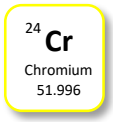
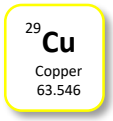
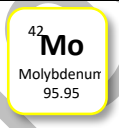
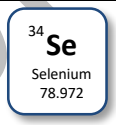
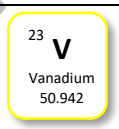
Metals & Traces®

HEAVY METALS & TRACE MINERALS EXTENDED PROFILE, URINE

Name:	Gender:	Age:
Date of Sampling:	Date of Analysis:	
Specimen: **Urine**	Patient Code Number:	

Essential Trace Elements

Code	TEST	RESULT		REFERENCE VALUES
1849	Chromium (**Cr**), Urine 	0.878 µg/g	●	0.550 – 4.830 µg/g Creatinine
1207	Cobalt (**Co**), Urine 	0.784 µg/g	●	< 5.000 µg/g Creatinine
1809	Copper (**Cu**), Urine 	9.717 µg/g	●	1.450 – 60.000 µg/g Creatinine
1313	Manganese (**Mn**), Urine 	**8.433 µg/g**	↑	< 4.500 µg/g Creatinine
1352	Molybdenum (**Mo**), Urine 	17.133 µg/g	●	9.700 – 100.000 µg/g Creatinine
1631	Selenium (**Se**), Urine 	**123.617 µg/g**	↑	12.000 – 90.000 µg/g Creatinine
348	Vanadium (**V**), Urine 	0.331 µg/g	●	< 1.000 µg/g Creatinine

Reference Values & Methods adapted from:
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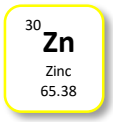
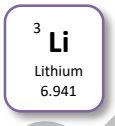
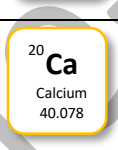
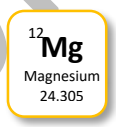
Name:	Gender:	Age:
Date of Sampling:	Date of Analysis:	
Specimen:	Patient Code Number:	

Essential Trace Elements & Microelements

Code	TEST	RESULT		REFERENCE VALUES
1854	Zinc (**Zn**), Urine 	**7.303 mg/g**	↑	0.060 – 0.780 mg/g Creatinine
1659	Strontium (**Sr**), Urine 	107.108 µg/g	●	< 200.000 µg/g Creatinine
1877	Iron (**Fe**), Urine 	23.115 µg/g	●	2.200 – 45.000 µg/g Creatinine
1878	Lithium (**Li**), Urine 	47.685 µg/g	●	< 175.000 µg/g Creatinine
328	Calcium (**Ca**), Urine 	180.304 mg/g	●	55.000 – 245.000 mg/g Creatinine
1318	Magnesium (**Mg**), Urine 	76.939 mg/g	●	12.000 – 150.000 mg/g Creatinine

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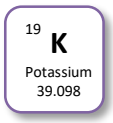
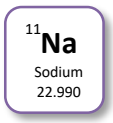
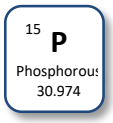
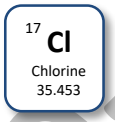
HEAVY METALS & TRACE MINERALS EXTENDED PROFILE, URINE

Name:	Gender:	Age:
Date of Sampling:	Date of Analysis:	
Specimen: **Urine**	Patient Code Number:	

Essential Trace Elements & Microelements

Code	TEST	RESULT		REFERENCE VALUES
1142	Potassium (**K**), Urine* 	47.119 mEq/g	●	22.00 – 82.00 mEq/g Creatinine
1411	Sodium (**Na**), Urine* 	117.202 mEq/g	●	43.50 – 226.00 mEq/g Creatinine
1806	Phosphorous (**P**), Urine** 	1282.843 mg/g	●	250.00 – 1300.00 mg/g Creatinine
1826	Chlorine (**Cl**), Urine** 	95.045 mg/g	●	40.00 – 224.00 mg/g Creatinine

***Method**: Ion-Selective Electrodes (ISE)
** **Method**: Spectrophotometry UV-Vis

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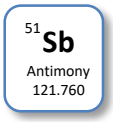
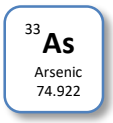
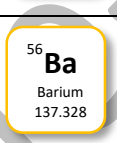
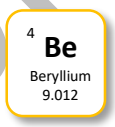
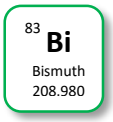
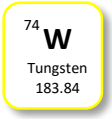
HEAVY METALS & TRACE MINERALS EXTENDED PROFILE, URINE

Name:	Gender:	Age:
Date of Sampling:	Date of Analysis:	
Specimen: **Urine**	Patient Code Number:	

Potentially Toxic Elements

Code	TEST	RESULT		REFERENCE VALUES
150	Aluminum (**Al**), Urine 	15.641 µg/g	●	< 40.000 µg/g Creatinine
228	Antimony (**Sb**), Urine 	0.009 µg/g	●	< 1.000 µg/g Creatinine
315	Silver (**Ag**), Urine 	0.315 µg/g	●	< 0.400 µg/g Creatinine
323	Arsenic (**As**)-Total, Urine 	**28.301 µg/g**	↑	< 15.000 µg/g Creatinine
359	Barium (**Ba**), Urine 	1.625 µg/g	●	< 5.700 µg/g Creatinine
380	Beryllium (**Be**), Urine 	0.183 µg/g	●	< 0.310 µg/g Creatinine
391	Bismuth (**Bi**), Urine 	0.066 µg/g	●	< 0.150 µg/g Creatinine
428	Tungsten (**W**), Urine 	0.502 µg/g	●	<0.790 µg/g Creatinine

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Name:	Gender:	Age:
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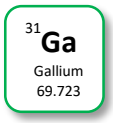
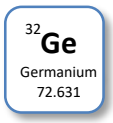
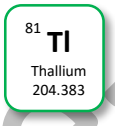
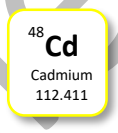
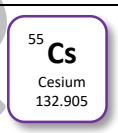
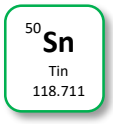
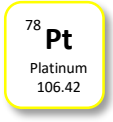
Specimen: **Urine**	Patient Code Number:	

Potentially Toxic Elements

Code	TEST	RESULT		REFERENCE VALUES
466	Gallium (**Ga**), Urine 	0.326 µg/g	●	< 0.500 µg/g Creatinine
481	Germanium (**Ge**), Urine 	0.022 µg/g	●	< 1.500 µg/g Creatinine
994	Zirconium (**Zr**), Urine 	0.109 µg/g	●	< 1.000 µg/g Creatinine
1031	Thallium (**Tl**), Urine 	0.224 µg/g	●	< 0.600 µg/g Creatinine
1129	Cadmium (**Cd**), Urine 	**0.834 µg/g**	↑	< 0.800 µg/g Creatinine
1138	Cesium (**Cs**), Urine 	5.270 µg/g	●	< 11.000 µg/g Creatinine
1185	Tin (**Sn**), Urine 	0.215 µg/g	●	< 2.000 µg/g Creatinine
1282	Platinum (**Pt**), Urine 	0.362 µg/g	●	< 0.600 µg/g Creatinine

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Name:	Gender:	Age:
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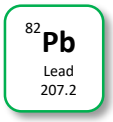
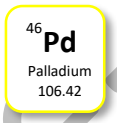
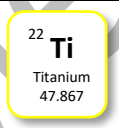
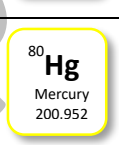
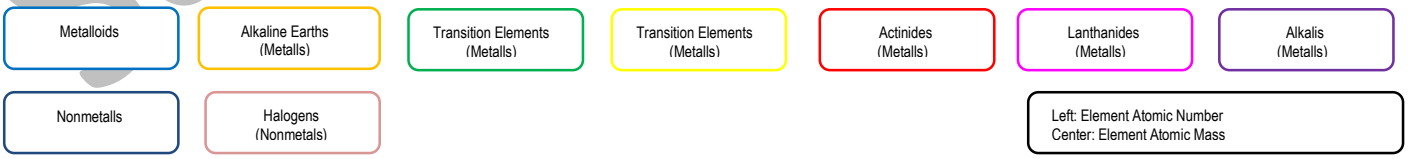
Specimen: **Urine**	Patient Code Number:	

Potentially Toxic Elements

Code	TEST	RESULT		REFERENCE VALUES
1359	Lead (**Pb**), Urine 	**10.766 µg/g**	↑	< 5.000 µg/g Creatinine
1416	Nickel (**Ni**), Urine 	**3.507 µg/g**	↑	< 3.000 µg/g Creatinine
1477	Uranium (**U**), Urine 	0.056 µg/g	●	< 0.060 µg/g Creatinine
1495	Palladium (**Pd**), Urine 	1.198 µg/g	●	< 1.400 µg/g Creatinine
1709	Titanium (**Ti**), Urine 	1.813 µg/g	●	< 13.000 µg/g Creatinine
1752	Mercury (**Hg**), Urine 	**2.175 µg/g**	↑	< 1.000 µg/g Creatinine

***ND** = Not Detected

The methodology used to measure all elements is **ICP-MS**: Inductively Coupled Plasma Mass Spectroscopy



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Metals & Traces®

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Name:	Gender:	Age:
Date of Sampling:	Date of Analysis:	
Specimen: **Urine**	Patient Code Number:	

General Notes and Comments

The information in this report is an interpretive aid to diagnostic procedures. The findings should relate to clinical examination, individual medical history, and possibly other diagnostic tests. Reference values have been obtained from the CDC (Center for Disease Control and Prevention, USA), WHO (World Health Organization), and other international agencies.

The results are reported in mg/g Creatinine for Macro-elements and in µg/g Creatinine for Trace elements and Heavy Metals. Normalization per g of Creatinine reduces the potentially large margin of error that can otherwise occur due to the sample collection process and sample volume variation. A Creatinine value of 0.30 mg/dL is the threshold for converting test results to mg/g and µg/g Creatinine. When Creatinine levels are lower (usually due to high fluid intake during urine collection), the cut-off value of 0.30 mg/dL is used for conversion.

High levels of toxic Heavy Metals in the urine usually reflect exposure to the corresponding poisonous metals in the past 48 hours. High levels of Trace Elements and Micro-elements are generally due to a high intake (from dietary supplements, mineral-fortified foods, superfoods, drugs, or certain foods). Heavy Metals become toxic to the body because they can replace other elements in the macromolecules of cells (e.g., enzymes, proteins, etc.). For example, Cadmium (Cd) toxicity occurs because it can replace Zinc (Zn) in many essential enzymes that contain Zinc in their structure, thus rendering them inactive.

Treatment with chelating agents or challenge with complexing agents increases urinary metal excretion. The maximum urinary excretion varies with the agent used since their ability to bind to the various metals differs considerably. Twenty-four hours before treatment, supplements containing mineral salts and products containing algae, drugs, or foods such as fish that may contain high levels of toxic metals, such as As or Hg, should be avoided. To maximize the detoxification process, it is crucial to understand the binding capacity of these factors. Since the maximum excretion of metals depends on the half-life of the chelating agent, an appropriate urine collection protocol must be followed.

All the above are general notes and comments.
Consult your Physician to interpret the results and administer the most appropriate treatment regimen for your case.

IMPORTANT NOTE: Special laboratory tests are performed for research purposes and as ancillary or supplementary tests in the context of a conventional laboratory test. They should only be used with other established medical data (e.g., medical history, symptoms, clinical examination, results of different lab tests, etc.).

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Metals & Traces®

HEAVY METALS & TRACE MINERALS EXTENDED PROFILE, URINE

Name:	Gender:	Age:
Date of Sampling:	Date of Analysis:	
Specimen: **Urine**	Patient Code Number:	

Special Comments

MANGANESE

The high manganese concentration in the urine may be due to excessive intake. Studies show that drinking black or green tea during chelating agent treatment and before urine collection can increase urinary excretion of manganese.

Biochemical basis

The main routes for manganese uptake, re-uptake, and excretion are bile, intestinal transport, and feces. Less than 0.5% of total manganese excretion typically occurs through urine, 3-5% through sweat, and the remaining approximately 95% through bile and feces. Therefore, the excretion of manganese in the urine may increase in patients with biliary obstruction or cirrhosis.

Manganese levels in urine can fluctuate without reflecting or affecting its stores in the body. In cases of overexposure to manganese, intravenous treatment with EDTA chelator is the method of choice. DMPS and DMSA have less binding capacity.

Pathophysiology

Excess manganese in urine without any challenge intervention is observed in patients with nephrotic syndrome, nephritis, biliary insufficiency or obstruction, and dietary overload or overuse of supplements. Some hormones and drugs inhibit the biliary excretion of manganese, resulting in increased excretion in the urine. Dopamine, glucagon, and cyclic AMP are some examples.

Environmental or industrial sources of manganese include mining, metal and ore cleaning and processing, foundries, glass industry, pigments, petrochemicals, plastics, synthetic industrial rubbers, certain types of batteries, and some gasoline additives. Groundwater used as drinking water may contain manganese, and studies show that manganese in urban drinking water ranges from < 5 to 350 µg/L. Manganese-rich waters promote bacterial growth.

Manganese neurotoxicity

Manganese can be neurotoxic. Symptoms of poisoning (especially after inhalation) include irritability, hallucinations, violence, tremors, Parkinson's symptoms, anorexia, sexual impotence, and speech disorders. Excess manganese can interfere with iron absorption, and if it occurs for a long time, it can lead to iron deficiency anemia.

Nutritional and Laboratory Information

Prolonged exposure to manganese increases the need for vitamin C. If manganese overexposure or toxicity is suspected, additional laboratory measurements are recommended, such as manganese levels in blood or hair.

SELENIUM

Urinary selenium measurements are good indicators of selenium toxicity, though in humans, toxicity is rare. The greatest level of selenium found in body fluids was 4900 mcg/L of selenium in urine. This was measured in a selenium worker after inhaling selenium dust. From these studies, Glover suggested that in public, rural and industrial health situations, selenium urinary levels should be below 100 mcg/l or approx. 100 mcg/g creatinine. Urine accounts for about one-half of the total body excretion of dietary selenium when normal amounts are ingested. Seafood, organ meats, cereal grains, and seleniferous vegetables (garlic, onions) are common dietary sources, but the Se-content of food greatly varies geographically. Because diets are highly variable in Se-content, urine measurements alone are not
diagnostically sufficient. Blood or hair levels may be used to substantiate findings. Selenium is also excreted in sweat, and in lesser amounts in fecal matters.

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Toxicity:
Excessive long-term exposure or excess supplementation can cause toxicity symptoms including alopecia, arthritis, atrophic, brittle nails, prevailing garlic breath and body odor, GI disorders, irritability, kidney impairment, metallic taste and yellowish skin. A garlic-like breath odor usually occurs. Because sulfur-containing amino acids and other sulfhydryl-based chelating agents such as DMSA have a capacity to bind selenium, they have the capacity to elevate urinary selenium levels in nonexposed persons above levels (100 mcg/l or mcg/g creatinine) of unprovoked urine.

ZINC

Elevated urinary levels may or may not correspond to excessive zinc intake or overload. They may also not correspond to zinc losses from body tissues because the major routes for zinc excretion are via bile, enteric transport, and feces. Typically, only 2 - 10% of total zinc excretion is via urine, with a similar proportion excreted in sweat. The remainder (approximately 80 to 95%) is excreted via biliary secretion and enteric transport.
Zinc Chelation Information: Urinary zinc levels (basal excretion, without the use of chelating agents) may fluctuate without reflecting or affecting body zinc stores. Very high urinary zinc levels are expected as a result of IV EDTA chelation therapy. On average, urinary zinc levels are increased to about 5 times above normal basal excretion levels, while DMPS chelation increases urinary zinc levels by 10-20 times. The binding capacity of DMSA for zinc is relatively low, with urinary levels being approximately 2 times that of basal excretion. Relatively insignificant zinc binding and loss are expected from using sulfhydryl-containing amino acids.
Pathophysiology: Tissue damage releases zinc into the extracellular fluid and increases urinary zinc levels. This can occur following or in association with injury, surgery, normal tissue catabolism following disease, starvation (ketosis), and diabetes mellitus. Significant zinc loss can occur in alcoholic cirrhosis.
Nutritional and biochemical information: True zinc overload and toxicity are rare. Depending on the chelating agent used, depletion of zinc stores in the body may occur if Zn supplementation is not used between chelation treatments.
Laboratory information: The measurement of zinc in serum or plasma is the most appropriate measure for assessing its status in the body. Hair levels reflect the zinc status in tissues.

ARSENIC

Environmental sources of arsenic exposure include food, water, soil and air. Arsenic is ubiquitous in the environment. Natural sources of the metal are mineral ores containing arsenic and groundwater (especially near areas with geothermal activity). In industry, arsenic is a by-product of the smelting process of many ores such as lead, gold, zinc, cobalt, and nickel. Other possible sources of arsenic exposure are:

- Commercial products: Wood preservatives, insecticides, herbicides, defoliants, fungicides, cotton desiccants, paints and pigments, ground paints, leaded gasoline, fireworks and flame-producing candy and beverage decorations
- Food: Wine (from grapes that have been sprayed with arsenic-containing pesticides) and seafood (especially bivalve mollusks, some cold-sea and bottom-feeding fish, algae)
- Smokers may also inhale small amounts of arsenic as a result of pesticide residues in tobacco leaves
- Industrial processes: purification of industrial gases (removal of sulfur), combustion of fossil fuels, combustion of wood treated with arsenic-containing preservatives, manufacture of electronics (microwave devices, lasers, LEDs, photoelectric panels and semiconductor devices), hardening of metal alloys, preservation of animal hides, copper industry, glass and ceramic industries
- Pharmaceutical: Fowler's solution (potassium arsenic), antiparasitic drugs (carbazone), Donovan's solution, some herbal remedies, superfoods, and preparations containing algae, some naturopathic remedies.

Laboratory Information
This reference value range only applies if no fish or products containing algae have been consumed 48 hours prior to urine collection. Mineral waters with high arsenic content may also increase levels of arsenic excretion in the urine. Eating foods such as those mentioned increases urinary arsenic levels significantly, at least 2-3 times above the given range.
Smoking can also increase urinary arsenic excretion levels.
Health Effects:

Reference Values & Methods adapted from:
1. Analytical Biochemistry, Holme & Peck, 3rd ed., 1998, Prentice Hall
2. Laboratory Tests and Diagnostic Procedures, Chernecky & Berger, 5th ed., 2008, Saunders Elsevier
3. Interpretation of Diagnostic Tests, Wallach, 8th ed., 2007, Lippincott

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- Acute arsenic toxicity may be associated with liver necrosis and elevated liver enzyme levels
- Gastrointestinal effects occur mainly after arsenic ingestion and less often after inhalation or absorption through the skin
- Arsenic can cause acute renal failure as well as chronic renal failure
- Long-term intake of arsenic in drinking water can lead to significant alterations of peripheral blood vessels
- Acute arsenic poisoning can cause both diffuse capillary leakage and cardiomyopathy resulting in shock
- Patients exposed to arsenic may develop axonal destruction leading to peripheral neuropathy
- Color changes and palmoplantar hyperkeratosis are characteristic of chronic arsenic exposure
- Benign arsenic keratosis can develop into malignancy
- Inhalation of high concentrations of arsenic compounds creates irritation of the respiratory mucosa
- Arsenic exposure has been linked to an increased incidence of spontaneous abortions and congenital malformations
- Arsenic carcinogenesis in humans has been documented, but no animal model has yet been developed
- The silent period for skin cancer associated with arsenic ingestion can be 3 to 4 decades, while non-cancerous skin effects usually develop several years after exposure

In workers exposed to arsenic, there is a systematic increase in lung cancer mortality rates, depending on the duration and intensity of exposure.
This measurement is for the total arsenic in the sample (i.e., inorganic arsenic which is toxic plus organic – protein bound – which is practically non-toxic).

CADMIUM
Cadmium is toxic to almost every system in the body. Only 1 to 5% of ingested cadmium is absorbed from the gastrointestinal tract. However, cadmium is inhaled (as smoke) and is rapidly transported from the blood to the liver, kidneys, intestinal mucosa and other tissues. Cadmium binds primarily to the low molecular weight protein metallothionein in all tissues where it inhibits enzymes and nutrient utilization and competes with zinc for binding sites on various enzymes and other proteins. After long-term exposure, cadmium accumulates in the kidneys and liver, and the cadmium-metallothionein complex has been shown to be toxic to the kidney.
Clinical signs and symptoms
Acute poisoning by inhalation of cadmium vapors causes severe pulmonary and bronchial irritation, nausea and vomiting. Repeated episodes can lead to emphysema of the lungs and death.
Excessive intake of contaminated food or drink in workers exposed to cadmium dust may result in acute gastrointestinal irritation, nausea, vomiting, abdominal cramps, and diarrhea.
Chronic poisoning mainly affects the kidneys. The first indications are glycosuria, aminooxyuria, hypertension and other symptoms of kidney disease. Lung diseases such as emphysema and pulmonary fibrosis may occur. Chronic poisoning can also affect the bones (itai-itai disease) and lead to cancer.
Laboratory investigation
Medical monitoring of exposed individuals includes screening for blood and urine cadmium, standard hematologic and biochemical tests, and measurement of urine proteins.
The excretion of cadmium is slow and mainly through the urine. Unexposed subjects excrete < 2 μg/g creatinine per day. Due to its stable tissue binding, cadmium has a long biological half-life, estimated to be between 20-30 years. The half-life in blood is estimated to be 2.5 months and therefore blood levels are only useful as indicators of recent exposure.
Smoking increases susceptibility to toxicity and tissue levels of cadmium are known to increase with age. Smokers have 50% higher rates of poisoning than non-smokers.
Hair testing for cadmium (as with other metals) reflects tissue levels, but when elevated levels of the metal are seen in hair, levels in the kidneys, liver and lungs are significantly higher.
Water contamination is a recognized source of cadmium in the body.
Chelation information
Various studies in smokers and non-smokers have shown that combined treatments with EDTA + DMSA and DTPAs + DMPS have a good capacity to bind cadmium, resulting in its removal from the body.

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LEAD

Occupational and environmental exposure are the most common causes of high lead concentrations in urine.
Common sources include leaded gasoline, canned goods, paints, newsprint, smoke, air pollution, and contaminated water.
Biochemical toxicity base: Only 8 to 12% of ingested lead is absorbed by the small intestine, but the toxic effects are severe. Lead can react with sulfhydryl groups in enzymes, resulting in the inactivation of essential enzymes such as aminolevulinic acid dehydrase (ALA) and ALA synthase, leading to hematological manifestations. Lead reduces the body's ability to use calcium, magnesium, zinc, iron, and other vital nutrients. Children readily absorb lead.
Clinical signs: The pathological effects of Pb have been recognized for centuries. Lead affects all physiological systems, including the kidney, nervous, reproductive, endocrine, immune, and hematopoietic systems. Exposure to lead, whether chronic or acute, presents a variety of symptoms, signs, and chemical signs. The exposed person may be asymptomatic or symptomatic.

- Mild symptoms include fatigue, lack of energy, constipation, mild abdominal pain and discomfort, anorexia, sleep disturbances, irritability, anemia, hair loss, paleness, and, less commonly, diarrhea and nausea. The formation and precipitation of lead sulfide can be displayed as a blue-black "lead line" at the base of teeth near the gums.
- Severe symptoms include colic, decrease in muscle strength, muscle tenderness, and symptoms of neuropathy and encephalopathy.
- Common symptoms in children are irritability, vomiting, abdominal pain, ataxia, anorexia, behavioral disorders, speech disorders, seizures, fever, and dehydration.

Treatment: Various chelating agents such as EDTA, dimercaprol (BAL), and penicillamine have been suggested. The FDA has approved the use of oral DMSA for detoxification of children. Various publications show that DMPS, ZnDTPA, and CaDTPA are highly effective chelating agents with a binding capacity equal to or greater than BAL and penicillamine. These data also show that combination therapy with EDTA + DMSA can bind lead and significantly increase its excretion. Vitamin C and cysteine have good lead-binding ability and can be used as supportive measures during chelation treatment.
Laboratory Analysis: There are two categories of exposure:

1. Professional exhibition
   - The case of occupational lead overexposure is defined (for adults - age 15 years and over) as the lead concentration in the blood greater than or equal to 25 μg/dL or 250 μg/L.
   - Lead poisoning in children is defined as lead blood levels above 10 μg/dL. Long-term effects can include slow growth, decreased IQ (IQ), learning disabilities, hearing loss, reduced height, and hyperactivity. Most children with lead overexposure have no symptoms. Symptoms resembling other childhood diseases often appear. Very severe exposure to lead (levels greater than 80 μg/dL) can cause coma, convulsions, and even death.

2. Chronic low-level exposure
   - Blood lead levels may or may not be elevated, depending on direct exposure.
   - Long-term exposed individuals usually have elevated metal levels in the hairs.

Lead concentrations in blood and urine are about 10 to 20 percent higher in men than women in children and adults.

NICKEL

Tobacco, smoking, and eating are the major sources of exposure to nickel.
Elevated urinary nickel levels reflect increased direct exposure.
Studies show that the greatest nickel binding has been seen with the combined EDTA + DMSA treatment.
Environmental / Professional Resources
Nickel is found in atmospheric air at very low levels as a result of emissions from manufacturing facilities, coal and oil burning, sewage sludge incineration and other sources.
Exposure can be through contact with objects of daily use, such as nickel containing jewelry, stainless steel cookware and metal elements in clothing (fasteners, buttons, etc.).
Toxicity and symptoms
Nickel carbonyl is the most toxic nickel compound, also found in cigarette smoke. Symptoms include headache, vertigo, nausea, vomiting, insomnia, and irritability, followed by chest pains, dry cough, cyanosis, gastrointestinal symptoms, sweating, visual disturbances and severe weakness.
The lungs and kidneys appear to be the target organs for acute nickel carbonyl toxicity in humans and animals.

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US and European health agencies consider nickel carbonyl to be a significant pollutant, based on the severe acute toxicity it causes.

Chronic effects (other than cancer)

Contact dermatitis is the most common effect from nickel exposure on humans and has been reported following occupational and non-occupational exposure, with symptoms of itching on the fingers, wrists, and forearms.

Chronic exposure to nickel in humans also leads to respiratory effects, including asthma due to primary irritation or allergic reaction and increased risk of chronic respiratory tract infections.

Risk of cancer

Human studies report an increased risk of lung and nasal cancer among workers in the nickel processing industry exposed to nickel dust. Nickel powder is a mixture of several nickel compounds, including nickel hyposulfite. The EPA (Environmental Protection Agency, USA) has classified nickel dust and nickel hyposulfite as carcinogens.

Nickel carbonyl has been reported to cause lung tumors in rats exposed through inhalation.

MERCURY

Mercury compounds easily react with sulfhydryl groups in proteins, resulting in the inhibition of their functional action. Both organic and inorganic mercury are equally potent toxic elements. Excretion levels < 20 μg/g creatinine may be seen in asymptomatic and symptomatic patients.

Signs and symptoms of toxicity: The most common causes of exposure to mercury at toxic levels are those related to industrial use, i.e., accidental, or chronic exposure. Exposure to mercury causes toxic effects due to its accumulation in the brain. Neurological signs may include increased agitation, severe behavioral and personality changes, insomnia, and memory loss. Hg levels > 100 μg/g creatinine in random urine prior to chelation are representative of acute exposure and indicative of toxicity. Gastrointestinal disturbances and kidney damage are likely to occur. A dose equal to 1 gram of mercury salt is considered fatal for humans.

Chronic mercury poisoning results in atrophy and degeneration of the sensory cerebral cortex. Other damage to the brain and nervous system, hearing and vision may also occur.

Early symptoms of chronic overexposure: Insomnia, dizziness, fatigue, drowsiness, weakness, depression, tremors, loss of appetite, memory loss, nervousness, headache, dermatitis, numbness and tingling of hands and feet, emotional instability, and kidney damage.

Laboratory information: Mercury remains in the bloodstream for 24 to 72 hours, and high blood levels confirm immediate and acute exposure.

Chelating Agents: All sulfhydryl chelating agents increase the urinary excretion of Mercury, even when there is a normal or even low concentration of the metal in the tissues or blood. Chelating agents such as DMPS (2,3-Dimercapto-1-Propenesulfonic Acid, Unithiol), DMSA (Dimercapto-Succinic Acid), ZnDTPA and cysteine are known to have good to excellent mercury binding capacity, resulting in increasing its excretion through urine. The chelating agent EDTA does not bind mercury significantly. Comparing urinary Hg levels before and after receiving the chelating agents allows monitoring the patient's response to treatment.

Sources: Overexposure can come from paints, explosives, electrical appliances, batteries, mercury-containing diuretics, fungicides, fluorescent lamps, cosmetics, hair dyes, dental amalgams, contaminated seafood, petroleum products, and thimerosal-containing vaccines. Broken mercury thermometers and other devices that use mercury, including batteries and fluorescent lamps, can also lead to excess mercury exposure.

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