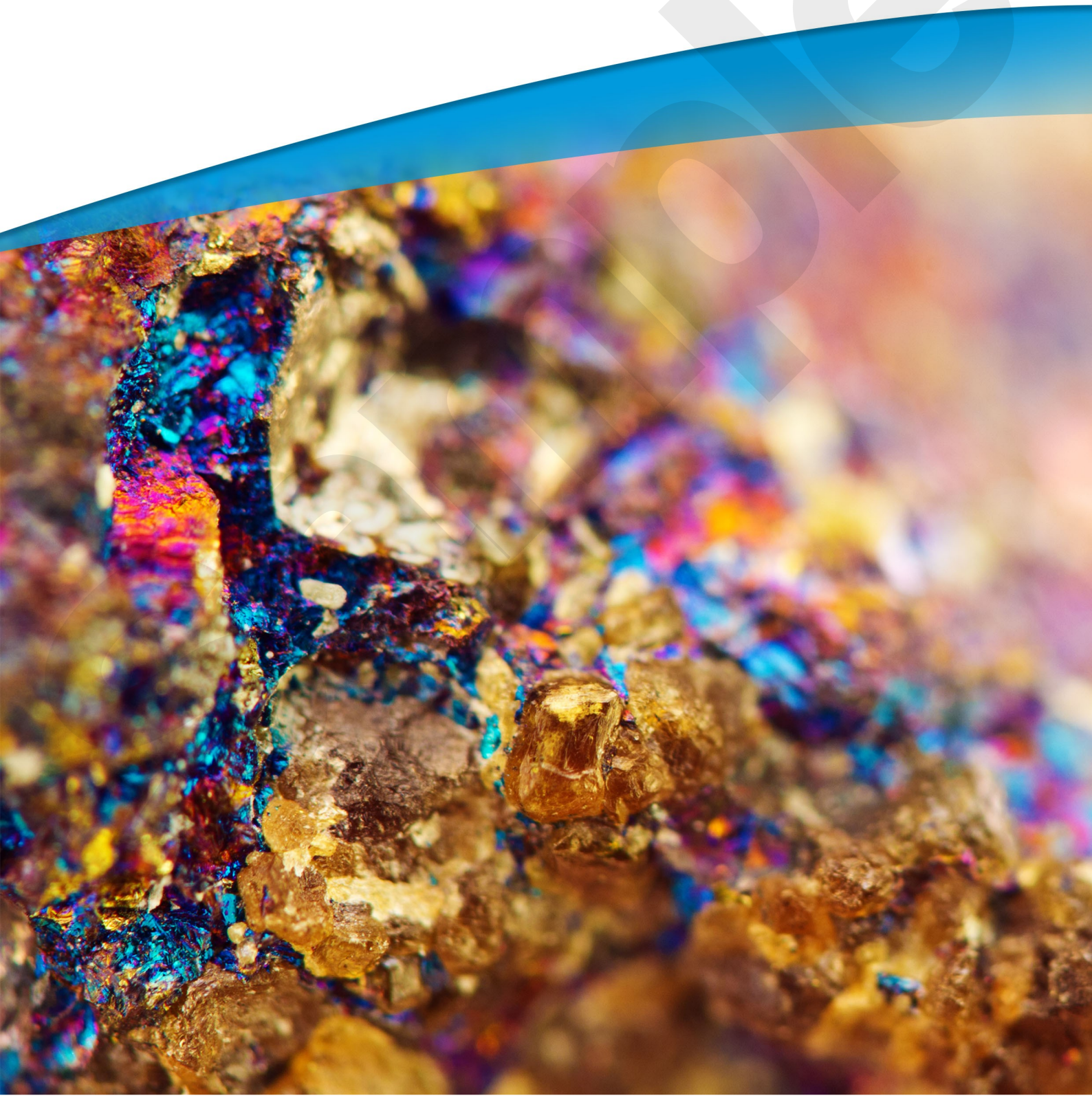
Lifecode GX ®
— Professional Genotype Analysis —

Metals and Minerals Report



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.

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 GT

Increased klotho activity. Good regulation of phosphorus (less risk of excess). Associated with better cognitive function, higher bone mineral density (and protection against osteoporosis) and longevity.

Ensure adequate calcium intake (at least equal to phosphorus). Limit consumption of processed foods - such as dark colas - which contain added phosphate.

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	AG	Higher ACE activity (G allele) 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	CC	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	GG	Higher angiotensinogen and increased risk of sodium and water retention, high blood pressure and hypertension. Limit dietary salt (sodium) and ensure good potassium intake.

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 [redacted] GG

No variance. Not a C282Y carrier (no A allele).

No risk of hereditary haemochromatosis unless at H63D carrier (see other SNP).

HFE [redacted] GC

[H63D] Heterozygous (one G allele). Likely not at risk of HH1 unless also an C282Y carrier (see other SNP). Although usually asymptomatic, carrier parents may pass on the risk allele to their child.

Test blood iron levels before adopting a low iron diet.

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omitted in the sample

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