# Results Overview

### ATM and DNA damage

Your ATM gene variants are linked to greater susceptibility to DNA damage and uncontrolled cell growth.

Higher Risk

### DNA repair and longevity (TP53)

Your gene variants suggest that you are less effective at repairing DNA damage, which is linked to reduced longevity.

Higher Risk

### APOE and health

You do not carry gene variants that may negatively impact your cardiovascular, metabolic, and neuronal health in older age.

Average Risk

### APOE and inflammation

You carry APOE gene variants associated with an average risk of oxidative stress and inflammation.

Average Risk

### Detoxification rate (NAT1)

You have a reduced susceptibility to cell damage and ageing, due to faster clearance of toxic compounds.

Lower Risk

### Detoxification rate (NAT2)

You have an increased susceptibility to cell damage and ageing, due to slower clearance of toxic compounds.

Higher Risk

**BDNF activity and cognition**

Your gene variants are associated with average BDNF activation. Increasing BDNF activity can improve mood, learning, and memory.

Average Risk

**SIRT1 and neuroprotection**

You do not carry a copy of the gene variant linked to increased protection from brain ageing and higher cognitive function in old age.

Average Risk

**Cholesterol and ageing (CETP)**

Your gene variants are linked to longevity, reduced cell ageing, and higher levels of "good" HDL cholesterol.

Lower Risk

**Bone mineral density (VDR)**

Your gene variants are linked to an average bone mineral density.

Average Risk

**WNT16 and age-related bone mineral density**

Your gene variants and age are linked to lower bone mineral density.

Higher genetic risk

**Sarcopenia risk**

Your genetic risk of sarcopenia is moderate.

Moderate Genetic Risk

## FOXO3 and longevity

You do not carry gene variants that are more frequently found in people who live beyond the age of 90.

Average Risk

# ATM and DNA damage

⏳ Biological Ageing

Did you know that our DNA is continually being damaged throughout our lives? When cells replicate, they often make mistakes copying genetic material, causing mutated or damaged DNA. Other normal processes, such as cell respiration, can also damage DNA through the generation of harmful reactive oxygen species (ROS) molecules. Exposure to radiation, such as the UV-B rays in sunlight, can cause breaks in the DNA strand. Luckily, we've evolved various repair mechanisms that can fix these types of DNA damage. In this trait, we look at ATM gene variants that may impair these repair mechanisms, increasing your susceptibility to DNA damage.

## Your result

- **Higher Risk**
- Average Risk
- No Data

**Your ATM gene variants are linked to greater susceptibility to DNA damage and uncontrolled cell growth.**

The ATM (ataxia telangiectasia mutated) gene is involved in repairing damaged DNA, particularly double-strand breaks in the DNA molecule.

In response to DNA damage, the ATM gene activates various DNA repair mechanisms and prevents damaged cells from replicating.

Mutations within the ATM gene can impair these repair mechanisms and increase susceptibility to DNA damage.

The 'A' allele (rs664143) of the ATM gene is linked to greater susceptibility to DNA damage and uncontrolled cell growth.

DNA damage is associated with accelerated ageing and greater risk of cancers.

You carry the 'A' risk allele linked to greater susceptibility to DNA damage.

'A' allele carriers are shown to have a higher risk of DNA damage to cells in lung tissue.

Some evidence suggests that higher intakes of antioxidants (including Vitamins A, C, and folic acid) may help to reduce susceptibility to DNA damage in 'A' allele carriers.

Modifiable lifestyle factors that can increase your risk of DNA damage include: smoking, alcohol, being overweight, physical inactivity, low intake of fruit and vegetables, and exposure to ionising radiation and UV in sunlight.

## Recommended Actions

**Swap your coffee for low caffeine alternatives such as tea, ashwagandha or decaf.** Caffeine can inhibit ATM and reduce protection against DNA damage so moderating intake can be beneficial.

**For 5 days each week, spend 30 minutes doing some moderate intensity activity.** Regular activity such as a walk or cleaning the house, upregulate your internal antioxidants, increasing protection against oxidative stress and DNA damage.

**Introduce a level of calorie restriction either through a dietary or time restricted feeding protocol.** Calorie restriction has been shown to reduce DNA damaging reactive oxygen species (ROS), slowing down cellular ageing.

**Take precautions in heavily polluted areas e.g wear a face covering.** Car exhaust fumes and other air pollutants can lead to toxin build up and DNA damage, enhancing cellular ageing.

**Add one large sweet potato or 1 cup of kale to one of your meals each week.** These foods are high in Vitamin A, helping to boost your levels of this important antioxidant.

**Eat your fruits and vegetables raw whenever possible.** Cooking, especially boiling, strips the food of much of its water-soluble vitamins, such as vitamin C, which is an important antioxidant that helps minimise DNA damage.

**Supplement with up to 200 mcg folic acid or 10-15 mg of L-methylfolate per day.** Ensuring you are consuming adequate folate helps increase protection against DNA damage and reduce the risk of certain cancers.

**Use gloves when at petrol stations or using cleaning products.** You are at greater risk of benzene toxicity, so taking precautions when using substances that contain benzene is important for reducing the risk of further DNA damage.

**Look to stop smoking if currently doing so.** Smoking increases DNA damage and the risk of associated disease states.

# DNA repair and longevity (TP53)

⏳ Biological Ageing

Do you have more effective DNA repair mechanisms linked to greater longevity?Nicknamed the "guardian of the genome", p53 is a tumour suppressor protein that senses damage to our genes, initiates DNA repair mechanisms, and then instructs our cells to stop dividing. p53 can also instruct cells to "self-destruct" by activating a programmed process of cell death known as apoptosis. Far from being damaging, these processes are often beneficial as they stop damaged cells from replicating, block harmful mutations from being passed on to new cells, and prevent uncontrolled cell growth. In fact, without apoptosis, it's thought that an 80 year-old person would accumulate 2 tonnes of bone marrow and lymph nodes, and have a 16 km-long gut!Variants of your TP53 gene, which encodes p53, can alter the balance of these DNA repair, growth arrest and cell death processes. This in turn can affect our longevity and health in older age.

## Your result

- Higher Risk
- Moderately Low Risk
- Lower Risk
- No Data

**Your gene variants suggest that you are less effective at repairing DNA damage, which is linked to reduced longevity.**

p53 is a tumour suppressor protein that repairs damaged DNA and prevents growth of damaged cells.

p53 also initiates apoptosis - a process of programmed cell death that allows tissues to eliminate damaged and abnormal cells.

The p53 protein is encoded by the TP53 gene. The 'G' allele (rs1042522) of this gene codes for a p53 protein that more effectively initiates apoptosis, but less effectively repairs DNA.

The 'C' allele (rs1042522) of the TP53 gene codes for a p53 protein that is better at repairing damaged DNA and preventing damaged cells from replicating. This allele has been linked to greater longevity.

You carry two copies of the 'G' allele linked to less effective DNA repair, but enhanced apoptosis. Your TP53 genotype is GG.

Enhanced apoptosis may be beneficial for preventing tissue damage in younger people, but may reduce lifespan and increase risk of critical illness in older individuals.

Your genotype is not linked to greater longevity and is associated with a 2-3 year shorter lifespan than 'C' allele carriers.

Some studies have associated your genotype (GG) with greater susceptibility to age-related illnesses, although findings are mixed.

## Recommended Actions

**Supplement with up to 200 mcg of folic acid or 10-15 mg of L-methylfolate per day.** Ensuring you are consuming adequate folate helps increase protection against DNA damage.

**Try supplementing with 1000 IU of vitamin D each day.** Vitamin D deficiency can cause DNA damage so ensuring you consume enough is important for protection.

**Add blueberries and grapes to your fruit bowl.** These fruits are high in important polyphenols which help protect against cellular damage, with one study showing a 20% reduction in DNA damage in those who consumed blueberries daily.

**Fill half your dinner plate with vegetables such as cabbage, Brussels spouts and cauliflower.** Cruciferous vegetables, such as these, have been shown to increase DNA repair and can help the liver detoxify potential cancer-producing compounds.

**Do one resistance workout in a fasted state.** Studies have shown resistance training when in a low glycogen state (e.g. when fasted) can acutely increase p53 activity, promoting DNA repair mechanisms.

**Eat plenty of broccoli.** Broccoli contains kaempferol and indole-3-carinol (I3C) which have both been shown to help increase DNA repair and protection against cellular damage.

**Switch out your morning coffee for a green tea.** Caffeine can interfere with some DNA repair processes, so switching to a low caffeine drink is beneficial. Green tea also upregulates p53 expression and reduces oxidative stress-induced damage.

**Take 400-500 mg of curcumin, with 10-20 mg of piperine to aid absorption, daily.** Curcumin has been shown to upregulate p53 expression and help increase protection against cellular damage.

# APOE and health

Brain Health

ApoE (apolipoprotein E) is a molecule involved in the transport, metabolism and clearance of lipids (fat and cholesterol) in the bloodstream. It also plays a key role in the delivery of cholesterol to the central nervous system for the growth and repair of nerves. ApoE is therefore important for maintaining the health of various tissues, including the brain, heart, liver and blood vessels. There are three major forms of ApoE (E2, E3, and E4), which are coded for by variants of your APOE gene.This trait analyzes which APOE gene variants you carry, which, in turn, influences the way you metabolize lipids. This has wider effects on your risk of cardiovascular and age-related illness, and physical and mental health.

## Your result

Higher Risk

Average Risk

Lower Risk

No Data

**You do not carry gene variants that may negatively impact your cardiovascular, metabolic, and neuronal health in older age.**

Apolipoprotein E (APOE) is a protein involved in the transport and metabolism of fat and cholesterol in the body.

APOE is the primary cholesterol carrier in the brain, and is important for cardiovascular, metabolic and neuronal health and function.

Genetic variants around the APOE locus impact the way lipoproteins are formed and how cholesterol is metabolised. These variants can increase the risk of age-related illness.

You have only a minor burden of variants linked to this pathway.

However, it is important to be aware of actions that promote good brain and metabolic health.

## Recommended Actions

**Lower the proportion of your dietary fat that comes from saturated fats.** Opt for poly and monounsaturated fats instead. Good sources are fish, nuts, seeds, avocados, olive oil and soybeans.

**If not regularly consuming two portions of oily fish a week, consider supplementing with an omega-3 supplement such as fish, krill or algal oil.** Omega-3 fatty acids have been shown to be increase learning, memory, cognitive well-being, and brain blood flow.

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

**Look to add more plant-based foods that are also high in soluble fibre to your diet.** Foods such as kidney beans, lentils, rolled oats, black beans, nuts and seeds can help reduce total and LDL cholesterol levels.

**Make sure you are involved in moderate-intensity exercise, such as 40 minutes of brisk walking, at least 3 times a week.** Physical activity will keep you metabolically healthy and boost brain health too.

**Increase the amount of non-exercise physical activity (e.g stair climbing, standing, carrying shopping) you perform daily.** Being active generally within our lifestyles is important for physical and mental health.

**Perform mental exercises such as reading, puzzles, or learning a new skill.** Regularly doing activities like these are beneficial for increasing connections and plasticity of the brain; keeping you neurologically healthy.

**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.

# APOE and inflammation

Inflammation

ApoE (apolipoprotein E) is a specialized type of protein involved in the transport and metabolism of lipids (fat and cholesterol) as well as the regulation of inflammation. There are three major forms of ApoE (E2, E3, and E4), which differ in their ability to suppress inflammation and the closely-related process of oxidative stress. This trait analyses variants of your APOE gene, which governs what form of ApoE you produce. In turn, this influences the susceptibility of various tissues to inflammation.

## Your result

Higher Risk

Moderately Higher Risk

**Average Risk**

Lower Risk

No Data

**You carry APOE gene variants associated with an average risk of oxidative stress and inflammation.**

ApoE stands for apolipoprotein E. Apolipoproteins are a class of molecules found on the surface of particles (called lipoproteins) that transport fat and cholesterol around the body.

ApoE influences the way different tissues (including those in the cardiovascular and central nervous systems) store and use fat and cholesterol.

ApoE has also been shown to protect against oxidative stress and suppress inflammation in brain tissue and blood vessel linings, partly by altering the way these tissues metabolise fat and cholesterol.

There are three different forms (or 'isoforms') of ApoE: ApoE2, ApoE3, ApoE4. These differ in their anti-inflammatory effects and therefore alter your risk of inflammation.

The ApoE isoform you produce, and therefore your risk of inflammation, depends on what variants of the APOE gene

you inherit.

You carry APOE variants associated with an average risk of oxidative stress and inflammation.

You are at a higher risk of damage to your cardiovascular system in response to smoking.

This trait looks at APOE gene variants only. Other traits (e.g. Inflammation and IL-6 levels, Oxidative stress risk), genes, and lifestyle factors (such as diet, exercise, sleep and psychological stress) also influence inflammation within the cardiovascular and central nervous systems.

## Recommended Actions

**Look to stop smoking if currently doing so.** Smoking contributes to increased inflammation as well as increasing the risk of poor metabolic health.

**Keep your body fat levels healthy.** Excess body fat can contribute to inflammation, and has been associated with increased neuroinflammation.

**Eat high antioxidant foods such as berries, lychee, grapes, broccoli, bok choi.** Antioxidants prevent certain types of cell damage and help to keep your risk of excess inflammation reduced.

# Detoxification rate (NAT1)

⏳ Biological Ageing

NAT1 (Arylamine N-acetyltransferase) is an enzyme that helps carry out acetylation, an important reaction involved in the detoxification of certain drugs and harmful environmental compounds. This trait looks at variants of your NAT1 gene, which affect your rate of acetylation. This, in turn, influences how effectively your body breaks down and clears damaging compounds, including heterocyclic amines (HCAs) found in tobacco smoke, diesel exhaust and meat cooked at high temperatures.

## Your result

Higher Risk

Moderately Higher Risk

**Lower Risk**

**You have a reduced susceptibility to cell damage and ageing, due to faster clearance of toxic compounds.**

NAT1 (Human arylamine N-acetyltransferase 1) is an enzyme responsible for a key class of reaction called acetylation.

Acetylation activates and detoxifies environmental chemicals - including HCAs (heterocyclic amines) found in tobacco smoke and meat that is cooked at a high temperature.

Acetylation is also important in the breakdown and elimination of several drugs from the body - particularly arylamine and hydrazine drugs (e.g. certain blood pressure, arrhythmia and TB medications).

This trait analyses several variants of your NAT1 gene, which are associated with differences in the level and activity of the NAT1 enzyme and rate of acetylation.

One of the key SNPs analyzed in this trait is rs4986782, which creates 'G' and 'A' alleles.

You carry two copies of the 'G' allele linked to higher NAT1 activity - i.e. your genotype is GG.

You are classified as a fast acetylator.

Your cells clear arylamines, HCAs and other potentially toxic compounds more effectively than slow and very slow acetylators.

Based on NAT1 gene variants alone, you are not significantly more susceptible to cell damage and ageing caused by compounds in tobacco smoke, grilled and pan-fried meat, and environmental pollutants.

This trait focuses on the effects of variation of the NAT1 gene on enzyme activity. Factors such as inadequate intake of B vitamins and exposure to oxidative stress can reduce NAT1 enzyme activity.

## Recommended Actions

**Eat foods rich in vitamin B2 such as beef, tofu, and oyster mushrooms.** Deficiencies in B2 can cause slowing of the acetylation process.

**Eat liver, egg yolk, broccoli, peanuts, legume and sweet potatoes.** These are great sources of vitamin B5, which is an important vitamin involved in the acetylation process.

# Detoxification rate (NAT2)

⏳ Biological Ageing

Like NAT1 in your previous trait, NAT2 (N-acetyltransferase 2) is an enzyme that carries out acetylation. This reaction is responsible for the breakdown and clearance of various potentially harmful compounds, including arylamines found in industrial toxins and HCAs (heterocyclic amines) found in tobacco smoke, vehicle exhaust and meat cooked at high temperatures. This trait analyses your ability to detoxify these compounds based on variants of your NAT2 gene, which affect your rate of acetylation.

## Your result

- Higher Risk
- Moderately Higher Risk
- Lower Risk
- No Data

**You have an increased susceptibility to cell damage and ageing, due to slower clearance of toxic compounds.**

NAT2 (Human arylamine N-acetyltransferase 2) is an enzyme responsible for a key class of reaction called acetylation.

Acetylation is important in the breakdown, detoxification and clearance of several environmental toxins - including arylamines used in the industrial production of epoxy polymers, explosives, fungicides, pesticides and colorants.

Acetylation also both activates and detoxifies various HCAs (heterocyclic amines) found in tobacco smoke, diesel exhaust and meat that is cooked at a high temperature.

NAT2 helps to break down paraxanthine (an active metabolite of caffeine) and histamine (an amine found in many foods).

This trait analyses several variants of your NAT2 gene, which are associated with differences in the level and activity of the NAT2 enzyme and rate of acetylation.

Some of the key SNPs analyzed in this trait include: rs1799930, rs1799931, rs1801279 and rs1801280.

You carry NAT2 gene variants associated with low enzyme activity.

You are classified as a slow acetylator.

Your cells clear arylamines and HCAs in the environment less rapidly than fast and medium acetylators. You therefore may be more susceptible to cell damage from these toxins. In particular, cells in your bladder may be at higher risk of damage from industrial arylamines and HCAs in cigarette smoke.

Some studies suggest that you may also more slowly activate certain arylamines, leading to a reduced risk of damage to cells in the colon and lung in comparison to fast acetylators.

Based on NAT2 variants alone, you may break down histamine in food more slowly. This may make you more likely to experience symptoms of histamine intolerance (e.g. headache, diarrhoea) when consuming foods that are high in histamine (e.g. fermented foods such as aged cheese, sauerkraut).

This trait focusses on NAT2 gene variants only. Several other genes (e.g. NAT1, CYP genes, DAO) and lifestyle factors affect your ability to break down and detoxify different compounds.

## Recommended Actions

**Take precautions in heavily polluted areas.** Due to slow acetylation, car exhaust fumes and other air pollutants can lead to toxin build-up and enhance cellular ageing.

**Consult a doctor if you experience negative symptoms from eating histamine-rich foods.** Symptoms such as headache and nasal congestion from eating foods such as cheese and alcohol suggest these foods may need to be avoided.

**Eat rice, eggs, lean meats and non-citrus fruit.** These are examples of low-histamine foods which will be easier to metabolise over high-histamine foods.

**Moderate your alcohol intake.** Your slower acetylation may influence the metabolism of alcohol.

**Avoid caffeine if you struggle with falling asleep.** Slow acetylation impacts on the breakdown of the caffeine metabolite, paraxanthine, therefore it's stimulating effects may be prolonged and could negatively impact your sleep.

**Look to stop smoking if currently doing so and avoid second-hand smoke exposure.** Your risk of smoking-induced diseases is higher than fast acetylators due to a greater chance of toxin build-up.

**Reduce your intake of grilled/barbecued foods.** The high temperatures used can lead to the formation of harmful HCA (heterocyclic amines) similar to those found in cigarette smoke.

# BDNF activity and cognition

Brain Health

BDNF (Brain Derived Neurotrophic Factor) is a protein that promotes the growth, development and survival of neurons (nerve cells). It is extremely important in learning, memory, processing of emotion and other cognitive functions. When a neuron is stimulated, it releases BDNF, which can encourage the formation of new synapses, new neurons and other structural changes in the brain that underlie learning.This trait analyzes variants of your BDNF gene that alter the release of BDNF by nerve endings. In turn, this can influence your cognition, response to exercise and risk of developing low mood.

## Your result

Higher Risk

Moderately Higher Risk

**Average Risk**

No Data

**Your gene variants are associated with average BDNF activation. Increasing BDNF activity can improve mood, learning, and memory.**

BDNF (Brain Derived Neurotrophic Factor) is a key protein that protects existing neurons (nerve cells in the brain) and also stimulates the growth of new neurons and synapses (connections between nerve cells).

BDNF plays an important role in learning and memory. It is secreted when neurons are stimulated. The cognitive benefits of exercise result in part from the production of BDNF in response to physical activity.

This trait looks at variants of your BDNF gene. In particular, we focus on a SNP (rs6265) that creates BDNF gene variants (alleles) associated with different activity of the BDNF protein.

You carry two copies of the 'G' allele. This genotype (GG) is associated with normal BDNF activation.

You are likely to have normal BDNF secretion in response to stimulation of neurons.

You will benefit from exercise - it has been shown to increase BDNF secretion, promote growth of new neurons, and improve memory, learning and other cognitive functions.

Inflammation, sleep deprivation, a high-fat diet, and exposure to stress can all reduce the secretion and activity of BDNF.

This trait is useful for understanding the extent to which lifestyle measures such as exercise, diet, and social activity can improve your cognitive function.

This trait focusses on BDNF gene variants. Learning, memory and other cognitive functions are complex processes influenced by several genes and environmental factors.

## Recommended Actions

**Maintain healthy levels of body fat.** This will help prevent reduced BDNF activity from inflammation, and optimise the regulatory role of BDNF in energy metabolism.

**Eat high antioxidant foods such as berries, lychee, grapes, broccoli, bok choi.** Inflammation can reduce BDNF so keeping your antioxidant defences high will prevent high levels of inflammation.

**Reduce stress with activities like meditation.** Stress leads to reductions in BDNF activity due to elevated glucocorticoids, such as cortisol, negatively influencing signalling.

**Optimise your sleep routine.** Sleep deprivation can reduce BDNF activation and impair cognitive function.

**Try to perform at least 150 minutes of moderate intensity activity every week.** Exercise is a great way to keep your BDNF levels boosted, minimize inflammation, improve sleep quality; and keep your body composition healthy.

**If not getting enough dietary zinc, supplement with at least 15 mg per day.** Zinc regulates the activity of the BDNF receptor influencing synaptic function and BDNF activity.

# SIRT1 and neuroprotection

Brain Health

You may have heard that calorie restriction (i.e. reducing the total amount of calories you eat without being undernourished) can extend lifespan, preserve brain health, and protect against age-related diseases. This effect may be due to activation of 'sirtuins' - enzymes that regulate our response to cellular stress and promote cell renewal and survival.In a key scientific discovery in 1999, it was found that overexpressing sirtuin (Sir2) in yeast extends their lifespans by up to 70%. In humans, sirtuins are also thought to have anti-ageing effects. In this trait, we look at gene variants that affect the expression of sirtuin 1 (SIRT1) - one of seven sirtuins produced by humans. Higher SIRT1 activity in the brain may protect against age-related damage to neurons and support healthy ageing.

## Your result

- **Average Risk**
- Moderately Low Risk
- Lower Risk
- No Data

**You do not carry a copy of the gene variant linked to increased protection from brain ageing and higher cognitive function in old age.**

SIRT1 (Sirtuin 1) is an enzyme that regulates several cell processes, including fat metabolism, inflammation, mitochondrial biogenesis, and response to cellular stress.

SIRT1 activity may have anti-ageing effects in the brain, partly by protecting against neuronal damage, promoting cell renewal and synaptic plasticity, and delaying cellular senescence (a state in which cells are unable to grow and divide).

SIRT1 expression decreases in the brain as we age. By contrast, calorie restriction, which may have anti-ageing effects, is shown to increase SIRT1 expression in various brain regions.

The 'T' allele (rs3758391) of the SIRT1 gene has been linked to higher SIRT1 expression in the brain, which may have a beneficial effect on age-related outcomes such as cognitive decline and neurodegeneration.

You do not carry the 'T' allele linked to higher SIRT1 activity and healthy brain-ageing. Your SIRT1 genotype is CC.

Your genotype (CC) is not linked to higher cognitive functioning in older age. One study found that older (>85 years) subjects with your genotype (CC) scored lower on tests of cognitive function (esp. delayed and immediate memory recall) compared to those with TT genotype.

Older (>70 years) men with your genotype (CC) were found to have higher BMI and poorer body composition compared to those with the T allele. This may reflect an effect of lower SIRT1 activity on brain circuits that regulate energy balance and metabolism.

Calorie restriction may help you to increase SIRT1 expression, which may benefit brain health. Some clinical trials report improvements in inflammatory markers, working memory and mood when reducing calorie intake by 12% - 30% over 3 - 24 months.

Animal studies suggest that exercise can also upregulate SIRT1 activity in the brain, which is protective against age-related damage to neurons.

# Recommended Actions

**Supplement with 500 mg of quercetin, with a mug of green tea.** Quercetin has been shown to increase SIRT1 activation, with green tea catechins helping increase it's potency so that you get benefits from lower doses.

**Treat yourself to a piece of dark chocolate (at least 70% cocoa) every now and again.** Dark chocolate is rich in flavanoids which have been shown to help reduce cognitive decline and benefit healthy brain ageing.

**Eat wild salmon, particularly sockeye (570 IU per 85 g/3 oz).** Salmon is rich in vitamin D3 and sockeye has the highest amounts; regular consumption will minimise the risk of vitamin D deficiency which can reduce SIRT1 activity.

**Eat portabello mushrooms or mushrooms that have been exposed to UV light (366 IU per 1/2 cup).** Once exposed to UV light, mushrooms are a good source of vitamin D2; helping minimise the risk of vitamin D deficiency which can reduce SIRT1 activity.

**Get enough vitamin B3 (niacin) in your diet by having 170 g (6 oz) of chicken breast or tuna fillet.** Niacin deficiencies can impair SIRT1 function by reducing NAD+ production.

**Eat plenty of red grapes.** Grape skins contain resveratrol which has been shown to increase SIRT1 activation.

**Walk, jog or cycle for 30-60 mins at least 3 times a week, at a moderate-vigorous intensity.** Aerobic exercise helps to stimulate pathways that upregulate SIRT1 levels as well other cardiometabolic benefits.

**Try adding extended periods of caloric restriction such as through intermittent fasting, with a fasting period of at least 12 hours.** Calorie restriction such as through intermittent fasting, has been shown to increase SIRT1 levels and induce beneficial neuroprotective processes.

**Incorporate 1-2 high intensity interval training workouts each week.** High intensity workouts help keep your insulin sensitivity healthy and increases SIRT1 activity.

**Perform resistance training at least 2 times a week, at a RPE greater than 12.** Resistance training has been shown to increase serum levels of SIRT1.

**Try having some haskap berries with your breakfast.** Haskap berries contain plenty of polyphenols which are an important antioxidant that benefit neuroprotection by keeping inflammation low.

# Cholesterol and ageing (CETP)

⏳ Biological Ageing

CETP (Cholesteryl Transfer Protein) is an enzyme that regulates the transport of fat and cholesterol in the bloodstream. Changes in the levels and activity of this enzyme are linked to differences in blood levels of 'good' HDL cholesterol, risk of cardiovascular disease, ageing and longevity. This trait looks at several variants of your CETP gene, which affect CETP enzyme activity, and analyzes your risk of enhanced (faster) ageing and low levels of 'good' HDL cholesterol.

## Your result

Higher Risk

Moderately Higher Risk

**Lower Risk**

**Your gene variants are linked to longevity, reduced cell ageing, and higher levels of "good" HDL cholesterol.**

CETP (Cholesteryl Ester Transfer Protein) is an enzyme involved in the transport of fat and cholesterol in the bloodstream.

CETP activity acts to reduce levels of "good" HDL cholesterol. Low activity and levels of CETP are associated with greater longevity and higher blood levels of HDL cholesterol.

This trait looks at several variants of your CETP gene, which affect the levels and activity of the CETP.

You carry one or two copies of the 'G' allele (rs5882) associated with decreased CETP activity, greater longevity / reduced aging and higher levels of "good" HDL cholesterol.

You do not carry other risk variants / alleles (e.g. rs708272, rs1864163) that are linked to higher CETP activity, enhanced aging and lower levels of "good" HDL cholesterol.

You carry CETP variants associated with reduced aging and greater longevity / lifespan.

You carry CETP variants that have been linked to higher levels of "good" HDL cholesterol. High levels of HDL may be protective against cardiovascular disease.

This trait is based on CETP gene variants only. A blood test (e.g. total lipid profile) is needed to more accurately assess your blood cholesterol levels.

This trait is useful for assessing your risk of aging and low HDL levels based on CETP gene variants.

Other genetic traits (e.g. Protein Synthesis and Hypertrophy), diet, exercise and lifestyle factors will also strongly influence your longevity and blood cholesterol levels.

## Recommended Actions

**Add plant sterols into your diet.** Often fortified into some foods or drinks, these sterols can help keep your triglyceride levels healthy; maintaining a lower risk of poor heart health.

**Ensure you are exercising enough and eating a balanced diet.** Your diet and activity levels can still significantly influence your heart health.

**Avoid the consumption of foods containing hydrogenated vegetable oils.** This trans fat source contains elaidic acid, which has been shown to raise CETP levels and may lead to poorer heart health.

# Bone mineral density (VDR)

⏳ Biological Ageing

Are you more likely to have fragile bones as you age? We all gradually lose bone tissue as we age, but, in a large proportion of people, this can lead to osteoporosis - a condition characterised by fragile bones, pathologically-low bone mineral density, and an increased risk of fractures. In fact, it is estimated that more than one in three women and one in five men will sustain one or more osteoporotic fractures in their lifetime. Advanced age is a strong risk factor, with the prevalence of osteoporosis rising from 2% at age 50 to 50% at age 80. Women are also at greater risk due to the decrease in oestrogen levels that occurs during menopause. Our genetics are important too, with genes thought to account for 60-80% of variance in bone mineral density.In this trait, we look at variants of the Vitamin D receptor (VDR) gene, which plays an important role in bone metabolism. Certain risk variants of this gene have been linked to reduced bone mineral density and a higher risk of osteoporosis and associated bone fractures.

## Your result

Higher Risk

Moderately Higher Risk

**Average Risk**

No Data

**Your gene variants are linked to an average bone mineral density.**

Vitamin D plays a crucial role in bone mineralisation - the process of laying down mineral (mostly calcium phosphate crystals) into the matrix of bones.

The active form of Vitamin D (1,25[OH]D3) stimulates bone mineralisation by binding to the vitamin D receptor (VDR) on bone cells called osteoblasts.

Variants of the VDR gene may affect bone mineralisation and have been tentatively linked to changes in bone mineral density (BMD) and risk of osteoporosis, a condition

characterised by fragile bones that are more susceptible to fracture.

Some studies have linked the BsmI 'B' variant (rs1544410) of the VDR gene with lower bone mineral density, greater susceptibility to osteoporosis, and a higher risk of fractures.

You do not carry the BsmI 'B' risk variant linked to lower bone mineral density. Your VDR BsmI genotype is bb.

Greater bone mineral density makes bones less prone to fracture. Some studies suggest people with your genotype have a lower risk of fracture compared to 'B' variant carriers.

Some studies suggest your genotype is linked to a lower risk of osteoporosis compared to 'B' variant carriers, although this association is less well studied in men.

Low testosterone levels as you age are also a risk factor for low bone mineral density and osteoporosis in men.

## Recommended Actions

**Do 2-4 sessions of high-impact exercise a week.** High-impact exercise such as running, squash or weight training are most effective for maintaining and improving bone density.

**Start your day with a brisk walk.** If you cannot do high-impact exercises like running, going for a brisk walk regularly is a good alternative that will still increase bone mineral density, albeit to a slightly lesser degree.

**Get outside as often as you can each day, particularly when the sun is out.** Sun exposure will naturally increase your vitamin D levels, which will allow more calcium to be absorbed. This will benefit your bone mineral density.

**If currently smoking, try nicotine replacement (for example, gums/patches) or talk to your clinician about stop smoking programmes.** Smoking has consistently been shown to lower bone mineral density, and increase the risk of fractures. Quitting smoking has the capability to partially reverse these negative effects on bone health.

**You may benefit from supplementing with vitamin D.** Making sure you are getting at least 10 mcg of vitamin D each day is important for bone health.

**If you don't consume dairy products, try supplementing with calcium citrate to help reach the recommended daily amount of 700 mg.** Adequate calcium intake is important for the maintenance of bone health.

**Add canned sardines to your salads or pasta.** Sardines are great sources of vitamin D (178 IU per can), and calcium (almost half your daily recommended amount!), two minerals that are beneficial for maintaining healthy bones.

**Try to have 3 servings of dairy each day.** Milk is full of calcium, phosphorous, magnesium and protein - all essential for healthy bones.

# WNT16 and age-related bone mineral density

⏳ Biological Ageing

Are you at risk of brittle bones? Roughly 1 in 2 women and 1 in 4 men aged over 50 are expected to have a bone fracture due to osteoporosis. Literally meaning “porous bone,” osteoporosis is a condition characterised by thinning of bones, making them weaker, more brittle and more prone to fracture. Although our risk of osteoporosis is greatest in advanced age, we all gradually start to lose bone material (causing a drop in bone mass and bone mineral density) from our mid thirties and forties onwards. Maintaining healthy bone metabolism to slow down this deterioration and minimise our risk of osteoporosis is therefore important at all stages of life. Women, in particular, stand to lose more bone material during the menopause as levels of oestrogen decline, and have a greater osteoporotic risk than men. Alongside our age and sex, genetics also play a role in how quickly we lose bone material. In this trait, we look at your WNT16 gene, which is involved in the remodelling of bone tissue. One risk variant of this gene has been linked to thinner, more brittle bones and a greater risk of osteoporotic fracture. Luckily, as explained in your actions, we can partially mitigate the impact of ageing, menopause, and genetics on bone loss with dietary, exercise, and lifestyle measures.

## Your result

Higher genetic risk

**Your gene variants and age are linked to lower bone mineral density.**

Moderate Genetic Risk

Average Genetic

Bone is constantly remodelled throughout your life. This involves destroying old bone tissue (resorption) and laying down new bone mineral (bone formation).

Risk

No Data

When bone formation fails to keep up with resorption, the mineral (mainly calcium and phosphorous) content of bone starts to fall. This drop in bone mineral density (BMD) makes your bones weaker and more susceptible to fracture.

In men, bone mineral density (BMD) reaches a peak between 25 and 30, before remaining stable for about 10 years. BMD then gradually declines from your 40s onwards as bone resorption outpaces bone formation.

Your genetics also affect how you maintain bone mineral density throughout life. Your WNT16 gene encodes a signalling molecule involved in the activity of osteoblasts: specialised bone cells that lay down new bone mineral.

The 'C' variant (rs2707466) of the WNT16 gene is thought to alter bone remodelling and has been linked to lower bone mineral density, reduced bone thickness, and a greater risk of bone fracture.

You carry two copies of the 'C' variant linked to lower bone mineral density and a greater risk of bone fracture. Your WNT16 (rs2707466) genotype is: CC.

Your genotype is linked to lower bone mineral density (BMD). Several studies have found that people with your genotype have lower BMD of bones in the forearm, thigh (neck of femur), and hip compared to other genotypes.

Studies have also found that the outer protective layer (cortex) of the tibia (shin) bone is thinner in people with your genotype. This is known as reduced cortical thickness.

The combination of lower bone mineral density and reduced cortical thickness makes bones weaker and more susceptible to fracture. One case-control study found that people with osteoporotic forearm fractures were 1.44 times more likely to have your genotype compared to other genotypes.

As you are over 40 years old, you will also be gradually losing 0.3-0.5% of your bone mineral density every year. This is because the rate of bone formation falls behind the rate of bone resorption as you get older.

Your lifestyle also has a major impact on how quickly you lose bone mineral density as you age. Smoking, physical inactivity, excessive alcohol use, and low intakes of calcium and vitamin D can all accelerate bone loss and increase your risk of osteoporosis (a condition charactersied by brittle bones) as you get older.

## Recommended Actions

**Do 2-4 sessions of high-impact exercise a week.** High-impact exercise which involves high amounts of force on the joints, such as running, squash or weight training, are most effective for maintaining and improving bone density.

**Start your day with a brisk walk.** If you cannot do high-impact exercises like running, going for a brisk walk regularly is a good alternative that will still increase bone mineral density, albeit to a slightly lesser degree.

**If you don't consume dairy products, try supplementing with calcium citrate to help reach the recommended daily amount of 700 mg.** Calcium is an important nutrient for the development and maintenance of bone health. Increased dietary intake can help to improve your bone mineral density.

**Introduce fermented foods such as miso and sauerkraut into your diet.** These foods are a rich source of vitamin K2, a vitamin that has been associated with protection against losses in bone mineral density.

**Add canned sardines to your salads or pasta.** Sardines are great sources of vitamin D (178 IU per can), and calcium (almost half your daily recommended amount!), two minerals that are beneficial for maintaining healthy bones.

**Try supplementing with 3 mg of boron (calcium fructoborate).** Boron supplementation has been shown in studies to prevent losses in bone mineral density, supporting bone health.

**Include fast, explosive movements in your weight training workouts such as squat jumps or cleans.** Weight training focused on increasing muscular power, which is what fast explosive exercises do, have been shown to improve bone mineral density.

**Supplement with up to 4000 UI of vitamin D3 per day, in the morning with breakfast in autumn/winter.** Vitamin D helps the absorption of calcium to keep your bones strong. Autumn and winter months provide less natural sun exposure which is usually a good source of vitamin D.

**Carry out three or four 30-minute sessions of weight-bearing exercises each week.** Doing activities such as dancing, jogging or playing a sport regularly can help to prevent bone loss.

**Make sure to eat plenty of fruits and vegetables that are high in fibre, such as currants, raspberries, mushrooms and artichokes.** Adequate intake of fibre has been associated with better bone mineral density.

**Sprinkle chia seeds on your smoothies and porridge.** Chia seeds have been shown to have 6 times more calcium than cow's milk! This makes them a great addition to your diet to increase your calcium intake and help you maintain strong bones.

# Sarcopenia risk

Muscle Building

Are you at greater risk of age-related muscle loss? By the year 2050, it is expected that 2 billion people around the globe will be aged 60 years or older. While this is due to welcome improvements in healthcare and living conditions, an as-yet inescapable fact of ageing is that our muscle tissue gradually deteriorates. As such, in the next 40 years, it is forecast that 200 million people worldwide will suffer from a condition called sarcopenia. From the Greek “sarx” (meaning “flesh”) and “penia” (meaning “loss”), sarcopenia refers to low muscle mass, strength, and performance relative to our age. Individuals with sarcopenia have lower grip strength, slower walking speeds, are less mobile, and have a higher overall mortality risk. Although more common in individuals aged over 60, research suggests that the early stages of sarcopenia can begin before this. Your lifestyle has a strong influence on your risk of developing sarcopenia as you get older, but your genetics are also important. In this trait we combine three different gene variants to assess your genetic risk of sarcopenia, while also factoring in lifestyle factors such as physical activity and body fat levels. Your personalised actions will help you to reduce your sarcopenia risk by building and maintaining muscle mass and slowing down age-related muscle loss.

## Your result

Higher genetic risk

High genetic risk

**Moderate Genetic Risk**

**Your genetic risk of sarcopenia is moderate.**

Sarcopenia is a condition characterised by low muscle strength, mass, and function. It is typically due to age-related loss of muscle tissue, with older individuals having the greatest sarcopenia risk. People who are physically inactive and/or obese are also more likely to develop sarcopenia.

Your genetic make-up also influences your risk of sarcopenia as you get older. One study found that variants of three

Lower Genetic Risk

genes, ACTN3, MTHFR, and NRF2, explained 39% of differences in sarcopenia risk between individuals.

The 'X' variant (rs1815739) of the ACTN3 gene causes reduced production of alpha-actinin-3, a protein involved in contraction of fast-twitch muscle fibres. This risk variant has been linked to lower muscle power, poorer physical performance, and higher risk of sarcopenia in older individuals.

The 'C' variant (rs1801131) of the MTHFR gene may elevate levels of homocysteine: a harmful amino acid that can can impair the production of new muscle proteins (muscle protein synthesis). Carriers of this risk variant have been found to have lower muscle mass and a higher risk of sarcopenia.

The NRF2 gene helps to maintain healthy muscle metabolism by promoting the growth of new mitochondria. The 'C' variant (rs12594956) of this gene has been linked to an increased risk of sarcopenia and may accelerate age-related muscle loss by negatively impacting mitochondrial function.

Your overall genetic risk of sarcopenia is: MODERATE

Your genetic risk is based on how many risk variants of the ACTN3, MTHFR, and NRF2 genes you carry in total. You carry some risk variants.

The risk of sarcopenia increases with age. Although you are under 60 years old, be aware that we all gradually start to lose muscle mass and strength from our 30s onwards and stand to lose 30-50% of our total muscle mass between the ages of 40 and 80. You carry some risk variants that may accelerate this age-related decline in muscle as you get older.

Your physically active lifestyle will help to slow down age-related loss of muscle and counteract your genetic sarcopenia risk as you get older. Resistance (strength)

training, in particular, is shown to help preserve muscle mass and strength as you age.

Maintaining a healthy body fat percentage minimises the release of pro-inflammatory molecules (adipokines) from fat cells that cause muscle breakdown. This will help you retain muscle mass, strength, and function as you get older.

In summary, you carry some sarcopenia risk variants, but this genetic risk can be counteracted by sustaining a healthy lifestyle as you get older.

## Recommended Actions

**Keep on moving.** Be wary of the 'exercise paradox', whereby your body will compensate for a hard workout by moving less throughout the rest of the day. To counteract this, keep your all-round activity elevated by walking short distances, taking the stairs where you can and doing some moderate intensity activities like cleaning.

**Take 5 g of creatine monohydrate post-workout.** Creatine has been extensively shown to help increase muscle strength when combined with resistance training.

**Try to include 2 days of resistance (strength) training with regular aerobic activity each week.** Combining resistance exercise with aerobic activities, such as walking, gardening or cycling, will optimise your ability to increase and maintain muscle strength.

**Consume 20 g of protein (for example, a scoop of protein powder or 3 eggs) in your post-exercise meal.** Ensuring you are consuming enough protein will help to optimally stimulate muscle growth following exercise.

**Get up and walk around for 5 minutes every 30-60 minutes when at work or watching tv.** Large periods of sedentary behaviour can increase your risk of sarcopenia, so it is important to include active periods when spending hours sitting down.

# FOXO3 and longevity

⏳ Biological Ageing

Could you live until 100?Although lifestyle factors such as our diet, physical activity, and daily habits are undoubtedly important, up to half of variation in human lifespan can be explained by genetic differences. When it comes to people with extremely long lifespans, it's thought that what genes you inherit are particularly influential, with the genetic contribution to longevity estimated to be between 20 and 30%.One of the best-studied candidate genes for longevity is FOXO3 - which encodes the forkhead box O3 (FOXO3) transcription factor. One particular variant of the FOXO3 gene, created by the rs2802292 SNP, is more frequently seen in people aged 95 years and older. Similarly, it's also been linked to healthy aging, reduced all-cause mortality, and lower risk of cardiovascular disease: a finding that has been reported in both men and women, and across different ethnic populations.

## Your result

- Average Risk
- Moderately Lower Risk
- Lower Risk
- No Data

**You do not carry gene variants that are more frequently found in people who live beyond the age of 90.**

FOXO3 is a transcription factor - a protein that switches different genes 'on' and 'off'.

FOXO3 switches on the genes that protect against oxidative stress, promote DNA repair, and stimulate cell renewal - all of which may prevent cell ageing.

The 'G' allele (rs2802292) of the FOXO3 gene is associated with increased expression of FOXO3 in response to cell stress/damage. This may protect against age-related cell damage.

The 'G' allele is linked to longevity and overrepresented in people aged >95 years old. People with the 'G' allele have also been found to have a reduced risk of age-related diseases, including coronary artery disease and stroke.

You do not carry the 'G' allele linked to longevity. Your FOXO3 genotype is TT.

Your genotype (TT) is not overrepresented in various groups of people with longer-than-average lifespans. A meta-analysis comparing long-lived individuals (aged >90 years old) to those with average lifespans found that long-lived individuals were 34% less likely to have your genotype (TT).

Your FOXO3 genotype is not shown to be protective against age-related illnesses. Compared to people with your genotype (TT), 'G' allele carriers have been shown to have a lower prevalence of coronary artery disease, hypertension, cancer, and bone fractures.

You may have poorer insulin sensitivity compared to G allele carriers. Studies have linked the 'G' allele to better insulin sensitivity and lower levels of IGF-1 (insulin-like growth factor 1), a marker of insulin resistance.

You can enhance FOXO3 expression through lifestyle changes, which can influence cell-ageing and longevity. Human and animal studies have shown that caloric restriction and consumption of phytonutrients (e.g. polyphenols) can increase FOXO3 expression and protect against age-related cell damage.

# Recommended Actions

**Do something you enjoy everyday such as reading a book or chatting with a friend.** Doing things we enjoy helps minimise stress and keeps us feeling more content; happiness has been associated with increased longevity.

**Eat plenty of red grapes.** Grape skins contain resveratrol which has been shown to have some promising longevity impacts.

**Add a handful of blueberries to your breakfast.** Blueberries contain plenty of polyphenols which are an important antioxidant that have been shown to have positive impacts on the function of FoxO proteins.

**Season your food with rosemary and cloves.** These herbs and spices are very rich in polyphenols, increasing the function of FoxO proteins.

**Supplement with 3 doses of 500 mg of berberine per day, taken with meals.** Berberine has been shown to activate AMPK which induces FOXO3 activation, so may have some benefits for longevity processes.

**Try fasting from 8pm to 10am.** Intermittent fasting lowers IGF-1 levels, and activates AMPK which leads to FOXO3 activation; promoting longevity processes.

**Start your day with a green tea.** Green tea contains epigallocatechin gallate (EGCG) which has been shown to increase FOXO3 activation while also reducing the activity of anti-inflammatory proteins like NF-kB.

**Take 400-500 mg of curcumin, with 10-20 mg of piperine to aid absorption, daily.** Curcumin increases FOXO3 activity and it's induced gene expression, helping to reduce inflammation and promote healthier ageing.

**Perform resistance exercise 3 times a week.** It has been shown that training regularly can improve FOXO3 expression and aid longevity.

**Try adding more root vegetables (like sweet potatoes) and soy products into your diet.** The Okinawan diet which has been associated with increased longevity is high in these foods.