Overview

Useful For
Detecting exposure to arsenic, lead, cadmium, and mercury

Profile Information

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<td>Mercury, B</td>
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<td>Patient Demographics</td>
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Testing Algorithm
See Porphyria (Acute) Testing Algorithm in Special Instructions.

Special Instructions
- Lead and Heavy Metals Reporting
- Trace Metals Analysis Specimen Collection and Transport
- Porphyria (Acute) Testing Algorithm

Method Name
Inductively Coupled Plasma-Mass Spectrometry (ICP-MS)

NY State Available
Yes

Specimen

Specimen Type
Whole blood

Necessary Information
If not ordering electronically, the Lead and Heavy Metals Reporting (T491) form is required. Send with specimen.

Specimen Required
Patient Preparation: High concentrations of gadolinium and iodine are known to interfere with most metals tests. If either gadolinium- or iodine-containing contrast media has been administered, a specimen should not be collected for 96 hours.

Supplies: Metal Free B-D Tube (EDTA), 6 mL (T183)

Container/Tube: Royal blue-top (EDTA) plastic trace element blood collection tube
**Specimen Volume:** Full tube

**Collection Instructions:**

1. See [Trace Metals Analysis Specimen Collection and Transport](#) in Special Instructions for complete instructions.

2. Send specimen in original collection tube.

**Additional Information:** If ordering the trace element blood collection tube from BD, order catalog #368381.

**Forms**

[Lead and Heavy Metals Reporting](#) (T491) in Special Instructions

**Specimen Minimum Volume**

0.3 mL

**Reject Due To**

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<td>Gross icterus</td>
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**Specimen Stability Information**

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**Clinical and Interpretive**

**Clinical Information**

Arsenic:

Arsenic (As) exists in a number of toxic and nontoxic forms. The toxic forms are the inorganic species As(V), also denoted as As(5+), the more toxic As(3+), also known as As(III), and their partially detoxified metabolites, monomethylarsine (MMA) and dimethylarsine (DMA). Detoxification occurs in the liver as As(3+) is oxidized to As(5+) and then methylated to MMA and DMA. As a result of these detoxification steps, As(3+) and As(5+) are found in the urine shortly after ingestion, whereas MMA and DMA are the species that predominate more than 24 hours after ingestion.

Blood concentrations of arsenic are elevated for a short time after exposure, after which arsenic rapidly disappears into tissues because of its affinity for tissue proteins. The body treats arsenic like phosphate, incorporating it wherever phosphate would be incorporated. Arsenic "disappears" into the normal body pool of phosphate and is excreted at the same rate as phosphate (excretion half-life of 12 days). The half-life of inorganic arsenic in blood is 4 to 6 hours, and the half-life of the methylated metabolites is 20 to 30 hours. Abnormal blood arsenic concentrations (>12 ng/mL) indicate significant exposure, but will only be detected immediately after exposure. Arsenic is not likely to be detected
in blood specimens drawn more than 2 days after exposure because it has become integrated into nonvascular tissues. Consequently, blood is not a good specimen to screen for arsenic, although periodic blood levels can be determined to follow the effectiveness of therapy. Urine is the preferred specimen for assessment of arsenic exposure.

A wide range of signs and symptoms may be seen in acute arsenic poisoning including headache, nausea, vomiting, diarrhea, abdominal pain, hypotension, fever, hemolysis, seizures, and mental status changes. Symptoms of chronic poisoning, also called arseniasis, are mostly insidious and nonspecific. The gastrointestinal tract, skin, and central nervous system are usually involved. Nausea, epigastric pain, colic (abdominal pain), diarrhea, and paresthesias of the hands and feet can occur.

Lead:

Lead is a heavy metal commonly found in man's environment that can be an acute and chronic toxin.

Lead was banned from household paints in 1978 but is still found in paint produced for nondomestic use and in artistic pigments. Ceramic products available from noncommercial suppliers (such as local artists) often contain significant amounts of lead that can be leached from the ceramic by weak acids such as vinegar and fruit juices. Lead is found in dirt from areas adjacent to homes painted with lead-based paints and highways where lead accumulates from use of leaded gasoline. Use of leaded gasoline has diminished significantly since the introduction of nonleaded gasolines that have been required in personal automobiles since 1972. Lead is found in soil near abandoned industrial sites where lead may have been used. Water transported through lead or lead-soldered pipe will contain some lead with higher concentrations found in water that is weakly acidic. Some foods (for example: moonshine distilled in lead pipes) and some traditional home medicines contain lead.

The typical diet in the United States contributes 1 to 3 mcg of lead per day, of which 1% to 10% is absorbed; children may absorb as much as 50% of the dietary intake, and the fraction of lead absorbed is enhanced by nutritional deficiency. The majority of the daily intake is excreted in the stool after direct passage through the gastrointestinal tract. While a significant fraction of the absorbed lead is rapidly incorporated into bone and erythrocytes, lead ultimately distributes among all tissues, with lipid-dense tissues such as the central nervous system being particularly sensitive to organic forms of lead. All absorbed lead is ultimately excreted in the bile or urine. Soft-tissue turnover of lead occurs within approximately 120 days.

Lead expresses its toxicity by several mechanisms. It avidly inhibits aminolevulinic acid dehydratase and ferrochelatase, 2 of the enzymes that catalyze synthesis of heme; the end result is decreased hemoglobin synthesis resulting in anemia. Lead also is an electrophile that avidly forms covalent bonds with the sulfhydryl group of cysteine in proteins. Thus, proteins in all tissues exposed to lead will have lead bound to them. The most common sites affected are epithelial cells of the gastrointestinal tract and epithelial cells of the proximal tubule of the kidney.

Avoidance of exposure to lead is the treatment of choice. However, chelation therapy is available to treat severe disease. Oral dimercaprol may be used in the outpatient setting except in the most severe cases.

Cadmium:

The toxicity of cadmium resembles the other heavy metals (arsenic, mercury, and lead) in that it attacks the kidney; renal dysfunction with proteinuria with slow onset (over a period of years) is the typical presentation. Breathing the fumes of cadmium vapors leads to nasal epithelial deterioration and pulmonary congestion resembling chronic emphysema.

The most common source of chronic exposure comes from spray painting of organic-based paints without use of a protective breathing apparatus; auto repair mechanics represent a susceptible group for cadmium toxicity. In addition, another common source of cadmium exposure is tobacco smoke.
Mercury:

Mercury (Hg) is essentially nontoxic in its elemental form. If Hg(0) is chemically modified to the ionized, inorganic species, Hg(2+), it becomes toxic. Further bioconversion to an alkyl Hg, such as methyl Hg ([CH3Hg](+)), yields a species of mercury that is highly selective for lipid-rich tissue such as neurons and is very toxic. The relative order of toxicity is:

Not Toxic -- Hg(0) < Hg(2+) << [CH3Hg](+) -- Very Toxic

Mercury can be chemically converted from the elemental state to the ionized state. In industry, this is frequently done by exposing Hg(0) to strong oxidizing agents such as chlorine. Hg(0) can be bioconverted to both Hg(2+) and alkyl Hg by microorganisms that exist both in the normal human gut and in the bottom sediment of lakes, rivers, and oceans. When Hg(0) enters bottom sediment, it is absorbed by bacteria, fungi, and small microorganisms; they metabolically convert it to Hg(2+), [CH3Hg](+), and (CH3)(2+)Hg. Should these microorganisms be consumed by larger marine animals and fish, the mercury passes up the food chain in rather toxic form.

Mercury expresses its toxicity in 3 ways:

-Hg(2+) is readily absorbed and reacts with sulfhydryl groups of protein, causing a change in the tertiary structure of the protein—a stereoisomeric change with subsequent loss of the unique activity associated with that protein. Because Hg(2+) becomes concentrated in the kidney during the regular clearance processes, this target organ experiences the greatest toxicity.

-With the tertiary change noted previously, some proteins become immunogenic, eliciting a proliferation of T lymphocytes that generate immunoglobulins to bind the new antigen; collagen tissues are particularly sensitive to this.

-Alkyl Hg species, such as [CH3Hg](+), are lipophilic and avidly bind to lipid-rich tissues such as neurons. Myelin is particularly susceptible to disruption by this mechanism.

Members of the public will occasionally become concerned about exposure to mercury from dental amalgams. Restorative dentistry has used a mercury-silver amalgam for approximately 90 years as a filling material. A small amount of mercury (2-20 mcg/day) is released from a dental amalgam when it was mechanically manipulated, such as by chewing. The habit of gum chewing can cause release of mercury from dental amalgams greatly above normal. The normal bacterial flora present in the mouth converts a fraction of this to Hg(2+) and [CH3Hg](+), which was shown to be incorporated into body tissues. The World Health Organization safety standard for daily exposure to mercury is 45 mcg/day. Thus, if one had no other source of exposure, the amount of mercury released from dental amalgams is not significant. (1) Many foods contain mercury. For example, commercial fish considered safe for consumption contain less than 0.3 mcg/g of mercury, but some game fish contain more than 2.0 mcg/g and, if consumed on a regular basis, contribute to significant body burdens.

Therapy is usually monitored by following urine output; therapy may be terminated after urine excretion is below 50 mcg/day.

Reference Values

ARSENIC

<13 ng/mL

Reference values apply to all ages.

LEAD
All ages: <5.0 mcg/dL

Critical values

Pediatrics (< or =15 years): > or =20.0 mcg/dL

Adults (> or =16 years): > or =70.0 mcg/dL

CADMIUM

<5.0 ng/mL

Reference values apply to all ages.

MERCURY

Â <10 ng/mL

Reference values apply to all ages.

**Interpretation**

**Arsenic:**

Abnormal blood arsenic concentrations (>12 ng/mL) indicate significant exposure.

Absorbed arsenic is rapidly distributed into tissue storage sites with a blood half-life of <6 hours. Unless a blood specimen is drawn within 2 days of exposure, arsenic is not likely to be detected in a blood specimen.

**Lead:**

The 95th percentile of the Gaussian distribution of whole blood lead concentration in a population of unexposed adults is <6.0 mcg/dL. For pediatric patients, there may be an association with blood lead values of 5.0 to 9.9 mcg/dL and adverse health effects. Follow-up testing in 3 to 6 months may be warranted. Chelation therapy is indicated when whole blood lead concentration is >25.0 mcg/dL in children or >45.0 mcg/dL in adults.

The Occupational Safety and Health Administration has published the following standards for employees working in industry:

- Employees with a single whole blood lead result >60.0 mcg/dL must be removed from workplace exposure.

- Employees with whole blood lead levels >50.0 mcg/dL averaged over 3 blood samplings must be removed from workplace exposure.

- An employee may not return to work in a lead exposure environment until their whole blood lead level is <40 mcg/dL.

New York State has mandated inclusion of the following statement in reports for children under the age of 6 with blood lead in the range of 5.0 to 9.9 mcg/dL: "Blood lead levels in the range of 5.0-9.9 mcg/dL have been associated with adverse health effects in children aged 6 years and younger."

**Cadmium:**
Normal blood cadmium is <5.0 ng/mL, with most results in the range of 0.5 to 2.0 ng/mL.

Acute toxicity will be observed when the blood level exceeds 50 ng/mL.

Mercury:

The quantity of mercury (Hg) found in blood and urine correlates with degree of toxicity. Hair analysis can be used to document the time of peak exposure if the event was in the past.

Normal whole blood mercury is usually <10 ng/mL.

Individuals who have mild exposure during work, such as dentists, may routinely have whole blood mercury levels up to 15 ng/mL.

Significant exposure is indicated when the whole blood mercury is >50 ng/mL if exposure is due to alkyl Hg, or >200 ng/mL if exposure is due to Hg(+2).

Cautions
No significant cautionary statements

Clinical Reference


Performance
Method Description
Arsenic (As), cadmium (Cd), mercury (Hg), and lead (Pb) are analyzed by inductively coupled plasma-mass spectrometry (ICP-MS) in kinetic energy discrimination (KED) mode using helium as a nonreactive gas to collide with polyatomic interferences such as argon chloride (ArCl). Internal standards used are gallium (Ga) for As, rhodium (Rh) for Cd, and lutetium (Lu) and iridium (Ir) summed for Hg and Pb. A salt matrix calibration is used. (Unpublished Mayo method)

PDF Report
No

Day(s) and Time(s) Test Performed
Monday through Saturday; 2 p.m.

Analytic Time
1 day

Maximum Laboratory Time
3 days

Specimen Retention Time
14 days

Performing Laboratory Location
Rochester

Fees and Codes

Fees
- Authorized users can sign in to Test Prices for detailed fee information.
- Clients without access to Test Prices can contact Customer Service 24 hours a day, seven days a week.
- Prospective clients should contact their Regional Manager. For assistance, contact Customer Service.

Test Classification
This test was developed and its performance characteristics determined by Mayo Clinic in a manner consistent with CLIA requirements. This test has not been cleared or approved by the U.S. Food and Drug Administration.

CPT Code Information
82175
82300
83655
83825

LOINC® Information
### Test Definition: HMDB

Heavy Metals Scrn with Demographics

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