

What the studies measured

The studies measured calcium in blood serum, partly from routine medical records. Hormones, vitamin D and the kidneys keep this level within a narrow range whatever the diet, so it says little about intake.

If intake is too low (general information)

A low calcium intake over many years does not lower the blood level, because the body takes calcium from the bones to keep it steady. Over time this can weaken the bones. A low blood calcium, with tingling or muscle cramps, usually has a medical cause. These signs have many causes, and only a blood test and a doctor can tell.

Too much (general information)	A high calcium intake alone rarely raises the blood level; very high intakes, mostly from supplements, can cause constipation and, in some people, kidney stones. EFSA has set a tolerable upper intake level of 2,500 mg a day for adults.
Reference intake	EFSA's population reference intake for adults is 950 mg a day, and 1,000 mg a day for adults aged 18 to 24.
Food sources	Milk, yogurt and cheese, calcium-rich mineral waters, tofu made with calcium salts, green vegetables such as kale and broccoli, almonds, and small fish eaten with their bones.

GENE HIGHLIGHT · CASR

Evidence A

Calcium set point (CASR)

YOUR GENOTYPE rs1801725 · GG

You have no copy of the Ser986 version, so both of your copies are the Ala986 version, the most common genotype. Studies found that people with this genotype tend to have a blood calcium level close to the population average.

Background CASR makes the calcium-sensing receptor, which the parathyroid glands use to keep blood calcium within a narrow range. Its Ser986 version (T on the forward strand) is linked with a slightly higher calcium set point. In a study of 12,865 people it was the strongest single signal for blood calcium, explaining about 1.3 percent of the differences between people, and in 39,400 Europeans each T copy went with blood calcium about 0.07 mg/dL higher.

Keep in mind This variant says nothing about how much calcium you eat or absorb, or about your bones. Blood calcium is tightly controlled by hormones; only a blood test shows your level.

SOURCES [20661308](#), [24068962](#), [33462484](#)

Magnesium

Magnesium is needed by hundreds of enzymes. Magnesium contributes to normal energy-yielding metabolism, normal muscle function, normal functioning of the nervous system, electrolyte balance and the maintenance of normal bones and teeth.

YOUR RESULT

— No meaningful difference  3/5

Independent variants known for this trait: 5; with a call in your file: 3; compared with the average person: 3; carrying at least one copy of the effect allele: 2.

Taken together, your scored variants point to no meaningful difference from the average person (net effect-allele copies +1.0; a total more than one standard deviation from the average counts as a tendency).

NOTE

The body keeps this blood level within a narrow range whatever you eat. A genetic tendency toward a slightly higher or lower level within the usual range says nothing about whether you eat enough magnesium.

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs13146355	AG	G	1	0.57	-0.01 mmol/L	20700443
rs4072037	CT	C	1	0.45	-0.01 mmol/L	20700443 , 26058915
rs3925584	TT	C	0	0.49	-0.01 mmol/L	20700443

What the studies measured

The studies measured magnesium in blood serum, partly from routine medical records. Less than 1 percent of the body's magnesium is in the blood, and the kidneys keep this level steady, so it says little about intake. Kidney function, diabetes and some medicines also affect it.

**If intake is too low
(general information)**

A low blood magnesium is uncommon in healthy people. It occurs mostly with digestive diseases or long use of some medicines, such as acid-reducing drugs and diuretics. It can cause muscle cramps and tremor. These signs have many causes, and only a blood test and a doctor can tell.

Too much (general information)

Magnesium from food causes no harm in people with healthy kidneys, but supplements can cause diarrhea. For magnesium from supplements, water and fortified foods, EFSA lists a tolerable upper intake level of 250 mg a day for adults. This limit does not count the magnesium naturally present in food.

Reference intake

EFSA's adequate intake for adults is 350 mg a day for men and 300 mg a day for women.

Food sources

Whole grains, pulses, nuts, seeds, green leafy vegetables, cocoa and some mineral waters.

Phosphorus

Phosphorus, present in the body mostly as phosphate, is a building block of bones and teeth, of DNA and of ATP, the molecule cells use to store energy. Phosphorus contributes to normal energy-yielding metabolism, the normal function of cell membranes and the maintenance of normal bones and teeth.

YOUR RESULT

▼ Slightly lower level  12/20

Independent variants known for this trait: 20; with a call in your file: 12; compared with the average person: 12; carrying at least one copy of the effect allele: 5.

Your scored variants point to a tendency toward a slightly lower blood level of phosphate than in the average person of European ancestry (net effect-allele copies -1.9; a total more than one standard deviation from the average counts as a tendency). The effect is small: genes explain only part of the differences between people, and diet, age, health and medicines usually matter more. This report does not measure your level; only a blood test can.

NOTE

The body keeps this blood level within a narrow range whatever you eat. A genetic tendency toward a slightly higher or lower level within the usual range says nothing about whether you eat enough phosphorus.

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs12654812 ~ rs10051765	AG	A	1	0.35	-0.05 unit	33462484
rs17265513	CT	C	1	0.19	+0.03 unit	33462484
rs453639	AA	A	2	0.60	-0.06 unit	39414775, 39789286
rs653178	TT	T	2	0.53	-0.03 unit	33462484
rs7208487	GT	G	1	0.16	-0.04 unit	33462484
rs10774232 ~ rs11611403	AA	G	0	0.22	-0.02 unit	33462484
rs112414002	CC	T	0	0.03	+0.10 unit	33462484
rs12745083 ~ rs7354905	CC	T	0	0.02	+0.10 unit	33462484
rs1801725 ~ rs73186030	GG	T	0	0.15	-0.08 unit	33462484, 39414775
rs72652923	AA	G	0	0.02	+0.12 unit	33462484
rs9469578 ~ rs147516727	CC	T	0	0.07	-0.06 mg/dL	20558539
rs975000	TT	C	0	0.28	+0.06 unit	33462484

rs1 ~ rs2: your file lacks the known variant rs2, so rs1, a nearby variant within 50,000 bases that studies also linked with this trait, was read in its place and scored with its own effect.

What the studies measured

The studies measured phosphate in blood serum. The kidneys and hormones keep it within a range, and it changes with age, the time of day and meals; kidney function has a large influence.

**If intake is too low
(general information)**

A low blood phosphate is rarely caused by diet, because phosphorus is in almost all foods. It usually has a medical cause, such as eating again after starvation or long use of some antacids. A low blood phosphate can cause muscle weakness. These signs have many causes, and only a blood test and a doctor can tell.

Too much (general information)

A high blood phosphate occurs mainly with reduced kidney function, not from diet in healthy people. EFSA has not set a tolerable upper intake level for phosphorus.

Reference intake

EFSA's adequate intake for adults is 550 mg a day.

Food sources

Dairy products, meat, fish, eggs, pulses, nuts and whole grains, and phosphate additives in processed foods and cola drinks.

Iron

Iron is part of hemoglobin, the protein in red blood cells that carries oxygen, and of many enzymes. Iron contributes to normal formation of red blood cells and hemoglobin, to normal oxygen transport in the body and to the normal function of the immune system.

YOUR RESULT▼ Slightly lower level  7/17

Independent variants known for this trait: 17; with a call in your file: 7; compared with the average person: 7; carrying at least one copy of the effect allele: 6.

Your scored variants point to a tendency toward a slightly lower blood level of iron (serum iron) than in the average person of European ancestry (net effect-allele copies -3.5 ; a total more than one standard deviation from the average counts as a tendency). The effect is small: genes explain only part of the differences between people, and diet, age, health and medicines usually matter more. This report does not measure your level; only a blood test can.

NOTE

Serum iron, ferritin, transferrin and transferrin saturation are four different blood markers of iron; doctors usually look at several of them together.

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs12718598	CT	C	1	0.50	+0.03 SD	33536631
rs2228145	AC	C	1	0.36	+0.03 SD	33536631
rs3811647 $\approx$ rs6762719	GG	G	2	0.65	-0.34 unit	39024449
rs7385804	CC	C	2	0.38	-0.06 SD	25352340 , 33536631 , 39024449
rs855791	AA	A	2	0.39	-0.18 unit	25352340 , 28334935 , 33536631 , 39024449
rs9402686 $\approx$ rs9399136	GG	G	2	0.73	-0.05 unit	39024449
rs77262773	CC	T	0	0.01	+0.08 SD	33536631

rs1 $\approx$ rs2: your file lacks the known variant rs2, so rs1, a nearby variant within 50,000 bases that studies also linked with this trait, was read in its place and scored with its own effect.

What the studies measured

The studies measured serum iron, the iron carried in blood by transferrin, which changes during the day and falls with infection. It does not show the body's iron stores, and diet, blood loss, menstruation, pregnancy and blood donation usually matter more than genes.

If intake is too low (general information)

Low iron over a long time can lead to anemia, with tiredness, paleness and shortness of breath on exertion. These signs have many causes, and only a blood test and a doctor can tell.

Too much (general information)

Very high iron intakes almost always come from supplements, not from food. EFSA has not set a tolerable upper intake level for iron. It considers intakes of up to 40 mg a day from all sources safe for adults.

Reference intake

EFSA's population reference intake for adults is 11 mg a day for men and for women after menopause, and 16 mg a day for women before menopause.

Food sources

Meat, fish, pulses, whole grains, nuts, seeds and green leafy vegetables; iron from meat and fish is absorbed best.

HFE iron-loading variants

YOUR GENOTYPE C282Y · rs1800562 · not in your file H63D · rs1799945 · CC S65C · rs1800730 · AA

Your file does not include C282Y, the main HFE variant; some DNA providers leave it out of their raw data files, so this card cannot say anything about it. Your file shows no copies of H63D.

Not a diagnosis. This card reads variants that are also studied in medicine. It describes a genetic background, not a condition, and is no reason to change your diet or any treatment. Only a doctor can diagnose a condition.

Background

HFE helps the liver sense how much iron the body holds and so how much iron the gut absorbs. Two copies of the C282Y variant are the most common genetic basis of hereditary hemochromatosis, a condition in which iron slowly builds up; H63D and S65C have much smaller effects. In large studies each C282Y copy raised transferrin saturation, a blood measure of how loaded the iron transport protein is, by about half of the usual spread between people. In a UK study of about 451,000 adults followed for 13 years on average, an estimated 56 in 100 men and 41 in 100 women with two C282Y copies had been diagnosed with hemochromatosis by age 80, against about 5 in 100 men with one C282Y and one H63D copy.

Keep in mind

This result is not a diagnosis and does not show your iron level, which also depends on diet, blood loss and health. If you have symptoms or questions, talk to a doctor; only a blood test of iron levels (transferrin saturation and ferritin) can tell.

SOURCES [38479735](#), [15858186](#), [18199861](#), [33536631](#)

Iron and the TMPRSS6 gene

YOUR GENOTYPE rs855791 · AA

You have two copies of the Val736 version. Studies found that people with this genotype tend to have slightly lower blood iron, transferrin saturation and hemoglobin than people with other genotypes; most still have values in the usual range.

Background

TMPRSS6 makes matriptase-2, a liver enzyme that holds back the iron hormone hepcidin and so helps set how much iron the gut absorbs. Lab and population studies suggest that the Val736 version (A on the forward strand that DNA files use, T in many papers) restrains hepcidin slightly less well than the Ala736 version (G). In up to about 160,000 Europeans, each Val736 copy went with blood iron and transferrin saturation lower by about one sixth of the usual spread between people, and in 16,001 people of European and Indian Asian ancestry with hemoglobin 0.13 g/dL lower per copy.

Keep in mind

Diet, menstruation, pregnancy, blood donation and health affect iron far more than this variant. Only a blood test (for example ferritin) shows your iron status.

SOURCES [19820699](#), [19820698](#), [21873547](#), [33536631](#)

Ferritin

Ferritin is the protein that stores iron inside cells, mainly in the liver. The small amount of ferritin in blood reflects how much iron the body has stored.

YOUR RESULT

▼ Slightly lower level  7/20

Independent variants known for this trait: 20; with a call in your file: 7; compared with the average person: 7; carrying at least one copy of the effect allele: 3.

Your scored variants point to a tendency toward a slightly lower blood level of ferritin than in the average person of European ancestry (net effect-allele copies -2.4 ; a total more than one standard deviation from the average counts as a tendency). The effect is small: genes explain only part of the differences between people, and diet, age, health and medicines usually matter more. This report does not measure your level; only a blood test can.

NOTE

Ferritin and transferrin move in opposite directions: when iron stores fall, ferritin goes down and transferrin goes up. A genetic tendency toward a slightly lower ferritin level is not the same as having low iron stores.

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs744653 $\approx$ rs12693541	TT	T	2	0.84	-0.09 unit	25352340
rs855791	AA	A	2	0.39	-0.04 SD	25352340 , 33536631
rs9921222	CC	C	2	0.58	$+0.03$ SD	33536631
rs1260326	CC	T	0	0.41	$+0.03$ SD	33536631
rs1542752	CC	T	0	0.16	$+0.03$ SD	33536631
rs17476364	TT	C	0	0.10	$+0.04$ SD	33536631
rs34523089	CC	T	0	0.16	$+0.07$ SD	33536631

rs1 $\approx$ rs2: your file lacks the known variant rs2, so rs1, a nearby variant within 50,000 bases that studies also linked with this trait, was read in its place and scored with its own effect.

What the studies measured

The studies measured ferritin in blood serum, partly from routine medical records. Besides genes, it depends on iron intake, blood loss, menstruation, pregnancy, age and sex, and it rises with inflammation, infection, liver problems, alcohol and excess body weight.

A low level (general information)

In a blood test, a low ferritin usually means that the body's iron stores are small. A low ferritin has many causes, and only a blood test and a doctor can tell.

A high level (general information)

In a blood test, a high ferritin is most often linked with inflammation, liver problems, alcohol or excess body weight, and less often with large iron stores. Only a doctor can tell which cause applies to one person.

Food sources

Ferritin is made by the body; the iron it stores comes from foods such as meat, fish, pulses, whole grains and green vegetables.

Transferrin

Transferrin is the protein that carries iron in the blood to the bone marrow and other tissues. The liver makes more of it when the body's iron stores are low.

YOUR RESULT

— No meaningful difference  7/7

Independent variants known for this trait: 7; with a call in your file: 7; compared with the average person: 7; carrying at least one copy of the effect allele: 7.

Taken together, your scored variants point to no meaningful difference from the average person (net effect-allele copies +0.9; a total more than one standard deviation from the average counts as a tendency).

NOTE

Transferrin and ferritin move in opposite directions, so a genetic tendency toward a higher transferrin level is not the same as having low iron stores.

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs174577	AC	A	1	0.36	+0.06 unit	25352340
rs4921915	AA	A	2	0.76	+0.08 unit	25352340
rs6486121	CT	T	1	0.66	−0.05 unit	25352340
rs744653	TT	T	2	0.84	+0.07 unit	25352340
rs8177240	TT	T	2	0.65	−0.38 unit	25352340
rs855791	AA	A	2	0.39	+0.04 unit	25352340
rs9990333	TT	T	2	0.47	−0.05 unit	25352340

What the studies measured

The studies measured transferrin in blood serum. Besides genes and iron stores, it rises in pregnancy and with estrogen-containing contraceptives, and falls with inflammation, liver disease and poor nutrition.

A high level (general information)

In a blood test, transferrin is often high when the body's iron stores are low, and it also rises in pregnancy and with estrogen-containing contraceptives. A high transferrin has many causes, and only a blood test and a doctor can tell.

Food sources

Transferrin is made in the liver, not eaten; the iron it carries comes from foods such as meat, fish, pulses and whole grains.

Transferrin saturation

Transferrin saturation is the share of transferrin's iron-binding sites that are carrying iron. It shows how much iron is available in the blood for making red blood cells and for other tissues.

YOUR RESULT

▼ Slightly lower level  4/7

Independent variants known for this trait: 7; with a call in your file: 4; compared with the average person: 4; carrying at least one copy of the effect allele: 4.

Your scored variants point to a tendency toward a slightly lower transferrin saturation than in the average person of European ancestry (net effect-allele copies −2.3; a total more than one standard deviation from the average counts as a tendency). The effect is small: genes explain only part of the differences between people, and diet, age, health and medicines usually matter more. This report does not measure your level; only a blood test can.

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs4895441 ~ rs9399136	AA	A	2	0.73	−0.06 unit	39024449
rs7385804	CC	C	2	0.38	−0.06 SD	25352340 , 33536631
rs8177240 ~ rs4854760	TT	T	2	0.65	+0.10 unit	25352340

rs1 = rs2: your file lacks the known variant rs2, so rs1, a nearby variant within 50,000 bases that studies also linked with this trait, was read in its place and scored with its own effect.

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs855791	AA	A	2	0.39	-0.17 SD	25352340 , 33536631 , 39024449

rs1 ≈ rs2: your file lacks the known variant rs2, so rs1, a nearby variant within 50,000 bases that studies also linked with this trait, was read in its place and scored with its own effect.

What the studies measured	The studies measured transferrin saturation, calculated from serum iron and the blood's iron-binding capacity, partly from routine medical records. Like serum iron, it changes during the day, after meals and with inflammation.
A low level (general information)	In a blood test, a low transferrin saturation suggests that too little iron reaches the bone marrow, for example when iron stores are low. A low result has many causes, and only a blood test and a doctor can tell.
A high level (general information)	In a blood test, transferrin saturation can be higher for a few hours after a meal or after iron supplements, and some medical conditions also raise it. A high result has many causes, and only a blood test and a doctor can tell.
Food sources	Transferrin saturation is a blood measure, not a nutrient; it reflects iron from foods such as meat, fish, pulses and whole grains.

Zinc

Zinc is a trace element needed by many enzymes and proteins. It contributes to normal DNA and protein synthesis and to the normal function of the immune system. Zinc also contributes to the maintenance of normal skin, hair, nails, bones and vision.

YOUR RESULT

— No meaningful difference  3/3

Independent variants known for this trait: 3; with a call in your file: 3; compared with the average person: 3; carrying at least one copy of the effect allele: 2.

Taken together, your scored variants point to no meaningful difference from the average person (net effect-allele copies +0.9; a total more than one standard deviation from the average counts as a tendency).

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs1532423	AG	A	1	0.37	+0.18 unit	23720494
rs4826508	T	T	1	0.73	+0.21 unit	23720494
rs2120019	TT	C	0	0.21	-0.29 unit	23720494

What the studies measured	This item rests on one study of about 2,600 adults in Australia, which measured zinc in red blood cells. Red blood cells live for about four months, so their zinc changes slowly. Zinc in blood serum, by contrast, shifts with meals, the time of day and inflammation. Besides genes, diet also affects the level.
If intake is too low (general information)	Low zinc intake over a long time can affect growth in children, the skin, the sense of taste and wound healing. These signs have many causes, and only a blood test and a doctor can tell.
Too much (general information)	Long-term high intakes, almost always from supplements, can lead to a lack of copper. EFSA lists a tolerable upper intake level of 25 mg a day for adults.
Reference intake	EFSA's population reference intake for adults is 9.4 to 16.3 mg a day for men and 7.5 to 12.7 mg a day for women. The higher figures apply to diets rich in phytate, a plant compound that reduces zinc absorption.
Food sources	Meat, shellfish, cheese, eggs, whole grains, pulses, nuts and seeds; zinc from animal foods is absorbed better.

Copper

Copper is a trace element that is part of several enzymes, including some antioxidant enzymes. Copper contributes to normal iron transport in the body, the maintenance of normal connective tissues and the protection of cells from oxidative stress.

YOUR RESULT

☐ Too few variants for a tendency  2/5

Independent variants known for this trait: 5; with a call in your file: 2; compared with the average person: 2; carrying at least one copy of the effect allele: 0.

Your file measures 2 of the 5 variants known for the blood level of copper. A tendency needs at least three, so this report gives none; the variants measured are listed below.

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs2769264	TT	G	0	0.16	+0.31 unit	23720494
rs4766566	TT	C	0	0.73	-0.19 unit	35501403

What the studies measured

Two studies measured copper: one in the red blood cells of about 2,600 adults in Australia, and one in the blood serum of about 2,500 people in China. In serum, most copper is bound to ceruloplasmin, a carrier protein, and the level rises with inflammation, pregnancy and estrogen-containing contraceptives.

If intake is too low (general information)

A lack of copper is rare. It has been seen after stomach surgery or long-term high zinc intake, and it can cause anemia and nerve problems. These signs have many causes, and only a blood test and a doctor can tell.

Too much (general information)

Too much copper, mostly from supplements or contaminated water, can upset the stomach and, over the long term, harm the liver. EFSA lists a tolerable upper intake level of 5 mg a day for adults.

Reference intake

EFSA's adequate intake for adults is 1.6 mg a day for men and 1.3 mg a day for women.

Food sources

Liver, shellfish, nuts, seeds, pulses, whole grains and cocoa.

Selenium

Selenium is a trace element built into selenoproteins, enzymes that protect cells and help activate thyroid hormones. Selenium contributes to normal thyroid function, the normal function of the immune system and normal spermatogenesis, the making of sperm. It also contributes to the maintenance of normal hair and nails and to the protection of cells from oxidative stress.

YOUR RESULT

☐ Too few variants for a tendency  1/1

Independent variants known for this trait: 1; with a call in your file: 1; compared with the average person: 1; carrying at least one copy of the effect allele: 0.

Only one variant is known for the selenium level in blood and toenails, and your file measures it. One variant is not enough for a tendency, so this report gives none; the variant is listed below.

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs921943	CC	T	0	0.30	+0.25 unit	23720494

What the studies measured

The studies measured selenium in blood and, in one study, in toenails, which reflect intake over several months. Besides genes, the level depends mainly on diet, and the selenium content of food depends on the soil where it was grown.

If intake is too low (general information)

A lack of selenium is rare in Europe. Severe lack, seen in regions with very poor soils, affects the heart muscle and the joints. These signs have many causes, and only a blood test and a doctor can tell.

Too much (general information)

Too much selenium, mostly from supplements or from eating many Brazil nuts, can cause hair and nail loss and a garlic smell on the breath. EFSA has set a tolerable upper intake level of 255 micrograms a day for adults.

Reference intake	EFSA's adequate intake for adults is 70 micrograms a day.
Food sources	Fish, seafood, meat, offal, eggs and Brazil nuts; the selenium in cereals and vegetables depends on the soil.

GENE HIGHLIGHT · DMGDH / BHMT

Evidence B

Selenium and the DMGDH region

YOUR GENOTYPE rs921943 · CC

You have no copy of the T version. Studies found that people with this genotype tend to have slightly lower blood selenium than people who carry it.

Background

A common variant in the DMGDH and BHMT gene region, which handles sulfur-containing amino acids and possibly their selenium counterparts, is linked with selenium levels. The T version (forward strand) went with higher selenium in red blood cells and whole blood in Australian and UK studies, and with higher toenail selenium, a longer-term measure, in 4,162 US adults. The variant explains only about 1 percent of the differences between people.

Keep in mind

The selenium content of food depends mostly on the soil where it was grown, and diet matters far more than this variant. Only a blood test shows your level.

SOURCES

[23720494](#), [25343990](#)

03

of 6

Fatty acids and homocysteine

Section 03 of 6 • 5 traits • 3 gene highlights

Fig. 03 – In this section		5 traits • 3 gene highlights
01	Omega-3 fatty acids	
02	DHA	
03	Omega-6 to omega-3 ratio	
04	Linoleic acid (omega-6)	
05	Homocysteine	
and 3 gene highlights		

Fatty acids and homocysteine

This section covers omega-3 and omega-6 fatty acids measured in blood, and homocysteine. Blood fatty acids mostly reflect what people eat, especially fish and vegetable oils. Among genes, variants in the FADS genes, which help the body turn shorter fatty acids into longer ones, have the largest effects. Homocysteine is not a nutrient: it is used in research as a marker of how the body uses folate, vitamin B12 and vitamin B6. Only a blood test can measure these levels.

Omega-3 fatty acids

Omega-3 fatty acids are polyunsaturated fats. The body cannot make alpha-linolenic acid (ALA), the plant omega-3, and turns only a small part of it into EPA and DHA, the omega-3 fatty acids of fish. ALA contributes to the maintenance of normal blood cholesterol levels, and EPA and DHA contribute to the normal function of the heart.

YOUR RESULT

No meaningful difference  9/20

Independent variants known for this trait: 20; with a call in your file: 9; compared with the average person: 9; carrying at least one copy of the effect allele: 2.

Taken together, your scored variants point to no meaningful difference from the average person (net effect-allele copies -1.4; a total more than one standard deviation from the average counts as a tendency).

NOTE

Variants in the FADS genes have the largest genetic effects on omega-3 and omega-6 fatty acids in blood. These genes help the body turn shorter fatty acids into longer ones.

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs174546	CT	T	1	0.35	-0.31 unit	38448586
rs62466318	CC	C	2	0.80	+0.04 unit	35213538 , 36764567 , 39091897
rs1260326	CC	T	0	0.41	+0.04 unit	35213538 , 36764567 , 39091897
rs1532085	GG	A	0	0.36	+0.08 unit	35213538 , 39091897
rs2229738	CC	T	0	0.09	-0.16 unit	35213538 , 38448586 , 39091897
rs445925 ≈ rs190921611	GG	A	0	0.09	+0.07 unit	39091897
rs499974 ≈ rs600626	CC	A	0	0.17	-0.06 unit	36764567
rs58542926	CC	T	0	0.07	-0.13 unit	35213538 , 36764567 , 39091897
rs9332238 ≈ rs61886804	GG	A	0	0.20	+0.06 unit	39091897

rs1 ≈ rs2: your file lacks the known variant rs2, so rs1, a nearby variant within 50,000 bases that studies also linked with this trait, was read in its place and scored with its own effect.

What the studies measured	The studies measured omega-3 fatty acids as a share of all fatty acids in blood plasma, mostly in the UK Biobank. Besides genes, diet matters most, especially oily fish and fish-oil supplements.
Too much (general information)	EFSA has not set a tolerable upper intake level for EPA and DHA. It considers long-term supplemental intakes of up to about 5 g a day of EPA and DHA combined to raise no safety concern for adults.
Reference intake	EFSA's adequate intakes for adults are 250 mg a day of EPA and DHA combined and 0.5 percent of energy intake from ALA.
Food sources	Oily fish such as salmon, mackerel, herring and sardines provide EPA and DHA; rapeseed oil, linseed, walnuts and chia seeds provide ALA.

Omega-3 and omega-6 conversion (FADS1 and FADS2)

YOUR GENOTYPE **rs174547 · CT**

You have one copy of each version. Studies found that people with this genotype tend to have blood fatty acid levels between those of the two other genotypes.

Background

FADS1 and FADS2 make the desaturase enzymes that turn the plant omega-3 and omega-6 fats (alpha-linolenic and linoleic acid) into long-chain forms such as EPA and arachidonic acid. Two common versions of this gene region differ strongly in how efficiently they do this. In 8,631 adults, each copy of the less active version (C at rs174547) went with arachidonic acid about 1.7 percentage points lower as a share of blood fatty acids, and in 8,866 adults with slightly less EPA and DPA (about 0.08 points per copy) and slightly more alpha-linolenic acid. The region explains about 9 percent of the differences between people in DPA.

Keep in mind

Blood omega-3 levels depend much more on what you eat, especially fish and seafood, than on this genotype; in the main study, fish intake was linked with blood EPA and DHA in the same way whatever the genotype. Only a blood fatty acid test shows your levels.

SOURCES [21829377](#), [24823311](#), [22503634](#)

DHA

Docosahexaenoic acid (DHA) is a long-chain omega-3 fatty acid and a major building block of the brain and of the retina of the eye. DHA contributes to the maintenance of normal brain function and of normal vision.

YOUR RESULT ▼ Slightly lower level  10/20

Independent variants known for this trait: 20; with a call in your file: 10; compared with the average person: 10; carrying at least one copy of the effect allele: 7.

Your scored variants point to a tendency toward a slightly lower share of DHA among blood fatty acids than in the average person of European ancestry (net effect-allele copies -2.0; a total more than one standard deviation from the average counts as a tendency). The effect is small: genes explain only part of the differences between people, and diet, age, health and medicines usually matter more. This report does not measure your level; only a blood test can.

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs10455872 ~ rs55730499	AA	A	2	0.93	-0.08 unit	35213538 , 36764567
rs16891156	AA	A	2	0.98	-0.10 unit	35213538
rs174528	CT	C	1	0.39	-0.25 unit	35213538 , 36764567
rs2144300 ~ rs2281721	CC	C	2	0.40	-0.03 unit	36764567
rs499974 ~ rs600626	CC	C	2	0.83	+0.04 unit	35213538
rs676210	AG	A	1	0.22	+0.04 unit	35213538 , 36764567 , 39091897
rs72836561	CC	C	2	0.96	+0.07 unit	35213538 , 39091897
rs1532085	GG	A	0	0.36	+0.06 unit	35213538
rs2229738	CC	T	0	0.09	-0.13 unit	35213538 , 36764567 , 38448586 , 39091897
rs439401	CC	T	0	0.38	+0.03 unit	35213538 , 39091897

rs1 ~ rs2: your file lacks the known variant rs2, so rs1, a nearby variant within 50,000 bases that studies also linked with this trait, was read in its place and scored with its own effect.

What the studies measured

The studies measured the share of DHA among all fatty acids in blood plasma, mostly in the UK Biobank. Besides genes, fish intake and fish-oil supplements have the largest influence.

Too much (general information)

EFSA has not set a tolerable upper intake level for EPA and DHA. It considers long-term supplemental intakes of up to about 5 g a day of EPA and DHA combined to raise no safety concern for adults.

Reference intake

EFSA's adequate intake for adults is 250 mg a day of EPA and DHA combined.

Food sources

Oily fish and other seafood, and oils made from algae; eggs and meat contain small amounts.

GENE HIGHLIGHT · ELOVL2

Evidence B

DHA and the ELOVL2 gene

YOUR GENOTYPE rs3734398 · CT

You have one copy of the C version. Studies found that people with this genotype tend to have DHA and DPA levels between those of the two other genotypes.

Background ELOVL2 makes an elongase enzyme that lengthens omega-3 fats on the last steps toward DHA. In 8,866 adults of European ancestry, each C copy at rs3734398 went with slightly more DPA in blood, the step before DHA (0.04 percentage points of total fatty acids per copy), and the same gene region went with slightly less DHA (0.11 points per copy, against average DHA levels of 2 to 5 percent). The region explains about 3 percent of the differences between people in DPA and less than 1 percent in DHA.

Keep in mind The link was clearer in people of European ancestry than in African, Chinese or Hispanic samples. Diet, especially fish and seafood, matters far more for blood DHA; only a blood test shows your level.

SOURCES 21829377, 37253714

Omega-6 to omega-3 ratio

This ratio compares the amount of omega-6 fatty acids in blood with the amount of omega-3 fatty acids. A higher ratio means relatively more omega-6 or less omega-3, and it mostly reflects how much oily fish and which vegetable oils people eat.

YOUR RESULT

No meaningful difference 9/20

Independent variants known for this trait: 20; with a call in your file: 9; compared with the average person: 9; carrying at least one copy of the effect allele: 5.

Taken together, your scored variants point to no meaningful difference from the average person (net effect-allele copies +1.3; a total more than one standard deviation from the average counts as a tendency).

NOTE

EFSA has not set a reference value for this ratio; it sets separate adequate intakes for the individual fatty acids.

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs174528	CT	T	1	0.61	-0.34 unit	35213538
rs2229738	CC	C	2	0.91	-0.13 unit	35213538, 39091897
rs58542926	CC	C	2	0.93	-0.14 unit	35213538, 36764567, 39091897
rs62466318	CC	C	2	0.80	-0.06 unit	35213538, 36764567, 39091897
rs9332238	GG	G	2	0.80	+0.03 unit	35213538, 39091897
rs1260326	CC	T	0	0.41	-0.07 unit	35213538, 36764567, 39091897
rs1532085	GG	A	0	0.36	-0.09 unit	35213538, 39091897

rs1 = rs2: your file lacks the known variant rs2, so rs1, a nearby variant within 50,000 bases that studies also linked with this trait, was read in its place and scored with its own effect.

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs445925	GG	A	0	0.09	-0.04 unit	39091897
rs499974 ≈ rs673335	CC	A	0	0.17	+0.06 unit	36764567

rs1 ≈ rs2: your file lacks the known variant rs2, so rs1, a nearby variant within 50,000 bases that studies also linked with this trait, was read in its place and scored with its own effect.

What the studies measured

The studies measured both groups of fatty acids in blood plasma, mostly in the UK Biobank, and divided one by the other. Besides genes, diet, especially fish and vegetable oils, has a large influence.

Food sources

Omega-6 fatty acids come mainly from sunflower, corn and soybean oil, nuts and seeds; omega-3 fatty acids from oily fish, rapeseed oil, linseed and walnuts.

Linoleic acid (omega-6)

Linoleic acid is the main omega-6 fatty acid, a type of polyunsaturated fat. It is essential: the body cannot make it and needs it from food. Linoleic acid contributes to the maintenance of normal blood cholesterol levels.

YOUR RESULT

— No meaningful difference  12/20

Independent variants known for this trait: 20; with a call in your file: 12; compared with the average person: 12; carrying at least one copy of the effect allele: 8.

Taken together, your scored variants point to no meaningful difference from the average person (net effect-allele copies +0.1; a total more than one standard deviation from the average counts as a tendency).

NOTE

Because this is a share of all fatty acids, it depends less on how much fat is in the blood than an absolute amount would. It still shifts with the overall mix of fats in the diet and in blood.

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs10195252	CT	C	1	0.44	+0.03 unit	38448586 , 40307439
rs10401969	TT	T	2	0.93	-0.06 unit	38448586
rs10455872	AA	A	2	0.93	-0.09 unit	38448586
rs174528 ≈ rs174564	CT	T	1	0.61	-0.17 unit	35213538 , 36764567
rs2972143 ≈ rs2972137	AA	A	2	0.37	+0.03 unit	35213538
rs4719841	AG	G	1	0.39	-0.03 unit	40307439
rs4810479	CT	C	1	0.29	-0.04 unit	35213538 , 39091897
rs738408 ≈ rs3747207	CC	C	2	0.77	-0.04 unit	35213538 , 36764567 , 39091897
rs116843064	GG	A	0	0.03	+0.10 unit	35213538 , 36764567 , 38448586 , 39091897 , 40307439
rs1260326	CC	T	0	0.41	-0.09 unit	35213538 , 36764567 , 38448586 , 39091897 , 40307439
rs17410962	GG	A	0	0.16	+0.07 unit	40307439
rs247616 ≈ rs11508026	CC	T	0	0.29	+0.04 unit	38448586

rs1 ≈ rs2: your file lacks the known variant rs2, so rs1, a nearby variant within 50,000 bases that studies also linked with this trait, was read in its place and scored with its own effect.

What the studies measured

The studies measured linoleic acid, the main omega-6 fatty acid, as a share of all fatty acids in blood plasma, mostly in the UK Biobank. Besides genes, the share depends mainly on diet, especially vegetable oils, nuts and seeds.

Reference intake

EFSA's adequate intake for adults is 4 percent of energy intake from linoleic acid.

Food sources

Sunflower, corn, soybean and other seed oils, nuts, seeds and foods made with these oils.

Homocysteine

Homocysteine is an amino acid that the body makes when it uses methionine, an amino acid from dietary protein. It is recycled or broken down with the help of folate, vitamin B12 and vitamin B6, and these three vitamins contribute to normal homocysteine metabolism.

YOUR RESULT

▼ Slightly lower level  11/12

Independent variants known for this trait: 12; with a call in your file: 11; compared with the average person: 11; carrying at least one copy of the effect allele: 6.

Your scored variants point to a tendency toward a slightly lower blood level of homocysteine than in the average person of European ancestry (net effect-allele copies -3.0 ; a total more than one standard deviation from the average counts as a tendency). The effect is small: genes explain only part of the differences between people, and diet, age, health and medicines usually matter more. This report does not measure your level; only a blood test can.

NOTE

Homocysteine is not a nutrient. Research uses it as a marker of one-carbon metabolism, the set of reactions that need folate, vitamin B12 and vitamin B6. A genetic tendency toward a higher level says nothing about whether you get enough of these vitamins.

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs1047891	AC	A	1	0.30	+0.09 unit	20154341 , 23824729
rs12780845	AA	A	2	0.66	+0.05 unit	23824729
rs2251468	AA	A	2	0.64	-0.05 unit	23824729
rs2275565	TT	T	2	0.20	-0.05 unit	23824729
rs234709	CT	T	1	0.40	-0.07 unit	23824729
rs9369898	AG	A	1	0.59	+0.04 unit	23824729
rs154657	GG	A	0	0.48	+0.10 unit	23824729
rs42648	GG	A	0	0.42	-0.04 unit	23824729
rs4660306	CC	T	0	0.30	+0.04 unit	23824729
rs7130284	CC	T	0	0.08	-0.12 unit	23824729
rs838133	GG	A	0	0.43	+0.04 unit	23824729

What the studies measured

The studies measured total homocysteine in blood plasma. Most findings come from one large analysis of about 44,000 people of European ancestry. Besides genes, the level depends on folate and vitamin B12 intake, kidney function, age, sex, smoking, coffee and some medicines.

A high level (general information)

In a blood test, a high homocysteine is most often linked with low folate or vitamin B12 levels, reduced kidney function, older age or some medicines. Only a doctor can tell which cause applies to one person.

Food sources

Homocysteine is not eaten; the body makes it from methionine, which comes with protein from foods such as meat, fish, eggs, dairy products and pulses.

MTHFR C677T, folate and homocysteine

YOUR GENOTYPE **rs1801133 • AG**

Your file shows one copy of the MTHFR 677T variant, like nearly half of people of European ancestry. Studies found that people with one copy tend to have about 7 percent lower blood folate and only slightly higher homocysteine than people with none. This is not a diagnosis, and the difference is small.

Not a diagnosis. This card reads a variant that is also studied in medicine. It describes a genetic background, not a condition, and is no reason to change your diet or any treatment. Only a doctor can diagnose a condition.

Background	MTHFR makes an enzyme that turns folate into the form the body uses to recycle homocysteine. The 677T version makes this enzyme less stable. In a CDC review of studies in women, those with two copies had about 13 percent lower serum folate and 16 percent lower red-cell folate than those with none, and a genome-wide study of 44,147 people found higher homocysteine with each copy. Across 59,995 people, the homocysteine gap between two copies and none was about 3 micromol/L in low-folate regions of Asia but close to zero where foods are fortified with folic acid.
Keep in mind	Medical genetics guidelines (ACMG 2013, reaffirmed 2020) say MTHFR genotyping has little medical use, and the US CDC states that people with this variant can process all types of folate, including folic acid. If you have symptoms or questions, talk to a doctor; only a blood test can tell your folate and homocysteine levels.

SOURCES [25788000](#), [23824729](#), [21803414](#), [23288205](#)

04 of 6

Food and drink habits

Section 04 of 6 • 5 traits • 4 gene highlights

Fig. 04 – In this section		5 traits • 4 gene highlights
01	Coffee intake	
02	Alcohol intake	
03	Protein intake	
04	Fat intake	
05	Carbohydrate intake	
and 4 gene highlights		

Food and drink habits

This section is about habits rather than blood levels. The genetic studies behind it asked large groups of adults how much coffee and alcohol they drink and what they eat. They used questionnaires, 24-hour diet recalls and diet records. From these answers, researchers worked out what share of energy came from protein, fat and carbohydrates. What people report partly reflects taste, culture, price, memory and how well they tolerate a drink. So a genetic tendency here describes what people with similar variants tended to report. It is not a measure of your diet and not a suggestion to change it.

Coffee intake

In most European countries, coffee is the main source of caffeine for adults, and it also contains many other plant compounds. Caffeine is a stimulant that the liver breaks down, mostly with the enzyme CYP1A2.

YOUR RESULT

▲ Slightly more  9/15

Independent variants known for this trait: 15; with a call in your file: 9; compared with the average person: 9; carrying at least one copy of the effect allele: 6.

Your scored variants point to a tendency toward a slightly higher reported coffee intake than in the average person of European ancestry (net effect-allele copies +2.5; a total more than one standard deviation from the average counts as a tendency). This describes what people with similar variants tended to report in the studies; it is not a measure of your diet and not a reason to change it.

NOTE

Some variants linked with drinking more coffee lie near the CYP1A2 and AHR genes, which affect how fast the liver breaks down caffeine. This report does not measure how fast your body breaks down caffeine or any medicine. EFSA gives about 90 mg of caffeine for a 200 ml cup of filter coffee.

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs1260326	CC	C	2	0.59	+0.03 cups per day	31837886
rs17685	AG	A	1	0.29	+0.08 unit	25288136 , 31837886
rs382140	AA	A	2	0.19	+0.08 unit	21876539
rs4410790	CT	T	1	0.38	-0.10 unit	21490707 , 25288136 , 31837886 , 40436827
rs7800944	CT	T	1	0.74	-0.05 unit	25288136
rs9902453	AG	A	1	0.57	-0.03 unit	25288136
rs1481012	AA	G	0	0.09	-0.07 cups per day	31837886
rs2472297	CC	T	0	0.21	+0.14 unit	21357676 , 25288136 , 31837886
rs476828	TT	C	0	0.25	+0.04 cups per day	31837886

What the studies measured

The studies counted the cups of coffee per day that adults reported in questionnaires. What people report also reflects taste, habit, culture, price and how well they tolerate caffeine.

Too much (general information)

Too much caffeine can cause restlessness, a fast heartbeat and poor sleep. EFSA considers habitual caffeine intakes of up to 400 mg a day from all sources to raise no safety concern for healthy adults. For pregnant and breastfeeding women, its figure is 200 mg a day.

Caffeine level and coffee drinking

YOUR GENOTYPE CYP1A1-CYP1A2 · rs2472297 · CC AHR · rs4410790 · CT

Copies linked with lower caffeine levels: 1 of 4

You carry fewer of these variants than most people of European ancestry. Studies found that people with fewer copies tend to have slightly higher caffeine levels in the blood after coffee and to drink a little less coffee. The difference is small, a fraction of a cup a day.

Background	Most caffeine is broken down in the liver by the enzyme CYP1A2, and the AHR protein turns CYP1A2 up. The T allele of rs2472297, between CYP1A1 and CYP1A2, and the C allele of rs4410790, near AHR, were linked in studies of up to 9,876 people to lower caffeine levels in the blood (Cornelis 2016), and in studies of up to 91,462 coffee drinkers to about 0.1 to 0.2 more cups of coffee a day per copy (Sulem 2011; Coffee and Caffeine Genetics Consortium 2015); in the largest later study, of 448,204 people, the difference was about 0.06 cups (Cole 2020). People seem to adjust how much coffee they drink to how much caffeine stays in their blood.
Keep in mind	The effect is small: smoking, hormones, some medicines, age and liver health change caffeine levels in the blood far more than these variants do. rs2472297 is almost absent in people of East Asian ancestry, so the count tells them less.

SOURCES [27702941](#), [21357676](#), [25288136](#), [32193382](#)

Alcohol intake

Alcohol (ethanol) is not a nutrient, and the body does not need it. It provides energy, and the liver breaks it down, first into acetaldehyde and then into acetate.

YOUR RESULT

▼ Slightly less  10/20

Independent variants known for this trait: 20; with a call in your file: 10; compared with the average person: 10; carrying at least one copy of the effect allele: 6.

Your scored variants point to a tendency toward a slightly lower reported alcohol intake than in the average person of European ancestry (net effect-allele copies -1.9; a total more than one standard deviation from the average counts as a tendency). This describes what people with similar variants tended to report in the studies; it is not a measure of your diet and not a reason to change it.

NOTE

Some of the variants with the largest effects change how fast the body processes alcohol and acetaldehyde. These variants are much more common in East Asian than in European populations.

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs11940694	AG	G	1	0.61	+0.02 unit	27911795 , 28937693 , 30643251 , 30643258 , 31358974 , 32193382 , 36477530
rs1229984	CC	C	2	0.97	+0.08 unit	28485404 , 30643251 , 30643258 , 30679032 , 32193382 , 36477530 , 40436827
rs1260326	CC	C	2	0.59	+0.02 unit	28937693 , 30643251 , 30643258 , 30679032

rs1 = rs2: your file lacks the known variant rs2, so rs1, a nearby variant within 50,000 bases that studies also linked with this trait, was read in its place and scored with its own effect.

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
						31358974, 32193382, 36477530
rs1421085 ~ rs11642015	CC	C	2	0.43	-0.01 unit	31358974, 36477530
rs7132908	AG	A	1	0.36	-0.01 unit	30643258, 36477530
rs838133 ~ rs2638282	GG	G	2	0.57	-0.01 unit	36477530
rs1217105 ~ rs10113566	TT	G	0	0.70	+0.01 unit	36477530
rs13107325	CC	T	0	0.08	-0.03 unit	30643251, 30643258, 30679032, 31358974, 36477530
rs362307	CC	T	0	0.06	-0.02 unit	36477530
rs4916723	AA	C	0	0.41	-0.01 unit	30643251, 30643258, 31358974, 36477530

rs1 ~ rs2: your file lacks the known variant rs2, so rs1, a nearby variant within 50,000 bases that studies also linked with this trait, was read in its place and scored with its own effect.

What the studies measured

The studies counted the alcoholic drinks per week that adults reported in questionnaires, mostly in people of European ancestry, and in some studies how often people drink. What people report also reflects culture, religion, social habits, age, sex and income, and people tend to report less than they drink.

Too much (general information)

The health risks of alcohol rise with the amount drunk, and national health authorities publish low-risk drinking guidelines.

GENE HIGHLIGHT • ALDH2

Evidence A

Alcohol flush

YOUR GENOTYPE rs671 • GG

Your file shows no copy of the ALDH2 variant rs671 A, so you have the usual ALDH2 enzyme and this variant gives no tendency to flush after alcohol. This is the case for almost everyone without East Asian ancestry.

Not a diagnosis. This card reads a variant that is also studied in medicine. It describes a genetic background, not a condition, and is no reason to change your diet or any treatment. Only a doctor can diagnose a condition.

Background

ALDH2 is the main enzyme that clears acetaldehyde, the substance alcohol is first turned into. The A allele of rs671 (Glu504Lys) makes an enzyme that barely works and also hampers the normal copy: in liver samples, one copy left about 17 percent of normal ALDH2 activity, and with two copies the activity was too low to measure (Lai 2014). About 8 in 100 people worldwide, around 540 million, carry it, nearly all of East Asian ancestry, and about 36 in 100 East Asians flush after drinking (Brooks 2009).

Keep in mind

The variant is very rare outside East Asia, and this result says nothing about how much alcohol is safe for anyone. It is not a medical diagnosis. If you have symptoms or questions, talk to a doctor.

SOURCES 23909789, 19320537, 24382882, 17718394

GENE HIGHLIGHT • ADH1B

Evidence A

Alcohol breakdown

YOUR GENOTYPE rs1229984 • CC

Your file shows no copy of the faster ADH1B variant rs1229984 T. You have the usual form of this enzyme, which turns alcohol into acetaldehyde, as do about 94 in 100 people of European ancestry.

Not a diagnosis. This card reads a variant that is also studied in medicine. It describes a genetic background, not a condition, and is no reason to change your diet or any treatment. Only a doctor can diagnose a condition.

Background ADH1B makes one of the liver enzymes that turn alcohol into acetaldehyde. The T allele of rs1229984 (Arg48His) gives an enzyme with about 90 times the turnover of the usual form in laboratory tests (Edenberg 2007), so acetaldehyde forms more quickly after drinking. In 261,991 people of European ancestry, carriers drank 17.2 percent fewer units of alcohol per week and were more likely not to drink at all (Holmes 2014).

Keep in mind This variant is one of several influences on how alcohol affects a person, alongside ALDH2, other genes and habits, and this result says nothing about how much alcohol is safe for anyone. It is not a medical diagnosis. If you have symptoms or questions, talk to a doctor.

SOURCES [25011450](#), [17718394](#), [18996923](#), [30643251](#)

GENE HIGHLIGHT · ADH1B Evidence B

Alcohol breakdown, second ADH1B variant

YOUR GENOTYPE [rs2066702](#) · AG

Your file shows one copy of the ADH1B variant rs2066702 A, a faster form of the enzyme found mainly in people of African ancestry. Studies in people of African ancestry found that each copy goes with drinking slightly less alcohol on average.

Not a diagnosis. This card reads a variant that is also studied in medicine. It describes a genetic background, not a condition, and is no reason to change your diet or any treatment. Only a doctor can diagnose a condition.

Background The A allele of rs2066702 changes ADH1B at position 370 (Arg370Cys). In laboratory tests the enzyme it makes can work about 75 times faster than the usual form, but only when alcohol levels are high (Edenberg 2007). It is found on about 19 in 100 gene copies in people of African ancestry and is almost absent elsewhere. In 95,343 people of African ancestry, each copy was linked with fewer drinks per week (Saunders 2022), and in 17,029 African American veterans with a lower largest amount usually drunk (Gelernter 2019).

Keep in mind Most studies of this variant are in people of African ancestry, the effect on drinking is small, and this result says nothing about how much alcohol is safe for anyone. It is not a medical diagnosis. If you have symptoms or questions, talk to a doctor.

SOURCES [36477530](#), [31151762](#), [17718394](#)

Protein intake

Protein supplies the amino acids the body uses to build and repair muscles, organs, enzymes and hormones. Protein contributes to the maintenance of muscle mass and of normal bones.

YOUR RESULT No meaningful difference  4/4

Independent variants known for this trait: 4; with a call in your file: 4; compared with the average person: 4; carrying at least one copy of the effect allele: 1.

Taken together, your scored variants point to no meaningful difference from the average person (net effect-allele copies -0.6; a total more than one standard deviation from the average counts as a tendency).

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs579459	TT	T	2	0.79	-0.07 unit	34426670
rs6601299	CC	T	0	0.09	+0.09 unit	34426670
rs780094	CC	T	0	0.41	+0.06 unit	34426670
rs838133	GG	A	0	0.43	-0.11 unit	34426670

What the studies measured	The studies calculated the share of total energy intake that came from protein, using food questionnaires or 24-hour diet recalls in large groups of adults. Reported diets also reflect culture, income, taste and memory. Some variants found, such as one near the FGF21 gene, are linked with liking sweet foods, which shifts the balance of the whole diet.
Reference intake	EFSA's population reference intake for adults is 0.83 g of protein per kilogram of body weight a day.
Food sources	Meat, fish, eggs, dairy products, pulses, soy foods, nuts and cereals.

Fat intake

Fat is the most concentrated source of energy in food. It supplies the essential fatty acids the body cannot make and helps it absorb vitamins A, D, E and K.

YOUR RESULT

▲ Slightly more  3/7

Independent variants known for this trait: 7; with a call in your file: 3; compared with the average person: 3; carrying at least one copy of the effect allele: 0.

Your scored variants point to a tendency toward a slightly higher reported share of energy from fat than in the average person of European ancestry (net effect-allele copies +1.2; a total more than one standard deviation from the average counts as a tendency). This describes what people with similar variants tended to report in the studies; it is not a measure of your diet and not a reason to change it.

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs1229984	CC	T	0	0.03	+0.52 unit	34426670
rs56131196	GG	A	0	0.20	-0.14 unit	34426670
rs838133	GG	A	0	0.43	-0.20 unit	34426670

What the studies measured	The studies calculated the share of total energy intake that came from fat, using food questionnaires or 24-hour diet recalls in large groups of adults. Reported diets also reflect culture, income, taste and memory.
Reference intake	EFSA's reference intake range for adults is 20 to 35 percent of energy from fat.
Food sources	Oils, butter, cheese, meat, nuts, seeds and many baked and processed foods.

Carbohydrate intake

Carbohydrates, mainly starches and sugars, are the main source of energy in most diets. Dietary fiber is also a carbohydrate, but it is not digested in the small intestine.

YOUR RESULT

○ Too few variants for a tendency  2/5

Independent variants known for this trait: 5; with a call in your file: 2; compared with the average person: 2; carrying at least one copy of the effect allele: 1.

Your file measures 2 of the 5 variants known for the reported share of energy from carbohydrates. A tendency needs at least three, so this report gives none; the variants measured are listed below.

Variant	Genotype	Effect allele	Copies	Frequency	Effect per copy	Studies (PubMed IDs)
rs2126259	CT	T	1	0.09	-0.23 unit	34426670
rs838133	GG	A	0	0.43	+0.21 unit	34426670

What the studies measured	The studies calculated the share of total energy intake that came from carbohydrates, using food questionnaires or 24-hour diet recalls in large groups of adults. Reported diets also reflect culture, income, taste and memory.
Reference intake	EFSA's reference intake range for adults is 45 to 60 percent of energy from carbohydrates.

Food sources

Bread, pasta, rice, potatoes, cereals, pulses, fruit, milk and foods with added sugar.

05 of 6

Food tolerance

Section 05 of 6 · 2 gene highlights

Fig. 05 – In this section

2 gene highlights

01 Lactase persistence

02 Celiac-linked HLA types

That is everything in this section.

Food tolerance

This section reads a few single, well-studied variants. Some are linked with whether the body keeps making lactase, the enzyme that digests milk sugar, after childhood. Others mark the immune types HLA-DQ2 and HLA-DQ8, which nearly everyone with celiac disease carries but which are also common in people without it. These results describe a genetic background, not a diagnosis. Most people who carry these immune types never develop celiac disease. How well people digest lactose also depends on how much lactose they eat and on other factors in the gut. Only a doctor can diagnose lactose intolerance or celiac disease, and this section is no reason to start or stop eating any food.

GENE HIGHLIGHT · LCT / MCM6

Evidence A

Lactase persistence

YOUR GENOTYPE rs4988235 · GG

Your file shows no copy of the lactase persistence variant. In people of European ancestry, studies found that this genotype usually goes with making much less lactase, the enzyme that digests milk sugar, after childhood; worldwide this is the most common pattern. In some other ancestries, variants not read here can keep lactase active. It is not a diagnosis of lactose intolerance: many people with this genotype have no symptoms with usual servings of milk products.

Not a diagnosis. This card reads a variant that is also studied in medicine. It describes a genetic background, not a condition, and is no reason to change your diet or any treatment. Only a doctor can diagnose a condition.

Background

The LCT gene makes lactase, the enzyme that digests lactose, the sugar in milk; in most people its activity falls after early childhood. The A allele of rs4988235, known in papers as LCT C/T-13910, keeps the gene active in adults: in the study that found it, it matched measured lactase activity in all 236 people tested from four populations (Enattah 2002). About two in three adults worldwide make much less lactase after childhood. Keeping lactase is most common in people of northern European descent and in some herding peoples of Africa and the Middle East, and rare in East Asia (Anguita-Ruiz 2020). Making less lactase does not always cause symptoms: in a small study of 13 such adults, symptoms did not rise with up to 6 g of lactose, and pain rose only from 12 g, about a cup of milk (Hertzler 1996).

Keep in mind

This variant explains lactase persistence well in people of European ancestry, but in parts of Africa, the Middle East and South Asia other nearby variants that are not read here, such as rs41380347 and rs145946881, also keep lactase active. If you have symptoms or questions, talk to a doctor; only a medical test, such as a hydrogen breath test, can tell how well you digest milk sugar.

SOURCES [11788828](#), [32899182](#), [17159977](#), [8613657](#)

Celiac-linked HLA types

YOUR GENOTYPE DQ2.5 · rs2187668 · CC DQ8 · rs7454108 · TT rs7775228 · TT rs2395182 · TT
rs4713586 · AA DQ7 · rs4639334 · not in your file

DQ2.5: no copy · DQ8: no copy · DQ2.2: no · DQ7: not read

Your file shows none of the three HLA types read here: DQ2.5, DQ8 and DQ2.2. In studies, about 3 in 100 people with celiac disease, or fewer, had none of the three. This is not a diagnosis, and it does not fully rule celiac disease out.

Not a diagnosis. This card reads variants that are also studied in medicine. It describes a genetic background, not a condition, and is no reason to change your diet or any treatment. Only a doctor can diagnose a condition.

Background

HLA-DQ genes shape how the immune system presents proteins, including gluten, to immune cells. In an Italian study of 437 children with celiac disease, 91 in 100 carried DQ2 or DQ8, 6 in 100 only the beta half of DQ2, usually as DQ2.2, 2 in 100 only its alpha half, as in DQ7, and fewer than 1 in 100 none of these (Megiorni 2009). Six marker SNPs read these HLA types with more than 99 percent sensitivity and specificity in Dutch and other European samples (Monsuur 2008). Celiac disease affects about 1 in 100 people.

Keep in mind

These markers were checked in people of European ancestry and may tag the HLA types less well in other ancestries. If you have symptoms or questions, talk to a doctor; only medical tests, starting with a blood test for celiac antibodies taken while gluten is still eaten, can tell.

SOURCES [18509540](#), [19255754](#), [19027045](#), [12651074](#)

06 of 6

Taste and smell

Section 06 of 6 · 4 gene highlights

Fig. 06 – In this section		4 gene highlights
01	Bitter taste (TAS2R38)	
02	Floral smell of beta-ionone (OR5A1)	
03	Smelling asparagus in urine	
04	Sweet preference (FGF21)	
That is everything in this section.		

Taste and smell

This section reads a few well-studied variants linked with how people perceive certain tastes and smells. Examples are the bitterness of some vegetables, a floral scent and the smell asparagus can give to urine. These are harmless differences between people. They can shape food likes and dislikes, but culture, upbringing and habit also play a large part.

GENE HIGHLIGHT • TAS2R38

Evidence A

Bitter taste (TAS2R38)

YOUR GENOTYPE A49P • rs713598 • CC V262A • rs1726866 • AA I296V • rs10246939 • TT

You have two copies of the non-taster version (AVI), like about 3 in 10 people of European ancestry. Studies found that most people with this combination taste little or no bitterness from PTC and PROP.

Background TAS2R38 is a bitter taste receptor for test compounds called PTC and PROP, which resemble substances in cabbage-family vegetables such as broccoli, Brussels sprouts and rocket. Three common changes in the gene form two main versions: PAV, the taster version, and AVI, the non-taster version. In the original studies these versions explained 55 to 85 percent of the differences between people in how sensitive they are to the bitter taste of PTC. In about 150,000 UK Biobank participants, taster copies went with slightly lower liking of horseradish and non-taster copies with slightly higher liking of grapefruit.

Keep in mind How bitter food tastes also depends on other bitter receptors, saliva and experience, and liking of bitter foods changes with cooking, habit and repeated tasting. In the same UK Biobank study, these versions were not clearly linked with liking of any vegetable.

SOURCES [12595690](#), [18209019](#), [35585065](#), [16979544](#)

GENE HIGHLIGHT • OR5A1

Evidence A

Floral smell of beta-ionone (OR5A1)

YOUR GENOTYPE rs6591536 • GG

You have two copies of the sensitive version. Studies found that people with this genotype usually notice beta-ionone easily and describe it as fragrant and floral.

Background OR5A1 is a smell receptor for beta-ionone, the violet-like floral note found in foods and drinks such as raspberries and some teas and wines, and often added to flavorings. A single change in this receptor, at rs6591536, explained more than 96 percent of the differences in beta-ionone sensitivity between people in the main study, and lab tests confirmed that it changes how the receptor responds. Sensitive people usually describe the smell as fragrant and floral, less sensitive people describe it differently, and the genotype was linked with which foods and products people chose.

Keep in mind This variant affects one smell note only; overall sense of smell and flavor liking depend on many genes, habits and health.

SOURCES [23910657](#), [23910658](#), [35113854](#)

GENE HIGHLIGHT • OR2M7

Evidence B

Smelling asparagus in urine

YOUR GENOTYPE **rs4481887 • GG**

You have no copy of the A version, the most common genotype. Studies found that people with this genotype are more likely than others not to notice the smell; in one study about 4 in 10 of them said they could not smell it.

Background After eating asparagus, some people notice a sulfur smell in their urine while many others cannot smell it. A variant in a cluster of smell receptor genes near OR2M7 was linked with this ability in 23andMe research participants and confirmed in a later study of 6,909 US adults, in which 58 percent of men and 62 percent of women did not strongly agree that they notice the smell. In the first study, 43 percent of people with no A copy said they could not smell it, against 29 percent with one copy and 26 percent with two.

Keep in mind Whether there is a smell to notice also depends on whether your body produces the odorous compounds, which this variant does not cover. The genotype only shifts the odds; people with every genotype fall on both sides.

SOURCES [20585627](#), [27965198](#), [20876394](#)

GENE HIGHLIGHT • FGF21

Evidence B

Sweet preference (FGF21)

YOUR GENOTYPE **rs838133 • GG**

You have no copy of the A version. Studies found that people with this genotype tend, on average, to get a slightly smaller share of their calories from carbohydrates and to like sweet foods slightly less than people who carry it.

Background FGF21 is a hormone made in the liver; its level rises after eating sugar, and in animal studies it reduces the appetite for sweets. The A version of a common variant in the FGF21 gene was linked with a slightly higher share of calories from carbohydrates in studies of 91,114 and 451,099 European adults, about 0.2 percentage points of daily calories per copy. In a Danish study of 6,514 adults it went with eating more sweets, and in about 160,000 UK Biobank participants with slightly higher liking of sweet foods.

Keep in mind The difference is a fraction of a percent of daily calories, far smaller than the effect of habits, culture and the food around you. The A version is common in Europe but almost absent in East Asia.

SOURCES [29988085](#), [29641994](#), [28467924](#), [35585065](#)

A1 • Nutrients without reliable genetic studies yet

These nutrients are as important as the others, but this report gives no genetic result for them. For some, the GWAS Catalog release used for this report holds no genome-wide study that the report could use. For others, such as sodium, potassium and chloride, the body keeps the blood level so tightly controlled that genetic studies of it say little about diet. Each paragraph gives what the nutrient does, where it is found and why there is no result.

Vitamin B1 (thiamine)

Thiamine is a water-soluble vitamin that cells need to turn carbohydrates into energy. Thiamine contributes to normal energy-yielding metabolism, normal functioning of the nervous system and the normal function of the heart.

Food sources	Whole grains, pork, pulses, nuts, seeds and potatoes.
Why there is no result	The GWAS Catalog release used for this report holds no genome-wide significant finding for thiamine levels.

Vitamin B2 (riboflavin)

Riboflavin is a water-soluble vitamin that forms coenzymes needed to release energy from food. Riboflavin contributes to normal energy-yielding metabolism, normal metabolism of iron, the maintenance of normal red blood cells, skin and vision and the protection of cells from oxidative stress.

Food sources	Milk and dairy products, eggs, meat, fish, mushrooms, almonds and whole grains.
Why there is no result	The GWAS Catalog holds no genome-wide study of riboflavin status. It holds only a few findings for FAD, a riboflavin-derived molecule in blood, and these are too few to give a result.

Vitamin B3 (niacin)

Niacin is a water-soluble vitamin that the body can also make from tryptophan, an amino acid in protein. Niacin contributes to normal energy-yielding metabolism, normal functioning of the nervous system and the maintenance of normal skin and mucous membranes.

Food sources	Meat, poultry, fish, peanuts, pulses and whole grains; foods rich in protein also supply tryptophan.
Why there is no result	The GWAS Catalog holds no genome-wide study of niacin status that this report could score.

Vitamin B5 (pantothenic acid)

Vitamin B5, pantothenic acid, is a water-soluble vitamin and part of coenzyme A, which cells need to release energy from food and to make fats. Pantothenic acid contributes to normal energy-yielding metabolism and to the normal synthesis and metabolism of steroid hormones, vitamin D and some neurotransmitters.

Food sources	Present in almost all foods; meat, liver, eggs, dairy products, whole grains, pulses and mushrooms are good sources.
Why there is no result	The GWAS Catalog holds a few genome-wide findings for pantothenate in blood, from studies that measured many small molecules at once. Consumer DNA chips measure only one of them, too few to give a result.

Vitamin B7 (biotin)

Biotin is a water-soluble vitamin that helps enzymes make glucose and fatty acids. Biotin contributes to the maintenance of normal skin, hair and mucous membranes. High-dose biotin supplements can distort some laboratory blood tests, including some heart and hormone tests. The US Food and Drug Administration has warned that this can lead to wrong results.

Food sources Eggs, liver, nuts, seeds, pulses, oats and some vegetables; raw egg white contains a protein that binds biotin, cooked egg does not.

Why there is no result The GWAS Catalog holds no genome-wide study of biotin levels.

Vitamin K

Vitamin K is a group of fat-soluble compounds that the liver needs to make blood-clotting proteins. K1 comes from green plants, and K2 is made by bacteria. Vitamin K contributes to normal blood clotting and to the maintenance of normal bones. It can also weaken the effect of some medicines that reduce blood clotting.

Food sources Green leafy vegetables such as kale, spinach and lettuce, broccoli and vegetable oils; cheese and fermented foods provide K2.

Why there is no result The genome-wide studies of vitamin K levels in the catalog found no variant at the strict significance threshold these studies use.

Sodium

Sodium, eaten mostly as table salt, controls the volume of blood and other body fluids and is needed for nerve signals and muscle contraction.

Food sources Salt added in cooking and at the table, and above all processed foods such as bread, cheese, cured meats, soups, snacks and ready meals.

Why there is no result Blood sodium is held within a narrow range by the kidneys and hormones, so genetic studies of it say little about how much salt people eat.

Potassium

Potassium is the main mineral inside cells. Potassium contributes to normal muscle function, normal functioning of the nervous system and the maintenance of normal blood pressure.

Food sources Vegetables, potatoes, fruit such as bananas and apricots, pulses, nuts, dairy products, meat and fish.

Why there is no result Blood potassium is held within a narrow range by the kidneys and hormones. Genetic studies of it mainly reflect kidney function and say little about how much potassium people eat.

Chloride

Chloride, eaten mostly as table salt, works with sodium to keep body fluids in balance. Chloride contributes to normal digestion by production of hydrochloric acid in the stomach.

Food sources Table salt and salty foods such as bread, cheese, cured meats and ready meals.

Why there is no result Blood chloride follows sodium and the body's acid-base balance, which the kidneys control, so genetic studies of it say nothing useful about diet.

Iodine

Iodine is needed to make thyroid hormones. Iodine contributes to the normal production of thyroid hormones and normal thyroid function, and to normal energy-yielding metabolism.

Food sources Iodized salt and foods made with it, fish and seafood, milk and dairy products, and eggs.

Why there is no result No genome-wide study in the catalog measures iodine status directly; thyroid hormone levels, which have been studied, reflect thyroid function rather than iodine intake.

Manganese

Manganese is a trace element that several enzymes need. Manganese contributes to normal energy-yielding metabolism, the maintenance of normal bones, the normal formation of connective tissue and the protection of cells from oxidative stress.

Food sources Whole grains, nuts, pulses, tea, leafy vegetables and fruit.

Why there is no result The catalog's few genome-wide findings for manganese in blood lack the variant details this report needs to compare them with a DNA file.

Sulfur

Sulfur is part of the amino acids methionine and cysteine, of the antioxidant glutathione and of the vitamins thiamine and biotin.

Food sources Protein foods such as meat, fish, eggs, dairy products, pulses and nuts; onions, garlic and cabbage-family vegetables also contain sulfur compounds.

Why there is no result There is no separate dietary requirement for sulfur, because the body gets it from the amino acids in protein. For this reason, there is no reference value or genetic study to report.

A2 · Popular nutrition-gene claims we leave out, and why

Many genetic nutrition tests report the variants below. We looked at the evidence for each and left them out of this report, for the reason given. Leaving a variant out does not mean it does nothing; it means the studies do not support reading it from a raw DNA file to say something about your nutrition.

CYP1A2 · rs762551

The claim

This CYP1A2 variant is often said to sort people into slow and quick caffeine processors, with coffee said to be riskier for the slow group.

Why we leave it out

Without smoking, a study found no clear difference in caffeine breakdown between its genotypes: in 185 non-smokers the caffeine test ratio did not differ (Sachse 1999). An early report that coffee carried different heart risks by genotype (Cornelis 2006) was not confirmed in 347,077 UK Biobank participants (Zhou 2019). Large genome-wide studies point to the two markers used in the caffeine card instead.

Sources

[10233211](#), [16522833](#), [30838377](#)

ADORA2A · rs5751876

The claim

This variant is said to make caffeine cause jitters and poor sleep.

Why we leave it out

Two experiments found stronger jitteriness after caffeine in people with the TT genotype: 94 people who rarely took caffeine (Alsene 2003) and 379 people (Rogers 2010). But in the second study the TT group drank more coffee, not less, and regular coffee drinkers became used to the effect whatever their genotype; in 1,023 workers the links with sleep were mixed (Erblang 2019). The variant has never reached genome-wide significance in large coffee studies.

Sources

[12825092](#), [20520601](#), [31817803](#)

ADH1C · rs698 · rs1693482

The claim

A slower ADH1C variant is said to change alcohol tolerance or how moderate drinking affects the heart.

Why we leave it out

Links between ADH1C and drinking are largely explained by its close tie to the ADH1B variant on the same stretch of DNA (Edenberg 2007). A single report that it changed the link between moderate drinking and heart outcomes (Hines 2001) was not confirmed in 9,584 Danish adults (Tolstrup 2009).

Sources

[17718394](#), [11207350](#), [20031627](#)

AOC1 (DAO) · rs10156191 · rs2052129 · rs1049742 · rs1049793

The claim

DAO gene variants are said to show histamine intolerance and which foods to avoid.

Why we leave it out

In 484 people, three of these variants were linked to lower DAO enzyme activity in the blood, but none was linked to histamine intolerance symptoms themselves (Maintz 2011). Histamine intolerance has no validated diagnostic test (Comas-Baste 2020), and one of the variants, rs1049793, is palindromic and cannot be read safely from raw files.

Sources

[21488903](#), [32824107](#)

HNMT · rs11558538

The claim

An HNMT variant is said to slow the breakdown of histamine from food.

Why we leave it out

The variant lowers HNMT activity in laboratory tests (Preuss 1998), but HNMT works inside cells, and histamine from food is broken down in the gut mainly by DAO (Comas-Baste 2020). No reliable link between it and reactions to food has been shown.

Sources

[9547362](#), [32824107](#)

ALDOB · rs1800546 · rs76917243 · rs78340951

The claim A raw DNA file is said to show fructose intolerance.

Why we leave it out This report does not read the ALDOB variants that cause hereditary fructose intolerance. They are rare, and SNP chips often miscall very rare variants (Weedon 2021). The common complaint of poor fructose absorption has no known genetic marker.

Sources [33589468](#), [20033295](#)

HLA-DQ / HLA-DR · rs9275596 · rs7192

The claim Genes are said to show whether a person is likely to be allergic to peanuts.

Why we leave it out The strongest common marker, near HLA-DQ, was found in 2,197 people of European ancestry and raised the odds about 1.7 times (Hong 2015); about half of Europeans carry it, and nearly all of them are not allergic. A study of Canadian children pointed to other HLA types, one of them linked with lower odds (Madore 2013), and allergy is diagnosed with skin or blood tests and food challenges, not from DNA.

Sources [25710614](#), [23443026](#)

HLA-DQ / HLA-DPB1 · rs59325236 · rs9271588 · rs9277630

The claim Genes are said to show a wheat allergy.

Why we leave it out The genome-wide findings so far come from two specific Japanese groups: people who became allergic to hydrolyzed wheat protein from a facial soap (Noguchi 2019) and people with wheat-dependent, exercise-induced reactions (Fukunaga 2021). They do not apply to wheat allergy in general or to other populations.

Sources [31301374](#), [34246321](#)

No single gene

The claim A DNA test is said to show gluten sensitivity without celiac disease.

Why we leave it out No genetic or other biological marker for gluten sensitivity without celiac disease is known (Catassi 2015). The HLA types in the celiac card are about celiac disease only.

Sources [26096570](#)

MTHFR · rs1801131

The claim MTHFR A1298C on its own, or together with one copy of C677T, slows methylation and calls for special forms of folate.

Why we leave it out In the first study of A1298C, the variant lowered enzyme activity but did not raise homocysteine or lower blood folate on its own; in that small study, higher homocysteine was seen only in people who carried one copy of each MTHFR variant. A larger study of 10,601 people did find links with homocysteine and folate, but genome-wide studies of homocysteine (44,147 people) and folate (37,341 people) did not find it as a separate signal, and the US CDC states there is not enough evidence that A1298C alone affects how the body processes folate.

Sources [9545395](#), [17436311](#), [23824729](#), [23754956](#)

MTR · rs1805087

The claim MTR A2756G disturbs B12-dependent methylation and raises homocysteine, so carriers need special forms of B12.

Why we leave it out A genome-wide study of 44,147 people found only a very small homocysteine effect at the MTR gene, about 0.05 standard deviations per copy, and it was measured through a nearby linked marker. An effect this small says nothing useful about one person.

Sources [23824729](#)

MTRR · rs1801394**The claim**

MTRR A66G slows B12 recycling and methylation.

Why we leave it out

About half of all gene copies in people of European ancestry carry this version. A study of 10,601 people that measured homocysteine, folate and B12 found no link with it, and genome-wide studies of homocysteine and vitamin B12 in tens of thousands of people did not find a signal at this gene. The claims rest on small candidate-gene studies with inconsistent results.

Sources

[17436311](#), [23824729](#), [23754956](#)

VDR · rs2228570 · rs1544410 · rs731236 · rs7975232**The claim**

VDR FokI, BsmI, TaqI and ApaI decide how well you respond to vitamin D and how strong your bones are.

Why we leave it out

A combined analysis of 26,242 people from nine European research teams found no link between these four VDR variants and bone density or fractures. Genome-wide studies of vitamin D levels in up to 79,366 people did not find VDR among the genes that matter. Other services still report them, which is why we explain our choice.

Sources

[16908916](#), [20541252](#), [29343764](#)

PEMT / CHDH / BHMT / MTHFD1 · rs12325817 · rs7946 · rs12676 · rs3733890 · rs2236225**The claim**

These variants mean you need more choline than other people.

Why we leave it out

The claims come from two small feeding studies of 54 and 57 adults on an almost choline-free diet, and in one of them the PEMT rs7946 and BHMT variants showed no link at all. The findings have not been repeated in large studies, and choline needs are set by age, sex and life stage, not by these variants.

Sources

[16816108](#), [16236726](#)

SLC19A1 (RFC1) · rs1051266**The claim**

The reduced folate carrier variant 80G to A lowers folate transport into cells.

Why we leave it out

Studies of this variant gave inconsistent results. A study of 10,601 people that measured folate and homocysteine found no link, and genome-wide studies of blood folate, including one of 37,341 people, did not find SLC19A1 among the genes that matter.

Sources

[17436311](#), [23754956](#), [19744961](#)

TCN2 · rs1801198**The claim**

TCN2 776C to G (Pro259Arg) lowers delivery of active vitamin B12 to cells.

Why we leave it out

The main study had 128 people and found a difference in the B12 carried by transcobalamin but not in total B12 or homocysteine. A study of 10,601 people found a link only with methylmalonic acid, another marker of B12 use, and genome-wide studies of B12 in up to 45,576 people pointed to other TCN2 variants. The marker is also a C/G site whose strand cannot be checked from the letters, so a reading could be wrong; we report a different, well-replicated TCN2 variant instead.

Sources

[12091374](#), [12107818](#), [17436311](#), [23754956](#)

BCO1 (BCM01) · rs12934922 · rs7501331**The claim**

These two BCO1 variants make you a poor converter of beta-carotene into vitamin A.

Why we leave it out

The poor-converter claim comes from one small study of female volunteers given a large dose of beta-carotene. In a later study of 2,344 women, R267S did go with higher blood beta-carotene, but A379V went the opposite way to the small study. R267S is an A/T site whose strand cannot be checked from the letters, and A379V is missing from some common chips, so we report the BCO1 marker rs6564851 instead.

Sources

[19103647](#), [23134893](#), [19185284](#)

FTO · rs9939609 · rs1558902 · rs1421085

The claim	Your FTO type shows which diet, for example low-fat or low-carb, will help you lose weight.
Why we leave it out	FTO variants are linked with average body weight, but an analysis of 8 randomized trials with 9,563 people found that weight loss after diet, exercise or drug programs was the same whatever the FTO genotype. In the Food4Me trial in seven European countries, adding genetic results to personalized advice did not improve diet changes.
Sources	27650503 , 27524815 , 17434869

APOA2 · rs5082

The claim	People with the APOA2 CC genotype gain weight from saturated fat and should eat less of it.
Why we leave it out	The link between this genotype, saturated fat and body weight comes from observational studies, largely by one research network, using a fixed intake cut-off. No trial has tested whether advice based on this genotype works better than general advice.
Sources	19901143 , 20975728

APOA5 · rs662799 · rs3135506

The claim	APOA5 variants decide whether a high-fat diet raises your blood fats.
Why we leave it out	APOA5 variants are strongly linked with blood triglycerides, but studies of diet interaction disagree: a Spanish clinic study of 1,465 people reported one, while a study of 5,487 Czech adults found none. Blood fat levels are a medical topic and are not covered by this report.
Sources	24462044 , 21209257

PPARG · rs1801282

The claim	The PPARG Pro12Ala variant decides whether you do better on a high-fat or a low-fat diet.
Why we leave it out	This variant is studied mainly for blood sugar control, where its effect is modest. Reports that it changes the response to dietary fat or exercise come from small observational studies, such as one of 506 people.
Sources	10973253 , 14681835

TCF7L2 · rs7903146

The claim	Carriers of the TCF7L2 variant should follow a low-carbohydrate diet.
Why we leave it out	In a large lifestyle trial, carriers and non-carriers lost similar amounts of weight, and the program helped both groups. There is no trial evidence that carbohydrate advice based on this variant works better.
Sources	16855264 , 21814547

MC4R · rs17782313

The claim	MC4R variants mean you need an appetite-control diet.
Why we leave it out	Common MC4R variants shift average body weight by a small amount. No trial shows that advice based on them works better than general advice.
Sources	18454148

SLC30A8 · rs13266634

The claim	Your zinc transporter gene SLC30A8 means you need more zinc.
Why we leave it out	Although the gene makes a zinc transporter, this variant is not among the signals found in a genome-wide study of blood zinc. A meta-analysis of 14 cohorts found only a weak hint that zinc intake changes the link between a variant in this gene and blood sugar.
Sources	23720494 , 21810599

CD36 · rs1761667**The claim**

CD36 shows whether you can taste fat and whether you crave fatty food.

Why we leave it out

The evidence comes from small studies, one of 21 people and one of 317, with measures that differ between studies. No large study has confirmed a link with fat taste or fat intake.

Sources

[22210925](#), [22240721](#)

TAS1R2 / TAS1R3 · rs35874116 · rs307355 · rs35744813**The claim**

TAS1R2 or TAS1R3 variants give you a sweet tooth.

Why we leave it out

The TAS1R2 results come from small studies whose effects differed by body weight and between studies, and the TAS1R3 finding rests on one small study. A UK study of about 160,000 people found little evidence that any of the sweet receptor genes affect food liking. FGF21, which has stronger evidence for sweet preference, is shown instead.

Sources

[20943793](#), [26279452](#), [19559618](#), [35585065](#)

CA6 (gustin) · rs2274333**The claim**

Your CA6 gustin variant makes you a supertaster.

Why we leave it out

The link was reported in small studies by one research group. An independent study of 243 people found no link between CA6 variants and bitter taste from PROP.

Sources

[20631203](#), [24534176](#)

ACE / AGT · rs4343 · rs699**The claim**

ACE or AGT variants show whether salt raises your blood pressure.

Why we leave it out

A meta-analysis found no link between these variants and how blood pressure responds to salt, although the studies were small. Blood pressure is a medical topic and is not covered by this report.

Sources

[26556555](#)

AMY1**The claim**

Your AMY1 copy number shows how well you digest starch and whether carbohydrates make you gain weight.

Why we leave it out

The number of AMY1 gene copies differs between people and populations, but it is a structural variation that SNP-based DNA files cannot measure reliably. A study of about 3,500 people, with 99 percent power to detect the claimed effect, found no link between AMY1 copy number and body mass index.

Sources

[17828263](#), [26098870](#)

GSTM1 / GSTT1**The claim**

Your GSTM1 and GSTT1 detox genes show whether you need more cabbage-family vegetables or detox support.

Why we leave it out

These genes vary mainly by whole-gene deletions, which are common in every population studied and which SNP-based DNA files do not report reliably. Controlled feeding trials gave mixed results on whether people with the deletions respond differently to cabbage-family vegetables.

Sources

[18056202](#), [19843669](#), [25165394](#)

APOE**The claim**

Eat for your APOE type.

Why we leave it out

APOE is studied mainly in medicine, for its links with disease, not in nutrition. This report does not read its variants.

OR6A2 · rs72921001**The claim**

Your genes show whether coriander tastes like soap to you.

Why we leave it out

One study of 14,604 23andMe research participants linked a variant near the smell receptor gene OR6A2 with reporting a soapy taste, with only weak confirmation in other participants of the same company. A UK study of about 160,000 people found its strongest signal for coriander liking in a different smell receptor region and did not report this variant. Common variants explain less than 9 percent of the differences between people.

Sources

[35585065](#); Eriksson N, Wu S, Do CB, et al. A genetic variant near olfactory receptor genes influences cilantro preference. Flavour 2012;1:22. doi 10.1186/2044-7248-1-22; arXiv 1209.2096

SLC2A2 (GLUT2) · rs5400**The claim**

Your GLUT2 gene gives you a sweet tooth.

Why we leave it out

The link with sugar intake comes from one report on two groups of people, and large genome-wide studies of diet and food liking have not reported it.

Sources

[18349384](#), [35585065](#)

GPX1 · rs1050450**The claim**

Your GPX1 variant means you need more selenium or antioxidants.

Why we leave it out

Genome-wide studies of blood and toenail selenium found their main signals near DMGDH, BHMT and CBS, and none in GPX1. Selenium needs are set by age, sex and life stage, not by this variant.

Sources

[23720494](#), [25343990](#)

A3 • Methods and sources

Data

This report uses the NHGRI-EBI GWAS Catalog, release 2026-06-01 (EFO v3.90.0), and allele frequencies of people of European ancestry from the 1000 Genomes Project. For its 27 traits it uses 305 independent lead variants and 311 neighboring variants that can stand in for them, counted per trait as in the table below (263 and 295 distinct variants, since a variant can serve more than one trait), from 67 studies. Its 24 gene highlights read 36 markers chosen from the literature.

How a tendency is given

For each nutrient we use up to 20 of the strongest independent findings in the GWAS Catalog, compare the alleles you carry at each with the average person of European ancestry, and report a slightly higher or lower tendency only when at least a third of those findings, and at least three variants, are measured in your file and your total lies more than one standard deviation from that average, which is the case for about one person in three.

Technical details. Each trait pools the catalog's genome-wide significant rows (p below 5×10^{-8}) for its ontology terms, after removing rows that measure something else or whose effect the catalog records in a form this report cannot use, and rows of mostly European studies whose allele frequency differs from the 1000 Genomes frequency by more than 0.25. Variants are removed when they are rare in Europeans (1000 Genomes minor allele frequency below 1 percent), when they are A/T or C/G variants, whose strand cannot be told from their letters, when they are insertions, deletions or other variants that are not a simple swap of one letter for another, and when no allele frequency is known to compare your copies with. The rest are grouped into regions of 250,000 bases, the strongest variant of each region is its lead, and the 20 strongest leads are kept. Every trait is scored by direction: each copy of an allele linked with a higher value counts plus one and each copy of an allele linked with a lower value minus one, both minus the copies the average person carries. The total is divided by its standard deviation in the reference population, the square root of the sum of $2p(1-p)$ over the scored variants, where p is the allele's frequency ($p(1-p)$ for a variant on a man's X chromosome, of which he has one copy); a result of 1 or more, or -1 or less, gives a tendency. Disease-defining variants and the HFE, MTHFR C677T and celiac HLA markers never enter these tables. Other gene-highlight markers can appear in a trait table too, where they count like any other variant; if the cards marked Not a diagnosis are left out of a report, their markers are left out of its tables as well.

On this release, vitamin B6, vitamin E and selenium have fewer than three known variants, so no file can get a tendency for them; their gene highlights explain what the variants mean.

Strand

DNA has two strands, and a file can report either. In the trait tables, a call is read on the strand it fits, judged from the variant's two letters; this is why A/T and C/G variants are left out of them. Gene highlights read their A/T and C/G markers as written, and only when every other highlight marker in the file reads on the plus strand.

In your file, 32 highlight markers and stand-ins were checked and all read on the plus strand, so the A/T and C/G markers were read.

Stand-ins

When your file lacks a lead, the first variant of the same trait within 50,000 bases that your file has stands in for it and is scored with its own study rows; the tables show it as rs1 = rs2. Gene highlights use a stand-in only when it is inherited with the marker so closely that it misreads fewer than 15 in 100 people of any 1000 Genomes population, and never for HFE C282Y.

Your file measures 127 of the 305 leads of this report directly and 44 more through a stand-in.

Population frequencies

Your copies are compared with the copies the average person carries, from 1000 Genomes frequencies of people of European ancestry. 864 of the 865 catalog rows use these frequencies. Rows for which 1000 Genomes gives none (1 on this release) use a frequency reported in the catalog; variants with no frequency at all are left out, since they cannot be compared.

Traits and catalog terms

The catalog term each trait is built on (the first of its terms), with its leads, the variants that can stand in for them, and the studies behind them.

Trait	Catalog term	Leads	Stand-ins	Studies
Vitamin A	vitamin A level (OBA_1000996)	5	1	4
Vitamin B6	vitamin B6 measurement (EFO_0004621)	1	1	2
Folate (vitamin B9)	folic acid level (OBA_1001015)	7	0	2
Vitamin B12	vitamin B12 measurement (EFO_0004620)	12	3	3
Vitamin C	vitamin C measurement (EFO_0600003)	10	0	1
Vitamin D	vitamin D level (OBA_1000968)	20	53	14
Vitamin E	Vitamin E level (OBA_1000955)	2	0	1
Calcium	calcium measurement (EFO_0004838)	20	74	8
Magnesium	magnesium measurement (EFO_0004845)	5	1	3
Phosphorus	phosphorus measurement (EFO_0004861)	20	34	6
Iron	serum iron amount (OBA_2050055)	17	10	6
Ferritin	ferritin measurement (EFO_0004459)	20	6	5
Transferrin	transferrin measurement (EFO_0006341)	7	2	2
Transferrin saturation	transferrin saturation measurement (EFO_0006333)	7	6	3
Zinc	serum zinc amount (OBA_2050076)	3	0	1
Copper	blood copper level (OBA_2045323)	5	0	2
Selenium	selenium amount (OBA_VT0001775)	1	2	2
Omega-3 fatty acids	omega-3 polyunsaturated fatty acid measurement (EFO_0010119)	20	11	4
DHA	docosahexaenoic acid to total fatty acids percentage (EFO_0022262)	20	10	4
Omega-6 to omega-3 ratio	omega-6:omega-3 polyunsaturated fatty acid ratio (EFO_0010732)	20	13	4
Linoleic acid (omega-6)	linoleic acid measurement (EFO_0006807)	20	18	5
Homocysteine	homocysteine measurement (EFO_0004578)	12	1	2
Coffee intake	cups of coffee per day measurement (EFO_0006782)	15	5	6
Alcohol intake	alcohol consumption quality (OBA_1000840)	20	59	11
Protein intake	protein intake measurement (EFO_0010810)	4	0	1
Fat intake	fat intake measurement (EFO_0010809)	7	0	1
Carbohydrate intake	carbohydrate intake measurement (EFO_0010811)	5	1	1

Gene highlights

Each gene highlight reads one or a few well-studied variants with a rule of its own, chosen and checked against the studies listed below. Evidence A means the finding was replicated in large studies; evidence B that it was replicated in smaller or less direct studies.

Studies cited by the gene highlights: [8613657](#), [11788828](#), [12595690](#), [12651074](#), [15858186](#), [16979544](#), [17159977](#), [17718394](#), [18199861](#), [18209019](#), [18509540](#), [18776911](#), [18996923](#), [19027045](#), [19185284](#), [19255754](#), [19297519](#), [19303062](#), [19320537](#), [19744961](#), [19820698](#), [19820699](#), [20519558](#), [20541252](#), [20585627](#), [20661308](#), [20876394](#), [21357676](#), [21729881](#), [21803414](#), [21829377](#), [21873547](#), [22113863](#), [22503634](#), [23134893](#), [23288205](#), [23393431](#), [23720494](#), [23754956](#), [23824729](#), [23909789](#), [23910657](#), [23910658](#), [24068962](#), [24382882](#), [24823311](#), [25011450](#), [25288136](#), [25343990](#), [25411281](#), [25527764](#), [25788000](#), [27702941](#), [27965198](#), [28467924](#), [29040465](#), [29343764](#), [29641994](#), [29988085](#), [30643251](#), [31151762](#), [31666285](#), [32193382](#), [32899182](#), [33203707](#), [33462484](#), [33536631](#), [35113854](#), [35585065](#), [36477530](#), [36635386](#), [36811574](#), [37253714](#), [38479735](#), [39414775](#)