

Urine Elements Resource Guide

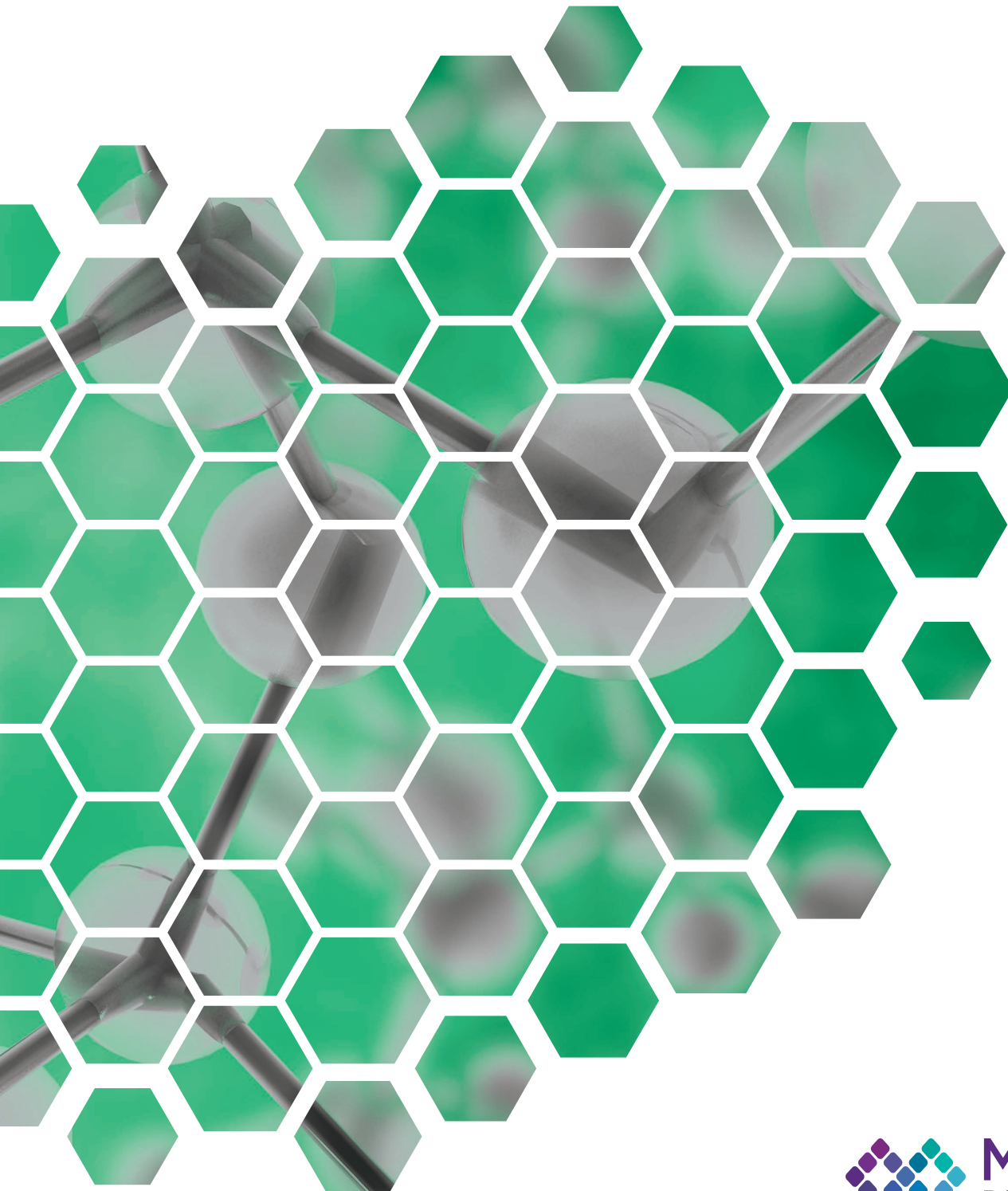


Table of Contents

Sample Report

<i>Sample Report</i>	1
--	---

Urine Toxic Metals Profile

Introduction	3
Aluminum	3
Antimony	4
Arsenic	4
Barium	5
Beryllium	5
Bismuth	5
Cadmium	6
Cesium	6
Gadolinium	7
Lead	7
Mercury	8
Nickel	9
Palladium	9
Platinum	9
Tellurium	10
Thallium	10
Thorium	11
Tin	11
Tungsten	12
Uranium	12

References

References	13
----------------------------------	----

Sample Report

Toxic Metals; urine



Order: 999999-9999

Test: U999999-9999-1
Client #: 999999
 Doctors Data Inc
 123 Main St.
 St. Charles, IL 60174 USA

Patient: Sample Patient
Id: 999999
Age: 60 **DOB:** 01/01/1960
Sex: Male
pH upon receipt: Acceptable

Sample Collection
Date Collected 08/01/2020
Collection Period Random
Date Received 08/03/2020
Date Reported 08/04/2020

Toxic Metals	Result	Unit	Within Reference	Outside Reference	Reference Interval
Aluminum	1.6	µg/g Creat			< 15
Antimony	0.074	µg/g Creat			< 0.18
Arsenic	12	µg/g Creat			< 40
Barium	1.6	µg/g Creat			< 5
Beryllium	<dl	µg/g Creat			< 0.10
Bismuth	0.24	µg/g Creat			< 0.8
Cadmium	0.35	µg/g Creat			< 0.6
Cesium	12	µg/g Creat			< 9
Gadolinium	<dl	µg/g Creat			< 0.5
Lead	0.52	µg/g Creat			< 1.1
Mercury	1.0	µg/g Creat			< 0.8
Nickel	3.9	µg/g Creat			< 4
Palladium	<dl	µg/g Creat			< 0.2
Platinum	<dl	µg/g Creat			< 0.1
Tellurium	<dl	µg/g Creat			< 0.2
Thallium	2.2	µg/g Creat			< 0.4
Thorium	<dl	µg/g Creat			< 0.007
Tin	0.34	µg/g Creat			< 3
Tungsten	<dl	µg/g Creat			< 0.4
Uranium	<dl	µg/g Creat			< 0.03

Urine Creatinine	Result	Unit	-2SD	-1SD	Mean	+1SD	+2SD	Reference Interval
Creatinine	17.9	mg/dL						87 – 190

Notes:
 < dl: less than detection limit
 Results are creatinine corrected to account for urine dilution variations. **Reference intervals are based upon NHANES (cdc.gov/nhanes) data if available, and are representative of a large population cohort under non-provoked conditions.** Chelation (provocation) agents can increase urinary excretion of metals/elements.
 Methodology: ICP-MS QQQ, Creatinine by Jaffe Reaction

Page: 1 of 7
Analyzed by DOCTOR'S DATA, INC. • 3755 Illinois Avenue, St. Charles, IL 60174-2420 USA • LAB DIR: Erlo Roth, MD • CLIA ID: 14D0646470

Reference intervals are based upon the NHANES 95th percentile, and are age and sex specific.

Analysis by ICP-MS triple quadrupole (QQQ)

Essential Elements; urine



Order: 999999-9999



Test: U999999-9999-1

Client #: 999999

Doctors Data Inc

123 Main St.

St. Charles, IL 60174 USA

Patient: Sample Patient

Id: 999999

Age: 60 DOB: 01/01/1960

Sex: Male

pH upon receipt: Acceptable

Sample Collection

Date Collected

Collection Period

Date Received

Date Reported

Date/Time

08/01/2020

Random

08/03/2020

08/04/2020

Essential Elements	Result	Unit	Percentile	Reference Interval
			2.5 th 16 th 50 th 84 th 97.5 th	
Sodium	125	mEq/g Creat		60 – 140
Potassium	99.2	mEq/g Creat		35 – 70
			2.5 th 16 th 50 th 84 th 97.5 th	
Phosphorus	611	µg/mg Creat		300 – 700
Calcium	177	µg/mg Creat		50 – 200
Magnesium	241	µg/mg Creat		40 – 100
Zinc	0.32	µg/mg Creat		0.2 – 0.7
Copper	0.0072	µg/mg Creat		0.007 – 0.015
Sulfur	607	µg/mg Creat		360 – 680
Molybdenum	0.0400	µg/mg Creat		0.02 – 0.07
Boron	5.1	µg/mg Creat		0.8 – 2.2
Lithium	0.0498	µg/mg Creat		0.015 – 0.07
Selenium	0.259	µg/mg Creat		0.045 – 0.12
Strontium	0.253	µg/mg Creat		0.065 – 0.18
			68 th 95 th	
Cobalt	0.70	µg/g Creat		< 0.5
Iron	<dl	µg/g Creat		< 10
Manganese	<dl	µg/g Creat		< 0.15
Chromium	0.98	µg/g Creat		< 0.5
Vanadium	<dl	µg/g Creat		< 0.1
			-2SD -1SD Mean +1SD +2SD	
Urine Creatinine				
Creatinine	17.9	mg/dL		87 – 190

Out of range results are accompanied by a comprehensive descriptive text inclusive of common sources of exposure and potential symptoms.

Notes:

< dl: less than detection limit

Results are creatinine corrected to account for urine dilution variations. Reference intervals are based upon NHANES (cdc.gov/nhanes) data if available, and are representative of a large population cohort under non-provoked conditions. Chelation (provocation) agents can increase urinary excretion of metals/elements.

Methodology: ISE, Spectrophotometry, ICP-MS QQQ, Creatinine by Jaffe Reaction

Page: 2 of 7

Analyzed by DOCTOR'S DATA, INC. • 3755 Illinois Avenue, St. Charles, IL 60174-2420 USA • LAB DIR: Erlo Roth, MD • CLIA ID: 14D0648470

Urine Toxic Metals Profile

Introduction

The analysis of urinary metals was performed by ICP-MS triple quadrupole (QQQ). Urine metal analysis is traditionally used for evaluation of very recent or ongoing exposure to potentially toxic metals. Reference intervals and corresponding graphs are age and sex specific, based upon NHANES (cdc.gov/nhanes) data if available, and representative of a large population cohort under non-provoked conditions. The urinary excretion of certain metals is known to be increased (provoked) to a variable extent after chelation. Reference intervals have not been established for post-chelation urine samples and are not recommended for comparison purposes with provoked test results.

For timed, random or first morning urine collections, metals are reported as $\mu\text{g}/\text{gram creatinine}$. Normalization per creatinine corrects for variability in the sample dilution (concentration). Under normal conditions, the rate of excretion of creatinine is quite constant and highly correlated with lean body mass (muscle). For 24 hour (h) urine collections elements are reported as $\mu\text{g}/24 \text{ h}$; results are also reported as μg element/gram creatinine to ensure clinically useful information in the event that an inaccurate 24 h urine volume was reported to the laboratory.

Descriptive texts appear in this report if detected levels of specific elements are abnormally high by comparison to the unprovoked reference values. If no descriptive texts follow this introduction, potentially toxic metals are within reference limits.

Aluminum

Urinary aluminum (Al) provides an indication of very recent or ongoing exposure to the potentially toxic metal. Urine accounts for greater than 95% of Al excretion from the body. Compromised renal function increases the risk of Al retention in the very young, elderly and patients with renal disease.

Al is ubiquitous, as the third most common element of the earth's crust. The general population is primarily exposed to Al through the consumption of food items, and lesser exposure may occur through ingestion of Al in drinking water and inhalation of urban air. Foods with high Al contents include imitation cheeses, cake mixes, frozen dough, pancake mixes, self-rising flours and pickled vegetables. Chewing gums may contain a significant amount of Al (up to 4 mg/stick). Food related uses of Al compounds include preservatives, fillers, coloring agents, anti-caking agents, emulsifiers and baking powders; some soy-based infant formulas have added Al. Cooking with aluminum foil or Al cookware may contribute to Al exposure; highly acidic foods increase leaching of Al. Al is present in many over-the-counter medications, such as antacids and buffered aspirin; chronic use of such may be a very significant source of Al exposure. Oral bioavailability of Al varies depending mainly on the chemical form of the ingested compound, and the concurrent intake of dietary acids that bind Al (e.g. citric acid, ascorbic acid, lactic acid and glycine). Al is present in a number of topically applied consumer products such as antiperspirants and cosmetics. Occupational exposures pertain primarily to absorption of Al-containing dust and fumes that may be associated with welding, fabrication and metallurgy.

Tissue concentrations of Al increase with age. The brain retains less Al than other tissues (e.g. bone), but the nervous system is the most sensitive target for toxic effects of Al. Some studies have reported that the Al concentration in the bulk brain samples, neurofibrillary tangles and plaques was higher in Alzheimer's disease subjects than controls, but other studies have found no difference.

Chelation may acutely increase urinary excretion of Al. Hair elemental analysis for assessment of Al exposure has not been documented, and external contamination of hair with Al may be a confounding factor.

Antimony

Urinary antimony (Sb) provides an indication of recent or ongoing exposure to the toxic metal, and endogenous detoxification to a lesser extent. Sb is chemically similar to, but less toxic than inorganic arsenic. Like arsenic, Sb is very sulfhydryl-reactive and is conjugated with glutathione and excreted in urine and feces.

Food and smoking are the most common sources of exposure to Sb. Sb has been shown to leach from “squishy” plastic (PET bottles) into bottled water; the extent of Sb contamination is temperature and time dependent. Some Sb containing pharmaceuticals are used to treat the intestinal parasite Leishmania. Gunpowder (ammunition) often contains Sb. Other possible sources of exposure include textiles (flame resistant material), metal alloys, brake pads, solder, paints, glass, ceramics, bearing metals and semiconductors.

Early signs of extensive exposure to Sb may include: gastrointestinal distress, fatigue, muscle weakness, myopathy, nausea, low back pain, headache, and metallic taste. Chronic Sb toxicity may be associated with cardiovascular effects including: hypotension, cardiac pain (angina pectoris), cardiac arrhythmia and faulty ventricular polarization.

Hair elements analysis may provide evidence of Sb exposure over the past few months. Chelation may acutely increase urinary excretion of Sb.

Arsenic

Urinary arsenic (As) provides an indication of recent or ongoing exposure to various forms of the metalloid. Urine As may also indicate, to a lesser extent, endogenous detoxification of inorganic As. The less toxic organic forms of As from seafood have a circulating half-life of about 48 hours, and are rapidly excreted from the body. Consider very recent shellfish consumption with a finding of high urinary As.

Food, well water, and air are common sources of exposure for As. The predominant dietary source of As is seafood, followed by rice/rice-based products, mushrooms, and poultry (antiparasitics in feed). While fish/shellfish contain the highest levels of As, most abundant are the much less harmful organic As species arsenobetaine and arsenocholine; they are rapidly and efficiently excreted. Some sea vegetables may contain the more toxic inorganic As. Other common sources of As include: tobacco, certain pesticides, fungicides (orchards, vineyards), smog, and older wood preservatives (CCA; pressure treated wood/sawdust). Copper and lead smelting, as well as hazardous waste sites contribute to environmentally-derived As exposure.

Significant exposure to inorganic As may be associated with malaise, muscle weakness, vomiting, diarrhea, and dermatitis. Long-term exposure may increase risks for diabetes, vascular disease and cancer (skin, bladder, kidney and lung). As may also adversely affect the peripheral nervous and hematopoietic systems. The W.H.O has stated that in utero and early childhood As exposure has been linked to negative impacts on cognitive development and increased deaths in young adults.

Physiological detoxification of inorganic As entails a sequence of oxidation/reduction and methylation reactions, with final conjugation to glutathione prior to excretion in urine and bile. Genetic and epigenetic factors may impair As detoxification. Compromised status of methionine, cysteine, glutathione, folate and B-12 may result in diminished capacity for endogenous detoxification of As.

Inorganic As accumulates in hair, nails, skin, thyroid gland, bone and the gastrointestinal tract. Exposure to inorganic As over the past few months may be assessed with hair elemental analysis. Chelation may acutely increase urinary excretion of inorganic As.

Barium

Urinary barium (Ba) provides an indication of recent or ongoing exposure to the toxic metal, and endogenous detoxification to a lesser extent. The main dietary sources of Ba include milk, flour, potatoes and some nuts and nut butters. Brazil nuts naturally contain high concentrations of Ba (e.g. 1,500 µg/g), and their recent consumption may be transiently associated with clinically insignificant elevations of urinary Ba.

Other sources of exposure to Ba include contaminated water, air or soil. Ba has been used in rodenticides and insecticides, and is used for medical testing (barium “swallow” for imaging). Mining, refining and combustion of coal and oil emit Ba. Mine-tailings and masonry products made from such can be a significant source of Ba to individuals producing or working with them (e.g. reconstruction, demolition). Arc-welding, metal fabrication, and occupational work with fireworks can be associated with abnormal exposure to Ba.

In the body excessive Ba may interfere with calcium metabolism and displace potassium. Very high-level acute exposure or chronic exposure to Ba may be associated with gastrointestinal distress (gastric pain, nausea, vomiting, and diarrhea), hypokalemia, ventricular tachycardia, hypertension and/or hypotension, muscle weakness, paralysis, bronchoconstriction, and painful swelling of the brain and testis.

Additional testing for excessive exposure to Ba includes hair elemental analysis and measurement of blood electrolytes; hypokalemia may be associated with elevated Ba. The Comprehensive Water Analysis test assesses the amount of Ba in drinking water.

Beryllium

Urinary beryllium (Be) provides an estimate of a recent or ongoing exposure to the metal, and endogenous detoxification to a lesser extent. After assimilation Be is slowly excreted in urine and may be found elevated many months after high level exposure.

Be is poorly absorbed in the gastrointestinal tract, but is readily absorbed across the lungs and skin. Inhalation is the primary route of significant exposure to Be, and may be associated with dyspnea, cough and pulmonary distress (berylliosis). Berylliosis, is an occupationally acquired lung disease that is associated with primary production, metal machining, and reclaiming scrap alloys. Other high-exposure occupations are in the nuclear power, aerospace, and electronics industries.

Tobacco contains Be, and smoking immediately increases Be levels in blood and urine. Be is a metal that is widely used in the manufacturing of cars, computers, golf clubs, and electrical equipment. Other possible sources of Be include electronic components (computers), metal alloys used in aircraft and aerospace applications (especially aluminum-copper-beryllium alloys), welding bearing sleeves, industrial ceramics, optical lens coatings, and some phosphors in fluorescent lights.

Be is a biological antagonist of magnesium. Be has a long-term effect of inducing abnormal activity in T lymphocytes, causing immune dysregulation and hypersensitivity reactions. Beryllium has been detected in hair but documentation correlating exposure, tissue levels and hair levels is lacking.

Bismuth

Urinary bismuth (Bi) provides an estimate of exposure to the metal, and endogenous detoxification to a lesser extent. Urinary Bi may be elevated for days to a few weeks after exposure.

Bi is a non-essential element with relatively low potential for toxicity. Use of Bi in medicine and health care is due to its high effectiveness in treating burns, intestinal disorders, and stomach ulcers as well as its potential activities against microorganisms, viruses and malignant tumors. Bismuth citrate-based compounds are the most common anti-H. pylori bismuth drugs and have been widely utilized in “cocktail” therapy to treat a variety of H. pylori-associated infections.

Sources of Bi include: cosmetics (lipstick), Bi containing medications such as ranitidine Bi-citrate, antacids (Pepto Bismol), pigments used in colored glass and ceramics, and dental cement. Several organometallic Bi compounds are used for bactericidal and fungicidal applications. Alloys of Bi have widespread commercial applications such as in the production of lubricating grease, chemicals, catalysts, shot bullets, fire sprinkler systems, solders, thermoelectric materials, and fishing sinkers. Bi is a by-product of processing lead, silver, tin, copper, and zinc.

Symptoms of moderate Bi toxicity include constipation or bowel irregularity, foul breath, blue/black gum line, and malaise. Unusually high levels of Bi retention in the body may be associated with nephrotoxicity (nephrosis, proteinuria) and neurotoxicity (tremor, memory loss, myoclonic jerks, dysarthria, and dementia).

Hair elemental analysis may provide information regarding Bi exposure over the past 2-4 months. Chelation may acutely increase the urinary excretion of Bi.

Cadmium

Urinary cadmium (Cd) provides an indication of recent or ongoing exposure to the toxic metal, and endogenous detoxification to a lesser extent. Most of absorbed Cd is retained in the liver and kidneys for many years. A small portion of assimilated Cd body leaves slowly in urine and bile/feces. Absorption, systemic transport and cellular uptake of Cd are mediated by metal transporters that the body uses for the essential elements iron, zinc and calcium.

For nonsmokers the primary source of Cd exposure is food. Cd is found in varying amounts in foods; from very low for fruits to high in some shellfish (oysters, anchovies) and organ meats. Root vegetables tend to have higher Cd content than other vegetables. Refined carbohydrates have very little zinc in relation to the Cd; zinc competes with Cd for absorption from the gut. Tobacco leaves accumulate high levels of cadmium, and Cd absorption across the lungs is very efficient. Urban particulate air pollution/dust (diesel smoke, tire wear) is a significant source of Cd exposure. Other sources of Cd include: human biosolids, tires, pigments and paints, plastics, and batteries (Ni-Cd). The majority of Cd-containing batteries are improperly disposed of and contribute to environmental exposure.

Excessive exposure to Cd may be symptomatically associated with fatigue, weight loss, osteomalacia, and lumbar pain. Chronic exposure to Cd may adversely affect the kidneys, lungs, testes, arterial walls, and bones. Chronic Cd excess may lead to hypertension, coronary and peripheral vascular disease, macular degeneration, kidney disease, obesity-independent diabetes, and microcytic, hypochromic anemia and functional zinc deficiency. Cd is known to damage the kidneys, especially the proximal tubular cells that actively reabsorb Cd along with zinc, glucose and amino acids. Cd is pro-inflammatory and pro-oxidative.

Urinary or serum beta-2-microglobulin is considered to be the best test for assessment of Cd-associated functional damage to the kidneys. Urine amino acid wasting may also be considered. Chelation may acutely increase urinary excretion of Cd.

Cesium

Urinary cesium (Cs) provides an indication of recent or ongoing exposure to the metal. Naturally occurring Cs, the isotope measured in this test, is not radioactive and is referred to as stable Cs (Cs133). Cesium is a naturally occurring element present at low levels in rocks, soil, and dust.

Humans may be exposed to Cs at relatively low levels from air and diet. Cesium-chloride is used as a lubricant to facilitate drilling for oil and natural gas. As such Cs may contaminate surface and ground water, and certain crops in close proximity to drilling sites. Very high levels of urinary Cs have been observed for people supplementing with oral Cs-chloride. Cesium chloride has been proclaimed to be a therapeutic treatment for cancer, but documentation to that effect is not available. Indiscriminant use Cs-chloride solutions have been reported to have very serious consequences when chronically ingested or injected at high levels. Like thallium, Cs is antagonistic to the essential element potassium, and Cs toxicity has been associated with hypokalemia, ventricular tachycardia and death.

Serum electrolytes and blood or red blood cell elements analysis are follow up tests for assessment of physiological impact of excessive exposure to Cs.

Gadolinium

Urinary gadolinium (Gd) provides an indication of recent or ongoing exposure to the metal, and endogenous detoxification to a lesser extent. Urinary Gd would be expected to be variably high if urine was collected within a week of medicinal Gd administration for imaging purposes.

Gd is found in the environment in geographically variable amounts, and usually at very low levels. It is widely used in industrial and household applications such as radar technologies, compact discs, and microwaves; direct exposure from such sources is not a concern. However disposal of Gd-containing devices contributes to greater potential for human exposure. The single greatest direct source of exposure to Gd is Gd-based contrast agents (GBCAs) that are widely used with magnetic resonance imaging (MRI). Concern has been raised regarding the use of unstable linear GBCAs, especially for patients with mild to severe kidney dysfunction. Urinary Gd levels vary with the time after administration, and the dose of the specific GBCA. There is much controversy regarding the safety of certain unstable GBCAs; Gd doesn't have physiological functions in the body.

Urinary levels of Gd normally decrease rapidly after administration for patients who have good kidney function (glomerular filtration rate; GFR). However, the rate of Gd clearance may be markedly slowed with compromised GFR. While the Gd levels normally decrease rather rapidly in urine and feces, it is clear that some Gd is retained in the body for a long time. Of greatest potential concern is Gd deposition in the brain, which is correlated with the number of GBCA-enhanced MRI procedures.

Gadolinium deposition disease has recently been described and may be associated with central and peripheral pain, headache, bone pain, skin thickening, muscle weakness, arthralgia, and persistent clouded mentation and headache. If such new symptoms appear 2-8 weeks after Gd-enhanced MRI, it is recommended to assess the level of Gd in urine (1st AM void or 24 hour collection).

Elemental analysis of hair or nails may be used to assess exposure to Gd over a longer period of time, but reference values are not available for nails. Chelation may acutely increase urinary excretion of Gd.

Lead

Urinary lead (Pb) provides an indication of recent or ongoing exposure to the toxic metal, and endogenous detoxification to a lesser extent. Low level Pb exposure is particularly problematic for the developing nervous system.

Dust and paint chips from Pb-based paint in older buildings are the most common sources of Pb exposure for children. Some types of artificial turf and rubber playground surfaces can also contain Pb. Imported toys and costume jewelry tainted with Pb can be an issue. Imported non-glossy, vinyl mini-blinds are known to contain lead. Water is a common source of exposure for Pb. Old plumbing, especially from 1930 or earlier, is a significant source for chronic exposure to Pb. Plumbing in very new homes may also be problematic, due to the use of leaded solder to join copper pipes. Well water may also be contaminated with Pb in pump components or the well seal. Old pipes that supply water to buildings may contaminate water with Pb.

Foods are not commonly contaminated with Pb, but imported candies or foods containing chili or tamarind may be contaminated with Pb. Lead has been reported to be present in chocolate (the darker the higher), cocoa powders, and some chocolate flavored whey protein concentrates. Some Ayurvedic medications have been found to contain high levels of Pb and other toxic metals. Pb may get into foods or liquids that have been stored in ceramics, pottery, china, or leaded-crystal.

Sources of air-borne Pb include: waste incinerators, utilities, and lead-acid battery manufacturer, and ore and metals processing. The highest air concentrations of Pb are usually found near lead smelters. Fishing tackle, ammunition, indoor firing ranges, and some art/hobby supplies may contribute to Pb exposure. Transdermal Pb absorption is slight, except for high absorption of lead acetate that may be present in hair

darkening products and certain tattoo inks.

Pb has pathological, neurotoxic, nephrotoxic, cardiovascular and carcinogenic effects that may be manifested with chronic low-level exposure. Pb may also affect the body's ability to utilize the essential elements calcium, magnesium, and zinc. Sustained Pb exposures may have adverse effects on memory, cognitive function, nerve conduction, and metabolism of vitamin D. Infants and children are especially vulnerable to Pb-induced developmental disorders, and behavior problems are associated with lower levels of blood Pb than previously acknowledged; lower of IQ, hearing loss, and poor growth. Pb is transferred across the placenta, and into breast milk.

Lead exposure and toxicity is commonly assessed by elevated blood lead. However blood lead may only reveal isolated exposures as the half-life of Pb in circulation is only about 1 month. Hair elemental analysis may provide information regarding Pb exposure over the past 2-4 months. Urine porphyrin analysis may reveal Pb-induced disruption of heme biosynthesis (physiological impact). Chelation may acutely increase the urinary Pb excretion.

Mercury

Urinary mercury (Hg) provides an indication of recent or ongoing exposure to the toxic element, and endogenous detoxification to a lesser extent. Potential toxic effects of Hg vary based on the form of the metal, the concentration, route, and duration of the exposure, and the individual's age and susceptibility.

The elemental (metallic) form of Hg is a dense liquid that volatilizes at room and body temperature. Elemental Hg is poorly absorbed from the gut. However Hg vapors are readily absorbed, with subsequent rapid distribution of Hg throughout the body. Very high exposures can result in central nervous system and renal toxicity. Possible sources of elemental Hg include: dental Hg-silver amalgam material, improper clean-up (vacuuming) of broken Hg-containing household items (compact fluorescent light bulbs, thermometers, thermostats, electrical switches), recent application of Hg-containing paint or caulk (marine), occupation and hobbies, and folk remedies. Elemental Hg may be higher in air downwind of coal fired power plants.

Symptoms of prolonged and/or acute exposures to elemental Hg may include: tremors, emotional changes (such as mood swings, irritability, nervousness), insomnia, headaches, neuromuscular weakness/atrophy, altered sensations, and poor cognitive function. Repeated or continuous high-level exposure to Hg vapor may result in accumulation of Hg in the body and permanent damage to the nervous system and kidneys.

Methylmercury (MeHg) is the most commonly encountered organic form of Hg. Exposure to MeHg and aquatic vegetables that concentrate MeHg. The highest concentrations of MeHg are generally found in larger predatory fish. Federal, state and local governments issue fish consumption advisories when fish are unsafe to eat. Detailed information regarding MeHg and fish consumption is readily available (e.g. <https://www.epa.gov/choose-fish-and-shellfish-wisely>). MeHg is almost completely absorbed in the gastrointestinal tract, enters the blood stream and distributes to all organs, including the brain.

Possible symptoms associated with excessive exposure to MeHg may include: loss of peripheral vision, paresthesias, vertigo, tinnitus, ataxia and muscle weakness. Infants and children are particularly susceptible to neurotoxic effects. Children exposed to MeHg in the womb may have impacts to their cognitive thinking, memory, attention, language, fine motor skills, and visual spatial skills.

Some medicines and laboratory reagents contain the Hg-containing preservative thimerosal. Hg is still used in skin lighteners and some anti-aging products for the skin; the imported products are sold illegally in the United States.

Elemental Hg has a short half-life in blood (3 days), so blood analysis for elemental Hg exposure would be confirmatory within the first 3 days after an acute high level exposure, or with current ongoing exposure. MeHg has a long half-life in blood (50-70 days) and MeHg partitions primarily in the red blood cells (RBCs). Elemental analysis of RBCs provides specific information regarding exposure to MeHg from fish.

MeHg accumulates in hair, and elemental analysis of hair provides a good indication of consumption of contaminated fish over the past 2-4 months. Chelation may acutely increase urinary excretion of Hg.

Nickel

Urinary nickel (Ni) provides an indication of recent or ongoing exposure to the metal, and endogenous detoxification to a lesser extent. There is substantial evidence that Ni is an essential trace element. However, excessive assimilation of Ni has been established to be nephrotoxic, and carcinogenic.

The general population may be exposed to Ni from ambient air, water and food. With the exception of specific occupational exposures, most absorbed Ni comes from food and beverages, and intakes can vary depending upon geographical location and water supply. Extensive Ni exposure may occur with cigarette smoking.

Ni is present at relatively higher levels in a large number of foods and food products, including: hydrogenated oils (margarine), black tea, nuts and seeds, soy milk and chocolate milk, chocolate and cocoa powders, certain canned and processed foods, and certain grains such as oats, buckwheat, whole wheat, and wheat germ. Other sources of Ni include: urban air/particulate diesel exhaust, Ni-Cd batteries, non-precious/ semiprecious dental materials, pigments (usually for ceramics or glass), arc welding, and nickel refining, metallurgical processes and electroplating.

Hair elemental analysis may confirm exposure to Ni over the past 2-4 months. Chelation may acutely increase urinary excretion of Ni.

Palladium

Urinary palladium (Pd) provides an indication of recent or ongoing exposure to the metal, and endogenous detoxification to a lesser extent.

As one of the rarest elements in the earth's crust, Pd is a precious metal associated with the platinum-group metals. The chemical and physical properties of Pd account for its widespread use in catalytic and automobile related industrial applications, electronic devices, dental applications, and fine jewelry (white gold). Currently Pd is used in the manufacture of the tiny multi-layer ceramic capacitors used in wide-screen television screens, computers and mobile phones. A significant increase in the industrial use of Pd has resulted in higher environmental levels of this metal, particularly in road dust, airborne microparticles, soil, and groundwater.

Elemental Pd from food and water is generally regarded as having low toxicity as it is poorly absorbed when ingested. However, respiratory exposures to Pd can cause acute toxicity or hypersensitivity with respiratory symptoms and urticaria. This effect on the immune system represents a health hazard. Observational studies suggest that Pd ions are one of the most common reactive sensitizers. Allergic contact dermatitis may be associated with Pd, and the main contact sources are jewelry and dental materials (alloyed with silver, gold, and copper). Very low doses are sufficient to cause allergic reactions in susceptible individuals.

Exposure to Pd over the past 2-4 months may be assessed with the Hair Toxic Element Exposure Profile. Chelation may acutely increase urinary Pd excretion.

Platinum

Urinary platinum (Pt) provides an indication of recent or ongoing exposure to the metal, and endogenous detoxification to a lesser extent. Significant exposure to this non-essential, precious metal is unusual except in association with chemotherapy.

Pt-containing, high-gold alloys have been used in dentistry for many decades. Platinum is poorly absorbed in the gut and high-level oral exposure is unlikely. Jewelers who make high-end jewelry may be exposed to

Pt. There may have been a slight increase in environmental Pt due to the use of Pt in automobile catalytic converters, but palladium has almost completely replaced Pt for that purpose. The platinum-based drugs cisplatin, carboplatin and oxaliplatin are commonly used intravenously in the treatment of cancer. As a sulfhydryl-reactive metal, Pt has a very long life-time in the body. The liver and kidneys are predominant sites of accumulation and potential toxic effects.

Hair elements analysis may reveal exposure to Pt over the past 2-4 months. Chelation may acutely increase urinary Pt excretion.

Tellurium

Urinary tellurium (Te) provides an indication of recent or ongoing exposure to the metal, and endogenous detoxification to a lesser extent. The metal has no physiological function in the body, and urinary excretion is predominant.

Te is a very rare element that is a byproduct of milled copper. The use of Te in industrial applications has increased in scope and scale. Te may be used as an additive in steel and it is often alloyed to aluminum, copper, lead and tin. It is also used in the manufacture of solar panels (cadmium-telluride), cast iron, ceramics, vulcanized rubber, blasting caps, and glass production.

Elemental Te is poorly absorbed so exposure to the metal from food and water is rare; organic Te compounds are found in relatively high concentration in fresh garlic buds. Soluble Te compounds can be absorbed through the skin, although ingestion or inhalation of fumes presents the greatest industrial hazard. Aerosolized Te is irritating to the eyes and is readily absorbed by the respiratory tract and can cause respiratory irritation, drowsiness, dry mouth, metallic taste, headache, abdominal pain, nausea and constipation. The main target sites for Te toxicity are the kidney, nervous system, skin, and the fetus. Chronic exposure to Te may be associated with garlic odor of the breath and sweat, as Te is methylated to dimethyl telluride in the body.

Thallium

Urinary thallium (Tl) provides an indication of recent or ongoing exposure to the metal, and endogenous detoxification to a lesser extent. About 35% of assimilated Tl is excreted in urine, and urinary Tl may be at elevated levels for up to 30 days after exposure.

Tl is rapidly and almost completely absorbed when ingested, inhaled or brought into contact with skin. Tl is a highly toxic heavy metal which is generally tasteless and odorless.

Currently the most common sources of dietary Tl are contaminated vegetables, fish and shellfish; particularly those obtained in close proximity to drilling sites for natural gas and oil. Kale, spinach, cabbage and other Brassicaceae family vegetables appear to be most highly contaminated. The highest levels of urine Tl observed at this laboratory have been associated with daily consumption of "green drinks" that were prepared at home from raw Brassicaceae vegetables. It should be noted that a labelling of "organic" generally does not provide any assurance that the produce is not contaminated with Tl. Contaminated water has apparently been used to irrigate crops in certain agricultural areas in California. Other possible sources of Tl include tobacco, fly ash (coal), cement dust, some fertilizers, some artists' paints, semiconductors, and hazardous waste sites and landfills. Tl is also a by-product of the smelting of copper, zinc and lead ores.

Symptoms associated with significant exposure to Tl may include: fatigue, headaches, sleep disturbance, neuropathy, ataxia, depression, psychoses, and extreme loss of hair. Tl follows potassium in the body and accumulates in tissues with high potassium content including skeletal/cardiac muscle, and central/peripheral nerves.

Hair elemental analysis may be utilized to assess exposure to Tl over the past 2-4 months.

Thorium

Urinary thorium (Th) provides an indication of recent or ongoing exposure to the radioactive metal, and endogenous detoxification to a lesser extent. This test measures Th232 which is the most abundant, naturally occurring radioactive isotope of Th.

Th is found almost everywhere in the earth's crust, so exposure to small amounts of Th from air, food and water is unavoidable. Th is a naturally occurring radioactive metal that is found at low levels in soil, rocks, water, plants, and animals. Th is almost as abundant in the earth's crust as lead, and three times more abundant than uranium (U238). Gastrointestinal absorption of Th from food and water is very low (0.1 to 1%). Most assimilation of Th occurs via inhalation of contaminated dust and microparticles in air.

Individuals who work in or near the mining, milling or Th industries may experience greater than normal Th exposure. Th is used to make ceramics, gas lantern mantles, welding rods, and camera and telescope lenses. It is also used as an alloying element with magnesium to coat tungsten wire that is used in electronic equipment, and metals used in the aerospace industry. The amounts of Th in the environment may be increased in association with accidental release from a processing facility. Large amounts of Th may be found near hazardous waste sites where Th has not been disposed of properly. People that live near these hazardous waste sites may be exposed to more Th than usual via inhalation of dust and consumption of locally grown food.

The radiation from Th232 and its decay products is in the form of alpha and beta particles and gamma radiation. While Th exposure for most people is well below levels associated with toxic effects, occupational exposure to Th dust increases risks for lung disease, lung cancer and pancreatic cancer. Th may have adverse health effects on bone and bone metabolism in association with long-term retention of Th-based radioactivity in the skeletal system.

Hair elemental analysis may be used to assess exposure to Th over the past 2-4 months. Chelation may acutely increase urinary Th excretion.

Tin

Urinary tin (Sn) provides an indication of recent or ongoing exposure to the metal, and endogenous detoxification to a lesser extent. Sn has no known physiological function in the body.

Inorganic Sn has a low potential for toxicity, while organic Sn may have appreciable toxic effects. Metallic Sn and inorganic Sn compounds are normally found in small amounts in soil, food and air. Exposure to Sn compounds may be much higher in close proximity to hazardous waste sites. Inorganic Sn is poorly absorbed from the gut. The main source of Sn is food. Canned tomatoes, tomato products, pineapple, pears and similar fruits contain the highest concentrations of Sn. The Sn concentrations of food increase with storage in opened cans. Other possible sources of inorganic Sn include: dinnerware, preservatives, food and beverage containers, cosmetics, pewter, bronze, and anticorrosive plating. Children who eat dirt (pica) may have increased exposure to Sn if the soil is contaminated. Inhalation of fumes (solder, welding) or dusts containing high amounts of Sn may result in irritation of the respiratory tract.

Inhalation, oral, or dermal exposure to some organotin compounds may have harmful effects. Potential toxic effects depend on the particular organotin compound. Organotin compounds, such as butyltin, are used in the manufacture of some plastics, food packages, certain PVC pipes, pesticides, paints, wood preservatives, and rodent repellants. Routine use of silicon-coated baking parchment paper may contribute to exposure to organotin. Toxic effects of Sn compounds are related, in part, to the interference with the iron and copper metabolism. Very high exposure to organotin compounds may be associated with skin and eye irritation, respiratory irritation, gastrointestinal effects, muscle weakness, anemia, and neurological and kidney problems.

Hair elemental analysis may be used to assess exposure to Sn over the past 2-4 months. Chelation may acutely increase urinary excretion of Sn.

Tungsten

Urinary tungsten (W) provides an indication of recent or ongoing exposure to the metal, and endogenous detoxification to a lesser extent. W doesn't have physiological functions in the body, and has low toxic potential with oral exposure.

About 50% of W appears to be rapidly absorbed from gastrointestinal tract, and excretion from the body is primarily via the urinary route. W is highly absorbed via inhalation of dust and fumes. In the body W is antagonistic to the essential element molybdenum which is important for the conversion of sulfite to essential sulfate, and for the production of uric acid. Thereby, excess W may impair physiological reactions and be associated with sulfite sensitivity (wine, eggs, etc.) and/or low levels of uric acid in blood. Low uric acid is not necessarily consequential, but rather may be an indicator of functional molybdenum insufficiency.

Most exposures to W are from foods, and tungstate salts that may contaminate drinking water. Unconfirmed information is suggestive that rice protein concentrates/rice-based gluten-free products may be contaminated with W during processing. If true, that might explain the higher levels of urine W after DMSA for autistic children who are commonly on gluten-free diets (unpublished observations, Doctor's Data). Other sources of W include catalysts and reagents in biochemical analysis, fire and waterproof materials, industrial lubricants, and ash from incineration of sewage sludge; "biosolids" may also be used to fertilize crops and pastures. W is used for producing hard metals, which are used in rock drills and metal-cutting tools, and for production of ferrotungsten in the steel industry. W-containing compounds are used as filaments for incandescent lamps, bronzes in pigments, and as catalysts in the petroleum industry.

Illness from low-level environmental or occupational W exposure has not been well documented. Little information is available regarding the toxicity of W. However, individuals occupationally exposed to W microparticles and/or fumes may develop serious lung disease known as "hard metal disease." Chelation may acutely increase urinary excretion of W.

Uranium

Urinary uranium (U) provides an indication of recent or ongoing exposure to the metal, and endogenous detoxification to a lesser extent. This test measures U238 which is the most abundant, naturally occurring U isotope.

All ten isotopes of U are radioactive; U-238 is the most abundant naturally occurring isotope and lowest energy emitter. It is important to note that the measured U-238 represents naturally occurring U, and does not indicate or imply exposure to highly enriched U-235 which is used in nuclear power and weaponry. U is a nonessential element that is abundant in rock, particularly granite. U is present at widely variable levels in drinking water, root vegetables, and high phosphate fertilizers. Some bottled waters may contain U, particularly those originating from mountain springs. Other sources of U include some ceramics, some colored glass, and some mine tailings. Uranium that is not excreted in urine may accumulate in bone and kidney tissues as well the liver. In excessive amounts, U can be nephrotoxic. Fatigue may be associated with chronic, low-level exposure to U.

Hair elements analysis may indicate exposure to U over the past 2-4 months. The level of U in tap and well water can be assessed with the Comprehensive Drinking Water Analysis test.

References

Aluminum

Krewski D, Yokel RA, Nieboer E et al. Human health risk assessment for aluminum, aluminum oxide and aluminum hydroxide. *J Toxicol Environ Hlth B Crit Rev* (2007)10:1-269.

Bassioni G, Mohammed FS, Zubaidy E et al. Risk assessment of using aluminum foil in food preparation. *Int J Electrochem Sci* (2012)7:4498-4519. <http://www.electrochemsci.org/papers/vol7/7054498.pdf>

Wang X, Cheng D, Jiang W et al. Mechanisms underlying aluminum neurotoxicity related to 14-3-3 ζ protein. *Toxicol Sci* (2018)163:45-56. doi: 10.1093/toxsci/kfy021

Antimony

Cooper RG and Harrison AP. The exposure to and health effects of antimony. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2822166/>

ATSDR Public Health Statement for Antimony <https://www.atsdr.cdc.gov/PHS/PHS.asp?id=330&tid=58>

Arsenic

ATSDR Public Health Statement for Arsenic <https://www.atsdr.cdc.gov/phs/phs.asp?id=18&tid=3>

W.H.O. Arsenic <https://www.who.int/news-room/fact-sheets/detail/arsenic>

Hong Y-S et al. Health effects of chronic arsenic exposure. *J Prev Med Public Hlth* (2014)47:245-52 <https://doi.org/10.3961/jpmph.14.035>

<https://fda.gov/files/food/published/Arsenic-in-Rice-and-Rice-Products-Risk-AssessmentReport-PDF.pdf>

Barium

A Review of the Health Impacts of Barium From Natural and Anthropogenic Exposure <https://pubmed.ncbi.nlm.nih.gov/24844320/>

ATSDR Tox Facts for Barium <https://www.atsdr.cdc.gov/toxfaqs/tf.asp?id=326&tid=57>

Beryllium

Beryllium Disease. <https://my.clevelandclinic.org/health/diseases/13807-beryllium-disease>

C.D.C. <https://www.cdc.gov/NIOSH/topics/beryllium/>

Bismuth

Wong H, Li H, Sun H. Bismuth: Environmental pollution and health effects. *Encyclopedia Environ Hlth* (2019) 415-23. <https://doi.org/10.1016/B978-0-12-409548-9.11870-6>

Cadmium

Mezynska M and MM Brzoska. Environmental exposure to cadmium- a risk for health of the general population in industrialized countries and preventive strategies. *Environ Sci Pollut Res Int* (2018)25:3211-32. <https://doi.org/10.1007/s11356-017-0827-z>

Wang Q and S Wei. Cadmium affects blood pressure and negatively interacts with obesity: Findings from NHANES 1999-2014. *Sci Total Environ* (2018)643:270-76. <https://doi.org/10.1016/j.scitotenv.2018.06.105>

Satarug S, Vesey DA, Gobe GC. Kidney cadmium toxicity, diabetes and high blood pressure: The perfect storm. *Tohoku J Expt Med* (2017)241:65-87. <https://doi.org/10.1620/tjem.241.65>

[Click here to return to the Table of Contents](#)

Cadmium in foods. ec.europa.eu/food/safety/chemical_safety/contaminants/catalogue/cadmium_en

Cesium

FDA Alert 2018 <https://fda.gov/drugs/human-drugs-human-drug-compounding/fda-alerts-health-care-professionals-significantsafety-risks-associated-cesium-chloride>

Session D et al. Fatal cesium chloride toxicity after alternative cancer treatment. *J Alt Comp Med* (2013)19:973-5. <https://doi.org/10.1089/ac m.2012.0745>

Gadolinium

Choi JW et al. Gadolinium deposition in the brain: Current Updates. *Korean J Radiol* (2019)20:134-147 <https://doi.org/10.3348/kjr.20180356>

Rogowski J et al. Gadolinium as a new emerging contaminant of aquatic environments. *Eviron Toxicol Chem* (2018) <https://doi.org/10.1002/etc.4116>

Gulani V et al. Gadolinium deposition in the brain: Summary of evidence and recommendations. *Lancet Neurol* (2017)16:564-70

Semelka RC et al. Gadolinium in humans: A family of disorders. *Am J Radiol* (2016)207 <https://doi.org/10.22/AJR.15.15842>

Lead

CDC. Lead Factsheet. <https://cdc.gov/biomonitoring/lead.factsheethtml>

Lead Exposure In Children Affects Brain And Behavior https://www.aacap.org/AACAP/Families_and_Youth/Facts_for_Families/FFF-Guide/Lead-Exposure-In-Children-Affects-Brain-And-Behavior-045.aspx

Abe E et al. Cadmium and lead in cocoa powder and chocolate products in the U.S. market. *Food Contam Part B Surrveil*(2018)2:92-102 <https://doi.org/10.1080/19393210.2017.1420700>

Taylor DA. Lead in cocoa products: Where does contamination come from? *Env Hlth Pespect* (2005)113:A687-88

Mercury

Ceccatelli S, Bose R, Edoff K et al. Long-lasting neurotoxic effects of exposure to methylmercury during development. *J Int Med* (2013)273:490-97. <https://doi.org/10.1111/joim.12045>

https://www.atsdr.cdc.gov/mercury/docs/physician_hg_flier.pdf (Accessed 7/6/20)

Health Effects of Exposures to Mercury <https://www.epa.gov/mercury/health-effects-exposures-mercury> (Accessed 7/6/20)

Nickel

Nickel Compounds. NCI (2019) <https://www.cancer.gov/about-cancer/causes-prevention/risk/substances/nickel>

<https://www.atsdr.cdc.gov/toxguides/toxguide-15.pdf>

Sharma AD. Low nickel diet in dermatology. *Ind J Dermatol* (2013)58:240 <https://doi.org/10.4103/0014-5154.110846>

Palladium

Chemical properties of palladium - Health effects of palladium - Environmental effects of palladium <https://www.lenntech.com/periodic/elements/pd.htm#ixzz6RWeYQV7B><http://www.lenntech.com/Periodic-chart-elements/Pd-en.htm>

International Programme on Chemical Safety (IPCS), Chemical Safety Information from Intergovernmental Organizations (INCHEM). Palladium. *Environmental Health Criteria* 226, 2002.

Platinum

Oun R, Moussa YE, Wheate NJ. The side effects of platinum-based chemotherapy drugs: A review for chemists. Dalton Trans (2018)47:6645-53. <https://doi.org/10.1039/c8dt00838h>

Tellurium

Tellurium -The Bright Future of Solar Energy <https://pubs.usgs.gov/fs/2014/3077/pdf/fs2014-3077.pdf>

<https://pubchem.ncbi.nlm.nih.gov/compound/Tellurium#section=Absorption-Distribution-and-Excretion>

<http://www.lenntech.com/Periodic-chart-elements/Te-en.htm> International Programme on Chemical Safety (IPCS), Chemical Safety Information from Intergovernmental Organizations (INCHEM). Tellurium. ICSC: 0986, April 2002.

Thallium

Karbowska B. Presence of thallium in the environment: sources of contaminations, distribution and monitoring methods. Environ Monit Assess (2016)188:640 <https://doi.org/10.1007/s10661-016-5647-y>

Nang Z et al. High accumulation and subcellular distribution of thallium in green cabbage. Int J Phytoremediation(2015)17:1097-104 <https://doi.org/10.1080/15226514.2015.1045633>

Thorium

EPA Facts about Thorium <https://semspub.epa.gov/work/HQ/175255.pdf>

ATSDR Toxicological Profile for Thorium 2019. <https://www.atsdr.cdc.gov/ToxProfiles/tp147.pdf>

<https://www.lenntech.com/periodic/elements/th.htm#ixzz6RXuXgl4k>

Wie Y, Li Z, Liu J et al. Levels of uranium and thorium in maternal scalp hair and risk of orofacial clefts in offspring. J Environ Radiac (2019)204:125-31. <https://doi.org/10.1016/j.jenvrad.2019.04.007>

Sahoo SK, Zunic Zs, Kritsananuwat R et al. Distribution of Uranium, Thorium and Some Stable Trace and Toxic Elements in Human Hair and Nails in Niška Banja Town, a High Natural Background Radiation Area of Serbia (Balkan Region, South-East Europe). J Environ Radioact (2015)145:66-77. <https://doi.org/10.1016/j.jenvrad.2015.03.020>

Tin

ATSDR/CDC. Toxic Substances Portal. <https://www.atsdr.cdc.gov/phs/phs.asp?id=541&tid=98>

NTP Research Report on Organotin and Total Tin Levels in Danish Women of Reproductive Age. Research Triangle Park (NC): National Toxicology Program. Natl Toxicol Program Res Rep Ser (2016)RR-02:1-27. <https://ntp.niehs.nih.gov/go/rr02abs>

Tungsten

Wasel O and JL Freeman. Comparative assessment of tungsten toxicity in absence and presence of other metals. Toxics (2018) <https://doi.org/10.3390/toxics6040066>

Consumer Wellness Center (2016). Heavy metals in rice protein products to be strictly limited thanks to a history-making agreement. <https://consumerwellness.org/PR21.html> [Accessed 4/28/20]

Uranium

Faroon KS et al. Public Health Statement for Uranium. <https://ncbi.nlm.nih.gov/books/NBK158804/>

Sahoo SK, Zunic Zs, Kritsananuwat R et al. Distribution of Uranium, Thorium and Some Stable Trace and Toxic Elements in Human Hair and Nails in Niška Banja Town, a High Natural Background Radiation Area of Serbia (Balkan Region, South-East Europe). J Environ Radioact (2015)145:66-77. <https://doi.org/10.1016/j.jenvrad.2015.03.020>

