PRACTITIONERS
INTERPRETATION GUIDE

IWDL

NUTRIENT AND TOXIC ELEMENTS

Measuring concentrations of elements in erythrocytes provides a look at view of well cellular levels are maintained because erythrocyte mineral levels reflect mineral status over a period of 3 months (the life cycle of an erythrocyte). Other specimens, like urine or plasma, are affected by daily dietary fluctuations. In the process of their formation in bone marrow, erythrocytes acquire nutrient elements, like zinc, according to the availability of each element.

The same may be said for the toxic elements, which means that high levels of these confirm chronic tissue penetration due to toxic exposure and inadequate detoxification mechanisms. All of the essential trace elements are dependent on stomach acid production for intestinal absorption.

Nutrient Elements	Abbreviation	Metabolic Association	Potential Intervention
Calcium	Ca	Myriad cell regulatory effects	See Calcium section
Chromium	Cr	Insulin target cell binding	200 - 400 µg/d
Copper	Cu	Detox pathways	3 - 5 mg/d
Magnesium	Mg	ATP energy transfer	200 - 400 mg/d
Manganese	Mn	Biosynthetic pathways	5 -15 mg
Potassium	K	Neuromuscular function	Fresh fruit & vegetables
Selenium	Se	Antioxidant protection	200 -1000 µg/d
Vanadium	V	Cholesterol, triglycerides	200 -1000 µg/d
Zinc	Zn	Cofactor for numerous enzymes	15 – 60 mg/d

Toxic Elements	Abbreviation	Preferential Accumulation Site	Potential Intervention
Aluminium	Al	Lung/Bone marrow	Magnesium/Phosphorus
Arsenic	As	CNS/GI	Chelation
Cadmium	Cd	Kidney	Zinc
Lead	Pb	Bone	Calcium
Mercury	Hg	PNS/CNS/Brain	Selenium

NUTRIENT ELEMENTS

Calcium (Ca)

Erythrocyte calcium is associated with the aetiology of heart disease and stroke. Total erythrocyte calcium is elevated in hypertension. Calcium levels in erythrocytes are not an accurate measure of calcium nutritional status because of the strong mechanisms maintaining the critical intracellular calcium concentrations, independent of the total body calcium regulation exerted by parathyroid hormone, vitamin D and other regulatory molecules.

Chromium (Cr)

Chromium accumulates primarily in spleen and heart tissue. The greatest number of chromium studies involves its role in glucose metabolism. Sugar metabolism was improved in over 80% of individuals with a slight glucose intolerance by using 200 µg/d chromium supplement. Chromium used in this way affects only those who are deficient in chromium. This nutrient impacts on sugar metabolism through its role in the uptake of insulin. Chromium also aids in lowering LDL cholesterol and raising HDL cholesterol.

Copper (Cu)

Most copper is concentrated in the liver, brain and hair, but is present in all other tissue. Most of the copper present in erythrocytes is bound to the enzyme superoxide dismutase (SOD), which protects the cells from oxidative damage. Dietary deficiency of copper is seen as low levels of erythrocyte copper and SOD, even in early stages of copper depletion. Impairment of function due to copper deficiency may result from reduced activities of the enzymes, ceruloplasmin, monoamine oxidase, lysyl oxidase in connective tissue and SOD. Loss of these biochemical functions can lead to anaemia, neural degeneration, lung and bone problems, CVD and accelerated ageing. In copper deficiencies, supplementation with 3 - 5 mg/d copper aspartate is helpful.

Chronically elevated plasma copper may result in elevation of erythrocyte copper levels as well, although the two specimens represent different copper utilisation. About 80% of erythrocyte copper is associated with SOD, while most plasma copper is bound to ceruloplasmin. Patients with Wilson's disease, an inherited copper – accumulating disease, show elevated erythrocyte copper resistant to copper-lowering treatments. In these cases, copper accumulates in liver and brain where it causes tissue degeneration, apparently due to the stimulation of protein and DNA oxidative damage.

Magnesium (Mg)

Magnesium serves as a cofactor in approximately three hundred enzyme systems, making this element a critically important nutrient for many bodily functions. Deficiency conditions can cause a wide variety of problems including hypertension, diabetes and PMS. Magnesium plays a vital role in normal cardiac function and deficiency has been increasingly associated with cardio-vascular disease. Magnesium deficiency in humans is rarely severe, although symptoms of marginal deficiency may be many and varied. Symptoms frequently associated with magnesium deficiency in humans are neuromuscular tremor and muscle spasms or twitches. The magnesium content of red blood cells is a good indicator of short-term magnesium status and low levels indicate nutritional deficiency. As the largest energy user, nervous tissue shows the earliest signs of deficiency, with the appearance of symptoms such as mental confusion, fatigue, irritability, dullness, listlessness, nausea, loss of appetite, alopecia (rapid hair loss), tremor and convulsions.

Manganese (Mn)

Manganese is necessary for the production of manganese superoxide dismutase, an antioxidant enzyme that quenches the superoxide radical. Many environmental and dietary factors, including alcohol, high polyunsaturated fat diets and oxidative stress, increase the need for manganese. Deficiency is rare, though decreased levels have been found in epilectics, diabetics, and persons with osteoporosis, high HDL cholestorol and diabetic-like glucose intolerance.

Potassium (K)

Erythrocyte potassium is the best single measure of body potassium status. Mild to moderate potassium deficiency is frequently found in those whose diet is low in fresh vegetables and fruit, especially if meat and fish intake is also low. Fortunately, the body has strong conservation mechanisms that dampen the effects of periods of low intake. Nervous and muscle tissues have strong requirements for potassium to maintain excitability. Depletion of body potassium can lead to a wide range of effects, including hypertension, heart arrythmias and muscle weakness. The use of vegetable juices, citrus juices, bananas, melons and other fruits and vegetables will increase potassium levels.

Selenium (Se)

Selenium has a fairly narrow window of safe effectiveness and works closely with vitamin E. Protein– containing foods, such as meats and seafood, in which the selenium is bound to amino acids are good sources of selenium. Evidence shows that dietary intake of selenium is directly related to levels of selenium in erythrocytes. Low levels indicate depleted selenium pools. Selenium functions primarily as an activator of enzymes necessary for cellular protection from oxidative damage and maintenance of normal redox potentials. A primary role of selenium in erythrocytes appears to be the activation of the enzyme glutathione peroxidase, whereby glutathione (a critical antioxidant and antitoxin for all cells) reacts with oxygen radicals. Similarly, selenium catalyses glutathione reductase, an enzyme that keeps glutathione in its reduced or active form.

Vanadium (V)

Recent data show vanadium to be essential for humans. Diets high in unsaturated oils have more vanadium than those high in saturated oils and unrefined brown sugar has 1000 times more vanadium than white sugar. Food is generally low in vanadium. Absorption of vanadium is highly dependent on its form. It is retained by liver and bone and transported on the blood protein transferrin. Vanadium lowers cholesterol synthesis and may lower plasma triglycerides in humans. It promotes mineralisation of bones and teeth and may protect against caries. Vanadium levels can become high due to environmental exposure to chemicals containing this element, as it is absorbed through inhalation.

Zinc (Zn)

Growth and repair of any tissue is dependent on zinc as an activating cofactor for DNA/RNA polymerase. For this reason, zinc is vital to the normal healing of wounds and skin disorders. Zinc is required for normal immune function. In fact, there are many similarities between the immunological effects of zinc deficiency and those of AIDS. Low zinc is associated with low T helper lymphocytes. If intake of calcium, copper or iron is excessive, tissue zinc may become depleted. If zinc is elevated, problems that might occur include iron-non-responsive anaemia, due to related copper deficiency and increased vascular disease risk from lowered HDL cholesterol.

TOXIC METALS

Toxic metals exchange between blood plasma and erythrocytes after a person is exposed. The concentration of metals in erythrocytes is also determined by the content of the tissue where erythrocytes originate – bone marrow. The marrow exchanges the metals with the mineral matrix of bone. Thus, elevated erythrocyte levels of a toxic metal reflect a deep tissue accumulation of the element. The distribution of elements between bone and soft tissues varies with each element. For example, lead tends to preferentially deposit in bone, while cadmium concentrations are usually highest in kidney. No single tissue or body fluid provides the whole answer to total body toxic metal load. The question of why a patient has developed a high toxic metal load is frequently difficult to answer. Obvious sources of high exposure are from industrial occupations where heavy metals used are easily identified, but chronic low-level exposure can escape notice.

The underlying mechanism of toxicity is often due to the ability of these metals to displace minerals from their binding sites. Metals, especially those that are lipid-soluble and can pass through cell membranes and the blood brain barrier, tend to be particularly neurotoxic. The mechanism for this is due to their ability to bind to sulphhydryl groups, generate free radicals and transform inorganic metals into organic metals – which allows metals to become more lipid-soluble. Free radical generation leads to lipid peroxidation, cellular membrane degeneration and untimely cell death. Finally, metals can also attach to proteins and become antigenic, such as occurs with 'metal hypersensitivity responses'. Mercury is particularly capable of producing such a response.

Aluminium (Al)

Toxic effects of aluminium include impaired memory (often Alzheimer's disease), dementia, osteomalacia, myoclonic jerks, aphasia (abnormal speech), ataxia (incoordination), convulsions and characteristic EEG changes. Urine is the major elimination route of aluminium, so once a chelating agent has mobilised and bound to the element, the latter is relatively easy to eliminate. Phosphorus can be protective in that it lowers intestinal absorption of aluminium. Sources include aluminium cooking utensils, baking powder (Al sulphate), certain antacids, antiperspirants, aluminium cans, drinking water (alum used as bacterioside), milk and milk products (from equipment), alum in pickled foods, nasal sprays, toothpaste, ceramics, dental amalgams, cigarette filters and tobacco smoke, exhaust fumes, pesticides, certain colourings, vanilla powder, table salt/ seasonings, bleached flour, American cheese, medication containing kaolin (Al silicate), sutures with wound healing coatings, rat poisons.

Cadmium (Cd)

The fact that cadmium is bound by the abundant zinc-sequestering protein, metallothionine, and that this protein occurs in high concentration in kidney, makes cadmium one of the most easily removed toxic elements. During chronic exposure, the kidney contains a major part of the body's burden of cadmium. Any intervention that increases the passage of metal-chelating agents through the kidney will lower total body burden of cadmium. Cadmium toxicity most clinically impacts the kidney, where damage to proximal tubules has been described. Also, cadmium compounds are classified as carcinogenic to humans. Other symptoms include yellow teeth, femoral pain, lumbago (backache), osteopenia, hypertension and vascular disease. Zinc is protective as it competes for cadmium binding sites. Sources include drinking water, soft water from galvanised pipes, soft drinks from dispensing devices with galvanised pipes, refined wheat flour, canned evaporated milk, processed foods, oysters, kidney, liver, cigarette smoke/ tobacco products, fertilizers, dental appliances, ceramic glazes, electroplating, paint pigments, silver polish, polyvinyl plastics, rubber carpet backing, nickel-Cd batteries, rust-proofing material.

Lead (Pb)

Lead toxicity causes paralysis and pain in the extremities due to effects on demyelinisation, axonal degeneration and presynaptic block. Microcytic hypochromic anaemia is the consequence of lead's inhibiting effects on enzymes in the heme-biosynthesis pathway. Other clinical signs associated with lead toxicity are kidney damage, hypertension, anorexia, muscle discomfort, constipation, metallic tastes, low IQ (children), epigastric pain and nausea, and male and female reproductive failure. Lead toxicity commonly affects sensory, visual, auditory and cerebellar (coordination) functions, reflecting the nervous system impact of this element. Patients may have paraesthesia in the region around the mouth and in the hands immediately after exposure. Various intravenous chelation agents, including penicillamine and EDTA, have been shown to be effective in reducing lead body burden.

Dietary calcium helps lower the intestinal absorption of lead. Whilst adults usually absorb only 1-10% of ingested lead, children can absorb up to 50%. Patients with reduced gastric integrity and reduced intakes of calcium, magnesium, iron, vitamin C and D will have increased absorption of lead. Serum levels of lead indicate current or chronic lead exposure. Hair levels are a good marker for lead toxicity as hair keratin has a high affinity for lead. Lead is widely distributed in the environment due to human activity. Sources include exhaust fumes, leaded house paint, drinking water from lead plumbing, vegetables grown in lead contaminated soil, canned fruit and juices, canned evaporated milk, crystal glassmaking, milk from animals grazing on contaminated land, bone meal, organ meats such as liver, pesticides, leaded caps on wine bottles, rainwater and snow, improperly glazed pottery, painted glassware, painted pencils, toothpaste, newsprint, coloured printed material, eating utensils, curtain weights, putty, car batteries, cigarette ash/ tobacco, lead shot/ firing ranges.

Mercury (Hg)

In its elemental form, mercury is non-toxic, however bacteria in the GI tract and environment transform it into its toxic inorganic form. As mercury passes up the food chain, it accumulates in deep-sea fish and man. Mercury has properties similar to lead and is toxic in three ways: it reacts with sulphhydryl groups and impairs the activity of enzymes; changes to tertiary structures and some proteins become immunogenic and elicit an autoimmune response (collagen in particular); these compounds are highly lipophillic and have a high affinity for tissue of the nervous system.

Conditions ranging from childhood autism to adult neurological dysfunction and dementia can result from the toxic effects of mercury. Other symptoms include hypertension, hearing loss, incoordination and loss of balance, dizziness, drowsiness, dermatitis, depression, irritability, insomnia, fatigue, poor short-term memory, tremor, stomatitis, gingivitis, renal disturbances, pain in joints/ limbs, weight loss, metallic tastes in mouth, decreased immunity, increase in antibiotic resistant strains of bacteria in the GI tract, chronic candidiasis. Removing the source of mercury should be the first treatment for toxicity. Sulphur-containing agents such as Dimercaptosuccinic acid (DMSA) are the most effective agents for removing mercury from tissues. Selenium is protective as it inhibits the cellular toxic effects of mercury.

Sources include dental amalgams, broken thermometers, calomel/ mercurious chloride (used in insecticides, fungicides, body powder, talc, laxatives), cosmetics, latex, solvent thinned paints, haemorrhoid suppositories, mercurochrome, fabric softener, floor waxes and polishes, air conditioning filters, wood preservatives, certain batteries, leather tanning products, felt, adhesives, skin lightening creams, certain ointments to treat psoriasis, tattooing, sewage sludge used as fertiliser, vaccinations (thiomersal), cosmetics and predator (eg tuna, swordfish, marlin) or fresh water fish.

Therapeutic considerations

1. Avoid exposure. Review sources as above and check contents of other material to which you have frequent exposure. In many cases it is difficult to identify specific sources; however care must be taken to reduce total toxic burden.
2. Reduce intestinal absorption by increasing dietary fibre, which restricts the amount of toxic metals absorbed. Include beans, cooked vegetables, whole grain breads and cereals, especially oatmeal and fresh fruits, particularly apples. If constipation is a symptom, work on this area first.
3. Increase elimination using natural chelating and complexing substances which increase the removal of toxic metals by forming more soluble complexes that can be eliminated by the kidneys. These include vitamin C (up to 3g/ day), chlorella, oral glutathione, methionine (3g/ day, ensure adequate folic acid and vitamin B12 to prevent elevation of homocysteine), lipoic acid (100mg TID). NAC should not be used in very high doses if mercury is elevated, as this may increase transport through the blood brain barrier.
4. Add competing minerals, as these displace toxic metals and may reduce toxicity. Selenium for mercury (maximum 200mg/d), zinc for cadmium (50µg/d), calcium for lead (1000mg/d) and magnesium for lead (500mg/d).

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