

Results Overview

Ergothioneine levels (SLC22A4)

Your SLC22A4 (L503F) genotype is associated with moderately higher blood and tissue levels of ergothioneine.



Moderately higher levels

Glutamine production and oxidative stress (GLUL)

Your genes are linked to significantly reduced glutamine production, which may increase your oxidative stress risk.



Higher Risk

Nitric oxide and blood flow

Your gene variants are linked to good production of NO, which can help enhance blood flow.



Good Production

Caffeine metabolism and heart health

Your gene variants are linked to an increased risk of adverse cardiovascular effects.



Higher Risk

MTHFR and folate conversion

You are a slightly slower converter of folate into its active form, 5-MTHF.



Slightly slower converter

MTHFR and blood pressure

You do not carry MTHFR gene variants linked to an increased risk of high blood pressure.



Average Risk

LDL cholesterol levels

Your genetic score is associated with average blood LDL-C levels.



Average levels

Lipoprotein (a) levels

Your genetic score is associated with mildly elevated Lp(a) levels.



Mildly elevated

Salt-sensitive hypertension

You do not carry risk genotypes linked to salt-sensitive hypertension.



Lower genetic risk

Familial Hypercholesterolaemia

You do not carry LDLR or APOB mutations linked to familial hypercholesterolaemia (FH).



Lower genetic risk

Inflammation and IL-6 levels

You carry gene variants associated with higher IL-6 production, which increases your susceptibility to chronic inflammation.



Higher Risk

Oxidative stress risk (NQO1)

You have average NQO1 activity, which helps to protect your cells from oxidative stress.



Lower Risk

Oxidative stress risk (GPx-1)

You carry gene variants linked to normal GPx-1 activity, making you less susceptible to cell damage from oxidative stress.



Lower Risk

Oxidative stress (SOD2)

You carry gene variants linked to average SOD2 activity, which may provide less protection against oxidative stress.



Average Risk

Homocysteine levels

Your genetic score is associated with slightly lower plasma homocysteine levels.



Below average levels

Blood triglyceride levels

Your genetic score is associated with slightly lower blood triglyceride levels.



Slightly lower

Ergothioneine levels (SLC22A4) ^



Brain Health

Dubbed the “longevity vitamin,” ergothioneine is a micronutrient found in mushrooms that has recently been lauded for its antioxidant, anti-ageing, and anti-inflammatory properties. After being absorbed from our diet and transported into cells, ergothioneine can neutralise harmful free radicals and protect against damage to DNA, proteins, and other important cell components. In particular, animal models show that ergothioneine can prevent forms of damage and degeneration to our neurons: nerve cells in our brain and nervous system. In humans, higher ergothioneine levels have been associated with a lower risk of neurodegenerative disease and slower rates of cognitive decline. Conversely, although there is no clinical syndrome of ergothioneine deficiency per se, lower tissue levels of ergothioneine have been correlated with faster rates of cognitive decline, increased dementia risk, and accelerated muscle ageing. In this report, we look at how variants of your SLC22A4 gene alter your ability to absorb ergothioneine from food and transport it into cells. This, in turn, can affect your body’s levels of ergothioneine and susceptibility of neurons and other tissues to oxidative stress, inflammation, and ageing.

Your result



Lower
levels,
low diet

Your SLC22A4 (L503F) genotype is associated with moderately higher blood and tissue levels of ergothioneine.






Lower
levels,
high
diet

Ergothioneine is an amino acid found in mushrooms and fermented foods. It is a potent antioxidant, meaning it can protect against damage to cells and tissues from highly reactive molecules known as free radicals.



Mod
higher
levels,
low diet

Ergothioneine may help to protect against certain types of age-related damage to neurons in our brain. Studies show

 **Lower levels****Mod higher levels,** **high diet** **Higher levels, low diet** **Moderate higher levels** **Higher levels, high diet** **Higher levels** **No data**

that blood and tissue ergothioneine levels are higher in healthy elderly individuals compared to those with Parkinson's disease, dementia, and mild cognitive impairment.

Ergothioneine may also protect the heart and blood vessels from damage caused by free radicals. In the Malmö Diet Cancer Study, higher ergothioneine levels were associated with a lower risk of cardiovascular disease.

To protect cells and exert its antioxidant properties, ergothioneine must be absorbed from our diet and moved into cells. This is carried out by a transporter protein called SLC22A4, which is coded for by our SLC22A4 gene.

People who carry the 'F' variant (L503F) of the SLC22A4 gene produce a transporter protein that can move greater amounts of ergothioneine into cells. This results in a higher blood and tissue levels of ergothioneine.

You carry one copy of the 'F' variant linked to higher ergothioneine uptake. Your SLC22A4 (L503F) genotype is: LF.

Your SLC22A4 (L503F) genotype is associated with moderately higher blood and tissue levels of ergothioneine.

Higher ergothioneine levels may protect neurons in our brain, as well as other tissues, against damage from free radicals (oxidative stress), inflammation, and ageing.

Case-control studies of older adults show that healthy individuals tend to have higher ergothioneine levels than people with Parkinson's disease, dementia, and mild cognitive impairment.

In a study of elderly individuals attending Singaporean memory clinics, those with higher ergothioneine levels experienced a slower decline in cognitive abilities such as attention, memory, and executive function.

We do not have adequate lifestyle data on your dietary intake of ergothioneine. In a meta-analysis of 5 studies, people who consumed mushrooms had a 6% lower risk of all-cause mortality compared to people who never ate mushrooms. This may partly be due to protective effects of ergothioneine (found in mushrooms) against ageing-related cell damage.

One study found that Crohn's disease patients with your SLC22A4 genotype (LF) were more likely to experience digestive discomfort and other symptoms of food intolerance when consuming mushrooms. SLC22A4 genotype, however, had no effect on intolerance to mushrooms in those without Crohn's disease.

Recommended Actions

If you don't like mushrooms, try a lion's mane powder or supplement. Lion's mane is a mushroom with a high concentration of L-ergothioneine. Having this as a tea or supplement may make consuming mushrooms more palatable.

Get your mushroom intake through lion's mane, cordyceps or chaga gummies. These mushrooms have high levels of ergothioneine so consuming these in gummy form is a great way to increase your ergothioneine levels.

Serve your meals with a side of sulphur-rich foods like kales, broccoli, or cauliflower. Sulphur-rich foods are rich in glutathione - another antioxidant that can help combat oxidative stress and inflammation.

Eat tomatoes, peppers and kale. These are great food sources of vitamin C. Vitamin C helps fight oxidative stress by boosting antioxidant defences such as raising glutathione levels.

Be active at a moderate intensity for at least 30 minutes, 3-5 days a week. This could be going for a run, lifting weights at the gym, or doing some gardening. Regular physical activity stimulates the hormesis response which increases endogenous antioxidant defences; providing increased protection against oxidative stress.

Switch to 0% alcohol beers to moderate your intake. The fermentation process of beer means it contains small levels of ergothioneine, but moderating alcohol intake is important as alcohol can promote neurodegeneration.

Glutamine production and oxidative stress (GLUL)



Heart Health

Your GLUL gene encodes the enzyme glutamine synthetase, which makes the amino acid glutamine. Glutamine and a related amino acid, glutamic acid (or glutamate), participate in several metabolic reactions in the body and can help prevent oxidative damage to your heart and blood vessels. This trait looks at variants of your GLUL gene that affect your production of glutamine. In turn, this can alter the risk of oxidative damage to your cardiovascular system, particularly when blood sugar levels are poorly controlled.

Your result



**Higher
Risk**

Your genes are linked to significantly reduced glutamine production, which may increase your oxidative stress risk.



**Moderately
Higher
Risk**



**Average
Risk**

Glutamine synthetase (GS) is an enzyme that produces glutamine from glutamic acid (glutamate).

Glutamine and glutamic acid are both key amino acids involved in several metabolic reactions, making proteins, insulin function and protection against oxidative stress.

This trait looks at variants of your GLUL gene, which encodes the glutamine synthetase (GS) enzyme.

This trait focuses on the rs10911021 C>T SNP, which creates 'T' and 'C' alleles.

You carry two copies of the risk 'C' allele.

Your genotype (CC) is associated with a 32% decrease in GS enzyme expression.

The cells lining your arteries (known as vascular endothelium) are at greater risk of damage from oxidative stress if you have poorly controlled blood sugar levels.

Your cardiovascular health is more sensitive to poor insulin function and blood sugar control.

Poor insulin function and blood sugar control can be caused by unhealthy diet (e.g. high-sugar, high GI carbs), lifestyle (e.g. physical inactivity) and body composition (e.g. excessive amounts of visceral fat).

This trait focuses on GS activity. Your diet, exercise habits, lifestyle factors and other genetic traits all influence your metabolic and cardiovascular health.

Recommended Actions

Try to perform at least 150 mins of moderate-intensity exercise each week. This will help keep your body fat levels healthy as well as your metabolic health.

Follow a resistance training program which incorporates compound exercises. Increasing your lean body mass will help keep you metabolically healthy, and limit negative impacts from this gene variant.

Eat lots of vegetables, fruits, beans, lentils, nuts and seeds. These contain antioxidants and do not cause large fluctuations in blood sugar levels.

Reduce the amount of sugar sweetened beverages in your diet. This will improve your blood sugar levels and insulin sensitivity.

Eat more red grapes in your diet. Their skins contain a chemical called resveratrol which has been shown to reduce fasting glucose and insulin concentrations.

Make sure you are getting enough magnesium through foods such as avocados, nuts and seeds or supplements. Magnesium deficiency can increase oxidative stress which can have negative impacts on your cardiovascular health.

Consume lots of insoluble fibre. This form of fibre can be found in brown rice and wheat bran. Fibre is effective at keeping LDL cholesterol levels low, improving blood sugar levels and increasing satiety.

Swap saturated fat for unsaturated fat, for example butter for olive oil spreads. These types of fats are anti-inflammatory and keep your cholesterol levels healthy.

Nitric oxide and blood flow ^



Heart Health

Nitric oxide synthase 3 (NOS3), also known as endothelial NOS (eNOS), is an enzyme that synthesises nitric oxide (NO). NO is a molecule that regulates vascular tone, and aids the dilation of blood vessels. It is therefore an important signalling molecule during exercise, in that it optimizes the blood flow to and from the working muscles. Furthermore, dysfunctional NO production has been linked to raised blood pressure and cardiovascular diseases.

Your result



**Higher
Risk**

Your gene variants are linked to good production of NO, which can help enhance blood flow.



Below

**Average
Production**

You do not have a burden of genetic variants that reduce NOS3 levels.



**Good
Productive**

You can produce enough NOS3, which is the enzyme that synthesizes nitric oxide (NO).

Poor NO production would mean your body is less able to modify and adapt to changes in blood pressure.

NOS3 level, which is also regulated by the peptide bradykinin, is only one factor in NO production.

In addition to NOS3 levels, the availability of the substrates arginine and BH4 are also important factors in nitric oxide production.

Increased oxidative stress can also reduce NO levels, and raise blood pressure.

Recommended Actions

Consuming meat and fish will increase your CoQ10 levels that helps preserve nitric oxide in your body. If you are vegetarian, you might want to consider supplementing with CoQ10.

Aim to get 90 mg for men and 75 mg for women of vitamin C into your diet each day. Food such as oranges, broccoli and bell peppers have a high amount of vitamin C. Getting enough vitamin C helps to enhance the bioavailability of nitric oxide and increase levels of nitric oxide synthase.

Be sure to get plenty of beets, celery, lettuce, radish and spinach into your diet. These foods are good natural sources of nitrate, so can increase the production of nitric oxide.

Increase your intake of flavonoid-rich foods such as apples, blueberries, broccoli, dark chocolate and strawberries. Flavanoids can increase the bioavailability of nitric oxide.

Make sure to add garlic to plenty of your home-cooked meals. Garlic can enhance the bioavailability of nitric oxide and can also increase the levels of nitric oxide synthase.

Try adding pomegranate to your breakfast or salads. Pomegranate has antioxidants that help to preserve nitric oxide and increase the levels of nitric oxide synthase.

Caffeine metabolism and heart health



Heart Health

Should I limit my coffee intake? Coffee is one of the most popular beverages across the world, with a staggering 2.25 billion cups of coffee consumed around the globe each day. The good news for coffee aficionados is that, according to several recent studies, drinking coffee (and caffeine more broadly) can overall reduce the risk of death from any cause, including cardiovascular disease, between 8 and 39%. Probe deeper, however, and it appears that the health benefits of coffee vary from person-to-person depending on various factors, including how much they consume and their genetic make-up. In fact, people that break down caffeine more slowly due to variants of their CYP1A2 gene may actually have a greater risk of heart attack and high blood pressure when drinking 4 or more cups of coffee per day. In this trait, you'll find out whether you're a fast, intermediate, or slow caffeine metaboliser and whether you should consider cutting down your caffeine intake for better heart health.

Your result



**Higher
Risk**

Your gene variants are linked to an increased risk of adverse cardiovascular effects.



**Low
Risk**

The effects of caffeine on your cardiovascular system are influenced by how quickly you break down or 'metabolise' caffeine.



No Data

Slow and intermediate metabolisers are shown in some studies to have a greater risk of heart attack (myocardial infarction) and high blood pressure (hypertension) with higher caffeine intake.

Fast metabolisers, by contrast, do not have an increased heart attack risk with higher caffeine intake. In fact, some

studies suggest that heavy caffeine consumption may even have a beneficial effect on blood pressure in these individuals.

Variants of the CYP1A2 gene determine whether you are a fast, intermediate, or slow caffeine metaboliser.

You are classified as an "intermediate metaboliser". This means you break down caffeine less quickly than fast metabolisers, but more quickly than slow metabolisers. Your CYP1A2 (rs762551) genotype is AC.

Some studies have found that higher caffeine intake increases the risk of heart attack (myocardial infarction) in your genotype. One study found that intermediate (and slow) metabolisers drinking 4 or more cups of coffee had a 1.64 times greater risk of heart attack compared to those drinking less than 1 cup per day.

Intermediate metabolisers may also have an increased risk of high blood pressure (hypertension) with greater caffeine intake. One study which followed subjects over 8 years found that intermediate (and slow) metabolisers drinking 4 or more cups of coffee per day were 3 times more likely to develop hypertension than those who didn't drink coffee.

Ingesting caffeine can cause a transient rise in blood pressure by causing temporary narrowing of arteries (vasoconstriction). Intermediate metabolisers are shown to have slightly higher rises in blood pressure compared to fast metabolisers.

Intermediate metabolisers may be more likely to experience high blood glucose levels when consuming caffeine. Over time, high blood glucose levels can damage the cardiovascular system.

Recommended Actions

If you drink caffeinated drinks, limit to 2 a day. If cutting caffeine out of your diet completely is not something you want to do, ensure you keep your total intake low, as you can experience greater rises in blood pressure from caffeine.

Opt for lower caffeine drinks such as herbal teas and decaf coffee. These drinks will still contain some caffeine but will be much lower than a black coffee or energy drink.

Avoid high-sugar varieties of coffee such as frappes or those with added syrups. As high caffeine intake can cause poorer blood glucose control, opting for lower sugar coffee drinks will be better for maintaining healthy blood sugar levels.

Black tea is a good addition to your day. Black tea is rich in polyphenols - anti-inflammatory compounds that help to improve cardiovascular health - but it is lower in caffeine than coffee.

Avoid caffeine at least 4 hours before going to sleep. Caffeine can negatively impact your sleep quality and, long-term, poor sleep can harm your cardiovascular health. Given your intermediate rate of caffeine metabolism, you will need to allow at least this much time for it to clear your system.

Give green tea a go. Green tea is lower in caffeine and richer in antioxidants than black tea, so if you can stomach this tea, it is a great addition to your day for improving your cardiovascular health.

Watch out for the caffeine in energy drinks and soft drinks.

Energy drinks such as Red Bull, and your everyday soft drink such as Coca Cola contain high amounts of caffeine, so be sure to take these into account when thinking about your daily caffeine intake.

Do not consume more than 400 mg of caffeine daily (approximately 5 or more cups of coffee). There is an increased risk of side effects such as insomnia and cardiovascular impacts when more than this amount is consumed daily.

MTHFR and folate conversion



Heart Health

Did you know that around 24% of us across the globe carry a mutation that impairs our ability to convert folate? Folate, or vitamin B9, is an important micronutrient that we use to make DNA, RNA, proteins, neurotransmitters, and red blood cells. It is also crucial for healthy foetal development during pregnancy, maintaining a healthy cardiovascular system by helping to limit the build-up of a harmful molecule called homocysteine, and switching genes on and off through a process called DNA methylation. We obtain folate from our diet, with dark, leafy greens and legumes being particularly rich sources. Many foods are also fortified with the synthetic form of folate, known as folic acid. Regardless of being natural or synthetic, folate first needs to be converted into its active form, 5-MTHF, before our bodies can use it. This conversion process is carried out by our MTHFR enzyme. Mutations in the gene encoding this enzyme, however, can affect how we convert folate into its active form. In this trait, we look at two common MTHFR gene mutations shown to reduce enzyme activity and impair folate conversion: C677T and A1298C. You will find out whether you carry these mutations and receive recommendations on how to optimise folate conversion and metabolism through dietary and lifestyle measures.

Your result



**Slow
converter**

You are a slightly slower converter of folate into its active form, 5-MTHF.

Moderately



**slower
converter**

In order to be used by the body, we need to convert folate from our diet into its active form: 5-MTHF (5-methyltetrahydrofolate).

Slightly



**slower
converter**

5-MTHF is crucial for a biological process called methylation. This process allows us to make and repair DNA, produce neurotransmitters, and switch genes on and off. It also helps



**Good
converter**

**No data**

us to reduce levels of homocysteine (Hcy) - an amino acid linked to cardiovascular disease.

The enzyme that converts and activates folate into 5-MTHF is called MTHFR (methylenetetrahydrofolate reductase). This enzyme is coded by our MTHFR gene.

Two common mutations in the MTHFR gene can reduce enzyme activity and slow our conversion of folate. These are C677T (rs1801133) and A1298C (rs1801131).

By slowing the conversion of folate into 5-MTHF, these MTHFR mutations may impair the process of methylation. In turn, this can lead to chronic inflammation, DNA damage, and elevated homocysteine levels.

Your MTHFR (C677T) genotype is: CC. You do not carry the 'T' variant linked to reduced MTHFR enzyme activity and slower folate conversion.

Your MTHFR (C677T) gene variant is not associated with lower methylation and higher homocysteine levels.

Your MTHFR (A1298C) genotype is AC. You carry one copy of the 'C' variant, which is linked to 17% lower MTHFR enzyme activity and slower folate conversion.

Overall, you are a: **SLIGHTLY SLOWER CONVERTOR** of folate into its active form, 5-MTHF.

A blood test is required to accurately assess your blood homocysteine levels and risk of cardiovascular disease.

Recommended Actions

Supplement with 7.5-12.5 mg of L-methylfolate per day. This can be taken with food and at any time of day. L-methylfolate is the active form of folate, so does not require conversion, which will be beneficial for you as a slightly slower converter.

Include plenty of dark green leafy vegetables in your diet such as spinach, asparagus, Brussels sprouts, and broccoli. These vegetables are good sources of folate (vitamin B9), which can help to reduce plasma homocysteine levels by driving the folate and methionine metabolic cycles.

Eat fresh tuna, salmon, and large eggs as part of your diet. These foods are great sources of vitamin B12, which plays an important role in your body's folate cycle.

Eat foods fortified with vitamin B12, such as cereals, grains or soy milk, at least four times a week. Nutritional yeast is also a good source of vitamin B12, with 1 tbsp containing 6 mcg of B12. These are good alternative dietary options if you don't eat animal products, and allow you maintain healthy B12 levels to support folate conversion.

Supplement with a B vitamin complex if you do not consume high amounts of folate or B12 in your diet (for example, if you do not regularly consume foods such as beef, salmon, eggs, lentils, and black beans). Supplementing will ensure you are getting enough of these key B vitamins to aid folate conversion, and limit homocysteine levels.

Moderate your alcohol intake or try alcohol-free alternatives.

Alcohol can reduce the absorption of folate, which can raise homocysteine levels.

MTHFR and blood pressure ^



Heart Health

Could low riboflavin (Vitamin B2) levels put you at greater risk of high blood pressure? Dietary factors can affect our health differently depending on our genetic make-up. Scientists call this 'gene-diet interaction'. In this trait, we look at an interaction between riboflavin (Vitamin B2) in your diet and your MTHFR gene. Your MTHFR gene is involved in the folate cycle, which activates folate (vitamin B9) from your diet; limits levels of homocysteine: an amino acid linked to cardiovascular disease; and plays a role in the regulation of blood pressure. When our dietary intake of riboflavin is low, people with certain MTHFR gene variants are at more risk of developing high blood pressure (hypertension). This, in turn, can put greater strain on our heart, damage blood vessels, kidneys and other organs, and is a risk factor for cardiovascular disease.

Your result



**Higher
Risk**

You do not carry MTHFR gene variants linked to an increased risk of high blood pressure.



**Average
Risk**

MTHFR is a key enzyme in the folate cycle, which plays a role in the production of nitric oxide (NO) - a molecule that dilates blood vessels and helps to regulate blood pressure.



No Data

The MTHFR enzyme requires riboflavin (Vitamin B2), found in meat, dairy, green vegetables and fortified foods, to function effectively.

This trait looks at the C677T SNP (rs1801133) in the MTHFR gene. People with two copies of the 'T' allele (TT genotype) have lower activity of the MTHFR enzyme and reduced NO production.

The TT genotype has been linked to an increased risk of high blood pressure (hypertension). This risk is enhanced when levels of riboflavin are low.

You do not have the TT genotype.

Your MTHFR genotype is not linked to a higher risk of hypertension.

Studies suggest that changes in riboflavin levels do not impact hypertension risk in people with your genotype.

Age is a risk factor for hypertension. Your (systolic) blood pressure and risk of hypertension increase as you get older, regardless of your MTHFR genotype.

Obesity (particularly abdominal obesity), smoking, and physical inactivity are major modifiable risk factors for hypertension. Targeting these factors can help to improve blood pressure.

Recommended Actions

Make your own sauces and dressings instead of using store-bought ones. This is an easy way to manage the amount of salt (sodium) in your diet, which helps to keep your blood pressure healthy.

Try yoga or meditation to reduce stress. High levels of stress can increase the risk of raised blood pressure.

Eat sweet potato. Sweet potato is a great source of potassium (448 mg per medium potato) helping to reduce the blood pressure raising impacts from salt (sodium) in your diet.

Look to stop smoking if currently doing so. Smoking can have negative affects on your blood vessels, contributing to raised blood pressure.

Exercise at a moderate intensity at least 30 mins, 5 times a week such as by going for a run or a brisk walk, or doing some household cleaning (vacuuming or mopping). Regular aerobic exercise provides multiple cardiovascular benefits that influence blood pressure regulation.

Perform muscle strengthening activities (for example, resistance training or carrying shopping) two times a week. Strength training is effective at keeping your weight healthy and benefits your cardiovascular health.

LDL cholesterol levels ^



Heart Health








What unites baboons, wild horses, and !Kung bushmen of the Kalahari desert? Compared to modern humans living in the industrialised world, they are all much less likely to develop fatty plaques that narrow their arteries (a process known as atherosclerosis). Interestingly, blood tests reveal that these groups, as well as other hunter-gatherer communities, also have significantly lower plasma levels of a particular lipid: LDL cholesterol. Cholesterol, a fat-like substance that is used to make cell membranes, certain hormones, and the insulation around our nerves, is transported around our body in particles known as lipoprotein particles. Smaller particles, such as low density lipoproteins (LDL), however, can easily penetrate into the linings of our arteries, where they deposit cholesterol that forms fatty plaques. In this respect, high levels of LDL cholesterol (LDL-C) are causally linked to atherosclerosis and associated cardiovascular diseases, such as coronary heart disease and stroke. Our lifestyle has a strong impact on our LDL-C level, and it is the healthier diets and greater physical activity that underlie the lower LDL-C level and cardiovascular disease risk observed in hunter-gatherer communities. Gene variants that affect how we absorb, transport, and metabolise cholesterol also affect our blood LDL-C level. In this trait, we give you a genetic score based on up to 37 gene variants linked to changes in LDL-C level. These were selected from a wider polygenic risk score shown to explain 12% of individual differences in LDL-C levels in the Framingham Heart Study. We also explain the impact of any lifestyle risk factors you have and give you advice on how to reduce your LDL-C level and lower your risk of cardiovascular disease.

Your result



Moderately
higher

Your genetic score is associated with average blood LDL-C levels.

-  **Slightly higher** LDL cholesterol is strongly linked to atherosclerosis: the formation of cholesterol-rich plaques in the lining of your
-  **Marginally higher** arteries. For every 1 mmol/L (39 mg/dL) increase in LDL-C level, your risk of cardiovascular diseases such as coronary
-  **Average levels** artery disease and stroke, is thought to increase by around 20%.
-  **Marginally lower** You should aim to maintain an LDL-C level below 3 mmol/L (115 mg/dL). If you have other cardiovascular risk factors, you
-  **Slightly lower** may need to target a lower LDL-C level. Speak to your GP or physician.
-  **Moderately lower** In this trait, we give you a genetic score based on up to 37 gene variants linked to small increases and decreases in
-  **No data** blood LDL-C level.

Your genetic score is associated with AVERAGE blood LDL-C levels.

Overall, your genetic score is associated with small changes in LDL-C level (roughly 0.01 mmol/L (0.4 mg/dL) compared to someone without your variants).

Based on our customer data, you carry an average number of gene variants linked to increases in LDL-C level. We would expect roughly 70% of the population to have a similar genetic score.

Your genetic score is associated with an average risk of cardiovascular disease (CVD). An unhealthy lifestyle, however, will elevate your blood LDL-C level and put you at higher CVD risk.

Several studies show that you can reduce your LDL-C levels by around 10-15% by adopting a healthy diet and performing regular exercise. This will also cut your risk of cardiovascular disease.

Your results are based on genetic and basic lifestyle data only. A blood test and other diagnostic tests are required to accurately measure your blood LDL cholesterol level and assess your cardiovascular risk.

Recommended Actions

Perform resistance exercise 3 times a week at a load of at least 75% of 1RM. High load resistance exercise is effective for maintaining healthy LDL cholesterol levels, particularly when combined with aerobic exercise.

Swap butter for avocado on your toast a few mornings each week. Foods high in saturated fat are shown to increase LDL cholesterol levels, but substituting these with foods rich in monounsaturated fats, such as avocados, will help you maintain healthy LDL levels.

If you are a heavy coffee drinker, avoid unfiltered coffee such as French press. Studies have found unfiltered coffee can cause increases in LDL cholesterol, particularly if you have more than 5 cups a day.

Have 1 or 2 plant-based meals a week. Swapping out meat with vegetables or beans is a great way to moderate your saturated fat intake, while also increasing your intake of fibre. This will help to keep your LDL cholesterol levels within a healthy range.

Moderate your intake of highly processed foods such as fast foods, deli meats, or microwave meals. These are usually very high in saturated and trans fats, which can cause increases in LDL cholesterol levels.

If currently smoking, try nicotine replacement (for example, gums/patches) or talk to your clinician about stop smoking programmes. Smoking reduces HDL cholesterol, which plays a role in removing excess LDL cholesterol, leading to raised LDL levels in smokers.

Perform some cardio exercise every day, such as going for a brisk walk, or cycling to work. Being active regularly will help you to maintain healthy cholesterol levels and minimise your risk of cardiovascular disease.

Eat an apple a day. Polyphenols, which are found in apples, may help to improve cholesterol regulation. Apples often contain pesticide residues, so ensure you rinse them thoroughly before eating.

Lipoprotein (a) levels ^



Heart Health

Lipoproteins are particles that transport cholesterol in the bloodstream. You've probably already heard of LDL (low density lipoprotein), which are what we term "pro-atherogenic" particles. This means they can penetrate the linings of your arteries, deposit cholesterol and form fatty plaques that obstruct blood flow to vital tissues and organs. As a result of this process, known as atherosclerosis, high LDL levels increase our risk of cardiovascular diseases such as heart attack and stroke. A lesser known particle that is even more pro-atherogenic than LDL is lipoprotein a (Lp(a)). In fact, a recent study estimated that a single Lp(a) particle is around 6 times more pro-atherogenic than an LDL particle. In line with this, high Lp(a) levels are an established risk factor for atherosclerotic cardiovascular disease. Lp(a) levels can vary by up to 1,000-fold between individuals, and it is thought that between 75% and 95% of this variance is driven by differences in our genetics. Although the majority of us will have Lp(a) levels towards the lower end of this spectrum, some of us carry genetic variants that can significantly increase Lp(a) levels. In this trait, you will find out whether you carry gene variants that elevate your Lp(a) levels and increase your risk of cardiovascular disease.

Your result



**Highly
elevated**

Your genetic score is associated with mildly elevated Lp(a) levels.



**Moderately
elevated**



**Mildly
elevated**

Lipoprotein (a) or Lp(a) is a particle that transports cholesterol in the bloodstream. Cholesterol in Lp(a) particles is more likely to be deposited in plaques in the lining of arteries: a process known as atherosclerosis.



Unelevated



No data

High Lp(a) levels therefore increase your risk of diseases linked to atherosclerosis, such as angina, heart attack, stroke,

and peripheral vascular disease. These are known collectively as atherosclerotic cardiovascular disease (ASCVD).

Rare variants of the LPA, PLG, and LPAL2 genes can increase the levels of Lp(a) circulating in your bloodstream. This can put carriers at greater risk of developing ASCVD.

In this trait, we look at up to 17 gene variants that have been associated with changes in Lp(a) level in subjects from UK Biobank and PROCARDIS studies.

Your genetic score is associated with MILDLY ELEVATED Lp(a) levels.

Your genetic score is linked to a 0 - 50 nmol/L (0 - 20 mg/dL) higher Lp(a) level (compared to someone without your gene variants).

Your genetic score is associated with up to roughly a 10% higher risk of atherosclerotic cardiovascular disease (ASCVD).

Your result is based on gene variants only. A blood test (serum Lipoprotein (a) level) is required to accurately assess your Lp(a) levels.

An optimal Lp(a) level is less than 25 nmol/L (10 mg/dL). An Lp(a) level greater than 75 nmol/L (30 mg/dL) is widely linked to a higher risk of ASCVD, and each 50 nmol/L (20 mg/dL) increase is associated with an 11% higher risk of ASCVD.

Studies suggest Lp(a) levels are difficult to target directly. Optimising your levels of LDL and HDL-cholesterol, however, will help to minimise your risk of atherosclerotic cardiovascular disease (ASCVD).

Recommended Actions

Consult your clinician about getting your Lp(a) and other lipid levels checked. Getting insight into your current blood levels of these lipid markers will provide a more accurate assessment of your current cardiovascular risk.

Add a tablespoon of flaxseeds to your oats, smoothies or yoghurt. Regular intake of flaxseeds has been shown to induce modest reductions (up to 4 mg/dL) in Lp(a) levels.

Flavour your meals with turmeric. Curcuminoids, found in turmeric, have Lp(a) lowering effects.

Include 200 g (1 cup) of lentils or kidney beans into one of your meals. These are great sources of soluble fibre which has been shown to help keep your total and LDL cholesterol levels healthy. Healthy cholesterol levels are important for minimising your lifetime risk of cardiovascular diseases.

Treat yourself to a piece of dark chocolate (at least 70% cocoa) every now and again. Flavanols found in dark chocolate have been shown to help keep cholesterol levels healthy, with chocolate being more effective than cocoa-containing drinks.

Add 2-3 days of moderate to high intensity resistance exercise each week. Resistance exercise, when combined with aerobic exercise, can further improve your cholesterol levels.

Perform moderate intensity activity for at least 30 mins, 5 times a week. Being active at a moderate intensity - where you can still speak but not sing - has been shown to have beneficial impacts on cholesterol levels and cardiovascular health.

Opt for fresh or frozen fruit, instead of jams or dried fruits, as these are lower in sugar. Making swaps like this can help to lower LDL cholesterol and improve your overall blood lipid profile.

Add a black or green tea into your day. Teas are rich in polyphenols - anti-inflammatory compounds that help to improve cardiovascular health, and have shown potential to have Lp(a) lowering effects.

Salt-sensitive hypertension ^



Heart Health

Are you more likely to be salt-sensitive? Over a quarter of us have “salt-sensitivity” - a trait whereby our blood pressure changes more greatly in response to how much salt (sodium) we consume in our diet. For salt-sensitive individuals, increasing salt intake leads to more drastic elevations in blood pressure and vice versa: cutting down on salt leads to greater reductions in blood pressure. Studies show that salt-sensitive individuals are also more likely to develop high blood pressure (known as hypertension). This combination of being salt sensitive and having high blood pressure is known as salt-sensitive hypertension and is strongly influenced by our genetic makeup. In particular, genes that affect the response of our kidneys and blood vessels to sodium intake may contribute to both salt sensitivity and the development of high blood pressure. In this report, you will find out if you carry some of these genes and receive advice on how to cut your sodium intake to healthy levels.

Your result



Higher
genetic
and
lifestyle
risk

You do not carry risk genotypes linked to salt-sensitive hypertension.

People who are salt-sensitive experience significant changes in blood pressure in response to how much salt (sodium) they consume in their diet.



Higher
genetic
risk





If you are salt-sensitive, your blood pressure rises sharply in response to increased salt intake, but falls when you reduce the salt content of your diet.



Low
genetic,
high
lifestyle
risk

Salt-sensitivity is thought to be due to an overactive renin-angiotensin aldosterone system (RAAS) - a network of hormones that acts to increase our blood pressure. In salt-

Moderate
genetic,

-  **high lifestyle risk** sensitive individuals, the activity of these hormones is not dampened down in response to high salt intake.
-  **Moderate genetic risk** Around 50% of individuals with high blood pressure (hypertension) are salt-sensitive. These people are described as having salt-sensitive hypertension.
-  **Lower genetic risk** Our genetic make-up strongly influences whether or not we are salt sensitive and our risk of developing high blood pressure. In this trait, we look at variants of your ADRB2, CYP11B2, PRKG1, and SLC8A1 genes that are linked to a higher risk of salt-sensitive hypertension.
-  **No data**

Your genetic risk score is: LOWER. You do not carry risk genotypes linked to salt-sensitive hypertension.

In a study of people with hypertension, those with your genetic risk score were not more likely to experience elevations in blood pressure when increasing their salt intake.

Your result is based on genetic data only. One method to assess salt sensitivity is to follow a low salt diet (e.g. less than 3 g per day) for 1 week, before switching to a high salt diet (e.g. 20 g per day) the following week. If your blood pressure changes by 3 to 5 mm Hg or more, you may be considered salt sensitive. (Please consult a doctor before trying this protocol).

High salt intakes can still increase your risk of developing hypertension (high blood pressure) and cardiovascular disease, regardless of your genetic make up. The World Health Organisation (WHO) currently recommends limiting your salt intake to below 5 g (just under a teaspoon) per day.

Recommended Actions

Keep your daily salt intake to below 5 grams (approximately 1 teaspoon). Consuming over this amount regularly can lead to poorer cardiovascular health.

Do not lower your salt intake to below 2 grams per day. Sodium - found in salt - is an essential nutrient that plays important roles in fluid balance, cell function and in our nervous system. Too low an intake can impair these vital functions.

Use herbs and spices to season food instead of adding salt. Opting for adding black pepper, chilli or rosemary for example are better alternatives to salt to help keep your sodium intake lower and your blood pressure healthy.

Make your own sauces and dressings instead of using store-bought ones. These can be high in salt so making your own allows you to better control the salt content.

Familial Hypercholesterolaemia ^



Heart Health

Affecting between 1 in every 200 to 500 people worldwide, familial hypercholesterolaemia (FH) is one of the most common inherited conditions to affect humans. The condition is typically caused by mutations that reduce the ability of the liver to clear LDL cholesterol circulating in the bloodstream. Consequently, people with familial hypercholesterolaemia have marked elevations in their blood LDL cholesterol levels from birth. If untreated, high LDL cholesterol levels significantly increase the risk of developing cardiovascular diseases such as coronary artery disease. As it is an inherited condition, those with FH will often have a first-degree relative who also has elevated LDL cholesterol levels or a history of premature cardiovascular disease (before the age of 60) in their family. Most people with FH have what is known as heterozygous familial hypercholesterolaemia. This means they carry one copy of a mutation that strongly elevates LDL cholesterol levels. More rarely, about 1 in 300,000 people carry two copies of a mutation and have homozygous familial hypercholesterolaemia, which results in extremely high LDL cholesterol levels from childhood. There have been thousands of mutations linked to FH, with the most common of these affecting the LDLR, APOB, and PCSK9 genes. In this trait, we look at up to 22 common mutations (21 in your LDLR gene and 1 in your APOB gene) known to cause FH. **IMPORTANT DISCLAIMER:** Please note this is not a diagnostic test for familial hypercholesterolaemia and the information in this report should not be used as a substitute for diagnosis, treatment, and guidance from a qualified medical practitioner. You are advised to consult a physician for further information, assessment, and support.

Your result



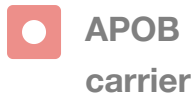
APOB

homozygote

LDLR

and

You do not carry LDLR or APOB mutations linked to familial hypercholesterolaemia (FH).



APOB
carrier



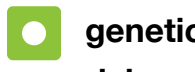
APOB
carrier



LDLR
carrier



LDLR
homozygote



Lower
genetic
risk



No data

Familial Hypercholesterolaemia (FH) is a genetic condition that causes abnormally high levels of LDL cholesterol (LDL-C) from birth. People with this condition have a higher risk of developing cardiovascular disease.

Between 60% and 80% of people with FH carry a mutation in their LDLR gene. Over 2000 different LDLR mutations are thought to cause FH; we analyse your DNA for up to 21 of the most common of these.

About 5% - 10% of people with FH carry a mutation in a different gene: APOB. Over 100 APOB mutations have been linked to FH; we analyse your DNA for one of the most common of these.

FH is typically an autosomal dominant condition. This means you only need to inherit one copy of an LDLR or APOB mutation to have the condition. More rarely, people can carry two copies of a mutation.

You do not carry LDLR or APOB mutations linked to familial hypercholesterolaemia (FH).

Please note that your result is based on analysis of up to 21 LDLR and 1 APOB mutation/s only. We have not analysed the over 2000 other mutations in LDLR, APOB, and other genes known to cause FH.

An unhealthy lifestyle that elevates your LDL cholesterol levels will also increase your risk of cardiovascular disease, regardless of your genetic makeup.

For a more accurate report on how your genetics and lifestyle affect your individual cardiovascular risk, please provide us with a recent blood LDL cholesterol level reading in your Lifestyle Profile.

High blood LDL-C levels, a family history of premature cardiovascular disease, and cholesterol deposits in your eyes

and tendons may also indicate the presence of FH.

Recommended Actions

Get your cholesterol levels checked. A blood test to measure your current cholesterol and triglyceride levels is important for understanding if they are raised or not. It also allows for better assessment of your cardiovascular risk.

Perform resistance exercise 3 times a week at a load of at least 75% of 1RM. High load resistance exercise is effective for maintaining healthy LDL cholesterol levels, particularly when combined with aerobic exercise.

Perform some cardio exercise every day, such as going for a brisk walk, or cycling to work. Being active regularly will help you to maintain healthy cholesterol levels and minimise your risk of cardiovascular disease.

Take the stairs over the elevator or escalators. Increasing physical activity in your everyday life is an easy way to increase your activity levels, which will have benefits for your cardiovascular health.

Cook homemade meals as much as you can, using fresh ingredients. Opting for homemade meals over pre-prepared or processed foods is an easy way to reduce saturated fat intake and better control your cholesterol levels.

Inflammation and IL-6 levels ^



Inflammation

IL-6 (interleukin 6) is a cell communication protein or 'cytokine' that helps coordinate the body's inflammatory response. Although inflammation can be a useful short-term response for dealing with harmful stimuli such as bacteria, toxic compounds and tissue injury, persistent or "chronic" inflammation can be damaging to our health. Chronic inflammation is thought to play a role in poor insulin sensitivity, type II diabetes, cardiovascular disease, ageing, and neurodegenerative disease. This trait analyzes gene variants related to the levels and activity of IL-6, which can influence your susceptibility to chronic inflammation.

Your result



**Higher
Risk**

You carry gene variants associated with higher IL-6 production, which increases your susceptibility to chronic inflammation.



**Moderately
Higher
Risk**



**Average
Risk**

IL-6 is key for controlling and modifying the inflammatory response. IL-6 is therefore an important modifier of inflammatory and auto-immune related diseases and conditions.

Genetic variants can alter the amount of IL-6 produced in response to inflammatory stimulus. This can make you more susceptible to chronic inflammation, thus impacting your metabolic health.

Genetic variants can also increase the amount of the IL-6 receptor, further modifying the acute and chronic inflammatory responses.

Our analysis shows that you have genetic variants that increase IL6 levels, and that increase IL6 receptor levels.

If you put on body fat, you will be more sensitive to metabolic issues that lead to further weight gain and suboptimal insulin function.

In addition, it is worth noting that many other genetic and non-genetic factors control inflammation, and thus can cause inflammatory and auto-immune related conditions.

Recommended Actions

Ensure that you are monitoring your training load effectively.

There is a direct correlation between training load and morning serum IL-6 levels.

Reduce intake of highly-processed and high glycemic load foods. Look to fill your diet with plenty of fruit and vegetables which contain important antioxidants and do not cause frequent fluctuations in blood sugar levels.

Try mindfulness meditation, tai-chi, qi-gong or breathing techniques. Mindfulness, which involves taking time to listen to your body, is a great way to de-stress and improve your mental well-being.

Ensure you are consuming vitamin C & vitamin E rich-foods within your diet as it can protect against free radical and exercise induced inflammation. Good food sources include broccoli, kiwis, berries, sunflower seeds, avocados.

Supplement daily with 400 mg of curcumin. Curcumin has been shown to improve insulin sensitivity by lowering blood sugar (and fatty acid) levels. The absorption and effects of curcumin are enhanced when it is combined with black pepper extract (piperine).

Eat foods high in antioxidants, such as berries, tea and soybeans. Antioxidants are molecules that can prevent certain types of cell damage and lower inflammation, which can help speed up your recovery after exercise.

Oxidative stress risk (NQO1) ^



Inflammation

Oxidative stress is a damaging process linked to inflammation, ageing, cardiovascular disease and cancer. It results from the accumulation of highly reactive, harmful substances in the body called free radicals and Reactive Oxygen Species (ROS). Our body has several mechanisms to protect against oxidative stress, including antioxidants, which neutralise free radicals and ROS. This trait looks at gene variants that affect the activity of NQO1: an enzyme that helps produce antioxidants and protect against oxidative stress.

Your result



Higher
Risk



Lower
Risk



No Data

You have average NQO1 activity, which helps to protect your cells from oxidative stress.

NQO1 is an enzyme that helps with the production of various antioxidants, including Coenzyme Q10 (CoQ10 - also known as ubiquinone) and certain forms of Vitamin E.

In addition, NQO1 protects against oxidative stress by preventing the production of damaging ROS molecules.

NQO1 is also involved in the detoxification of benzene, a harmful chemical found in tobacco smoke, vehicle exhaust, and some glues, paints and detergents.

A SNP (rs1800566) of the NQO1 gene creates two variants (alleles): 'C' and 'T'. Inheriting two copies of the 'T' allele (i.e. TT genotype) results in significantly lower NQO1 enzyme activity.

You carry at least one copy of the C allele.

You have average NQO1 activity.

Based on NQO1 gene variants alone, you are not more susceptible to cell damage from oxidative stress.

Based on NQO1 gene variants alone, your body effectively detoxifies benzene found in tobacco smoke, vehicle exhaust and some glues, paints and detergents.

This trait focuses specifically on NQO1 gene variants. Other traits (e.g. SOD2), genes (e.g. GPx-1) and lifestyle factors (e.g. diet, smoking status) influence your susceptibility to oxidative stress.

Recommended Actions

Optimise your vitamin B2 levels. B2 (riboflavin) is a precursor for FAD, which is a key coenzyme for stabilising NQO1.

Make sure you are consuming enough vitamin E. NQO1 reduces vitamin E into its active antioxidant form, providing the beneficial effects against oxidative stress.

Try to perform at least 150 minutes of moderate-intensity activity every week. Regular physical activity keeps your endogenous antioxidant defences high, as well as activating the Nrf2 pathway to keep NQO1 stimulated.

Eat high antioxidant foods such as berries, lychee, grapes, broccoli, bok choy. These will help to keep inflammation levels low.

Oxidative stress risk (GPx-1) ^



Inflammation

GPx-1 (glutathione peroxidase 1) is an enzyme that helps to prevent the build up of Reactive Oxygen Species (ROS) - highly reactive substances that can damage cells. When ROS accumulate at a greater rate than they are neutralised and cleared, we call it 'oxidative stress'. Oxidative stress is a highly damaging process and linked to negative health outcomes such as inflammation, aging and cancer. This trait looks at variants of your GPX1 gene, which affects the activity of your GPx-1 enzyme. In turn, this influences your susceptibility to oxidative stress.

Your result



**Higher
Risk**



**Lower
Risk**

You carry gene variants linked to normal GPx-1 activity, making you less susceptible to cell damage from oxidative stress.

GPx-1 is an antioxidant enzyme that helps your cells remove a harmful substance called hydrogen peroxide.

Hydrogen peroxide is an example of a Reactive Oxygen Species (ROS) - an oxygen-derived substance that can damage important molecules such as DNA, proteins and lipids.

Levels of hydrogen peroxide (and other ROS) can rise if they are not cleared effectively by antioxidant systems. This is known as 'oxidative stress'.

Oxidative stress is a damaging process linked to inflammation, ageing, cardiovascular disease and cancer.

The activity of your GPx-1 enzyme influences the levels of hydrogen peroxide that may accumulate within cells. This affects your susceptibility to oxidative stress.

This trait looks at variants of your GPX1 gene, which codes for and affects the activity of your GPx-1 enzyme.

This trait focuses on the rs1050450 C>T SNP. You carry two copies of 'C' allele associated with normal GPx-1 enzyme activity.

Based on this gene result alone, you are not more susceptible to oxidative stress.

Be sure to also check your 'SOD2 and metabolism' trait result. SOD2 is another antioxidant enzyme that clears ROS substances.

Reduced SOD2 activity can increase your susceptibility to oxidative stress.

This trait focuses on GPx-1 activity. Your susceptibility to oxidative stress is affected by the function of other antioxidant systems in your body, as well as lifestyle factors e.g. diet, exercise, alcohol intake, smoking and exposure to pollutants.

Recommended Actions

Monitor your copper and iron levels. High levels of these minerals can impair the action of glutathione peroxidases.

Keep your diet high in sulfur-rich foods like eggs, onions, garlic or broccoli. Sulfur is found in the amino acids required to synthesise glutathione, and therefore glutathione peroxidases.

Eat foods high in selenium, such as brazil nuts, shellfish, eggs and mushrooms. Chronic low levels of selenium can impair the function of glutathione peroxidase and increase the risk of oxidative stress.

Consume vitamin C & vitamin E rich-foods within your diet. These vitamins are antioxidants as well as aiding the formation of glutathione, and therefore glutathione peroxidases.

Perform frequent moderate to high intensity exercise. One of the adaptations induced by regular exercise is the increased levels of antioxidant defences including glutathione peroxidase.

Oxidative stress (SOD2) ^



Inflammation

SOD2 (superoxide dismutase 2) is an enzyme that helps to clear away harmful free radical molecules. Free radicals are highly reactive substances that can damage DNA, proteins, lipids and other cell components. During respiration, the process by which cells generate energy, our mitochondria produce free radicals called reactive oxygen species (ROS). Your SOD2 enzyme prevents the build-up of ROS by converting them into less harmful molecules. Reduction in the activity of SOD2, however, can lead to an imbalance between the production and clearance of ROS - a state known as oxidative stress. This trait looks at variants of your SOD2 gene, which affects the activity of the SOD2 enzyme and your susceptibility to cell damage from oxidative stress.

Your result



**Higher
Risk**

You carry gene variants linked to average SOD2 activity, which may provide less protection against oxidative stress.



**Average
Risk**



**Lower
Risk**

SOD2 removes toxic superoxide anion radicals (reactive oxygen species (ROS)) which can be toxic to biological systems.



No Data

SOD2 controls processes that have been linked to premature aging, motor neuron dysfunction and cardiovascular issues.

SNPs associated with the SOD2 locus have been linked to higher SOD2 activity.

You have variants that are not linked to increased SOD2 activity.

You may therefore respond to chronic oxidative stress less favourably.

However, you may have better acute ROS signalling, an important process for muscle growth and repair.

Recommended Actions

Make sure you are consuming enough vitamin E as this vitamin has a positive impact in the prevention of damage caused by free radicals.

Consume manganese rich foods such as nuts, seeds, beans, legumes, oats, and leafy green vegetables. Manganese is a cofactor required for mitochondrial SOD activity.

Cook with the herbs rosemary and sage. These contain compounds that increase the activity of superoxide dismutase, reducing inflammation and oxidative stress.

Consider adding a high dose vitamin C supplement (1000-2000 mg per day). Vitamin C plays a role in reducing inflammation through increasing SOD activity levels.

Try to perform at least 150 mins of moderate-intensity exercise each week. Regular exercise upregulates SOD levels in the mitochondria, protecting against oxidative stress.

Take 400 mg of curcumin, with 10 mg of piperine to aid absorption, daily. Curcumin has been shown to increase superoxide dismutase activity, increasing antioxidant effects.

Drink green tea. It, and other teas, contain EGCG (epigallocatechin gallate) which can increase the activity of SOD2 and reduce levels of inflammation.

Avoid taking vitamins C and E near a workout. This will prevent the exercise-induced free radical increase needed to stimulate the upregulation of SOD mRNA from exercise.


Homocysteine levels ^



Heart Health

In 1969, the pathologist Kilmer McCully performed a post-mortem on a child who had suffered from homocystinuria, a rare inherited condition characterised by a high amount of the amino acid homocysteine found in urine. McCully discovered that the arteries of the child were thickened, narrowed, and full of plaques. Building on this observation, McCully came up with the homocysteine theory of arteriosclerosis, in which elevated homocysteine levels in the body (not necessarily restricted to those with rare genetic disorders) cause damage to blood vessels and, more broadly, increase the risk of cardiovascular disease. While this theory was contentious at first, the following decades have seen more evidence linking homocysteine to cardiovascular conditions such as stroke and coronary artery disease. In this trait, you'll receive a genetic risk score based on several gene variants associated with changes in blood plasma levels of homocysteine. Based on your score, we provide personalised recommendations to limit the accumulation of homocysteine in your bloodstream, optimise your intake of folate and other B vitamins (which help to lower homocysteine levels), and improve your cardiovascular health.


Your result

 Above average levels

Your genetic score is associated with slightly lower plasma homocysteine levels.

 Average levels

Homocysteine (Hcy) is an intermediate amino acid that is formed when our bodies convert methionine, an essential amino acid that we need to obtain from protein in our diet, into cysteine, another amino acid used to make various proteins.

 Below average levels

 No data

Hcy circulating in our bloodstream has been shown to damage the cells lining the inside of our blood vessels

(vascular endothelium). This can trigger the formation of fatty plaques in arteries (atherosclerosis) and increase the risk of cardiovascular diseases, such as stroke and coronary artery disease.

In this respect, several observational studies show that elevated plasma Hcy levels are associated with a higher risk of cardiovascular disease. For example, a 2020 meta-analysis of 6 prospective cohort studies found that every 1 micromol/L increase in plasma Hcy level (above 6.5 micromol/L) was associated with a 5-6% higher risk of stroke.

A healthy plasma Hcy level is typically considered to be less than 15 micromol/L. A plasma Hcy level of 15 micromol/L and above is diagnostic of hyperhomocysteinemia, which is linked to an increased risk of cardiovascular disease.

Plasma Hcy levels are influenced by your intake of various B vitamins, namely B6 (pyridoxine), B9 (folate), and B12 (cobalamin). These vitamins play important roles in two key metabolic pathways that regulate Hcy levels: the methionine and folate cycles. Low intakes of these vitamins have been linked to higher Hcy levels and a greater risk of cardiovascular disease.

Variants of genes involved in the methionine and folate cycles (e.g. MTHFR, MTR), as well as the wider absorption and metabolism of B vitamins (e.g. FUT2, NOX4), are shown to have small effects on your plasma Hcy levels.

In this trait, we calculate a genetic risk score based on variants of 13 genes linked to differences in Hcy level.

Together, these gene variants have been shown to explain 5.9% of differences in plasma Hcy levels in a sample of 5500 people from the Rotterdam Study.

Your Hcy level genetic risk score is: **BELOW AVERAGE**. Your genetic score is estimated to be lower than roughly 70% of the population.

Your genetic risk score is associated with: **SLIGHTLY LOWER PLASMA HOMOCYSTEINE (Hcy) LEVELS**. In the Rotterdam Studies I and II, which analysed Hcy levels in approx. 8000 people aged 55 and over, those with below average genetic risk scores had a plasma Hcy level roughly 1-3 micromol/L lower than those with the highest 30% of genetic risk scores.

A below average genetic risk score is also associated with a slightly lower risk of hyperhomocysteinaemia (defined as a plasma Hcy level of 15 micromol/L or higher). Analysis of the Rotterdam Studies I and II found that individuals with a below average genetic risk score were roughly 40% less likely to have hyperhomocysteinemia compared to someone with an average genetic risk score.

Lifestyle factors, including your diet, also have a large influence on your plasma homocysteine levels. Low folate intake, high coffee consumption, alcohol intake, and smoking have all been linked to elevations in plasma Hcy levels.

Several observational and interventional studies suggest that increasing your dietary intake of folate (vitamin B9), folic acid (the synthetic version of folate), and pyridoxine (vitamin B6) can help to reduce your plasma Hcy levels and lower your risk of cardiovascular disease. A 2020 meta-analysis of 12 observational studies found that a 100 microgram/day increase in folate intake and a 0.5 mg/day increase in B6 intake were associated with a 6% lower risk of stroke.

A fasting plasma homocysteine (Hcy) blood test is required to accurately assess your current Hcy levels. A normal plasma Hcy level is considered to be less than 15 micromol/L. Some

medical experts suggest a level of 10 micromol/L or less is recommended for reducing the risk of stroke.

Recommended Actions

Include plenty of dark green leafy vegetables in your diet such as spinach, asparagus, Brussels sprouts, and broccoli. These vegetables are good sources of folate (vitamin B9), which can help to reduce plasma homocysteine levels by driving the folate and methionine metabolic cycles.

Eat fresh tuna, salmon, and large eggs as part of your diet. These foods are great sources of vitamin B12. Deficiencies in vitamin B12 can raise homocysteine levels.

Supplement with a B vitamin complex if you do not consume high amounts of folate or B12 in your diet (for example, if you do not regularly consume foods such as beef, salmon, eggs, lentils, and black beans). Supplementing will ensure you are getting enough of these key B vitamins to limit homocysteine levels.

Moderate your alcohol intake or try alcohol-free alternatives. Alcohol intake can cause increases in homocysteine levels.

If currently smoking, try nicotine replacement (for example, gums/patches) or talk to your clinician about stop smoking programmes. Smoking increases homocysteine levels and has several other negative impacts on your cardiovascular health.

Blood triglyceride levels ^



Heart Health

Triglycerides are the most common form of fat in our bloodstream. When we consume fat in our diet, it is broken down into fatty acids. These are then reassembled into triglycerides and packaged into particles (called triglyceride-rich lipoproteins, TRLs) that are transported in our bloodstream to various organs, such as our muscles and fat tissue. After transporting triglycerides, however, TRLs can become enriched with cholesterol, another fat-like substance. These cholesterol-laden TRL particles, known as “remnants,” then deposit cholesterol in the walls of our arteries, forming fatty plaques that obstruct blood flow: a process known as atherosclerosis. Our blood triglyceride levels serve as a marker of the amount of TRLs and cholesterol-rich remnant particles circulating in our bloodstream. High blood triglyceride levels therefore reflect an elevated number of TRLs and remnant particles, and are an established risk factor for atherosclerotic cardiovascular diseases, such as coronary artery disease and stroke. In this trait, in addition to looking at your lifestyle risk factors, we analyse up to 30 gene variants linked to differences in blood triglyceride level. These were selected from a wider panel of gene variants shown to explain 9.6% of individual differences in triglyceride levels in the Framingham Heart Study.

Your result



Moderately
higher

Your genetic score is associated with slightly lower blood triglyceride levels.



Marginally
higher

An optimal (fasting) blood triglyceride level is less than 1.2 mmol/L (100 mg/dL). This is recommended by the European Atherosclerosis Society.






Marginally
lower



Slightly
lower

High blood triglyceride levels can promote atherosclerosis: the development of cholesterol-rich plaques in the walls of our arteries. A fasting triglyceride level greater than 1.7

-  **Moderately lower** 150 mmol/L (150 mg/dL) significantly increases the risk of atherosclerosis.
-  **Lower** In this trait, we give you a genetic score which collates several gene variants linked to small increases and decreases in blood triglyceride level.
-  **No data**

Your genetic score is associated with SLIGHTLY LOWER blood triglyceride levels.

Overall, your genetic score is associated with a roughly 0.006 to 0.018 mmol/L (0.23 to 0.69 mg/dL) lower blood triglyceride level (compared to someone without your variants).

Based on our customer data, you carry an average number of gene variants linked to changes in blood triglyceride level. We would expect roughly 70% of the population to have a similar genetic score.

Lower blood triglyceride levels are linked to a decreased risk of cardiovascular disease. A 2007 meta-analysis found that, after adjusting for other risk factors, people with triglyceride levels in the bottom third of the study population were 1.72 times less likely to have coronary artery disease (CAD) compared to those in the top third.

Your lifestyle also has a strong impact on your blood triglyceride levels. The National FINRISK Study found that someone with a BMI greater than 30 kg/m², who smokes, consumes excess alcohol, and undertakes less than 30 mins physical activity a week would be 14.4 times (men) and 9.7 times (women) more likely to have elevated triglyceride levels (>1.7 mmol/L (150mg/dL)) compared to someone with a healthy lifestyle.

Your results are based on genetic and basic lifestyle data only. A (fasting or non-fasting) blood test is required to accurately measure your blood triglyceride level. This is

usually part of a blood lipid profile, which also assesses your levels of total, LDL, and HDL cholesterol.

Recommended Actions

Try reducing your alcohol intake by opting for alcohol-free alternatives. Excessive alcohol intake is associated with hypertriglyceridemia (high levels of triglycerides).

When cooking, opt to bake, steam, or grill foods (over frying). This is an easy way to reduce the amount of fat in your diet, which will help to keep your blood triglyceride levels healthy.

Add steamed cauliflower or green beans to meals. These are great sources of insoluble fibre, which binds to dietary fats and slows down their absorption in the gut. This helps to lower blood triglyceride levels.

Exercise at a moderate intensity for at least 30 mins, 5 times a week. This can be going for a run or a brisk walk, or doing some household cleaning (vacuuming or mopping). Regular aerobic exercise helps to lower and maintain healthy triglyceride levels.