WAC 296-62-07441 Appendix A, substance safety data sheet—Cadmium. (1) Substance identification.

(a) Substance: Cadmium.

(b) 8-Hour, time-weighted-average, permissible exposure limit (TWA PEL):

(c) TWA PEL: Five micrograms of cadmium per cubic meter of air 5 $\mu g/m^3$, time-weighted average (TWA) for an 8-hour workday.

(d) Appearance: Cadmium metal—soft, blue-white, malleable, lustrous metal or grayish-white powder. Some cadmium compounds may also appear as a brown, yellow, or red powdery substance.

(2) Health hazard data.

(a) Routes of exposure. Cadmium can cause local skin or eye irritation. Cadmium can affect your health if you inhale it or if you swallow it.

(b) Effects of overexposure.

(i) Short-term (acute) exposure: Cadmium is much more dangerous by inhalation than by ingestion. High exposures to cadmium that may be immediately dangerous to life or health occur in jobs where workers handle large quantities of cadmium dust or fume; heat cadmium-containing compounds or cadmium-coated surfaces; weld with cadmium solders or cut cadmium-containing materials such as bolts.

(ii) Severe exposure may occur before symptoms appear. Early symptoms may include mild irritation of the upper respiratory tract, a sensation of constriction of the throat, a metallic taste and/or a cough. A period of one to ten hours may precede the onset of rapidly progressing shortness of breath, chest pain, and flu-like symptoms with weakness, fever, headache, chills, sweating, and muscular pain. Acute pulmonary edema usually develops within twenty-four hours and reaches a maximum by three days. If death from asphyxia does not occur, symptoms may resolve within a week.

(iii) Long-term (chronic) exposure. Repeated or long-term exposure to cadmium, even at relatively low concentrations, may result in kidney damage and an increased risk of cancer of the lung and of the prostate.

(c) Emergency first-aid procedures.

(i) Eye exposure: Direct contact may cause redness or pain. Wash eyes immediately with large amounts of water, lifting the upper and lower eyelids. Get medical attention immediately.

(ii) Skin exposure: Direct contact may result in irritation. Remove contaminated clothing and shoes immediately. Wash affected area with soap or mild detergent and large amounts of water. Get medical attention immediately.

(iii) Ingestion: Ingestion may result in vomiting, abdominal pain, nausea, diarrhea, headache, and sore throat. Treatment for symptoms must be administered by medical personnel. Under no circumstances should the employer allow any person whom they retain, employ, supervise, or control to engage in therapeutic chelation. Such treatment is likely to translocate cadmium from pulmonary or other tissue to renal tissue. Get medical attention immediately.

(iv) Inhalation: If large amounts of cadmium are inhaled, the exposed person must be moved to fresh air at once. If breathing has stopped, perform cardiopulmonary resuscitation. Administer oxygen if available. Keep the affected person warm and at rest. Get medical attention immediately.

(v) Rescue: Move the affected person from the hazardous exposure. If the exposed person has been overcome, attempt rescue only after no-

tifying at least one other person of the emergency and putting into effect established emergency procedures. Do not become a casualty yourself. Understand your emergency rescue procedures and know the location of the emergency equipment before the need arises.

(3) Employee information.

(a) Protective clothing and equipment.

(i) Respirators: You may be required to wear a respirator for nonroutine activities; in emergencies; while your employer is in the process of reducing cadmium exposures through engineering controls; and where engineering controls are not feasible. If air-purifying respirators are worn, they must have a label issued by the National Institute for Occupational Safety and Health (NIOSH) under the provisions of 42 C.F.R. part 84 stating that the respirators have been certified for use with cadmium. Cadmium does not have a detectable odor except at levels well above the permissible exposure limits. If you can smell cadmium while wearing a respirator, proceed immediately to fresh air. If you experience difficulty breathing while wearing a respirator, tell your employer.

(ii) Protective clothing: You may be required to wear impermeable clothing, gloves, foot gear, a face shield, or other appropriate protective clothing to prevent skin contact with cadmium. Where protective clothing is required, your employer must provide clean garments to you as necessary to assure that the clothing protects you adequately. The employer must replace or repair protective clothing that has become torn or otherwise damaged.

(iii) Eye protection: You may be required to wear splash-proof or dust resistant goggles to prevent eye contact with cadmium.

(b) Employer requirements.

(i) Medical: If you are exposed to cadmium at or above the action level, your employer is required to provide a medical examination, laboratory tests and a medical history according to the medical surveillance provisions under WAC 296-62-07423. (See summary chart and tables in this section, appendix A.) These tests must be provided without cost to you. In addition, if you are accidentally exposed to cadmium under conditions known or suspected to constitute toxic exposure to cadmium, your employer is required to make special tests available to you.

(ii) Access to records: All medical records are kept strictly confidential. You or your representative are entitled to see the records of measurements of your exposure to cadmium. Your medical examination records can be furnished to your personal physician or designated representative upon request by you to your employer.

(iii) Observation of monitoring: Your employer is required to perform measurements that are representative of your exposure to cadmium and you or your designated representative are entitled to observe the monitoring procedure. You are entitled to observe the steps taken in the measurement procedure, and to record the results obtained. When the monitoring procedure is taking place in an area where respirators or personal protective clothing and equipment are required to be worn, you or your representative must also be provided with, and must wear the protective clothing and equipment.

(c) Employee requirements. You will not be able to smoke, eat, drink, chew gum or tobacco, or apply cosmetics while working with cadmium in regulated areas. You will also not be able to carry or store tobacco products, gum, food, drinks, or cosmetics in regulated areas because these products easily become contaminated with cadmium from the workplace and can therefore create another source of unnecessary

cadmium exposure. Some workers will have to change out of work clothes and shower at the end of the day, as part of their workday, in order to wash cadmium from skin and hair. Handwashing and cadmium-free eating facilities must be provided by the employer and proper hygiene should always be performed before eating. It is also recommended that you do not smoke or use tobacco products, because among other things, they naturally contain cadmium. For further information, read the labeling on such products.

(4) Physician information.

(a) Introduction. The medical surveillance provisions of WAC 296-62-07423 generally are aimed at accomplishing three main interrelated purposes: First, identifying employees at higher risk of adverse health effects from excess, chronic exposure to cadmium; second, preventing cadmium-induced disease; and third, detecting and minimizing existing cadmium-induced disease. The core of medical surveillance in this standard is the early and periodic monitoring of the employee's biological indicators of:

(i) Recent exposure to cadmium;

(ii) Cadmium body burden; and

(iii) Potential and actual kidney damage associated with exposure to cadmium. The main adverse health effects associated with cadmium overexposure are lung cancer and kidney dysfunction. It is not yet known how to adequately biologically monitor human beings to specifically prevent cadmium-induced lung cancer. By contrast, the kidney can be monitored to provide prevention and early detection of cadmium-induced kidney damage. Since, for noncarcinogenic effects, the kidney is considered the primary target organ of chronic exposure to cadmium, the medical surveillance provisions of this standard effectively focus on cadmium-induced kidney disease. Within that focus, the aim, where possible, is to prevent the onset of such disease and, where necessary, to minimize such disease as may already exist. The by-products of successful prevention of kidney disease are anticipated to be the reduction and prevention of other cadmium-induced diseases.

(b) Health effects. The major health effects associated with cadmium overexposure are described below.

(i) Kidney: The most prevalent nonmalignant disease observed among workers chronically exposed to cadmium is kidney dysfunction. Initially, such dysfunction is manifested as proteinuria. The proteinuria associated with cadmium exposure is most commonly characterized by excretion of low-molecular weight proteins (15,000 to 40,000 MW) accompanied by loss of electrolytes, uric acid, calcium, amino acids, and phosphate. The compounds commonly excreted include: Beta-2-microglobulin (β_2-M), retinol binding protein (RBP), immunoglobulin light chains, and lysozyme. Excretion of low molecular weight proteins are characteristic of damage to the proximal tubules of the kidney (Iwao et al., 1980). It has also been observed that exposure to cadmium may lead to urinary excretion of high-molecular weight proteins such as albumin, immunoglobulin G, and glycoproteins (Ex. 29). Excretion of high-molecular weight proteins is typically indicative of damage to the glomeruli of the kidney. Bernard et al., (1979) suggest that damage to the glomeruli and damage to the proximal tubules of the kidney may both be linked to cadmium exposure but they may occur independently of each other. Several studies indicate that the onset of low-molecular weight proteinuria is a sign of irreversible kidney damage (Friberg et al., 1974; Roels et al., 1982; Piscator 1984; Elinder et al., 1985; Smith et al., 1986). Above specific levels of β_2-M associ-

ated with cadmium exposure it is unlikely that β_2-M levels return to normal even when cadmium exposure is eliminated by removal of the individual from the cadmium work environment (Friberg, Ex. 29, 1990). Some studies indicate that such proteinuria may be progressive; levels of β_2-M observed in the urine increase with time even after cadmium exposure has ceased. See, for example, Elinder et al., 1985. Such observations, however, are not universal, and it has been suggested that studies in which proteinuria has not been observed to progress may not have tracked patients for a sufficiently long time interval (Jarup, Ex. 8-661). When cadmium exposure continues after the onset of proteinuria, chronic nephrotoxicity may occur (Friberg, Ex. 29). Uremia results from the inability of the glomerulus to adequately filter blood. This leads to severe disturbance of electrolyte concentrations and may lead to various clinical complications including kidney stones (L-140-50). After prolonged exposure to cadmium, glomerular proteinuria, glucosuria, aminoaciduria, phosphaturia, and hypercalciuria may develop (Exs. 8-86, 4-28, 14-18). Phosphate, calcium, glucose, and amino acids are essential to life, and under normal conditions, their excretion should be regulated by the kidney. Once low molecular weight proteinuria has developed, these elements dissipate from the human body. Loss of glomerular function may also occur, manifested by decreased glomerular filtration rate and increased serum creatinine. Severe cadmium-induced renal damage may eventually develop into chronic renal failure and uremia (Ex. 55). Studies in which animals are chronically exposed to cadmium confirm the renal effects observed in humans (Friberg et al., 1986). Animal studies also confirm problems with calcium metabolism and related skeletal effects which have been observed among humans exposed to cadmium in addition to the renal effects. Other effects commonly reported in chronic animal studies include anemia, changes in liver morphology, immunosuppression and hypertension. Some of these effects may be associated with co-factors. Hypertension, for example, appears to be associated with diet as well as cadmium exposure. Animals injected with cadmium have also shown testicular necrosis (Ex. 8-86B).

(ii) Biological markers. It is universally recognized that the best measures of cadmium exposures and its effects are measurements of cadmium in biological fluids, especially urine and blood. Of the two, CdU is conventionally used to determine body burden of cadmium in workers without kidney disease. CdB is conventionally used to monitor for recent exposure to cadmium. In addition, levels of CdU and CdB historically have been used to predict the percent of the population likely to develop kidney disease (Thun et al., Ex. L-140-50; WHO, Ex. 8-674; ACGIH, Exs. 8-667, 140-50). The third biological parameter upon which WISHA relies for medical surveillance is beta-2-microglobulin in urine (β_2-M), a low molecular weight protein. Excess β_2-M has been widely accepted by physicians and scientists as a reliable indicator of functional damage to the proximal tubule of the kidney (Exs. 8-447, 144-3-C, 4-47, L-140-45, 19-43-A). Excess β_2-M is found when the proximal tubules can no longer reabsorb this protein in a normal manner. This failure of the proximal tubules is an early stage of a kind of kidney disease that commonly occurs among workers with excessive cadmium exposure. Used in conjunction with biological test results indicating abnormal levels of CdU and CdB, the finding of excess β_2-M can establish for an examining physician that any existing kidney disease is probably cadmium-related (Trs. 6/6/90, pp. 82-86, 122, 134). The upper limits of normal levels for cadmium in urine and cadmium in

blood are 3 µg Cd/gram creatinine in urine and 5 µgCd/liter whole blood, respectively. These levels were derived from broad-based population studies. Three issues confront the physicians in the use of β_2-M as a marker of kidney dysfunction and material impairment. First, there are a few other causes of elevated levels of β_2-M not related to cadmium exposures, some of which may be rather common diseases and some of which are serious diseases (e.g., myeloma or transient flu, Exs. 29 and 8-086). These can be medically evaluated as alternative causes (Friberg, Ex. 29). Also, there are other factors that can cause β_2-M to degrade so that low levels would result in workers with tubular dysfunction. For example, regarding the degradation of β_2-M, workers with acidic urine (pH<6) might have β_2-M levels that are within the "normal" range when in fact kidney dysfunction has occurred (Ex. L-140-1) and the low molecular weight proteins are degraded in acid urine. Thus, it is very important that the pH of urine be measured, that urine samples be buffered as necessary (See WAC 296-62-07451, appendix F.), and that urine samples be handled correctly, i.e., measure the pH of freshly voided urine samples, then if necessary, buffer to Ph>6 (or above for shipping purposes), measure Ph again and then, perhaps, freeze the sample for storage and shipping. (See also WAC 296-62-07451, appendix F.) Second, there is debate over the pathological significance of proteinuria, however, most world experts believe that β_2-M levels greater than 300 µg/g Cr are abnormal (Elinder, Ex. 55, Friberg, Ex. 29). Such levels signify kidney dysfunction that constitutes material impairment of health. Finally, detection of β_2-M at low levels has often been considered difficult, however, many laboratories have the capability of detecting excess β_2-M using simple kits, such as the Phadebas Delphia test, that are accurate to levels of 100 µg β_2-M/g Cr U (Ex. L-140-1). Specific recommendations for ways to measure β_2-M and proper handling of urine samples to prevent degradation of β_2-M have been addressed by WISHA in WAC 296-62-07451, appendix F, in the section on laboratory standardization. All biological samples must be analyzed in a laboratory that is proficient in the analysis of that particular analyte, under WAC 296-62-07423 (1)(d). (See WAC 296-62-07451, appendix F). Specifically, under WAC 296-62-07423 (1)(d), the employer is to ensure that the collecting and handling of biological samples of cadmium in urine (CdU), cadmium in blood (CdB), and beta-2 microglobulin in urine (β_2-M) taken from employees is collected in a manner that ensures reliability. The employer must also ensure that analysis of biological samples of cadmium in urine (CdU), cadmium in blood (CdB), and beta-2 microglobulin in urine (β_2-M) taken from employees is performed in laboratories with demonstrated proficiency for that particular analyte. (See WAC 296-62-07451, appendix F).

(iii) Lung and prostate cancer. The primary sites for cadmium-associated cancer appear to be the lung and the prostate (L-140-50). Evidence for an association between cancer and cadmium exposure derives from both epidemiological studies and animal experiments. Mortality from prostate cancer associated with cadmium is slightly elevated in several industrial cohorts, but the number of cases is small and there is not clear dose-response relationship. More substantive evidence exists for lung cancer. The major epidemiological study of lung cancer was conducted by Thun et al., (Ex. 4-68). Adequate data on cadmium exposures were available to allow evaluation of dose-response relationships between cadmium exposure and lung cancer. A statistical-

ly significant excess of lung cancer attributed to cadmium exposure was observed in this study even when confounding variables such as co-exposure to arsenic and smoking habits were taken into consideration (Ex. L-140-50). The primary evidence for quantifying a link between lung cancer and cadmium exposure from animal studies derives from two rat bioassay studies; one by Takenaka et al., (1983), which is a study of cadmium chloride and a second study by Oldiges and Glaser (1990) of four cadmium compounds. Based on the above cited studies, the U.S. Environmental Protection Agency (EPA) classified cadmium as "B1," a probable human carcinogen, in 1985 (Ex. 4-4). The International Agency for Research on Cancer (IARC) in 1987 also recommended that cadmium be listed as "2A," a probable human carcinogen (Ex. 4-15). The American Conference of Governmental Industrial Hygienists (ACGIH) has recently recommended that cadmium be labeled as a carcinogen. Since 1984, NIOSH has concluded that cadmium is possibly a human carcinogen and has recommended that exposures be controlled to the lowest level feasible.

(iv) Noncarcinogenic effects. Acute pneumonitis occurs 10 to 24 hours after initial acute inhalation of high levels of cadmium fumes with symptoms such as fever and chest pain (Exs. 30, 8-86B). In extreme exposure cases pulmonary edema may develop and cause death several days after exposure. Little actual exposure measurement data is available on the level of airborne cadmium exposure that causes such immediate adverse lung effects, nonetheless, it is reasonable to believe a cadmium concentration of approximately 1 mg/m^3 over an eight hour period is "immediately dangerous" (55 FR 4052, ANSI; Ex. 8-86B). In addition to acute lung effects and chronic renal effects, long term exposure to cadmium may cause other severe effects on the respiratory system. Reduced pulmonary function and chronic lung disease indicative of emphysema have been observed in workers who have had prolonged exposure to cadmium dust or fumes (Exs. 4-29, 4-22, 4-42, 4-50, 4-63). In a study of workers conducted by Kazantzis et al., a statistically significant excess of worker deaths due to chronic bronchitis was found, which in his opinion was directly related to high cadmium exposures of 1 mg/m^3 or more (Tr. 6/8/90, pp. 156-157). Cadmium need not be respirable to constitute a hazard. Inspirable cadmium particles that are too large to be respirable but small enough to enter the tracheobronchial region of the lung can lead to bronchoconstriction, chronic pulmonary disease, and cancer of that portion of the lung. All of these diseases have been associated with occupational exposure to cadmium (Ex. 8-86B). Particles that are constrained by their size to the extra-thoracic regions of the respiratory system such as the nose and maxillary sinuses can be swallowed through mucocillary clearance and be absorbed into the body (ACGIH, Ex. 8-692). The impaction of these particles in the upper airways can lead to anosmia, or loss of sense of smell, which is an early indication of overexposure among workers exposed to heavy metals. This condition is commonly reported among cadmium-exposed workers (Ex. 8-86-B).

(c) Medical surveillance. In general, the main provisions of the medical surveillance section of the standard, under WAC 296-62-07423 (1) through (16), are as follows:

(i) Workers exposed above the action level are covered;

(ii) Workers with intermittent exposures are not covered;

(iii) Past workers who are covered receive biological monitoring for at least one year;

(iv) Initial examinations include a medical questionnaire and biological monitoring of cadmium in blood (CdB), cadmium in urine (CdU), and Beta-2-microglobulin in urine (β_2-M);

(v) Biological monitoring of these three analytes is performed at least annually; full medical examinations are performed biennially;

(vi) Until five years from the effective date of the standard, medical removal is required when CdU is greater than 15 µg/gram creatinine (g Cr), or CdB is greater than 15 µg/liter whole blood (lwb), or β_2-M is greater than 1500 µg/g Cr, and CdB is greater than 5 µg/lwb or CdU is greater than 3 µg/g Cr;

(vii) Beginning five years after the standard is in effect, medical removal triggers will be reduced;

(viii) Medical removal protection benefits are to be provided for up to eighteen months;

(ix) Limited initial medical examinations are required for respirator usage;

(x) Major provisions are fully described under WAC 296-62-07423; they are outlined here as follows:

(A) Eligibility.

(B) Biological monitoring.

(C) Actions triggered by levels of CdU, CdB, and β_2-M (See Summary Charts and Tables in WAC 296-62-07441(5).)

(D) Periodic medical surveillance.

(E) Actions triggered by periodic medical surveillance (See appendix A Summary Chart and Tables in WAC 296-62-07441(5).)

(F) Respirator usage.

(G) Emergency medical examinations.

(H) Termination examination.

(I) Information to physician.

(J) Physician's medical opinion.

(K) Medical removal protection.

(L) Medical removal protection benefits.

(M) Multiple physician review.

(N) Alternate physician review.

(O) Information employer gives to employee.

(P) Recordkeeping.

(Q) Reporting on OSHA form 200.

(xi) The above mentioned summary of the medical surveillance provisions, the summary chart, and tables for the actions triggered at different levels of CdU, CdB and β_2-M (in subsection (5) of this section, Attachment 1) are included only for the purpose of facilitating understanding of the provisions of WAC 296-62-07423(3) of the final cadmium standard. The summary of the provisions, the summary chart, and the tables do not add to or reduce the requirements in WAC 296-62-07423(3).

(d) Recommendations to physicians.

(i) It is strongly recommended that patients with tubular proteinuria are counseled on: The hazards of smoking; avoidance of nephrotoxins and certain prescriptions and over-the-counter medications that may exacerbate kidney symptoms; how to control diabetes and/or blood pressure; proper hydration, diet, and exercise (Ex. 19-2). A list of prominent or common nephrotoxins is attached. (See subsection (6) of this section, Attachment 2.)

(ii) DO NOT CHELATE; KNOW WHICH DRUGS ARE NEPHROTOXINS OR ARE ASSOCIATED WITH NEPHRITIS.

(iii) The gravity of cadmium-induced renal damage is compounded by the fact there is no medical treatment to prevent or reduce the ac-

cumulation of cadmium in the kidney (Ex. 8-619). Dr. Friberg, a leading world expert on cadmium toxicity, indicated in 1992, that there is no form of chelating agent that could be used without substantial risk. He stated that tubular proteinuria has to be treated in the same way as other kidney disorders (Ex. 29).

(iv) After the results of a workers' biological monitoring or medical examination are received the employer is required to provide an information sheet to the patient, briefly explaining the significance of the results. (See subsection (7) of this section.)

(v) For additional information the physician is referred to the following additional resources:

(A) The physician can always obtain a copy of the OSHA final rule preamble, with its full discussion of the health effects, from OSHA's Computerized Information System (OCIS).

(B) The OSHA Docket Officer maintains a record of the OSHA rulemaking. The Cadmium Docket (H-057A), is located at 200 Constitution Ave. NW., Room N-2625, Washington, DC 20210; telephone: (202) 219-7894.

(C) The following articles and exhibits in particular from that docket (H-057A):

Exhibit number	Author and paper title
8-447	Lauwerys et. al., Guide for physicians, "Health Maintenance of Workers Exposed to Cadmium," published by the Cadmium Council.
4-67	Takenaka, S., H. Oldiges, H. Konig, D. Hochrainer, G. Oberdorster. "Carcinogenicity of Cadmium Chloride Aerosols in Wistar Rats." JNCI 70:367-373, 1983. (32)
4-68	Thun, M.J., T.M. Schnoor, A.B. Smith, W.E. Halperin, R.A. Lemen. "Mortality Among a Cohort of U.S. Cadmium Production Workers—An Update." JNCI 74(2):325-33, 1985. (8)
4-25	Elinder, C.G., Kjellstrom, T., Hogstedt, C., et al., "Cancer Mortality of Cadmium Workers." Brit. J. Ind. Med. 42:651-655, 1985. (14)
4-26	Ellis, K.J. et al., "Critical Concentrations of Cadmium in Human Renal Cortex: Dose Effect Studies to Cadmium Smelter Workers." J. Toxicol. Environ. Health 7:691-703, 1981. (76)
4-27	Ellis, K.J., S.H. Cohn and T.J. Smith. "Cadmium Inhalation Exposure Estimates: Their Significance with Respect to Kidney and Liver Cadmium Burden." J. Toxicol. Environ. Health 15:173-187, 1985.
4-28	Falck, F.Y., Jr., Fine, L.J., Smith, R.G., McClatchey, K.D., Annesley, T., England, B., and Schork, A.M. "Occupational Cadmium Exposure and Renal Status." Am. J. Ind. Med. 4:541, 1983. (64)

Exhibit number	Author and paper title
8-86A	Friberg, L., C.G. Elinder, et al., "Cadmium and Health a Toxicological and Epidemiological Appraisal, Volume I, Exposure, Dose, and Metabolism." CRC Press, Inc., Boca Raton, FL, 1986. (Available from the OSHA Technical Data Center)
8-86B	Friberg, L., C.G. Elinder, et al., "Cadmium and Health: A Toxicological and Epidemiological Appraisal, Volume II, Effects and Response." CRC Press, Inc., Boca Raton, FL, 1986. (Available from the OSHA Technical Data Center)
L-140-45	Elinder, C.G., "Cancer Mortality of Cadmium Workers," Brit. J. Ind. Med., 42, 651-655, 1985.
L-140-50	Thun, M., Elinder, C.G., Friberg, L, "Scientific Basis for an Occupational Standard for Cadmium, Am. J. Ind. Med., 20; 629-642, 1991.

(5) Information sheet. The information sheet (subsection (8) of this section, Attachment 3) or an equally explanatory one should be provided to you after any biological monitoring results are reviewed by the physician, or where applicable, after any medical examination.

(6) Attachment 1—Appendix A, summary chart and Tables A and B of actions triggered by biological monitoring.

(a) Summary chart: WAC 296-62-07423(3) Medical surveillance—Categorizing biological monitoring results.

(i) Biological monitoring results categories are set forth in Table A for the periods ending December 31, 1998, and for the period beginning January 1, 1999.

(ii) The results of the biological monitoring for the initial medical exam and the subsequent exams must determine an employee's biological monitoring result category.

(b) Actions triggered by biological monitoring.

(i) The actions triggered by biological monitoring for an employee are set forth in Table B.

(ii) The biological monitoring results for each employee under WAC 296-62-07423(3) must determine the actions required for that employee. That is, for any employee in biological monitoring category C, the employer will perform all of the actions for which there is an X in column C of Table B.

(iii) An employee is assigned the alphabetical category ("A" being the lowest) depending upon the test results of the three biological markers.

(iv) An employee is assigned category A if monitoring results for all three biological markers fall at or below the levels indicated in the table listed for category A.

(v) An employee is assigned category B if any monitoring result for any of the three biological markers fall within the range of levels indicated in the table listed for category B, providing no result exceeds the levels listed for category B.

(vi) An employee is assigned category C if any monitoring result for any of the three biological markers are above the levels listed for category C.

(c) The user of Tables A and B should know that these tables are provided only to facilitate understanding of the relevant provisions of WAC 296-62-07423. Tables A and B are not meant to add to or subtract from the requirements of those provisions.

Table A
Categorization of Biological Monitoring Results
Applicable Through 1998 Only

	Monitoring result categories		
Biological marker	A	B	C
Cadmium in urine (CdU) (μg/g creatinine)	≤=3	>3 and ≤=15	>15
β_2-microglobulin (β_2-M) (μg/g creatinine)	≤=300	>300 and ≤=1500	>1500*
Cadmium in blood (CdB) (μg/liter whole blood)	≤=5	>5 and ≤=15	>15

* If an employee's β_2-M levels are above 1,500 μg/g creatinine, in order for mandatory medical removal to be required (See WAC 296-62-07441, Appendix A Table B.), either the employee's CdU level must also be >3 μg/g creatinine or CdB level must also be >5 μg/liter whole blood.

Applicable Beginning January 1, 1999

	Monitoring result categories		
Biological marker	A	B	C
Cadmium in urine (CdU) (μg/g creatinine)	≤=3	>3 and ≤=7	>7
β_2-microglobulin (β_2-M) (μg/g creatinine)	≤=300	>300 and ≤=750	>750*
Cadmium in blood (CdB) (μg/liter whole blood)	≤=5	>5 and ≤=10	>10

* If an employee's β_2-M levels are above 750 μg/g creatinine, in order for mandatory medical removal to be required (See WAC 296-62-07441, Appendix A Table B.), either the employee's CdU level must also be >3 μg/g creatinine or CdB level must also be >5 μg/liter whole blood.

Table B—Actions determined by biological monitoring.

This table presents the actions required based on the monitoring result in Table A. Each item is a separate requirement in citing noncompliance. For example, a medical examination within ninety days for an employee in category B is separate from the requirement to administer a periodic medical examination for category B employees on an annual basis.

Table B
Monitoring result category

	A[1]	B[1]	C[1]
Required actions			
(1) Biological monitoring:			
(a) Annual.	X		
(b) Semiannual		X	
(c) Quarterly			X
(2) Medical examination:			
(a) Biennial	X		
(b) Annual.		X	
(c) Semiannual.			X
(d) Within 90 days		X	X

	A[1]	B[1]	C[1]
(3) Assess within two weeks:			
(a) Excess cadmium exposure		X	X
(b) Work practices		X	X
(c) Personal hygiene		X	X
(d) Respirator usage		X	X
(e) Smoking history		X	X
(f) Hygiene facilities		X	X
(g) Engineering controls		X	X
(h) Correct within 30 days		X	X
(i) Periodically assess exposures			X
(4) Discretionary medical removal		X	X
(5) Mandatory medical removal			X[2]

[1] For all employees covered by medical surveillance exclusively because of exposures prior to the effective date of this standard, if they are in Category A, the employer shall follow the requirements of WAC 296-62-07423 (3)(a)(ii) and (4)(e)(i). If they are in Category B or C, the employer shall follow the requirements of WAC 296-62-07423 (4)(e)(ii) and (iii).

[2] See footnote in Table A.

(7) Attachment 2, list of medications.

(a) A list of the more common medications that a physician, and the employee, may wish to review is likely to include some of the following:

(i) Anticonvulsants: Paramethadione, phenytoin, trimethadone;

(ii) Antihypertensive drugs: Captopril, methyldopa;

(iii) Antimicrobials: Aminoglycosides, amphotericin B, cephalosporins, ethambutol;

(iv) Antineoplastic agents: Cisplatin, methotrexate, mitomycin-C, nitrosoureas, radiation;

(v) Sulfonamide diuretics: Acetazolamide, chlorthalidone, furosemide, thiazides;

(vi) Halogenated alkanes, hydrocarbons, and solvents that may occur in some settings: Carbon tetrachloride, ethylene glycol, toluene; iodinated radiographic contrast media; nonsteroidal anti-inflammatory drugs; and

(vii) Other miscellaneous compounds: Acetaminophen, allopurinol, amphetamines, azathioprine, cimetidine, cyclosporine, lithium, methoxyflurane, methysergide, D-penicillamine, phenacetin, phenendione.

(b) A list of drugs associated with acute interstitial nephritis includes:

(i) Antimicrobial drugs: Cephalosporins, chloramphenicol, colistin, erythromycin, ethambutol, isoniazid, para-amin-osalicylic acid, penicillins, polymyxin B, rifampin, sulfonamides, tetracyclines, and vancomycin;

(ii) Other miscellaneous drugs: Allopurinol, antipyrine, azathioprine, captopril, cimetidine, clofibrate, methyldopa, phenindione, phenylpropanolamine, phenytoin, probenecid, sulfinpyrazone, sulfonamide diuretics, triamterene; and

(iii) Metals: Bismuth, gold. This list has been derived from commonly available medical textbooks (e.g., Ex. 14-18). The list has been included merely to facilitate the physician's, employer's, and employee's understanding. The list does not represent an official OSHA opinion or policy regarding the use of these medications for particular

employees. The use of such medications should be under physician discretion.

(8) Attachment 3—Biological monitoring and medical examination results.

Employee _____
Testing
Date _____

Cadmium in Urine ___ µg/g Cr—Normal Levels: ≤=3 µg/g Cr.
Cadmium in Blood ___ µg/lwb—Normal Levels: ≤=5 µg/lwb.
Beta-2-microglobulin in Urine ___ µg/g Cr-Normal Levels: ≤=300 µg/g Cr.
Physical Examination Results:
N/A ___ Satisfactory ___
Unsatisfactory ___ (see physician again).
Physician's Review of Pulmonary Function Test:
N/A ___ Normal ___ Abnormal ___.
Next biological monitoring or medical examination scheduled for _____

(a) The biological monitoring program has been designed for three main purposes:

(i) To identify employees at risk of adverse health effects from excess, chronic exposure to cadmium;

(ii) To prevent cadmium-induced disease(s); and

(iii) To detect and minimize existing cadmium-induced disease(s).

(b) The levels of cadmium in the urine and blood provide an estimate of the total amount of cadmium in the body. The amount of a specific protein in the urine (beta-2-microglobulin) indicates changes in kidney function. All three tests must be evaluated together. A single mildly elevated result may not be important if testing at a later time indicates that the results are normal and the workplace has been evaluated to decrease possible sources of cadmium exposure. The levels of cadmium or beta-2-microglobulin may change over a period of days to months and the time needed for those changes to occur is different for each worker.

(c) If the results for biological monitoring are above specific "high levels" (cadmium urine greater than 10 micrograms per gram of creatinine µg/g Cr), cadmium blood greater than 10 micrograms per liter of whole blood (µg/lwb), or beta-2-microglobulin greater than 1000 micrograms per gram of creatinine (µg/g Cr)), the worker has a much greater chance of developing other kidney diseases.

(d) One way to measure for kidney function is by measuring beta-2-microglobulin in the urine. Beta-2-microglobulin is a protein which is normally found in the blood as it is being filtered in the kidney, and the kidney reabsorbs or returns almost all of the beta-2-microglobulin to the blood. A very small amount (less than 300 µg/g Cr in the urine) of beta-2-microglobulin is not reabsorbed into the blood, but is released in the urine. If cadmium damages the kidney, the amount of beta-2-microglobulin in the urine increases because the kidney cells are unable to reabsorb the beta-2-microglobulin normally.

An increase in the amount of beta-2-microglobulin in the urine is a very early sign of kidney dysfunction. A small increase in beta-2-microglobulin in the urine will serve as an early warning sign that the worker may be absorbing cadmium from the air, cigarettes contaminated in the workplace, or eating in areas that are cadmium contaminated.

(e) Even if cadmium causes permanent changes in the kidney's ability to reabsorb beta-2-microglobulin, and the beta-2-microglobulin is above the "high levels," the loss of kidney function may not lead to any serious health problems. Also, renal function naturally declines as people age. The risk for changes in kidney function for workers who have biological monitoring results between the "normal values" and the "high levels" is not well known. Some people are more cadmium-tolerant, while others are more cadmium-susceptible.

(f) For anyone with even a slight increase of beta-2-microglobulin, cadmium in the urine, or cadmium in the blood, it is very important to protect the kidney from further damage. Kidney damage can come from other sources than excess cadmium-exposure so it is also recommended that if a worker's levels are "high" they should receive counseling about drinking more water; avoiding cadmium-tainted tobacco and certain medications (nephrotoxins, acetaminophen); controlling diet, vitamin intake, blood pressure and diabetes; etc.

[Statutory Authority: RCW 49.17.010, 49.17.040, 49.17.050, and 49.17.060. WSR 19-01-094, § 296-62-07441, filed 12/18/18, effective 1/18/19. Statutory Authority: RCW 49.17.010, [49.17].040 and [49.17].050. WSR 99-10-071, § 296-62-07441, filed 5/4/99, effective 9/1/99. Statutory Authority: Chapter 49.17 RCW. WSR 94-15-096 (Order 94-07), § 296-62-07441, filed 7/20/94, effective 9/20/94; WSR 93-21-075 (Order 93-06), § 296-62-07441, filed 10/20/93, effective 12/1/93; WSR 93-07-044 (Order 93-01), § 296-62-07441, filed 3/13/93, effective 4/27/93.]