HAIR MINERAL ANALYSIS

Hair Mineral Analysis.

SUBJECT

Demo Patient

Male Adult · Human — Head · DOB 1980-01-01

LAB NUMBER

DEMO-HIGHBURDEN

Previous

SAMPLE COLLECTED

2026 · 07 · 03

RECEIVED IN LAB

2026 · 07 · 03

REPORT RELEASED

2026 · 07 · 03

For practitioner use only. Not for direct distribution to the patient.

REQUISITIONING

Dr. Demo Practitioner

Practitioner ID · 200

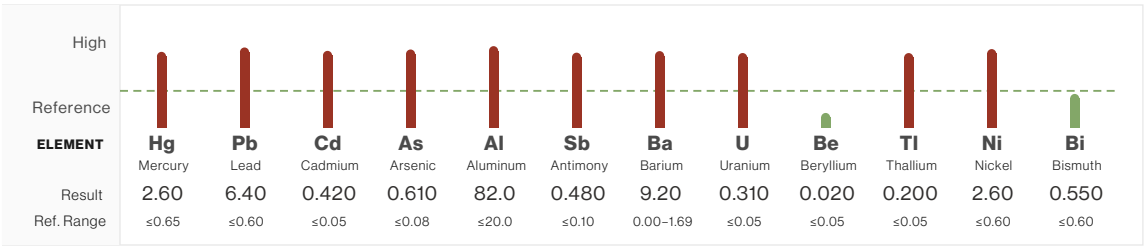
Hair Mineral Analysis | Demo Patient | Head hair | Male Adult

CanAlt Health Laboratories

Sample ID **DEMO-HIGHBURDEN** New Sample Practitioner **Dr. Demo Practitioner (200)**

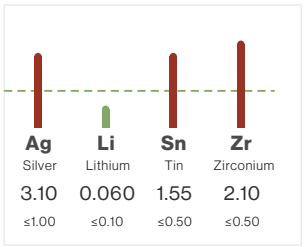
DOB **1980-01-01** Collected **2026-07-03** Received **2026-07-03** Report **2026-07-03**

HEAVY METALS



ELEMENT	Hg	Pb	Cd	As	Al	Sb	Ba	U	Be	Tl	Ni	Bi
	Mercury	Lead	Cadmium	Arsenic	Aluminum	Antimony	Barium	Uranium	Beryllium	Thallium	Nickel	Bismuth
Result	2.60	6.40	0.420	0.610	82.0	0.480	9.20	0.310	0.020	0.200	2.60	0.550
Ref. Range	≤0.65	≤0.60	≤0.05	≤0.08	≤20.0	≤0.10	0.00–1.69	≤0.05	≤0.05	≤0.05	≤0.60	≤0.60

OTHER ELEMENTS



ELEMENT	Ag	Li	Sn	Zr
	Silver	Lithium	Tin	Zirconium
Result	3.10	0.060	1.55	2.10
Ref. Range	≤1.00	≤0.10	≤0.50	≤0.50

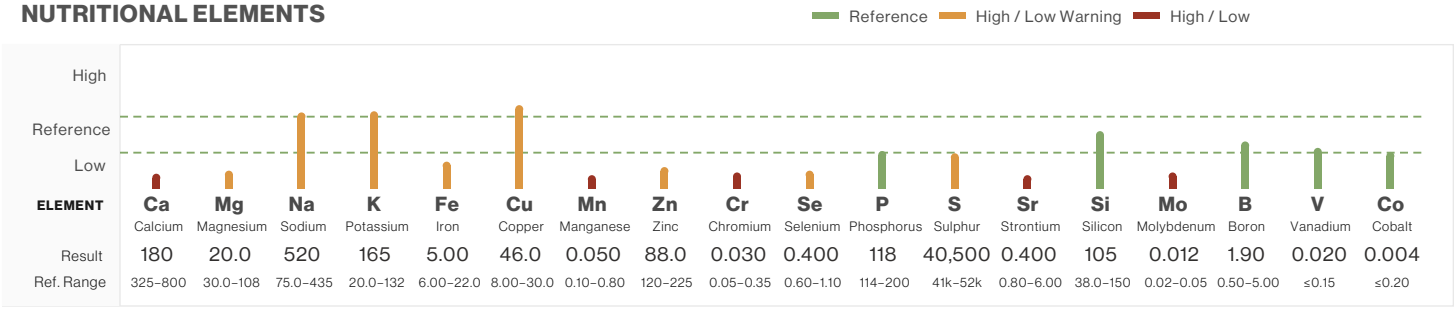
MINERAL-HEAVY METAL RATIOS

Protective mineral-to-toxic metal ratios reflect the body's capacity to buffer against heavy metal burden.

Ratio	Current	Range
Ca : Cd	428.6 ▼	>800
Fe : Pb	0.78 ▼	>20.0
Zn : Hg	33.8 ▼	>200
Se : Hg	0.15 ▼	>1.00

Ratio	Current	Range
Se : As	0.66 ▼	>10.0
Fe : Hg	1.9 ▼	>22.0
Ca : Pb	28.1 ▼	>84.0
Zn : Cd	209.5 ▼	>800

NUTRITIONAL ELEMENTS



ELEMENT	Ca	Mg	Na	K	Fe	Cu	Mn	Zn	Cr	Se	P	S	Sr	Si	Mo	B	V	Co
	Calcium	Magnesium	Sodium	Potassium	Iron	Copper	Manganese	Zinc	Chromium	Selenium	Phosphorus	Sulphur	Strontium	Silicon	Molybdenum	Boron	Vanadium	Cobalt
Result	180	20.0	520	165	5.00	46.0	0.050	88.0	0.030	0.400	118	40,500	0.400	105	0.012	1.90	0.020	0.004
Ref. Range	325–800	30.0–108	75.0–435	20.0–132	6.00–22.0	8.00–30.0	0.10–0.80	120–225	0.05–0.35	0.60–1.10	114–200	41k–52k	0.80–6.00	38.0–150	0.02–0.05	0.50–5.00	≤0.15	≤0.20

ENERGY & METABOLISM RATIOS

Mineral patterns governing thyroid, adrenal, and cellular energy function.

Ratio	Current	Range
Ca : Mg	9.0	5.00–20.0
Ca : Na	0.35 ▼	0.50–5.00
Ca : K	1.1 ▼	2.00–6.00
Na : Mg	26.0 ▲	0.50–5.00
Na : K	3.2	2.00–4.00
K : Mg	8.2 ▲	0.50–5.00

STRUCTURAL RATIOS

Ratios tied to bone density, connective tissue, and iron–copper balance.

Ratio	Current	Range
Ca : P	1.5 ▼	1.60–4.00
Ca : Sr	450.0	150–650
Ca : Zn	2.0	2.00–8.00
Fe : Cu	0.11 ▼	0.26–1.20
Zn : Cu	1.9 ▼	4.00–17.0
Ca : Fe	36.0	20.0–85.0

ADDITIONAL RATIOS

These ratios are provided for additional information only.

Ratio	Current
Mg : P	0.17
Fe : Mn	100.0
Cu : Mo	3,833
Cu : Se	115.0

Ratio	Current
S : Cu	880.4
Zn : Fe	17.6
Zn : P	0.75
Zn : Se	220.0

Disclaimer: Results for Ca, Mg, Na, K, and Li as well as related ratios and patterns are provided for research and informational purposes only — see full disclaimer.

OXIDIZER TYPE Extreme Fast

Cut-offs: Ca/K < 1 | Na/Mg > 16.6

This marks a strongly accelerated pattern. In the report this extreme fast signature means the entire mineral profile must be viewed through the lens of very high metabolic speed. Sodium or potassium elevations often signal acute stress responses, while calcium magnesium depressions are pronounced, and toxic metals may appear elevated due to rapid but unsustainable turnover. The body experiences intense alarm stage metabolism with very high sympathetic activity and rapid nutrient depletion. A nutritional and lifestyle approach that strongly emphasizes stabilization, deep rest, and reduction of any stimulating factors is usually most appropriate to help moderate the intensity and protect long term reserves.

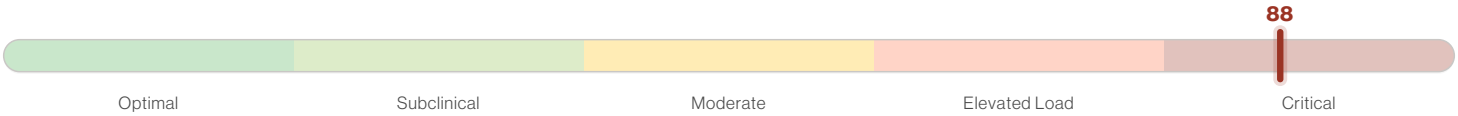
Notable Observations

- Intense anxiety, panic-like episodes, or extreme irritability and aggression commonly seen.
- Very high sympathetic drive; may include heart palpitations or strain.
- Rapid exhaustion after even short bursts of activity.
- High body temperature, excessive sweating, and strong inflammatory responses are typical.
- Quick nutrient burnout and risk of acute stress collapse are significant concerns.
- Often observed in high-stress, acute alarm phase presentations.

MACROMINERAL PATTERNS

	STATUS	CRITERIA
Calcium Shell	—	Ca > 2000 µg/g
Magnesium Shell	—	Mg > 200 µg/g and Mg > 0.15 × Ca
Extreme Na/K	—	Na/K < 1.0 or Na/K > 10.0
Sympathetic Dominance	—	Ca/K < 4.0 and Ca/Mg < 6.0
Parasympathetic Dominance	—	Ca/Mg > 9.5 and Ca/K > 10.0
Four-Low	—	Ca < 400, Mg < 60, Na < 250, K < 100 µg/g
Four-High	—	Ca > 875, Mg > 100, Na > 370, K > 115 µg/g
Reduced Buffering Capacity	—	Ca > 875, Mg > 100, Sr > 6.0, Ba > 3.0 µg/g
Bio-unavailable Ca and Mg	—	Ca > 2000 µg/g, Mg > 100 µg/g

ELEMENTAL BURDEN INDEX High



The Elemental Burden Index summarizes overall load from essential and toxic elements; it is covered in detail separately. The Advanced HMA Report provides a comprehensive breakdown spanning Stress, Nutrition, Lifestyle factors, Cellular Metabolism, and Hormone activity, all derived from the patient's hair mineral and toxic metal profile.

Disclaimer: Any information regarding Ca, Mg, Na, K and Li is for research and informational purposes only.

HMA Methodology.

Interpretive guidance draws on the measured element values, their relationships to one another, and the peer-reviewed literature describing their patterns of utilization, deposition, exposure, and excretion.

01 · ESTABLISH
Pattern
Macromineral framework: Ca·Mg for direction, Na·K for stress response.

02 · CLARIFY
Ratios
Balance, compensation, antagonism — relationships rather than absolutes.

03 · OBSERVE
Trend
Months-long view across repeats — coherence and directional change.

04 · APPLY
Sequence
Prioritize, sequence, and pace foundational regulation work.

INTERPRETIVE APPROACH

Trace nutritional minerals are evaluated as indicators of nutritional status, longer-term demand, and utilization, with attention to relationships among elements rather than to single values in isolation. Toxic and non-essential elements are evaluated as indicators of exposure and elimination patterns — not measures of total body burden, and not diagnostic of poisoning or any specific toxicological condition.

LIMITS OF HAIR MINERAL ANALYSIS

Hair mineral analysis is a longitudinal, statistical method. Its results are subject to hair treatments, environmental exposures, individual physiology, washing and preparation protocols, and analytical variability. It is well suited to identifying patterns and trends across repeat analyses, and is not a substitute for clinical history, physical assessment, or confirmatory testing where clinically indicated.

DISCLAIMER

The following terms apply to all information in this hair mineral analysis report. **By using this report, the requisitioning practitioner accepts these terms.**

PRACTITIONER-ONLY DISTRIBUTION

Intended solely for the requisitioning practitioner — a regulated health professional whose scope authorizes ordering and interpreting laboratory tests for the named patient. Not for direct distribution to the patient without the practitioner's own clinical interpretation.

NOT DIAGNOSTIC

The analytical results and literature summaries do not constitute a medical diagnosis. Results may be influenced by environmental exposures, hair treatments, individual physiology, and methodological variables. Interpret alongside medical history, physical assessment, and confirmatory testing as clinically appropriate. No diagnosis or course of treatment is implied.

LIMITS OF HAIR MINERAL ANALYSIS

A longitudinal, statistical method whose results are subject to environmental exposures, hair treatments, individual physiology, washing and preparation protocols, and analytical variability. Well suited to identifying patterns and trends across repeat analyses; not a substitute for clinical history, physical assessment, or confirmatory blood, urine, or imaging testing.

PROFESSIONAL RESPONSIBILITY

Nothing here replaces or diminishes the practitioner's professional obligations under applicable laws, regulations, and standards of practice. The practitioner retains full responsibility for any clinical decisions made in connection with this report. Literature references are provided for convenience, not as laboratory endorsements; consult the originals and exercise independent judgment.

ISSUING LABORATORY

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CanAlt Health Laboratories

DEMO PATIENT

CALCIUM (Ca) **LOW · OUT OF RANGE**

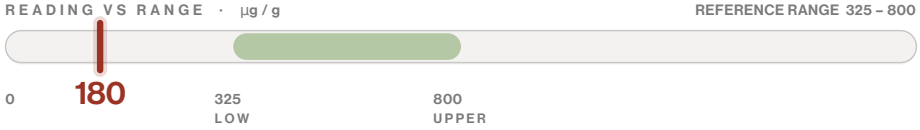
The information on this page is provided to support the practitioner's independent clinical assessment, drawn from the published mineral balancing literature, alongside clinical history, physical assessment, and confirmatory testing where clinically appropriate.

20 40.078
Ca
Calcium
Below range

HAIR MINERAL ANALYSIS · ELEMENT REFERENCE

READING VS RANGE · µg / g

REFERENCE RANGE 325 – 800



Reference range · Current 180 µg/g

SCAN FOR REFERENCED LITERATURE

Calcium underpins skeletal structure, neuromuscular signalling, cellular stability, and alkaline buffering.

The mineral-balancing literature has described calcium as a slow-acting structural mineral; depletion of the hair value has been reported to reflect long-term shifts in metabolic pacing rather than dietary insufficiency alone. The literature has described a low hair calcium reading as a marker of fast oxidation or exhaustion-phase physiology: reserves shed under prolonged sympathetic drive, or globally suppressed when adaptive capacity is depleted. Hair calcium is read alongside the stress markers (Na, K) and the broader macromineral pattern rather than the calcium value alone.

SCENARIO REFERENCE

Scenarios described in the published mineral-balancing literature. These are descriptions of the taxonomy, not characterizations of the patient.

RELATES TO	LITERATURE DESCRIPTION	RELATED READINGS
Adrenal & stress response	A low calcium reading has been associated with fast-oxidation, sympathetic-alarm physiology, where calcium is shed as the Ca:K and Ca:Na ratios collapse under adrenal drive. It is interpreted against the stress ratios and the oxidation-rate markers. ***Watts DL, Trace Elements Inc. — HTMA fast-oxidation convention***	Sympathetic Dominance · Oxidizer Type: Fast/Extreme Fast · Ca 180 (▼) · K 165 (▲) · Na 520 (▲) · Ca:K 1.09 (·) · Ca:Na 0.346 (▼)
Nervous system & neuromuscular tone	Low calcium has been linked with heightened neuromuscular excitability, research describing reduced calcium as lowering the excitability/seizure threshold. It is read holistically against magnesium and the Ca:Mg balance. ***PMID 25810356***	Ca 180 (▼) · Mg 20 (▼) · Ca:Mg 9 (·) · K 165 (▲) · P 118 (·)
Toxic-metal displacement & body burden	A low calcium reading has been associated with lead and cadmium displacing calcium from bone through chemical mimicry; the protective Ca:Pb and Ca:Cd ratios are read alongside the reading. ***PMID 41782652***	Ca 180 (▼) · Pb 6.40 (▲) · Cd 0.420 (▲) · Ca:Pb 28.1 (▼) · Ca:Cd 429 (▼) · Al 82 (▲)

☐ IDENTIFIED ☐ BORDERLINE ☐ NOT IDENTIFIED

NUTRITIONAL COFACTOR NOTES

SYNERGISTS

Vitamin D: drives intestinal calcium absorption through TRPV6 and calbindin pathways. PMID 12748856 · Nijenhuis et al. 2003

Vitamin K2: activates osteocalcin to deposit calcium into bone rather than soft tissue. PMID 39125301 · Aaseth et al. 2024

Gastric acid: is required to ionize dietary calcium for absorption; PPI use and achlorhydria reduce uptake. PMID 32718584 · Koyyada et al. 2021

Magnesium: is required for parathyroid hormone secretion and vitamin D activation, regulating calcium availability. PMID 1939521 · Fatemi et al. 1991

ANTAGONISTS

Vitamin D insufficiency caps active calcium transport across the gut wall. ***PMID 17913228*** *· Cashman 2007*

Caffeine: increases renal calcium loss; alcohol disrupts vitamin D metabolism. ***PMID 8360789*** *· Massey 1993*

High dietary phosphorus shifts the Ca: P ratio and drives PTH-mediated bone calcium release. ***PMID 8360792*** *· Calvo 1993*

LITERATURE OBSERVATIONS

PEER-REVIEWED

Lower hair calcium has been consistently reported in metabolic-syndrome research and tracked inversely with insulin resistance (HOMA-IR), pointing to altered mineral handling in metabolic dysfunction.

Children in autism-spectrum research cohorts have shown significantly lower hair calcium than neurotypical controls, often alongside elevated toxic metals.

Patients in schizophrenia research cohorts have shown lower hair calcium than controls, with reduced reserves or increased turnover described in the literature.

Lower hair calcium has been reported alongside more severe coronary artery disease scores (Syntax) and elevated systemic inflammation markers in angiography studies.

Bone mass predictive studies: Reduced hair calcium has appeared in predictive models for low bone mass and osteoporosis risk. ***PMID 33745523*** *· Park et al., 2021*

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DEMO PATIENT

MANGANESE (Mn) **LOW · OUT OF RANGE**

The information on this page is provided to support the practitioner's independent clinical assessment, drawn from the published mineral balancing literature, alongside clinical history, physical assessment, and confirmatory testing where clinically appropriate.

25 54.938

Mn

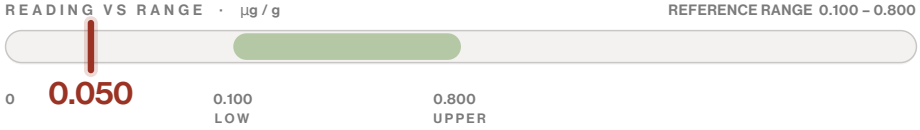
Manganese

Below range

HAIR MINERAL ANALYSIS · ELEMENT REFERENCE

READING VS RANGE · µg / g

REFERENCE RANGE 0.100 – 0.800



Reference range · Current 0.050 µg/g

SCAN FOR REFERENCED LITERATURE

Manganese underpins mitochondrial antioxidant defence through MnSOD, connective tissue integrity, and carbohydrate metabolism.

In HMA, low Mn has been described as a marker of reduced antioxidant resilience and compromised metabolic buffering rather than dietary insufficiency alone. The literature has described it most often in iron-displacement patterns, in restrictive diets, and alongside zinc imbalances.

SCENARIO REFERENCE

Scenarios described in the published mineral-balancing literature. These are descriptions of the taxonomy, not characterizations of the patient.

RELATES TO	LITERATURE DESCRIPTION	RELATED READINGS
Mitochondrial antioxidant defence	A low manganese reading has been associated with reduced capacity of manganese superoxide dismutase, the mitochondrial antioxidant enzyme that depends on manganese as its catalytic metal. Research has linked depressed manganosuperoxide dismutase activity with diminished free-radical scavenging, and it has been read holistically against the other antioxidant trace minerals. ***PMID 3517203***	Mn 0.050 (▼) · Zn 88 (▼) · Cu 46 (▲) · Se 0.400 (▼) · Zn:Cu 1.91 (▼)
Glucose & carbohydrate metabolism	Low manganese has been linked in experimental work with impaired glucose handling, where manganese deficiency was associated with reduced insulin secretion and altered carbohydrate homeostasis. It has been read alongside the other glucose-related trace minerals rather than as a standalone metabolic marker. ***PMID 6379130***	Mn 0.050 (▼) · Cr 0.030 (▼) · Zn 88 (▼) · Mo 0.012 (▼) · Fe:Mn 100 (▲)
Connective tissue & GAG synthesis	Reduced manganese has been associated with the manganese-dependent glycosyltransferases that build glycosaminoglycans, with studies suggesting low manganese relates to impaired connective-tissue and cartilage matrix synthesis. It has typically been framed against the broader trace-mineral pattern that supports structural integrity. ***PMID 3882911***	Mn 0.050 (▼) · Zn 88 (▼) · Cu 46 (▲) · Ca 180 (▼) · P 118 (·)

☐ IDENTIFIED ☐ BORDERLINE ☐ NOT IDENTIFIED

NUTRITIONAL COFACTOR NOTES

SYNERGISTS

Zinc and Mn function together in superoxide dismutase isoforms.

B vitamins (B1, B3, B6) and vitamin C support Mn-dependent enzymes.

Sustained protein intake: supports transferrin and alpha-2-macroglobulin Mn carriers.

ANTAGONISTS

Iron and Mn compete for DMT1 and tissue uptake sites.

High Ca intake: reduces intestinal Mn absorption; excess Cu interferes with Mn enzymes.

Phytic acid: binds Mn at the intestinal wall; low stomach acid compounds the loss.

LITERATURE OBSERVATIONS

PEER-REVIEWED

Children with ASD consistently show significantly lower hair Mn than neurotypical controls, part of broader essential trace element imbalances.

Lower hair Mn is observed in children with ADHD, correlating with symptom severity and neurodevelopmental challenges.

Consistently lower hair Mn in T2DM, linked to glucose metabolism and insulin sensitivity challenges.

Patients (especially pharmacoresistant cases) exhibit markedly lower hair Mn, highlighting Mn's role in neurological stability.

Women with fibromyalgia have significantly lower hair Mn vs. controls, suggesting reduced antioxidant and metabolic support.

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DEMO PATIENT

CHROMIUM (Cr) **LOW · OUT OF RANGE**

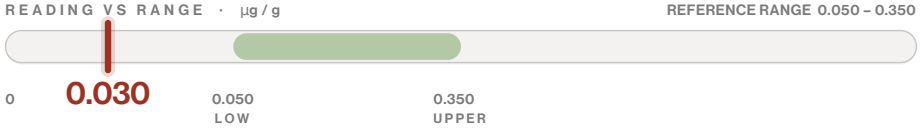
The information on this page is provided to support the practitioner's independent clinical assessment, drawn from the published mineral balancing literature, alongside clinical history, physical assessment, and confirmatory testing where clinically appropriate.

24 51.996
Cr
Chromium
Below range

HAIR MINERAL ANALYSIS · ELEMENT REFERENCE

READING VS RANGE · µg / g

REFERENCE RANGE 0.050 – 0.350



SCAN FOR REFERENCED LITERATURE

Chromium supports insulin sensitivity and efficient cellular glucose uptake.

In HMA, low Cr has been described as a marker of reduced tissue availability from sustained metabolic demand: typically refined-carbohydrate stress, chronic dysglycemia, or high physical activity outpacing intake. Rarely has it been described as a simple dietary insufficiency.

SCENARIO REFERENCE

Scenarios described in the published mineral-balancing literature. These are descriptions of the taxonomy, not characterizations of the patient.

RELATES TO	LITERATURE DESCRIPTION	RELATED READINGS
Glucose & insulin metabolism	A low chromium reading has been associated with reduced tissue availability under sustained metabolic demand, where research has linked repeated insulin surges from refined-carbohydrate stress and chronic dysglycemia to chromium depletion. It has been read holistically against the insulin-cofactor minerals and the Mg:P ratio rather than as simple dietary insufficiency. ***PMID 15208835***	Cr 0.030 (▼) · Mg 20 (▼) · Zn 88 (▼) · Mg:P 0.169 (·) · Na:K 3.15 (▲)
Dietary intake & absorption	A low chromium reading has been associated with sustained low intake of chromium-rich whole foods and with the poor absorption that studies have described for dietary chromium, often paired with broader trace-mineral insufficiency. It has been read alongside the other intake-sensitive trace minerals to frame a whole-pattern shortfall. ***Watts DL, Trace Elements Inc. — HTMA trace-mineral intake patterns***	Cr 0.030 (▼) · Mn 0.050 (▼) · Mo 0.012 (▼) · Zn 88 (▼) · Se 0.400 (▼)
Excretory & metabolic demand	A low chromium reading has been associated with accelerated urinary excretion and sweat losses, where research has linked insulin-stimulated chromodulin turnover and high physical activity to increased chromium loss outpacing intake. It has been read against the cofactor minerals and the Na:K stress ratio to gauge demand. ***PMID 11472024***	Cr 0.030 (▼) · Zn 88 (▼) · Mg 20 (▼) · Na:K 3.15 (▲) · K 165 (▲)

☐ IDENTIFIED ☐ BORDERLINE ☐ NOT IDENTIFIED

NUTRITIONAL COFACTOR NOTES

SYNERGISTS

Zinc and Mn support enzymes that incorporate Cr into chromodulin.

Magnesium: is required for insulin receptor autophosphorylation, the pathway Cr amplifies.

B vitamins (B1, B3, biotin) and vitamin C support glucose-handling enzymes.

ANTAGONISTS

Refined carbohydrates trigger insulin spikes that mobilize Cr to urinary excretion.

Iron and Cr compete for transferrin binding; Fe has much higher affinity.

Low gastric acidity (PPI use, age) reduces uptake of an already poorly absorbed mineral.

LITERATURE OBSERVATIONS

PEER-REVIEWED

Lower hair Cr in MetS patients vs. controls, with negative correlations to fasting glucose, triglycerides, and insulin resistance (HOMA-IR).

Juvenile diabetes: Children with juvenile (type 1) diabetes have significantly reduced hair Cr (0.56 ug/g) vs. healthy peers (0.85 ug/g), linked to altered glucose handling. ***PMID 25192728*** · *Wacewicz et al., 2014*

Reduced hair Cr is associated with elevated insulin resistance markers in adult males, including higher fasting insulin and HOMA-IR.

Lower hair Cr observed in vascular disease patients, correlating with atherogenic lipid profiles (low HDL, high LDL) and increased CVD risk.

Frequent intense exercise correlates with lower hair Cr due to accelerated urinary excretion and sweat losses, exacerbating depletion under metabolic demand.

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DEMO PATIENT

STRONTIUM (Sr) **LOW · OUT OF RANGE**

The information on this page is provided to support the practitioner's independent clinical assessment, drawn from the published mineral balancing literature, alongside clinical history, physical assessment, and confirmatory testing where clinically appropriate.

38 87.62

Sr

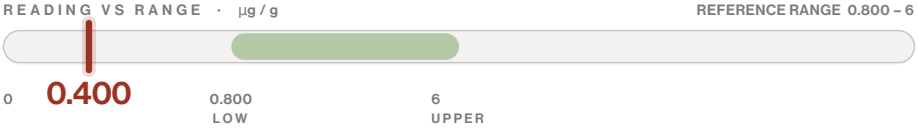
Strontium

Below range

HAIR MINERAL ANALYSIS · ELEMENT REFERENCE

READING VS RANGE · µg / g

REFERENCE RANGE 0.800 – 6



Reference range · Current 0.400 µg/g

SCAN FOR REFERENCED LITERATURE

Strontium is normally present in hair at low concentrations, reflecting its trace role.

In HMA, low Sr has rarely been described as a deficiency. The literature has described very low Sr most often as a marker of reduced mineral elimination capacity, or as a parallel finding to low calcium absorption (Ca and Sr share absorption pathways).

SCENARIO REFERENCE

Scenarios described in the published mineral-balancing literature. These are descriptions of the taxonomy, not characterizations of the patient.

RELATES TO	LITERATURE DESCRIPTION	RELATED READINGS
Bone mineral density & matrix	A low strontium reading has been associated with a thinner alkaline-earth contribution to the bone mineral fraction, where strontium normally tracks calcium in hydroxyapatite and supports matrix resilience. It has been read holistically against calcium, magnesium and the Ca:Sr ratio rather than as a stand-alone deficiency. ***PMID 34207344***	Sr 0.400 (▼) · Ca 180 (▼) · Ca:Sr 450 (·) · Mg 20 (▼) · Ca:Mg 9 (·) · P 118 (·)
Calcium absorption & gut	Low strontium has been linked with the same intestinal handling that governs calcium, since research has shown strontium and calcium share absorption pathways and strontium has been used as a stable tracer of fractional calcium absorption. It has been read alongside calcium, phosphorus and magnesium to frame whether a co-suppressed alkaline-earth group reflects reduced uptake. ***PMID 3115389***	Sr 0.400 (▼) · Ca 180 (▼) · P 118 (·) · Mg 20 (▼) · Ca:P 1.53 (▼) · Mg:P 0.169 (·)
Mineral elimination & turnover	In hair-analysis practice a low strontium reading has been interpreted as part of a poor-eliminator pattern, where strontium falls alongside other minerals that depend on elimination efficiency and turnover. It has been read together with magnesium and the calcium ratios that frame overall mineral mobilisation. ***Watts DL, Trace Elements Inc. — HTMA mineral elimination convention***	Sr 0.400 (▼) · Mg 20 (▼) · Ca 180 (▼) · Ca:Mg 9 (·) · P 118 (·)

☐ IDENTIFIED ☐ BORDERLINE ☐ NOT IDENTIFIED

NUTRITIONAL COFACTOR NOTES

SYNERGISTS

Sr absorbs and deposits via the same pathways as Ca; restoring Ca/Mg/D pulls Sr along.

Stomach acid solubilizes Sr and Ca; low gastric acidity (PPI, age, atrophic gastritis) contributes.

Vitamin K2: directs Sr (alongside Ca) into bone rather than soft tissue.

ANTAGONISTS

Sr, Ca, and Mg all require gastric acid for solubilization; PPI use depletes the group.

High phosphate intake: binds Sr in the gut; aluminium competes with Sr at bone deposition sites.

Restrictive diets low in dairy, leafy greens, and grains reduce Sr intake.

LITERATURE OBSERVATIONS

PEER-REVIEWED

Lower hair Sr is inversely associated with greater complexity and severity of coronary artery disease, suggesting a potential protective role of adequate Sr in vascular health.

Reduced bone mineral density and structural support: Low Sr is linked to diminished bone mineral density and reduced connective tissue resilience, contributing to patterns of skeletal aging and structural weakness. ***PMID 33745523*** · *Park et al., 2021*

Low Sr reduces the body's capacity to limit aluminium absorption and promote its excretion, potentially increasing the risk of aluminium deposition in the brain and associated neurotoxicity.

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DEMO PATIENT

MOLYBDENUM (Mo) **LOW · OUT OF RANGE**

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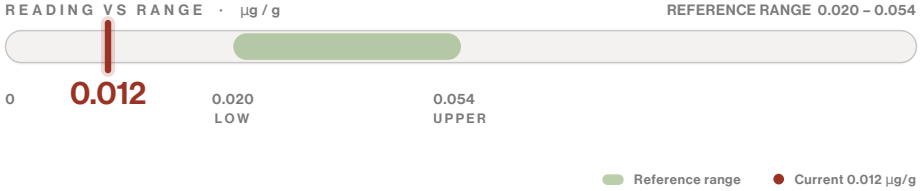
42 95.95
Mo
Molybdenum
Below range

HAIR MINERAL ANALYSIS · ELEMENT REFERENCE

READING VS RANGE · µg / g

REFERENCE RANGE 0.020 – 0.054



SCAN FOR REFERENCED LITERATURE

Molybdenum supports long-term detoxification capacity through sulfite oxidation, aldehyde metabolism, and purine regulation.

In HMA, low Mo has been described less as dietary deficiency and more as a marker of suppression by excess copper, increased detoxification demand, or restrictive diets. Copper dominance has been described as the most clinically important context to evaluate.

SCENARIO REFERENCE

Scenarios described in the published mineral-balancing literature. These are descriptions of the taxonomy, not characterizations of the patient.

RELATES TO	LITERATURE DESCRIPTION	RELATED READINGS
Copper regulation & antagonism	A low molybdenum reading has been associated with sustained copper dominance, since research has shown copper and molybdenum act reciprocally and copper-rich states suppressed molybdenum retention. It is read holistically against an elevated Cu:Mo ratio and a low Zn:Cu rather than as simple dietary lack. ***PMID 2998442***	Mo 0.012 (▼) · Cu 46 (▲) · Cu:Mo 3,833 (▲) · Zn:Cu 1.91 (▼) · Fe:Cu 0.109 (▼)
Sulfur & detox metabolism	Low molybdenum has been linked to reduced reserve for the sulfite-oxidase pathway, where studies of molybdenum cofactor loss showed impaired sulfur amino-acid catabolism and sulfite handling. It is read alongside sulfur and the toxic-metal panel as a possible marker of heavy detoxification demand outpacing supply. ***PMID 39669634***	Mo 0.012 (▼) · S 40,500 (▼) · Hg 2.60 (▲) · Pb 6.40 (▲) · Cd 0.420 (▲)
Dietary intake & status	Reduced molybdenum has been described in the context of low intake of molybdenum-rich whole foods such as legumes, grains, and leafy greens, and balance studies in healthy adults showed intake tracked these protein sources. It is read alongside other trace minerals where a broader insufficiency pattern is suspected. ***PMID 17098584***	Mo 0.012 (▼) · Cr 0.030 (▼) · Mn 0.050 (▼) · Zn 88 (▼) · Se 0.400 (▼)

☐ IDENTIFIED ☐ BORDERLINE ☐ NOT IDENTIFIED

NUTRITIONAL COFACTOR NOTES

SYNERGISTS

Methionine and cysteine supply sulfur for Moco biosynthesis and sulfite oxidase.

Iron pairs with Mo in xanthine and aldehyde oxidase; Zn supports the broader network.

Sustained protein intake: supports albumin and alpha-2-macroglobulin Mo transport.

ANTAGONISTS

Cu and Mo: are reciprocal; Cu forms tetrathiomolybdate complexes that remove Mo.

Mo-dependent enzymes detoxify sulfite, aldehydes, and reactive nitrogen species from toxic metals.

Mo: is concentrated in legumes, whole grains, and leafy greens (often reduced in restrictive diets).

LITERATURE OBSERVATIONS

PEER-REVIEWED

Children with ASD consistently show significantly lower hair Mo than neurotypical controls, part of broader essential trace element imbalances.

Lower hair Mo is associated with worse verbal communication, social interaction, and overall impression scores in children with ASD.

Reduced hair Mo is observed in children with neurodevelopmental conditions, correlating with behavioural difficulties and cognitive issues.

NUTRITIONAL ELEMENTS

LOW MAGNESIUM (Mg)

READING	REFERENCE RANGE
20 µg/g	**30.0–108** µg/g

The most common hair finding â€" usually chronic under-repletion combined with stress-driven drain. Produces the familiar deficiency picture: muscle tension, restless sleep, mild anxiety or irritability, cramping, reduced tolerance for caffeine or alcohol. Sustained sympathetic activation burns through magnesium faster than most dietary patterns replace it. **Na/Mg** separates the two mechanisms: elevated Na/Mg confirms active stress drain; normal Na with low Mg points to dietary gap. Responds well to **glycinate, malate, or threonate** titrated to bowel tolerance; persistent low readings despite repletion warrant checking gut absorption and renal loss.

HIGH SODIUM (Na)

READING	REFERENCE RANGE
520 µg/g	**75.0–435** µg/g

Active adrenal output â€" sympathetic drive is still running, not yet depleted. Salt cravings, early waking, heightened reactivity to minor stressors, and jaw or neck tension through the day fit. This is the acute-to-chronic transition zone â€" still responsive rather than exhausted. The **Na/Mg ratio** is the confirmatory lens: both elevated means the stress axis is actively working; elevated Na alone is usually dietary (processed foods, electrolyte drinks). Responds well to foundational **magnesium + adrenal support** before the pattern transitions into the lower-sodium exhaustion phase that follows sustained strain.

HIGH POTASSIUM (K)

READING	REFERENCE RANGE
165 µg/g	**20.0–132** µg/g

Suggests **early thyroid overactivity or increased cellular turnover** rather than dietary excess â€" hair potassium doesn't track intake linearly. Subtle palpitations on exertion, early waking with a racing mind, unintended weight loss, or looser stools than baseline fit. A **full thyroid panel (TSH, fT3, fT4, antibodies)** is warranted when paired with heat intolerance or cardiovascular symptoms. **Ca/K** is the tighter thyroid marker: low Ca/K amplifies the hypothesis; high Ca/K steers toward turnover from intensive training, injury recovery, or recent illness rather than endocrine activation.

LOW IRON (Fe)

READING	REFERENCE RANGE
5 µg/g	**6.00–22.0** µg/g

Less reliable than serum â€" confirm with **ferritin and CBC** before acting. Ferritin below 30 Âµg/L with a symptom match (fatigue, cold extremities, breathlessness on exertion, brittle nails, reduced exercise tolerance) justifies repletion; in-range ferritin with a low hair reading usually reflects distribution or oxidative-stress consumption rather than depleted stores. Copper status matters â€" low Cu can cause **functional iron deficiency** independent of iron level because copper is required for iron loading onto transferrin. Dietary iron paired with vitamin C for absorption, away from calcium and tannin blockers, is first-line.

Entries shown mildly out of reference range — see bar chart on page 1 for context.

NUTRITIONAL ELEMENTS

HIGH COPPER (Cu)

READING	REFERENCE RANGE
46 µg/g	**8.00–30.0** µg/g

Biounavailable copper accumulating in tissues while the ceruloplasmin-bound functional form may still be inadequate â€” copper excess and functional deficiency can coexist. Anxiety, mood lability, fatigue with cold extremities, and heavy or intensely premenstrual periods in women fit. **Estrogen-dominant states** (oral contraceptives, HRT, perimenopause) and copper plumbing are the common drivers. **Serum ceruloplasmin** with a copper-to-ceruloplasmin ratio calculation settles functional status â€” the raw hair value alone doesn't. Support with **zinc, molybdenum, and vitamin C** to mobilise unbound copper, rather than dietary copper restriction alone.

LOW ZINC (Zn)

READING	REFERENCE RANGE
88 µg/g	**120–225** µg/g

Either **increased demand** (growth, pregnancy, recovery from illness) or **chronic GI loss** from low stomach acid, malabsorption, or stress-driven burn. Slow wound healing, skin changes (acne, eczema flares), reduced immune resilience, or loss of taste/smell acuity fit; in men, fertility-related concerns may accompany. Worth taking seriously even at this level â€” zinc sits at the centre of immune, wound-healing, and hormonal function. Check **Zn/Cu** and GI history. **Picolinate or bisglycinate** forms are well-tolerated; pair with adequate copper to preserve ratio balance.

LOW SELENIUM (Se)

READING	REFERENCE RANGE
0.4 µg/g	**0.600–1.10** µg/g

Reduced **antioxidant and thyroid-conversion reserves** rather than an acute problem. Fatigue, reduced immune resilience, dry or brittle hair and nails, and â€” on labs â€” impaired T4-to-T3 conversion fit. **Brazil nuts (1-2/day)** is usually sufficient dietary support; selenomethionine supplementation when dietary shifts aren't feasible or thyroid antibodies are elevated. Check **Zn/Se and Cu/Se ratios** for antioxidant-system context.

LOW SULFUR (S)

READING	REFERENCE RANGE
40500 µg/g	**41,000–52,000** µg/g

Reduced **glutathione production capacity** or inadequate sulphur-amino-acid intake â€” both clinically significant. Reduced detoxification resilience (chemical sensitivity, slow medication clearance, histamine intolerance) is the clinical tell. Sulphur-rich foods (eggs, cruciferous vegetables, garlic, onions, grass-fed proteins) support dietary repletion. **NAC or glutathione precursors** when clinical signs are present.

Entries shown mildly out of reference range — see bar chart on page 1 for context.

CanAlt Health Laboratories

DEMO PATIENT

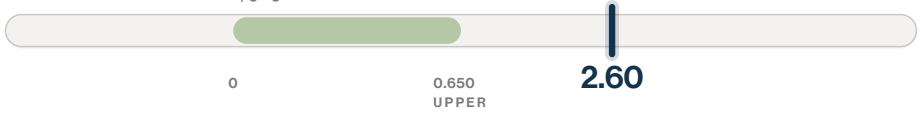
MERCURY (Hg) **HIGH · OUT OF RANGE**

The information on this page is provided to support the practitioner's independent clinical assessment, drawn from the published mineral balancing literature, alongside clinical history, physical assessment, and confirmatory testing where clinically appropriate.

Hg
Mercury
Above range

HAIR MINERAL ANALYSIS · ELEMENT REFERENCE

READING VS RANGE · µg / g



SCAN FOR REFERENCED LITERATURE

Mercury is a nonessential metal with no known biological role, a strong affinity for sulfur- and selenium-dependent systems, and a tendency toward long-term tissue persistence.

In the hair-analysis literature an elevated reading has been described as reflecting longer-term exposure patterns and the ongoing exchange between soft tissues, organ stores, and the growing hair shaft, with stored mercury reported to mobilize into hair during physiological stress, mineral rebalancing, or metabolic adjustment. The reading has been read most meaningfully not in isolation but against the minerals that buffer it (selenium, zinc, sulfur), the Na:K pattern that reflects adaptive capacity, and any co-occurring toxic metals that raise overall elimination demand.

SCENARIO REFERENCE

Scenarios described in the published mineral-balancing literature. These are descriptions of the taxonomy, not characterizations of the patient.

RELATES TO	LITERATURE DESCRIPTION	RELATED READINGS
Exposure & body burden	An elevated mercury reading has been described as reflecting longer-term exposure and the ongoing exchange between soft-tissue and organ stores and the growing hair shaft, with stored mercury reported to mobilize into hair during stress or metabolic adjustment. It has been read holistically against the protective minerals that buffer it and the Na:K pattern that frames adaptive and eliminative capacity, rather than as an isolated value. ***Watts DL, Trace Elements Inc. — HTMA toxic-metal exposure and mobilization convention***	Reduced Buffering Capacity · Hg 2.60 (▲) · Se 0.400 (▼) · Zn 88 (▼) · S 40,500 (▼) · Na:K 3.15 (▲)
Target-organ effects	Sustained mercury exposure has been associated in population studies with neurobehavioral, renal, and cardiovascular findings, with hair mercury used as a long-term exposure biomarker in these cohorts. The reading has been interpreted holistically alongside the protective minerals and co-occurring toxic metals that frame overall burden and elimination demand. ***PMID 9392777***	Hg 2.60 (▲) · Se 0.400 (▼) · Pb 6.40 (▲) · Cd 0.420 (▲) · Ca 180 (▼) · Na:K 3.15 (▲)
Protective & antagonist minerals	Research has linked selenium, zinc, and sulfur-dependent (glutathione) systems with the buffering, sequestration, and conjugation pathways that neutralize and eliminate mercury, so the same burden has been reported to carry greater biological impact when these elements read low. The mercury reading has been weighed holistically against these antagonist minerals rather than against its own value alone. ***PMID 20561558***	Hg 2.60 (▲) · Se 0.400 (▼) · Zn 88 (▼) · S 40,500 (▼) · Hg:Se 6.50 · Zn:Cu 1.91 (▼)

☐ IDENTIFIED ☐ BORDERLINE ☐ NOT IDENTIFIED

NUTRITIONAL COFACTOR NOTES

SYNERGISTS

Selenium: has been described as central to mercury buffering and antioxidant defense, forming biologically inert mercury-selenide complexes; lower selenium has been reported to reduce neutralization capacity.

Zinc: has been described as competing with mercury at enzymatic binding sites and supporting metallothionein-mediated sequestration, immune regulation, and neurological stability.

Sulfur-containing (thiol) compounds such as glutathione have been described as central to mercury binding and to the conjugation pathways involved in its elimination.

Adequate adaptive mineral status, reflected in a balanced Na:K pattern, has been associated in the mineral-balancing literature with greater capacity for endogenous toxic-metal handling. Wilson L, Nutritional Balancing 1998

LITERATURE OBSERVATIONS

PEER-REVIEWED

Chronic mercury exposure, particularly methylmercury, has been strongly associated with cognitive deficits, memory complaints, tremor, and neurobehavioral impairment in population studies that used hair as a long-term exposure biomarker.

Sustained mercury exposure has been linked to elevated blood pressure, endothelial dysfunction, and increased cardiovascular disease risk in dose-response meta-analyses, with hair mercury reported as a tracking exposure marker.

Long-term mercury exposure has been associated with renal tubular dysfunction, proteinuria, and chronic kidney disease risk in exposed populations.

Chronic mercury exposure has been associated with altered thyroid hormone levels and endocrine disruption, described in the literature as proceeding primarily through selenium displacement.

Prenatal and early-life mercury exposure has been linked to neurodevelopmental variability, lower cognitive scores, and behavioral findings in epidemiological studies of maternal hair mercury.

CanAlt Health Laboratories

DEMO PATIENT

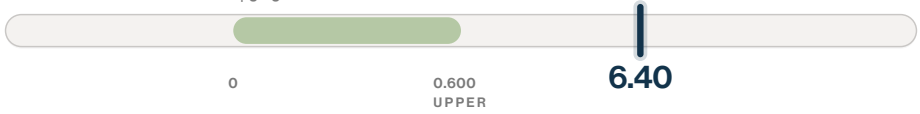
LEAD (Pb) **HIGH · OUT OF RANGE**

The information on this page is provided to support the practitioner's independent clinical assessment, drawn from the published mineral balancing literature, alongside clinical history, physical assessment, and confirmatory testing where clinically appropriate.

Pb
Lead
Above range

HAIR MINERAL ANALYSIS · ELEMENT REFERENCE

READING VS RANGE · µg / g



SCAN FOR REFERENCED LITERATURE

Lead is a toxic metal with no known biological role in human physiology and a strong affinity for bone, giving it long-term biological persistence.

In hair mineral analysis, an elevated lead reading has been described as reflecting longer-term exposure patterns and the ongoing exchange between skeletal stores, soft tissues, and the growing hair shaft. Through competition with essential minerals such as calcium, iron, zinc, and magnesium, lead has been reported to disrupt enzyme activity, energy metabolism, antioxidant defences, and mineral regulation even when individual mineral values fall within reference ranges. Elevated hair lead is therefore read in the context of mineral interactions, skeletal storage dynamics, and the broader metabolic patterns reflected across the profile rather than as an isolated value.

SCENARIO REFERENCE

Scenarios described in the published mineral-balancing literature. These are descriptions of the taxonomy, not characterizations of the patient.

RELATES TO	LITERATURE DESCRIPTION	RELATED READINGS
Exposure & skeletal body burden	An elevated lead reading has been associated with longer-term exposure and an accumulated body burden, since lead is laid down in bone and exchanged slowly with soft tissues and the growing hair shaft. Research has linked this skeletal store to ongoing endogenous re-release during physiological stress, pregnancy, lactation, and bone turnover, so the hair value has been read against the calcium-storage minerals rather than as a single point. ***PMID 28811684***	Pb 6.40 (▲) · Ca 180 (▼) · Ca:Pb 28.1 (▼) · Ca:Mg 9 (·) · Sr 0.400 (▼) · Ca:Sr 450 (·)
Target-organ effects & heme/ erythropoiesis	Elevated lead has been associated with disruption of heme synthesis and erythropoiesis, with studies reporting that it inhibits the zinc-dependent enzyme delta-aminolevulinic acid dehydratase and interferes with iron incorporation into haemoglobin. Research has linked this pathway to lead's broader competition with iron and zinc, so the reading has been framed alongside those antagonists and the iron-utilisation indicators. ***PMID 22864587***	Pb 6.40 (▲) · Fe 5 (▼) · Zn 88 (▼) · Zn:Fe 17.6 (·) · Fe:Cu 0.109 (▼) · S 40,500 (▼)
Protective & antagonist minerals	Studies suggested that adequate calcium, iron, and zinc status has been associated with reduced lead absorption, retention, and biological impact, with dietary calcium in particular linked to lower circulating lead during pregnancy and lactation. In hair-analysis practice the lead reading has therefore been weighed against the buffering minerals and their ratios rather than interpreted in isolation. ***PMID 17296490***	Pb 6.40 (▲) · Ca:Pb 28.1 (▼) · Ca 180 (▼) · Zn 88 (▼) · Zn:Pb 13.8 · Fe 5 (▼) · Mg 20 (▼)

☐ IDENTIFIED ☐ BORDERLINE ☐ NOT IDENTIFIED

NUTRITIONAL COFACTOR NOTES

SYNERGISTS

Calcium reflects skeletal buffering capacity and the bone-related storage dynamics that govern how lead is retained and released.

Iron: shares absorption pathways with lead, influencing its tissue distribution and oxygen-handling; adequacy has been associated with reduced lead uptake.

Zinc: competes with lead at enzymatic binding sites and is often observed reduced where lead burden is present.

Magnesium: supports neuromuscular and energy-related stability affected by mineral displacement.

Sulphur: is relevant to lead's affinity for sulphur-based binding and elimination pathways.

ANTAGONISTS

LITERATURE OBSERVATIONS

PEER-REVIEWED

Studies using hair and blood biomarkers have reported associations between chronic lead exposure and reduced IQ, attention deficits, and developmental variability in children.

Cohort and dose-response research has linked sustained lead exposure to elevated blood pressure, hypertension, and higher cardiovascular disease risk.

Population cohorts have associated chronic lead burden with renal tubular dysfunction, proteinuria, and a higher risk of chronic kidney disease.

Long-term exposure has been linked to reduced bone mineral density and increased fracture risk, through interference with calcium metabolism.

Lead exposure has been associated with impaired heme synthesis, anemia, and altered oxygen transport through disrupted iron utilisation.

CanAlt Health Laboratories

DEMO PATIENT

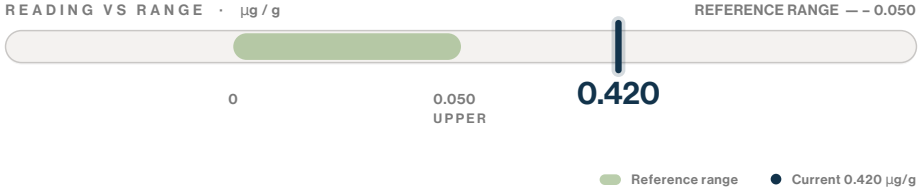
CADMIUM (Cd) **HIGH · OUT OF RANGE**

The information on this page is provided to support the practitioner's independent clinical assessment, drawn from the published mineral balancing literature, alongside clinical history, physical assessment, and confirmatory testing where clinically appropriate.

Cd
Cadmium
Above range

HAIR MINERAL ANALYSIS · ELEMENT REFERENCE

READING VS RANGE · µg / g



SCAN FOR REFERENCED LITERATURE

Cadmium is a toxic metal with no known biological role in human physiology.

In HMA, an elevated reading has been described as reflecting cumulative exposure and tissue retention, with hair levels representing longer-term environmental contact given cadmium's slow accumulation and prolonged biological half-life. The literature has reported that cadmium competes with essential minerals including zinc, iron, calcium, and copper, interfering with enzyme activity, antioxidant defenses, and mineral regulation even when individual mineral values fall within reference ranges; an elevated reading is therefore read alongside those mineral relationships, metabolic pacing, and any co-occurring toxic elements rather than as an isolated value.

SCENARIO REFERENCE

Scenarios described in the published mineral-balancing literature. These are descriptions of the taxonomy, not characterizations of the patient.

RELATES TO	LITERATURE DESCRIPTION	RELATED READINGS
Exposure & body burden	An elevated cadmium reading has been associated with cumulative environmental exposure and tissue retention, with hair levels reflecting longer-term contact given cadmium's slow accumulation and prolonged biological half-life. It is read holistically alongside co-occurring toxic elements and the stress pacing of the Na:K ratio rather than as an isolated value. ***PMID 19409405***	Cd 0.420 (▲) · Pb 6.40 (▲) · As 0.610 (▲) · Na:K 3.15 (▲) · Zn:Cd 210
Target-organ effects	Sustained cadmium burden has been linked in epidemiological research to renal tubular dysfunction, reduced bone mineral density, and cardiovascular and respiratory strain, the organs where retained cadmium has been observed to concentrate. The reading is framed holistically against calcium status and the Ca:P structural ratio rather than read in isolation. ***PMID 16759980***	Cd 0.420 (▲) · Ca 180 (▼) · Ca:P 1.53 (▼) · Zn 88 (▼) · Ca:Cd 429 (▼)
Protective & antagonist minerals	Research has linked adequate zinc, iron, and selenium to lower cadmium retention, since zinc competes at enzyme binding sites and supports metallothionein, iron shares intestinal transport with cadmium, and selenium aids thiol-based antioxidant defense. An elevated reading is therefore read against these protective minerals and the Zn:Cd and Cd:Fe ratios rather than alone. ***PMID 33572579***	Zn 88 (▼) · Fe 5 (▼) · Se 0.400 (▼) · Cu 46 (▲) · Cd 0.420 (▲) · Zn:Cd 210 · Cd:Fe 0.084

☐ IDENTIFIED ☐ BORDERLINE ☐ NOT IDENTIFIED

NUTRITIONAL COFACTOR NOTES

SYNERGISTS

Zinc: competes with cadmium at enzyme binding sites and supports metallothionein activity; adequate zinc has been associated with reduced cadmium retention.

Iron status: influences cadmium absorption through shared intestinal transport (DMT1); iron sufficiency has been associated with lower cadmium uptake.

Selenium and sulfur-dependent pathways support antioxidant defense and thiol-based binding relevant to cadmium handling.

Calcium sufficiency and vitamin-D-related regulation have been linked to skeletal buffering that moderates cadmium's competition at structural sites.

ANTAGONISTS

Tobacco smoke (active and second-hand) has been described as a dominant non-occupational cadmium exposure route.

Low dietary iron and zinc have been associated with increased intestinal cadmium absorption.

LITERATURE OBSERVATIONS

PEER-REVIEWED

Chronic cadmium exposure has been strongly associated with renal tubular dysfunction, proteinuria, and increased risk of chronic kidney disease in large population and occupational studies.

Sustained cadmium exposure has been linked to decreased bone mineral density, osteoporosis patterns, and higher fracture risk in epidemiological research.

Meta-analyses and cohort studies have observed associations between cadmium body measures and elevated blood pressure, hypertension, and increased cardiovascular disease risk.

Chronic cadmium exposure has been associated with reduced lung function and emphysema-like changes, particularly in smoking-related and occupational contexts.

Long-term cadmium exposure has been associated with increased lung cancer risk in occupational cohorts; cadmium has been classified as a Group 1 human carcinogen in international reviews.

CanAlt Health Laboratories

DEMO PATIENT

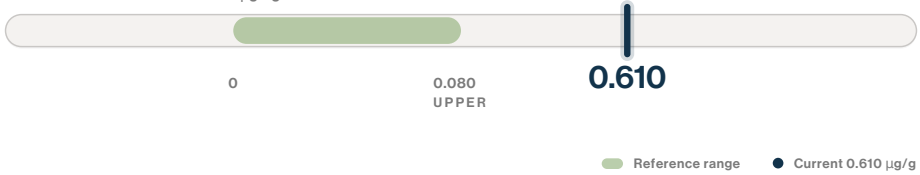
ARSENIC (As) **HIGH · OUT OF RANGE**

The information on this page is provided to support the practitioner's independent clinical assessment, drawn from the published mineral balancing literature, alongside clinical history, physical assessment, and confirmatory testing where clinically appropriate.

As
Arsenic
Above range

HAIR MINERAL ANALYSIS · ELEMENT REFERENCE

READING VS RANGE · µg / g



SCAN FOR REFERENCED LITERATURE

Arsenic is a toxic metalloid with no known biological role in human physiology and a strong affinity for sulfur-containing proteins that has been associated with longer-term biological persistence.

In hair mineral analysis, an elevated reading has been described as reflecting longer-term exposure patterns and the exchange between environmental intake, tissue retention, and incorporation into the growing hair shaft. The literature has reported that arsenic competes with minerals such as selenium, zinc, and sulfur and may be mobilized from tissue stores during periods of metabolic or mineral rebalancing, so the reading is read alongside those mineral relationships and the broader metabolic pattern rather than as an isolated value.

SCENARIO REFERENCE

Scenarios described in the published mineral-balancing literature. These are descriptions of the taxonomy, not characterizations of the patient.

RELATES TO	LITERATURE DESCRIPTION	RELATED READINGS
Exposure & body burden	An elevated arsenic reading has been associated with longer-term intake from groundwater, rice, and seafood and with incorporation of body burden into the growing hair shaft. It is read holistically alongside the other toxic elements to distinguish an isolated arsenic exposure from a broader environmental burden carried together. ***PMID 32650499***	As 0.610 (▲) · Pb 6.40 (▲) · Cd 0.420 (▲) · Hg 2.60 (▲) · U 0.310 (▲) · Se:As 0.656
Target-organ effects	Chronic arsenic exposure has been associated with skin, vascular, and neurocognitive changes in epidemiological research, and hair arsenic has shown correlation with dermatological manifestations in exposed populations. The reading is framed against co-occurring toxic metals and the protective minerals rather than interpreted on its own. ***PMID 20214082***	As 0.610 (▲) · Se 0.400 (▼) · Zn 88 (▼) · Pb 6.40 (▲) · Hg 2.60 (▲) · Cd 0.420 (▲)
Protective & antagonist minerals	Lower availability of selenium, zinc, and sulfur has been associated with greater arsenic retention, since selenoproteins, metallothionein, and sulfhydryl-based conjugation are the pathways through which arsenic has been reported to be bound and eliminated. The reading is interpreted against these antagonist minerals and their ratios to arsenic. ***PMID 29058732***	As 0.610 (▲) · Se 0.400 (▼) · Zn 88 (▼) · S 40,500 (▼) · Se:As 0.656 · Zn:As 144

☐ IDENTIFIED ☐ BORDERLINE ☐ NOT IDENTIFIED

NUTRITIONAL COFACTOR NOTES

SYNERGISTS

Selenium: has been described as central to arsenic binding and regulation through selenoproteins and antioxidant enzymes; lower selenium availability has been frequently associated with greater arsenic retention.

Zinc: has been reported to compete with arsenic at enzymatic and cellular binding sites and to support metallothionein activity involved in toxic-metal regulation.

Sulfur: has been described as relevant because of arsenic's strong affinity for sulfhydryl groups and its reliance on sulfur-based pathways for conjugation and elimination.

ANTAGONISTS

Groundwater and private wells containing naturally occurring inorganic arsenic have been described as a primary source of longer-term exposure.

Dietary intake from rice, seafood, and seaweed has been reported to contribute both organic and inorganic arsenic compounds.

Occupational and industrial activity — mining, smelting, glass production, metal processing, and the historical use of arsenic-based pesticides, herbicides, and wood preservatives — has been associated with elevated exposure.

LITERATURE OBSERVATIONS

PEER-REVIEWED

Population studies and meta-analyses have observed associations between chronic arsenic exposure and impaired glucose regulation, with an increased risk of type 2 diabetes reported in exposed populations.

Epidemiological research and dose-response meta-analyses have linked chronic arsenic exposure to elevated blood pressure and increased cardiovascular disease risk.

Chronic arsenic exposure has been associated with characteristic skin changes, including hyperpigmentation and hyperkeratosis, and hair arsenic levels have shown correlation with these skin manifestations in exposed populations.

Environmental arsenic exposure has been associated with cognitive and neurodevelopmental variability, particularly in children and during early-life stages.

Studies have reported associations between sustained arsenic exposure and markers of kidney-function decline, including an increased risk of chronic kidney disease.

CanAlt Health Laboratories

DEMO PATIENT

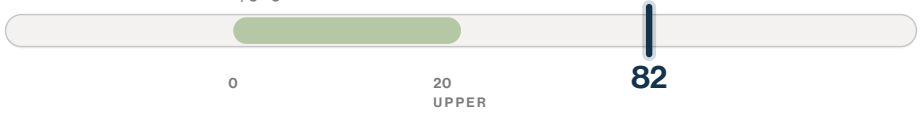
ALUMINUM (Al) **HIGH · OUT OF RANGE**

The information on this page is provided to support the practitioner's independent clinical assessment, drawn from the published mineral balancing literature, alongside clinical history, physical assessment, and confirmatory testing where clinically appropriate.

Al
Aluminum
Above range

HAIR MINERAL ANALYSIS · ELEMENT REFERENCE

READING VS RANGE · µg / g



SCAN FOR REFERENCED LITERATURE

Aluminum is a toxic environmental metal with no known biological role in human physiology.

In HMA, an elevated aluminum reading has been described as reflecting a sustained exposure and elimination pattern interacting with mineral regulation, often where the capacity to bind, buffer, and clear aluminum over time appears reduced. The literature has reported that aluminum competes with calcium, magnesium, phosphorus, iron, and zinc, and that this competition can place strain on enzyme activity, energy metabolism, and oxidative balance even when individual essential minerals fall within reference ranges. The reading is read alongside those competing minerals and co-occurring toxic metals rather than as an isolated value.

SCENARIO REFERENCE

Scenarios described in the published mineral-balancing literature. These are descriptions of the taxonomy, not characterizations of the patient.

RELATES TO	LITERATURE DESCRIPTION	RELATED READINGS
Exposure & body burden	An elevated aluminum reading has been associated with a sustained exposure-and-elimination pattern, with higher tissue burden more commonly observed in occupational and urban populations exposed to welding, smelting, airborne dust, cookware, antacids, and aluminum-treated water. It is read holistically as a marker of cumulative load alongside co-occurring toxic metals rather than as a single isolated exposure. ***PMID 16280209***	Al 82 (▲) · Pb 6.40 (▲) · Cd 0.420 (▲) · Ni 2.60 (▲) · S 40,500 (▼)
Neurocognitive target-organ effects	Research has linked higher aluminum exposure to neurological and cognitive strain, with meta-analytic data reporting reduced processing speed, working memory, and attention in exposed cohorts. The reading is framed holistically against magnesium and calcium, the competing minerals whose displacement at neural and skeletal sites has been described as part of this pattern. ***PMID 37777128***	Al 82 (▲) · Mg 20 (▼) · Ca 180 (▼) · Ca:Mg 9 (·) · P 118 (·) · Ca:P 1.53 (▼)
Oxidative stress & antagonist minerals	Studies have reported elevated oxidative-stress and DNA-damage biomarkers in aluminum-exposed workers, a pattern described as placing demand on antioxidant defence where zinc, selenium, and magnesium serve as protective antagonists competing with aluminum at enzymatic binding sites. The reading is interpreted holistically against those minerals and the iron-handling balance. ***PMID 29239217***	Al 82 (▲) · Zn 88 (▼) · Se 0.400 (▼) · Mg 20 (▼) · Fe 5 (▼) · Zn:Cu 1.91 (▼) · Cu:Se 115 (▲)

☐ IDENTIFIED ☐ BORDERLINE ☐ NOT IDENTIFIED

NUTRITIONAL COFACTOR NOTES

SYNERGISTS

Magnesium: has been described as protective against aluminum retention; low magnesium has been reported to increase susceptibility to aluminum accumulation and impaired energy metabolism.

Calcium reflects aluminum competition at bone and cellular sites, influencing skeletal buffering and neuromuscular regulation.

Zinc: competes with aluminum at enzymatic binding sites and supports antioxidant and immune regulation.

Gastrointestinal elimination efficiency and hydration status have been described as relevant to long-term aluminum clearance.

ANTAGONISTS

Dietary exposure: has been reported from aluminum cookware, foil, cans, baking trays, baking powders, processed baked goods, and processed cheese.

LITERATURE OBSERVATIONS

PEER-REVIEWED

Meta-analyses of human occupational cohorts have reported associations between aluminum exposure and neurological and cognitive strain, including measurable reductions in processing speed, working memory, attention, and reaction time among exposed workers compared with controls.

Large-scale nationwide epidemiological data-linkage studies in general populations have linked higher exposure to airborne aluminum, such as in particulate matter, to poorer sleep quality, including difficulties with sleep maintenance and overall sleep disturbance.

Longitudinal cohort studies of aluminum workers have reported that chronic occupational exposure correlated with elevated blood pressure and an increased risk of developing hypertension over time.

Occupational studies have reported elevated biomarkers of oxidative stress and DNA damage in aluminum-exposed workers, a pattern described as contributing to systemic regulatory strain on vascular, renal, and hepatic processes.

Elevated aluminum body burden, measured in urine, serum, or tissue, has more commonly been observed in industrial, occupational, and urban populations with greater airborne or workplace exposure sources, including welding and smelting settings.

CanAlt Health Laboratories

DEMO PATIENT

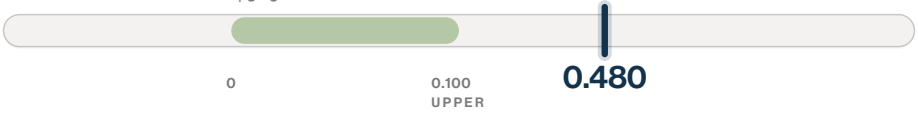
ANTIMONY (Sb) **HIGH · OUT OF RANGE**

The information on this page is provided to support the practitioner's independent clinical assessment, drawn from the published mineral balancing literature, alongside clinical history, physical assessment, and confirmatory testing where clinically appropriate.

Sb
Antimony
Above range

HAIR MINERAL ANALYSIS · ELEMENT REFERENCE

READING VS RANGE · µg / g



SCAN FOR REFERENCED LITERATURE

Antimony is a metalloid with no known biological role, commonly encountered through industrial processes, manufactured materials, and certain consumer products.

In hair mineral analysis an elevated reading has been interpreted in terms of how this element has interacted with tissue-level mineral balance and elimination pathways over time rather than as a measure of total body burden. Antimony has been described as competing with minerals such as zinc, selenium, magnesium, and sulfur-containing compounds, which has been associated with added demand on the systems responsible for maintaining mineral balance and supporting natural elimination.

SCENARIO REFERENCE

Scenarios described in the published mineral-balancing literature. These are descriptions of the taxonomy, not characterizations of the patient.

RELATES TO	LITERATURE DESCRIPTION	RELATED READINGS
Exposure & body burden	An elevated antimony reading has been associated with cumulative environmental and occupational contact reported in the biomonitoring literature, where smelting, metal processing, solder and battery work, and flame-retardant materials such as antimony trioxide were described as exposure routes. It has been read holistically alongside the sulfur-handling and selenium pathways involved in metal binding and elimination rather than as a measure of total body burden. *PMID 16307078*	Sb 0.480 (▲) · S 40,500 (▼) · Se 0.400 (▼) · Zn 88 (▼) · Cu:Se 115 (▲)
Target-organ & cardiovascular strain	Higher antimony exposure has been linked in population studies with elevated blood pressure and increased hypertension risk, and occupational research has associated inhaled antimony with respiratory and oxidative-stress effects. The reading has been considered holistically against the calcium-magnesium buffering minerals and the sodium-to-potassium pacing relationship that frame vascular and adaptive tone. *PMID 24945898*	Sb 0.480 (▲) · Na:K 3.15 (▲) · Ca:Mg 9 (·) · Mg 20 (▼) · Ca 180 (▼)
Protective & antagonist minerals	Studies of essential and toxic elements in the hair of occupationally exposed workers have described antimony in relation to the protective minerals that compete with it at binding sites and support antioxidant defense, notably selenium, zinc, and sulfur-containing compounds. The reading has been read holistically against these antagonists and the copper-selenium balance reflecting antioxidant reserve. *PMID 30456579*	Sb 0.480 (▲) · Se 0.400 (▼) · Zn 88 (▼) · S 40,500 (▼) · Cu:Se 115 (▲) · Zn:Cu 1.91 (▼)

☐ IDENTIFIED ☐ BORDERLINE ☐ NOT IDENTIFIED

NUTRITIONAL COFACTOR NOTES

SYNERGISTS

Zinc: has been described as competing with antimony at enzyme and binding sites and as supporting antioxidant balance. Wilson L, Nutritional Balancing and Hair Mineral Analysis 1998

Selenium: has been described as relevant to neutralizing oxidative stress and supporting sulfur-related pathways involved in metal handling. Wilson L, Nutritional Balancing and Hair Mineral Analysis 1998

Sulfur-containing compounds: have been described as important for the binding and elimination pathways involved in handling metals such as antimony. Watts DL, Trace Elements and Other Essential Nutrients

Magnesium: has been described as supporting energy metabolism and neuromuscular stability that may be affected by mineral displacement. Wilson L, Nutritional Balancing and Hair Mineral Analysis 1998

LITERATURE OBSERVATIONS

PEER-REVIEWED

- Population studies have observed associations between higher urinary antimony and elevated blood pressure or increased hypertension risk.
- Higher urinary antimony has been linked in population data to insufficient sleep, longer sleep latency, and possible obstructive sleep apnea symptoms.
- Occupational inhalation exposure has been associated with respiratory irritation, chronic bronchitis-like symptoms, and pneumoconiosis-like changes in exposed workers.
- Occupational studies have reported elevated oxidative stress and DNA damage biomarkers in antimony-exposed workers.
- Hair antimony levels have correlated strongly with urine (r = 0.870) and blood (r = 0.713) in occupational research, supporting hair mineral analysis for assessing longer-term exposure and related regulatory patterns.

CanAlt Health Laboratories

DEMO PATIENT

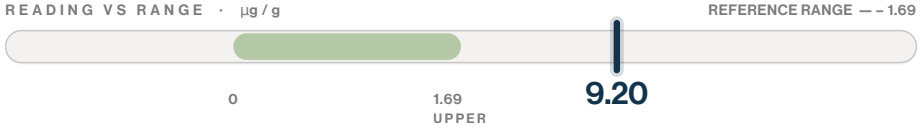
BARIUM (Ba) **HIGH · OUT OF RANGE**

The information on this page is provided to support the practitioner's independent clinical assessment, drawn from the published mineral balancing literature, alongside clinical history, physical assessment, and confirmatory testing where clinically appropriate.

Ba
Barium
Above range

HAIR MINERAL ANALYSIS · ELEMENT REFERENCE

READING VS RANGE · µg / g



SCAN FOR REFERENCED LITERATURE

Barium is a non-essential alkaline-earth metal with no known biological role in human physiology, and elevated hair barium has been read in HMA as a marker of environmental or occupational exposure and the body's longer-term handling of that load rather than as total body burden.

The literature has described barium as competing with calcium, potassium, and magnesium — the minerals central to nerve conduction, muscle contraction, and cardiac rhythm — and as acting on potassium channels in ways that have been observed to influence cellular membrane potentials and metabolic pacing even when individual mineral values fell within reference ranges. Hair barium has been interpreted most meaningfully alongside the alkaline-earth group, electrolyte ratios, and other toxic elements, since barium may be incorporated into hair during periods of metabolic adjustment or mineral rebalancing.

SCENARIO REFERENCE

Scenarios described in the published mineral-balancing literature. These are descriptions of the taxonomy, not characterizations of the patient.

RELATES TO	LITERATURE DESCRIPTION	RELATED READINGS
Exposure & body burden	An elevated barium reading has been associated with sustained environmental, water-source, or occupational intake of this non-essential alkaline-earth metal, with biomonitoring studies treating hair and tissue barium as a marker of longer-term handling of that load rather than total body burden. It has been read holistically alongside the alkaline-earth group and co-occurring toxic metals, since the regulatory and oxidative demand of a mixed metal load has been described as shared across elimination pathways. ***PMID 36402181***	Ba 9.20 (▲) · Sr 0.400 (▼) · Ca:Sr 450 (·) · Ca:Pb 28.1 (▼) · Ca:Cd 429 (▼) · Pb 6.40 (▲)
Cardiac & neuromuscular targets	Because soluble barium has been observed to act on potassium channels and drive potassium into cells, an elevated reading has been linked in case and occupational series with hypokalemia, flaccid muscle weakness, and cardiac arrhythmias. It has been read holistically against potassium and the electrolyte ratios, which frame the membrane-potential and metabolic-pacing strain barium has been described as imposing. ***PMID 40729295***	Ba 9.20 (▲) · K 165 (▲) · Na:K 3.15 (▲) · Mg 20 (▼) · Ca 180 (▼) · Ca:Mg 9 (·)
Protective & antagonist minerals	In hair-analysis practice barium has been read against the minerals it competes with, where calcium and magnesium have been described as buffering the alkaline-earth load through the Ca:Sr and Ca:Mg relationships and where sulfur-based pathways have been noted to participate in endogenous binding and elimination. Adequate potassium and these antagonist minerals have been interpreted holistically as the framework against which tolerance to barium is gauged. ***Watts DL, Trace Elements Inc. — HTMA alkaline-earth antagonist and buffering convention***	Ca 180 (▼) · Mg 20 (▼) · Ca:Mg 9 (·) · Ca:Sr 450 (·) · S 40,500 (▼) · K 165 (▲)

☐ IDENTIFIED ☐ BORDERLINE ☐ NOT IDENTIFIED

NUTRITIONAL COFACTOR NOTES

SYNERGISTS

Calcium: has been described as competing with barium in neuromuscular signalling and tissue distribution, with the Ca:Ba relationship reflecting buffering against the alkaline-earth load.

Magnesium: has been noted to support neuromuscular stability and energy metabolism under electrolyte stress where barium interferes with calcium- and potassium-dependent processes.

Potassium status: has been read alongside barium because barium has been observed to act on potassium channels and alter cellular electrical activity.

Sulfur-based pathways: have been described as participating in the endogenous binding and elimination of barium and other metals.

LITERATURE OBSERVATIONS

PEER-REVIEWED

Acute or high-level exposure to soluble barium has been associated with cardiac arrhythmias, blood pressure changes, and ECG abnormalities in case reports and occupational-exposure series.

High-level barium exposure has been linked to muscle weakness, paresthesia, and flaccid paralysis, frequently described alongside hypokalemia in documented cases.

Ingestion of soluble barium compounds has been associated with severe vomiting, abdominal pain, diarrhea, and gastrointestinal irritation in acute exposure incidents.

Case reports of significant barium exposure have noted acute kidney injury or renal insufficiency in severe intoxication scenarios.

Chronic or environmental barium exposure has been observed in association with potential strain on metabolic, neurological, and stress-response systems in populations near industrial sources, though evidence for low-level chronic effects has been described as limited.

CanAlt Health Laboratories

DEMO PATIENT

URANIUM (U) **HIGH · OUT OF RANGE**

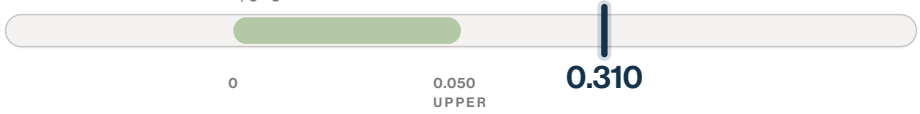
The information on this page is provided to support the practitioner's independent clinical assessment, drawn from the published mineral balancing literature, alongside clinical history, physical assessment, and confirmatory testing where clinically appropriate.

U
Uranium
Above range

HAIR MINERAL ANALYSIS · ELEMENT REFERENCE

READING VS RANGE · µg / g

REFERENCE RANGE — – 0.050



SCAN FOR REFERENCED LITERATURE

Uranium is a toxic heavy metal with no known biological role in human physiology, encountered through uranium-rich groundwater, agricultural inputs, and occupational or industrial activity.

Once retained, it has been observed to show affinity for bone and mineralized tissues, where it interacts with calcium and phosphorus and influences skeletal storage dynamics. By competing with essential minerals such as calcium, magnesium, zinc, and selenium, retained uranium has been described as disrupting enzyme activity, mitochondrial energy production, and antioxidant regulation, so an elevated hair reading is interpreted alongside related mineral patterns rather than as an isolated value.

SCENARIO REFERENCE

Scenarios described in the published mineral-balancing literature. These are descriptions of the taxonomy, not characterizations of the patient.

RELATES TO	LITERATURE DESCRIPTION	RELATED READINGS
Exposure & body burden	An elevated uranium reading has been associated with retained body burden from uranium-rich groundwater, agricultural inputs, or occupational exposure, with chronic ingestion studies linking accumulation to renal tubular strain as the principal target-organ signal. It is read holistically against the other toxic elements and the pacing ratios that frame elimination capacity rather than as an isolated value. ***PMID 9629621***	Reduced Buffering Capacity · U 0.310 (▲) · Ca:Mg 9 (·) · Na:K 3.15 (▲) · Mg 20 (▼) · K 165 (▲)
Skeletal storage & bone	Uranium has been observed to show affinity for bone and mineralized tissue, where it interacts with calcium and phosphorus, and sustained exposure has been linked to altered skeletal mineral handling and reduced bone density. The reading is framed against calcium, phosphorus, and the Ca:P relationship so that retained uranium is read as exposure overlaid on changing skeletal buffering rather than a fixed value. ***PMID 15626650***	U 0.310 (▲) · Ca 180 (▼) · P 118 (·) · Ca:P 1.53 (▼) · Ca:Sr 450 (·) · Mg 20 (▼)
Protective & antagonist minerals	In hair-analysis practice a high uranium reading has been read alongside the essential minerals that compete with retained metals at enzyme and antioxidant sites, where displacement of magnesium, zinc, and selenium has been described as raising metabolic and antioxidant demand. Calcium, magnesium, zinc, and selenium status frames the body's endogenous handling of the burden rather than the uranium value alone. ***Watts DL, Trace Elements Inc. — HTMA toxic-metal antagonist-mineral convention***	U 0.310 (▲) · Mg 20 (▼) · Zn 88 (▼) · Se 0.400 (▼) · Ca 180 (▼) · Zn:Cu 1.91 (▼)

☐ IDENTIFIED ☐ BORDERLINE ☐ NOT IDENTIFIED

NUTRITIONAL COFACTOR NOTES

SYNERGISTS

Calcium reflects skeletal storage dynamics and buffering capacity relevant to where retained uranium has been observed to deposit and release.

Phosphorus interacts with uranium in bone and mineralized tissue and has been described as influencing structural mineral balance.

Magnesium: supports energy metabolism and neuromuscular stability that mineral displacement has been reported to affect.

Antioxidant capacity and balanced calcium, magnesium, and phosphorus status have been associated with the body's endogenous handling of retained metals.

Adequate hydration and regular renal and gastrointestinal clearance have been associated with supporting physiological excretion of retained uranium.

LITERATURE OBSERVATIONS

PEER-REVIEWED

Chronic uranium exposure has been strongly associated with renal tubular dysfunction, proteinuria, and increased risk of chronic kidney disease in population and occupational studies.

Sustained exposure has been linked to altered calcium and phosphorus handling, reduced bone mineral density, and skeletal mineral imbalance in long-term exposure contexts.

Epidemiological research has observed associations between uranium exposure and elevated blood pressure or hypertension risk in general population cohorts.

Chronic exposure has been associated with cognitive variability, neurological effects, and potential central nervous system strain in environmental and occupational populations.

Long-term exposure has been linked to endocrine disruption and reproductive regulatory effects in population-based studies.

CanAlt Health Laboratories

DEMO PATIENT

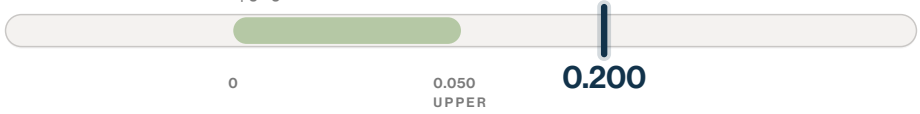
THALLIUM (TI) **HIGH · OUT OF RANGE**

The information on this page is provided to support the practitioner's independent clinical assessment, drawn from the published mineral balancing literature, alongside clinical history, physical assessment, and confirmatory testing where clinically appropriate.

Tl
Thallium
Above range

HAIR MINERAL ANALYSIS · ELEMENT REFERENCE

READING VS RANGE · µg / g



SCAN FOR REFERENCED LITERATURE

Thallium is an ultra-trace element that is not required for normal human biochemistry and is most often encountered through environmental and industrial exposure.

Because of its chemical similarity to potassium, thallium has been observed to follow potassium-dependent transport systems and enzymes. In HMA, an elevated thallium reading has been described as reflecting longer-term exposure patterns — integrating environmental input with tissue retention and elimination capacity across the period of hair growth — so it is read most meaningfully in relation to potassium dynamics and metabolic pacing rather than as an isolated exposure marker.

SCENARIO REFERENCE

Scenarios described in the published mineral-balancing literature. These are descriptions of the taxonomy, not characterizations of the patient.

RELATES TO	LITERATURE DESCRIPTION	RELATED READINGS
Exposure & body burden	An elevated thallium reading has been associated with longer-term environmental or industrial exposure, integrating ongoing input with tissue retention across the period of hair growth. In population research thallium burden has been linked with combustion, metal-refining, and tobacco-particulate sources, so it has been read holistically against co-occurring toxic metals and overall elimination demand rather than as an isolated value. ***PMID 39211414***	Tl 0.200 (▲) · K 165 (▲) · Na:K 3.15 (▲) · Ca:Pb 28.1 (▼) · S 40,500 (▼)
Target-organ effects	Sustained thallium exposure has been associated with peripheral neuropathy, alopecia and nail change, and renal tubular dysfunction, described in the literature as arising from interference with potassium-dependent nerve conduction and energy processes. The reading has been framed against potassium dynamics and metabolic pacing that mark the systems most challenged by an elevated body burden. ***PMID 9684026***	Tl 0.200 (▲) · K 165 (▲) · Na:K 3.15 (▲) · Mg 20 (▼) · S 40,500 (▼)
Protective & antagonist minerals	Because thallium follows potassium transport pathways and has been observed to drive oxidative and excitotoxic stress in experimental models, its handling has been read against potassium status and the antioxidant minerals selenium, zinc, and magnesium that support enzymatic stability and defence. Studies suggested that antioxidant agents tempered thallium toxicity, so these minerals frame the antagonist context of an elevated reading. ***PMID 29313218***	Tl 0.200 (▲) · K 165 (▲) · Se 0.400 (▼) · Zn 88 (▼) · Mg 20 (▼) · Cu:Se 115 (▲)

☐ IDENTIFIED ☐ BORDERLINE ☐ NOT IDENTIFIED

NUTRITIONAL COFACTOR NOTES

SYNERGISTS

Potassium: shares cellular transport and enzyme sites with thallium; potassium status has been used to contextualise how thallium interacts with energy production and nerve-related processes.

Magnesium: supports enzymatic stability and energy metabolism that may be challenged when thallium interferes with potassium-dependent reactions.

The sodium-to-potassium relationship: has been read as a marker of metabolic pacing and adaptive capacity that frames thallium retention and elimination.

ANTAGONISTS

Hydration and regular bowel and renal clearance have been described in the elimination literature as routes supporting physiological excretion of thallium.

Co-occurring toxic-metal elevations: have been observed to increase overall regulatory and elimination demand when thallium is also elevated.

LITERATURE OBSERVATIONS

PEER-REVIEWED

Chronic thallium exposure has been associated with peripheral neuropathy, paresthesia, and cognitive change, described in the literature as arising from interference with potassium-dependent nerve conduction and energy processes.

Chronic thallium exposure has been linked to hair loss (alopecia), nail changes such as Mees' lines, and alterations in skin and hair structure in exposed populations.

Longer-term thallium burden has been associated with renal tubular dysfunction, proteinuria, and increased risk of chronic kidney disease in population cohorts.

Sustained thallium exposure has been linked to digestive variability, abdominal discomfort, and gastrointestinal distress patterns in environmental and occupational studies.

Cardiovascular associations: Chronic thallium exposure has been associated with elevated cardiovascular mortality risk and blood-pressure changes in general-population research. *General-population research*

CanAlt Health Laboratories

DEMO PATIENT

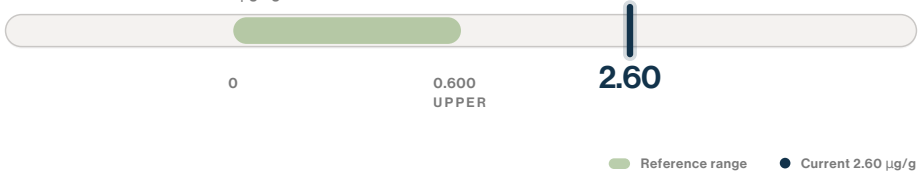
NICKEL (Ni) **HIGH · OUT OF RANGE**

The information on this page is provided to support the practitioner's independent clinical assessment, drawn from the published mineral balancing literature, alongside clinical history, physical assessment, and confirmatory testing where clinically appropriate.

Ni
Nickel
Above range

HAIR MINERAL ANALYSIS · ELEMENT REFERENCE

READING VS RANGE · µg / g



SCAN FOR REFERENCED LITERATURE

Nickel is a widely distributed trace metal with no established essential biological role in human physiology, and an elevated hair reading has been described as reflecting longer-term exposure and gradual tissue accumulation rather than recent intake.

In hair mineral analysis, a high nickel reading has been read as the ongoing exchange between environmental and dietary intake, tissue stores, and the growing hair shaft, with nickel reported to compete with essential elements at shared absorption, transport, and enzymatic binding sites. Nickel is interpreted alongside its mineral relationships and the broader metabolic pattern rather than as an isolated value, since stored nickel may be mobilised and reflected in hair during periods of metabolic adjustment or mineral rebalancing.

SCENARIO REFERENCE

Scenarios described in the published mineral-balancing literature. These are descriptions of the taxonomy, not characterizations of the patient.

RELATES TO	LITERATURE DESCRIPTION	RELATED READINGS
Exposure & body burden	An elevated nickel reading has been associated with longer-term environmental, dietary, or occupational exposure and gradual tissue accumulation rather than recent intake, with research linking sustained nickel contact to allergic contact dermatitis and skin reactivity in exposed individuals. It is read holistically against co-retained toxic metals and the sulfur-dependent handling pathways rather than as an isolated value. ***PMID 31524759***	Ni 2.60 (▲) · Pb 6.40 (▲) · Cd 0.420 (▲) · Al 82 (▲) · S 40,500 (▼) · Ca:Pb 28.1 (▼) · Ca:Cd 429 (▼)
Target-organ effects	A high nickel burden has been associated with cardiovascular strain in population studies and with renal tubular changes in exposed cohorts, where research has linked sustained nickel to endothelial dysfunction, elevated blood pressure, and proteinuria-type kidney patterns. The reading is framed alongside calcium, magnesium, and the fluid-balance and neuromuscular ratios rather than read in isolation. ***PMID 37360515***	Ni 2.60 (▲) · Ca 180 (▼) · Mg 20 (▼) · Ca:Mg 9 (·) · Na:K 3.15 (▲) · K 165 (▲)
Protective & antagonist minerals	In hair-analysis practice an elevated nickel reading has been read against the minerals reported to compete with it at shared absorption, transport, and enzymatic binding sites, with zinc and iron described as antagonists and sulfur-bearing compounds framing thiol-based binding and elimination. The pattern is interpreted as the balance between nickel and these protective minerals rather than nickel alone. ***Watts DL, Trace Elements Inc. — nickel mineral antagonism (Zn / Fe / S) in HTMA***	Ni 2.60 (▲) · Zn 88 (▼) · Fe 5 (▼) · S 40,500 (▼) · Zn:Cu 1.91 (▼) · Fe:Cu 0.109 (▼) · S:Cu 880 (▼)

☐ IDENTIFIED ☐ BORDERLINE ☐ NOT IDENTIFIED

NUTRITIONAL COFACTOR NOTES

SYNERGISTS

Zinc: has been described as competing with nickel at enzymatic binding sites and supporting antioxidant enzyme activity.

Iron: shares absorption and transport pathways with nickel and has been reported to influence its tissue distribution and metabolic efficiency.

Magnesium: has been described as supporting the enzymatic and neuromuscular stability that may be affected by nickel displacement.

Sulfur-containing compounds: have been reported as relevant to nickel binding and endogenous handling through gastrointestinal and biliary elimination routes.

ANTAGONISTS

Hydrogenated oils processed with nickel catalysts, canned foods, and stainless steel cookware and utensils have been described as dietary and consumer contributors to nickel exposure.

LITERATURE OBSERVATIONS

PEER-REVIEWED

Chronic nickel exposure has been strongly associated with allergic contact dermatitis, skin reactivity, and hand eczema in occupationally and environmentally exposed individuals.

Sustained inhalation exposure has been linked to respiratory irritation, lung fibrosis, and chronic bronchitis-like symptoms in occupational cohorts.

Chronic nickel burden has been associated with endothelial dysfunction, elevated blood pressure, and potential cardiovascular disease risk in population and occupational studies.

Long-term exposure has been linked to renal tubular dysfunction, proteinuria, and kidney disease patterns in exposed workers and general populations.

Occupational exposure to certain nickel compounds has been associated with increased risk of lung and nasal cancers in refinery and processing cohorts.

Information on this page is provided for educational and reference purposes only — drawn from the published mineral balancing literature — and should not be relied upon as the basis for clinical diagnosis or treatment decisions.

SCAN FOR REFERENCES

Protective Ratios

Hair mineral analysis provides a tissue-level snapshot of mineral balance and the elimination of toxic metals such as mercury, lead, cadmium, and arsenic. These toxic elements often appear elevated in hair exactly when the body is actively mobilizing and excreting them. The resulting pattern offers insight into metabolic activity and current detoxification capacity.

MINERAL-TO-TOXIC-METAL RATIOS

HIGHER = STRONGER PROTECTIVE BUFFER

Ca:Cd **428.6**	Fe:Pb **0.78**	Zn:Hg **33.8**	Se:Hg **0.15**
Se:As **0.66**	Fe:Hg **1.9**	Ca:Pb **28.1**	Zn:Cd **209.5**

WHY RATIOS MATTER

Ratios between protective nutrient minerals and toxic metals (e.g., Ca/Pb, Se/As, Zn/Cd, Fe/Pb) are usually more clinically meaningful than absolute levels alone. These ratios reflect how well essential minerals compete at absorption sites, induce metallothionein proteins that sequester toxins, and maintain glutathione-dependent antioxidant pathways.

A favorable ratio suggests effective elimination; an unfavorable ratio suggests mineral depletion, reduced binding capacity, or greater retention of toxins in deeper tissues.

INTERPRETING RESULTS

Mineral levels and ratios should never be read in isolation; they belong in the context of the complete mineral pattern. Low nutrient-mineral values may indicate depleted reserves or a reduced metabolic rate after chronic stress, both of which limit detoxification efficiency and allow toxic metals to remain stored in organs and tissues.

Conversely, very low toxic-metal readings do not always mean low exposure. They can reflect poor mobilization and ongoing internal storage. Serial hair analyses over months or years usually provide the most reliable view of progress: as mineral balance improves and metabolic activity rises, previously stored toxins are often released, temporarily raising hair toxic-metal levels during elimination.

KEY PROTECTIVE MINERALS

Zinc: Induces metallothionein proteins that bind cadmium and mercury and promote their elimination. Also supports immune function and tissue repair.

Selenium: Strengthens cellular antioxidant defenses and works with glutathione to neutralize oxidative stress when stored toxins are mobilized.

Calcium: Serves as a marker of metabolic state. Persistently elevated hair calcium often reflects soft-tissue deposition that can slow detoxification.

Iron: Competes with lead and cadmium at the DMT1 transporter. Adequate iron limits absorption; deficiency increases it. Excess iron should be avoided due to Fenton-reaction oxidative stress.

Sulfur: Sulfur-containing amino acids from dietary protein drive glutathione synthesis and metallothionein function. Cruciferous vegetables, garlic, onions, and quality protein support this pathway.

CanAlt Health Laboratories

DEMO PATIENT

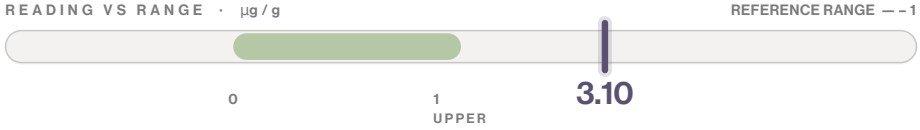
SILVER (Ag) HIGH · OUT OF RANGE

The information on this page is provided to support the practitioner's independent clinical assessment, drawn from the published mineral balancing literature, alongside clinical history, physical assessment, and confirmatory testing where clinically appropriate.

Ag
Silver
Above range

HAIR MINERAL ANALYSIS · ELEMENT REFERENCE

READING VS RANGE · µg/g



SCAN FOR REFERENCED LITERATURE

Silver is a nonessential metal with no known physiological requirement, and excess tissue accumulation has been reported to interfere with normal biochemical processes.

The experimental and observational literature has described silver as a broad enzyme inhibitor with affinity for sulfur-containing proteins, and elevated levels have been associated with disruption of antioxidant activity, cellular respiration, and membrane stability. In hair mineral analysis, an elevated silver reading has been described as reflecting longer-term tissue incorporation rather than acute exposure, offering insight into sustained presence and the body's handling of the metal.

SCENARIO REFERENCE

Scenarios described in the published mineral-balancing literature. These are descriptions of the taxonomy, not characterizations of the patient.

RELATES TO	LITERATURE DESCRIPTION	RELATED READINGS
Exposure & body burden	An elevated silver reading has been associated with cumulative exposure and tissue incorporation from sources such as colloidal silver, occupational dust, dental and wound-care products, and jewellery rather than with acute contact. In hair-analysis practice it has been read holistically as a long-term body-burden marker, framed against the oxidation-rate pattern and the major minerals to distinguish sustained retention from temporary mobilisation. ***PMID 15964881***	**Oxidizer Type: Slow/Extreme Slow** Ag 3.10 (▲) · Ca 180 (▼) · Mg 20 (▼) Na 520 (▲) · K 165 (▲)
Target-organ deposition	Chronic silver accumulation has been associated with deposition in skin, mucous membranes, and the eye, where the literature has described argyria and ocular argyrosis as the most commonly reported and largely cosmetic effects of sustained exposure. The reading has been interpreted holistically alongside total burden and the structural minerals that frame the tissue-retention pattern. ***PMID 32591303***	Ag 3.10 (▲) · Ca 180 (▼) · S 40,500 (▼) Se 0.400 (▼) · Zn 88 (▼)
Protective & antagonist minerals	Elevated silver has been associated with interference at selenium- and copper-dependent antioxidant enzyme systems such as glutathione peroxidase and superoxide dismutase, and with competition for zinc at sulfur-rich metallothionein binding sites. Research has linked these antagonist relationships to disrupted oxidative balance, so the reading has been framed holistically against selenium, copper, and zinc status. ***PMID 30046482***	**Hg:Se** · Ag 3.10 (▲) · Se 0.400 (▼) Cu 46 (▲) · Zn 88 (▼) · Cu:Zn 0.523 S 40,500 (▼)

☐ IDENTIFIED ☐ BORDERLINE ☐ NOT IDENTIFIED

NUTRITIONAL COFACTOR NOTES

SYNERGISTS

Selenium: has been reported to support glutathione peroxidase activity and antioxidant capacity within systems where silver has been described as an inhibitor.

Copper-dependent enzyme systems, including superoxide dismutase, have been described in the literature as participating in the oxidative balance with which silver interacts.

Sulfur-containing amino acids such as cysteine have been described as the protein binding context relevant to silver's affinity and to conjugation and elimination pathways.

ANTAGONISTS

Silver: has been reported to interfere with copper- and selenium-dependent antioxidant enzymes (superoxide dismutase, glutathione peroxidase), a pattern associated with disrupted oxidative balance and reduced cellular defense.

Silver: has been reported to compete with zinc for metallothionein binding sites, a pattern associated with altered zinc distribution and metal-sequestration capacity.

LITERATURE OBSERVATIONS

PEER-REVIEWED

Chronic silver exposure has been associated with argyria, a permanent blue-gray discoloration of the skin and mucous membranes that has been described as irreversible and mainly cosmetic, and as the most commonly reported effect of sustained exposure.

Silver exposure from occupational dust or colloidal silver ingestion has been associated with ocular argyrosis, a gray-blue pigmentation of the cornea, conjunctiva, and sclera that has typically been reported without major vision impairment.

Inhalation of silver dust or compounds in higher occupational exposures has been associated with upper airway, throat, and lung irritation, with breathing difficulty reported in some settings.

Silver ions and nanoparticles have been reported to generate reactive oxygen species and to inhibit antioxidant enzymes such as superoxide dismutase and glutathione peroxidase, a pattern that has been linked with cellular damage and inflammation in experimental models.

High or chronic silver exposure has occasionally been associated with liver and kidney changes, gastrointestinal symptoms, mild anemia, or neurological effects, though these have been reported as uncommon and secondary to argyria at typical exposure levels.

CanAlt Health Laboratories

DEMO PATIENT

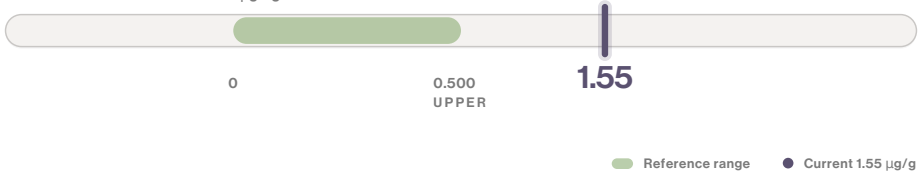
TIN (Sn) HIGH · OUT OF RANGE

The information on this page is provided to support the practitioner's independent clinical assessment, drawn from the published mineral balancing literature, alongside clinical history, physical assessment, and confirmatory testing where clinically appropriate.

Sn
Tin
Above range

HAIR MINERAL ANALYSIS · ELEMENT REFERENCE

READING VS RANGE · µg / g



SCAN FOR REFERENCED LITERATURE

Tin is a naturally occurring metal with no clearly established essential role in human physiology; in hair mineral analysis its relevance has been described as that of an environmental and industrial metal that can accumulate in tissue under conditions of repeated exposure or reduced elimination efficiency.

The mineral-balancing literature has read an elevated tin alongside zinc, iron, copper, and selenium, since tin has been reported to interact with these minerals in enzymatic activity and cellular energy processes. An elevated reading has been interpreted in the broader context of oxidation rate and antioxidant capacity rather than as an absolute value, and is read to distinguish active exposure from reduced clearance or the mobilisation of stored metal during mineral rebalancing.

SCENARIO REFERENCE

Scenarios described in the published mineral-balancing literature. These are descriptions of the taxonomy, not characterizations of the patient.

RELATES TO	LITERATURE DESCRIPTION	RELATED READINGS
Exposure & body burden	An elevated tin reading has been associated with environmental or dietary exposure to inorganic tin and with reduced elimination efficiency rather than with any established physiological need, since tin has no clearly essential role in humans. In hair-analysis practice it has been read within the wider toxic-metal pattern to distinguish an isolated exposure from a broader body burden, and against oxidation rate to gauge active uptake versus mobilisation of stored metal during rebalancing. ***Watts DL, Trace Elements Inc. — HTMA toxic-metal body-burden pattern***	Sn 1.55 (▲) · Pb 6.40 (▲) · Cd 0.420 (▲) · Al 82 (▲) · Hg 2.60 (▲) · As 0.610 (▲)
Target-organ effects	Elevated tin has been associated with gastrointestinal irritation following ingestion of inorganic tin from contaminated or unlacquered canned foods, and chronic high intake has been linked in animal models with reduced iron status, lower haemoglobin and haematocrit. The reading has therefore been cross-referenced with iron and the haematological minerals to frame whether a target-organ effect is plausible alongside the exposure. ***PMID 14563391***	Sn 1.55 (▲) · Fe 5 (▼) · Cu 46 (▲) · Fe:Cu 0.109 (▼) · Ca:Fe 36 (·) · Mn 0.050 (▼)
Protective & antagonist minerals	An elevated tin has been read holistically against zinc, selenium, and iron, since dietary tin has been reported to inhibit zinc retention at the intestinal level and reduced antioxidant and mineral status has been associated with a greater tendency to retain tin. Adequate zinc and selenium have been described as protective considerations, so the reading has been framed by the Zn:Cu balance and the zinc-to-lead relationship rather than by tin alone. ***PMID 6837491***	Zn:Pb · Sn 1.55 (▲) · Zn 88 (▼) · Se 0.400 (▼) · Zn:Cu 1.91 (▼) · Cu:Se 115 (▲)

☐ IDENTIFIED ☐ BORDERLINE ☐ NOT IDENTIFIED

NUTRITIONAL COFACTOR NOTES

SYNERGISTS

Zinc: has been reported to compete with tin at absorption and enzyme-regulation sites; adequate zinc status has been associated with a reduced tendency to retain tin.

Selenium: supports antioxidant buffering capacity, which the literature has linked to the body's handling of retained tin.

Iron status: has been read alongside tin because chronic inorganic tin exposure has been reported to interfere with iron metabolism, and adequate iron has been described as a consideration where tin is retained.

Oxidation-rate and antioxidant-capacity context: has been used to read whether an elevated tin reflects active exposure or reduced elimination.

LITERATURE OBSERVATIONS

PEER-REVIEWED

Acute ingestion of inorganic tin from contaminated canned foods, often from unlacquered cans, has been associated with gastrointestinal irritation including nausea, vomiting, abdominal pain, cramping, and diarrhoea, described as gastric irritation at high doses.

High dietary tin has been reported to impair zinc absorption and retention, increasing faecal zinc excretion, decreasing plasma zinc, and potentially producing negative zinc balance, a pattern described as disrupting zinc-dependent enzymes.

Neurodevelopmental indicators: Elevated hair tin has been reported alongside other toxic metals in children with autism spectrum disorder, where it has been described as a potential marker of broader environmental burden; the links have been characterised as associative and not causal for tin alone. *Hair multi-element autism-spectrum biomonitoring research*

Chronic or high inorganic tin exposure has been associated with interference in iron metabolism, with reduced serum iron and contributions to anaemia including decreased haemoglobin and haematocrit reported primarily in animal models and described as plausible in humans with compromised iron status.

CanAlt Health Laboratories

DEMO PATIENT

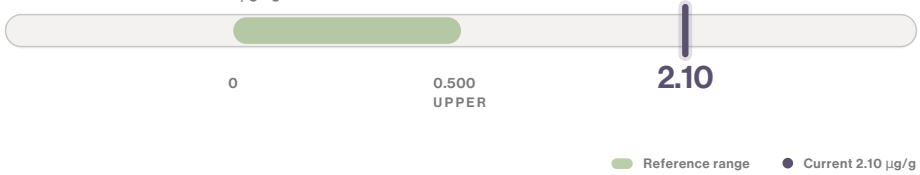
ZIRCONIUM (Zr) **HIGH · OUT OF RANGE**

The information on this page is provided to support the practitioner's independent clinical assessment, drawn from the published mineral balancing literature, alongside clinical history, physical assessment, and confirmatory testing where clinically appropriate.

Zr
Zirconium
Above range

HAIR MINERAL ANALYSIS · ELEMENT REFERENCE

READING VS RANGE · µg / g



SCAN FOR REFERENCED LITERATURE

Zirconium is a transition metal naturally present in soil, water, and rock, with no established essential biological role in human physiology.

In the mineral-balancing literature it has been treated as an ultra-trace environmental element rather than a metabolically regulated mineral, and an elevated hair reading has been described as a reflection of external exposure or surface deposition rather than internal biochemical demand. Population studies have reported very low baseline hair zirconium, with variability linked to environmental contact and personal-care product use, so the reading has been read against exposure history, elimination efficiency, and the broader mineral pattern rather than as an indicator of physiological requirement.

SCENARIO REFERENCE

Scenarios described in the published mineral-balancing literature. These are descriptions of the taxonomy, not characterizations of the patient.

RELATES TO	LITERATURE DESCRIPTION	RELATED READINGS
Exposure & body burden	An elevated zirconium reading has been associated with external and dermal exposure rather than an essential biological role, most often from zirconium-containing personal-care products and airborne particulate contact. In hair-analysis practice it has been read against aluminium and the co-occurring toxic-metal load so an exogenous deposition signal is distinguished from a broader environmental burden. ***PMID 9308573***	Zr 2.10 (▲) · Al 82 (▲) · Ni 2.60 (▲) · Ca:Pb 28.1 (▼) · Ca:Cd 429 (▼)
Target-organ & tissue effects	Persistently elevated zirconium has been linked in occupational research to inhalation of zirconium particulates, where exposure was associated with pulmonary changes and zirconium granulomas, and in personal-care studies to cutaneous granuloma formation. The reading has been interpreted holistically alongside other respiratory-relevant toxic metals rather than as a stand-alone tissue marker. ***PMID 2043021***	Zr 2.10 (▲) · Al 82 (▲) · Ni 2.60 (▲) · Be 0.020 (·) · Oxidation Rate
Protective minerals & total load	Research using hair mineral analysis has linked elevated toxic-element profiles with reduced concentrations of protective minerals, reflecting oxidative-stress and antagonist dynamics. A high zirconium reading has therefore been framed against zinc, selenium, copper, and the antagonist ratios so adequacy of the protective mineral pool is read alongside the overall body burden. ***PMID 41465571***	Zr 2.10 (▲) · Zn 88 (▼) · Se 0.400 (▼) · Cu 46 (▲) · Zn:Cu 1.91 (▼) · Cu:Se 115 (▲)

☐ IDENTIFIED ☐ BORDERLINE ☐ NOT IDENTIFIED

NUTRITIONAL COFACTOR NOTES

SYNERGISTS

Oxidation-rate context: has been used to distinguish chronic surface retention or reduced elimination from temporary mobilization during mineral rebalancing.

Overall mineral: balance has been read alongside zirconium so an exogenous reading is interpreted against the full profile rather than as an isolated value.

Adequate hydration and regular elimination have been described as supportive of endogenous handling of trace environmental metals.

ANTAGONISTS

Stick-form ('stift') antiperspirants containing zirconium compounds have been reported to produce 3-to-5-times higher hair zirconium than roll-on or spray forms, reflecting dermal-exposure differences.

Zirconium from antiperspirant compounds (e.g., aluminum zirconium tetrachlorohydrex) has been reported to be absorbed through the skin and subsequently detected in hair.

Occupational exposure to zirconium particulates has been linked to high hair zirconium and to pulmonary changes in industrial settings.

LITERATURE OBSERVATIONS

PEER-REVIEWED

Respiratory-illness observations: Studies have reported elevated hair zirconium in association with respiratory conditions, with higher zirconium content observed in individuals with current respiratory infections or asthma compared with healthy subjects. *Hair-element observational research*

Antiperspirant delivery-form observations: Research has reported that use of stick-form antiperspirants containing zirconium compounds was associated with hair zirconium 3-to-5-times higher than roll-on or spray forms, reflecting dermal-exposure differences. *Personal-care exposure research*

Dermal-absorption observations: Studies have reported that zirconium from antiperspirant compounds (e.g., aluminum zirconium tetrachlorohydrex) was absorbed through the skin, unlike aluminum in comparable products, and was subsequently detected in hair. *Dermal-absorption research*

Occupational research has linked high hair zirconium to exposure to zirconium particulates, which has been associated with zirconium pneumoconiosis or pulmonary fibrosis in industrial settings.

Hair-treatment observations: Studies have reported that hair-dryer use was associated with approximately 50 percent lower hair zirconium, likely from heat-induced cuticle damage allowing elution or washout of the element. *Hair-handling methodological research*

CanAlt Health Laboratories

DEMO PATIENT

CA/K RATIO · LOW

Information on this page is provided for educational and reference purposes only — drawn from the published mineral balancing literature — and should not be relied upon as the basis for clinical diagnosis or treatment decisions.

SCAN FOR REFERENCES

Ca/K

Calcium · Potassium

▼ LOW

HAIR MINERAL ANALYSIS · RATIO REFERENCE

1.1

REFERENCE RANGE	MULTIPLE OF UPPER	READING COMPONENTS
2.00–6.00	—	Ca 180 · K 165

NORMAL BAND 2.00–6.00

0 **1.1** CURRENT

The calcium potassium (Ca/K) ratio, known as the thyroid ratio, measures long term metabolic rhythm and cellular thyroid efficiency.

This pattern often emerges during the alarm stage of stress, where the body removes stabilizing calcium influences to maximize responsiveness and drive. Biochemically, elevated potassium increases cell membrane permeability and amplifies tissue sensitivity to thyroid hormones, leading to heightened cellular thyroid effect even when blood thyroid levels (TSH, T4, T3) appear normal. Over time, this characterizes fast oxidation, marked by rapid nutrient consumption, biochemical friction, and symptoms like restlessness, anxiety, irritability, tachycardia, heat intolerance, and emotional volatility.

Balancing a reduced Ca/K ratio focuses on reintroducing stabilizing calcium influences to soften the metabolic pace and shift the body out of constant alarm mode. This involves supporting calcium through nutrient dense foods (for example, properly prepared dairy or vegetables), addressing sympathetic overdrive and adrenal factors, and following targeted nutritional balancing protocols. As the ratio normalizes toward ideal, the system can transition from stress driven, catabolic energy toward more sustainable and grounded vitality, protecting against eventual endocrine exhaustion.

CLINICAL ASSOCIATIONS

LITERATURE REVIEW

Sympathetic Overdrive Symptoms: Frequently linked in HMA practice to pronounced signs of acute sympathetic nervous system activation, including restlessness, anxiety, irritability, tachycardia (elevated heart rate), heat intolerance, and emotional volatility; these reflect the body's heightened fight-or-flight state and accelerated cellular energy turnover during the alarm phase of stress. *Wilson L · Nutritional Balancing and Hair Mineral Analysis*

Thyroid-Related Patterns: In HMA interpretation, a reduced Ca/K ratio is commonly associated with increased thyroid hormone effectiveness at the cellular and tissue level (often described as heightened thyroid expression or hyperthyroid-like cellular trends), which can contribute to patterns of accelerated metabolism and sympathetic drive even when circulating blood thyroid panels (TSH, T4, T3) remain within normal reference ranges. *Wilson L · Nutritional Balancing and Hair Mineral Analysis*

Glucose and Insulin Dynamics: Low tissue calcium combined with high potassium patterns are associated with initially heightened insulin sensitivity and rapid glucose uptake; however, if the catabolic stress persists, this can transition into impaired glucose regulation, elevated insulin resistance markers, and metabolic instability. *Wilson L · Nutritional Balancing and Hair Mineral Analysis*

Electrolyte and Nutrient Flux: Elevated intracellular potassium tends to increase the electrical gradient across cell membranes, promoting renal excretion of magnesium and sodium; this ongoing electrolyte loss amplifies autonomic alarm responses, perpetuates nutrient depletion cycles, and further supports the fast oxidation state. *Wilson L · Nutritional Balancing and Hair Mineral Analysis*

Inflammatory and Catabolic Drive: Fast oxidation patterns characterized by reduced Ca/K are linked to elevated production of pro-inflammatory cytokines (e.g., IL-6), increased sympathetic-adrenal-medullary axis activity, accelerated tissue turnover, and challenges in maintaining lean muscle mass or structural integrity over time. ***PMID 36880228*** *· Xie T 2023*

CanAlt Health Laboratories

DEMO PATIENT

NA/MG RATIO · HIGH

Information on this page is provided for educational and reference purposes only — drawn from the published mineral balancing literature — and should not be relied upon as the basis for clinical diagnosis or treatment decisions.

SCAN FOR REFERENCES

Na/Mg

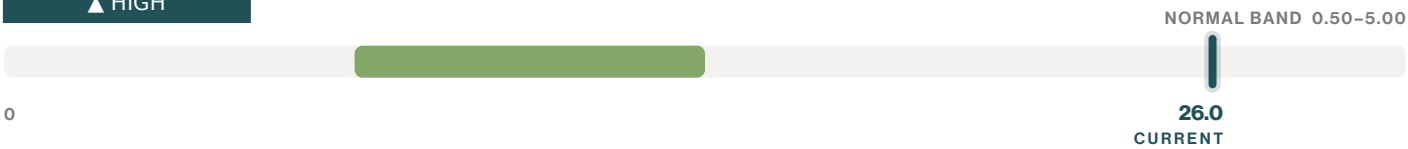
Sodium · Magnesium

▲ HIGH

HAIR MINERAL ANALYSIS · RATIO REFERENCE

26.0

REFERENCE RANGE	MULTIPLE OF UPPER	READING COMPONENTS
0.50–5.00	5.2×	Na 520 · Mg 20



The sodium magnesium (Na/Mg) ratio reveals the current intensity of adrenal drive and the body's readiness for external demands.

This configuration is typical of the alarm phase of stress, when immediate action takes priority. With magnesium relatively suppressed, activation proceeds with minimal internal moderation. This results in a quicker internal rhythm, tighter stimulation thresholds, and faster consumption of recovery reserves. Prolonged elevation creates sharp energy spikes followed by deep fatigue, straining the nervous system and metabolic foundation.

Correction restores magnesium's moderating power so adrenal response becomes less forceful and more controlled. This is accomplished through magnesium rich whole foods (such as leafy greens and balanced seeds), intentional stress calming practices, and carefully chosen protocols. When the ratio returns to balance, the body regains effective responsiveness without constant overdrive, supporting steadier energy, stronger tissue resilience, and lower burnout risk.

CLINICAL ASSOCIATIONS

LITERATURE REVIEW

Adrenal Hyper function: Strong adrenal cortical output and elevated aldosterone signaling promote sodium retention and magnesium wasting at the tissue level, typical of acute stress overload and sympathetic activation. ***PMID 15692156*** · *Matsuoka H 2005*

Neuromuscular Excitability: Weakened magnesium restraint leads to muscle tightness, cramping, or exaggerated sensitivity to stimulants and stressors, reflecting reduced membrane stabilization. ***PMID 38709369*** · *Stanojevic M 2025*

Hyper metabolic Trends: Fast glucose turnover and immediate stress activation, characteristic of the early General Adaptation Syndrome phase with high energy demand and increased catecholamine influence. ***PMID 18406806*** · *Nonogaki K 1997*

Vascular and Inflammatory Tension: Heightened vascular tone and pro inflammatory activity, frequently felt as internal pressure, symptom amplification, or inflammatory flare during peak demand; linked to higher IL-6 and cytokine signaling. ***PMID 1584207*** · *Weglicki WB 1992*

Cardiovascular Reactivity: Increased sympathetic influence and myocardial excitability, contributing to functional blood pressure swings, tension under load, and higher risk of premature ventricular complexes in stress states. ***PMID 22584491*** · *Falco CN 2012*

CanAlt Health Laboratories

DEMO PATIENT

FE/CU RATIO · LOW

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SCAN FOR REFERENCES

Fe/Cu

Iron · Copper

▼ LOW

HAIR MINERAL ANALYSIS · RATIO REFERENCE

0.11

REFERENCE RANGE
0.26–1.20

MULTIPLE OF UPPER
—

READING COMPONENTS
Fe 5 · Cu 46

NORMAL BAND 0.26–1.20

0 **0.11** CURRENT

The iron copper (Fe/Cu) ratio serves as a sensitive gauge of copper burden and iron availability.

Viral infections, chronic copper exposure (diet, water, birth control, copper IUDs), or zinc/copper supplementation often underlie this imbalance. Excess copper disrupts iron transport (ceruloplasmin overload or biounavailability), promotes viral replication, and generates unbound copper-induced oxidative stress. The long-term picture includes fatigue, brain fog, estrogen dominance, anemia despite normal iron, and risks of neurological or cardiovascular complications.

Correction centers on supporting iron availability and gently lowering copper excess. Include iron-rich foods (red meat, spinach in moderation), limit copper sources, and apply targeted protocols to improve copper bioavailability. When the ratio normalizes, iron utilization improves, toxicity risk decreases, and the body regains stable energy and hormonal equilibrium.

CLINICAL ASSOCIATIONS — LITERATURE REVIEW

Viral Infection Tendency: Low iron relative to copper favors viral proliferation by limiting iron-dependent antiviral defenses and supporting copper-mediated replication. *Wilson L · Nutritional Balancing and Hair Mineral Analysis*

Copper Toxicity Symptoms: Copper predominance is associated with biounavailable copper, leading to fatigue, brain fog, anxiety, depression, and estrogen dominance. *Wilson L · Nutritional Balancing and Hair Mineral Analysis*

Iron Deficiency Anemia: High copper interferes with iron oxidation and transport, contributing to functional anemia even when iron stores appear normal. *Wilson L · Nutritional Balancing and Hair Mineral Analysis*

Cardiovascular and Metabolic Strain: Reduced Fe/Cu correlates with hidden copper toxicity, linked to hypertension, metabolic syndrome, and cardiovascular risk in hair mineral studies. ***PMID 37367837*** *· Vigna L 2023*

Hormonal and Neurological Imbalance: Copper excess promotes estrogen dominance and thyroid overstimulation, associated with PCOS-like patterns, mood disorders, and neurological irritability. ***PMID 39139929*** *· Liu Y 2024*

CanAlt Health Laboratories

DEMO PATIENT

ZN/CU RATIO · LOW

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SCAN FOR REFERENCES

Zn/Cu

Zinc · Copper

▼ LOW

HAIR MINERAL ANALYSIS · RATIO REFERENCE

1.9

REFERENCE RANGE	MULTIPLE OF UPPER	READING COMPONENTS
4.00–17.0	—	Zn 88 · Cu 46

NORMAL BAND 4.00–17.0

0 **1.9** CURRENT

The zinc copper (Zn/Cu) ratio is a critical marker in hair mineral analysis for immune modulation, hormonal balance, and thyroid function.

This pattern frequently develops in chronic stress, copper overload (for example, from water pipes or IUDs), or zinc deficiency from diet malabsorption. Physiologically, high copper relative to zinc disrupts zinc dependent enzymes, promotes pro inflammatory cytokines, and leads to estrogen dominance. Serum levels may show transient low Zn or high Cu/Zn during acute inflammation, but hair levels extend these observations by revealing chronic copper dominance, commonly interpreted in HMA as hidden (biounavailable) copper toxicity, where copper accumulates in tissues (liver, brain, thyroid) despite normal or low normal serum copper. Over time, it reflects trends toward chronic inflammation, immune weakness, hypothyroidism, and risks of metabolic syndrome or cancer.

Balancing a reduced Zn/Cu ratio emphasizes supporting zinc status to restore enzymatic and hormonal function. This involves zinc rich nutrient dense foods (for example, pumpkin seeds or beef in moderation), reducing copper sources, and targeted protocols. Normalization promotes stronger immune competence, reduced inflammation, and a shift from sluggish metabolism to efficient cellular activity.

CLINICAL ASSOCIATIONS

LITERATURE REVIEW

Immune Suppression: Low zinc relative to copper correlates with reduced immune competence, increased infection susceptibility, and delayed wound healing; associated with zinc deficiency in recurrent infections or ADHD in children. ***PMID 2200472*** · *Keen CL 1990*

Chronic Inflammation: Copper predominance promotes pro inflammatory cytokine elevation (IL 6, TNF), linked to chronic conditions like rheumatoid arthritis or metabolic syndrome. ***PMID 21223959*** · *Guo CH 2011*

Thyroid Disorders: Reduced Zn/Cu is strongly associated with hypothyroidism and Hashimoto's thyroiditis, as copper excess and zinc deficiency impair thyroid hormone synthesis, T3/T4 conversion, and TSH signaling. Hair levels extend serum findings by confirming chronic imbalance, often interpreted as hidden copper toxicity, in persistent thyroid autoimmunity. ***PMID 8157857*** · *Nishiyama S 1994*

Metabolic Syndrome and Insulin Resistance: Lower Zn/Cu ratios in hair are linked to metabolic syndrome components, including insulin resistance and obesity in population studies. ***PMID 32545948*** · *Jeong SY 2021*

Hormonal Imbalance: Reduced Zn/Cu is associated with estrogen dominance, hypothyroidism trends, and reduced testosterone; common in androgenetic alopecia and PCOS like states, especially when hidden copper toxicity is present. *Wilson L · Nutritional Balancing and Hair Mineral Analysis*

CanAlt Health Laboratories

DEMO PATIENT

CU/MO RATIO · LOW

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SCAN FOR REFERENCES

Cu/Mo

Copper · Molybdenum

▼ LOW

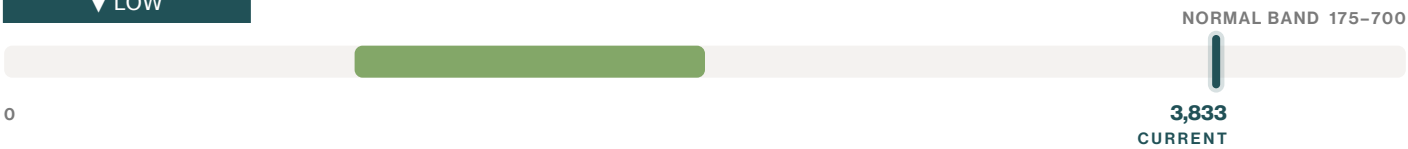
HAIR MINERAL ANALYSIS · RATIO REFERENCE

3,833

REFERENCE RANGE
175–700

MULTIPLE OF UPPER
5.5×

READING COMPONENTS
Cu 46 · Mo 0.012



In hair mineral analysis (HMA), the copper-to-molybdenum (Cu/Mo) ratio serves as a reliable gauge of copper handling and excretion efficiency.

When the Cu/Mo ratio is elevated, it frequently points to copper retention or biounavailable copper, meaning copper is present in tissues but not properly utilized or cleared. This pattern is especially common in slow oxidation states, where metabolic rate is lower, energy feels sluggish, and parasympathetic dominance prevails. Individuals with high Cu/Mo often experience chronic fatigue, hormonal imbalances (such as estrogen excess or thyroid sluggishness), mood swings or emotional volatility, and heightened oxidative stress from unchecked copper activity.

A lower Cu/Mo ratio, by contrast, generally reflects smoother copper metabolism, better excretion, and more balanced energy dynamics.

This ratio offers practical insight into how well the body regulates one of its most influential trace elements—copper—while highlighting potential links to fatigue, endocrine function, and overall detoxification capacity. As with all HMA ratios, interpret it within the complete mineral pattern and individual context for meaningful understanding.

CanAlt Health Laboratories

DEMO PATIENT

CU/SE RATIO · LOW

Information on this page is provided for educational and reference purposes only — drawn from the published mineral balancing literature — and should not be relied upon as the basis for clinical diagnosis or treatment decisions.

SCAN FOR REFERENCES

Cu/Se

Copper · Selenium

▼ LOW

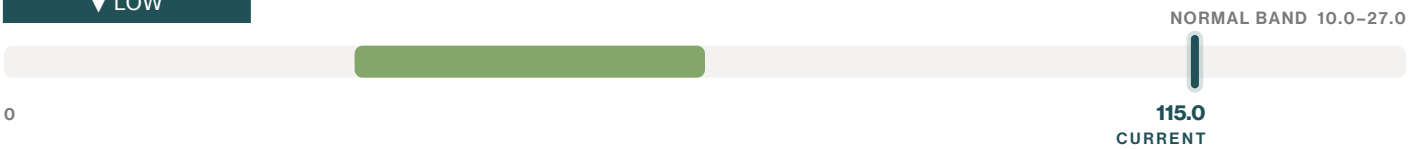
HAIR MINERAL ANALYSIS · RATIO REFERENCE

115.0

REFERENCE RANGE
10.0–27.0

MULTIPLE OF UPPER
4.3×

READING COMPONENTS
Cu 46 · Se 0.4



Hair mineral analysis (HMA) uses the copper-to-selenium (Cu/Se) ratio to capture how well the body balances copper load against its protective antioxidant and thyroid-support mechanisms.

When the Cu/Se ratio climbs high, selenium is often outmatched by copper. This setup shows up frequently in slow-oxidation profiles, where metabolism runs cooler, energy feels heavy, and tissues tend to hold onto minerals. The fallout can include creeping oxidative stress, sluggish thyroid conversion, subtle hormonal shifts, brain fog, or a general sense of being "wired but tired." Copper's unchecked influence starts to tip the scales toward irritation and fatigue rather than smooth function.

A more favorable (lower) Cu/Se ratio, by contrast, usually means selenium is holding its ground: copper stays in check, oxidative pressure eases, thyroid signaling flows better, and the whole system breathes a little easier.

In short, this ratio quietly reveals whether antioxidant defenses and thyroid conversion are keeping pace with copper demands. Like every HMA marker, its real story emerges only when you zoom out and look at the full mineral landscape alongside the person's symptoms and history.

CanAlt Health Laboratories

DEMO PATIENT

S/CU RATIO · LOW

Information on this page is provided for educational and reference purposes only — drawn from the published mineral balancing literature — and should not be relied upon as the basis for clinical diagnosis or treatment decisions.

SCAN FOR REFERENCES

S/Cu

Sulfur · Copper

▼ LOW

HAIR MINERAL ANALYSIS · RATIO REFERENCE

880.4

REFERENCE RANGE
1,500–6,000

MULTIPLE OF UPPER
1.8×

READING COMPONENTS
S 40500 · Cu 46

NORMAL BAND 1,500–6,000

In hair mineral analysis (HMA), the sulfur to copper (S/Cu) ratio offers a clear view of how well the body manages copper clearance.

A low S/Cu ratio usually means sulfur is insufficient compared to the copper present. This pattern appears frequently during prolonged stress or in slow oxidation states, where metabolism slows and detoxification becomes less efficient. Without adequate sulfur, copper tends to linger, often contributing to persistent fatigue, skin sensitivities, mood fluctuations, neurological fog, or the classic sense of feeling wired yet exhausted.

When the S/Cu ratio is more balanced or higher, sulfur pathways generally keep pace with copper demands. Copper gets properly bound and excreted, which helps reduce oxidative strain and supports steadier energy and emotional balance.

Overall, this ratio highlights whether the body's copper detoxification system has the necessary building blocks. As with every HMA marker, its meaning deepens when considered alongside the complete mineral profile, symptoms, and personal context.

RATIOS

LOW Ca/Na RATIO

READING	REFERENCE RANGE
0.35	0.50–5.00

Active adrenal output with sodium rising while calcium holds or drops. The clinical picture aligns with acute or early-chronic stress rather than the recovery patterns seen with elevated Ca/Na. Read against Na/K and Na/Mg â€" all three elevated is the full sympathetic-dominance picture; low Ca/Na with elevated Na/K but normal Na/Mg is more benign dietary-sodium excess (processed foods, sports drinks, electrolyte supplements) rather than true stress drive. Stress modulation, sleep, and dietary sodium review are practical entry points.

HIGH K/Mg RATIO

READING	REFERENCE RANGE
8.2	0.50–5.00

Magnesium burn-through with potassium holding steady â€" typically sustained stress, intensive training without adequate recovery, or chronic dietary magnesium insufficiency. Muscle tension, cramping, sleep-onset difficulty, restless legs, and carbohydrate cravings fit the picture. **Na/Mg** confirms active stress drive; normal sodium values suggest the issue is more dietary than stress-driven. Magnesium supplementation (**glycinate, malate, or threonate**) titrated to bowel tolerance resolves most cases over 8-12 weeks; pair with potassium-rich whole foods.

LOW Ca/P RATIO

READING	REFERENCE RANGE
1.5	1.60–4.00

Phosphorus running relatively high over calcium â€" suggests **cellular catabolism, protein-heavy dietary pattern, or intracellular phosphate release from tissue breakdown**. Common in athletes in heavy training, during recovery from illness, or with diets disproportionately high in meat and processed foods (phosphate additives). Sustained low Ca/P can compromise bone density over time as phosphate buffering pulls calcium from skeletal reserves. **Serum Ca, phosphate, PTH, and dietary review** clarify the driver. Rebalancing often requires reducing phosphate-additive intake and ensuring adequate calcium-rich foods rather than supplementing either mineral directly.

Fe/Mn RATIO

READING	REFERENCE RANGE
100.0	15.0–80.0

Absorption-level competition â€" iron and manganese compete for the same transporter (DMT1). One typically suppresses the other when either is supplemented heavily. **Don't supplement both simultaneously**; separate dosing by at least several hours if both repletions are needed.

Entries shown mildly out of reference range — see bar chart on page 1 for context.