Toxic Metals; Stool Resource Guide

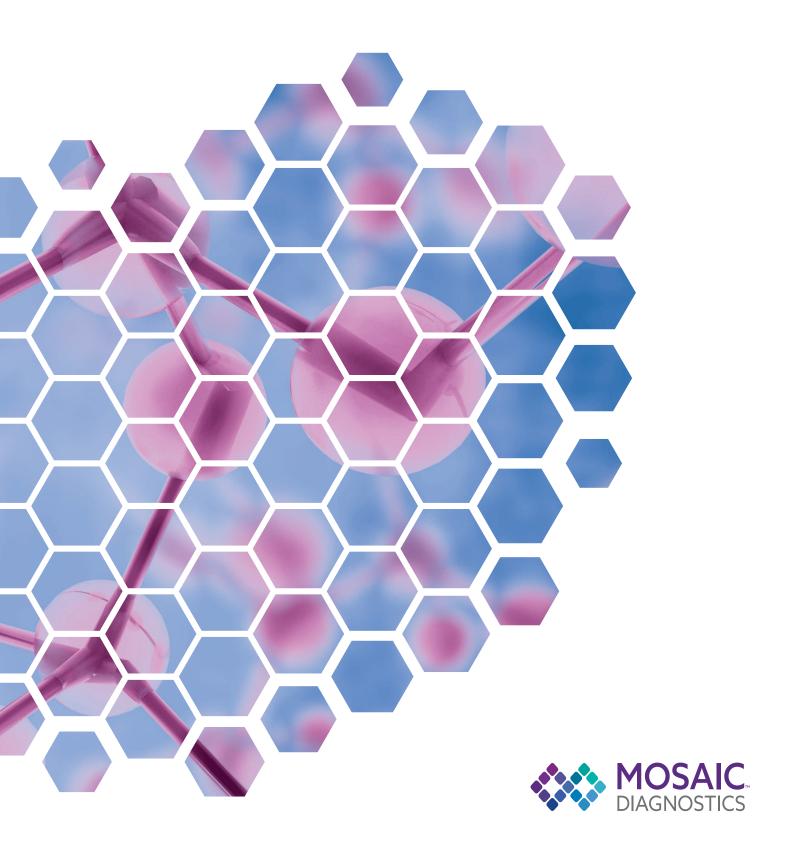


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Why test toxic metals in stool?

Oral exposure:

A Gram of Prevention Is Worth a Kilogram of Cure

Minding exposure to toxic substances is a primary goal for practitioners of preventive medicine and health conscious individuals. Contamination of food and water with toxic elements is the primary source of exposure for the general population.

Detoxification from the body:

The Biliary-fecal Route

Biliary secretion of several toxic elements from the liver into stool is the primary natural route of elimination from the body.

The results of stool elemental analysis can help identify and reduce oral exposure to toxic metals. Even healthy foods such as vegetables may be variably contaminated with toxic elements. It is not feasible to analyze all foods that one habitually consumes, but knowledge of metals in stool while consuming one's typical diet may help "clean up" the diet. Toxic elements such as mercury, cadmium, lead, antimony and uranium are primarily excreted naturally via the biliary-fecal route. However, oral exposure and relatively low absorption of toxic elements likely contributes substantially more to metals in stool than does biliary excretion.

Elements are measured by ICP-MS and expressed on a dry weight basis to eliminate variability related to water content of the specimen.

Sample Report



Toxic Metals; Stool

ANTIMONY

Fecal antimony (Sb) provides an indication of recent oral exposure to the element, and to a much lesser extent Sb that has been excreted from the body in bile. Sb is a nonessential element that is chemically similar to but less toxic than inorganic arsenic. Like arsenic, Sb is conjugated with glutathione and excreted in urine and feces.

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

Early signs of extensive exposure to Sb include: fatigue, muscle weakness, myopathy, nausea, low back pain, headache, and metallic taste. Hair elements analysis may provide evidence of Sb exposure over the past 2-4 months.

ARSENIC

Fecal Arsenic (As) provides an estimate of a very recent oral exposure to the element, and to a much lesser extent, As that has been excreted from the body in bile. Inorganic As accumulates in hair, nails, skin, thyroid gland, bone and the gastrointestinal tract. Non-toxic organic As (shellfish) is rapidly excreted, primarily in the urine and to a lesser extent in the feces.

Common sources of As include some rice and rice-based products, insecticides (calcium and lead arsenate), fungicides (orchards, vineyards), well water, smog, shellfish (arsenobetaine), and older wood preservatives (CCA; pressure treated wood/sawdust). There have been reports of arsenate exposure to pets and children associated with soil under older decks. Significant As exposure may be associated with malaise, muscle weakness, vomiting, diarrhea, and dermatitis. Extended exposure to As may increase risks for diabetes and cancer, and adversely affect the peripheral nervous, cardiovascular and hematopoietic systems.

Exposure to inorganic As over the past 2-4 months may be assessed with hair elemental analysis.

BERYLLIUM

Fecal beryllium (Be) provides an estimate of a recent oral exposure to the element. 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. Fecal Be is not diagnostic for berylliosis.

Possible sources of Be include electronic components, metal alloys used in aircraft and aerospace applications (especially aluminum-copper-beryllium alloys), bearing sleeves, optical lens coatings, and some phosphors in fluorescent lights. Tobacco contains Be, and smoking immediately increases Be levels in blood and urine.

Beryllium has been detected in hair but documentation correlating exposure, tissue levels and hair levels is lacking.

BISMUTH

Fecal bismuth (Bi) provides an estimate of a recent oral exposure to the element. Bi is a non-essential element of relatively low toxicity. Absorption is dependent upon solubility of the Bi compound, with insoluble Bi excreted in the feces while soluble forms are excreted primarily in the urine. Sources of Bi include: cosmetics (lipstick), Bi containing medications such as ranitidine Bi-citrate, antacids (Pepto-Bismol), pigments used in colored glass and ceramics, dental cement, and dry cell battery electrodes. Several organometallic Bi compounds are used for bactericidal and fungicidal applications.

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 result in nephrotoxicity (nephrosis, proteinurea) and neurotoxicity (tremor, memory loss, monoclonic jerks, dysarthria, dementia).

Urine elements analysis may be used to corroborate Bi absorption for a period of days or a few weeks after the exposure.

CADMIUM

Fecal cadmium (Cd) provides an estimate of very recent oral exposure to the element, and to a much lesser extent the biliary excretion of Cd from the body.

Cd absorption is suppressed to some extent with appropriate intake of zinc, calcium, and selenium. Cd is found in varying amounts in foods, from very low for some 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. Other sources of Cd include human biosolids, pigments and paints, batteries (Ni-Cd), plastics and synthetic rubber (tires).

Extensive bodily retention of Cd may be symptomatically associated with fatigue, weight loss, osteomalacia, and lumbar pain (Atai-Atai disease). Extensive exposure to Cd may adversely affect the kidneys, lungs, testes, arterial walls, and bones. Chronic Cd excess may lead to microcytic, hypochromic anemia, proteinuria, and functional zinc deficiency.

Urinary or serum beta-2-microglobulin is considered to be the best test for assessment of Cd-associated functional damage to the kidneys.

CESIUM

Fecal cesium (CS) provides an indication of recent oral exposure to the element, and to a much lesser extent Cs that has been excreted from the body in bile.

Naturally occurring Cs, the isotope measured at Doctor's Data, is not radioactive and is referred to as stable Cs (Cs133). Cesium is a naturally occurring element found combined with other elements in rocks, soil,

and dust in low amounts. Humans may be exposed to Cs at relatively low levels from air and diet. Cesiumchloride 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 fecal Cs have been observed at Doctor's Data for patients self-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.

Confirmatory tests for excessive exposure to Cs include serum electrolytes and blood or red blood cell elements analysis.

COPPER

Fecal copper (Cu) provides an estimate of very recent exposure to Cu, and to some extent biliary Cu excretion. The biliary / fecal route is the main route of excretion for Cu from the body.

Foods highest in Cu include organ meats (liver), shellfish, firm tofu, spirulina, beans/legumes, seeds and nuts, and dark chocolate. Other potential sources of Cu include contaminated food or drinking water, excessive Cu supplementation, occupational / environmental exposures, and wood preservatives. "Green" wood that has been treated with copper (AAC, ACQ, ACZA), and possibly chromium and arsenate (CCA) can be a source of those elements; respiratory and contact protection should be implemented when working with such exterior wood. There have been reports of significant metal exposure to pets and children associated with soil under decks.

Elemental analysis of whole blood, red blood cells and serum elements analysis provide direct indication of Cu status.

GADOLINIUM

Fecal gadolinium (Gd) provides an indication of Gd that has been excreted from the body in bile, and to a lesser extent oral exposure.

Gadolinium can be found in the environment in geographically variable amounts, and usually at very low levels. Gadolinium 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. Fecal 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 typically decrease very 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. Fecal Gd levels have not been well studied, but preliminary observations indicate that fecal levels of Gd also normally decrease sharply with time after administration of GBCAs (unpublished, Doctor's Data). 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 MRIs.

Gadolinium deposition disease (GDD) 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).

LEAD

Fecal lead (Pb) provides an indication of recent oral exposure to the element, and to a much lesser extent Pb that has been excreted from the body in bile. Absorbed Pb is excreted primarily in urine (76%) and bile (16%). Lead remains the most common clinically problematic toxic metal despite long past termination of its use in gasoline and paint. However, high levels of Pb have been found in soil under older bridges and overpasses due to sand blasting and refurbishing.

Most lead contamination occurs via oral ingestion of contaminated food or water, or by children mouthing or eating lead-containing objects such as imported children's trinkets and toys. Municipal drinking water

has become a significant source of Pb in certain parts of the country. Lead has been reported to be present in chocolate (the darker the higher), cocoa powders, and some chocolate flavored whey protein concentrates. In addition to some glazed pottery and lead crystal glass (drinking glasses/carafes), Pb may be present in dinnerware. Other sources of lead include: old lead paint (dust/chips), bullets and fishing tackle, batteries, computers, industrial smelting and alloying, ceramics, and artist paints and pigments (including certain tattoo inks).

The extent of oral absorption of Pb depends upon stomach contents (empty stomach increases uptake), and upon essential element status and dietary intake. Deficiency of zinc, calcium or iron may increase lead uptake. Transdermal exposure is slight, except for high absorption of lead acetate that may be present in hair darkening dyes.

Lead (Pb) has pathological, neurotoxic, nephrotoxic and carcinogenic effects that may be manifested with even 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.

The medical standard of care for assessment of lead exposure and toxicity is 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 P-induced disruption of heme biosynthesis (physiological impact). Doctor's Data offers a comprehensive drinking water test inclusive of lead.

MANGANESE

Fecal manganese (Mn) provides an indication of recent oral exposure to the element, and to a much lesser extent Mn that has been excreted from the body in bile.

Manganese is an essential trace element that is naturally present in many foods and available as a dietary supplement. Oral absorption is very low (< 3%), so the vast majority of fecal Mn represents unabsorbed Mn from foods and beverages. Low oral absorption is protective because excessive retention of Mn in the body may have neurological consequences.

Manganese is present in a wide variety of foods, including whole grains, clams, oysters, mussels, nuts, soybeans and other legumes, rice, leafy vegetables, coffee, tea, and many spices, such as black pepper. Drinking water also contains Mn at variable levels, and well water Mn levels may be high. Chronic consumption of water containing high levels of Mn may lead to Mn toxicity. Caffeinated and herbal teas tend to be high in Mn; content is higher with longer brewing/steeping time. Ninety-eight percent of absorbed Mn is normally excreted from the body via the biliary fecal route. High dietary Mn intake is not commonly associated with toxic effects of Mn unless excretion of Mn by the liver is compromised (biliary obstruction, liver failure). Manganese toxicity has occurred in people exposed to high amounts of Mn from chronic inhalation of Mn dust/fumes (e.g. welding and mining).

Hair elemental analysis may indicate the extent of Mn exposure over the past 2-4 months.

MERCURY

Fecal mercury (Hg) provides a good indication of recent or ongoing exposure to elemental Hg, and to a much lesser extent Hg that has been excreted from the body in bile. Data collected at Doctor's Data indicates a linear association between fecal Hq concentration and the number of amalgams currently in the mouth. Fecal Hq for subjects with 9 to 11 dental amalgams in place was 20-times greater than that of subjects without any dental amalgams in place (0.60 and 0.03 g/gram dry weight, respectively). Dental amalgams typically contain about 50% elemental Hq, and constant abrasion associated with chewing and bruxism releases very small particles of Hg which are poorly absorbed (about 5%) in the gastrointestinal tract. A direct association between fecal Hg levels and health has not been established, but a land mark study of amalgam placement in monkeys indicated there was an associated induction of co-resistance to both Hq and antibiotics by pathogenic bacteria in the gastrointestinal tract, particularly for species in the Enterobacteriaceae family. Such was also reported for miners exposed to elemental Hg while working in gold mines.

Methylmercury, which is abundant in predatory fish, is almost entirely absorbed and thereby does not show up nearly as prevalent in feces as does amalgam-derived inorganic Hg. In fact the presence of just a few dental amalgams precludes definitive contribution of fish consumption to total fecal Hq. The study conducted at Doctor's Data indicates that consumption of greater than 36 ounces of fish per month in subjects without dental amalgams was associated with a level of fecal Hg equivalent to that of subjects with only 1-2 amalgams (0.16 mg/kg dry weight).

The use of Hq in fungicides and pesticides (including that in marine paints) has declined due to environmental concerns, but residual Hq persists from past use. Except for fish, the human dietary intake of mercury is negligible unless food is contaminated with one of the previously mentioned forms/sources.

Analysis of Hg in red blood cells and hair provides a good estimate of sustained exposure to methymercury from fish; methylmercury partitions into those two matrices to a far greater extent than does inorganic Hg.

NICKEL

Fecal nickel (Ni) provides an estimate of very recent or ongoing oral exposure to the element. One to 10% of dietary Ni is be absorbed from the gastrointestinal tract into the blood; that which is not absorbed is excreted in the feces. Nickel is present to a minor extent in most dietary items and food is considered to be a major source of nickel exposure for the general population.

There is substantial evidence that Ni is an essential trace element which may be required in extremely low amounts. However, excessive assimilation of Ni has been well established to be nephrotoxic, and carcinogenic. With the exception of specific occupational exposures, most absorbed nickel comes from food and beverages, and intakes can vary due to a multitude of factors depending upon geographical location and water supply.

Nickel 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. Nickel is the most common cause of metal allergy among the people, and sensitization tends to persist life-long. Studies indicate the benefit of low dietary Ni diet in the management of nickel eczema. Careful selection of food with relatively low nickel concentration can help to control nickel dermatitis.

Other sources of Ni include: cigarettes, particulate diesel exhaust, Ni-Cd batteries, non-precious/ semiprecious dental materials, "fashion" jewelry, pigments (usually for ceramics or glass), arc and nickel refining and metallurgical processes.

PLATINUM

Platinum (Pt) is a nonessential element that is sometimes detected in feces. However, the clinical significance of high levels of Pt in feces has not been well studied.

Platinum is poorly absorbed in the gut and high level of oral exposure is unusual. Since it is a relatively rare element, most Pt exposures are of occupational origin. There may have been a slight increase in environmental Pt due to the use of Pt in automobile catalytic converters. Pt is a byproduct of copper refining and used as an alloy in some dental and orthopedic materials. High urinary Pt post-chelation has been reported at Doctor's Data for a few patients who had received platinum-based chemotherapy, and individuals who had been making high quality jewelry for years.

The platinum-based drugs cisplatin, carboplatin and oxaliplatin are commonly used intravenously in the treatment of cancer and while they are effective, their use is limited by their severe, dose-limiting side effects. The extent to which different platinum-based drugs and their metabolites are excreted in bile after infusion has not been well characterized, but Pt has a very long life-time in the body. The liver is a prominent site of retention.

Hair elements analysis may reveal exposure to Pt over the past 2-4 months.

THALLIUM

Fecal thallium (TI) provides an indication of TI that has been excreted from the body in bile, and to a lesser extent recent oral exposure to the element. The biliary fecal route is the primary route of TI excretion from the body, although about 35% is excreted in urine. Tl is rapidly and near completely absorbed when ingested, inhaled or brought into contact with skin. Thallium is a highly toxic heavy metal which is generally tasteless and odorless, and doesn't have physiological functions in the body.

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 TI observed at Doctor's Data have been associated with daily consumption of "green drinks" that were prepared at home from raw Brassicaceae vegetables. It should be noted that a statement 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 TI include tobacco, fly ash (coal), cement dust, some fertilizers, some artists' paints, semiconductors, and hazardous waste sites and landfills (nearby drinking water/soil). Thallium is also a byproduct from the smelting of copper, zinc and lead ores.

Symptoms associated with significant exposure to TI may include: fatigue, headaches, sleep disturbance, neuropathy, ataxia, depression, psychoses, and extreme loss of hair. Thallium 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.

TUNGSTEN

Fecal tungsten (W) provides an indication of recent oral exposure to the element, and to a much lesser extent W that has been excreted from the body in bile. About 50% of W appears to be rapidly absorbed from gastrointestinal tract, and excretion from the body is primarily via the urinary route. Tungsten is highly absorbed via inhalation.

W doesn't have physiological functions in the body. 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 background environmental exposures to W are from foods, and tungstate salts that may occur in drinking water. Unconfirmed information is suggestive that rice protein concentrates/rice-based glutenfree 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 (such "biosolids" may also used as fertilizer). Industrially W is used for producing hard metals, which are used in rock drills and metal-cutting tools, and for producing ferrotungsten in the steel industry. Tungsten compounds are used as lubricating agents, filaments for incandescent lamps, bronzes in pigments, and as catalysts in the petroleum industry.

Human illness from low-level environmental or occupational W exposure has not been well established. 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."

URANIUM

Fecal uranium (U) provides an indication of recent oral exposure to the element, and to a much lesser extent U that has been excreted from the body in bile. The levels of U in feces has been used to estimate total daily intake of U. Most U passes through the intestine unabsorbed. Excretion of U from the body occurs via bile and urine.

All 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 represents naturally occurring U-238, 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 very abundant in rock, particularly granite. Uranium is present at widely variable levels in drinking water, root vegetables, and present in 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.

Hair elements analysis may indicate exposure to U over the past 2-4 months.

References

Antimony

Fan Y-Y et al. Effect of storage temperature and duration on release of antimony and bisphenol A from polyethylene terephthalate drinking water bottles of China. Environ Pollution (2014)192:113-20 https://doi.org/10.1016/jenvpol.2014.05.012

Arsenic

Hong Y-S et al. Health effects of chronic arsenic esposure. 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

Beryllium

Public Health Statement for Beryllium. https://atsdr.cdc.gov/phs/phs.asp?id=339&tid=33

Bismuth

Borbinah C et al. Bismuth encephalopathy- a rare complication of long standing use of bismuth subsalicylate. BMC Neurol (2019)19:article #212

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

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-significant-safety-riskes-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

Copper

NIH 2020. Copper. https://ods.nih.gov/factsheets/Copper-HealthProfessional/

Public Health Statement for Copper. https://atsdr.cdc.gov/phs/phs.asp?id=204&tid=37

EPA 2019. Overview of wood preservative chemicals. https://epa.gov/ingredients-used-pesticide-products/overview-wood-preservative-chemicals

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/1022/AJR.15.15842

Lead

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

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

Manganese

Williams M et al. Toxicological Profile for Manganese. https://ncbi.nlm.nih.gov/books/NBK1588681

O'Neal SL and W Zheng. Manganese toxicity upon overexposure: a decade in review. Curr Environ HIth Rev (2015) https://10.1007/s40572-015-0056-x

Schwalfenberg G et al. The benefits and risks of consuming brewed tea: beware of toxic element contamination. J Toxicol (2013) https://doi.org/10.1155/2013/370460

Mercury

Skurink D et al. Is exposure to mercury a driving force for the carriage of antibiotic resistance genes? J Med Microbiol (2010)59:804-807 https://doi.org/10.1099/jmm.0.017665-0

Public Health Statement for Mercury. https://atsdr.cdc.gov/PHS/PHS.asp?id=112&tid=24

Mercury, hair. https://Mayomedicalabs.com/test-catalog/Overview/8498

Nickel

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

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

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

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/



