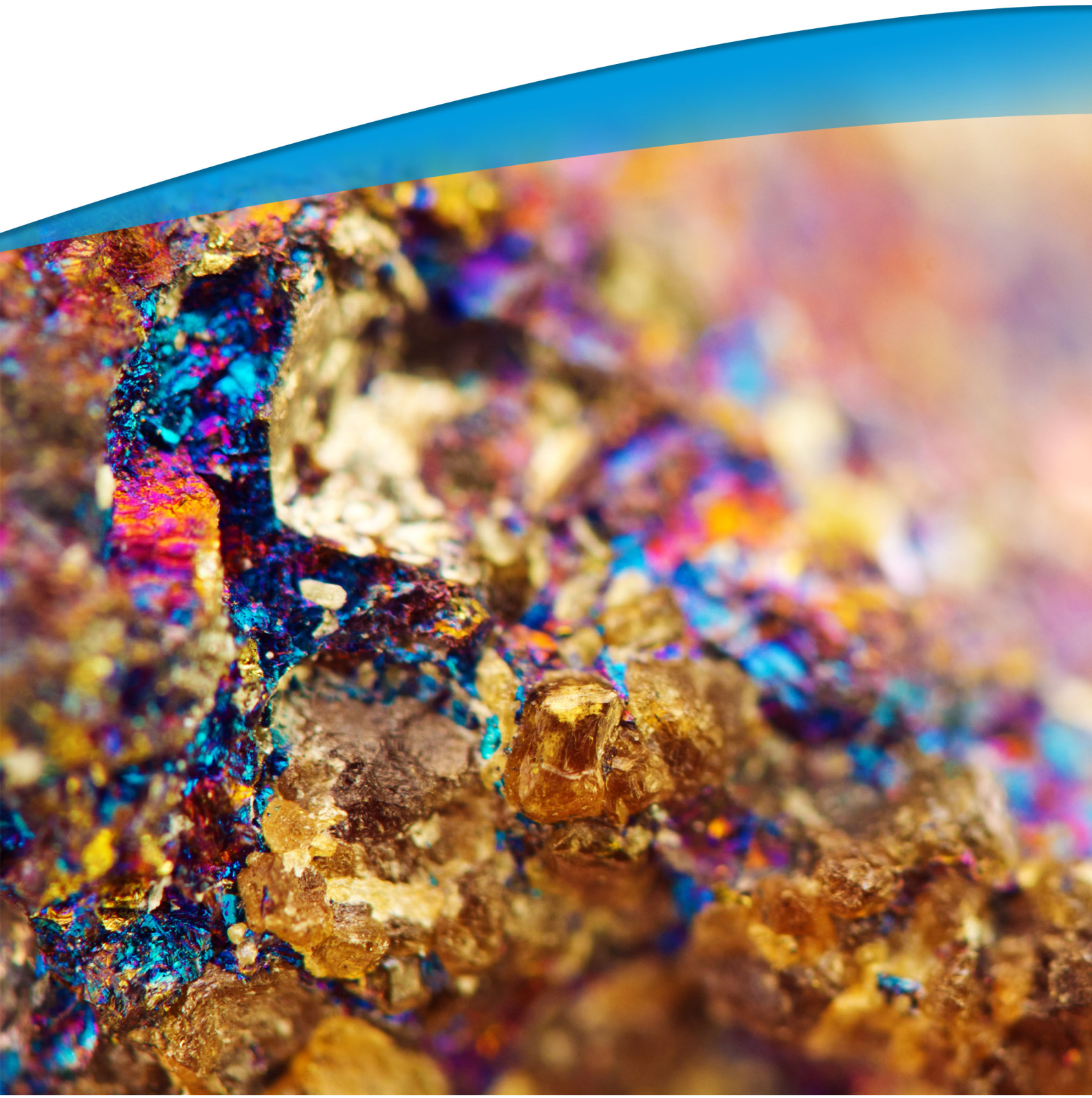
Lifecode GX ®
— Professional Genotype Analysis —

Metals and Minerals Report



Report for Vanessa Kimbell (CP00120420)

Welcome to your unique, personalised Metals and Minerals DNA report!

Mineral nutrients are chemical elements essential for human life. Distinct from vitamins, which are organic compounds made by plants and animals, minerals are inorganic and originate from rocks, soil or water. They are vital for providing structural support to bones and teeth, maintaining pH and fluid balance, enabling nerve conduction and muscle contraction, and supporting the function of hormones and enzymes, as well as the immune system.

Whilst essential minerals are necessary in adequate amounts for health, excessive intake and accumulation can be detrimental. Additionally, environmental heavy metals are toxic to humans and pose serious health risks.

Metals and minerals have complex interactions with one another as well as with vitamin metabolism. Genetic variants – as well as nutrition, age, gender and lifestyle habits – can affect the absorption, distribution and excretion of metals and minerals, impacting their balance and status in the body. In this report we present elements of your DNA profile that have been shown to influence your need, status and metabolism of:

- **Major minerals** (RDI >100mg): calcium, magnesium, phosphorus, potassium and sodium
- **Trace minerals** (RDI <100mg): copper, iron, manganese, selenium and zinc
- **Heavy metals** (toxic elements): arsenic, cadmium, lead, and mercury

The nutritional genomics team at Lifecode Gx® are experts in interpreting DNA results, examining the most up-to-date research in the field and distilling it into relevant, meaningful and practical advice. To benefit the most from this report, we recommend you work with a qualified nutrition professional.

Calcium

Calcium is the most abundant mineral in the human body. Best known for keeping teeth and bones strong and healthy, calcium also has other vital roles in muscle contraction, blood clotting, nerve transmission, hormone secretion and blood pressure regulation.

Blood calcium is tightly regulated by calcium-sensing receptors, encoded by the CASR gene. When levels drop, CASRs induce parathyroid hormone (PTH), activating vitamin D to promote calcium absorption, and reducing calcium excretion. PTH also triggers calcium release from bone (resorption).

Two CASR SNPs alter receptor sensitivity in opposite directions. The A986S SNP results in a receptor which more readily stimulates PTH to raise blood calcium. Increased resorption is linked to osteoporosis.

Conversely, the R990G SNP acts to lower blood calcium. This is associated with excess calcium excetion (hypercalciuria) and the risk of developing kidney stones.

Plasma membrane calcium ATPase type 1 (PMCA1), encoded by ATP2B1, helps fine-tune calcium levels by pumping calcium out of cells. Higher calcium intake is effective in reducing blood pressure in SNP carriers.

Nutrition Advice

Opt for dietary sources of calcium. Dairy products – milk, yoghurt, cheese etc., are good sources, as well as bony fish – sardines and salmon, and vegetables – kale and broccoli. Oxalic acid and phytic acid (in some plant foods) inhibit calcium absorption. Optimise vitamin D levels to support calcium absorption, along with vitamin K to direct calcium into bones and protect from vascular calcification.

Your Results

ATP2B1 rs17249754	--	No result. It was not possible to read the genetic code at this location.
CASR rs1042636	AA	[R990G] Normal (not higher) CASR sensitivity. Not associated with low blood calcium or kidney stones. Ensure regular intake of calcium preferably from dietary sources, with vitamin D to aid absorption.
CASR rs1801725	TG	[A986S] Slightly lower CASR sensitivity and tendency to raise blood calcium. Risk of increased bone resorption, osteoporosis and soft-tissue calcification. Higher intake of calcium may be needed. Optimise other bone building nutrients - vitamins D and K, magnesium and protein, along with weight bearing exercise.

Magnesium

Magnesium is essential for the function of hundreds of enzymes involved in energy (ATP) production, DNA synthesis, blood glucose control, and blood pressure regulation. It promotes vitamin D synthesis, and bone formation, as well as glutathione synthesis. Magnesium also plays a role in the active transport of calcium and potassium ions across cell membranes, a process that is important to nerve impulse conduction, muscle contraction, and normal heart rhythm.

The TRPM6 gene encodes the transient receptor potential cation channel, subfamily M, member 6 protein. TRPM6 regulates magnesium levels by forming ion channels that allow magnesium, and small amounts of calcium, to be absorbed in the gut and kidney. SNPs are associated with magnesium deficiency and lower insulin response. Risk is greater when dietary intake of magnesium is low; or absorption is impaired (e.g. by the use of proton pump inhibitors – PPIs); or excessive loss via the kidneys (e.g. due to alcohol use disorder, diabetes, diuretic drugs, kidney disease).

Symptoms of magnesium deficiency (hypomagnesaemia) include muscle spasms, cramps, numbness, tremors, migraines, loss of appetite, fatigue and weakness and, in the longer term, cardio vascular disease, type 2 diabetes and osteoporosis. Severe deficiency can result in seizures, abnormal heart rhythms and delirium. Hypomagnesaemia can often disrupt homeostasis of other minerals, resulting in calcium and potassium deficiency.

Nutrition Advice

Green leafy vegetables are good sources of magnesium. It is also prevalent in nuts – cashews, Brazil nuts and almonds; seeds – chia and pumpkin; dark chocolate and coffee; and whole grains. Supplementary magnesium as glycinate, malate and taurate form is easily absorbed through the digestive tract. Magnesium sulphate (Epsom salts) and chloride may be absorbed through the skin.

Your Results

TRPM6 rs2274924	TT	Normal activity and efficient magnesium absorption. No increased risk of magnesium deficiency.
TRPM6 rs3750425	CC	Normal activity and efficient magnesium absorption. No increased risk of magnesium deficiency.

Phosphorus

Phosphorus is an essential mineral, and structural component of nucleic acids (DNA and RNA), cell membranes (phospholipids), adenosine triphosphate (ATP), and bones and teeth. In the body, almost all phosphorus is combined with oxygen, forming phosphate.

Phosphate is essential for life, but it can also be toxic at very high levels. Like calcium, phosphate homeostasis is due to a delicate balance between intestinal absorption, renal excretion, and influx to and efflux from bone. Intestinal absorption occurs passively and actively – regulated by calcitonin. High blood phosphate induces parathyroid hormone (PTH) which stimulates urinary excretion via klotho-FGF23 (fibroblastic growth factor 23) binding, and bone resorption.

The klotho (KL) gene is named after the Fate in Greek mythology who spins the thread of life. It is best known for its anti-ageing effects. In addition to its role in phosphate excretion, klotho regulates insulin and insulin-like growth factor signalling, and protects from oxidative stress and inflammation.

The KL variant, known as F352V, can increase or decrease klotho activity. The heterozygous genotype (single G allele) has higher KL activity and is linked to longer lifespan. However the homozygous genotype (two G alleles) has lower KL function and risk of accelerated ageing. Many of the age-related effects of impaired klotho – vascular calcification, mitochondrial oxidative stress, disrupted ATP supply, and apoptosis (cell death) are linked to chronically high phosphate.

Other than genetics, CKD (Chronic Kidney Disease) and hyperparathyroidism increase the risk of phosphate excess.

Nutrition Advice

Phosphorus is naturally present in many foods, and deficiency is rare. A high phosphate diet can have a significant effect on phosphate status. Processed foods often contain high levels of phosphate additives – to preserve moisture or colour, enhance flavour or extend their shelf life. To manage phosphate levels, minimise intake of pre-baked pastries and cakes, sliced meats and cheeses, and soda drinks – particularly dark colas. Eat freshly cooked, unprocessed food more often.

Your Results

KL rs9536314	TT	Most common genotype. Normal (not increased) function. Normal phosphorus levels but potential for excess with age, or with kidney disease or injury. Ensure adequate calcium intake (at least equal to phosphorus). Limit consumption of processed foods - such as dark colas - which contain added phosphate. Regular aerobic exercise has been shown to increase klotho.

Sodium and Potassium

Sodium and potassium are electrolytes critical for nerve impulse transmission, muscle contraction, cardiac function, and blood pressure regulation.

Levels are regulated by the renin-angiotensin-aldosterone system (RAAS). In response to low blood pressure, the kidneys release renin, which splits angiotensinogen precursor (AGT) to make angiotensin I (Ang I). Angiotensin converting enzyme (ACE) converts Ang I into the physiologically active angiotensin II (Ang II). The AGTR1 gene which encodes a receptor for Ang II regulates aldosterone secretion. Aldosterone promotes reabsorption of sodium and excretion of potassium, thereby increasing water retention, blood volume and blood pressure.

SNPs on AGT, ACE and AGTR1 genes confer higher activity and risk of essential hypertension and cardiovascular diseases, osteoporosis and kidney disease. Symptoms include headaches, nose bleeds, anxiety, irritability, fatigue and muscle cramping.

Risk of developing hypertension is higher with age, African heritage (the AGT risk variant is twice as common), family history, obesity, a sedentary lifestyle, smoking, high salt-low potassium diet, heavy alcohol use, stress and comorbidity with other chronic conditions such as kidney disease, diabetes or sleep apnea.

Nutrition Advice

Although sodium (salt) is an essential nutrient, excess is associated with high blood pressure. Reduce intake of condiments and seasonings (soy sauce, Worcestershire sauce, bouillon cubes), processed-meats (bacon, sausage and ham), canned soups and vegetables, and ready meals, which are usually high in salt.

Increasing potassium intake can help to counterbalance the blood-pressure-raising effects of sodium. The best sources of potassium are plant foods (avocados, parsnips, turnips, potatoes) and particularly high levels are found in fruits (apricots, bananas, kiwifruit and coconuts).

Your Results

ACE rs4343	GG	Higher ACE activity and angiotensin II levels. Risk of electrolyte imbalance (high sodium-potassium ratio), high blood pressure and cardiovascular disease. Limit dietary salt (sodium) and ensure good potassium intake.
AGTR1 rs5186	CA	Increased sensitivity to angiotensin II. Risk of sodium and water retention and high blood pressure. Limit dietary salt (sodium) and ensure good potassium intake.
AGT rs699	GA	Higher angiotensinogen and increased risk of sodium and water retention and high blood pressure. Limit dietary salt (sodium) and ensure good potassium intake.

Copper

Copper is an essential mineral and component of 'cupro' enzymes involved in energy production, iron metabolism, connective tissue synthesis and neurotransmitter production.

Copper homeostasis is maintained by regulating absorption and excretion. Copper toxicity is rare in healthy individuals, occurring mainly in those with high environmental exposure or genetic variance. It can cause gastrointestinal distress (e.g. abdominal pain, cramps and nausea); and neurological, liver and kidney damage. Symptoms of copper deficiency include anaemia, hypopigmentation, hypercholesterolaemia, connective tissue disorders, osteoporosis/other bone defects, ataxia, and increased risk of infection.

ATPase copper transforming beta, encoded by the ATP7B gene, transports copper out of cells (including from the liver into bile), preventing its accumulation.

ATP7B also incorporates copper into ceruloplasmin (CP) which transports copper in the blood, facilitates iron efflux from cells (protecting them from iron overload) and acts as a free radical scavenger.

Whilst rare ATP7B mutations cause Wilson's disease (the build up of copper in the liver, brain, eyes and other organs), common SNPs can have milder effects. Reduced activity increases the risk of copper excess and lower CP activity. Conversely, a beneficial CP SNP protects against copper and iron excess, selenium depletion, oxidative stress and mitochondrial damage.

Nutrition Advice

Copper rich foods include beef liver, oysters, cashew nuts and dark chocolate. If copper excess is suspected, investigate sources of exposure – water pipes, cookware etc. and diet. As zinc competes with copper for absorption, it can be used therapeutically to lower copper levels.

Your Results

ATP7B rs1061472	CC	Lower ATP7B. Reduced copper transport, excretion and ceruloplasmin formation. Higher risk of copper and iron accumulation in liver and other organs. If high copper levels, limit copper exposure - from cookware and diet, and increase zinc which limits copper absorption.
ATP7B rs732774	TT	Lower ATP7B. Reduced copper transport, excretion and ceruloplasmin formation. Higher risk of copper and iron accumulation in liver and other organs. If high copper levels, limit copper exposure - from cookware and diet, and increase zinc which limits copper absorption.
CP rs11708215	AA	Most common genotype. Risk of low ceruloplasmin. Less protection against copper and iron accumulation, and more risk of selenium depletion. Avoid excess copper exposure. Increase selenium intake (e.g. Brazil nuts) to protect against oxidative stress.

Iron (Excess)

Iron is an essential element for almost all living organisms, from bacteria to humans. It is needed for oxygen transport – as a component of haemoglobin (in red blood cells), and myoglobin (in muscles); energy (ATP) production; growth and development; immune system functioning; and hormone and neurotransmitter synthesis.

Although iron is vital for many metabolic processes it can also be toxic. Hepcidin, a hormone produced by the liver, is a major regulator of iron metabolism. When iron levels are sufficient (or high), hepcidin inhibits its intestinal absorption and recycling. Dysregulation of hepcidin or of other regulatory proteins can result in iron overload or deficiency.

Hereditary haemochromatosis type 1 (HH1) is an iron overload disorder caused by defects on the HFE (homeostatic iron regulator) gene. The HFE protein detects high iron levels and triggers hepcidin production to lower it. Two common HFE SNPs can impair hepcidin production leading to excess absorption of dietary iron and accumulation in organs (liver, pancreas, heart, and brain) and joints. The C282Y homozygous genotype (AA result) carries the highest risk of developing HH1, however any combination of C282Y and H63D variants can result in iron excess. The C282Y variant is common in Northwestern Europe, occurring at a frequency of more than 10% in people of Irish heritage.

Other risk factors for iron overload include being male or menopausal (not losing iron through menstruation), liver damage (e.g. due to hepatitis), heavy alcohol use, or high dietary intake. Symptoms of HH1 include fatigue, skin bronzing/ darkening, diabetes, arthritis, cardiomyopathy, and liver and neurodegenerative diseases.

Nutrition Advice

If HH1 is suspected, consult with a health professional. If iron excess is confirmed, limit intake of iron-rich foods – particularly haem iron from animal sources (e.g. oysters, mussels, red meat, and liver) which is easily absorbed. Some plant foods such as dried fruits (e.g. apricots and prunes) and pulses (e.g. beans, peas, and lentils) are also high in iron. Limit or avoid alcohol. Avoid high dose vitamin C as it increases iron absorption and mobilisation. Routine phlebotomy (blood letting) treatment is effective in reducing iron levels and preventing further damage.

Your Results

HFE rs1800562	AG	[C282Y] Heterozygous (one A allele). Increased risk of HH1 if also a H63D carrier (see other SNP). Although usually asymptomatic, carrier parents can pass on the risk allele to their child. Test blood iron levels before adopting a low iron diet.
HFE rs1799945	CC	[H63D] No variance (no G allele). No risk of hereditary haemochromatosis unless a C282Y carrier (see other SNP).

Iron (Deficiency)

Iron deficiency is the most common nutritional deficiency worldwide. It occurs when iron loss is not adequately compensated by dietary intake, eventually leading to iron deficiency anaemia (IDA).

Symptoms of IDA include fatigue, weakness, pale skin, chest pain, heart palpitations, cold hands and feet, brittle nails, hair loss, tongue soreness, and poor immune function.

The enzyme matriptase-2 (MT-2), encoded by the TMPRSS6 (transmembrane protease serine 6) gene, suppresses the iron regulatory hormone hepcidin, enabling iron absorption and recycling (the opposite effect of the HFE gene). A common variant of TMPRSS6 (known as A736V) lowers MT-2 production resulting in elevated hepcidin, reduced iron absorption, and lower iron and haemoglobin levels. The variance can result in a spectrum of conditions, from mild iron deficiency to severe Iron-Refractory Iron Deficiency Anaemia (IRIDA) – which is usually only found in homozygotes. A key hallmark of IRIDA is microcytic anaemia (small, pale-red blood cells with low haemoglobin content).

Non-genetic risk factors for iron deficiency include low dietary intake, malabsorptive conditions (e.g. coeliac disease, Crohn's disease or inflammatory bowel disease), excessive bleeding (including menstruation), pregnancy, exercise (iron loss due to haemolysis and sweating), and inflammation (due to chronic diseases such as rheumatoid arthritis or obesity). These risk factors shoud be considered alongside genetics.

Nutrition Advice

If iron deficiency is suspected, the underlying cause should be identified before using iron supplements. Mild deficiency can usually be corrected by consuming iron rich foods – particularly haem iron from animal sources (e.g. oysters, mussels, red meat, and liver) which are most easily absorbed. Some plant foods such as dried fruits (e.g. apricots and prunes) and pulses (e.g. beans, peas, and lentils) are also high in iron. Vitamin C can help increase iron absorption and mobilisation. As IRIDA is not responsive to dietary/oral iron, intravenous iron treatment may be needed.

Your Results

TMPRSS6 rs855791	GA	Lower matriptase-2 production, elevated hepcidin and inhibition of iron absorption. Risk of mild iron deficiency and anaemia. Slightly increased risk of Iron-Refractory Iron Deficiency Anaemia (IRIDA). Review alongside HFE (genotype) results. Mild deficiency can usually be corrected by consuming iron rich foods - particularly haem iron from animal sources, or supplementation. If IRIDA is suspected consult with a health professional.

Manganese

Manganese is an essential trace element involved in energy metabolism, antioxidant function, detoxification, immunity, fertility, bone health, and regulation of brain and nerve function. Both deficiency and excess can lead to adverse health outcomes. Excessive exposure is associated with increased risk of behavioural disorders in children and neurodegenerative diseases in adults.

The ZIP8 protein, encoded by the SLC39A8 (solute carrier family 39 member 8) gene, is a divalent metal ion transporter which mediates the cellular uptake of manganese and zinc and contributes to the uptake of selenium. It promotes manganese reabsorption (from bile in the liver, and in the kidneys), impacting systemic levels and thereby regulating the activity of manganese dependent enzymes. It also imports (cytotoxic) cadmium, which is found in cigarette smoke. A SNP on SLC39A8 is associated with manganese deficiency, which can have wide-ranging health impacts. These can be mitigated by increasing manganese intake.

Superoxide dismutase 2 (encoded by SOD2) is a manganese-dependent mitochondrial protein. It plays a key role in limiting the negative effects of reactive oxygen species (ROS) by binding to superoxide, a byproduct of the electron transport chain (ETC), and converting it to hydrogen peroxide and oxygen. A common variant on SOD2 is associated with reduced function and increased DNA damage, leading to premature ageing, organ deterioration and pathologies such as cancer and heart disease.

Nutrition Advice

Dietary sources of manganese include whole grains, oats, legumes, nuts, cloves, cinnamon, seafood, and tea. Occupational exposure in industries such as mining, welding, and battery manufacturing can lead to manganese accumulation and toxicity. Manganese is also a component of fungicides, petrol additives, paints and cosmetics.

Your Results

SLC39A8 rs13107325	CC	Normal ZIP8 activity and absorption and uptake of manganese into cells. No increased need for manganese.
SOD2 rs4880	GA	Reduced SOD2 activity and more potential for excess superoxides and free radical damage. Increased risk of oxidative stress, premature ageing and some cancers. Optimise manganese levels via diet - nuts, beans and shellfish, or supplementation.

Selenium

Selenium is an essential trace element, which plays critical roles in thyroid hormone metabolism, DNA synthesis, fertility, and protection from oxidative damage and infection.

Deficiency is linked to risk of autoimmune conditions – scleroderma, Hashimoto's thyroiditis, rheumatoid arthritis and Raynaud's; as well as allergies and asthma, epilepsy, osteoarthritis, male infertility; and higher risk of mercury, arsenic and cadmium accumulation. Selenium toxicity can occur with excess dietary intake. Symptoms include hair loss, nail brittleness, headaches, fatigue, skin rashes and nausea.

Selenium is a constituent of twenty-five selenoproteins including glutathione peroxidase (GPX) and thioredoxin reductase (TxnRd) families – involved in antioxidant defence; iodothyronine deiodinases (DIO) – thyroid hormone metabolism; and Selenoprotein P (SEPP1).

SEPP1, encoded by SELENOP, transports selenium to peripheral tissues – the brain, testis and pancreas. It also functions as an extracellular antioxidant. SELENOP SNPs confer lower function and risk of selenium deficiency.

GPX proteins use glutathione to protect cells from oxidative damage. GPX1, the most abundant, targets hydrogen peroxide, while GPX4 protects against damage induced by iron (ferroptosis) and oxidised fats (from high-temperature frying) and acts as a structural protein in sperm, impacting male fertility.

Nutrition Advice

Brazil nuts, seafood, meat, poultry, and organ meats are the richest sources of selenium. Sulphur-rich foods like broccoli, onions, and garlic, or supplements such as N-acetylcysteine (NAC), provide cysteine for glutathione synthesis. Limit fried foods, which increase oxidative stress.

Your Results

GPX1 rs1050450	--	No result. It was not possible to read the genetic code at this location.
GPX4 rs713041	CC	Normal (good) GPX4 function and protection of cells from oxidative damage. Ensure adequate intake of selenium and glutathione.
SELENOP rs3877899	CC	Normal Selenoprotein P activity and transport of selenium into cells. This is not associated with selenium deficiency.
SELENOP rs7579	CC	Normal Selenoprotein P activity and transport of selenium into cells. This is not associated with selenium deficiency.

Zinc

Zinc is an essential trace mineral found in all cells and serves as a cofactor for over three-hundred enzymes. It plays crucial roles in immune regulation, wound healing, blood clotting and thyroid function. Zinc is also necessary for taste, smell, and normal growth and development. Since only limited amounts can be stored in muscles and bones, regular intake is essential.

Zinc homeostasis is tightly regulated by zinc transporters ZIP8 and ZNT8. The ZIP8 protein, encoded by SLC39A8 (solute carrier family 39 member 8), mediates import of zinc, manganese and cadmium into cells. A ZIP8 SNP is associated with reduced function and increased risk of inflammatory conditions including Crohn's disease, obesity, schizophrenia, and bacterial infections. ZNT8 (SLC30A8, solute carrier family 30 member 8) transports zinc out of cells. A SNP on ZNT8 upregulates it, depleting pancreatic beta-cells of zinc and increasing the risk of type 2 diabetes. Both SNPs indicate higher zinc requirements.

Zinc deficiency can lead to many symptoms including canker sores, acne, dermatitis, thin and brittle hair, weak nails, recurrent infections, inflammatory disorders, diabetes, menstrual irregularities, fatigue, depression, anxiety, ADHD-type symptoms and constipation. It is more common in individuals with hypothyroidism, gastrointestinal diseases and following restricted diets (e.g. vegetarian or vegan).

Although much rarer than deficiency, zinc overload can also be problematic and lead to digestive issues, headaches, and copper and iron deficiencies.

Nutrition Advice

Meat, fish and shellfish (especially oysters), poultry, cereals, beans, nuts, seeds, and dairy products are good sources of zinc. Be aware of excess zinc intake as it can lead to copper insufficiency.

Your Results

SLC30A8 rs13266634	CC	Higher ZNT8 activity and more transport of zinc out of pancreatic beta cells. Risk of zinc deficiency and type 2 diabetes. Higher (up to 25%) zinc intake may be needed.
SLC39A8 rs13107325	CC	Normal ZIP8 activity - normal import of zinc into cells. Not linked to risk of deficiency. Normal (not higher) zinc requirements.

Arsenic

Arsenic, a naturally occurring element, is highly toxic to humans. Most long-term exposure to inorganic arsenic is via contaminated ground water used for drinking, food preparation and irrigation of crops. Other sources are industrial processing, eating contaminated food and smoking tobacco. Rice is particularly susceptible to arsenic uptake.

Symptoms of arsenic toxicity (arsenicosis) include fatigue, skin thickening, neuropathy, diabetes, heart disease, and cognitive impairment. Chronic exposure also increases the risk of developing several cancers (e.g. skin, lung, liver and bladder).

Inorganic arsenic (IA) is metabolised by arsenite methyltransferase, encoded by the AS3MT gene. It initially converts IA to highly-toxic monomethylarsonous acid (MMA (III)), and subsequently to less-harmful dimethylarsinic acid (DMA (V)).

Variants on the AS3MT gene affect the ability to detoxify arsenic. Carriers of the M287T SNP (C allele) have higher first-step methylation capacity, increasing the risk of MMA (III) toxicity. Whereas, the 529-56G>C SNP (C allele) confers a faster conversion of MMA (III) to DMA (V) reducing the risk of arsenicosis. Additionally, a SNP on the MTHFR gene can impact the availability of SAMe (S-adenosyl-methionine), the cofactor needed to support AS3MT activity.

Nutrition Advice

To reduce risk, choose safe (low arsenic) sources of drinking-water, particularly in regions where arsenic contamination is significant, and avoid excessive consumption of rice (especially brown rice), fish and shellfish. Support AS3MT activity by optimising methylation – with B vitamins, zinc and magnesium. Glutathione can help with detoxification and counter oxidative stress due to arsenic exposure.

Your Results

AS3MT rs11191439	TT	[M287T] Normal (not faster) first methylation step - conversion from inorganic arsenic to more toxic MMA (III). No increased risk of arsenic toxicity. Limit arsenic exposure from contaminated drinking water and foods (particularly brown rice), as well as tobacco and cosmetics.
AS3MT rs3740393	GG	[529-56G>C] Wild-type (most common). Less efficient second methylation step - slower conversion from toxic MMA (III) to DMA (V). Increased risk of arsenic toxicity. Minimise arsenic exposure from contaminated drinking water and foods (particularly brown rice), tobacco and cosmetics. Optimise methylation and antioxidants (glutathione etc.).
MTHFR rs1801133	AG	[C677T] Reduced methyl-folate synthesis - a key step in providing SAMe to support AS3MT activity. Increase intake of B vitamins (including folate) and cofactors - magnesium and zinc to optimise methylation (and SAMe).

Mercury

Mercury is a heavy metal that is toxic to humans. It exists in different forms – elemental, inorganic and organic. Methylmercury, found in contaminated fish and seafood, is the major source of organic mercury for humans. Elemental mercury is used in dental amalgams, thermometers and industrial processes such as paper production. All forms of mercury can accumulate in organs causing damage to kidneys, brain, heart and lungs.

The effects of mercury toxicity vary depending on the type and duration of exposure. Organic mercury can impact fertility and pregnancy and result in developmental delays and behavioural issues (such as excitability and restlessness) in children. It can also cause a broad range of neurological symptoms – headaches, depression, memory loss, mood changes, tremors, numbness and cognitive and motor dysfunction – at any life stage. Elemental mercury can have toxic effects on the digestive and immune systems, as well as skin and eyes.

The glutathione S-transferase (GST) genes use glutathione to bind mercury for excretion via urine and bile. SNPs on GSTP1 and GSTT1 impact mercury clearance increasing risk of toxicity. Metallothionein proteins are also involved in mercury detoxification (see Metallothioneins).

Nutrition Advice

To reduce exposure to mercury limit consumption of large fish (such as swordfish and tuna) which may contain high levels, and avoid (or remove) amalgam fillings. Sulphur-rich foods such as garlic, cruciferous vegetables and leeks help replenish glutathione levels and support GST activity. Consider supplementing N-acetyl Cysteine (NAC) or glutathione alongside other antioxidants – alpha lipoic acid (ALA) and vitamins C, E and selenium.

Chelation therapy using agents such as dimercaptosuccinic acid (DMSA) can be effective at removing mercury, but should be used under medical supervision.

Your Results

GSTP1 rs1138272	CC	Normal (good) glutathione S-transferase Pi 1 function and ability to detoxify mercury. If exposed to mercury, increase intake of antioxidants - glutathione, vitamins C and E, and selenium.
GSTP1 rs1695	AA	Normal (good) glutathione S-transferase Pi 1 function and ability to detoxify mercury. If exposed to mercury, increase intake of antioxidants - glutathione, vitamins C and E, and selenium.
GSTT1 GSTT1	II	The GSTT1 gene is present. Normal (good) glutathione S-transferase Theta 1 function and ability to detoxify mercury. If exposed to mercury, increase intake of antioxidants - glutathione, vitamins C and E, and selenium.

Metallothioneins

Metallothioneins (MTs) are a family of cysteine-rich proteins whose main function is to maintain metal homeostasis, especially the storage and availability of zinc. They protect from heavy metal toxicity by binding to cadmium, copper, arsenic, lead and mercury. Additionally they have a major role in controlling oxidative stress. MT production is dependent on dietary minerals such as zinc, copper and selenium as well as the amino acids histidine and cysteine.

Variants on MT1A and MT2A genes are associated with lower activity, reduced free radical scavenging and increased oxidative stress. The most studied SNP on MT2A, known as 5A/G, is linked to increased risk of type 2 diabetes, kidney disease, age-related macular degeneration, and some cancers. Variance on MT1A can disrupt zinc availability for superoxide dismutase (CuZn-SOD) and insulin production, increasing the risk of type 2 diabetes.

Symptoms of poor metallothionein function include chronic fatigue, headaches, irritability, cognitive impairment, behavioural disorders (in children), dementia (in adults), abdominal discomfort, joint and bone pain, osteoporosis, dental caries, skin issues (flushing, rashes), metabolic syndrome, obesity, type 2 diabetes, cancers and cardiovascular diseases.

Nutrition Advice

To support MT function optimise intake of zinc (oysters and other shellfish are good sources) and selenium – found in Brazil nuts. Include high protein foods (e.g. meat, eggs and yoghurt) to provide the amino acids to make MTs.

Limit heavy metal exposure – arsenic (contaminated water, pesticides and herbicides, rice from certain origins), cadmium (cigarette and vape smoke), lead (old paint), and mercury (from seafood and amalgam dental fillings).

Your Results

MT1A rs8052394	AA	Normal (good) metallothionein function and antioxidant defence. Less risk of heavy metal toxicity. Ensure adequate zinc and selenium intake.
MT2A rs28366003	--	No result. It was not possible to read the genetic code at this location.

How to Read the Report

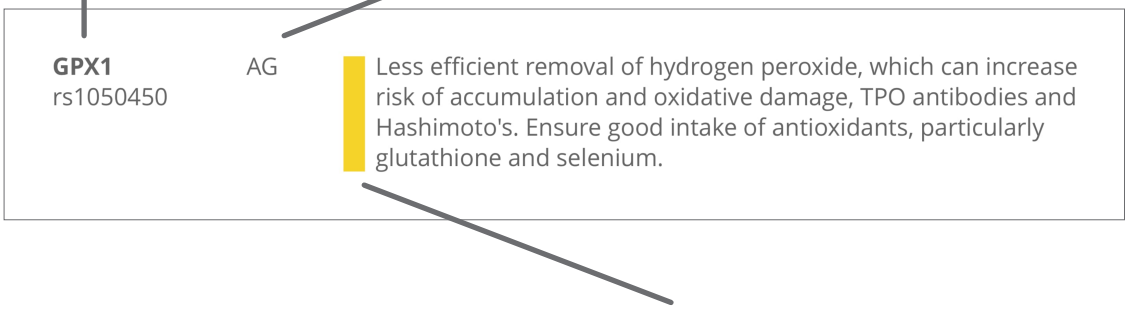
Genes

Results are listed in order of the gene short name. The 'rs' number is the reference sequence number that identifies a specific location on the genome. It is also known as a SNP (Single Nucleotide Polymorphism) pronounced 'snip', polymorphism or mutation.

Personalised Result

Your genotype result is shown as two letters (A,G,T or C) which represent the DNA bases present at that location.

Multiple attempts are made to achieve the required level of statistical confidence, but if that cannot be met it is reported as **'No result'**.



Highlight Colour

The genotype result highlight indicates the potential effect of the SNP on gene function in a particular context.

RED the effect of the variant is negative

AMBER the effect of the variant is somewhat negative

GREEN no variation, or the effect of the variant is positive

References

ACE Angiotensin I Converting Enzyme

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AGTR1 Angiotensin II Receptor Type 1

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AGT Angiotensinogen

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AS3MT Arsenite Methyltransferase

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ATP2B1 ATPase Plasma Membrane Ca2+ Transporting 1

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ATP7B ATPase Copper Transporting Beta

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CASR Calcium Sensing Receptor

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CP Ceruloplasmin

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GPX1 Glutathione Peroxidase 1

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GPX4 Glutathione Peroxidase 4

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GSTP1 Glutathione S-Transferase Pi 1

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GSTT1 Glutathione S-Transferase (GST) Theta 1

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HFE Homeostatic Iron Regulator

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KL Klotho

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MT1A Metallothionein 1A

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MT2A Metallothionein 2A

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MTHFR Methylenetetrahydrofolate Reductase (NAD(P)H)

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SELENOP Selenoprotein P

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SLC30A8 Solute Carrier Family 30 (zinc transporter), Member 8

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SLC39A8 Solute Carrier Family 39 Member 8

Dragasevic S, Stankovic B, Kotur N, Milutinovic AS, Milovanovic T, Stojkovic Lalosevic M, Stojanovic M, Pavlovic S, Popovic D. Genetic Aspects of Micronutrients Important for Inflammatory Bowel Disease. Life (Basel). 2022 Oct 18;12(10):1623. doi: 10.3390/life12101623. PMID: 36295058; PMCID: PMC9604584. (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9604584/)

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SOD2 Superoxide Dismutase 2, Mitochondrial

Anagianni S, Tuschl K. Genetic Disorders of Manganese Metabolism. Curr Neurol Neurosci Rep. 2019 May 14;19(6):33. doi: 10.1007/s11910-019-0942-y. PMID: 31089831; PMCID: PMC6517356. (https://pmc.ncbi.nlm.nih.gov/articles/PMC6517356/)

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TMPRSS6 Transmembrane Serine Protease 6

Jallow MW, Cerami C, Clark TG, Prentice AM, Campino S. Differences in the frequency of genetic variants associated with iron imbalance among global populations. PLoS One. 2020 Jul 1;15(7):e0235141. doi: 10.1371/journal.pone.0235141. PMID: 32609760; PMCID: PMC7329092. (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7329092/)

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TRPM6 Transient Receptor Potential Cation Channel Subfamily M Member 6

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