



HEAVY METAL TESTING

HAIR / URINE / BLOOD

PRACTITIONER MANUAL



NutriPATH Pty. Ltd.

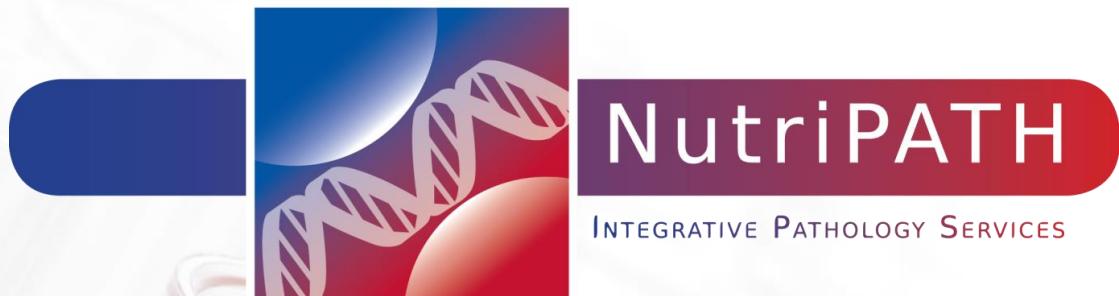
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HEAVY METALS & MINERALS ANALYSES

Dietary elements (commonly known as dietary minerals or mineral nutrients) are the chemical elements required by living organisms, other than the four elements carbon, hydrogen, nitrogen and oxygen present in common organic molecules.

Chemical elements in order of abundance in the human body include the seven major dietary elements calcium, phosphorus, potassium, sulphur, sodium, chlorine and magnesium. Important trace or minor dietary elements include iron, cobalt, copper, zinc, manganese, molybdenum, iodine and selenium.

Trace and essential elements are vital for nearly all of the chemical reactions in the body.

With the enormous amounts of toxic metals in the environment and the widespread nutrient mineral insufficiencies of the modern diet, assessing patients for element imbalances and excesses is an increasingly important tool. Heavy metal toxicity can also lead to numerous health conditions including those affecting the various body systems - cardiovascular, endocrine, immune, musculo-skeletal, integumentary and nervous systems. Mechanisms of toxicity include antagonism of mineral balance, alterations in enzyme activity, changes in neuronal membrane potential, increased oxidation and affects on the immune system.

The term 'heavy metals' is loosely defined: it is related to the periodic table of elements and refers to a variety of elements with high density or metallic properties. These elements are found naturally throughout the environment and are also used by industries to manufacture a wide range of common products. Some of them, such as iron, copper, selenium, molybdenum and zinc, are required in trace amounts by the body for normal function but can be toxic at higher levels.

Significant concentrations of any of the heavy metals can be irritating or damaging to the body and can contaminate soil, air, food and water, persisting indefinitely in the environment. Because they are a source of potential injury, the term 'heavy metals' is frequently used interchangeably with the term 'toxic metals'.

The signs and symptoms that a person may experience depend upon the type of metal, its form, the quantity, the length of exposure, the type of exposure, the age of the person and the person's general state of health. Some metals are much more toxic than others, and one form of a metal may be more harmful than other forms, such as an organic versus an inorganic metal compound. How a person is exposed can influence the amount of metal absorbed and the part(s) of the body that are affected. For example, a metal that does little when it is held in someone's hand, or is only moderately harmful and poorly absorbed when swallowed, may be much more toxic and cause severe lung damage when its vapours are inhaled.

Severe acute exposure can cause damage and, in some cases, can be life-threatening, but moderate exposures over time should also be monitored. The body is able to process small amounts of heavy metals, but moderate to large quantities can accumulate in the kidneys, liver, bones and brain. Some metals are considered carcinogenic and some can affect the body's ability to produce red and white blood cells. Foetuses and young children are at the highest risk because exposures to low or moderate concentrations can affect physical and mental development and can permanently damage the organs and brain. Many of the metals can be passed from the mother to the foetus, and some can be passed to the infant in breast milk.

Importance of measuring minerals and metals

For biological purposes, minerals, metals and elements are often grouped in two categories: those that are required in our diets in amounts greater than 100 milligrams per day and those that are required in amounts less than 100 milligrams daily. The term 'mineral' is applied to the former group, while 'trace element' is applied to the latter.

The vast majority of chemical reactions that govern cellular processes are in turn regulated by enzymatic reactions. Enzyme catalysts most often require mineral cofactors to operate. Magnesium and zinc, for instance, are cofactors in hundreds of enzymatic reactions. Toxic elements, on the other hand, can interfere with enzymatic reactions and disrupt cellular activities.

What is your Nutritional and Mineral Status?

Mineral analysis can be assessed in hair, urine, serum and red cell. Mineral imbalances can indicate sub clinical status prior to presentation of a medical condition. Pathology assessments provide a precise indicator for the activity of many metabolic processes within the body.

Mineral Analysis can provide an important insight into supportive strategies for many conditions ranging from depression and behaviour disorders to cardiovascular and neurological illnesses. Hair Mineral Analysis can provide a basis to adopt a more holistic, comprehensive, and targeted approach in curing many ailments, health concerns and diseases.

Why Test for Minerals?

Minerals are used in practically every metabolic process in the body. Some considerations:

- High levels of Copper and Mercury can lead to mental health disturbances, cognitive and learning difficulties.
- Free calcium excess is associated with loss of Calcium from our bodies can be so advanced that severe osteoporosis might have already set in even without any noticeable change in blood calcium levels.
- Symptoms of iron deficiency can be present long before low iron levels are detected in our blood serum levels.
- Zinc, a very essential element that many of us are most commonly deficient in, is involved in the production, storage, and secretion of insulin and is necessary for growth hormone production.
- Magnesium works synergistically with Calcium and is required for normal muscular function (great for soreness after exercise), especially the heart. Magnesium deficiency has been linked to an increased incidence of heart attacks, anxiety and nervousness.

Why test for Metals?

Heavy metals can enter the body through the skin or by inhalation or ingestion. Toxicity occurs when the metals displace the essential elements in the body and begin to affect the normal function of various organs. Acute or chronic exposures occur in the workplace, especially in industries that use metals to manufacture products; such as cadmium, lead, and mercury used in batteries and the arsenic used in some pesticides. Exposures can also occur in agricultural workers, in people whose job it is to clean up contaminated environmental sites, in those who work with certain products – such as auto mechanics working with car batteries, and in those with hobbies that involve the use of metals – such as the lead used by stained glass artisans.

Signs of metal toxicity:

- Chronic pain and fatigue
- Brain fog – state of forgetfulness and confusion
- Chronic infections such as Candida
- Gastrointestinal complaints, such as diarrhoea, constipation, bloating, gas, and indigestion
- Food allergies
- Dizziness
- Migraines and/or headaches
- Mood swings, depression, and/or anxiety
- Nervous system malfunctions – burning extremities, numbness, tingling, paralysis, and/or an electrifying feeling throughout the body.

HAIR, BLOOD OR URINE ANALYSIS

Which Test is Best?

For more than 30 years, the significance of measuring element concentrations in scalp hair, blood and urine has been studied. These biological samples reflect the body's dynamic equilibrium.

Hair mineral analysis is more stable than blood tests as blood is subject to daily and even sometimes, hourly variations. Also, our bodies have an in-built mechanism to maintain blood mineral levels within a narrow 'near normal to normal' range at the expense of other tissues. This explains why blood pH levels are always within a narrow range - serving as a natural defence for the body, drawing minerals from tissues to maintain this pH range. Heavy metals are not found in the blood or urine as the body soon clears the blood by placing the majority of the toxic heavy metal(s) into cells – e.g. liver, brain, kidney, hair. So a blood or urine test will generally only show heavy metal toxicity for acute exposure of heavy metal(s) and will be a negative test within days to weeks of exposure. Therefore, hair analysis is an ideal base line test.

Red cell mineral levels are useful to reveal physiologically active levels and are useful to determine mitochondrial function.

Urine tests measure what metals are being excreted reflecting metabolic status of minerals and metals.

Blood tests measure what is being absorbed and temporarily in circulation before excretion and/or transportation into storage depots.

Urine and blood can both be used for heavy metal testing, but they do not necessarily test for the same forms of the metal. For instance, methyl mercury – organic mercury found in fish – can be detected in the blood but not in urine. Urine is the preferred sample for measuring inorganic forms of mercury and for measuring arsenic.

Hair and fingernail analysis can give an indication of exposure that has occurred over time or in the past but will not show recent exposures. Blood and urine will reflect exposures that are chronic or that have happened in the last few days.

Any abnormalities in metal toxicities present clinical criteria for DMPS challenge testing.

The medical approach to diagnosing heavy metal toxicity is the **POST CHELATION METALS CHALLENGE** (also known as provocation testing). DMPS is a chelating agent that can be administered intravenously, that will then capture heavy metal(s) and then be excreted by the kidneys. A urine sample is taken before administration of DMPS as a baseline. After DMPS is given intra-venously, the patient collects urine for the following 24 hour period.

HAIR ANALYSIS

Hair mineral analysis is the analysis of hair tissue to determine its chemical content. To understand how hair retains elements, it is important to know the structure of hair and how hair protein is synthesized and traps minerals.

Because of the chemical make-up of human hair, it traps and stores elements between strands of keratin, the protein that comprises around 95% of hair tissue. Minerals become trapped within salt bonds, whilst heavy metals attach to sulphhydryl (-SH) groups between adjacent cysteine molecules that form part of the protein structures of individual keratin strands.

These constituents remain attached to the keratin for the life of that individual hair, enabling them to be analysed when a hair sample is submitted for testing.

The growing hair follicle is richly supplied with blood vessels, and the blood that bathes the follicle is the transport medium for both essential and potentially toxic elements. As these elements reach hair follicles, they are then incorporated into the growing hair protein. Unlike other body tissues, hair is a metabolic end product that incorporates elements into its structure while growing. As hair approaches the skin surface, it undergoes a hardening process, or keratinisation, and the elements accumulated during its formation are sealed into the protein structure of the hair. Because of the exposure of hair follicles to the blood supply during growth, element concentrations of the hair reflect concentrations in other body tissues.

On average, human hair grows at a rate of about 1cm per month; therefore the analysis of a 3-4cm cut sample represents what that growing hair has absorbed over the last 3-4 month period.

By contrast, fingernail samples typically represent growth over a 5-6 month period, whilst toenail samples accumulate minerals and metals over a 10-12 month timeframe.

By comparing the results of both hair and nail samples, we can check whether exposure to a toxic metal has occurred recently (hair) or over a longer period (nails).

There are numerous papers on the accuracy and efficacy of hair testing, particularly for toxic metals such as mercury.^{4,5} The Environmental Protection Agency (E.P.A.) published an authoritative study in 1979 in which more than 400 reports on hair testing were reviewed. The authors concluded that hair is a "meaningful and representative tissue for biological monitoring of most of the toxic metals."⁶ Hair analysis is also useful as a prognostic tool to ascertain whether an individual has a specific biochemical uniqueness, which can then be addressed in a therapeutic or prophylactic program.

Hair element testing is best viewed as a means to monitor element imbalances and environmental toxicity. Follow-up blood testing or provocative urine testing is useful to confirm hair element findings.

In recent years, hair evaluation for toxic elements such as lead, mercury, cadmium, and arsenic has received scientific validation. Studies confirm that toxic elements can directly influence behaviour by impairing brain function, influencing neurotransmitter production and utilization, and altering metabolic processes. Gastro-intestinal, neurological, cardiovascular and urological systems are susceptible to impairment and dysfunction induced by elements.



Environmental exposure to toxic metals may be infrequent and highly variable, and hair element concentrations are most meaningful when cumulative intake and exposure over time is the case.¹³⁻¹⁵ Research suggests hair metal content provides a better estimate for long-term accumulation when compared to blood metal levels.¹⁶ Hair is an excellent medium because concentrations often are up to 300 times higher than those of serum or urine. Because hair stores these elements, it is a barometer of early, chronic exposure and often reflects excess exposure before symptoms appear.

URINE / BLOOD ANALYSIS

Urine analysis can provide important information to the clinician that may not be readily available with blood analysis. Minerals can be stored in various tissues where they may cause damage or metabolic interference in the depot structures (kidney, bone, nerve tissue) without causing particularly elevated blood levels. Toxic elements are often cleared rapidly from the blood, leaving only a relatively brief time window in which blood levels reflect actual body burden.

Provocative testing can help determine such instances of toxic element deposition and provide the clinician with clear therapeutic direction and accurate monitoring of treatment response. This method allows a sampling of the stored deposits of toxic elements, which have been sequestered from the blood.

Levels of nutrient elements in the blood and the excreted urine are tightly controlled via metabolic, re-absorptive and excretory mechanisms. Consequently, most urine testing is not helpful in nutritional element assessment. However, inclusion of nutritional elements among the analytes reported can support the study of nutrient/toxic interactions.

Note that measured results never reveal exactly how much to supplement when a level is abnormal. Analyses measure excretion or saturation levels and are indicative of excess or deficiency. Individual supplementation needs to be determined on a case-by-case basis.

ADVANTAGES & DISADVANTAGES OF METAL AND/OR MINERAL ANALYSIS

HAIR

Hair is perhaps the best specimen through which to evaluate mineral imbalances and toxicities. It provides good long-term exposure assessment, is non-invasive, is inexpensive and allows for investigation of nutrient/toxic interactions, which are only beginning to be determined for the other samples mentioned.

It is a test with an easy specimen collection; making it ideal for testing children, the elderly and non-compliant patients.

It provides information on long term levels of nutrients and exposure to heavy metals. Since hair grows at approximately 1cm per month, the hair assessed during this diagnostic procedure provides data on the toxic exposure and mineral levels in the body from the prior 2-4 months. Therefore, re-testing of the patient must occur no sooner than 2-4 months after any treatment for mineral deficiencies or metal toxicities has been finished to ensure an adequate period for hair re-growth.

It requires no dietary changes prior to specimen collection.

Special consideration of hair treatments is necessary however. Some shampoos (especially anti-dandruff), dyes and swimming pool water treatments can lead to external contamination of the hair and show false evaluations due to hair preparations. Perm, dye, and bleach treatments typically raise levels of aluminium, cadmium, lead, nickel, silver and tin. These elevated levels reflect a combination of the true body level and an extra addition from the hair preparation or treatment. Selenium, when it is elevated, almost always reflects use of a selenium-containing shampoo. Selsun Blue and similar shampoos add selenium to the hair. The resulting level is not representative of the body's real selenium status. If hair treatment may have influenced the test results, wait eight to ten weeks and repeat the test with a new hair sample.

Elevated levels of copper often reflect exposure to swimming pool water treated with algaecide. Occasionally, elevated copper occurs from hair treatments, perm, dye, or bleach. If these conditions do not apply to your patient, then look for possible sources of copper in the environment that may be causing the elevated level. If copper levels are low, the safest course is to measure blood serum ceruloplasmin. Do not supplement with single copper supplements without measuring ceruloplasmin levels first. Low ceruloplasmin may indicate a serious disorder in copper metabolism. Combinations of nutrients that include RDI levels of copper are adequate.

Furthermore, in certain hobbies or occupations, heavy metals can be found in the air, solvent or liquids used which can be a high risk for hair contamination. In these circumstances a urinary provocation test should be considered.



Patient with long hair may find this specimen type unsuitable for aesthetic reasons. If head hair is not available, pubic hair may be used.

Hair mineral analysis is more stable than any blood test as blood tests are subject to daily and even sometimes, hourly variations. Also, our bodies have an in-built mechanism to maintain blood mineral levels within a narrow 'near normal to normal' range at the expense of other tissues.

Hair mineral analysis reflects how efficiently the root was nourished (or intoxicated) via the blood stream. As long as metals circulate, hair tissue will receive them. This feeding and storing mechanism continues over time. Therefore, hair mineral levels reflect how well or poorly the hair tissue was supplied over time. HMA values DO NOT reflect present variations as seen in blood or urine.

Hair tissue storage depends on the body's protein-metal binding ability, which decreases with age. This actually means that an older, grey-haired person is less likely to supply hair tissue with nutrients and toxins. Therefore, elevated levels of any toxins in a grey-haired person are a sign of concern. Light-haired children have a lower protein-metal-binding capacity, and again, elevated levels of any toxin are a sign of concern.

A 'normal' hair level of mercury or lead does not necessarily exclude a metal burden. If a metal such as mercury (Hg) has fully crossed the blood brain barrier and no additional exposure exists, the metal will no longer be detected in the circulating blood stream. Since the metal is no longer circulating, it cannot supply the hair root and hair shaft. As a result, the metal can no longer be detected in the outgrowing hair shaft.

This principle applies to all metals capable of crossing the blood brain barrier. Some chelating agents may cross the blood brain barrier, binding metals, re-routing them back into the bloodstream where these metals, notably mercury, temporarily, and only for a few hours, circulate until excreted.

This temporary circulation is often misinterpreted. Some chelation therapists believe that the redistribution of brain metals into the blood stream causes these circulating toxins to be transported to organ cells where they are stored, causing intoxication of other organ systems. This is a theoretical, but unlikely probability. If this possibility would exist, all chelation would lead to redistribution of chelator-bound metals into other organ cells. This is a highly unlikely scenario, because a metal which is tightly bound to a chelating agent will not be easily 'dropped'. If that were the case, chelation therapy would be endangering patients rather than detoxifying them; it would not cause detoxification but metal redistribution and the end effect would not be an improvement in metal-related health problems, but an increase in metal-related disease. This is not the case.

Some points to consider with HMA results

Element antagonism is the relationship between certain elements that leads one to become deficient as another increases in tissue concentration or vice versa. There are a number of element antagonisms that need to be recommended in order to use HMA to best effect.

When one mineral is high and its natural antagonist is correspondingly low, this is a good indication of a valid test. Use blood and/or urine to validate your findings.

In general, hair levels of boron, chromium, copper, cobalt, lithium, manganese, molybdenum and zinc correlate with tissue levels.

External contamination should not be excluded when the HMA shows an abnormally high level of any one element, particularly if it is not accompanied by low levels of its antagonist elements e.g. Copper contamination is common and is confirmed if blood ceruloplasmin levels and serum copper are normal. However if zinc and molybdenum are low, then copper intake is likely to be excessive.

Cobalt is a good indicator of vitamin B12 status. Low cobalt could indicate high homocysteine levels. Check folate and vitamin B12 status with blood tests.

Calcium levels are best checked with blood tests and bone densitometry. Electrolytes (**sodium, chloride, potassium**) should be cross-reference with blood tests.

Mercury: Selenium - Often when selenium is low, mercury will tend to be high. This is because in cases of mercury excess, selenium will be used up as it binds in a 1:1 ratio to form a relatively inert selenomeric salt.

Cadmium: Zinc - Cadmium is antagonistic to zinc. High levels of cadmium will lower the tissue level of zinc. Selenium deficiency may enhance cadmium toxicity. If low levels of selenium, supplementation is also indicated.

Copper: Molybdenum, Zinc, Manganese - High copper levels often lead to low levels of zinc and molybdenum. Manganese levels may rise to compensate for low molybdenum and zinc and then normalize as Cu, Zn and Mo levels improve.

Common antagonistic elements:

Aluminium	Si
Arsenic	Se, Zn
Cadmium	Ca, Zn
Copper	Mn, Mo, Zn
Lead	Ca, Zn
Mercury	Se, Zn

URINE



The advantage of this test is it provides a good measure of existing body burden of heavy metals, especially when a chelating agent is administered prior to testing **POST CHELATION METALS CHALLENGE**.

A chelating agent e.g. DMPS, EDTA or DMSA leads to a release of the metals from the body into the urine.

Compared to hair, follow up tests can be performed at any time to determine if therapy has reduced toxic element load.

This test shows what elements the body is currently excreting. It provides good qualitative information if a person has been recently exposed to a toxic element (days to weeks) and it gives quantitative information of excreted elements before, during and after provocative challenge.

Follow up tests can be performed at any time to determine the success of any therapies used to reduce the heavy metal toxicity. They are useful for assessing exposure prior and after exposure to a potential hazard, for example, in the mining industry.

Caution should be taken when using provocation and/or chelation therapy in sensitivity patients, those with chronic illnesses or individuals with poor kidney function. In these instances, it may be more appropriate to avoid the provocation test and consider hair mineral analysis.

BLOOD

The toxic metals screen in blood is the standard test for measuring toxic metals, however, it is only able to detect recent exposure (hours to days). Blood levels do not represent the level of toxic metals within tissues.

As for urine element analysis of toxic elements without provocation, blood analysis does not accurately reflect total body metal burden. For example, blood lead levels appear to peak 4 to 5 hours after exposure and then decrease exponentially with a half-life of about 27 days. Thus, levels of lead in blood are limited to detection of only very recent or ongoing exposure.

Blood provides information about what the body has currently or recently absorbed. Blood levels are largely independent of tissue deposition. Blood levels vary according to the actual component analysed (plasma, serum, red cell). They can be transient in nature, and are subject to the body's homeostatic mechanisms to maintain levels within narrow ranges.

The ability of the blood to counter changes in element presentation keeps nutritional and many toxic levels within a narrow range unless under heavy exposure. This homeostatic response illustrates the effective clearance mechanisms in the blood, and largely explains the short-term utility of blood analysis.

Some Points to Consider With Heavy Metal Testing

Research during the past three decades suggests the relationship between hair, blood, and urine element concentrations and human health is an important and complex process. Variables of key clinical relevance include exposure sources, absorption dynamics, and tissue distribution of the various essential and toxic elements. As our understanding of the sophisticated relationships between nutrient and toxic elements increases, so will the utility of tissue elemental analysis for diagnosis and treatment applications.

Studies of the relationship of mineral status and behavioural disorders, cardiovascular disease, and cancer offer exciting possibilities - continuing to expand the range of applications for mineral and metal analysis far beyond its long-accepted use in cases of acute toxic exposure.

COPPER is a case in point. A doctor may order a serum copper, ceruloplasmin and 24 hour urinary copper, but this does not provide the entire information one really requires determining tissue copper status. Hair analysis is essential for such purposes, in order to ascertain the relative proportions of bioavailable versus bio-unavailable copper in the system. Note that serum copper represents all of the forms of copper present in serum, including that being stored within one's ceruloplasmin molecules. Neither free copper nor the copper transporter, GHK-Cu, can be readily measured at the present time.

Another example is **MAGNESIUM**. Red cell magnesium and 24 hour urinary magnesium excretion are the investigations of choice. Serum magnesium is really only of use in Emergency Departments, when a patient attends with chest pain, often accompanied by a cardiac arrhythmia. Red cell magnesium is by far the better measure of tissue stores. Hair magnesium bears no relationship to red cell magnesium.

Likewise, red cell **ZINC** is individually the most useful of zinc measurements; however, hair zinc levels are also reliable and both can be used in determining zinc status. Pfeiffer protocol however base therapy on plasma zinc, serum copper and ceruloplasmin levels.

Hair **CALCIUM** needs to be combined with 24 hour urinary calcium to give an overall picture of tissue calcium status. A Vitamin D3 blood test is also essential as Vitamin D regulates the absorption of calcium from the gut.

Other trace elements such as **MANGANESE, MOLYBDENUM, CHROMIUM, LITHIUM, VANADIUM** and **SELENIUM** can be reliably measured by hair analysis. Serum or red cell selenium is also a worthwhile confirmatory test of selenium status.

IODINE needs to be measured by a urinary **IODINE LOADING TEST**. Iodine spot urines are only of use in ascertaining the likelihood of iodine deficiency. Only the loading test can ascertain iodine sufficiency. Deficiency and sufficiency are two entirely different concepts. Prevention of iodine deficiency relates to the level of iodine required to prevent the development of goitre. Iodine sufficiency relates to the adequacy of tissue stores to supply iodine to tissues other than the thyroid gland. Such knowledge is essential in the prevention of breast & prostate cancers that are both highly dependent on sufficient stores of iodine (I_2) - not potassium iodide (KI) that is required for normal thyroid function.



Note that approximately around 10% of the population cannot secrete such metals into their hair, due to polymorphisms in their MDR1, MRP1, MRP2 and ABCG2 transporter genes. These genes and the proteins they encode, form an essential component of Phase III detoxification pathways.

Such patients may require **PRE & POST METALS CHALLENGE testing** with, for example, intravenous DMPS 3mg/kg in normal saline (preferred). In order to gain a full appreciation of the likely accumulation of heavy metals in bodily tissues you may need to perform each of the following:

1. Hair mineral analysis
2. Pre-challenge (provocation) 24 hour urine
3. Post-challenge (provocation) 24 hour urine

All three are required to properly evaluate the extent of heavy metal toxicity in any one given patient.

Red cell **MERCURY** is of value in ascertaining the contribution of total mercury from seafood. Serum mercury is only of use in the toxicological assessment of acute metal exposure. Post-challenge urinary mercury is by far the best single test of total mercury burden in any given individual. However, this test should always be performed in combination with both hair and 24 hour pre-challenge urine for a full and proper assessment to be made.

Amino acids can be measured in either plasma or urine. We recommend a 24 hour urine collection particularly if the patient is taking varying amounts of protein in their diet, or an overnight, early morning urine sample may be used. As amino acids are primary precursors to neurotransmitters, assessment of neurotransmitters is recommended in any patient requiring a proper nutritional and mental health analysis. Amino acids analysis is recommended with organic acid testing (a metabolic assessment of the function of the Krebs cycle) in a patient with mitochondrial dysfunction and chronic fatigue.

Conclusion

Research during the past three decades suggests the relationship between hair, blood, and urine element concentrations and human health is an important and complex process. Variables of key clinical relevance include exposure sources, absorption dynamics and tissue distribution of the various essential and toxic elements. As our understanding of the relationships between nutrient and toxic elements increases, so will the utility of tissue elemental analysis for diagnosis and treatment applications.

Studies of the relationship of mineral status and behavioural disorders, cardiovascular disease, and cancer offer exciting possibilities, expanding the range of applications for mineral and metal analysis far beyond its long-accepted use in cases of acute toxic exposure.

Practitioners with patients with non-specific symptoms with no known aetiology should consider testing for and treating mineral deficiencies and/or heavy metal toxicities.

ANALYTES TESTED BY NUTRIPATH

METALS & MINERALS	HAIR Level 1	HAIR Level 2	URINE Mineral	URINE Heavy Metals	URINE Metals & Minerals	BLOOD red cell Metals	BLOOD red cell Mineral	URINE
Test Codes	5013	5014	5019	5020 5022 5024	5021 5023 5025	5026	5027	5015 5016
Aluminium (Al)	✓	✓		✓	✓	✓		
Antimony (Sb)		✓		✓	✓			
Arsenic (As)	✓	✓		✓	✓	✓		
Barium (Ba)		✓	✓		✓			
Beryllium (Be)		✓		✓	✓			
Bismuth (Bi)		✓		✓	✓			
Boron (B)		✓						
Cadmium (Cd)	✓	✓		✓	✓	✓		
Calcium (Ca)	✓	✓	✓		✓		✓	
Chromium (Cr)	✓	✓	✓		✓		✓	
Cobalt (Co)		✓	✓		✓			
Copper (Cu)	✓	✓	✓		✓	✓		
Iodine (I)		✓						✓
Iron (Fe)	✓	✓	✓		✓			
Lead (Pb)	✓	✓		✓	✓	✓		
Lithium (Li)		✓	✓		✓			
Magnesium (Mg)	✓	✓	✓		✓		✓	
Manganese (Mn)	✓	✓	✓		✓			
Mercury (Hg)	✓	✓		✓	✓	✓		
Molybdenum (Mo)		✓	✓		✓			
Nickel (Ni)	✓	✓		✓	✓			
Palladium (Pd)		✓						
Platinum (Pt)		✓		✓	✓			
Potassium (K)								✓
Selenium (Se)	✓	✓	✓		✓		✓	
Silver (Ag)		✓						
Sodium (Na)								✓
Strontium (Sr)		✓	✓		✓			
Thallium (Tl)		✓		✓	✓			
Tin (Sn)	✓	✓		✓	✓			
Titanium (Ti)		✓						
Tungsten (W)		✓						
Uranium (U)		✓						
Vanadium (V)		✓	✓		✓			
Zinc (Zn)	✓	✓	✓		✓		✓	
Zirconium (Zr)		✓						

NUTRIPATH TESTS

Test Name	Code	Analytes
Hair Mineral Analysis - Level 1	5013	<i>8 minerals:</i> Ca, Cr, Cu, Fe, Mg, Mn, Se, Zn; ratios. <i>8 toxic metals:</i> Al, As, Cd, Hg, Ni, Pb, Ag, Sn
Hair Mineral Analysis - Level 2	5014	<i>17 minerals:</i> B, Ca, Co, Cr, Cu, Fe, Ge, I, Li, Mg, Mn, Mo, Se, Sr, V, W, Zn; ratios. <i>18 toxic metals:</i> Al, Sb, As, Ba, Be, Bi, Cd, Hg, Ni, Pb, Pd, Pt, Ag, Tl, Sn, Ti, U, Zr
Essential Mineral Analysis (Urine)	5019	<i>14 minerals:</i> Ba, Ca, Co, Cr, Cu, Fe, Li, Mg, Mn, Mo, Se, Sr, V, Zn
Heavy Metal Analysis (Urine)	5020	<i>12 toxic metals:</i> Al, As, Be, Bi, Cd, Hg, Ni, Pb, Pt, Sb, Sn, Tl
Essential Mineral & Heavy Metal Analysis (Urine)	5021	<i>14 minerals:</i> Ba, Ca, Co, Cr, Cu, Fe, Li, Mg, Mn, Mo, Se, Sr, V, Zn <i>12 toxic metals:</i> Al, As, Be, Bi, Cd, Hg, Ni, Pb, Pt, Sb, Sn, Tl
Pre Chelation Metals Challenge (Urine)	5022	<i>Toxic Metals -</i> Al, As, Be, Bi, Cd, Hg, Ni, Pb, Pt, Sb, Sn, Tl
Pre Chelation Metals & Minerals Challenge (Urine)	5023	<i>26 Toxic Metals & Essential Minerals -</i> Ba, Ca, Co, Cr, Cu, Fe, Li, Mg, Mn, Mo, Se, Sr, V, Zn; Al, As, Be, Bi, Cd, Hg, Ni, Pb, Pt, Sb, Sn, Tl
Post Chelation Metals Challenge (Urine)	5024	<i>Toxic Metals -</i> Al, As, Be, Bi, Cd, Hg, Ni, Pb, Pt, Sb, Sn, Tl
Post Chelation Metals & Minerals Challenge (Urine)	5025	<i>26 Toxic Metals & Essential Minerals -</i> Ba, Ca, Co, Cr, Cu, Fe, Li, Mg, Mn, Mo, Se, Sr, V, Zn; Al, As, Be, Bi, Cd, Hg, Ni, Pb, Pt, Sb, Sn, Tl
Red Cell Metals (Blood)	5026	Al, As, Cd, Cu, Hg, Pb
Red Cell Minerals (Blood)	5027	Ca, Cr, K, Mg, Na, Se, Zn
Iodine – Loading Test (Urinary)	5015	Iodine (random), Iodine (post loading), Iodine excretion %

TOXIC METALS

ALUMINIUM (Al)

A disturbing pattern of aluminium accumulation and interference with normal neurological function appears to be supported in the literature. Dyslexic children were shown to have higher levels of aluminium in their hair compared with controls, and other behavioural difficulties in school also correlated with elevated levels of this element.^{17,18} Urine levels of aluminium are observed to be elevated in people with a history of antacid intake.¹⁹ The estimated half-life of aluminium found in the urine is 7.5 hours.²⁰

There are geographical links between Alzheimer's disease and high aluminium in drinking water. Elevated hair aluminium has been observed in Alzheimer's patients. Amyotrophic lateral sclerosis, another neurodegenerative disease, may also be linked to aluminium content of water supplies.

Habitual underarm antiperspirant application may result in aluminium absorption. All intramuscularly injected Al, e.g. from vaccines, may be absorbed over time. Al distributes unequally to all tissues. Distribution and renal excretion appear to be enhanced by citrate. Brain uptake of Al may be mediated by Al transferrin and Al citrate complexes. Aluminium is deposited in bone tissue and elimination half-lives of several years have been reported. Al elimination is primarily renal with ~ 2% excreted through the bile.

Most ingested aluminium comes from food and drink, while additional amounts may come from pharmaceuticals. Whilst the gastrointestinal absorption of aluminium is fairly minimal, its absorption is typically decreased by the presence of dietary phosphates (from animal protein sources), but may be increased by the presence of citric or malic acids (carboxylic acids) present in foods or drink. Excretion of aluminium from the bloodstream is predominantly by urine.

It is also implicated in the development of the autoimmune ASIA Syndrome, particularly from parenteral use of vaccines, where aluminium is used as an adjuvant to enhance the desired immune response. Fluoride treated water can increase Al bioavailability and uptake.

Administration of chelating agent EDTA can release Al from bloodstream and increase urine levels.

SOURCES: include cookware (coffee pots, pizza pans, utensils), tomato sauce cooked in aluminium pots or pans, antiperspirants/deodorants and cosmetics, aluminium hydroxide antacids (when G.I. phosphate is inadequate), aluminium baking powder, drinking water clarified by 'alum', and drinking from surface-damaged or very old aluminium soft drink cans. Both calcium citrate and fluoride-treated municipal water increase aluminium bioavailability and uptake.

ANTIMONY (Sb)



Symptoms associated with low level or chronic Sb contamination may have delayed or insidious onset and can depend upon the chemical form. Sb rapidly clears the blood (typically within two hours of a point-in-time exposure) and accumulates in the adrenals, thyroid, kidney, liver, spleen, and bone. The Sb (+3) that is not promptly excreted (urine) is preferentially distributed in the liver and is primarily excreted via bile and faeces. Sb (+5), if not excreted via the kidneys in urine, can deposit in bone. Antimony interferes with cellular metabolism by combining with sulphhydryl groups (-SH) on enzymes. Antimony may also disrupt purine metabolism, leading to elevated uric acid, ammonia. By inhibiting the enzyme, monoamine oxidase (MAO), the element can disrupt adrenal catecholamine metabolism. Signs and symptoms consistent with chronic antimony toxicity are variable and may include metallic taste, anorexia, fatigue, myopathy, gout-like symptoms, MAO dysfunction, hypotension, erythrocyte fragility, and angina. "Antimony spots" may result from skin contact with antimony compounds; inhalation of antimony may result in nosebleeds, rhinitis, and pneumonitis.

Hair is a preferred tissue for analysis of Antimony (Sb) exposure and body burden. Elevated hair Sb levels have been noted as long as a year after exposure.

Like arsenic, Sb has a high affinity for sulphhydryl groups on many enzymes. Sb is conjugated with glutathione and excreted in urine and faeces. Therefore, excessive exposure to Sb has the potential to deplete intracellular glutathione pools.

Early signs of Sb excess include fatigue, muscle weakness, myopathy, nausea, low back pain, headache, and metallic taste. Later symptoms include haemolytic anaemia, myoglobinuria, haematuria and renal failure. Transdermal absorption can lead to 'antimony spots' which resemble chicken pox. Respiratory tissue irritation may result from inhalation of Sb particles or dust.

SOURCES: include solders, metal type (printing), antifriction alloys, small-arms ammunition, lead batteries, paints, enamels, glass and pottery glazes, flame-proofing/retardants for textiles and carpets, mordants for textiles and leather dyes, vulcanizing and colouring agent for rubber, tobacco, mines and smelting operations.

Food and smoking are the usual sources of Sb. Thus cigarette smoke can externally contaminate hair, as well as contribute to uptake via inhalation. Gunpowder (ammunition) often contains Sb. Firearm enthusiasts often have elevated levels of Sb in hair. Other possible sources are textile industry, metal alloys, and some antihelminthic and antiprotozoan drugs. Sb is also used in the manufacture of paints, glass, ceramics, solder, batteries, bearing metals and semiconductors.

ARSENIC (As)

In general, hair provides a rough estimate of exposure to Arsenic (As) absorbed from food and water. However, hair can be contaminated externally with Arsenic from air, water, dust, shampoos and soap. Inorganic As, and some organic Arsenic compounds, can cause toxicity. Some research suggests that Arsenic may be essential at extremely low levels but its function is not understood. Inorganic As accumulates in hair, nails, skin, thyroid gland, bone and the gastrointestinal tract. Organic As is rapidly excreted in the urine.

Most forms of ingested arsenic are excreted in urine, and variations in dietary intake, such as a single meal of arsenic containing shellfish, can cause urine levels to temporarily increase by a factor of 50 to 100. Therefore, increased urine arsenic indicates exposure but does not necessarily imply tissue accumulation or toxicity. Besides ingestion, arsenic can be assimilated by inhalation and via contact with the skin. Detoxification occurs via methylation requiring S-adenosylmethionine (SAMe). Arsenic can be increased in urine following administration of sulphhydryl (-SH) detoxifying agents such as DMSA, DMPS, or D-Penicillamine.

Arsenic has multiple toxic effects including inhibition of mitochondrial function, including metabolism of pyruvate, succinate and alpha-ketoglutarate (Kreb's Cycle metabolites), inactivation of lipoic acid, impairment of lymphocyte stimulation and proliferation, and interference with DNA repair processes. Symptoms consistent with excessive arsenic ingestion include garlic breath and increased salivation, fatigue, chest pain, diarrhoea and hypotension. Long term or chronic signs may include hair loss, skin hypopigmentation, white-streaked fingernails, anorexia, peripheral neuropathy, leukopaenia, and erythrocyte fragility.

Arsenic can cause malaise, muscle weakness, vomiting, diarrhoea, dermatitis, and skin cancer. Long-term exposure may affect the peripheral nervous, cardiovascular and hematopoietic systems.

Arsenic is a major biological antagonist to selenium.

SOURCES: include food, water, soil and air, especially around arsenic-containing mineral ores. In industry, arsenic is a by-product of the smelting process for many metal ores such as lead, gold, zinc, cobalt, and nickel. Arsenic is used for purifying industrial gases (removal of sulphur), burning fossil fuels, electronics manufacturing (microwave devices, lasers, light-emitting diodes, photoelectric cells, and semiconductor devices), hardening metal alloys, preserving animal hides, bronze plating, and clarifying glass and ceramics.

Other **potential sources** are wood preservatives, insecticides, herbicides (weed killers and defoliants), fungicides, cotton desiccants, cattle and sheep dips, paints and pigments, antifouling paints, leaded gasoline, and fire salts (multi-coloured flame). Wine (grapes sprayed with arsenic-containing pesticides), or seafood (especially certain cold water and bottom-feeding finfish) and seaweed can be cause of dietary exposure. Smokers inhale small amounts of arsenic as a result of pesticide residue on tobacco leaves.



Commonly encountered sources of arsenic include contaminated shellfish or other seafood, edible seaweeds, production of semiconductor or photoelectric components (particularly, gallium arsenide), electroplating, galvanizing and etching processes, certain fungicides and pesticides, chemical process industry (reagents, catalysts), fireworks (intense white and blue colours), leather tanning and taxidermy, textile printing, lead and copper alloys (cable sheaths, solders, shot), and specialty glass manufacture (opal glass, IR transmitting, decolorizing).

Common sources of Arsenic are insecticides (calcium and lead arsenate), well water, smog, shellfish (arsenobetaine), and industrial exposure, particularly in the manufacture of electronic components (gallium arsenide).

NOTE: To ensure true measures, ensure 48 hours prior to the urine collection no fish or algae products were consumed. Mineral waters high in arsenic may also raise urinary excretion levels. Consumption of any of these sources raises urine levels considerably, at least 2-3 times above the given range. Smoking may also raise urinary excretion levels or arsenic.

BARIUM (Ba)

Under normal conditions, most absorbed barium is excreted via the bile and faeces (about 90%), with urine and sweat accounting for the remainder. Excessive body burden, especially from ingestion of soluble barium salts, can increase urine levels, and chelating agents used for detoxification (such as EDTA) can increase urinary barium levels. Insoluble forms of barium, such as barium sulphate, are routinely used in medical diagnostic scanning and x-ray procedures because barium is radio-opaque. These forms are considered to be nontoxic. Soluble forms of barium (chloride, sulphide, nitrate and carbonate) may be encountered occupationally or in contaminated water or food. These forms are bioavailable and can be quite toxic.

Barium appears to negatively influence calcium absorption, and has properties similar to lead and cadmium. In order to normalize barium levels, consideration must be given to the calcium balance. Antioxidants such as Vitamin C, E, and A, Se and zinc can be used successfully in therapy. Barium sulphate is sometimes used in medical tests and to take x-rays of the gastrointestinal tract.

Few studies relate barium levels in the hair to pathologic processes, although one retrospective study indicated that high levels in the hair along with an elevated calcium/magnesium ratio correlated with myocardial infarction.²⁵ The insoluble form of this element, barium sulphate, is used as an X-ray contrast medium and is non-problematic. Absorbable barium salts (hydroxide, chloride, or carbonate) may occur in some pesticides.²⁶

Barium compounds that do not dissolve well, such as barium sulphate, are generally not harmful. Barium has been found to potentially cause gastrointestinal disturbances and Muscular weakness, changes in heart rhythm or paralysis and possibly death.

Once absorbed, barium has a digitalis-like activity. Tingling in the extremities and muscle stimulation followed by paralysis can occur, as can ventricular fibrillation and respiratory distress. If the exposure is oral, nausea, vomiting and colic can precede muscle and myocardial effects. Toxic effects of barium include displacement of potassium (extracellular potassium is decreased, intracellular potassium may be increased) and hyper secretion of adrenal catecholamines. Symptoms of low-level or chronic barium vapour exposure include bronchoconstriction and increased blood pressure.

Symptoms of low level/chronic Barium vapour exposure include bronchoconstriction and increase BP.

Potassium is an effective antagonist of the cardiotoxic and paralysing effects of barium (Dart R., Medical Toxicology 3rd ed. Lippincott Williams & Wilkins, 2004).

SOURCES: include arc-welding and metal fabrication work, contact with welding flux materials, some rat poisons and insecticides, manufacture and use of flares and fireworks (barium nitrate and chlorate which produce green fumes), manufacture of paints, pigments and glazes, manufacturing of cathode ray tubes and photo cells.

Barium gets into the air during the mining, refining, and production of barium compounds, and from the burning of coal and oil. Fish and aquatic organisms can accumulate barium.

Sources include arc-welding, metal fabrication work, some rat poisons and insecticides, manufacture and use of fireworks/flares, paint/pigment/glaze manufacture.

BERYLLIUM (Be)

Beryllium ores are used to make speciality ceramics for electrical and high-technology applications, also used in nuclear weapons and reactors, aircraft and space vehicle structures, instruments, x-ray machines, and mirrors

Beryllium alloys are used in automobiles, computers, sports equipment (golf clubs and bicycle frames), and dental bridges. Lung damage has been observed in people exposed to high levels of beryllium in the air. Beryllium blocks several hepatic enzyme systems. Marcotte and Witschi (1972) suggested that this element binds to chromatin and interferes with DNA synthesis. Preventive measures such as avoiding skin contact with beryllium to prevent sensitization are most important. Careful irrigation and debridement are recommended for wounds.

Agency for Toxic Substances and Disease Registry (ATSDR). 2002. Toxicological Profile for Beryllium. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service

BISMUTH (Bi)

Inorganic bismuth compounds have been shown to elicit low toxicity, as between 92-97% of ingested bismuth is excreted in the stools. However, methylation by intestinal bacteria convert this relatively harmless substance into potentially toxic trimethyl-bismuth $[(CH_3)_3Bi]$ and bismuth trihydride. These substances are known to accumulate in bodily tissues with established risk of neurotoxicity.

SOURCES: Is used as colouring agent in cosmetics and burn ointments. It is used in x-ray analysis as a fungicide, in wart treatments and to regulate stool odour and consistency in colostomy patients. Bismuth containing medications are used to treat peptic ulcers, H.pylori and to treat diarrhoea. Besides food, drink and pharmaceuticals; sources include cosmetics and lipstick (pearlescent), low melting temperature alloys in fuses, automatic fire sprinklers and solders, pigments and paints, semiconductors, electronic components and batteries, metal casting, production of Cu and Pb.

BORON (B)

Boron is normally found in hair but the correlations among Boron absorption, and tissue and hair levels of Boron have yet to be determined. Boron has a low order of toxicity, but excessive intake induces riboflavinuria. Boron is frequently high in hair in association with high levels of potentially toxic elements (i.e. lead, mercury, and cadmium) and exposure to toxic chemicals.

Exogenous contamination of hair with B is possible since B is present in some soap. Boron is also present in some cleaners, cements, ceramics and glass.

CADMIUM (Cd)

Hair Cadmium (Cd) levels provide an excellent indication of body burden. Moderately high Cd levels may be associated with hypertension, while very severe Cd toxicity may cause hypotension. Cd adversely affects the kidneys, lungs, testes, arterial walls, and bones and interferes with many enzymatic reactions. Chronic Cd excess can lead to microcytic, hypochromic anaemia and proteinuria, and functional zinc deficiency. Cd excess is also commonly associated with fatigue, weight loss, osteomalacia, and lumbar pain.

Cadmium absorption is reduced by zinc, calcium, and selenium. Cigarette smoking significantly increases Cd intake. Refined carbohydrates have very little zinc in relation to the Cd.

If hair zinc is not abnormal, external contamination may have caused the elevated hair Cd level. Exogenous contamination may come from permanent solutions, dyes, bleach, and some hair sprays.

Hair analysis is useful for evaluating cadmium in smoker and non-smoker populations of industrially non-exposed urban and rural areas.⁵¹ Smoking itself causes significant elevation of toxic element levels in hair, particularly cadmium, lead, and nickel.⁵² The urine level of cadmium is also a good measure of body stores.⁵³ Under most circumstances, measurement of urine levels is a clinically useful technique. Once the renal threshold has been exceeded, however, urine levels become less trustworthy.⁵⁴

Cadmium exposure has been associated with hypertension, and studies show that hair levels of hypertensives are higher than controls.⁵⁵ Cadmium appears to inhibit sulphhydryl-containing enzymes so that relatively low doses depress levels of norepinephrine, serotonin, and acetylcholine.⁵⁷

Kidneys are main target organ. Detox agents e.g. EDTA/DMSA can increase urinary excretion. Presence of mercury and/or lead dramatically increases toxicity.

SOURCES: include plants which readily assimilate it, e.g. veges especially potatoes and leafy greens; contaminated soils and sewerage sludge. Others include cadmium-plated hardware (nuts/bolts), electroplating, Ni-Cd batteries, some photovoltaic cells, brazes, solders, pigments (paints, ink, glazes), cigarettes, photographic and engraving chemicals.

LEAD (Pb)

Lead is the best-known example of problems associated with chronic low-level toxic element exposure.

Mainly excreted via urine and less via bile. Small amounts via sweat, hair and nails. Non-provoked urine Pb levels can fluctuate according to dietary and physiological factors and doesn't necessarily reflect body burden. Provoked levels can indicate excess Pb in body. Pb toxicity is increased with presence of Hg or Cd.

Most uptake via contaminated food and water, inhalation of Pb dust, transdermal absorption.

Primarily deposits in aorta, liver, kidneys, adrenal, thyroid, bones and teeth. Can reduce Vitamin D synthesis, neuropathy and hypertension.

Lead has neurotoxic and nephrotoxic effects in humans as well as interfering with heme biosynthesis. Pb may also affect the body's ability to utilize the essential elements calcium, magnesium, and zinc.

At moderate levels of body burden, Pb may have adverse effects on memory, cognitive function, nerve conduction, and metabolism of vitamin D. Children with hair Pb levels greater than 1 µg/g have been reported to have a higher incidence of hyperactivity than those with less than 1 µg/g. Children with hair Pb levels above 3 µg/g have been reported to have more learning problems than those with less than 3 µg/g. Detoxification therapy by means of chelation results in transient increases in hair lead. Eventually, the hair Pb level will normalize after detoxification is complete.



Occupational and environmental exposure is the common causes of exposure. Lead reduces the body's ability to utilize calcium, magnesium, zinc, iron, and other important nutrients. It is a known cause of anaemia. Children are easily affected by lead exposure.

Symptoms associated with excess Pb are somewhat nonspecific, but include: anaemia, headaches, fatigue, weight loss, cognitive dysfunction and decreased coordination.

SOURCES: include leaded petrol, canned goods, lead paint, newsprint, tobacco smoke, air pollution and contaminated water. old lead-pigment paints, batteries, industrial smelting and alloying, some types of solders, some toys and products from China, glazes on (foreign) ceramics, leaded (antiknock compound) fuels, bullets and fishing sinkers, artist paints with lead pigments, and leaded joints in some municipal water systems. Deficiency of zinc, calcium or iron may increase lead uptake. Transdermal exposure is slight. Inhalation has decreased significantly with almost universal use of non-leaded automobile fuel.

Although historic **uses of lead** (house paint, anti-knock gasoline additives, and soldered joints in water systems) have been discontinued, old building materials, paint chips, plumbing and the environment may contain residual amounts from these sources. Other sources include batteries in cars, trucks, boats, and power backup systems, art supplies, coloured glass kits, bullets, fishing sinkers, balance weights, radiation shields, bearing alloys, some ceramic glazes or pigments and sewage sludge.

Sources of exposure to Pb include: welding, old leaded paint (chips/dust), drinking water, some fertilizers, industrial pollution, lead-glazed pottery, and newsprint.

Elevated levels of Pb in head hair can be an artefact of hair darkening agents or dyes, e.g. lead acetate in Grecian 2000. Although these agents can cause exogenous contamination, some transdermal absorption does occur.

Consider vitamin C, sulphur amino acids and other oral chelating agents can increase urinary excretion.

LITHIUM (Li)

Hair appears to be a reliable indicator of lithium status. A direct association was observed between hair lithium and cobalt concentrations, suggesting a role for lithium in the transport and distribution of vitamin B12. Hair lithium levels increase in response to supplementation, but lithium is a marker only in some subjects.⁶⁹⁻⁷¹ It may be that certain behavioural defects, depression, and learning disabilities are caused, or aggravated, by low nutritional lithium intake coupled with marginal deficiencies of B12 and folic acid, whose transport is also modulated by lithium.

This is trace element required to modulate nerve transmission throughout the central nervous system. It generally has a calming effect. Lithium deficiency is very common and under-recognized and may be easily corrected by low-dose supplementary lithium orotate. This should not be confused with the high-dose pharmacological lithium carbonate that is prescribed for patients with Bipolar Disorder.

Please note that, even if your hair lithium levels exceed the stated reference range, this may not necessarily be abnormal. The optimal level of lithium in hair has not been clinically determined and may be as high as 0.1 – 0.3 mg%. Results in excess of 0.3 mg%, that cannot be explained by the use of either supplementary or prescription lithium, must be further investigated.

MERCURY (Hg)

At high concentrations, mercury causes liver and kidney damage and neurological symptoms.³⁵ Interest has grown in the possible ill health effects of mercury liberated from dental amalgam fillings as well as the increased consumption of fish contaminated with mercury.^{36,37} Hair is used as an index of internal accumulation of mercury provided it was not externally contaminated by exposure to mercury vapor.^{38,39}

There is intriguing research correlating increased hair mercury levels with certain disease conditions. For instance, chronic mercury ingestion may be related to cardiovascular disease. Recent data suggests that a high intake of mercury from non-fatty freshwater fish and the consequent accumulation of mercury in the body are associated with an increased incidence of acute myocardial infarction, as well as death from cardiovascular disease in general.⁴⁰

Collaborative evidence for this finding comes from a Finnish case-controlled study in which higher numbers of dental fillings in individuals was associated with increased occurrence of acute myocardial infarction.⁴¹

There is additional support that mercury from dental fillings results in increased body burden: scalp hair of British dentists and dental hygienists were 2-3 times higher in mercury than those of the support staff.⁴² A study of dentists, dental nurses, and assistants showed the average elevation of urine mercury levels were significantly related to the number of amalgam fillings the subjects had.⁴³

Chronic mercury ingestion may be a risk factor for cardiovascular disease. This increased risk has been proposed to be due to the promotion of lipid peroxidation of mercury. Elevated levels of mercury in hair have been associated with inducement of autoimmune disease.

Hair is the best measure of seafood sources of mercury.

Urine (challenge test) is the best measure of dental amalgam sourced mercury.

For elemental and inorganic Hg, biliary excretion predominates, but urinary excretion increases as degree of exposure and burden increases. Inorganic Hg concentrates mainly in kidneys. For organic Hg (methyl, ethyl, alkyl), biliary excretion predominates with some urinary excretion.

Urinary excretion of Hg is increased following DMSA/DMPS; EDTA results in minor urinary excretion.



Low levels can cause immune dysregulation; lymphocyte inhibition and dysfunction, immunosuppression and autoimmune conditions. Hg can induce cytotoxicity, oxidative stress (via loss of GSH) and increased secretion of beta-amyloid in neuronal cells – linking it to Alzheimer's.

Inside cells Hg binds to ALA, GSH, and CoA and can impair pyruvate metabolism and citric acid cycle function leading to impaired energy production. Chronic Hg exposure may produce increased excitability and tremor, memory loss, insomnia, lassitude, anorexia, weight loss.

Early signs of mercury contamination include: decreased senses of touch, hearing, vision and taste, metallic taste in mouth, fatigue or lack of physical endurance, and increased salivation. Symptoms may progress with moderate or chronic exposure to include: anorexia, numbness and paresthesias, headaches, hypertension, irritability and excitability, and immune suppression, possibly immune dysregulation.

Methyl mercury accumulates in aquatic animals and fish and is concentrated up the food chain reaching high concentrations in large fish and predatory birds.

SOURCES: Fish, shellfish and edible seaweed are dietary sources. Other sources include pre-1990 paint, antifungal/antifouling paints (marine), some fluorescent tubes, Thimerosal in some vaccinations, batteries, electrical switches, thermostats, thermometers, barometers. Dental amalgams are primary source – Hg vapour from fillings can be absorbed and excreted in urine but is less than found in food sources. Increased Hg in environment e.g. soils, sediments, water. Coal-fired power plants emit over 30%, other industrial sources include cement plants, pulp/paper mills, municipal and medical waste incinerators.

NICKEL (Ni)

Given nickel's ability to cause contact dermatitis, and its observed perturbation of immunoglobulin levels (IgG, IgA, IgM), elevated hair levels may serve as an indicator of possible immune dysfunction, as well as a potentially useful marker of cardiovascular problems.

Up to 90% of absorbed nickel is excreted via urine; sweat and bile/faeces account for the remainder. Administration of sulphhydryl agents (D-Penicillamine, DMPS) can mobilize sequestered nickel and increase its concentration in urine.

SOURCES: most absorbed nickel is of dietary origin; hydrogenated oils, cocoa and chocolate are notable sources. Batteries (nickel-cadmium), non-precious dental materials, costume jewellery, and nickel-plated hardware are other sources that may be of concern in nickel dermatitis. Smoke, cigarette smoking and food are major sources of nickel exposure.

PALLADIUM (Pd)

Palladium is alloyed and used in jewellery. White gold is an alloy of gold de-colorized by the addition of palladium. The metal is used in watch making, in making surgical instruments and electrical contacts. Like mercury, it has been used in dental fillings since 1986.

All palladium compounds should be regarded as highly toxic and carcinogenic. Palladium chloride is harmful if swallowed, inhaled or absorbed through the skin. It causes bone marrow, liver and kidney damage in laboratory animals.

The statistical evaluation of so-called non-exposed patients indicates that EDTA may be considered the chelator of choice.

PLATINUM (Pt)

Most exposure is via industrial inhalation or cisplatin administration. Elevated urine platinum after administration of DMSA/DMPS.

SOURCES: Include cisplatin, catalytic converters on gasoline engines (cars, trucks), electroplating operations, catalyst production and catalytic equipment in the chemical process industries and petroleum refineries, precious dental materials, jewellery, smelting and refining of nickel and copper, purification of gold ores, and electronic parts such as thermocouples, resistance wires and contacts.

SILVER (Ag)

Silver interacts metabolically with copper and selenium. Animal studies by Whanger and Weswigh established it as a dietary copper antagonist. Hill et al showed that silver accentuated signs of copper deficiency. Silver has been shown to alleviate selenium toxicity.

Skin contact and breathing of silver-containing dust can occur in industrial environments involved in silver production or utilization. While a high silver intake markedly depresses copper tissue levels, which suggests a depressed copper absorption, studies indicate that total copper excretion was unaffected by silver treatment.

Argyria, a characteristic and irreversible gray or blue-gray discoloration of the skin and mucous membranes, has been observed in individuals that have ingested both metallic silver and silver compounds in small doses over periods of months or years.

DMPS is the preferred chelator.



STRONTIUM (Sr)

Approximately 99% of absorbed strontium is stored in bone tissue, where it plays a vital role in the stabilization of bone structure. Strontium is excreted in the urine and has been reported to correlate with tissue levels.

Deficiencies of strontium may contribute to the development of osteoporosis, whilst potentially toxic levels of strontium may invariably be seen with the use of Strontium ranelate for the pharmaceutical treatment of this condition.

THALLIUM (Tl)

This highly toxic element can enter the body via ingestion, inhalation or skin contact. Most thallium is excreted via urine.

Inside cells, thallium enters the mitochondria where it inhibits cellular respiration by blocking oxidative phosphorylation. The principal effect of such poisoning is marked fatigue. Indeed, some patients with Chronic Fatigue Syndrome may in fact be suffering from thallium (and/or arsenic) toxicities.

SOURCES: Include manufacture of electronic component parts (switches and contact materials that function at low temperatures), photoelectric cells, semiconductors, radiation counters, pigments, luminous paints, coloured glass and artificial gems. Thallium may also emit from coal-fired power stations, depending on the source of the coal, together with the combustion of both petrol & diesel fuels, as thallium is often included in the formulation of fuel additives. Hence, people who work or live in close proximity to main roads or freeways may be at increased risk of thallium intoxication. A low dietary potassium intake, e.g. by a lack of fresh fruits that are rich in potassium, seems to predispose to this condition.

Most is excreted via urine. Administration of K may increase urinary excretion. As Thallium has affinity for sulphhydryl compounds, cysteine and/or GSH may spread contamination.

Literature is lacking re use of DMPS/DMSA.

TIN (Sn)

Organic tin can be absorbed dermally. Elemental tin is poorly absorbed from GIT. Absorbed inorganic tins are at least 85% excreted via urine. Administration of -SH detoxification agents lessens toxic effect and increases urine and bile excretion.

The repeated ingestion of elemental tin may result in its accumulation in bodily tissues. Tin is excreted by the kidneys in both its elemental and organic forms. Some people are unable to excrete tin effectively and this may show as elevated tin levels on a hair analysis. Thus, hair is an excellent tissue for assessing tin levels in the body.

High tin levels influence the metabolism of several minerals including calcium, zinc and ALP activity in liver.

Therefore, consider testing calcium and zinc levels.

SOURCES: Most encountered tin sources are elemental or inorganic salts for which toxicity is low: tin from tin cans with damaged (or absent) polymer coatings, stannous fluoride in toothpaste, food and drinking water exposed to bronze, brass or tin-containing solders. Anti-corrosion electroplating of metals, pewter (tin, antimony and copper), cosmetics, dental amalgams, glazes and pigments and some plastics (e.g. PVC) are other potential sources of inorganic tin.

Of more concern environmentally are the potential sources of organic or organo-inorganic tins. These include: rodent poisons, fungicides, marine antifouling paints and coatings, wood preservatives, herbicide manufacture and acaricides (mite & tick sprays used agriculturally on almonds, hops, apples, citrus, peaches, pears, nectarines and plums.) The marine sediments of boat, harbours & marinas accumulate these organic tins with devastating effects to micro-organisms and marine fauna.

TUNGSTEN (W)

Tungsten is chemically related to both chromium and molybdenum.

Elevated levels in hair are relatively uncommon. Any elevation should be confirmed by a 24 hour urinary Pre & Post Challenge test by means of DMSA or EDTA

SOURCES: Most exposures to this metal occur as the result of industrial or mining operations. TIG welding and metal cutting lead to the release of microscopic particles of tungsten that can then be readily inhaled with a resulting pulmonary alveolitis, with or without fibrosis. Cemented tungsten carbide, used to make grinding wheels, is the most common tungsten compound. Other tungsten compounds are used in ceramic pigments, as fire retardant coatings for fabrics and as fade-resistant fabric dyes. Tungsten and its alloys are used as light bulb filaments, as the part of x-ray cathode tubes, as a catalyst to speed up chemical reactions, as a component of steel in high-speed tools & tungsten carbide drill bits, in turbine blades and welding electrodes.

URANIUM (U)

Uranium has both radiochemical and toxic chemical hazards.

Excretion is mostly by urine, but the biological residence time is long with measurable uranium in the urine for up to 18 months after an acute exposure. Urine levels may reflect body burden.

Fatigue, a commonly-observed symptom of chronic, low-level uranium exposure, may be due to impairment of cellular energy metabolism, by disruption of high energy phosphate bonds.

The uranyl ion also bonds to phosphates in bone, thereby displacing calcium. Uranium bound to bicarbonate may cause renal damage leading to renal failure.

SOURCES: Most common mode of exposure is by drinking water that has passed through rock strata, typically of igneous origin. Ground water aquifers may become contaminated with microscopic particles of uranium and regular use of well water from these aquifers has been known to cause uranium poisoning.

Inhaled uranium from rock dust as a result of mining activities is another major source, whilst exposure to dust particles containing depleted uranium is also a recognised cause in war zones. Exposure from leakages at nuclear power plants & waste sites are additionally potential sources of exposure.

MINERALS



Nutritional elements serve a variety of metabolic functions. As structural components, they are part of the skeletal system, vitamin B12, haemoglobin, and thyroid hormone. As cellular regulators, they are involved in nerve transmission, maintenance of cell membrane permeability, and regulation of osmotic pressure, water balance, and acid-base equilibrium.

Additionally, nutritional elements serve as cofactors in a wide array of enzymatic reactions. Various circumstances may result in inadequate status of nutritional elements: insufficient intake, poor digestion, poor absorption, and competitive inhibition by toxic elements. Maintaining optimal digestive function can therefore be a critically important aspect in elemental nutrition. (Comprehensive Digestive Stool Analysis testing may offer significant benefit in improving digestion.)

Research into the relationship of nutrient hair mineral status and behavioural disorders, cardiovascular disease, and cancer offer exciting new and emerging possibilities. Firm correlations between element concentrations in hair and the abnormal physiology for various diseases are as yet incompletely understood. However, some significant relationships have been found.

CALCIUM (Ca)

This nutrient mineral has been studied extensively in the hair. Perhaps the most useful of the hair calcium correlations is its link to cardiovascular disease.

Hypocalcaemia causes elevated urinary Ca except when glomerular function is impaired. Use of Ca supplements, excessive use of Vitamin A or D may elevate urinary Ca. Changes in dietary habits such as abrupt decrease in protein intake can cause transient urinary Ca loss. Other reasons include EDTA, citric acid, corticosteroid use, oestrogen therapy.



CHROMIUM (Cr)

Is an essential trace element that is required for the sugar and fat metabolism and is part of the glucose tolerance factor. Low chromium levels are often found in the elderly and pregnant women whose diet is rich in sugars and refined foods. Alcoholics and 'sugarholics' are often chromium deficient. Deficiency conditions are atherosclerotic plaque, elevated LDL cholesterol levels, increased insulin need, impaired glucose tolerance and a reduced stress response. Deficiency causes are diets rich in highly processed foods, alcoholism, malabsorption and insufficient intake of B vitamins.

Sources include wholegrains, brewers yeast, wheat germ, meat, cheeses.

Chromium is a naturally occurring element found in rocks, animals, plants, soil, and in volcanic dust and gases. Chromium is present in the environment in several different forms. The most common forms are chromium (III), and chromium (VI), which enter into air, water and soil. Chromium (VI) at high levels can damage the nose and can cause cancer. Chromium has been found at 1,036 of the 1,591 National Priority List sites identified by the Environmental Protection Agency (EPA).

Chromium (III) occurs naturally in the environment, is an essential nutrient and considered non-toxic. Chromium III helps the body use sugar, protein, and fat.

Chromium (VI) and chromium (III) are used for chrome plating, dyes and pigments, leather tanning, and wood preserving.

Chromium 51 is a radioactive isotope of chromium used to label red blood cells for measurement of mass or volume, survival time, and sequestration studies, for the diagnosis of gastrointestinal bleeding, and to label platelets to study their survival.

COBALT (Co)

Is part of the vitamin B12 molecule and is necessary for B12 activity and function. Cobalt which is mainly stored in the liver activates numerous enzymes and is excreted in bile. High levels increase the toxic effect of selenium and suppress iron absorption.

Elevated cobalt on a hair test is a sensitive indicator of selenium deficiency. Conversely, low hair cobalt may reflect a B12 deficiency that may only be detected on a urinary methylmalonic acid (MMA) test via ORGANIC ACIDS – METHYLATION COFACTORS urine test.

Consider B vitamins and enzymes to support liver function. Fatty acids stimulate the cobalt excretion via bile.

SOURCES: Sources include animal products including meats, fish, cheese, yeast extracts. Strict vegetarians (vegans) and those who lack intrinsic factor risk vitamin B12 and cobalt deficiency.

Toxic accumulation of Co can occur via inhalation of metal dusts and fumes in industrial environments, hip replacements. Possible sources include contaminated food and drink, smoking. Cobalt blue is used in paints and pottery glazes. It is used as catalyst in chemical process industries, for manufacture of polyesters and phenolics. It is also released into the environment by burning coal and heavy fuel oil.

Use of GSH, NAC, DPTA increase urinary excretion; while EDTA and DMSA increase biliary/faecal excretion (possibly lowering urine levels).

COPPER (Cu)

Small amount excreted via urine but most normally excreted via bile/faeces. Biliary insufficiency, cirrhosis or liver disease with cholestasis can decrease biliary Cu excretion and increase urinary levels. D-penicillamine (Cuprimine) greatly increases urinary Cu. In Wilson's Disease, urinary Cu is increased by 3-25 times.

Conditions that may result from excess Cu include abnormal renal transport, glucosuria, hyperaminoaciduria, tremor, dementia, haemolytic anaemia, jaundice, hypotension, reduced blood level of Vitamin A, molybdenum deficiency.

Unbound copper is known to be an even more reactive pro-oxidant than iron, especially in the presence of strong reducing agents such as ascorbate or homocysteine. High levels of copper can induce oxidative damage. Small amounts are required for CuZnSOD and ceruloplasmin.

Toxic levels cause nausea, behaviour problems, vomiting and diarrhoea. Elevated levels of copper often reflect exposure to swimming pool water treated with algaecide. Occasionally elevated copper occurs from hair treatments, perm, dye or bleach.

Brain and liver are the main storage sites, while the liver is the main organ for excretion.

High hair levels of copper suggest elevated liver storage and the body's inability to complex copper with amino acids such as histidine, threonine and glutamine. This insufficient complexing prevents the transport of copper between the liver and various peripheral tissues. High hair copper levels have been linked to headache, dizziness, depression and mood disorder, migraines, an increased sensitivity to pain, collagen disease, leukaemias and other malignancies.

The high level of Copper (Cu) in hair may be indicative of excess Cu in the body. However, it is important first to rule out exogenous contamination sources: permanent solutions, dyes, bleaches, swimming pool/hot tub water, and washing hair in acidic water carried through Cu pipes. In the case of contamination from hair preparations, other elements (aluminium, silver, nickel, titanium) are usually also elevated.

High copper levels increase the toxic effect of selenium and suppress iron absorption. High copper levels are often accompanied by low zinc levels.



Medical conditions that may be associated with excess Cu include: biliary obstruction (reduced ability to excrete Cu), liver disease (hepatitis or cirrhosis), and renal dysfunction. Symptoms associated with excess Cu accumulation are muscle and joint pain, depression, irritability, tremor, haemolytic anaemia, learning disabilities, and behavioural disorders.

SOURCES: Dietary include shellfish, nuts, organ meats, eggs, cocoa, chocolate. Environmental sources such as Chlorination of water has doubled Cu content. Cu is in electrical systems, brass and bronze alloys, antifouling paints, pigments, algaecides, fungicides. Most excess Cu intake is from drinking acidic water via copper pipes.

Sources of excessive Cu include contaminated food or drinking water, excessive Cu supplementation, and occupational or environmental exposures. Insufficient intake of competitively absorbed elements such as zinc or molybdenum can lead to, or worsen Cu excess.

Consider evaluating iron, manganese, zinc and molybdenum levels. Vitamin C increase copper excretion especially when used with amino acids and vitamin B6.

IRON (Fe)

Is essential for he oxygen transport and utilization. Iron is regulated in the body primarily by absorption rather than by excretion. Gastrointestinal function is important in controlled totally body iron. Transferrin is the transport protein for iron in blood. The most common sign of deficiency is anaemia. Predisposing factors to iron deficiency may be excessive intake of copper, manganese, zinc, carbonates, oxalates, phosphates, phytates, antibiotics, coffee.

Excess ingested Fe is excreted in stool, bile and urine. Iron storage problems evaluated with serum ferritin. Fe overload can cause liver and pancreas disturbances, lipid peroxidation, endocrine effects and CVD.

While iron excretion occurs in the urine, primary elimination is via stool. Deficient hair iron may be a manifestation of iron deficiency. If low hair iron levels are present, further evaluation of potential iron deficiency is recommended. This involves a simple blood test for iron studies (iron, transferrin, ferritin and transferrin saturation).

Desferrioxamine is chelating agent of choice for acute Fe toxicity.

MAGNESIUM (Mg)

There are many studies involving this macro mineral nutrient in the hair, and levels do increase with even fairly low amounts of supplementation.⁸⁶

Low Mg may be due to fasting or poor diet. Radiation enteritis or GIT surgery can result in poor Mg uptake and therefore low urinary levels. Low urinary Mg is often due to GIT disorders including prolonged diarrhoea, celiac, hypochlorhydria, and pancreatic insufficiency.

Other pathology leading to low urinary Mg include renal insufficiency, alkalosis, biliary insufficiency, occasional hypothyroidism and Addison's.

Possible dietary or nutritional reasons include inappropriate supplementation of magnesium, supplementation of magnesium when the amino acid taurine is deficient or excessively excreted in urine (taurine is magnesium-sparing), excessive supplementation of calcium, and alcoholism. Serum levels of magnesium are homeostatically controlled by the urinary excretion of excess magnesium. However, electrolyte imbalances can provoke urinary loss of magnesium. Loss of magnesium is expected with administration of thiazide diuretics, corticosteroids, and certain other drugs or medications.

Low hair Magnesium (Mg) levels may be indicative of Mg deficiency, but this has not been unequivocally demonstrated. When hair Mg is low, dietary intake and malabsorption should be considered. Mg is an essential element/electrolyte that is necessary for the activity of many important enzymes. Low hair Mg may or may not be associated with physiological dysfunction.

Causes of Mg deficiency include: consumption of a 'junk food' diet or Mg-deficient foods, intestinal malabsorption, hypocalcaemia with decreased Mg retention, chemical toxicity with renal wasting, alcoholism, alkalosis, prolonged diarrhoea/laxative abuse, and iatrogenic causes (digoxin therapy, occasionally from oral contraceptives, hypercalcemic drugs, gentamicin, neomycin).

Symptoms of Mg deficiency include: muscle twitching, cramps, tremor or muscle spasms, paraesthesia, and mental depression. Low Mg status is associated with arrhythmias and increased cardiovascular risk.

Mg status can be difficult to assess; whole blood and red cell levels are more indicative than serum/plasma levels. Amino acid analysis can be helpful in showing rate-limiting steps that are Mg-dependent such as phosphorylation. Taurine deficiency is often associated with urinary loss of Mg.

MANGANESE (Mn)

At physiological levels, manganese is an essential element that functions as an activator of certain enzymes. In excess, this element can accumulate in cell mitochondria in the pancreas, liver, kidneys, and intestines, and also deposits in bone and in the brain. In the nervous system, manganese decreases dopamine and its function. Emotional instability, compulsive and aberrant behaviours are also attributed to excess manganese.

Over 90% of ingested Mn is excreted via bile/faeces. Only 1% is excreted via urine.

High hair manganese has also recently been associated with violent behaviour, possibly because of its link to dopamine and serotonin depletion.⁷⁷ Biliary dysfunction, chelating agents (EDTA and D-penicillamine) can cause increase urinary Mn. Excess amounts will accumulate in pancreas, liver, kidney and intestine mitochondria. Mn decreased dopamine and function. Mn used in batteries, electronic components, matches, welding, glazes, dyes and pigments.



Biliary dysfunction and administration of chelating agents, especially EDTA and, to a lesser extent, D-penicillamine, can result in increased or elevated urine manganese.

The toxic accumulation of manganese – usually occupationally related – is now known to cause a Parkinsonian syndrome.

Low Mn may be due to highly refined diet, intestinal malabsorption, and renal insufficiency.

SOURCES: Include the manufacture of steel and bronze alloys, batteries, electronic components, water conditioning systems (potassium permanganate for high-iron water), matches, welding rods, glazes, dyes and pigments.

MOLYBDENUM (Mo)

Mo is an essential trace element that is an activator of specific enzymes such as: xanthine oxidase (catalyses formation of uric acid), sulphite oxidase (catalyses oxidation of sulphite to sulphate), and aldehyde dehydrogenase (catalyses oxidation of aldehydes). Possible effects or symptoms consistent with Mo deficiency are: subnormal uric acid in blood and urine, sensitivity or reactivity to sulphites, protein intolerance (specifically to sulphur-bearing amino acids), and sensitivity or reactivity to aldehydes.

Mo deficiency has been linked to gout. Low levels in heavy meat eaters reflect digestive disorder, the need for digestive enzymes and dietary changes. Such patients should avoid pork, beef, wholegrain and rather eat poultry, fish and other lighter proteins. Vegetarians should either add some meat to their diet or take molybdenum with B vitamins which aid the absorption of molybdenum. Dietary molybdenum is readily absorbed by the intestine and is excreted in the urine and bile.

Excess intake of copper, zinc and sulphates can depress Mo uptake, causing disturbances in the uric acid cycle.

Sources include wholegrains, legumes, leafy veges and organ meats.

Deficiency of Molybdenum is one of the commonest abnormalities seen on a hair mineral analysis. Although a low result may indicate dietary deficiency or intestinal malabsorption, it may simply reflect the fact that Australian soils are often deficient in this mineral and hence our food supply is likewise deficient. Molybdenum competes with copper for absorption; therefore if copper levels or copper stores are high, molybdenum levels are invariably low. This is reflected in the copper/molybdenum ratio, which is normally 650:1.

Levels above 850:1 usually indicate copper accumulation in extracellular tissues. The DMSA Challenge test will provide confirmation if this is occurring in your case.

Low Molybdenum (Mo) in hair is a possible indication of Mo deficiency. Hair is very rarely contaminated with exogenous Mo.

True Mo deficiency is uncommon but may result from: a poor-quality diet, gastrointestinal dysfunctions, or tungsten exposure. Tungsten can be a powerful antagonist of Mo retention in the body. Copper overload can also reduce Mo retention.

Because normal blood and blood cell Mo levels are very low (a few parts per billion), blood measurement is not an appropriate tissue for confirmation of subnormal molybdenum.

SODIUM (Na)

The concentration of sodium is carefully regulated within a well-defined range in both situations. Serum (blood) concentrations are primarily maintained by the actions of the adrenal hormone aldosterone and to a lesser extent cortisol and indirectly by the actions of other hormones such as ACTH and Angiotensin II.

The measurement of sodium in hair reflects the transfer of salt (NaCl) across the germinal basement membrane of the growing hair follicle. Sodium and potassium are trapped by salt bonds within the strands of keratin.

High hair sodium levels generally reflect periods of stress in a person's life, or may result from a high salt diet. Conversely, low sodium levels are often associated with adrenal fatigue, depression or a low sodium diet. Elevated sodium and potassium levels are frequently found together with low levels of calcium and magnesium in hair.

SELENIUM (Se)

There are clear relationships between selenium levels in blood, urine and hair samples. Hair selenium reflects dietary intake over time.⁶⁰ Hair selenium has been observed to increase with supplementation.⁶⁴

Low hair selenium may be a useful marker to explore in the relationship between nutrients and thyroid function. Low hair selenium levels may reflect a clinical worsening of the iodine deficiency state.⁶⁵

High urinary levels may result from overuse of Se supplements or after admin of D-penicillamine, DMSA, DMPS or the use of cysteine, NAC, penicillin antibiotics.

Low urinary Se can be secondary to dietary insufficiency. Diet of highly processed foods or foods grown in Se-deficient soils can lead to low Se. GIT dysfunction/malabsorption can lead to low Se. Renal insufficiency would result in elevated blood Se with low urinary Se.

Selenium salts may be toxic in large quantities, but trace amounts are essential for normal cellular function. It is a component of the enzymes glutathione peroxidase and thioredoxin reductase, which convert thyroid hormones from one form to another.



Frank selenium toxicity, called selenosis, is rare in humans. However, cases have been noted, primarily caused by contaminated soil. Symptoms of selenosis include garlic breath odor, thick brittle fingernails, dry brittle hair, red swollen skin of the hands and feet, and nervous system abnormalities including numbness, convulsions or paralysis.

Dietary sources: Garlic, onions, broccoli, Brazil nuts, brewer's yeast. Although yeast primarily contains selenomethionine, garlic, onions and broccoli are rich food sources of mixed selenocompounds. Of these, garlic contains the widest variety of selenocompounds, including low-dose inorganic selenium, in ratios that are safe and effective. However, selenium content of foods is highly variable, depending on soil content.

Most processed selenium is used in the electronics industry, but it is also used as a nutritional supplement, in the glass industry, as a component of pigments in plastics, paints, enamels, inks, and rubber; in the preparation of pharmaceuticals; as a nutritional feed additive for poultry and livestock; in pesticide formulations; in rubber production; as an ingredient in antidandruff shampoos; and as a constituent of fungicides. Radioactive selenium is used in diagnostic medicine.

Because the antioxidant role of selenium parallels that of vitamin E, the nutritional amount of selenium needed is inversely proportional to the dietary supply of vitamin E.

VANADIUM (V)

This is an essential trace mineral required for normal blood glucose and lipid control, as well as promoting glutathione-S-transferase activity – an essential component of the body's detoxification mechanisms. Only ~10% of dietary vanadium is absorbed, and is excreted primarily through the bile. However, excessive amounts of the vanadium salt, Vanadate (V^{5+}) may inhibit Na^+K^+ ATPase activity, thereby decreasing cellular energy utilization. This may result in fatigue or symptoms resembling hypothyroidism. Therefore, correct Vanadium balance, as measured in a hair analysis, is essential for good health.

ZINC (Zn)

A nutritionally essential element, zinc is needed as an activator for a number of enzymes in human tissues. Most absorbed zinc is normally disposed of via bile/faeces with a lesser and relatively constant degree in urine.

Zn toxicity may be due to displacement of Cu and inhibition of membrane ATPase, leading to disrupted Na and K transport. Hyperglycaemia, hypercholesterolemia, decreased haem synthesis and decreased serum albumin:globulin ratio can occur.

Zinc's toxicity, at high body burdens, may be due to displacement of copper and inhibition of membrane ATPases, leading to disrupted sodium and potassium ion transport.

Hyperglycaemia, hypercholesterolemia, decreased heme synthesis, and decreased albumin/globulin ratio in serum can occur. Zinc 'fume fever', from industrial exposure to freshly-formed zinc vapour, produces chills and fever, muscle weakness, fatigue and profuse sweating, usually for 24 to 48 hours following the exposure.

High tissue zinc levels are usually the result of excessive use of nutritional supplements, or industrial exposure, or less commonly from chelation therapy with EDTA or malignancy.

Habitual use of galvanised containers or the collection of rainwater from galvanised roofing is also uncommon causes of zinc excess.

The regular use of pyrithione-zinc based anti-dandruff shampoos may increase hair zinc levels by 30-40%. Avoid these shampoos if possible, during the 3-4 months prior to hair mineral analysis test.

RELATED TESTS

Test Name	Code	Analytes
Comprehensive Digestive Stool Analysis		
• CDSA Level 1	2003	<i>Level 1:</i> Macroscopic & Microscopic Description, Beneficial & Bacteria, Yeasts, Parasites (visual detection)
• CDSA Level 2	2004	<i>Level 2:</i> Macroscopic & Microscopic Description; Digestive, Absorption & Metabolic Markers; Beneficial & Other Bacteria; Yeasts; Parasites (visual detection)
• CDSA Level 3	2005	<i>Level 3:</i> Macroscopic & Microscopic Description; Digestive, Absorption & Metabolic Markers; Beneficial & Other Bacteria; Yeasts; Parasites (visual detection) & Sensitivities
• CDSA Level 3+	2006	<i>Level 3+:</i> Macroscopic & Microscopic Description; Digestive, Absorption & Metabolic Markers; Inflammation Markers; Tumour/Ulcer Markers; Beneficial & Other Bacteria; Yeasts; Parasites (visual detection) & Sensitivities
• CDSA Level 4	2007	<i>Level 4:</i> Macroscopic & Microscopic Description; Digestive, Absorption & Metabolic Markers; Beneficial & Other Bacteria; Yeasts; Parasites (visual & chemical EIA detection) & Sensitivities
• CDSA Level 4+	2008	<i>Level 4+:</i> Macroscopic & Microscopic Description; Digestive, Absorption & Metabolic Markers; Inflammation Markers; Tumour/Ulcer Markers; Beneficial & Other Bacteria; Yeasts; Parasites (visual & chemical EIA detection) & Sensitivities
• CDSA Level 5	2009	<i>Level 5:</i> Macroscopic & Microscopic Description; Beneficial & Other Bacteria; Yeasts; Parasites (visual & chemical EIA detection) & Sensitivities

RELATED TESTS Contd.

Test Name	Code	Analytes
Liver Detoxification Profile	4010	<i>Phase I Detoxification:</i> Caffeine Clearance; <i>Phase II Detoxification:</i> Measures of Glucuronidation, Glycation, Glutathionation & Sulphation
Organic Acids – Metabolic Panel (Urinary)	4016	Organic acids for Bacterial dysbiosis, Yeast & fungal dysbiosis, Citric acid cycle metabolites, Ketone/fatty acid metabolites, Cofactor sufficiency markers, Neurotransmitter metabolites (HVA, VMA, 5HIAA)
Organic Acids – Environmental Pollutant Panel (Urinary)	4014	<i>Environmental Pollutants:</i> Metabolites of Benzene, Xylene, Toluene, Trimethylbenzene, Styrene, Phthalate exposure
Organic Acids – Metabolic & Environmental Pollutant Panel (Urinary)	4017	Organic acids for Bacterial dysbiosis, Yeast & fungal dysbiosis, Citric acid cycle metabolites, Ketone/fatty acid metabolites, Cofactor sufficiency markers, Neurotransmitter metabolites AND Environmental Pollutants
Oxidative Damage Markers - Comprehensive	4019	8-OHdG, Allantoin, Carbonyl Proteins, MDA
Porphyrins (Urinary)	4024	Uroporphyrins I & III, 7-Carboxy Porphyrin, 6-Carboxy Porphyrin, 5-Carboxy Porphyrin, Precoproporphyrins, Coproporphyrins I & III
Amino Acids (Plasma)	5003	29 Amino Acids: Alanine, Arginine, Asparagine, Aspartic Acid, Citrulline, Cysteine, GABA, Glutamate, Glutamine, Glycine, Histidine, 1-Methyl Histidine, 3- Methyl Histidine, Isoleucine, Leucine, Lysine, Methionine, Ornithine, Phenylalanine, Proline, hydroxy Proline, Serine, Taurine, Threonine, Tryptophan, Tyrosine, Valine
Amino Acids (Urinary)	5004	29 Amino Acids: Alanine, Arginine, Asparagine, Aspartic Acid, Citrulline, Cysteine, GABA, Glutamate, Glutamine, Glycine, Histidine, 1-Methyl Histidine, 3- Methyl Histidine, Isoleucine, Leucine, Lysine, Methionine, Ornithine, Phenylalanine, Proline, hydroxy Proline, Serine, Taurine, Threonine, Tryptophan, Tyrosine, Valine
Iodine – Random (Urinary)	5016	Iodine (random)

POINTS OF INTEREST

Zinc increase Copper excretion

Wilson's disease is a hereditary disease associated with copper toxicity. Previously Wilson's disease was considered to be a disease of copper accumulation in the liver and other organs. 'De-coppering' with the chelating agent penicillamine was the treatment of choice, often with worsening of symptoms during the first weeks or months of therapy. It has since been revealed that the symptoms of Wilson's disease (e.g. involuntary shaking, easy bruising, drooling, difficulty swallowing) arise from copper intoxication (free copper in the blood) rather than copper accumulation in the liver and other organs. Free copper is toxic whereas accumulated copper and copper that is bound to ceruloplasmin and metallothionein is not. Therefore, treatment is aimed at the normalisation of free copper in the blood, rather than the removal of accumulated copper. This can be achieved safely and effectively with oral zinc therapy. Zinc induces metallothionein, a highly effective detoxification protein that binds copper. Oral zinc therapy leads to storage of metallothionein-bound copper in the mucosa of the GIT and to the excretion of copper via the faeces. New treatment guidelines advise against the use of chelating agents as initial treatment since they may aggravate copper intoxication.

Conclusion: Zinc therapy is considered to be a safe and effective first-line treatment for symptomatic Wilson's disease.

Hoogenraad TU. Paradigm shift in treatment of Wilson's disease: zinc therapy now treatment of choice.
Brain Dev 2006 Apr;28(3):141-146.

Antioxidants in Arsenic and Cadmium toxicity

Both cadmium and arsenic are ubiquitous in the environment. Exposure through food and water as well as occupation sources can contribute to symptoms of dermal lesions and anaemia for arsenic toxicity, or osteoporosis and osteomalacia from cadmium toxicity. The mechanisms and excretion of these heavy metals depend on the presence of antioxidants and thiols that aid arsenic methylation and both arsenic and cadmium metallothionein-binding. SAMe, ALA, GSH, Se, Zn, NAC, methionine, cysteine, Vit E and ascorbic acid have been found to be especially beneficial in mitigating the toxic effects of arsenic and cadmium. Several antioxidants including NAC, Zn, methionine and cysteine when used in conjunction with standard chelating agents can improve the mobilization and excretion of As and Cd. Se - forms insoluble complex with As; supports As methylation; forms inert complexes with Cd and decreases toxicity; reduces tissue retention of Cd. ALA - hepatic protection from Cd-induced damage; forms a complex with Cd for enhanced mobilization and excretion. Zn - increased metallothionein levels in the liver, kidneys and intestines.

Conclusion: Antioxidant nutrients may be used in As and Cd toxicity to limit metal induced free radical damage and to assist in the metabolism and excretion of the toxic metals. Patrick L. Toxic metals and antioxidants: part II. The role of antioxidants in arsenic and cadmium toxicity. Altern Med Rev 2003 May;8(2):106-128.

Wheat bran binds heavy metals

Dietary fibres are known to bind minerals such as calcium, Fe, and Zn. Wheat bran in particular has an exceptionally high binding potential. This study investigated the potential of wheat bran to bind the heavy metals Hg, Cd and Pb. Water soluble dietary fibres (WSDF) and water insoluble dietary fibre (WIDF) from wheat bran were shown to effectively bind the heavy metals separate pH conditions equivalent to those found in the stomach (pH 2.0) and small intestine (pH 7.0) with great binding potential exhibited at the higher pH level. The presence of amino acids, Ca, Fe and Zn slightly affected the binding capacity of wheat bran while the presence of copper significantly affected the binding capacity. Colon fermentation released part of the heavy metal ions from the dietary fibres.

Conclusion: Dietary fibres from wheat bran can effectively bind Hg, Cd, and Pb to prevent the body from being affected by their toxicity.

Ou S, Gao K, Li Y. An in vitro study of wheat bran binding capacity for Hg, Cd and Pb. J Agric Food Chem 1999 Nov;47(11):4714-4717.

Selenium's double actions in Mercury toxicity

There are limited human studies available looking at the relationship between selenium and Hg; most of the data regarding Se-Hg interaction has been obtained from in vivo studies. This human study investigated the Se and Hg content of serum and urine samples collected from 72 subjects; 37 were selected from an area of high exposure in China (including 25 miners) and 35 from a non-contaminated area. A relationship between Se and Hg was found in urine but not in serum. It is proposed that long term Hg exposure may affect Se concentration in human serum. Glutathione peroxidase activity increased in the serum of the exposed group. The Hg miners exhibited greater expression of both selenoprotein P and glutathione peroxidase as well as elevated Se concentrations in serum. Selenoprotein P was found to bind more Hg at higher Hg exposure concentrations.

Conclusion: Selenoproteins play two important roles in protecting against Hg toxicity. Firstly their highly reactive selenol group enables binding to Hg. Secondly, their antioxidant properties help eliminate Hg-induced reactive oxygen species.

Chen C, Yu H, Zhao J, Li B, Qu L, Liu S, et al. The roles of serum selenium and selenoproteins on mercury toxicity in environmental and occupational exposure. Environ Health Perspect 2006 Feb;114(2):297-301.

Toxic elements

Exposure to heavy metals or even excessive levels of certain essential minerals can lead to adverse health effects including neurotoxicity, genotoxicity and carcinogenicity. For example, elevated levels of iron, copper and zinc are related to neurodegenerative diseases such as Alzheimer's disease and Parkinson's disease. Certainly many other factors may contribute to



the aetiology and progression of these diseases, requiring a multifactorial treatment approach. Metal chelation is one option gaining increasing support in the scientific literature and driving new research into effective chelating agents.

Perez LG, Franz KJ. Minding metals: tailoring multifunctional chelating agents for neurodegenerative disease. *Dalton trans* 2010 Mar 7;39(9):2177-2187.

Modified citrus pectin enhances toxic metal excretion

Pilot trial examined effect of modified citrus pectin (MCP) on the urinary excretion of toxic elements in healthy subjects. Subjects were given 15g of MCP for 5 days. On the sixth and final day they ingested 20g. Toxic and essential elements were examined using 24 hour urine samples collected on the first and final days of the study. The first 24hour period of MCP infection saw a significant increase in urinary arsenic excretion (130%). The results from the final day of the study show a significant increase in cadmium excretion (150%) as well as a dramatic increase in lead excretion (%560%).

Conclusion: Oral administration of MCP at a dose of 15g/day over 5 days, followed by 20g on 6th day, dramatically increased the urinary output of toxic metals, namely arsenic, cadmium and lead, in healthy subjects with a 'normal' body load of metals.

Eliaz I, Hotchkiss AT, Fishman ML, Rode D. The effect of modified citrus pectin on urinary excretion of toxic elements. *Phytother Res* 2006 Oct;20(10):859-864.

Antioxidants support heavy metal chelation

The unifying factor in the toxic manifestations of heavy metal exposure (particularly of arsenic, lead, cadmium and mercury) is oxidative stress. Heavy metals also have an affinity for thiol groups in enzymes and proteins that are responsible for normal cellular defence mechanisms. Chelating agents such as EDTA (ethylenediamine tetraacetic acid), DMPS (2,3-dimercaptopropane-1-sulphonic acid) and DMSA (dimercaptosuccinic acid) are considered to be the best known treatment for metal poisoning but may have serious side effects. Supplementation with antioxidants alongside chelation therapy has proven to be a better treatment regime than chelation therapy alone. Certain antioxidants are especially beneficial in heavy metal toxicity: Vit E – protects membrane lipids; prevents lead-induced oxidation in proteins. Vit C – reduces lead absorption; increases lead excretion. Betacarotene – low serum levels predispose to arsenic-related ischaemic heart disease; spares glutathione in cadmium toxicity. ALA – chelates Fe, Zn, Cu, Hg and Cd; contributes thiol groups for the detoxification of Cd.

Conclusion: Further studies are recommended to ascertain optimal dosages and treatment duration of antioxidant therapy in heavy metal-induced oxidative stress.

Flora SJ, Mittal M, Mehta A. Heavy metal induced oxidative stress and its possible reversal by chelation therapy. *Indian J Med Res* 2008 Oct;128(4):501-523.

ARTICLES

Blood Mercury Levels in US Children and Women of Childbearing Age, 1999-2000

Susan E. Schober, PhD; Thomas H. Sinks, PhD; Robert L. Jones, PhD; P. Michael Bolger, PhD, DABT; Margaret McDowell, MPH, RD; John Osterloh, MD, MS; E.Spencer Garrett, MS; Richard A. Canady, PhD, DABT; Charles F. Dillon, MD, PhD; Yu Sun, PhD; Catherine B. Joseph, MSPH; Kathryn R. Mahaffey, PhD JAMA. 2003;289:1667-1674.

Blood and Hair Mercury Levels in Young Children and Women of Childbearing Age --- United States, 1999

Reported by Center for Food Safety and Applied Nutrition, Food and Drug Administration. US Environmental Protection Agency. National Energy Technology Laboratory, Dept of Energy. National Marine Fisheries Laboratory, National Oceanic and Atmospheric Administration. National Center for Health Statistics; National Center for Environmental Health, CDC.

Higher fish consumption in pregnancy was associated with better infant cognition, but higher mercury levels were associated with lower cognition. Women should continue to eat fish during pregnancy but choose varieties with lower mercury contamination.

Emily Oken, Robert O. Wright, Ken P. Kleinman, David Bellinger, Chitra J. Amarasiriwardena, Howard Hu, Janet W. Rich-Edwards, and Matthew W. Gillman

Mercury and Selenium Concentrations in Maternal and Neonatal Scalp Hair: Relationship to Amalgam-Based Dental Treatment Received During Pregnancy

Razagui I.B-A.; Haswell S.J. Biological Trace Element Research, Volume 81, Number 1, July 2001 , pp. 1-19(19)

Comment: This is another study speaking against dental treatment or removal of amalgam during pregnancy. It also supports infant hair testing.

Hair Element Concentrations in Females in One Acid and One Alkaline Area in Southern Sweden

Ingegerd Rosborg, Bengt Nihlgard, and Lars Gerhardsson. AMBIO: A Journal of the Human Environment. Vol 32, Issue No7, Nov. 2003 pp. 440-446

Comment: Metal uptake is influenced by pH.



The U.S. Environmental Protection Agency concluded in a 1980 report that "human hair can be used effectively for the biological monitoring of the highest priority toxic metals - lead, cadmium, mercury and arsenic," and "For toxic exposure.. (testing) hair appears to be superior to (testing) blood and urine."

Comment: Hair mineral analysis reflects long term exposure. It can be used to evaluate the mercury status in women before pregnancy occurs, allowing for nutritional corrections. To further promote an understanding it should be noted that during the digestion (ashing) of hair, all organic mercury is broken down. Hence, hair mineral analysis results reflect total inorganic mercury.

Maternal Fish Consumption, Hair Mercury, and Infant Cognition in a U.S. Cohort

Emily Oken, Robert O. Wright, Ken P. Kleinman, David Bellinger,- Chitra J. Amarasiriwardena, Howard Hu, Janet W. Rich-Edwards, and Matthew W. Gillman

Fish and other seafood may contain organic mercury but also beneficial nutrients such as n-3 polyunsaturated fatty acids. We endeavored to study whether maternal fish consumption during pregnancy harms or benefits fetal brain development. We examined associations of maternal fish intake during pregnancy and maternal hair mercury at delivery with infant cognition among 135 mother-infant pairs in Project Viva, a prospective U.S. pregnancy and child cohort study. We assessed infant cognition by the percent novelty preference on visual recognition memory (VRM) testing at 6 months of age. Mothers consumed an average of 1.2 fish servings per week during the second trimester. Mean maternal hair mercury was 0.55 ppm, with 10% of samples > 1.2 ppm. Mean VRM score was 59.8 (range, 10.9-92.5). After adjusting for participant characteristics using linear regression, higher fish intake was associated with higher infant cognition. This association strengthened after adjustment for hair mercury level: For each additional weekly fish serving, offspring VRM score was 4.0 points higher [95% confidence interval (CI), 1.3 to 6.7].

However, an increase of 1 ppm in mercury was associated with a decrement in VRM score of 7.5 (95% CI, -13.7 to -1.2) points. VRM scores were highest among infants of women who consumed > 2 weekly fish servings but had mercury levels \leq 1.2 ppm.

Higher fish consumption in pregnancy was associated with better infant cognition, but higher mercury levels were associated with lower cognition.

Women should continue to eat fish during pregnancy but choose varieties with lower mercury contamination.

Comment: Hair mineral analysis reflects long term exposure. It can be used to evaluate the mercury status in women before pregnancy occurs, allowing for nutritional corrections. To further promote an understanding it should be noted that during the digestion (ashing) of hair, all organic mercury is broken down. Hence, hair mineral analysis results reflect total inorganic mercury.

Mercury and Selenium Concentrations in Maternal and Neonatal Scalp Hair: Relationship to Amalgam-Based Dental Treatment Received During Pregnancy. Razagui I.B-A.; Haswell S.J. Biological Trace Element Research, Volume 81, Number 1, July 2001 , pp. 1-19(19)

Mercury and selenium concentrations were determined in scalp hair samples collected postpartum from 82 term pregnancy mothers and their neonates. Maternal mercury and selenium had median concentrations of 0.39 g/g (range 0.1-2.13 g/g) and 0.75 g/g (range 0.1-3.95 g/g), respectively, and corresponding median neonatal values were 0.24 g/g (range 0.1-1.93 g) and 0.52 g/g (range (0.1-3.0 g/ g). Amalgam-based restorative dental treatment received during pregnancy by 27 mothers (Group I) was associated with significantly higher mercury concentrations in their neonates ($p < 0.0001$) compared to those born to 55 mothers (Group II) whose most recent history of such dental treatment was dated to periods ranging between 1 and 12 yr prior to pregnancy. In the Group I mother/neonate pairs, amalgam removal and replacement in 10 cases was associated with significantly higher mercury concentrations compared to 17 cases of new amalgam emplacement. Selenium concentrations showed no significant intergroup differences. The data from this preliminary study suggest that amalgam-based dental treatment during pregnancy is associated with higher prenatal exposure to mercury, particularly in cases of amalgam removal and replacement. The ability of a peripheral biological tissue, such as hair, to elicit such marked differences in neonatal mercury concentrations provides supporting evidence of high fetal susceptibility to this form of mercury exposure.

Comment: This is another study speaking against dental treatment or removal of amalgam during pregnancy. It also supports infant hair testing.

...Free copper is toxic, whereas accumulated copper and copper that is bound to ceruloplasmin and metallothionein is not.

Therefore, treatment is aimed at normalization of free copper in the blood rather than the removal of accumulated copper. This can be achieved safely and effectively with oral zinc therapy....

Hoogenraad TU. Paradigm shift in treatment of Wilson's disease: zinc therapy now treatment of choice. Brain Dev 2006 Apr;28(3):141-146.

...The metabolism and excretion of these heavy metals depend on the presence of antioxidants and thiols that aid arsenic methylation and both arsenic and cadmium metallothionein-binding. SAMe, lipoic acid, glutathione, selenium, zinc, N-acetyl cysteine, methionine, cysteine, alpha tocopherol and ascorbic acid have been found to be especially beneficial in mitigating the toxic effects of arsenic and cadmium...

Patrick L. Toxic metals and antioxidants: part II. The role of antioxidants in arsenic and cadmium toxicity. Altern Med Rev 2003 May;8(2):106-128.



...Dietary fibres from wheat bran can effectively bind mercury, cadmium and lead to prevent the body from being affected by their toxicity...

Ou S, Gao K, Li Y. **An invitro study of wheat bran binding capacity for Hg, Cd and Pb.** J Agric Food Chem 1999 Nov;47(11):4714-4717.

...Curcumin and green tea polyphenols have been found to significantly affect brain iron levels

Curcumin, in addition to providing protection to the brain by direct binding and complex formation with toxic metals, could also afford protection against oxidative damage and inflammation.

The ability of curcumin to bind effectively with redox-active metals such as iron and copper and the enhanced radical scavenging efficacy of the curcumin-metal complexes further indicate a neuroprotective role.

Green tea polyphenols act as potent metal chelators binding to ions such as iron...

Hedge ML, Bharathi P, Suram A, Venugopal C, Jagannathan R, Poddar P, et al. **Challenges associated with metal chelation therapy in Alzheimer's disease.** J Alzheimers Dis 2009 Jul;17(3):457-468

...Chelating agents such as EDTA (ethylenediamine tetraacetic acid), DMPS (2,3-dimercaptopropane-1-sulphonic acid) and DMSA (dimercaptosuccinic acid) are considered to be the best known treatment for metal poisoning but many have serious side effects.

Supplementation with antioxidants alongside chelation therapy has proven to be a better treatment regimen than chelation therapy alone.

Certain antioxidants are especially beneficial in heavy metal toxicity as details below:

- Vitamin E – protects membrane lipids; prevents lead-induced oxidation of proteins.
- Vitamin C – reduces lead absorption; increases lead excretion.
- Betacarotene – low serum levels predispose to arsenic-related ischaemic heart disease; spares glutathione in cadmium toxicity.
- Alpha lipoic acid – chelates iron, zinc, copper, mercury and cadmium; contributes thiol groups for the detoxification of cadmium.

Further studies are recommended to ascertain optimal dosages and treatment duration of antioxidant therapy in heavy metal-induced oxidative stress...

Flora SJ, Mittal M, Mehta A. **Heavy metal induced oxidative stress and its possible reversal by chelation therapy.** Indian J Med Res 2008 Oct;128(4):501-523

...Selenoproteins play two important roles in protecting against mercury toxicity. Firstly, their highly reactive selenol group enables binding to mercury. Secondly their antioxidant properties help eliminate mercury-induced reactive oxygen species...

Chen C, Yu H, Zhao J, Li B, Qu L, Liu S, et al. **The roles of serum selenium and selenoproteins on mercury toxicity in environmental and occupational exposure.** Environ Health Perspect 2006 Feb;114(2):297-301.

K. Jomova, Z. Jenisova, M. Feszterova, S. Baros, J. Liska, D. Hudecova, C. J. Rhodes and M. Valko. **Arsenic: toxicity, oxidative stress and human disease.** Journal of Applied Toxicology. Published online in Wiley Online Library: 14 February 2011