6C-1

. 1 - HSDB

HAZARDOUS SUBSTANCES DATABANK NUMBER	1650
LAST REVISION DATE	931105
REVIEW DATE	Reviewed by SRP on 8/26/88
UPDATE HISTORY	11/05/93, 1 field
UPDATE HISTORY	10/28/93, 1 field
UPDATE HISTORY	08/07/93, 1 field
UPDATE HISTORY	05/25/93, 1 field
UPDATE HISTORY	01/20/93, 1 field
UPDATE HISTORY	12/21/92, 1 field
UPDATE HISTORY	11/23/92, 1 field
UPDATE HISTORY	08/18/92, 1 field
UPDATE HISTORY	05/15/92, 1 field
UPDATE HISTORY	04/01/92, 1 field
UPDATE HISTORY	01/23/92, 1 field
UPDATE HISTORY	07/11/91, 1 field
UPDATE HISTORY	12/19/90, 1 field
UPDATE HISTORY	11/09/90, 1 field
UPDATE HISTORY	10/23/90, 3 fields
UPDATE HISTORY	08/23/90, 1 field
UPDATE HISTORY	06/21/89, 77 fields
UPDATE HISTORY	03/08/88, 3 fields
UPDATE HISTORY	04/28/86
RECORD LENGTH	225252
NAME OF SUBSTANCE	LEAD CHROMATE
CAS REGISTRY NUMBER	7758-97-6
RELATED HSDB RECORDS	231 [LEAD]
RELATED HSDB RECORDS	5800 [TUNGSTEN TRIOXIDE1
SYNONYMS	CANARY CHROME YELLOW 40-2250 **PEER REVIEWED**
SYNONYMS	CHROMATE DE PLOME (FRENCH) **PEER REVIEWED**
SYNONYMS	CHROME LEMON **PEER REVIEWED**
SYNONYMS	CHROME YELLOW **PEER REVIEWED**
SYNONYMS	CHROME YELLOW 5G **PEER REVIEWED**
SYNONYMS	CHROME YELLOW G **PEER REVIEWED**
SYNONYMS	CHROME YELLOW GF **PEER REVIEWED**
SYNONYMS	CHROME YELLOW LF **PEER REVIEWED**
SYNONYMS	CHROME YELLOW LIGHT 1066 **PEER REVIEWED**
SYNONYMS	CHROME YELLOW LIGHT 1075 **PEER REVIEWED**
SYNONYMS	CHROME YELLOW MEDIUM 1074 **PEER REVIEWED**
SYNONYMS	CHROME YELLOW MEDIUM 1085 **PEER REVIEWED**
SYNONYMS	CHROME YELLOW MEDIUM 1298 **PEER REVIEWED**
SYNONYMS	CHROME YELLOW PRIMROSE 1010 **PEER REVIEWED**
SYNONYMS	CHROME YELLOW PRIMROSE 1015 **PEER REVIEWED**
SYNONYMS	CHROMIC ACID (H2CRO4), LEAD(2+) SALT (1:1) **PEER REVIEWED**
SYNONYMS	CHROMIUM YELLOW **PEER REVIEWED**
SYNONYMS	CI 77600 **PEER REVIEWED**
SYNONYMS	CI PIGMENT YELLOW 34 **PEER REVIEWED**
SYNONYMS	COLOGNE YELLOW **PEER REVIEWED**
SYNONYMS	CP CHROME YELLOW LIGHT **PEER REVIEWED**
SYNONYMS	CP CHROME YELLOW MEDIUM **PEER REVIEWED**
SYNONYMS	CP CHROME YELLOU PRIMOSE **PEER REVIEWED**
SYNONYMS	DAINICHI CHROME YELLOW G **PEER REVIEWED**
SYNONYMS	KING'S YELLOW **PEER REVIEWED**
SYNONYMS	LEAD CHROMATE (PBCRO4) **PEER REVIEWED**
SYNONYMS	LEAD CHROMATE(VI) **PEER REVIEWED**
SYNONYMS	LEIPZIG YELLOW **PEER REVIEWED**
SYNONYMS	LEMON YELLOU **PEER REVIEWED**
SYNONYMS	PARIS YELLOW **PEER REVIEWED**
SYNONYMS	PLUMBWS CHROMATE **PEER REVIEWED**
SYNONYMS	PURE LEMON CHROME L3GS **PEER REVIEWED**
SYNONYMS	Phoenicochroite **PEER REVIEWED**
MOLECULAR FORMULA	Cr-04-Pb **QC REVIEWED**
WISWESSER LINE NOTATION	PB CR-02-02 **PEER REVIEWED**

RTECS NUMBER NIOSH/GB2975000

EPA HAZARDOUS WASTE NUMBER D007; A waste containing lead chromate (chromium ion) may (or may not) be characterized a hazardous waste following testing by the Toxicant Extraction Procedure as prescribed by the Resource Conservation and Recovery Act (RCRA) regualtions.

EPA HAZARDOUS UASTE NUMBER D008; A waste containing lead chromate (lead ion) may (or may not) be characterized a hazardous waste following testing by the Toxicant Extraction Procedure as prescribed by the Resource Conservation and Recovery Act (RCRA) regualtions.

METHODS OF MANUFACTURING

LEAD CHROMATE CAN BE PRODUCED BY REACTING SODIUM CHROMATE UITH LEAD NITRATE, OR BY REACTING LEAD MONOXIDE UITH CHROMIC ACID SOLN. DETAILS OF VARIWS COMMERCIAL PROCEDURES FOR MFR OF LEAD CHROMATES ARE NOT GENERALLY REVEALED BY PRODUCERS. [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: Uorld Health Organization, International Agency for Research on Cancer, 1972-PRESENT. (Multivolume work). V2 105 (1973)] **PEER REVIEWED**

FORMULATIONS/PREPARATIONS

Chrome yellows /contain/ ... 52-98% lead chromate ... [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-PRESENT. (Multivolume work). V23 233 (1980)] **PEER REVIEWED**

MANUFACTURERS

Heubach Inc, Hq, Heubach Ave, Newark, NJ 07114, (201) 242-1800; Production sites: 105 Bedford Ave, Brooklyn, NY 11211; Newark, NJ 07114 [SRI. 1989 Directory of Chemical Producers - United States of America. Menlo Park, CA: SRI International, 1989. , p. 8621 **UNREVIEWED**

MANUFACTURERS

Kikuchi Color & Chemicals Corp, USA, Hq, 19 E Fifth St, Paterson, NJ 07524, (201) 278-0206 [SRI. 1989 Directory of Chemical Producers - United States of America. Menlo Park, CA: SRI International, 1989. , p. 8621 **UNREVIEWED**

MANUFACTURERS

Maxxam Group Inc, Hq, 1088 O Uilshire Blvd, Los Angeles, CA 90024, (213) 474-6264; Kaiser Tech Limited, Harshaw/Filtrol Partnership, 30100 Chagrin Boulevard, Cleveland, OH 44124; Production site: 3400 Bank St, Louisville, KY 40212 [SRI. 1989 Directory of Chemical Producers - United States of America. Menlo Park, CA: SRI International, 1989. , p. 8621 **UNREVIEWED**

MANUFACTURERS

National Industries Chemical Co, Hq, 600 West 52nd St, Chicago, IL 60609, (312) 924-3700 [SRI. 1989 Directory of Chemical Producers - United States of America. Menlo Park, CA: SRI International, 1989. , p. 8621 **UNREVIEWED**

MANUFACTURERS

NL Chemicals, Inc, Hq, Uyckoffs Mill Rd, Hightstown, NJ 08520, (609) 443-2000; 5548 Manchester Ave, St Louis, MO 63110 CSRI. 1989 Directory of Chemical Producers - United States of America. Menlo Park, CA: SRI International, 1989. , p. 8621 **UNREVIEWED**

MANUFACTURERS

Rockwood Industries Inc, Hq, 12116 Conway Rd, Beltsville, MD 20705, (301) 470-3366; Subsidiaries: Frank D Davis Co, 3700 E Olympic Blvd, Los Angeles, CA 90023, (213) 269-7311; Production site: Los Angeles, CA 90023; Mineral Pigments Corp, 7011 Muirkirk Rd, Beltsville, MD 20705, (301) 776-2400; Chemical Color Division; Production site: Beltsville, MD 20705 [SRI. 1989 Directory of Chemical Producers - United States of America. Menlo Park, CA: SRI International, 1989. , p. 8621 **UNREVIEWED**

MANUFACTURERS

Wayne Pigment Corp, Hq, 546 South Uater St, Milwaukee, UI 53204, (414) 278-8844 CSRI. 1989 Directory of Chemical

Producers - United States of America. Menlo Park, CA: SRI International, 1989. , p. 8621 **UNREVIEWED**

OTHER MANUFACTURING INFORMATION
LEAD CHROMATE-BASED INKS ARE USED FOR PRINTING COLORED PAGES OF SOME CHILDREN'S MAGAZINES (COMICS). THIS PAPER REPORTS INVESTIGATIONS OF 48 UNITED KINGDOM, 9 SPANISH, & 5 AUSTRIAN COMICS. [EATON DF ET AL; ENVIRON SCI TECHNOL 9 (8): 768-70 (1975)] **PEER REVIEWED**

OTHER MANUFACTURING INFORMATION
... USA EPA HAS ORDERED BAN ON INTERSTATE SHIPMENTS OF PAINTS FOR DOMESTIC USE CONTAINING MORE THAN 0.06% LEAD, EFFECTIVE FROM 31 DECEMBER 1973. THIS BAN WILL ... REDUCE US CONSUMPTION OF LEAD CHROMATE PIGMENTS & ... REDUCE WHATEVER CONTRIBUTION LEAD CHROMATE ... MAKES TO LEAD & CHROMIUM POLLUTION PROBLEMS. [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-PRESENT. (Multivolume work). V2 106 (1973)] **PEER REVIEWED**

OTHER MANUFACTURING INFORMATION
Lead based pigment colors include green ... . /Lead chromate based pigments/ [USEPA; The Health and Enviromental Impacts of Lead: p.87 (1979) EPA 560/2-79-001] **PEER REVIEWED**

OTHER MANUFACTURING INFORMATION
Considerable energy is released by the mixture /of lead chromate and aluminum dinitronaphthalene/ derived from chromate-catalyzed exothermic decomposition of the nitro compound, coupled with a thermite-type reaction of the aluminum and chromate. It is useful for cracking concrete. [Bretherick, L. Handbook of Reactive Chemical Hazards. 3rd ed. Boston, MA: Butterworths, 1985. , p. 10271 **PEER REVIEWED**

MAJOR USES
DECORATING CHINA; IN CHEMICAL ANALYSIS OF ORG SUBSTANCES [The Merck index. 10th ed. Rahway, New Jersey: Merck Co., Inc., 1983. , p. 7771 **PEER REVIEWED**

MAJOR USES
PIGMENT IN INDUSTRIAL PAINTS, RUBBER, PLASTICS [Sax, N.I. and R.J. Lewis, Sr. (eds.). Hawley's Condensed Chemical Dictionary. 11th ed. New York: Van Nostrand Reinhold Co., 1987. , p. 6891 **PEER REVIEWED**

MAJOR USES
PIGMENT IN: OIL PAINTS & WATER COLORS, PRINTING FABRICS, DECORATING PORCELAIN [SRI 1 **PEER REVIEWED**

U.S. PRODUCTION
(1972) 3.06X10+10 G [SRI 1 **PEER REVIEWED**

U.S. PRODUCTION
(1975) 1.18X10+10 G (CHROME YELLOW & ORANGE) [SRI 1 **PEER REVIEWED**

COLOR/FORM	YELLOW OR ORANGE-YELLOW POWDER [The Merck Index. 10th ed. Rahway, New Jersey: Merck Co., Inc., 1983. , p. 7771 **PEER REVIEWED**
COLOR/FORM	MONOCLINIC CRYSTALS [Weast, R.C. (ed.) Handbook of Chemistry and Physics, 68th ed. Boca Raton, Florida: CRC Press Inc., 1987-1988. B-991 **PEER REVIEWED**
MELTING POINT	844 DEG C [The Merck Index. 10th ed. Rahway, New Jersey: Merck Co., Inc., 1983. , p. 777] **PEER REVIEWED**
MOLECULAR UEIGHT	323.22 [The Merck Index. 10th ed. Rahway, New Jersey: Merck Co., Inc., 1983. , p. 777] **PEER REVIEWED**
DENSITY/SPECIFIC GRAVITY	6.12 @ 15 deg C [Weast, R.C. (ed.) Handbook of Chemistry and Physics, 68th ed. Boca Raton, Florida: CRC Press Inc., 1987-1988. B-99] **PEER REVIEWED**
SOLUBILITIES	0.2 MG/L WATER [The Merck Index. 10th ed. Rahway, New Jersey: Merck Co., Inc., 1983. , p. 7771 **PEER REVIEWED**
SOLUBILITIES	INSOL IN ACETIC ACID; SOL IN DIL NITRIC ACID & IN SOLN OF FIXED ALKALI HYDROXIDES [The Merck Index. 10th ed. Rahway, New Jersey: Merck Co., Inc., 1983. , p. 7771 **PEER REVIEWED**

SOLUBILITIES
SOL IN ACID; INSOL IN AMMONIA [Weast, R.C. (ed.) Handbook of Chemistry and Physics, 68th ed. Boca Raton, Florida: CRC Press Inc., 1987-1988. B-99] **PEER REVIEWED**

SPECTRAL PROPERTIES
INDEX OF REFRACTION: 2.31, 2.37 (LI), 2.66 [Weast, R.C. (ed.) Handbook of Chemistry and Physics, 68th ed. Boca Raton, Florida: CRC Press Inc., 1987-1988. B-991 **PEER REVIEWED**

OTHER CHEMICAL/PHYSICAL PROPERTIES
Hexavalent chromiun is acidic. /Hexavalent chromium/ [USEPA; Health Assessment Document: Chromiun p.2-2 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

OTHER CHEMICAL/PHYSICAL PROPERTIES
Divalent lead has a strong affinity for inorganic ions containing oxygen (eg, carbonate) or sulfur (sulfide). Lead can also complex uith electron rich ligands in many organic cmpd such as amino acids, proteins, and humic acid. /Inorganic lead/ [Kayser, R., D. Sterling, D. Viviani (eds.). Intermedia Priority 'Pollutant Guidance Documents. Washington, DC: U.S.Environmental Protection Agency, July 1982. 1-11 **PEER REVIEWED**

FIRE FIGHTING PROCEDURES
Self-contained breathing apparatus uith a full facepiece, operated in pressure-demand or other positive pressure mode. /Inorganic lead/ [NIOSH; Criteria Docwnt: Inorganic Lead p.I-10 (1978) DHEU Pub. NIOSH 78-1581 **PEER REVIEWED**

TOXIC COMBUSTION PRODUCTS
DANGEROUS; WHEN HEATED IT EMITS HIGHLY TOXIC FUMES. /INORGANIC LEAD/ [Sax, N.I. Dangerous Properties of Industrial Materials. 4th ed. Neu York: Van Nostrand Reinhold, 1975. , p. 8661 **PEER REVIEWED**

REACTIVITIES & INCOMPATIBILITIES
Hydrazine is decomposed explosively by chromates. /Chromates/ [National Fire Protection Association. Fire Protection Guide on Hazardous Materials. 9th ed. Boston, MA: National Fire Protection Association, 1986. 491M-65] **PEER REVIEWED**

REACTIVITIES & INCOMPATIBILITIES
Incompatibilities: combustible, organic, or other readily oxidizable materials .... /Chromic acid and chromates/ [NIOSH. Pocket Guide to Chemical Hazards. 5th Printing/Revision. DHHS (NIOSH) Publ. No. 85-114. Washington, D.C.: U.S. Dept. of Health and Human Services, NIOSH/Supt. of Docunents, GPO, Sept. 1985. , p. 821 **PEER REVIEWED**

REACTIVITIES & INCOMPATIBILITIES
During grinding operations, the intimate mixture /of lead chromate and iron(3+) hexacyanoferrate(4-)/ was ignited by a spark and burned fiercely. Spontaneous ignition of Brunswick Green pigment (which also contains lead sulfate) soon after grinding was not uncommon, and similar incidents had led to the loss of ships uith cargoes of Prussian Blue or Brunswick Green in wooden casks. The mixture /of lead chromate and sulfur/ is pyrophoric. The mixture /of lead chromate and tantalum/ is a pyrotechnic composition. [Bretherick, L. Handbook of Reactive Chemical Hazards. 3rd ed. Boston, MA: Butterworths;, 1985. , p. 10281 **PEER REVIEWED**

REACTIVITIES & INCOMPATIBILITIES
Considerable energy is released by the mixture /of lead chromate and aluminum dinitronaphthalene/ derived from chromate-catalyzed exothermic decomposition of the nitro compound, coupled with a thermite-type reaction of the aluminum and chromate. [Bretherick, L. Handbook of Reactive Chemical Hazards. 3rd ed. Boston, MA: Butterworths, 1985. , p. 10271 **PEER REVIEWED**

REACTIVITIES & INCOMPATIBILITIES
Under certain conditions, dry mixtures of lead chromate pigments with the G & O azo-dyes ... dinitroaniline orange or ... chlorinated para-red may lead to violent explosions during mixing or blending operations. [Bretherick, L. Handbook of Reactive Chemical Hazards. 3rd ed. Boston, MA: Butterworths, 1985. , p. 10271 **PEER REVIEWED**

REACTIVITIES & INCOMPATIBILITIES

Incompatibilities: Strong oxidizers, hydrogen peroxide, and the active metals, sodium and potassium. /Inorganic lead/ [NIOSH. Pocket Guide to Chemical Hazards. 5th Printing/Revision. DHHS (NIOSH) Publ. No. 85-114. Washington, D.C.: U.S. Dept. of Health and Human Services, NIOSH/Supt. of Documents, GPO, Sept. 1985. , p. 1461 **PEER REVIEWED**

SKIN, EYE AND RESPIRATORY IRRITATIONS

... Chromate dusts may cause severe irritation of the nose, throat, bronchial tubes, and lung. /Chromate dusts/ [Mackison, F. W., R. S. Stricoff, and L. J. Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) Publication No. 81-123 (3 VOLS). Washington, DC: U.S. Government Printing Office, Jan. 1981. , p. 1] **PEER REVIEWED**

PROTECTIVE EQUIPMENT & CLOTHING

Impervious gloves, aprons, and footwear shall be worn at operations where solutions of chromium(VI) may contact the skin. Protective gloves shall be worn at operations where dry compounds of chromium(VI) are handled and may contact the skin. /Hexavalent chromium/ [NIOSH; Criteria Document: Chromium (VI) p.8 (1976) DHEW Pub. NIOSH 76-129] **PEER REVIEWED**

PROTECTIVE EQUIPMENT & CLOTHING

Upper limit respirator devices permitted: at 5 mg/cu m: 1) High-efficiency particulate respirator with a full facepiece; or 2) Supplied-air respirator with a full facepiece, helmet, or hood; or 3) Self-contained breathing apparatus with a full facepiece; at 30 mg/cu m: 1) Powered air-purifying respirator with an organic vapor cartridge and a high-efficiency particulate filter with a full facepiece; or 2) Type C supplied-air respirator with a full facepiece, operated in pressure-demand or other positive pressure mode, or with a full facepiece, helmet, or hood, operated in continuous-flow mode; escape: 1) High-efficiency particulate respirator; or 2) Self-contained breathing apparatus. /Chromic acid and chromates/ [NIOSH. Pocket Guide to Chemical Hazards. 5th Printing/Revision. DHHS (NIOSH) Publ. No. 85-114. Washington, D.C.: U.S. Dept. of Health and Human Services, NIOSH/Supt. of Documents, GPO, Sept. 1985. , p. 821 **PEER REVIEWED**

PROTECTIVE EQUIPMENT & CLOTHING

Not in excess of 0.5 mg/cu m: Dust mask, except single use. Not in excess of 1 mg/cu m: Dust and mist respirator, except single use and quarter mask respirators. Any fume respirator or high efficiency particulate filter respirator. Any supplied air respirator or any self contained breathing apparatus. Not in excess of 5 mg/cu m: A high efficiency particulate filter respirator with a full facepiece. Any supplied air respirator with a full facepiece. Any self contained breathing apparatus with a full facepiece. Not in excess of 100 mg/cu m: Type C supplied-air respirator operated in pressure-demand or other positive pressure or continuous flow mode. A powered air-purifying respirator with high efficiency particulate filter. Not in excess of 200 mg/cu m: Type C supplied air respirator with a full facepiece operated in pressure demand or other positive pressure mode, or with a full facepiece, helmet, or hood, operated in continuous-flow mode. Greater than 200 mg/cu m, or entry and escape from unknown concn: Self-contained breathing apparatus with a full facepiece operated in pressure demand or other positive pressure mode. A combination respirator, which includes a type C supplied air respirator with a full facepiece, operated in pressure demand or other positive pressure or continuous flow mode and an auxiliary, self contained, breathing apparatus operated in pressure demand or other positive pressure mode. /Inorganic lead/ [NIOSH; Criteria Document: Inorganic Lead p.I-7 (1978) DHEW Pub. NIOSH 78-1581 **PEER REVIEWED**

PROTECTIVE EQUIPMENT & CLOTHING

Overalls or other full body protective clothing shall be worn in areas where there is occupational exposure to inorganic lead. /Inorganic lead/ [NIOSH; Criteria Document: Inorganic Lead p.I-9 (1978) DHEW Pub. NIOSH 78-1581 **PEER REVIEWED**

PROTECTIVE EQUIPMENT & CLOTHING

Workers who are exposed to lead in any of its forms should wear

personal protective equipnent, which should be washed or renewed at least once a week. Protective clothing made of certain man made fibers retains much less dust than cotton overalls and should be used where the conditions of work render it possible; turn ups, pleats and pockets in which lead dust may collect should be avoided. /Inorganic lead/ [International Labour Office. Encyclopedia of Occupational Health and Safety. Vols. I&II. Geneva, Switzerland: International Labour Office, 1983. , p. 12041 **PEER REVIEWED**

PROTECTIVE EQUIPMENT & CLOTHING

Full facepiece is required if the lead aerosols cause eye or skin irritation at the use concn. Full facepiece, self-contained breathing apparatus operated in positive-pressure mode should be used when lead is at a concn greater than of 100 mg/cu m, at an unknown concn, or when fire fighting. /Inorganic lead cmpd/ [29 CFR 1910.1025 07/01/87] **PEER REVIEWED**

PROTECTIVE EQUIPMENT & CLOTHING

PRECAUTIONS FOR "CARCINOGENS": ... Dispensers of liq detergent /should be available./ ... Safety pipettes should be used for all pipetting. ... In animal laboratory, personnel should ... wear protective suits (preferably disposable, one-piece & close-fitting at ankles & wrists), gloves, hair covering & overshoes. ... In chemical laboratory, gloves & gowns should always be worn ... however, gloves should not be assumed to provide full protech[{s. Carefully fitted masks or respirators may be necessary when working with particulates or gases, & disposable plastic aprons might provide addnl protection. ... Gowns ... /should be/ of distinctive color, this is a reminder that they are not to be worn outside the laboratory. /Chemical Carcinogens/ [Montesano, R., H. Bartsch, E.Boyland, G. Della Porta, L. Fishbein, R. A. Griesemer, A.B. Swan, L. Tomatis, and W. Davis (eds.). Handling Chemical Carcinogens in the Laboratory: Problems of Safety. IARC Scientific Publications No. 33. Lyon, France: International Agency for Research on Cancer, 1979. , p. 81 **PEER REVIEWED**

OTHER PREVENTIVE MEASURES

Showers and wash basins should be located in the locker area to encourage good personal hygiene. /Hexavalent chromium/ [NIOSH; Criteria Document: Chromium (VI) p.16 (1976) DHEW Pub. NIOSH 76-1291 **PEER REVIEWED**

OTHER PREVENTIVE MEASURES

Covered containers should be provided for work clothing discarded at the end of the shift or after a contamination incident. The clothing will be held in these containers until removed for decontamination or disposal. /Hexavalent chromium/ [NIOSH; Criteria Document: Chromium (VI) p.16 (1976) DHEW Pub. NIOSH 76-1291 **PEER REVIEWED**

OTHER PREVENTIVE MEASURES

Good industrial hygiene practices recommend that engineering controls be used'to reduce environmental concentrations to the permissible exposure level. However, there are some exceptions where respirators may be used to control exposure. Respirators may be used when engineering and work practice controls are not technically feasible, when such controls are in the process of being installed, or when they fail and need to be supplemented. Respirators may also be used for operations which require entry into tanks or closed vessels, and in emergency situations. If the use of respirators is necessary, the only respirators permitted are those that have been approved by the Mine Safety and Health Administration (formerly Mining Enforcement and Safety Administration) or by the National Institute for Occupational Safety and Health. In addition to respirator selection, a complete respiratory protection program should be instituted which includes regular training, maintenance, inspection, cleaning, and evaluation. /Chromic acid and chromates/ [Mackison, F. U., R. S. Stricoff, and L. J. Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) Publication No. 81-123 (3 VOLS). Washington, DC: U.S. Government Printing Office, Jan. 1981. , p. 31 **PEER REVIEWED**

OTHER PREVENTIVE MEASURES

Eating and smoking should not be permitted in areas where

solids or liquids containing chromic acid or chromates are handled, processed, or stored. Employees who handle solids or liquids containing chromic acid or chromates should wash their hands thoroughly with soap or mild detergent and water before eating, smoking, or using toilet facilities. /Chromic acid and chromates/ [Mackison, F. W., R. S. Stricoff, and L. J. Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) Publication No. 81-123 (3 VOLS). Washington, DC: U.S. Government Printing Office, Jan. 1981. , p. 41 **PEER REVIEWED**

OTHER PREVENTIVE MEASURES

Areas in which exposure to a carcinogenic form of chromium (VI) may occur should be identified by signs or other appropriate means, and access to these areas should be limited to authorized personnel only. /Chromic acid and chromates/ [Mackison, F. W., R. S. Stricoff, and L. J. Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) Publication No. 81-123 (3 VOLS). Washington, DC: U.S. Government Printing Office, Jan. 1981. , p. 41 **PEER REVIEWED**

OTHER PREVENTIVE MEASURES

Employees should wash imnediately when skin becomes contaminated. Work clothing should be changed daily, if it is reasonably probable that the clothing is contaminated. Immediately remove non-impervious clothing that becomes contaminated. /Chromic acid and chromates/ [NIOSH; Pocket Guide to Chemical Hazards p.69 (1981) DHEW (NIOSH) Pub No. 78-2101 **PEER REVIEWED**

OTHER PREVENTIVE MEASURES

A quick drench eyewash should be available. /Chromic acid and chromates/ [NIOSH; Pocket Guide to Chemical Hazards p.69 (1981) DHEW (NIOSH) Pub No. 78-2101 **PEER REVIEWED**

OTHER PREVENTIVE MEASURES

Persons not wearing protective equipment and clothing should be restricted from areas of spills until cleanup has been completed. [Mackison, F. W., R. S. Stricoff, and L. J. Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) Publication No. 81-123 (3 VOLS). Washington, DC: U.S. Government Printing Office, Jan. 1981. , p. 51 **PEER REVIEWED**

OTHER PREVENTIVE MEASURES

Cloakroom accomnodation should be provided for this personal protective equipment with separate accomnodation for clothing taken off during working hours. Washing accomnodation, including bathing accomnodation with warm water, should be provided and used. Time should be allowed for washing before eating. Arrangements should be made to prohibit eating and smoking in the vicinity of lead processes and suitable messrooms should be provided. /Inorganic lead/ [International Labour Office. Encyclopedia of Occupational Health and Safety. Vols. I&II. Geneva, Switzerland: International Labour Office, 1983. , p. 12041 **PEER REVIEWED**

OTHER PREVENTIVE MEASURES

SRP: Contaminated protective clothing should be segregated in such a manner so that there is no direct personal contact by personnel who handle, dispose, or clean the clothing. Quality assurance to ascertain the completeness of the cleaning procedures should be implemented before the decontaminated protective clothing is returned for reuse by the workers. All contaminated clothing should not be taken home at end of shift, but should remain at employee's place of work for cleaning. [CITATION 1 **PEER REVIEWED**

OTHER PREVENTIVE MEASURES

SRP: Local exhaust ventilation should be applied wherever there is an incidence of point source emissions or dispersion of regulated contaminants in the work area. Ventilation control of the contaminant as close to its point of generation is both the most economical and safest method to minimize personnel exposure to airborne contaminants. [Sax, N.I. Dangerous Properties of Industrial Materials. 4th ed. New York: Van Nostrand Reinhold, 1975. 1 **PEER REVIEWED**

OTHER PREVENTIVE MEASURES

1) The employer shall ensure that all personal protective

devices are inspected regularly and maintained in clean and satisfactory working condition. 2) Working clothing and shoes shall not be taken home by employees. The employer shall provide for maintenance and laundering of protective clothing. 3) The employer shall ensure that precautions necessary to protect laundry personnel are taken when soiled protective clothing is laundered. /Inorganic lead/ [NIOSH; Criteria Document: Inorganic lead p.1-9 (1978) DHEW Pub. NIOSH 78-158] **PEER REVIEWED**

OTHER PREVENTIVE MEASURES

Employees should wash daily at the end of each work shift. /Inorganic lead/ [Sittig, M. Handbook of Toxic and Hazardous Chemicals and Carcinogens, 1985. 2nd ed. Park Ridge, NJ: Noyes Data Corporation, 1985. , p. 5441 **PEER REVIEWED**

OTHER PREVENTIVE MEASURES

POISONING SOLELY OR MAINLY FROM THE INGESTION OF LEAD COMPOUNDS IS OF RELATIVELY INFREQUENT OCCURRENCE IN AMERICAN INDUSTRY ... /MAINLY/ BECAUSE OF THE VIRTUAL INEVITABILITY OF THE CONTAMINATION OF THE ATMOSPHERE OF WORKROOMS IN THE LEAD-USING INDUSTRIES WITH LEAD COMPOUNDS, & THE DIFFICULTIES THAT ATTEND THE AVOIDANCE OF SOME DEGREE OF RESPIRATORY EXPOSURE TO, & ABSORPTION OF, THESE COMPOUNDS. ... INGESTION OF LEAD COMPOUNDS IN THE COURSE OF THE DAY'S WORK CAN & SHOULD ALWAYS BE PREVENTED THROUGH EXERCISE OF REGULAR, ATTENTIVE CARE IN MATTERS OF PERSONAL CLEANLINESS & GOOD HOUSEKEEPING, WHILE PREVENTION OF THE INHALATION OF LEAD IS MUCH MORE DIFFICULT. ... THE LARGEST PROPORTION OF THE LEAD ABSORBED BY THE ... VICTIM OF INDUSTRIAL LEAD POISONING IS DERIVED FROM THE RESPIRED AIR. FOR THESE REASONS THE DETERMINATION OF THE RESPIRABLE LEAD IN THE ATMOSPHERE OF WORKROOMS MAY YIELD FAIRLY ADEQUATE INFORMATION AS TO THE MAGNITUDE OF THE OCCUPATIONAL LEAD HAZARD IN SUCH ROOMS. /INORGANIC AND ALKYL LEAD COMPOUNDS/ [Patty, F. (ed.). Industrial Hygiene and Toxicology: Volume 11: Toxicology. 2nd ed. New York: Interscience Publishers, 1963. , p. 9491 **PEER REVIEWED**

OTHER PREVENTIVE MEASURES

OSHA has recomnended engineering controls over administrative controls and protective equipment to reduce exposures to Chemicals in the workplace. The application of employee training and motivation programs (such as job safety analysis) to reduce exposures to chemicals has not been emphasized. To determine the effectiveness of such programs, a pilot project in an alkyl lead production facility was conducted with 35 employees in an effort to reduce exposures to organic and inorganic lead. Results after 12 mo *showed* a *40%* reduction in lead in urine and a 24% reduction in lead in blood, both indicators of total exposure to organic and inorganic Pb. /Inorganic lead/ [Maples TU et al; Am Ind Hyg Assoc J 43 (9): 692-4 (1982)] **PEER REVIEWED**

OTHER PREVENTIVE MEASURES

PRECAUTIONS FOR "CARCINOGENS": Smoking, drinking, eating, storage of food or of food & beverage containers or utensils, & the application of cosmetics should be prohibited in any laboratory. All personnel should remove gloves, if worn, after completion of procedures in which carcinogens have been used. They should ... wash ... hands, preferably using dispensers of liq detergent, & rinse ... thoroughly. Consideration should be given to appropriate methods for cleaning the skin, depending on nature of the contaminant. No standard procedure can be recomnended, but the use of organic solvents should be avoided. Safety pipettes should be used for all pipetting. /Chemical Carcinogens/ [Montesano, R., H. Bartsch, E.Boyland, G. Della Porta, L. Fishbein, R. A. Griesemer, A.B. Swan, L. Tomatis, and W. Davis (eds.). Handling Chemical Carcinogens in the Laboratory: Problems of Safety. IARC Scientific Publications No. 33. Lyon, France: International Agency for Research on Cancer, 1979. , p. 8] **PEER REVIEWED**

OTHER PREVENTIVE MEASURES

PRECAUTIONS FOR "CARCINOGENS": In animal laboratory, personnel should remove their outdoor clothes & wear protective suits (preferably disposable, one-piece & close-fitting at ankles & wrists), gloves, hair covering & overshoes. ... clothing should be changed daily but ... discarded imnediately if obvious

contamination occurs ... /also,/ workers should shower immediately. In chemical laboratory, gloves & gowns should always be worn ... however, gloves should not be assumed to provide full protection. Carefully fitted masks or respirators may be necessary when working with particulates or gases, & disposable plastic aprons might provide addnl protection. If gowns are of distinctive color, this is a reminder that they should not be worn outside of lab. /Chemical Carcinogens/ [Montesano, R., H. Bartsch, E.Boyland, G. Della Porta, L. Fishbein, R. A. Griesemer, A.B. Swan, L. Tomatis, and W. Davis (eds.). Handling Chemical Carcinogens in the Laboratory: Problems of Safety. IARC Scientific Publications No. 33. Lyon, France: International Agency for Research on Cancer, 1979. , p. 8L **PEER REVIEWED**

OTHER PREVENTIVE MEASURES

PRECAUTIONS FOR "CARCINOGENS": ... Operations connected with synth & purification ... should be carried out under well-ventilated hood. Analytical procedures ... should be carried out with care & vapors evolved during ... procedures should be removed. ... Expert advice should be obtained before existing fume cupboards are used ... & when new fume cupboards are installed. It is desirable that there be means for decreasing the rate of air extraction, *so* that carcinogenic powders can be handled without ... powder being blown around the hood. Glove boxes should be kept under negative air pressure. Air changes should be adequate, so that concn of vapors of volatile carcinogens will not occur. /Chemical Carcinogens/ [Montesano, R., ti. Eartsch, E.Boyland, G. Della Porta, L. Fishbein, R. A. Griesemer, A.E. Swan, L. Tomatis, and W. Davis (eds.). Handling Chemical Carcinogens in the Laboratory: Problems of Safety. IARC Scientific Publications No. 33. Lyon, France: International Agency for Research on Cancer, 1979. , p. 8L **PEER REVIEWED**

OTHER PREVENTIVE MEASURES

PRECAUTIONS FOR "CARCINOGENS": Vertical laminar-flow biological safety cabinets may be used for containment of in vitro procedures ... provided that the exhaust air flow is sufficient to provide an inward air flow at the face opening of the cabinet, & contaminated air plenums that are under positive pressure are leak-tight. Horizontal laminar-flow hoods or safety cabinets, where filtered air is blown across the working area towards the operator, should never be used ... Each cabinet or fume cupboard to be used ... should be tested before work is begun (eg, with fume bomb) & label fixed to it, giving date of test & avg air-flow measured. This test should be repeated periodically & after any structural changes. /Chemical Carcinogens/ [Montesano, R., H. Bartsch, E.Boyland, G. Della Porta, L. Fishbein, R. A. Griesemer, A.B. Swan, L. Tomatis, and W. Davis (eds.). Handling Chemical Carcinogens in the Laboratory: Problems of Safety. IARC Scientific Publications No. 33. Lyon, France: International Agency for Research on Cancer, 1979. , *p.* 91 **PEER REVIEWED**

OTHER PREVENTIVE MEASURES

PRECAUTIONS FOR "CARCINOGENS": Principles that apply to chem or biochern lab also apply to microbiological & cell-culture labs ... Special consideration should be given to route of admin. ... Safest method of administering volatile carcinogen is by injection of a soln. Admin by topical application, gavage, or intratracheal instillation should be performed under hood. If chem will be exhaled, animals should be kept under hood during this period. Inhalation exposure requires special equipment. ... unless specifically required, routes of admin other than in the diet should be used. Mixing of carcinogen in diet should be carried out in sealed mixers under fune hood, from which the exhaust is fitted with an efficient particulate filter. Techniques for cleaning mixer & hood should be devised before expt begun. Uhen mixing diets, special protective clothing &, possibly, respirators may be required. /Chemical Carcinogens/ [Montesano, R., H. Bartsch, E.Boyland, G. Della Porta, L. Fishbein, R. A. Griesemer, A.E. Swan, L. Tomatis, and W. Davis (eds.). Handling Chemical Carcinogens in the Laboratory: Problems of Safety. IARC Scientific Publications No. 33. Lyon, France: International Agency for Research on Cancer, 1979. , p.

91 **PEER REVIEWED**

OTHER PREVENTIVE MEASURES

PRECAUTIONS FOR "CARCINOGENS": When ... admin in diet or applied to skin, animals should be kept in cages with solid bottoms & sides & fitted with a filter top. When volatile carcinogens are given, filter tops should not be used. Cages which have been used to house animals that received carcinogens should be decontaminated. Cage-cleaning facilities should be installed in area in which carcinogens are being used, to avoid moving of ... contaminated /cages/. It is difficult to ensure that cages are decontaminated, & monitoring methods are necessary. Situations may exist in which the use of disposable cages should be recomnended, depending on type & amt of carcinogen & efficiency with which it can be removed. /Chemical Carcinogens/ [Montesano, R., H. Bartsch, E.Boyland, G. Della Porta, L. Fishbein, R. A. Griesemer, A.B. Swan, L. Tomatis, and W. Davis (eds.). Handling Chemical Carcinogens in the Laboratory: Problems of Safety. IARC Scientific Publications No. 33. Lyon, France: International Agency for Research on Cancer, 1979. , p. 10] **PEER REVIEWED**

OTHER PREVENTIVE MEASURES

PRECAUTIONS FOR "CARCINOGENS": To eliminate risk that ... contamination in lab could build up during conduct of expt, periodic checks should be carried out on lab atmospheres, surfaces, such as walls, floors & benches, & ... interior of fume hoods & airducts. As well as regular monitoring, check must be carried out after cleaning-up of spillage. Sensitive methods are required when testing lab atmospheres for chem such as nitrosamines. Methods ... should ... where possible, be simple & sensitive. ... /Chemical Carcinogens/ [Montesano, R., H. Bartsch, E.Boyland, G. Della Porta, L. Fishbein, R. A. Griesemer, A.B. Swan, L. Tomatis, and W. Davis (eds.). Handling Chemical Carcinogens in the Laboratory: Problems of Safety. IARC Scientific Publications No. 33. Lyon, France: International Agency for Research on Cancer, 1979. , p. 10] **PEER REVIEWED**

OTHER PREVENTIVE MEASURES

PRECAUTIONS FOR "CARCINOGENS": Rooms in which obvious contamination has occurred, such as spillage, should be decontaminated by lab personnel engaged in expt. Design of expt should ... avoid contamination of permanent equipment. ... Procedures should ensure that maintenance workers are not exposed to carcinogens. ... Particular care should be taken to avoid contamination of drains or ventilation ducts. In cleaning labs, procedures should be used which do not produce aerosols or dispersal of dust, ie, wet mop or vacuum cleaner equipped with high-efficiency particulate filter on exhaust, which are avail comnercially, should be used. Sweeping, brushing & use of dry dusters or mops should be prohibited. Grossly contaminated cleaning materials should not be re-used ... If gowns or towels are contaminated, they should not be sent to laundry, but ... decontaminated or burnt, to avoid any hazard to laundry personnel. /Chemical Carcinogens/ [Montesano, R., H. Bartsch, E.Boyland, G. Della Porta, L. Fishbein, R. A. Griesemer, A.B. Swan, L. Tomatis, and W. Davis (eds.). Handling Chemical Carcinogens in the Laboratory: Problems of Safety. IARC Scientific Publications No. 33. Lyon, France: International Agency for Research on Cancer, 1979. , p. 101 **PEER REVIEWED**

OTHER PREVENTIVE MEASURES

PRECAUTIONS FOR "CARCINOGENS": Doors leading into areas where carcinogens are used ... should be marked distinctively with appropriate labels. Access ... limited to persons involved in expt. ... A prominently displayed notice should give the name of the Scientific Investigator or other person who can advise in an emergency & who can inform others (such as firemen) on the handling of carcinogenic substances. /Chemical Carcinogens/ [Montesano, R., H. Bartsch, E.Boyland, C. Della Porta, L. Fishbein, R. A. Griesemer, A.B. Swan, L. Tomatis, and W. Davis (eds.). Handling Chemical Carcinogens in the Laboratory: Problems of Safety. IARC Scientific Publications No. 33. Lyon, France: International Agency for Research on Cancer, 1979. , p. 11] **PEER REVIEWED**

OTHER PREVENTIVE MEASURES

Protective clothing shall be changed at least daily at the end of the shift and more frequently if it should become grossly contaminated. /Inorganic lead/ [NIOSH: Criteria Document: Inorganic Lead p.1-9 (1978) DHEW Pub. NIOSH 78-1581 **PEER REVIEWED**

STABILITY/SHELF LIFE

CMPD OF CHROMIUM CONSIDERED /LEAD CHROMATE/ ARE ALL STABLE MATERIALS. CIARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-PRESENT. (Multivolume work). V2 1031 **QC REVIEWED**

STABILITY/SHELF LIFE

Trivalent chromium is the most stable oxidation state and hexavalent chromiun is the second most stable oxidation state. /Trivalent and hexavalent chromium/ [USEPA; Health Assessment Document: Chromium p.2-2 (1984) EPA 600/8-83-014F] **QC REVIEWED**

SHIPMENT METHODS AND REGULATIONS

Whenever hazardous materials are to be transported, Title 49 CFR, Transportation, Parts 100-180, published by the US Dept of Transportation, contain the regulatory requirements and must be consulted. [52 FR 16482 (5/5/87)] **PEER REVIEWED**

STORAGE CONDITIONS

IN GENERAL, /TO AVOID INDUSTRIAL FIRES/ MATERIALS WHICH ARE TOXIC AS STORED OR WHICH CAN DECOMPOSE INTO TOXIC COMPONENTS DUE TO CONTACT WITH HEAT, MOISTURE, ACIDS, OR ACID FUMES, SHOULD BE STORED IN COOL, WELL VENTILATED PLACE, OUT OF THE DIRECT RAYS OF THE SUN, AWAY FROM AREAS OF HIGH FIRE HAZARD, & SHOULD BE PERIODICALLY INSPECTED & MONITORED. INCOMPATIBLE MATERIALS SHOULD BE ISOLATED FROM EACH OTHER. /INORGANIC LEAD/ [Sax, N.I. Dangerous Properties of Industrial Materials. 5th ed. New York: Van Nostrand Rheinhold, 1979. , p. 2481 **PEER REVIEWED**

STORAGE CONDITIONS

PRECAUTIONS FOR "CARCINOGENS": Storage site should be as close as practicable to lab in which carcinogens are to be used, so that only small quantities required for ... expt need to be carried. Carcinogens should be kept in only one section of cupboard, an explosion-proof refrigerator or freezer (depending on chemicophysical properties ...) that bears appropriate label. An inventory ... should be kept, showing quantity of carcinogen & date it was acquired ... . Facilities for dispensing ... should *be* contiguous to storage area. /Chemical Carcinogens/ [Montesano, R., H. Bartsch, E.Boyland, G. Della Porta, L. Fishbein, R. A. Griesemer, A.B. Swan, L. Tomatis, and W. Davis (eds.). Handling Chemical Carcinogens in the Laboratory: Problems of Safety. IARC Scientific Publications No. 33. Lyon, France: International Agency for Research on Cancer, 1979. , p. 13] **PEER REVIEWED**

CLEANUP METHODS

Trivalent chromium can be effectively removed from drinking water by conventional coagulation techniques, but these techniques are inadequate when chromium is in the hexavalent form. Reverse osmosis is effective for removal of both forms of chromium. /Trivalent & hexavalent chromium/ [48 FR 45512 (10/5/83)] **PEER REVIEWED**

CLEANUP METHODS

If chromic acid or chromates are spilled, the following steps should be taken: 1. Ventilate area of spill. 2. Collect spilled material in the most convenient and safe manner and deposit in sealed containers for reclamation or for disposal in a certified sanitary landfill. Liquid containing chromic acid or chromates should be absorbed in vermiculite, dry sand, earth, or a similar material. /Chromic acid and chromates/ [Mackison, F. W., R. S. Stricoff, and L. J. Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) Publication No. 81-123 (3 VOLS). Uashington, DC: U.S. Government Printing Office, Jan. 1981. , p. 51 **PEER REVIEWED**

CLEANUP METHODS

PRECAUTIONS FOR "CARCINOGENS": A high efficiency particulate arrestor (HEPA) or charcoal filters can be used to minimize amt of carcinogen in exhausted air ventilated safety cabinets, lab

hoods, glove boxes or animal rooms ... Filter housing that is designed so that used filters can be transferred into plastic bag without contaminating maintenance staff is avail comnercially. Filters should be placed in plastic bags imnediately after removal. ... The plastic bag should be sealed imnediately. ... The sealed bag should be labelled properly ... Waste liquids ... should be placed or collected in proper containers for disposal. The lid should be secured & the bottles properly labelled. Once filled, bottles should be placed in plastic bag, so that outer surface ... is not contaminated. ... The plastic bag should also be sealed & labelled. ... Broken glassware ... should be decontaminated by solvent extraction, by chemical destruction, or in specially designed incinerators. /Chemical Carcinogens/ [Montesano, R., H. Bartsch, E.Boyland, G. Della Porta, L. Fishbein, R. A. Griesemer, A.B. Swan, L. Tomatis, and W. Davis (eds.). Handling Chemical Carcinogens in the Laboratory: Problems of Safety. IARC Scientific Publications No. 33. Lyon, France: International Agency for Research on Cancer, 1979. , p. 151 **PEER REVIEWED**

CLEANUP METHODS

... Described treatment of wastewater from a tetraethyl lead manufacturing process. Two major categories of waste were inorganic lead wastewaters and organic lead wastewaters. After sedimentation in a holding basin to recover solid Lead and lead oxide, the inorganic lead waste fraction (66.1 mg/l) was effectively treated by coagulation with ferric and ferrous sulfate. /Inorganic lead/ [Patterson JW, Industrial Wastewater Treatment Technolgy 2nd Edition p.75 (1985)] **PEER REVIEWED**

CLEANUP METHODS

Environmental considerations for 1) Lard spill: Dig a pit, pond, or lagoon holding area to contain liquid or solid material. /SRP: If time permits, pits, ponds, lagoons, soak holes, or holding areas should be sealed with an impermeable flexible membrane Liner./ Cover solids with a plastic sheet to prevent dissolving in rain or fire fighting water. /Inorganic lead/ [Association of American Railroads. Emergency Handling of Hazardous Materials in Surface Transportation. Washington, D.C.: Assoc. of American Railroads, Hazardous Materials Systems (BOE), 1987. , p. 4071 **PEER REVIEWED**

CLEANUP METHODS

Environmental considerations for 2) Water spill: neutralize with agricultural lime (CaO), crushed limestone (CaCO3), or sodium bicarbonate (NaHCO3). Adjust pH to neutral (pH= 7). Use mechanial dredges or lifts to remove immobilized masses of pollutants & precipitates. /Inorganic lead/ [Association of American Railroads. Emergency Handling of Hazardous Materials in Surface Transportation. Washington, D.C.: Assoc. of American Railroads, Hazardous Materials Systems (BOE), 1987. , p. 4071 **PEER REVIEWED**

DISPOSAL METHODS

SRP: At the time of review, criteria for land treatment or burial (sanitary landfill) disposal practices are subject to significant revision. Prior to implementing Land disposal of waste residue (including uaste sludge), consult with environmental regulatory agencies for guidance on acceptable disposal practices. [CITATION 1 **PEER REVIEWED**

DISPOSAL METHODS

Waste disposal method: Chromic acid or chromates may be disposed of in sealed containers in a secured sanitary landfill. /Chromic acid and chromates/ [Mackison, F. W., R. S. Stricoff, and L. J. Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) Publication No. 81-123 (3 VOLS). Washington, DC: U.S. Government Printing Office, Jan. 1981. , p. 51 **PEER REVIEWED**

DISPOSAL METHODS

PRECAUTIONS FOR "CARCINOGENS": There is no universal method of disposal that has been proved satisfactory for all carcinogenic compounds & specific methods of chem destruction ... published have not been tested on all kinds of carcinogen-containing waste. ... Summary of avail methods & recommendations ... /given/ must be treated as guide only. /Chemical Carcinogens/

[Montesano, R., H. Rartsch, E.Boyland, C. Della Porta, L. Fishbein, R. A. Griesemer, A.B. Swan, L. Tomatis, and W. Davis (eds.). Handling Chemical Carcinogens in the Laboratory: Problems of Safety. IARC Scientific Publications No. 33. Lyon, France: International Agency for Research on Cancer, 1979. , p. 14] **PEER REVIEWED**

DISPOSAL METHODS

PRECAUTIONS FOR "CARCINOGENS": Total destruction ... by incineration may be only feasible method for disposal of contaminated laboratory waste from biological expt. However, not all incinerators are suitable for this purpose.The most efficient type ... is probably the gas-fired type, in which a first-stage combustion with a less than stoichiometric air:fuel ratio is followed by a second stage with excess air. Some ... are designed to accept ... aqueous & organic-solvent solutions, otherwise it is necessary ... to absorb soln onto suitable combustible material, such as sawdust. Alternatively, chem destruction may be used, esp when small quantities ... are to be destroyed in laboratory. /Chemical Carcinogens/ [Montesano, R., H. Bartsch, E.Boyland, G. Della Porta, L. Fishbein, R. A. Griesemer, A.E. Swan, L. Tomatis, and W. Davis (eds.). Handling Chemical Carcinogens in the Laboratory: Problems of Safety. IARC Scientific Publications No. 33. Lyon, France: International Agency for Research on Cancer, 1979. , p. 151 **PEER REVIEWED**

DISPOSAL METHODS

PRECAUTIONS FOR "CARCINOGENS": HEPA (high-efficiency particulate arrestor) filters ... can be disposed of by incineration. For spent charcoal filters, the adsorbed material can be stripped off at high temp & carcinogenic wastes generated by this treatment conducted to & burned in an incinerator. ... LIQUID WASTE: ... Disposal should be carried out by incineration at temp that ... ensure complete combustion. SOLID WASTE: Carcasses of lab animals, cage litter & misc solid wastes ... should be disposed of by incineration at temp high enough to ensure destruction of chem carcinogens or their metabolites. /Chemical Carcinogens/ [Montesano, R., H. Bartsch, E.Boyland, G. Della Porta, L. Fishbein, R. A. Griesemer, A.B. Swan, L. Tomatis, and U. Davis (eds.). Handling Chemical Carcinogens in the Laboratory: Problems of Safety. IARC Scientific Publications No. 33. Lyon, France: International Agency for Research on Cancer, 1979. , p. 151 **PEER REVIEWED**

DISPOSAL METHODS

PRECAUTIONS FOR "CARCINOGENS": ... Small quantities of ... some carcinogens can be destroyed using chem reactions ... but no general rules can be given. ... As a general technique ... treatment with sodium dichromate in strong sulfuric acid can be used. The time necessary for destruction ... is seldom known ... but 1-2 days is generally considered sufficient when freshly prepd reagent is used. ... Carcinogens that are easily oxidizable can be destroyed with milder oxidative agents, such as sat soln of potassium permanganate in acetone, which appears to be a suitable agent for destruction of hydrazines or of compounds containing isolated carbon-carbon double bonds. Concn or 50% aqueous sodium hypochlorite can also be used as an oxidizing agent. /Chemical Carcinogens/ [Montesano, R., H. Bartsch, E.Boyland, G. Della Porta, L. Fishbein, R. A. Griesemer, A.B. Swan, L. Tomatis, and U. Davis (eds.). Handling Chemical Carcinogens in the Laboratory: Problems of Safety. IARC Scientific Publications No. 33. Lyon, France: International Agency for Research on Cancer, 1979. , p. 161 **PEER REVIEWED**

DISPOSAL METHODS

Chemical Treatability of Lead; Concentration Process: Biological Treatment; Chemical Classification: Metal; Scale of Study: Respirometer Study; Results of Study: Oxygen uptake inhibited. IUSEPA; Management of Hazardous Waste Leachate, EPA Contract No. 68-03-2766 p.E-53 (1982)] **PEER REVIEWED**

DISPOSAL METHODS

The report describes a bench scale fluidized bed incinerator that will capture trace metals on the bed material when firing hazardous waste. The design is based on limited tests at an

existing laboratory facility. Operating conditions, operating procedures, and equipnent design are established for greater than 90 percent trace metal capture on the bed material. A surrogate hazardous waste, paint containing lead chromate, was used in the tests. Other trace metals were identified that can be captured by agglomeration on a silica bed material. The design provides the capability of operating in either a single or double stage configuration so that various bed materials or operating conditions can be used to capture different trace metals or to provide more effective capture. [Litt RD, Tewksbury TL; Govt Reports Announcement 3: Index 5 (1985)] **PEER REVIEWED**

TOXIC HAZARD RATING

A) Evidence for carcinogenicity to humans (sufficient for hexavalent chromium compounds); B) Evidence for carcinogenicity to animals (sufficient for hexavalent chromium compounds). Overall evaluation is Group 1. The working group concluded that hexavalent chromium compounds are carcinogenic to humans. (The evaluation applies to the group of chemicals as a whole & not necessarily to all individual chemicals within the group ...). /Hexavalent chromium compounds/ [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-PRESENT. (Multivolume work). S7 165 (1987)] **PEER REVIEWED**

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Overview Title: LEAD

EMERGENCY MEDICAL TREATMENT

o LIFE SUPPORT :

This overview assumes that basic life support measures have been instituted.

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o CLINICAL EFFECTS :

SUMMARY

- o Signs and symptoms of lead poisoning are referable to the nervous, hematologic, renal, gastrointestinal, and cardiac systems (Cullen et al, 1983). There may be no unique signs or symptoms to suggest lead poisoning as the diagnosis. Serious injury can occur when neither signs nor symptoms have been present. Case detection is best done by screening populations at risk, which include, for example, operatives in lead industries, their children, and children living in homes built before 1980 where lead-containing paint may be the source. Universal screening of children beginning at 6 months of age, is recommended by the CDC.
- o Most lead poisoning is slow in onset and results from gradual accumulation of lead from sources of low solubility such as lead paint or industrial dusts. The ingestion of rapidly absorbed salts causes an acute syndrome of hepatic injury and hemolysis as well as chronic exposure effects.

VITAL SIGNS

- o Chronic lead exposure may cause hypertension.

CARDIOVASCULAR

- o Chronic exposure in adults leads to renal hypertension and secondary cardiac effects.

NEUROLOGIC

- o In young children, developnental defects, including learning disabilities, Lowered IQ, and behavioral abnormalities, can occur without symptoms. At higher levels of exposure, non-specific signs occur. At high levels, encephalopathy with imminent risk of death, permanent mental retardation, and motor deficits.

GASTROINTESTINAL

- Anorexia, constipation, and diarrhea may be present due to direct effects or the result of altered CNS control.

HEPATIC

- Liver injury occurs from poisoning with readily available lead salts but rarely with chronic inorganic lead exposure.

GENITOURINARY

- In acutely ill patients, proteinuria, glucosuria, and aminoaciduria may occur. Chronic exposure leads to tubular damage, azotemia, and gout.
- Decreased sperm count in lead exposed males has been described.

METABOLISM

- Hyperuricemia may be seen after chronic lead poisoning.

HEMATOLOGIC

- Lead interrupts several steps in heme synthesis resulting in anemia. Assay of substrates of blocked enzymes is used in diagnosis. With some lead salts, hemolysis is possible. RBCs show endoplasmic clumping known as stippling.

MUSCULOSKELETAL

- Lead is deposited in teeth and bones. In children, lead deposition can be seen as an opacity in the metaphyseal plate.

OTHER

- Lead sulfide precipitates in gum margins causing a blue-black line. Present in older children and adults; rarely in children <5 years as the sulfide comes from bacteria rarely found in the mouths of young children.

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o LABORATORY :

- Elevation of blood lead level is essential to the diagnosis of childhood and industrial cases. Children with a blood lead level of *45* or greater require medical treatment.
- Children with blood lead levels between 25 and *44* mcg/dL may need evaluation with a CaNa2EDTA challenge test and children with levels of 10 to *24* mcg/dL require medical monitoring and environmental evaluation.

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o TREATMENT OVERVIEW :

SUMMARY

- Preventing futher lead exposure is paramount. Treatment may include the removal of lead from the body; this is complicated by the large amount which is in the pharmacokinetically deep compartments of bone and teeth. In encephalopathy, treatment of cerebral edema must be concurrent with chelation therapy.

ORAL EXPOSURE

- ACUTE INGESTION
  3. Cleansing enema should be administered if lead is found by abdominal x-ray or significant recent oral ingestion is suspected.
- ACUTE/CHRONIC INGESTION
  1. OBTAIN BLOOD LEAD LEVEL
     a. Hospitalize any patient with significant CNS or protracted gastrointestinal symptoms, or with a blood lead level (BLL) of *50* micrograms/100 milliliters or greater, or with a positive EDTA challenge (provocative test) even if no symptoms are present (Piomelli et al, *1984).* Chelation therapy should be instituted in all patients with BLL of *45* micrograms/deciliter *(2.2* micromoles/liter) or greater. Patients treated as outpatients must be discharged to a lead-free environment.
     b. Children with a BLL of 20 to *44* micrograms/deciliter should receive a complete medical examination including testing for iron deficiency; environmental lead sources should be identified and removed. Children with blood lead levels of *45* to *69* micrograms/deciliter need to have medical treatment

and environmental assessment and remediation instituted within *48* hours. Children whose BLL are >69 micrograms/deciliter must receive medical treatment and have environmental concerns addressed imnediately (CDC, *1991*).

c. Admit all with evidence of CNS involvement.,
d. Consider admission for work-up when diagnosis in doubt (Piomelli et al, *1984).*

2. CALCIUM EDTA PROVOCATION TEST: May be useful in determining necessity for therapeutic chelation in children with blood lead levels of *25* to *44* mcg/dL. This may be performed as an outpatient if the patient remains in the clinic (Piomelli et al, 1984).

3. CHELATION THERAPY

a. Should be instituted in all patients with blood lead level equal to or greater than *45* mcg/dL *(2.2* mcmol/L) even if asymptomatic, and in patients with a positive calcium EDTA provocation test. Symptoms and signs, along with the EP and blood lead level, determine the route, dose, and agent to be used for chelation.
b. BAL (dimercaprol): *3* to 5 mg/kg/dose deep IM every *4* hours for 2 days; then every *4* to 6 hours for *2* more days; then every *4* to 12 hours up to an additional 7 days.
c. CALCIUM EDTA: *50* to *75* mg/kg/day deep IM in 3 to 6 divided doses for up to *5* days. EDTA should only be administered after BAL in patients with encephalopathy or children with levels >69 mcg/dL.
d. D-PENICILLAMINE: *250* mg *4* times a day PO for up to *5* days. Do not exceed 40 mg/kg/day. OSHA prohibits prophylactic chelation therapy in workers occupationally exposed to lead.

4. ESTABLISH ADEQUATE FLUID BALANCE with a urine flow of *1* to 2 mL/kg/hour. Do not force fluids.

5. PERFORM A NEUROLOGICAL EXAM with particular reference to the presence of encephalopathy. Spinal tap may be dangerous in the presence of increased intracranial pressure.

6. CEREBRAL EDEMA: May be managed by ventilation and the administration of *1.5* g/kg of *20%* mannitol IV over *20* minutes. Dexamethasone: up to 1 to *2* mg/kg/day IV in divided doses.

8. FOR ENCEPHALOPATHY, institute BAL and EDTA simultaneously in the maximum dose.

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o REFERENCE : [Rumack BH & Spoerke DG: POISINDEX(R) Information System. Micromedex Inc., Denver, CO, *1993;* CCIS CD-ROM Volume 78, edition exp November, *1993.* 1 **PEER REVIEWED**

EMERGENCY MEDICAL TREATMENT

o LIFE SUPPORT :

This overview assumes that basic life support measures have been instituted.

.....

o CLINICAL EFFECTS :

SUMMARY

o Oral ingestion produces gastrointestinal corrosion and acute multisystem shock, followed by renal failure, hemorrhagic diathesis, and hepatic injury within several days.

HEENT

o Oral burns and severe corneal injury may result from acute exposure. Chronic inhalation produces deep perforating nasal ulcers (chrome holes).

CARDIOVASCULAR

o Circulatory collapse and shock are frequently reported following overdose.

RESPIRATORY

o Pulmonary edema, pneumonoconiosis, metal fume fever, and

bronchial asthma may occur.

NEUROLOGIC
- Hepatic encephalopathy may occur.

GASTROINTESTINAL
- Gastroenteritis and hemorrhage frequently occur imnediately following oral ingestion.

HEPATIC
- Acute hepatitis may be a late manifestation.

GENITOURINARY
- Renal failure is comnonly observed during the first few days after ingestion.

HEMATOLOGIC
- Thrombocytopenia, and anemia usually occur after 3 to 7 days. Methemoglobinemia has been reported.

DERMATOLOGIC
- Deep perforating ulcers and hypersensitivity dermatitis may be noted. Systemic toxicity has resulted from minimal dermal exposure.

CARCINOGENICITY
- An increased incidence of lung cancer has been associated with chronic exposure to hexavalent chromium.

IMMUNOLOGIC
- Exposure to chromium vapors may result in an anaphylactoid reaction.

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o LABORATORY :

- The presence of chromium and chromium complexes in biological specimens can be determined using chromatographic and colorimetric techniques. Patch testing and lymphocyte proliferation testing have been used to determine chromium sensitivity.

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o TREATMENT OVERVIEW :

ORAL EXPOSURE
- DO NOT induce vomiting.
- Administer ascorbic acid (1 gram per 0.135 gram of elemental chromium).
- Exchange transfusion is recomnended in all substantial ingestions.
- FORCED DIURESIS: After initial hydration administer furosemide 1 mg/kg up to *40* mg/dose.
- MONITOR VOLUME STATUS, HEMATOCRIT, AND PLATELET COUNT.

INHALATION EXPOSURE
- Monitor for respiratory distress for 72 hours.
- Obtain baseline chest x-ray and vital signs.
- Pulmonary edema may be managed with PEEP and short-term steroid therapy.

EYE EXPOSURE

DERMAL EXPOSURE
- Wash the exposed area with water or 10 to 20% ascorbic acid solution for 15 minutes. A physician may need to examine the exposed area if irritation or pain persists.
- Excision of affected skin is recomnended in severe exposures.

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o RANGE OF TOXICITY :

- Serious toxicity has resulted from ingestion of 0.5 gm of hexavalent chromium. Death has resulted from 1 to *8* gm and survival with 15 gm. Dermal involvement of 10% of body surface has been fatal. Trivalent chromium has not been associated with toxicity.

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o REFERENCE : [Rumack BH & Spoerke DC: POISINDEX(R) Information System. Micromedex Inc., Denver, CO, 1993; CCIS CD-ROM Volume *78,* edition exp November, 1993.]
PEER REVIEWED

ANTIDOTE AND EMERGENCY TREATMENT

Exptl Therapy: Potentials of 2,3,-dimercaptosuccinic acid (DMS) and D-penicillamine, the currently acclaimed heavy metal

chelating drugs, to enhance urinary excretion of lead and restore altered levels of urinary delta-aminolevulinic acid (delta-ALA) and blood lead, zinc protoporphyrin (ZPP), and delta-aminolevulinic acid dehydratase (delta-ALA-D) in Pb poisoned rabbits, were compared. The better performance of 2,3-dimercaptosuccinic acid than D-penicillamine was attributed to two -SH groups as stronger metal binding sites in the 2,3-dimercaptosuccinic acid molecule. /Inorganic lead/ [Tandon SK et al; Res Comnun Chem Pathol Pharmacol 32 (3): 557-60 (1981)] **PEER REVIEWED**

ANTIDOTE AND EMERGENCY TREATMENT

EXPTL THERAPY: THERAPEUTIC DOSES OF THIAMINE MAY BE OF VALUE IN PREVENTION & TREATMENT OF LEAD POISONING IN CATTLE & IN OTHER ANIMALS, OR HUMANS EXPOSED TO HIGH ENVIRONMENTAL LEVELS OF LEAD. [BRATTON GR ET AL; TOXICOL APPL PHARMACOL 59 (1): 164 (1981)] **PEER REVIEWED**

MEDICAL SURVEILLANCE

Initial Medical Examination: 1) A complete history and physical examination: The purpose is to detect pre-existing conditions that might place the exposed employee at increased risk, and to establish a baseline for future health monitoring. ... Exam of the respiratory system, blood, liver, and kidneys should be stressed. The skin should be examined for evidence of chronic disorders. 2) A complete blood count: Chromates have been shoun to cause blood changes in humans. A complete blood count should be performed, including a red cell count, a white cell count, a differential count of a stained smear, as well as hemoglobin and hematocrit. 3) 14" x 17" chest roentgenogram: Chromates may cause human lung damage and are associated with a high incidence of lung cancer. Surveillance of the lung is indicated. 4) FVC and FEV (1 sec): Chromates are reported to cause decreased pulmonary function. Periodic surveillance is indicated. 5) Urinalysis: Since chromates may cause kidney damage, a urinalysis should be obtained, including, at a minimum specific gravity, albumin, glucose, & a microscopic ... /examination of/ centrifuged sediment. 6) Liver function tests: Chromates may cause liver damage. A profile of liver function should be obtained by utilizing a medically acceptable array of biochemical tests. 7) Skin disease: Chromates are defatting agents and can cause dermatitis on prolonged exposure. Persons with pre-existing skin disorders may be more susceptible to the effects of these agents. Periodic Medical Examination: The aforementioned medical examination should be repeated on annual basis. /Chromic acid and chromates/ [Mackison, F. U., R. S. Stricoff, and L. J. Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) Publication No. 81-123 (3 VOLS). Washington, DC: U.S. Government Printing Office, Jan. 1981. , p. 21 **PEER REVIEWED**

MEDICAL SURVEILLANCE

Periodic Medical Examination: ... Emphasis should be placed on observation for changes in the mucous membranes of the upper respiratory tract, ulceration of the skin, and surveillance for malignancy of the respiratory tract. /Chromic acid and chromates/ [Mackison, F. U., R. S. Stricoff, and L. J. Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) Publication No. 81-123 (3 VOLS). Washington, DC: U.S. Government Printing Office, Jan. 1981. , p. 2] **PEER REVIEWED**

MEDICAL SURVEILLANCE

Reporting Signs and Symptoms: A physician should be contacted if anyone develops any signs or symptoms and suspects that they are caused by exposure to chromic acid or chromates. /Chromic acid and chromates/ [Mackison, F. W., R. S. Stricoff, and L. J. Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) Publication No. 81-123 (3 VOLS). Washington, DC: U.S. Government Printing Office, Jan. 1981. , p. 21 **PEER REVIEWED**

MEDICAL SURVEILLANCE

... If environmental sampling and analysis show that environmental levels are at or greater than the recomnended environmental levels, ... blood analysis shall be conducted quarterly ... until blood levels have returned to below the

acceptable limit. ... If a blood level of 0.060 mg Pb/100 g or greater is found and confirmed by a second sample to be taken within two weeks, steps to reduce absorption of lead shall be taken as soon as the high levels are confirmed and a medical examination for possible lead poisoning shall be made available to workers with unacceptable blood lead levels. /Inorganic lead/ INIOSH; Criteria Document: Inorganic Lead p.I-2 (1978) DHEW Pub. NIOSH 78-158] **PEER REVIEWED**

MEDICAL SURVEILLANCE

Medical records shall include information on all biologic determinations and on all required medical examinations. These records shall be available to the medical representatives of the employer, the Secretary of Labor, the Secretary of Health, Education, and Welfare and at the employee's request, to the employee's physician. These records shall be kept for at least 30 years after the last occupational exposure to inorganic lead. /Inorganic lead/ INIOSH; Criteria Document: Inorganic Lead p.I-2 (1978) DHEW Pub. NIOSH 78-158] **PEER REVIEWED**

MEDICAL SURVEILLANCE

... Comparative advantages of /the biological indicator/ delta-aminolevulinic acid dehydratase (ALAD) in typical (variable) occupational exposure conditions included: the highest sensitivity at low and relatively high lead exposure levels; better reflection of biologically active Pb as opposed to blood lead (PbB) (particularly compared to delta-aminolevulinic acid (ALAU) and coproporphyrin (CPU)); higher specificity compared to other indicators of lead effect; and generally higher reliability with regard to biologically and methodologically induced variations. ... /Inorganic lead/ [Telisman S et al; Int Arch Occup Environ Health 50 (4): 397-412 (1982)] **PEER REVIEWED**

MEDICAL SURVEILLANCE

The employer shall make available biological monitoring in the form of blood sampling and analysis for lead and zinc protoporphyrin levels to each employee who is or may be exposed above the action level for more than 30 days/yr. /Inorganic lead/ [29 CFR 1910.1025 (7/1/87)] **PEER REVIEWED**

MEDICAL SURVEILLANCE

A thorough physical exam, with particular attention to teeth, gums, hematologic, gastrointestinal, renal, cardiovascular, neurological, ... /and pulmonary systems/. /Inorganic lead/ t29 CFR 1910.1025 (7/1/87)] **PEER REVIEWED**

MEDICAL SURVEILLANCE

PRECAUTIONS FOR "CARCINOGENS": ... In relation specifically to cancer hazards, there are at present no health monitoring methods that may ensure the early detection of preneoplastic lesions or lesions which may preclude them. Whenever medical surveillance is indicated, in particular when exposure to a carcinogen has occurred, ad hoc decisions should be taken concerning additional tests that might become useful or mandatory. /Chemical Carcinogens/ [Montesano, R., H. Bartsch, E.Boyland, G. Della Porta, L. Fishbein, R. A. Griesemer, A.B. Swan, L. Tomatis, and W. Davis (eds.). Handling Chemical Carcinogens in the Laboratory: Problems of Safety. IARC Scientific Publications No. 33. Lyon, France: International Agency for Research on Cancer, 1979. , p. 231 **PEER REVIEWED**

MEDICAL SURVEILLANCE

... Unacceptable absorption of lead posing a risk of lead poisoning is demonstrated at levels equal to or greater than 0.060 mg Pb/100 g whole blood. All workers subject to exposure to inorganic lead shall be offered biologic monitoring at least every 6 months. ... A medical examination for possible lead poisoning shall be made available to workers with unacceptable blood lead levels. Medical examination: Medical examinations shall be made available prior to employee placement and annually thereafter, unless a different frequency is indicated by professional medical judgement, based on such factors as emergencies, variations in work periods, and the existing health status of the individual worker. These examinations should focus on the blood forming elements, the kidneys, and the nervous and reproductive systems. They should include a physical examination, complete blood counts, blood lead determinations, routine urinalysis (specific gravity, sugar and

protein determinations, and microscopic examination), and should record any signs or symptoms of plumbism present. ... Each employee who absorbs unacceptable amounts of lead, as indicated by biologic monitoring, shall be examined as soon as practical after such absorption is demonstrated and confirmed, and at least every three months thereafter until blood levels have returned to below the acceptable limit, ie below 0.060 mg/100 g blood. If clinical evidence of plumbism is obtained from the medical examinations, the worker shall be kept under a physician's care until completely recovered, or maximal improvement has occurred. Medical records shall include information on all biologic determinations and on all required medical examinations. ... These records shall be kept for at least 30 years after the last occupational exposure to inorganic lead. /Inorganic lead/ INIOSH; Criteria Document: Inorganic Lead p.1-2 (1978) DHEW Pub. NIOSH 78-158] **PEER REVIEWED**

MEDICAL SURVEILLANCE

A complete and detailed work history is important in the initial evaluation. A listing of all previous employment with information on work processes, exposure to fumes or dust, known exposures to lead or other toxic substances, respiratory protection used, and previous medical surveillance should all be included in the worker's record. Where exposure to lead is suspected, information concerning on the job personal hygiene, smoking or eating habits in work areas, laundry procedures, and use of any protective clothing or respiratory protection equipment should be noted. A complete work history is essential in the medical evaluation of a worker with suspected lead toxicity, especially when long term effects such as neurotoxicity and nephrotoxicity are considered. The medical history is also of fundamental importance and should include a listing of all past and current medical conditions, current medications including proprietary drug intake, previous surgeries and hospitalizations, allergies, smoking history, alcohol consumption and also non-occupational lead exposures such as hobbies (hunting, riflery). Also known childhood exposures should be elicited. Any previous history of hematological, neurological, gastrointestinal, renal, psychological, gynecological, genetic, or reproductive problems should be specifically noted. A careful and complete review of systems must be performed to assess both recognized complaints and subtle or slowly acquired symptoms which the worker might not appreciate as being significant. /Lead and lead cmpd/ [29 CFR 1910.1025 (7/01/87)] **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

Potential symptoms as a result of exposure: Respiratory & nasal septum irritations; leukocytosis, leukopenia, monocytosis, eosinophilia; eye injury, conjunctivitis, skin injury, sensitization dermatitis. INIOSH. Pocket Guide to Chemical Hazards. 5th Printing/Revision. DHHS (NIOSH) Publ. No. 85-114. Washington, D.C.: U.S. Dept. of Health and Human Services, NIOSH/Supt. of Documents, GPO, Sept. 1985. , p. 831 **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

TOXIC BY INGESTION & INHALATION. [Sax, N.I. and R.J. Lewis, Sr. (eds.). Hawley's Condensed Chemical Dictionary. 11th ed. New York: Van Nostrand Reinhold Co., 1987. , p. 6891 **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

... HIGH RELATIVE FREQUENCY OF DEATH FROM RESP CANCER AMONG WORKERS IN CHROMATE PRODUCING INDUSTRY /REPORTED/. AMONG 193 DEATHS FROM ALL CAUSES AT 6 CHROMATE-PRODUCING PLANTS IN US, 21.8% RESULTED FROM RESP CANCER, AS COMPARED TO AN EXPECTED FREQUENCY OF 1.4% IN CONTROL GRWP FROM OTHER INDUSTRIES. ... IN ADDITION, CLINICAL OBSERVATIONS FROM GERMANY HAVE SUGGESTED POSSIBLE RELATION OF LUNG CANCER TO CHROME PIGMENT INDUSTRY ... /CHROMATES/ IIARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-PRESENT. (Multivolume work). V2 118 (1973)] **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

Using the IARC classification scheme, the level of carcinogenic evidence available for the combined animal and human data would place hexavalent chromium (Cr(VI)), compounds into Group 1, meaning that there is decisive evidence for the human carcinogenicity of these compounds. /Hexavalent chromium/ [USEPA; Health Assessment Docunent: Chromium p.2-11 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

The lifetime cancer risk due to air containing 1 ug/cu m of hexavalent chromium compounds is estimated *to* be 1.2x10-2. This would place hexavalent chromium (Cr(VI)) in the first quartile of the ... compounds evaluated by the Carcinogen Assessment Group (CAG) for relative carcinogenic potency. /Hexavalent chromium/ [USEPA; Health Assessment Document: Chromium p.2-11 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

Effects on the upper respiratory tract have been observed in workers in chromium-related industries. The major effects of chromium on this system include ulceration of the nasal septum, with subsequent perforation, and chronic rhinitis and pharingitis. Early studies indicated that approximately one-half to four-fifths of the workers in chromate plants had perforated nasal septa, at levels of exposure that approached 1 mg/cu m. Subsequent work indicated that chromic acid levels exceeding 0.1 mg/cu m also caused perforated septa in some workers. /Chromates/ [USEPA; Health Assessment Document: Chromium p.2-8 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

Allergic contact dermatitis may arise from exposure to either trivalent or hexavalent chromium, although hexavalent chromium is responsible for most of the reported cases. Cr(VI) penetrates undamaged skin, and subsequently reduces to Cr(III) which combines with proteins or other skin components to form a whole skin allergen. /Hexavalent chromium/ [USEPA; Health Assessment Document: Chromium p.2-8 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

Acute lead poisoning is relatively infrequent and occurs from ingestion of acid soluble lead compounds or inhalation of lead vapors. Local actions in the mouth produce marked astringency, thirst, and a metallic taste. Nausea, abdominal pain, and vomiting ensue. The vomitus may be milky from the presence of lead chloride. Although the abdominal pain is severe, it is unlike that of chronic poisoning. Stools may be black from lead sulfide, and there may be diarrhea or constipation. If large amounts of lead are absorbed rapidly, a shock syndrome may develop secondary to massive gastrointestinal loss of fluid. Acute central nervous system symptoms incl paresthesia, pain, and muscle weakness. An acute hemolytic crisis sometimes occurs and causes severe anemia and hemoglobinuria. The kidneys are damaged, and oliguria and urinary changes are evident. Death may occur in 1 or 2 days. If the patient survives the acute episode, characteristic signs & symptoms of chronic lead poisoning are likely to appear. /Inorganic lead cmpd/ [Gilman, A.G., L.S.Goodman, and A. Gilman. (eds.). Goodman and Gilman's The Pharmacological Basis of Therapeutics. 7th ed. New York: Macmillan Publishing Co., Inc., 1985. , p. 16071 **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

Signs & symptoms of chronic lead poisoning (plumbism) can be divided into 6 categories: gastrointestinal, neuromuscular, CNS, hematological, renal, & other. They may occur separately or in combination. The neuromuscular & CNS syndromes usually result from intense exposure, while the abdominal syndrome is a more comnon manifestation of a very slowly & insidiously developing intoxication. In the United States, the CNS syndrome is usually more comnon among children, while the GI syndrome is more prevelant in adults. /Inorganic lead/ [Gilman, A.G., L.S.Goodman, and A. Gilman. (eds.). Goodman and Gilman's The Pharmacological Basis of Therapeutics. 7th ed. New York: Macmillan Publishing Co., Inc., 1985. , p. 16071 **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

... /IN CHRONIC LEAD POISONING/ THE ABDOMINAL SYNDROME OFTEN BEGINS WITH VAGUE SYMPTOMS, SUCH AS ANOREXIA, MUSCLE DISCOMFORT, MALAISE, AND HEADACHE. CONSTIPATION IS USUALLY AN EARLY SIGN, ESP IN ADULTS, BUT DIARRHEA OCCASIONALLY OCCURS. A PERSISTENT METALLIC TASTE APPEARS EARLY IN THE COURSE OF THE SYNDROME. AS INTOXICATION ADVANCES, ANOREXIA AND CONSTIPATION BECOME MORE MARKED. INTESTINAL SPASM, WHICH CAUSES SEVERE ABDOMINAL PAIN, OR LEAD COLIC, IS THE MOST DISTRESSING FEATURE OF THE ADVANCED ABDOMINAL SYNDROME. THE ATTACKS ARE PAROXYSMAL AND GENERALLY EXCRUCIATING. THE ABDOMINAL MUSCLES BECOME RIGID, & TENDERNESS IS ESP MANIFESTED IN THE REGION OF THE UMBILICUS. /INORGANIC LEAD/ [Gilman, A.G., L.S.Goodman, and A. Gilman. (eds.). Goodman and Gilman's The Pharmacological Basis of Therapeutics. 7th ed. New York: Macmillan Publishing Co., Inc., 1985. , p. 16071 **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

NEUROMUSCULAR SYNDROME OR LEAD PALSY IS NOW RARE IN THE UNITED STATES. IT IS A MANIFESTATION OF ADVANCED SUBACUTE POISONING. MUSCLE WEAKNESS & EASY FATIGUE OCCUR LONG BEFORE ACTUAL PARALYSIS AND MAY BE THE ONLY SYMPTOMS. WEAKNESS OR PALSY MAY NOT BECOME EVIDENT UNTIL AFTER EXTENDED MUSCLE ACTIVITY. THE MUSCLE GROUPS INVOLVED ARE USUALLY THE MOST ACTIVE ONES (EXTENSORS OF THE FOREARM, WRIST, & FINGERS & EXTRAOCULAR MUSCLES), & THE PALSY OFTEN OCCURS ONLY ON THE DOMINANT SIDE. WRIST DROP &, TO A LESSER EXTENT, FOOT DROP WITH APPROPRIATE HISTORY OF EXPOSURE HAVE BEEN CONSIDERED ALMOST PATHOGNOMONIC FOR LEAD POISONING. THERE IS USUALLY NO SENSORY INVOLVEMENT. DEGENERATIVE CHANGES IN MOTONEURONS & THEIR AXONS HAVE BEEN DESCRIBED. /INORGANIC LEAD/ [Gilman, A.G., L.S.Goodman, and A. Gilman. (eds.). Goodman and Gilman's The Pharmacological Basis of Therapeutics. 7th ed. New York: Macmillan Publishing Co., Inc., 1985. , p. 16071 **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

THE CNS SYNDROME HAS BEEN TERMED LEAD ENCEPHALOPATHY. IT IS THE MOST SERIOUS MANIFESTATION OF LEAD POISONING, AND IS MUCH MORE COMMON IN CHILDREN THAN IN ADULTS. THE EARLY SIGNS OF THE SYNDROME MAY BE CLUMSINESS, VERTIGO, ATAXIA, FALLING, HEADACHE, INSOMNIA, RESTLESSNESS, AND IRRITABILITY. AS THE ENCEPHALOPATHY DEVELOPS, THE PATIENT MAY FIRST BECOME EXCITED AND CONFUSED; DELIRIUM WITH REPETITIVE TONIC-CLONIC CONVULSIONS OR LETHARGY & COMA FOLLOW. VOMITING, A COMMON SIGN, IS USUALLY PROJECTILE. VISUAL DISTURBANCES ARE ALSO PRESENT. ... TREATMENT FOR CEREBRAL EDEMA MAY BECOME NECESSARY. THERE MAY BE PROLIFERATIVE MENINGITIS, INTENSE EDEMA, PUNCTATE HEMORRHAGES, GLIOSIS AND AREAS OF FOCAL NECROSIS. ... THE MORTALITY RATE AMONG PATIENTS WHO DEVELOP CEREBRAL INVOLVEMENT IS ABOUT 25%. WHEN CHELATION THERAPY IS BEGUN AFTER THE SYMPTOMS OF ACUTE ENCEPHALOPATHY APPEAR, APPROXIMATELY 40% OF SURVIVORS HAVE NEUROLOGICAL SEQUELAE, SUCH AS MENTAL RETARDATION, EEG ABNORMALITIES OR ... SEIZURES, CEREBRAL PALSY, OPTIC ATROPHY, OR DYSTONIA MUSCULORUM DEFORMANS. /INORGANIC LEAD/ [Gilman, A.G., L.S.Goodman, and A. Gilman. (eds.). Goodman and Gilman's The Pharmacological Basis of Therapeutics. 7th ed. New York: Macmillan Publishing Co., Inc., 1985. , p. 16081 **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

EXPOSURE TO LEAD OCCASIONALLY PRODUCES ... PROGRESSIVE MENTAL DETERIORATION IN CHILDREN. THE HISTORY OF THESE CHILDREN INDICATES NORMAL DEVELOPMENT DURING THE FIRST 12-18 MONTHS OF LIFE OR LONGER, FOLLOWED BY A STEADY LOSS OF MOTOR SKILLS & SPEECH. THEY MAY HAVE SEVERE HYPERKINETIC AND AGGRESSIVE BEHAVIOR DISORDERS & A POORLY CONTROLLED CONVULSIVE DISORDER. THE LACK OF SENSORY PERCEPTION SEVERELY IMPAIRS LEARNING. CONCN OF LEAD IN BLOOD EXCEED 60 UG/DL OF WHOLE BLOOD, & X-RAY MAY SHOW HEAVY, MULTIPLE BANDS OF INCR DENSITY IN THE GROWING BONES. ... AN INCREASED INCIDENCE OF HYPERKINETIC BEHAVIOR & A STATISTICALLY SIGNIFICANT, ALTHOUGH MODEST, DECREASE IN IQ HAVE BEEN SHOWN IN CHILDREN WITH BLOOD LEAD CONCN OF 30-50 UG/DL. /INORGANIC LEAD/ [Gilman, A.G., L.S.Goodman, and A. Gilman. (eds.). Goodman and Gilman's The Pharmacological Basis of Therapeutics. 7th ed. New York: Macmillan Publishing Co., Inc., 1985. , p. 16081 **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

Recent research work on the clinical effects of lead has

focused on the subtle neuropsychiatric, reproductive, & renal effects of chronic low dose lead exposure. Children are particularly susceptible to lead induced impairment of neuropsychological development because of their reduced ability to excrete lead & their enhanced absorption of lead compared to adults. Death in children from undetected lead poisoning may be greater than heretofore suspected. /Inorganic lead/ [Ellenhorn, M.J. and D.G. Barceloux. Medical Toxicology - Diagnosis and Treatment of Human Poisoning. New York, NY: Elsevier Science Publishing Co., Inc. 1988. , p. 10301 **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

Renal toxicity occurs in two forms: a reversible renal tubular disorder (usually seen after acute exposure of children to lead) and an irreversible interstitial nephropathy (more comnonly observed in chronic industrial lead exposure). Clinically, a Fanconi like syndrome seen ... (proteinuria, hematuria, and casts in the urine; glycosuria and aminoaciduria). In some patients, hyperuricemia may be associated with renal insufficiency. Histologically, lead nephropathy is revealed by a characteristic nuclear inclusion body, composed of a lead protein complex; this appears early & resolves after chelation therapy. Such inclusion bodies have been reported in the urine of uorkers exposed to lead in an industrial setting. /Inorganic lead/ [Gilman, A.G., L.S.Goodman, and A. Gilman. (eds.). Goodman and Gilman's The Pharmacological Basis of Therapeutics. 7th ed. New York: Macmillan Publishing Co., Inc., 1985. , p. 16091 **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

Lead systemic poisoning can cause a wide variety of visual & ocular disturbances according to many case reports during the past 3.5 centuries. Much of what ... /is known/ of the clinical aspects of ocular effects comes from early literature which has been condensed in several reviews. ... Of 172 eye cases surveyed by Lewin & Guillery /& reported in 1913/, 79 had colic, 52 had headache, 47 had convulsions, delirium, stupor, or coma, & 27 had paralysis of extensor muscles. In general ocular disturbances appeared most commonly after many months or years of chronic poisoning, & at least after several weeks of poisoning. As a rule, ocular involvement was not among the early symptoms in adults, although it could be one of the first symptoms, esp in children. On the basis of clinical & exptl observations, it has been possible to recognize disturbances of the following sites: visual cortex & suprageniculate pathways; optic nerve, both retrobulbar & bulbar; retina; intraocular muscles; & extraocular muscles. Unfortunately many reports describing visual symptoms provide insufficient information for determination of site or mechanism. /Inorganic lead/ [Grant, U.M. Toxicology of the Eye. 3rd ed. Springfield, IL: Charles C. Thomas Publisher, 1986. , p. 550] **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

OTHER SIGNS & SYMPTOMS OF PLUMBISM ARE ASHEN COLOR OF FACE & PALLOR OF LIPS; RETINAL STIPPLING; APPEARANCE OF "PREMATURE AGING," WITH STOOPED POSTURE, POOR MUSCLE TONE, & EMACIATION; & BLACK OR GRAYISH SO CALLED LEAD LINE ALONG THE GINGIVAL MARGIN. ... THE CARCINOGENICITY OF LEAD IN MAN IS NOT WELL ESTABLISHED, BUT IT HAS BEEN SUGGESTED, & SEVERAL CASE REPORTS OF RENAL ADENOCARCINOMA IN LEAD WORKERS HAVE BEEN PUBLISHED. /INORGANIC LEAD/ [Gilman, A.G., L.S.Goodman, and A. Gilman. (eds.). Goodman and Gilman's The Pharmacological Basis of Therapeutics. 7th ed. New York: Macmillan Publishing Co., Inc., 1985. , p. 16091 **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

THERE IS NO EVIDENCE TO SUGGEST THAT EXPOSURE TO LEAD SALTS CAUSES CANCER OF ANY SITE IN MAN. HOWEVER, ONLY ONE EPIDEMIOLOGICAL STUDY OF THE RELATIONSHIPS BETWEEN EXPOSURE TO LEAD & THE OCCURRENCE OF CANCER HAS BEEN REPORTED. ... THE LEVEL OF HUMAN EXPOSURE EQUIVALENT TO THE LEVEL OF LEAD ACETATE PRODUCING RENAL TUMORS IN RATS IS 810 MG/DAY (550 MG PB). THIS LEVEL APPEARS TO EXCEED BY FAR THE MAX TOLERATED DOSE FOR MAN. /INORGANIC LEAD/ [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer,

1972-PRESENT. (Multivolune work). V1 48 (1972)] **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

A few clinical studies have found increased chromosomal defects in workers with blood lead levels above 60 ug/dl. /Inorganic lead/ [Doull, J., C.D.Klassen, and M.D. Amdur (eds.). Casarett and Doull's Toxicology. 3rd ed., New York: Macmillan Co., Inc., 1986. , p. 6031 **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

RESIDUAL NEUROLOGICAL SEQUELAE MAY AFFECT AS HIGH AS 82% OF SURVIVORS. /INORGANIC LEAD/ [PERLSTEIN; CLINICAL PED 5: 292 (1966)] **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

IN MANY CASES THERE IS NO MACROSCOPIC OR MICROSCOPIC EVIDENCE OF BRAIN EDEMA. EDEMA IS RARELY THE CAUSE OF DEATH. /INORGANIC LEAD/ [KRIGMAN E AL; EXP CLIN NEUROTOXICOLOGY, WILLIAMS & WILLIAMS (1980)] **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

MICROCYTOSIS & ANEMIA EVIDENTLY OCCUR MUCH LESS COMMONLY THAN PREVIOUSLY REPORTED IN CHILDHOOD LEAD POISONING UNCOMPLICATED BY OTHER HEMATOLOGIC DISORDERS. /INORGANIC LEAD/ CCOHEN AR ET AL; PEDIATRICS 67 (6): 904-6 (1981)] **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

... The incidence of premature fetal membrane rupture in term and preterm infants is much higher (17%) 30 to 50 miles west of a lead mining area of Missouri than in a Missouri urban area remote from lead mining activities (0.41%). Maternal and fetal blood lead levels (PbB) at birth also differed significantly for normal births vs births with premature membrane rupture. Maternal and fetal blood lead levels for the normal deliveries were about 14 and 4 ug/dl respectively, whereas they were about 26 and 13 ug per dl for mothers and infants with membrane rupture. /Inorganic lead/ [Fahim MS et al; Res Comun Chem Pathol Pharmacol 13: 309 (1976) as cited in USEPA; Ambient Water Quality Criteria Doc: Lead p.C-67 (1980) EPA 440/5-80-057] **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

... Significant levels of teratospermia occured among men in a lead storage battery factory (PbBs 30 to 80 ug/dl). /Inorganic lead/ [Lancranjan I et al; Arch Environ Health 30: 396-401 (1975) as cited in USEPA; Ambient Water Quality Criteria Doc: Lead p.C-67 (1980) EPA 440/5-80-057] **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

INCREASED SERUM ARGINASE ACTIVITY MAY INDICATE LIVER DAMAGE, UHILE DECR KALLIKREIN ACTIVITY MAY INDICATE KIDNEY DAMAGE IN WORKERS EXPOSED TO LEAD. /INORGANIC LEAD/ [CHMIELNICKA J ET AL; ER J IND MED 38 (2): 175-8 (1981)] **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

Blood lead values in children ranging from 40 to 80 ug/100 ml seem to be associated with adverse neuropsychological effects. Landrigan and coworkers ... compared a group of 70 children, aged 3 to 15 years (mean blood lead 48 ug/100 ml). They found that higher lead levels were associated with decreased intelligence and slowing in a finger wrist tapping test. /Inorganic lead/ [Landrigan PJ et al; Lancet 29: 708-12 (1975) as cited in NIOSH; Criteria Docunent: Inorganic Lead p.XI-56 (1978) DHEW Pub. NIOSH 78-1581 **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

The reproductive ability of men was ... shown to be adversely affected by moderate absorption of lead. Concn of lead in blood greater than 52 ug/100 ml were associated with a high frequency of altered spermatogenesis. Disorders of sexual dynamics were evident with blood lead values greater than 41 ug/100 ml. Among the workers with the highest concn of lead in blood (mean 74.50 + or - 26 ug/100 ml), 75% were judged to be hypofertile, 50% being even infertile. It was not possible, however, to demonstrate a reliable association between lead absorption in these men and the number of normal pregnancies per couple, or the frequency of miscarriages, ectopic pregnancies, or premature births. Nevertheless, these results were interpreted to indicate that lead clearly has a direct toxic action on the male gonads at relatively low Levels of absorption. /Inorganic Lead/ [Lancranjan I et al; Arch Environ Health 30: 396-401

(1975) as cited in NIOSH; Criteria Document: Inorganic Lead p.XI-52 (1978) DHEW Pub. NIOSH 78-158] **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

... WHEN AN EXCESSIVE AMT OF LEAD HAS BEEN DEPOSITED IN BONE, EITHER FROM INGESTION OR FROM INDUSTRIAL EXPOSURE, & HAS REMAINED IMMOBILIZED FOR YEARS, INTERCURRENT FACTORS CAUSING MOBILIZATION MAY CAUSE A SUDDEN OUTBREAK OF CLINICAL SYMPTOMS OF LEAD POISONING. /INORGANIC LEAD/ [Browning, E. Toxicity of Industrial Metals. 2nd ed. New York: Appleton-Century-Crofts, 1969. , p. 1741 **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

... In rural Scotland, four people developed clinical lead poisoning and others showed biochemical evidence of grossly elevated lead exposure. Lead concn in the domestic water supply was 2-3 mg/l. In this case the reason for the extreme contamination was that the water was stored in lead tanks. /Inorganic lead/ [Goldberg A; Environ Health Perspect Exp Issue 7: 103-7 (1974) as cited in WHO; Environ Health Criteria: Lead p.49 (1977)] **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

... Studies correlating elevated blood lead levels to hypertension are conflicting. Concern exists over possibility that soft acid water increases the solubility of lead & that elevated levels account for the increased incidence of hypertension & cardiovascular mortality in areas with soft-water supplies. The contribution of increased lead absorption to gout, hypertension, nephropathy, & neurotoxicity remains to be determined. /Inorganic lead/ [Ellenhorn, M.J. and D.G. Barceloux. Medical Toxicology - Diagnosis and Treatment of Human Poisoning. New York, NY: Elsevier Science Publishing Co., Inc. 1988. , p. 10301 **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

Although per day children ingest less lead (Pb) in their diets and inhale less Pb than do adults, on a dose per body weight basis children may have 2-3 times the exposure. Furthermore children because of their poor oral hygiene are more likely than adults to absorb Pb from extraneous sources: ingestion of foreign objects, dust, paint chips, inhalation of resuspended settled dusts. Preliminary data indicate that children absorb 50% of ingested Pb, ie at a rate 5 times greater than adults. Animal studies support the hypothesis that the young retain more of a Pb dose than do adults and that this is reflected in soft-tissue Pb levels. The portions of the Pb body burden found in soft tissues of children and adults are 27.5% and 5% respectively. Hence, a higher fraction of a child's body burden of Pb is available to produce toxic effects in soft tissues. /Inorganic lead/ [USEPA; Ambient Water Quality Criteria Doc: Lead p.C1-9 (1984) EPA 440/5-84-027] **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

... Zinc protoporphyrin (ZPP) concn was used as an index of blood lead concn. Analysis of zinc protoporphyrin consists of diluting whole blood (1:500), adding **dimethyldodecylamineoxide** and measuring the fluorescence at 594 nm (excitation at 424 nm). A correlation coefficient of 0.87 for zinc protoporphyrin and blood lead concn (by AAS) /was found/. Iron deficiency anemia will also reduce zinc protoporphyrin concn. /Inorganic lead/ [Lamola AA et al; Clin Chem 21 (1): 93-7 (1975) as cited in NIOSH; Criteria Document: Inorganic Lead p.XI-29 (1978) DHEW Pub. NIOSH 78-1581 **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

... EXPOSURE ... SHOULD BE TERMINATED ... WHEN CONCN /IN BLOOD/ ... IN EXCESS OF 0.08 MG/100 G ... OR OF URINE ... 0.18 MG/L ... OR WHEN CONCN ... IN BLOOD ... APPROACHING THRESHOLD OF DANGER, & ... CONCN IN URINE REMAINS ... IN LOWER THIRD OF NORMAL ... THUS GIVING EVIDENCE OF IMPAIRMENT OF RENAL FUNCTION ... /LEAD CMPD/ [Patty, F. (ed.). Industrial Hygiene and Toxicology: Volume II: Toxicology. 2nd ed. New York: Interscience Publishers, 1963. , p. 9771 **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

ON BASIS OF CLINICAL OBSERVATION ... DISTURBANCES /OCCUR IN/ VISUAL CORTEX & SUPRAGENICULATE PATHWAY; OPTIC NERVE ... RETROBULBAR & BULBAR; RETINA; INTRAOCULAR MUSCLE; LENS; & EXTRAOCULAR MUSCLE. INDIVIDUAL PORTION OF VISUAL SYSTEM MAY BE

AFFECTED ... MORE /OFTEN/ SEVERAL PORTIONS. ... /LEAD CMPDS/ [Grant, W. M. Toxicology of the Eye. 2nd ed. Springfield, Illinois: Charles C. Thomas, 1974. , p. 6211 **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

... Lead Levels were elevated in the liver of individuals having liver siderosis or other diseases involving altered iron metabolism .... Hemosiderin induced by high levels of iron, may act as a binding agent not only for iron but for Pb as well. Thus these diseases may incr Pb retention in the body and especially in the Liver. The Pb(+2) may later be released by being replaced with Fe(+3). /Inorganic lead/ [Butt EM et al; Metal binding in medicine p.43-9 (1960) as cited in Nat'l Research Council Canada; Effects of Lead in the Canadian Envir p.572 (1978) NRCC No.16736] **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

Lead containing particles in ambient air have an aerodynamic diameter of approx 0.1-1.0 um, & the predicted deposition in the airway is about 35%. This is questionable for smaller particles (< 0.1 um) which are mainly deposited by diffusion. Actual measurements of deposition in human volunteers gave results that differed considerably depending on the physical & chemical properties of the inhaled aerosol. ... A deposition of approx 25% /was observed/ after exposure to particles with a mass median aerodynamic diameter of 0.25 um. ... A deposition in the resp tract of about 60% /was observed/ in persons close to a motorway, where particles were about 0.03 um in diameter. This figure is consistent with lab expt carried out by the same authors, in which subjects inhaled radioactively labeled particles of about the same size. Uhen volunteers inhaled lead particles near urban roads where the particle size was larger (0.2-2.0 um), deposition was about 50%. Based on available data, it seems reasonable to conclude that the rate of deposition of airborne lead in the general population is approx 30-50%, depending on particle size & ventilation rates. /Inorganic lead/ [Friberg, L., Nordberg, G.F., Kessler, E. and Vouk, V.B. (eds). Handbook of the Toxicology of Metals. 2nd ed. Vols I, II.: Amsterdam: Elsevier Science Publishers B.V., 1986. V2 3111 **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

OCCUPATIONAL EXPOSURE TO CHROMIUM CMPD (CR6+) CAUSES DERMATITIS, PENETRATING ULCERS ON HANDS & FOREARMS, PERFORATION OF NASAL SEPTUM, & INFLAMMATION OF LARYNX & LIVER. [Doull, J., C.O. Klaassen, and M. D. Amdur (eds.). Casarett and Doull's Toxicology. 2nd ed. New York: Macmillan Publishing Co., 1980. , p. 4421 **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

RECENT EPIDEMIOLOGICAL EVIDENCE HAS BEEN INTERPRETED AS INDICATING THAT INHALATION OF LEAD CHROMATE DUST MAY LEAD TO AN INCR INCIDENCE OF LUNG CANCER. [American Conference of Governmental Industrial Hygienists. Documentation of the Threshold Limit Values for Substances in Uorkroom Air. Third Edition, 1971. Cincinnati, Ohio: American Conference of Governmental Industrial Hygienists, 1971. (Plus supplements to 1979) , p. 4501 **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

LEAD CHROMATE ... UNDOUBTEDLY LESS TOXIC THAN MORE SOL ACETATE, CHLORIDE & OXIDE ... CHRONIC INDUSTRIAL LEAD POISONING IS CAUSED AS READILY BY DUST OF ... METALLIC LEAD AS BY THESE SOL CMPD. [Browning, E. Toxicity of Industrial Metals. 2nd ed. New York: Appleton-Century-Crofts, 1969. , p. 1781 **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

LEAD CHROMATE IS LESS HAZARDOUS THAN ANY MEMBER OF THIS GROUP WHETHER GIVEN PARENTERALLY, ORALLY OR BY INHALATION. PERHAPS CHROMATE POISONING PLAYS ROLE IN ACUTE REACTION BUT SIGNS OF CHRONIC LEAD POISONING MAY APPEAR WK OR MO AFTER INITIAL INSULT. [Gosselin, R.E., H.C. Hodge, R.P. Smith, and M.N. Gleason. Clinical Toxicology of Comnercial Products. 4th ed. Baltimore: Williams and Wilkins, 1976. II-96] **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

THERE IS EXCESSIVE RISK OF LUNG CANCER AMONG WORKERS IN CHROMATE-PRODUCING INDUSTRY. IT IS LIKELY THAT EXPOSURE TO 1 OR MORE CHROMIUM CMPD IS RESPONSIBLE, BUT IDENTITY OF THIS OR

THESE IS NOT KNOWN. THERE IS NO EVIDENCE THAT NON-OCCUPATIONAL EXPOSURE TO CHROMIUM CONSTITUTES CANCER HAZARD. /CHROMIUM CMPD/ [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-PRESENT. (Multivolume work). V2 120 (1973)] **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

LEAD CHROMATE-BASED INKS ARE USED FOR PRINTING COLORED PAGES OF SOME CHILDREN'S MAGAZINES (COMICS) & TESTS HAVE SHOWN THAT HIGH LEVELS OF LEAD & CHROMIUM CAN BE EXTRACTED FROM COLORED MAGAZINE PAGES BY DIL (0.1 MOLAR) HYDROCHLORIC ACID @ BODY TEMP. [EATON DF ET AL; ENVIRON SCI TECHNOL 9 (8): 768-70 (1975)] **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

The strong oxidizing potential of Cr(VI) compounds explain much of their irritating and toxic properties. /Hexavalent chromium/ [USEPA; Health Assessment Document: Chromium p.2-7 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

Target organs: Blood, respiratory system, liver, kidneys, eyes, and skin. /Chromic acid and chromates/ [NIOSH. Pocket Guide to Chemical Hazards. 5th Printing/Revision. DHHS (NIOSH) Publ. No. 85-114. Washington, D.C.: U.S. Dept. of Health and Human Services, NIOSH/Supt. of Documents, GPO, Sept. 1985. , p. 831 **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

LEAD IS POISONOUS IN ALL FORMS. IT IS ONE OF THE MOST HAZARDOUS OF THE TOXIC METALS BECAUSE THE POISON IS CUMULATIVE & THE TOXIC EFFECTS ARE MANY & SEVERE. /LEAD CMPDS/ [Gosselin, R.E., R.P. Smith, H.C. Hodge. Clinical Toxicology of Commercial Products. 5th ed. Baltimore: Williams and Wilkins, 1984. 111-2261 **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

INHALATION OF HEXAVALENT CHROMIUM CMPD MAY CAUSE BRONCHIAL CARCINOMAS. ABILITY OF HEXAVALENT CHROMIUM CMPD TO INDUCE BRONCHOGENIC CANCER IN HUMANS IS WELL ESTABLISHED. [Friberg, L., G.R. Nordberg, and V.B. Vouk. Handbook on the Toxicology of Metals. New York: Elsevier North Holland, 1979. , p. 3831 **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

The strong oxidizing powers of Cr(VI) compounds explain much of their irritating and toxic properties. [USEPA; Health Assessment Document: Chromium p.2-7 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

Lung cancer mortality among workers at three English chromate pigment factories was investigated. Workers at factories 1 and 2 were exposed to both lead chromate and zinc chromate; those at factory 3 were exposed to lead chromate alone. A total of 152 workers made up the cohort of males completing at least 1 year of work by June 1975. Jobs were allocated to high, medium, and low exposure grades; total worker exposure was determined where possible. Observed deaths from respiratory cancers were compared to expected numbers of deaths taken from a national death rate schedule. Mortality from all causes except lung cancer showed a modest excess of deaths from nonmalignant respiratory diseases. For other respiratory cancers the ratio of observed to expected cases was 2.65. No lung cancer deaths had so far occurred among post 1967 work force entrants of any factory. At factory 1, among workers with high and medium exposure, the lung cancer mortality ratio was 2.22 among workers entering from 1932 to 1945 and 2.23 among 1946 to 1954 entrants. No excess was apparent among Later entrants. At factory 2 the lung cancer mortality ratio among high and medium exposure workers entering service from 1948 to 1967 was 3.73. Two deaths occurred among all low exposure workers, compared with 1.03 expected. For factory 3, there were no excess deaths from lung cancer for any exposure groups at any time of entry. Examination of mortality data from factories 1 and 2 in 5 year followup intervals showed high and medium exposure worker lung cancer mortality significantly increased during the first 9 years after entry, with the excess most pronounced 10 and 24 years after entry, and diminishing thereafter. When high and

medium exposure groups were examined separately results did not differ significantly. Occupational exposure to zinc chromate produces lung cancer among high and medium exposure workers after as little as 1 year, but no excess cancer was found in lead chromate workers. [Davies JM; Br J Ind Med 41 (2): 158-69 (1984)] **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

The history of working conditions and occupational health of female workers in China and improvements which have been made were presented. In 1954, the average air lead level in a privately owned lead plant was 1000 times the current standard, and all female employees were found to have lead poisoning. Lead poisoing was no longer found among employees after the plant merged into a government enterprise. Females were found to be more sensitive to lead poisoning than were men. Rates of threatened abortions were significantly higher among female workers exposed to lead chromate, and nursing babies of mothers with lead poisoning were diagnosed as having lead poisoning. [Wang Y-L, Zhaw X-H; Occupatinal Health Bulletin 1: 15-26 (1985)] **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

Arsenic, nickel, and chromium compounds that are human carcinogens were tested in a transformation assay using cultured primary human diploid foreskin cells. All nickel cmpd tested, lead chromate, potassium chromate, chromic oxide, disodium arsenate, sodium arsenous oxide and N-methyl-N'-nitro-nitrosoguanidine caused significant (p= 0.001) dose dependent inducticns of anchorage independent colonies in human diploid foreskin cells. ... Optimal expression times for induction of anchorage independence in human diploid foreskin cells were observed as early as 11 days following treatment with N-methyl-N'-nitro-N-nitrosoguanidine, trinickel disulfide, nickel acetate, or nickel sulfate. Cell strains derived from anchorage-independent colonies showed 33 to 429-fold higher plating efficiencies in soft agar than parental populations, and the anchorage independent phenotype was stable for eight passages, at which time cells senesced. Anchorage independent cell strains derived from metal salt treated cells were not resistant to the cytotoxicity of metal salts, indicating metal salts induced rather than selected for anchorage independence. Nine of 10 cell strains derived from metal compound or N-methyl-N'-nitro-N-nitrosoguanidine induced anchorage independent colonies displayed the same or lower saturation densities than untreated hman fibroblasts. None of these cell strains escaped senescence or showed definitive morphological transformation. [Biedermann KA, Landolph JR; Cancer Res 47 (14): 3815-23 (1987)] **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

Many carcinogenic chemicals are used in tanning processes in the tanneries of the 'leather area' of Tuscany near to the town of S Croce (Italy). Until recently increased risk of cancer has been demonstrated for tannery workers, but there has been a growing concern in S Croce on the health effects in leather tannery procedures. Proportional mortality data on a cohort of tannery workers showed an increased risk of lung cancer, esophageal cancer, sarcomas, and leukemias. From a cohort of workers and controls in the same area, data on urinary mutagenic activity using Salmonella typhimurium TA1538 and TAIOO as tester strains with and without metabolic activation were reported. Only non-smokers were included in the study *to* avoid interference from known urinary mutagens. The mutagenic activity of the urine of tanning workers did not differ from that of clerical workers. Data were also obtained on bladder desquamative cytology, which did not show any difference between clerical controls and workers. Then data were obtained on pulmonary cytology, analysed and classified under different headings. The specimens with normal cytology (class 1), with cellular hyperplasia and normal metaplasia (class 2.1) were regarded as normal. Specimens with mild cellular atypical metaplasia (class 2.2), moderate cellular atypical metaplasia (class 2.3) were regarded as pathological. No cases with atypical cells (class 3) or obvious neoplastic changes (class 4) were found. In the group of workers, a higher relative risk

of developing pathological desquamative cytology in the lung /was noted/ ... in chemical refining workers but not in tannery workers. [Constantini AS et al; Ann Occup Hyg 31 (1): 21-30 (1987)] **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

SYMPTOMATOLOGY: A. Acute poisoning by ingestion only. 1) An astringent and metallic taste in the mouth, dry throat, thirst. 2) Burning abdominal pain, nausea, and vomiting. The vomitus may appear milky due to the presence of lead chloride. The abdominal pain may become colicky and severe. 3) Sometimes diarrhea, less often constipation. The stools may be bloody, or black due to the presence of lead sulfide. 4) Peripheral circulatory collapse. 5) Neuromuscular symptoms include muscular weakness, pain, and cramps, especially in the legs. 6) Central nervous system manifestations include headache, insomnia, paresthesias, depression, coma, and death. 7) Though usually of secondary concern, kidney damage may result in oliguria, albuminuria, and cylindruria. The renal lesion may be due to the mildly nephrotoxic action of lead, to disturbances in kidney circulation, or to the products of intravascular hemolysis. Renal lesions may assume increased importance if edetate calcium disodium therapy is instituted. 8) An acute hemolytic crisis sometimes develops and results in anemia and hemoglobinuria. 9) Death may occur within 1 or 2 days, but recovery is the rule. Convalescence is slow and may be interrupted by episodes like those seen in typical chronic poisoning. /Lead cmpd/ [Gosselin, R.E., R.P. Smith, H.C. Hodge. Clinical Toxicology of Commercial Products. 5th *ed.* Baltimore: Williams and Wilkins, 1984. 111-2321 **PEER REVIEWED**

HUMAN TOXICITY EXCERPTS

SYMPTOMATOLOGY: B. Lead encephalopathy in chronic lead poisoning. 1) Headache and insomnia. 2) Persistent vomiting, which is sometimes projectile. A typical lead colic may or may not be present. 3) Visual disturbances, choked optic disks. 4) Irritability, restlessness, delirium, hallucinations. 5) Convulsions and coma. 6) The intracranial pressure is characteristically high. The cerebrospinal fluid is generally unremarkable except for and elevation of total protein. 7) Death from exhaustion and respiratory failure. The mortality rate is high; Recovery is slow and frequently incomplete. /Lead cmpd/ [Gosselin, R.E., R.P. Smith, H.C. Hodge. Clinical Toxicology of Commercial Products. 5th ed. Baltimore: Williams and Wilkins, 1984. 111-2331 **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

OF THE CHROMIUM SALTS TESTED IN THE RAT BY IM & IP ADMIN, CHROMIC CHROMATE & ZINC CHROMATE HYDROXIDE WERE HIGHLY EVOCATIVE OF SARCOMAS @ SITE OF INJECTION IN RAT, WHILE ... LEAD CHROMATE ... /WAS/ INACTIVE OR PRACTICALLY INACTIVE. [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-PRESENT. (Multivolume work). V2 119 (1973)] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

... EXPERIMENTS /WITH RATS/ LASTING 27 MO GAVE FOLLOWING NUMBER OF TUMORS @ SITE OF INTRAPLEURAL INJECTION (DETAILS OF DOSE & TUMOR TYPE NOT GIVEN): ... LEAD CHROMATE, 3/34 (32 ALIVE @ 1 YR) ... NONE OF THE CONTROL RATS SHOWED TUMORS (HUEPER, 1961). [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-PRESENT. (Multivolume work). V2 115 (1973)] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

/INVESTIGATORS/ ... TESTED ... /SEVERAL/ COMPOUNDS BY IM IMPLANTATION & FOUND THE FOLLOWING INCIDENCE OF IMPLANTATION-SITE SARCOMAS AFTER 27 MO IN GROUPS OF 22-34 RATS ... ... LEAD CHROMATE, 1/33 (28 ALIVE @ 1 YR) ... NONE OF THE 35 CONTROL RATS GIVEN IMPLANTS OF SHEEP FAT ALONE DEVELOPED LOCAL TUMORS. [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-PRESENT. (Multivolume work). V2 114 (1973)] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

NO LUNG TUMORS WERE FOUND ... IN 8 RABBITS EXPOSED ON 4 DAYS/WK FOR UP TO 50 MO TO A MIXED CHROMATE DUST BY INHALATION OR IN 10 RABBITS EXPOSED TO SIMILAR MATERIAL BY INTRATRACHEAL INJECTION. SIMILAR STUDIES WITH ... LEAD CHROMATE IN 7 RABBITS ALSO GAVE NEGATIVE RESULTS. CIARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-PRESENT. (Multivolune work). V2 111 (1973)] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

... 13 ... /GUINEA PIGS/ WERE GIVEN 6 ONCE-MONTHLY INTRATRACHEAL INSTILLATIONS OF LEAD CHROMATE. ALL ANIMALS WERE OBSERVED UNTIL THEY DIED ... NONE DEVELOPED CARCINOMAS OF LUNG (STEFFEE & BAETKER. 1965). CIARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-PRESENT. (Multivolune work). V2 111 (1973)] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

THE POTENTIAL MUTAGENICITY OF LEAD CHROMATE WAS TESTED BY FOLLOWING BATERRY OF MICROBIAL TESTS: THE ESCHERICHIA COLI POLA+/POLA- SURVIVAL TEST; THE SALMONELLA/MICROSOME HIS+ REVERSION ASSAY; THE E COLI TRP+ REVERSION TEST AS A PLATE ASSAY; THE E COLI GAL+ FORWARD MUTATION TEST & THE SACCHAROMYCES CEREVISIAE ASSAY FOR MITOTIC RECOMBINATION. LEAD CHROMATE WAS MUTAGENIC IN SALMONELLA & IN SACCHAROMYCES. METABOLIC ACTIVATION BY RAT LIVER HOMOGENATE (S9) WAS NOT REQUIRED. APPARENTLY, CHROMATE ION IS RESPONSIBLE FOR MUTAGENICITY OF LEAD CHROMATE. [NESTMANN ER ET AL; MUTAT RES 66 (4): 357-65 (1979)] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

Several hexavalent chromium compounds have been shown to be carcinogenic in cancer bioassay studies. Only calcium chromate has consistently produced lung tumors in rats by several routes of administration. Other chromium compounds: strontium chromate, zinc chromate, sodium dichromate, lead chromate, lead chromate oxide, and sintered chromium trioxide, have produced sarcomas or lung tumors in rats at the site of intrabronchial, intratracheal, intramuscular, subcutaneous and intraperitoneal application. Hexavalent chromium compounds have not induced lung tumors by inhalation; however, studies have not been reported in detail. ... Animal cancer bioassay studies suggest that hexavalent chromium compounds (particularly soluble and sparingly soluble compounds) are probably the etiologic agent in chromium related human cancer. Under the IARC criteria, the animal bioassay studies would constitute sufficient evidence of the carcinogenicity of hexavalent chromium compounds. /Hexavalent chromium/ [USEPA; Health Assessment Document: Chromium p.2-10 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

In general, soluble Cr(VI) compounds are less active in the presence of metabolic activating systems. The reduction of Cr(VI) to Cr(III) by cellular agents in metabolic activation systems, in part, explains the reduced mutagenic activity of Cr(VI) in the presence of such activating systems. Some recent evidence implicating both Cr(VI) and Cr(III) in induced mutagenesis has been reported in DNA interaction and DNA polymerase infidelity assays, and several tests with apparently pure Cr(III) samples have produced chromosomal aberration. /Hexavalent chromium/ CUSEPA; Health Assessment Document: Chromium p.2-9 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

Limited work has been reported on the reproductive effects of chromium. Cr(VI) and Cr(III) have been found to cross the placental barrier in animals (hamsters and mice) and enter the fetus during mid to late gestation. Fetal uptake of Cr(VI), however, was much greater than that of Cr(III). Developmental effects attributed to both Cr(VI) and Cr(III) differed between hamsters and mice, and included such external abnormalities as cleft palate and skeletal defects, and (in one study of a Cr(III) compound) neural tube defects. One researcher concluded that Cr(VI) was present at sufficiently high fetal concentrations to cause direct effects on embryonic structures,

but also questioned whether all of the teratogenicity and fetal toxicity associated with exposure to Cr(III) might be attributed to extra-embryonic effects, for example, those on placental tissues. /Trivalent and hexavalent chromium/ [USEPA; Health Assessment Document: Chromium p.2-8 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

The main toxic action of chromium takes place in the roots of soybeans. More than 0.1 ppm Cr(6+) in a solution culture interfered with the uptake and translocation of essential elements by the roots of soybeans. In solution culture, 1-5 ppm of either Cr(3+) or Cr(6+) was the toxic threshold for a number of plant species. When compost was used for the growth medium, the toxic threshold was increased to 500 ppm for Cr(6+) and 5000 ppm for Cr(3+). In contrast, the use of sand as the growth medium did not change the toxic thresholds much from those for the solution cultures. This increase in the toxic threshold was related to the greater ion exchange capacity of compost over that of sand. Thus, the greatest risk of chromium toxicity to plants is in acidic, sandy soil having low organic content. /Chromium/ [Nat'l Research Council Canada; Executive Reports: Effects of Chromium, Alkali Halides, Arsenic, Asbestos, Mercury, Cadmium in the Canadian Evironment p.13 (1980) NRCC No. 175851 **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

The dietary toxic threshold for Cr(6+) in experimental animals is higher than in man. In dogs, fatal exposure was 0.1 g Cr(6+)/day in food for 2-3 months; 11.2 ppm in drinking water for 4 years caused significant tissue accunulation of chromium, but no obvious pathological effects; 6 ppm was the threshold for accumulation in the tissues. In rats, 134 ppm Cr(6+) in drinking water for 2-3 months caused kidney and liver lesions, while the toxic threshold for Cr(6+) in the food of young rats was 0.1% (1000 ppm). Hence Cr(6+), when blended with food, is much less available than Cr(6+) in drinking water. /Hexavalent chromium/ [Nat'l Research Council Canada; Executive Reports: Effects of Chromium, Alkali Halides, Arsenic, Asbestos, Mercury, Cadmium in the Canadian Environment p.14 (1980) NRCC No. 175851 **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

The acute toxicity of chromium(VI) to 23 saltwater vertebrate and invertebrate species ranged from 2,000 ug/l for a polychaete worm and a mysid shrimp to 105,000 ug/l for a mud snail. The chronic values for a polychaete ranged from < 13 to 36.74 ug/l, whereas that for a mysid shrimp was 132 ug/l. The acute-chronic ratios ranged from 15.38 to > 238.5 ug/l. Toxicity to macroalgae was reported at 1,000 and 5,000 ug/l. /Hexavalent chromium/ [USEPA; Ambient Uater Quality Criteria Doc: Chromium p.18 (1984) EPA 440/5-84-029] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

/The authors/ compared the toxicities of dichromate and chromate to the bluegill in soft water. The 96-hr LC50s were 110,000 ug/l for dichromate and 170,000 ug/l for chromate. They concluded that acidic dichromate was more toxic than basic chromate, because the greatest part of the chromium(VI) was *in* the form of the hydrochromate ion at the lower pH of the dichromate solutions, whereas at the higher pH of the chromate solutions, most of the chromium(VI) was in the form of the chromate ion. /Hexavalent chromium/ [Trama FB, Benoit RJ; Uater Pollut Control Fed 32: *868* (1960) as cited in USEPA; Ambient Uater Quality Criteria Doc: Chromium p.6 (1984) EPA 440/5-84-029] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

The toxicity of chromium(VI) apparently increases as pH is Lowered or as hardness is lowered or both. Although there are exceptions, softer surface waters usually have a lower pH than harder surface waters. However, the available data are insufficient to develop criteria on the basis of water quality characteristics. /Hexavalent chromium/ [USEPA; Ambient Uater Quality Criteria Doc: Chromium p.7 (1984) EPA 440/5-84-029] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

/Several authors/ reported that the toxicity of chromium(VI) to

fish, shrimp, crab, and clam increased as salinity decreased (Tables 1 and 6). The change was usually less than a factor of two, except when salinity was about 1 g/kg. /Hexavalent chromium/ IUSEPA; Ambient Water Quality Criteria Doc: Chromium p.7 (1984) EPA 440/5-84-029] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

Selected ranked Species Mean Acute Values for saltwater species are as follows: Nassarius obsoletus (mud snail) 105,000 ug/l, Fundulus heteroclitus (mummichog) 74,010 ug/l, Menidia menidia (Atlantic silverside) 15,280 ug/l, Crassostrea gigas (Pacific oyster) 4,538 ug/l, Mytilus edulis (blue mussel) 4,469 ug/l, Mysidopsis bahia (mysid shrimp) 2,030 ug/l, and Nereis virens (polychaete worm) 2,000 ug/l. The saltwater Final Acute Value of 2,158 ug/l for Cr(VI) was calculated from the Genus Mean Acute Values. /Hexavalent chromium/ IUSEPA; Ambient Water Quality Criteria Doc: Chromium p.47-49 (1984) EPA 440/5-84-029] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

Selected ranked Species Mean Acute Values for freshwater species are as follows: Neophasganophora capitata (stonefly) 1,870,000 ug/l, Lepomis macrochirus (bluegill) 132,900 ug/l, Carassius auratus (goldfish) 119,500 ug/l, Salmo gairdneri (rainbow trout) 69,000 ug/l, Pimephales promelas (fathead minnow) 41,050 ug/l, Poecilia reticulata (guppy) 30,000 ug/l, and Daphnia magna (cladoceran) 23.07 ug/l. A freshwater Final Acute Value of 31.49 ug/l was calculated from the Genus Mean Acute Values, using the procedure described in the guidelines. /Hexavalent chromium/ IUSEPA; Ambient Water Quality Criteria Doc: Chromium p.7, 43-5 (1984) EPA 440/5-84-029] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

A single sc injection of 30 mg lead chromate(VI) or lead chromate(VI) oxide in water to groups of 40 Sprague-Dauley rats gave rise to 26/40 & 27/40 sarcomas, respectively, at site of injection within 117-150 wk. No Local sarcoma occurred in 60 vehicle-treated control rats ... (Maltoni, 1974, 1976). [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-PRESENT. (Multivolume work). V23 258 (1980)] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

Groups of 25 male & 25 female Fischer-344 rats received monthly im injections of 8 mg lead chromate(VI) suspended in trioctanoin for 9 mo or 4 mg calcium chromate(VI) suspended in trioctanoin for 12 mo. Calcium chromate produced 3 fibrosarcomas & 2 rhabdomyosarcomas at injection site in 5/45 rats, whereas lead chromate induced 14 fibrosarcomas & 17 rhabdomysarcomas at the site of injection in 31/47 rats. In addition, 3/24 lead chromate-treated rats had renal carcinomas. No such tumors appeared in a similar group of 22 controls injected with the vehicle (Furst et al, 1976). [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-PRESENT. (Multivolume work). V23 258 (1980)] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

Lead poisoning can result when curious animals ingest lead based paints (either old dry paint or paint from "empty" paint cans), glazier's putty or other caulking materials, used crankcase oil, greases, linoleum, leaded gasoline ... solder, roofing materials, asphalt, or industrial effluents in streams or on forage. Grass near busy highways may contain toxic amounts of lead from auto exhausts. Licking of discarded storage batteries can also result in lead poisoning. Water from lead plumbing or glazed crockery pots may contain toxic amounts. ... Natural sources of lead such as galena or soils are not particularly toxic but can add to the total body burden. Milk secreted from lead poisoned animals can be dangerous for the young animal. Paint seems to be a comnon source of lead in poisoned animals. In addn, cattle & calves are often poisoned by ingesting crankcase oil drained from internal combustion engines. The farm dump is often a smorgasbord of poisons for animals that gain access to it.

/Inorganic lead/ [Booth, N.H., L.E. McDonald (eds.). Veterinary Pharmacology and Therapeutics. 5th ed. Ames, Iowa: Iowa State University Press, 1982. , p. 10051 **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

The acute oral lethal single dose of lead in various species is: calves, 50-600 mg/kg as lead or lead salts; cattle ... 600-800 mg/kg from lead salts ... sheep & goats, 600-800 mg Pb/kg from lead salts ... fowl, 160-600 mg/kg absorbed from lead salts. Chronic toxicosis can arise when lead is ingested over a period of days, weeks, or months. The chronic oral lethal dose in different species is: calves, 1-3 g total/day; cattle ... may not get ill with 6 mg/kg/day for 60 weeks from lead paint. ... Prolonged ingestion of small amounts of lead can eventually cause mild to severe illness in animals, although the definition of "small amounts" may vary with species. ... The no effect level of lead intake for sheep is about 0.1 mg/kg/day; biochemical aberrations occur in this species when the intake is 0.3 to 3 mg/kg/day. In mammals (rats), the no effect level for prenatal exposure is about 1 ppm in the diet. Some pet foods contain more lead than this. ... Cats are not poisoned by lead very often because they do not chew on foreign objects, lick painted surfaces, or eat materials that are not foodstuffs. Cats can be poisoned if their food or water is contaminated or if they lick lead containing matter such as grease or oil from their fur. /Inorganic lead/ [Booth, N.H., L.E. McDonald (eds.). Veterinary Pharmacology and Therapeutics. 5th ed. Ames, Iowa: Iowa State University Press, 1982. , p. 1005] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

Forage samples normally contain 3-7 ppm lead or Less. Lambs have safely eaten forage containing lead at 45-60 ppm, but no studies have been made of possible subtle effects. ... Silage containing 140 ppm lead has poisoned cattle, & herbage containing lead at 216-914 ppm has killed calves. /Inorganic lead/ [Booth, N.H., L.E. McDonald (eds.). Veterinary Pharmacology and Therapeutics. 5th ed. Ames, Iowa: Iowa State University Press, 1982. , p. 10111 **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

Onset of clinical signs of lead poisoning /in cattle/ may take a few hours, days, or weeks, depending on the amount ingested, species, & other factors. ... Chronic poisoning may take weeks or months to develop. Clinical signs manifested by different species do have some differences, but the overall impression is of an encephalopathy preceded & accompanied by GI malfunction. ... When signs are classified /in the literature/, the classification looks very much like that of chlorinated hydrocarbon insecticides & urea which also produce encephalopathy and GI signs. /Inorganic lead/ [Booth, N.H., L.E. McDonald (eds.). Veterinary Pharmacology and Therapeutics. 5th ed. Ames, Iowa: Iowa State University Press, 1982. , p. 10081 **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

Behavorial aberrations are manifested, beginning with apparent anxiety or apprehension & proceeding to such things as hyperexcitability, bellowing or other vocalization, rolling of eyes & apparent fear or terror, possible belligerence, pressing of the head against a wall or post, attempts to climb the wall, sudden jumping into the air, & frenzied or manical behavior. One fascinating aspect of this category is the effect of small amounts of lead in exptl animals. Lead can disrupt conditioned (learned) behavior in adult rats, rabbits & sheep. It can also disrupt learning & memory in young & adult rats & in lambs born of lead treated ewes. /Inorganic lead/ [Booth, N.H., L.E. McDonald (eds.). Veterinary Pharmacology and Therapeutics. 5th ed. Ames, Iowa: Iowa State University Press, 1982. , p. 1008] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

Nervous phenomena such as depression (particularly in sheep & horses, or in chronic poisoning), muscle fasciculations, twitching of ears or muzzle, excessive blinking, muscle spasms, rhythmic jerking of the head & neck, jaw champing, pharyngeal paralysis, laryngeal hemiplegia (causing roaring in horses), tongue lolling, nystagmus, blindness (causing the animal to

stumble & blunder into objects), opisthotonos, torticollis, clonic-tonic convulsions, normal body temp in most cases but occasionally a temp of *42-44* deg C, coma, & either a quiet or convulsive death are also manifested. /Inorganic lead/ [Booth, N.H., L.E. McDonald **(eds.).** Veterinary Pharmacology and Therapeutics. 5th ed. Ames, Iowa: Iowa State University Press, 1982. , p. 10081 **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

Some autonomic manifestations of lead poisoning are salivation (or loss of saliva from inability to swallow), lacrimation, possible sweating, colic (tucked abdomen, tooth gnashing, groaning), thirst, diarrhea or constipation, rumen atony, urinary incontinence, & possibly vomiting (even in cattle). /Inorganic lead/ [Booth, N.H., L.E. McDonald **(eds.).** Veterinary Pharmacology and Therapeutics. 5th ed. Ames, Iowa: Iowa State University Press, 1982. , p. 10081 **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

The locomotor disturbances of lead poisoning range from a stiff, stilted gait with ataxia & incoordination through rigidity of all postural muscles, swaying, & posterior weakness to compulsive hypermotility (circling, pacing, running). In mice & rats, offspring of animals fed low levels of lead developed hypermotility & stereotyped movements. /Inorganic lead/ [Booth, N.H., L.E. McDonald **(eds.).** Veterinary Pharmacology and Therapeutics. 5th ed. Ames, Iowa: Iowa State University Press, 1982. , p. 10081 **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

Some dependent signs, obviousiy secondary to other effects of lead, include rapid labored breathing, anorexia, wt loss, decreased milk production, dehydration, emaciation, fetal death with either resorption or abortion of the fetus, & general weakness. The veterinarian does not necessarily see all these signs. The typical calf or cow has diarrhea & is anorexic, dull or excited, colicky, possibly trembling or blind or pressing the head, bawling or bellowing, & beginning to manifest hypermotility. More severe signs may occur later if the condition is not treated. Sometimes animals ingest so much lead that no signs are observed; they may be found dead. /Inorganic lead/ [Booth, N.H., L.E. McDonald **(eds.).** Veterinary Pharmacology and Therapeutics. 5th ed. Ames, Iowa: Iowa State University Press, 1982. , p. 10081 **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

In acute poisoning of calves, cattle, or sheep (farm species most often poisoned by lead) there may be no lesions. Alternatively, there may be marked gastroenteritis & severe kidney damage (sheep) or only pale liver & hyperemic kidneys (cattle). The stomach may contain paint, paint flakes, black colored motor oil, or other foreign materials. Microscopically, the liver of calves & cattle may manifest centrilobular necrosis. Hepatocytes & renal tubular epithelium may contain eosinophilic, acid fast intranuclear inclusions. In the brain, histologic changes ranging from diffuse capillary activation to scattered foci of status spongiosus & necrosis of the cerebral cortex may occur. /Inorganic lead/ [Booth, N.H., L.E. McDonald **(eds.).** Veterinary Pharmacology and Therapeutics. 5th ed. Ames, Iowa: Iowa State University Press, 1982. , p. 10081 **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

In subacute poisoning of calves & cattle, few lesions may be manifested. However, there may be pale muscles, liver, & kidneys & pronounced rigor if the animal had a high body temp or convulsions prior to death. Transudational lesions such as fluid in the body cavities or pericardium, excess CSF, lung edema, & cerebral edema may also be seen. Vascular failure may be further manifested by hemolysis ... Degenerative lesions such as softening of the kidney cortex or cerebral softening & cavitation might be seen. Microscopically, the lesions would be consistent with the gross ones. There may be acid-fast eosinophilic intranuclear inclusions in proximal tubular renal epithelium; necrosis of renal tubules; & cerebral status spongiosus with astrocyte swelling, nerve cell degeneration, & vascular proliferation. The brain histopathology may not be readily differentiated from bovine polioencephalomalacia.

/Inorganic lead/ [Booth, N.H., L.E. McDonald (eds.). Veterinary Pharmacology and Therapeutics. 5th ed. Ames, Iowa: Iowa State University Press, 1982. , p. 10091 **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

In calves & cattle chronically poisoned over 2 or more weeks, lesions may be quite minimal. The carcass may ... be emaciated & lacking in fat. Otherwise, lesions of subacute toxicosis may be seen, possibly in a more advanced state; ie, hemolysis & hemorrhagic lesions may be more widespread & include hemorrhagic gastroenteritis (very comnon in abomasum & small gut) & meningitis. The kidneys may manifest fibrotic changes & the brain may be more obviously softened, yellow, & necrotic, particularly at the tips of cerebral gyri. Microscopically, there may be blood loss anemia with basophilic stippling of erythrocytes & juvenile forms of erythrocytes. More obvious renal & cerebral degenerative changes ... /similar to those seen in subacute toxicosis/ may occur. There may also be an increased number of inclusions in renal tubular epithelium. Neither basophilic stippling of erythrocytes nor acid fast eosinophilic inclusions in kidney or liver cells is pathognomonic of lead poisoning, but these microscopic lesions are highly suggestive of it. /Inorganic lead/ [Booth, N.H., L.E. McDonald (eds.). Veterinary Pharmacology and Therapeutics. 5th ed. Ames, Iowa: Iowa State University Press, 1982. , p. 10091 **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

In dogs, acute lead poisoning (eg, from ingestion of paint) may not cause gross lesions. Paint may be found in the stomach or intestines. If dogs have been manifesting clinical signs of poisoning for several days or weeks, a number of gross microscopic lesions might be found. ... The gross lesions consist of meningeal congestion; very red bone marrow; radiopacity of long bone metaphyses in young animals ("lead lines"); delayed closure of thoracic vertebral epiphyses; & possibly an enlarged fatty liver, enlarged spleen, & swollen brain. /Inorganic lead/ [Booth, N.H., L.E. McDonald (eds.). Veterinary Pharmacology and Therapeutics. 5th ed. Ames, Iowa: Iowa State University Press, 1982. , p. 10091 **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

In brain of dog, microscopic Lesions may include cerebral vasodilation (congestion) with swelling & necrosis of vascular endothelium, capillary proliferation in cortical gray matter, gliosis, vacuolation, hyalinization & necrosis of some arterioles, thrombi in some capillaries, perivascular edema, fibrin deposition, hemorrhage, & necrosis of cortical neurons. EEG abnormalities probably reflect these pathologic changes, although they could precede lesions that are visible by light microscopy. It is assumed that in many tissues cellular ultrastructural lesions precede microscopic & gross lesions. /Inorganic lead/ [Booth, N.H., L.E. McDonald (eds.). Veterinary Pharmacology and Therapeutics. 5th ed. Ames, Iowa: Iowa State University Press, 1982. , p. 10091 **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

12 male and 12 female beagle dogs (one year old) were fed a basic ground meal diet consisting of 100 ppm lead for 46 weeks, or 500 ppm for 30 weeks, followed by 1000 ppm for 16 weeks. Increased blood and urine levels, increased urinary delta-aminolevulinic acid (ALA) excretion and decreased erythrocyte ALA-dehydratase activity were observed in all groups. No effect on blood regeneration (red cell count, hemoglobin, hematocrit ratio) was noted following replacement of one-half of the blood volume. /Inorganic lead/ [Maxfield ME et al; Am Ind Hyg Assoc J 33 (5): 326-7 (1972) as cited in NIOSH; Criteria Document: Inorganic Lead p.XI-60 (1978) DHEU Pub. NIOSH 78-1581 **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

The effects of acute lead poisoning on rat kidney included formation of intranuclear inclusion bodies, mitochondrial swelling with impairment of oxidative and phosphorylative processes, and aminoaciduria (apart from the long recognized delta-aminolevulinic aciduria). The intranuclear inclusion bodies were a lead protein complex that may have adaptive function in excessive lead exposure. Rats developed

hyperuricemia and in chronic lead poisoning, renal adenocarcinoma. In all, but the last, the findings in rats, paralleled those seen in man. /Inorganic lead/ [Goyer RA et al; Current Topics in Pathology 55: 147-76 (1971) as cited in NIOSH; Criteria Document: Inorganic Lead p.III-14 (1978) DHEW Pub. NIOSH 78-158] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

Chronic exposure through ingestion of 6 to 7 milligrams of lead per kg of body weight, can result in poisoning in cattle. /Inorganic lead/ [USEPA; The Health and Environmental Impacts of Lead: p.152 (1979) EPA 560/2-79-001] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

Paraplegia was reported in suckling rats. The disease was produced by transfer of lead from the mothers milk until weaning, with subsequent post weaning feeding of lead to the young. /Inorganic lead/ [Pentschew A, Garro F; Acta Neuropathol 6: 266-78 (1966) as cited in WHO; Environ Health Criteria: Lead p.89 (1977)] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

Hypertension can occur with heavy exposure to lead /in rats, rabbits, or dogs/. /Inorganic lead/ [WHO; Environ Health Criteria: Lead p.92 (1977)] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

Alveolar macrophages from guinea pigs are damaged in vitro by inorganic lead cmpd (3 ug/lx10+6 cells) ... more than 90% of the cells /were/ damaged within 20 hr. /Inorganic lead/ [Beck EG et al; Proceedings of the International Symposium: Environ Health Aspects of Lead p.451-61 (1973)] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

The effects of various centrally active drugs were studied in lead (6.25 mg lead/kg per day, orally) and control treated pigeons responding under a multiple fixed ratio 30 response, fixed interval 5 min schedule of food presentation. ... Chronic low-Pb exposure in adult pigeons produced functional alterations in central catecholaminergic but not cholinergic, GABAergic, glycinergic, or serotonergic neuronal systems as revealed by schedule controlled responding. /Inorganic lead/ [Carter RB, Leander JD; Neurobeh Toxicol 2 (4): 345-54 (1981)] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

Rana pipens @ 25 ppm lead exhibited a permanent loss of normal semi-erect posture. After 20 days of exposure to the same concn, there was a marked sloughing of the skin, excretion of bile, hypertrophy of the liver, spleen, and stomach. Pb deposits were present in the liver. Incidence of mortality was significantly increased over controls when Pb concn exceeded 25 ppm. /Inorganic lead/ [Kaplan HM; Lab Anim Care 17: 140 (1967) as cited in Nat'l Research Council Canada; Effects of Lead in the Canadian Envir p.245 (1978) NRCC No.0316-01143 **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

Levels of lead above the allowable drinking water standard of 50 ug/l are toxic, or result in morphological changes in aquatic vertebrates. ... Embryo and fingerling stages of fish are more susceptible to lead poisoning than adults. ... Fish are more susceptible to poisoning in soft water than in hard water. /Inorganic lead/ [USEPA; The Health and Environmental Impacts of Lead: p.152 (1979) EPA 560/2-79-001] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

... Animal studies indicate that relatively high levels of lead exposure interfere with resistance to infectious disease. /Inorganic lead/ [Gainer JH; Environ Health Perspect Exp 7: 113-9 (1974) as cited in USEPA; Ambient Water Quality Criteria Doc: Lead p.C-71 (1980) EPA 440/5-80-057] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

Lead is not considered to be an essential element for plant growth and development. Lead inhibits growth, reduces photosynthesis (by inhibiting enzymes unique to photosynthesis), interferes with cell division and respiration, reduces water absorption and transpiration, accelerates abscission or defoliation and pigmentation, and reduces chlorophyll and ATP synthesis. /Lead cmpds/ [USEPA; The Health

and Environmental Impacts of Lead: p.156 (1979) EPA 560/2-79-001] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

... 25 micromoles of lead applied to spinach leaves induced a pronounced inhibition of gas turnover with a greater inhibition of CO2 uptake in the light period than C02 release in the dark period. The intensity of (14)CO2 fixation by isolated spinach chloroplasts and ATP synthesis was reduced by lead quantities of 2 to 200 micromoles. /Inorganic lead/ [Holl A, Hamp R; Residue Reviews 54: 79-111 (1975) as cited in USEPA; The Health and Environmental Impacts of Lead p.157 (1979) EPA 560/2-79-001] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

Freshwater algae are affected by concentrations of lead above 500 ug/l based on data for four species. /Inorganic lead/ [USEPA; Ambient Water Quality Criteria Doc: Lead p.1-9 (1984) EPA 440/5-84-027] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

... PERIPHERAL NEUROPATHY ... ANATOMIC LESION IS CHARACTERIZED BY SEGMENTAL DEMYELINATION & BY AXONAL DEGENERATION. FUNCTIONALLY, NERVE CONDUCTION VELOCITY IS SLOUED, EVEN IN THE ABSENCE OF PALSY ... /HOWEVER, THERE IS GREAT SPECIES SUSCEPTIRILITY; RATS & GUINEA PIGS MAINLY SEGMENTAL DEMYELINATION; RABBITS SHOW WALLERIAN DEGENERATION; PRIMATES ARE INCONSISTENT/. /Inorganic lead/ [Doull, J., C.D. Klaassen, and M. D. Amdur (eds.). Casarett and Doull's Toxicology. 2nd ed. New York: Macmillan Publishing Co., 1980. , p. 4181 **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

ORAL ADMIN OF LEAD TO RATS INHIBITED ACTIVITY OF DELTA-AMINOLEVULINIC ACID DEHYDRATASE (ALAD). EDTA TREATMENT OF RATS GIVEN LEAD CAUSED REACTIVATION OF LIVER ALAD. /Inorganic lead/ [HAMMOND PB; TOXICOL APPL PHARMACOL 26 (3): 466 (1973)] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

V79 Chinese hamster cell line was used to detect the induction by nitrilotriacetic acid of 6-thioguanine resistance, due to mutation at the HGPRT locus, with direct and indirect mutagens as positive controls. Nitrilotriacetic acid was tested within the 10.4-1.5 above the 10-2 M dose, it did not increase the frequency of mutations at any of the tested concentrations, independently of metabolic activation (rat liver S9 fraction). Nitrilotriacetic acid is known to dissolve heavy metals and therefore to increase their genotoxicity. An insoluble chromic compound, lead chromate (PbCr04), was not cytotoxic nor mutagenic on V79 cells, probably because it is taken up by the cells very slowly. This effect was due to solubilization of the chromate anion by nitrilotriacetic acid, as determined by comparing spectrophotometric determinations of chromic in lead chromate treatment solutons with a mutagenicity titration curve obtained with a completely soluble chromic salt (potassium dichromate, K2Cr207). [Celotti L et al; Mutat Res 190 (1): 35-40 (1987)] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

By using the sex linked recessive lethal mutation test in Drosophila melanogaster, the mutagenic effects of treatments by feeding with nitrilotriacetic acid (5xM-1M), with the insol chromic cmpd lead chromate (PbCr04) (supernatant of 4.6x10-4M suspension in which the actual concn was 0.06 gamma/ml as chromic), and with both cmpd preincubated at 3 relative ratios (nitrilotriacetic acid: 5x10-2M; PbCr04: 64.6x10-4, 4.6x10-5 and 9.2x10-M, respectively), were analyzed. ... EMS (5x10-3M) was used as the ref pos control, with clearly mutagenic results. Treatment with nitrilotriacetic acid or with PbCr04 alone did not induce any significant increase of the mutation frequency. PbCr04 at the 3 concn tested was completely sol in the 5x10-2M nitrilotriacetic acid soln, and the mixt of nitrilotriacetic acid and PbCr04 induced significant increases of the frequency of sexlinked lethal mutations, with a significant dose effect relationship with respect to PbCr04. [Costa R et al; Mutat Res 204 (2): 257-61 (1988)] **PEER REVIEWED**

NON-HUMAN TOXICITY EXCERPTS

The influence of nitrilotriacetic acid on the mutagenic and clastogenic activity of several water insoluble or poorly soluble chromium compounds was detected by means of Salmonella/microsome assay (plate test on TA100 strain) and the sister chromatid exchange test in mammalian cell cultures (CHO line). Nitrilotriacetic acid in itself did not induce gene mutations nor did it increase the frequency of the sister chromatid exchange. Chromic cmpd (lead, barium, zinc, strontium, and calcium chromates) and an industrial chromic pigment, chromium orange, were inactive or scarcely active mutagens in the Salmonella/microsome test when dissolved in water, but they were increasingly mutagenic when solubilized by 0.5N sodium hydroxide or nitrilotriacetic acid (10 or 100 mg/ml). Also, the mutagenic activity of chromic, contaminating an industrial chromous pigment (chromite), was slightly enhanced by nitrilotriacetic acid. Mutagenicity of chromate was correlated with the amounts of chromic solubilized by nitrilotriacetic acid or alkali, as detected by the colorimetric reaction with diphenylcarbazide and atomic absorption spectrophotometry, and was decreased by incubation with microsomes, due to reduction of chromic to the genetically inactive chromous form. [Venier P et al; Mutat Res 156 (3): 219-28 (1985)] **PEER REVIEWED**

POPULATIONS AT SPECIAL RISK

The effects of chromium compounds on the skin are caused primarily by direct contact. Most of the effects have occurred in occupational settings, and, as expected, with more men than women reporting these effects. /Chromium compounds/ [USEPA; Health Assessment Document: Chromium p.2-8 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

POPULATIONS AT SPECIAL RISK

Persons with a history of asthma, allergies, or known sensitizations to chromic acid or chromates would be expected to be at increased risk from exposure. /Chromic acid or chromates/ [Mackison, F. W., R. S. Stricoff, and L. J. Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) Publication No. 81-123 (3 VOLS). Washington, DC: U.S. Government Printing Office, Jan. 1981. , p. 21 **PEER REVIEWED**

POPULATIONS AT SPECIAL RISK

Individuals having genetically caused glucose-6-phosphate dehydrogenase deficiency are at higher risk of lead induced hemolysis than normal individuals. Various iron storage diseases may incr lead in the human liver. /Inorganic lead/ [Nat'l Research Council Canada; Effects of Lead in the Canadian Envir p.67 (1978) NRCC No.0316-01141 **PEER REVIEWED**

POPULATIONS AT SPECIAL RISK

Children with pica for paint chips or for soil may experience an elevation in blood lead ranging from marginal to sufficiently high levels to cause clinical illness. ... Women in workplace are more likely to experience adverse effects from lead exposure than men due to the fact that their hematopoietic system is more lead sensitive than men's. /Inorganic lead/ [Sittig, M. Handbook of Toxic and Hazardous Chemicals and Carcinogens, 1985. 2nd ed. Park Ridge, NJ: Noyes Data Corporation, 1985. , p. 5431 **PEER REVIEWED**

POPULATIONS AT SPECIAL RISK

... Health hazard of Lead poisoning in police officers employed in detection of fingerprints. /Inorganic lead/ [Rabjerg L et al; Scand J Work, Environ Health 9(6): 511-13 (1983)] **PEER REVIEWED**

POPULATIONS AT SPECIAL RISK

Children with sickle-cell anemia are at greater risk of developing nervous system effects due to lead absorption ... /because the disease results in/ an increased erythrocyte turnover, ... altered renal functioning ... and a zinc deficiency. ... /Lead cmpd/ [Erenberg G et al; Pediatrics 54 (4): 428-441 (1974) as cited in Nat'l Research Council Canada; Effects of Lead in the Canadian Envir p.571 (1978) NRCC No.16736] **PEER REVIEWED**

POPULATIONS AT SPECIAL RISK

Pregnant women undergoing lithium (Li) psychotherapy were considered ... especially susceptible to lead (Pb) exposure ...

/since/ both Li and Pb cross the placental barrier and could interact to harm the fetus. /Inorganic lead/ [Boulos BM; NIOSH 134: 39 (1976) as cited in Nat'l Research Council Canada; Effects of Lead in the Canadian Envir p.563 (1978) NRCC No.16736] **PEER REVIEWED**

POPULATIONS AT SPECIAL RISK

/Individuals/ having hemoglobin S or O or thalassemia ... have increased susceptability to lead and other poisons affecting the synthesis and life span of red blood cells. /Inorganic lead/ [Goyer RA, Mahaffey KR; Eniron Health Perspect 2: 73-80 (1972) as cited in Nat'l Research Council Canada; Effects of Lead in the Canadian Envir p.571 (1978) NRCC No.16736] **PEER REVIEWED**

POPULATIONS AT SPECIAL RISK

... If a pregnant woman is exposed to lead, it can be carried to the unborn child and cause premature birth, low birth weight, or even abortion. /Lead cmpd/ [DHHS/ATSDR; Toxicological Profile for Lead p. 2 (1990) TP-88/17] **PEER REVIEWED**

POPULATIONS AT SPECIAL RISK

Young children are at risk because they swallow lead when they put toys or objects soild with lead containing dirt in their mouths. For infants and young children, lead exposure has been shown to decrease intelligence (IQ) scores, slow their growth, and cause hearing problems. /Lead cmpd/ [DHHS/ATSDR; Toxicological Profile for Lead p.2-3 (1990) TP-88/17] **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETICN

(51)Cr-labelled sodium, zinc and lead chromates were studied. Sodium chromate and the less soluble zinc chromate were absorbed by the blood, resulting in increased urinary excretion of chromium. ... The less water-soluble the chromate, the higher was its elimination via the feces. Absorbed chromium was retained in the spleen and bone marrow in all three cases, and also in the liver and kidneys in the case of sodium chromate. Chromium levels in blood and urine are not indicative of inhalation exposure to insoluble chromates. [Bragt PC, van Dura EA; Annals of Occupational Hygiene 27 (3): 315-22 (1983)] **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

Unlike the trivalent compounds, those of Cr(VI) tend to cross biological membranes fairly easily, and are somewhat more readily absorbed through the gut or through the skin. /Hexavalent chromium/ [USEPA; Health Assessment Document: Chromium p.2-7 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

In the airways and in the GI tract, soluble Cr(VI) compounds are apparently taken up by epithelial cells, by simple diffusion, through the plasma membrane. After entry, Cr(VI) reduction occurs from the action of enzymatically mobilized electrons, which are available from GSH, NADPH, and NADH. The reducing capacity inside the cell is limited, so that Cr(VI) and Cr(III) exist simultaneously inside the cytoplasm; Cr(VI) is then released from the cell by simple diffusion into the bloodstream and taken up by blood cells. /Hexavalent chromium/ [USEPA; Health Assessment Document: Chromium p.2-6 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

In spite of the refined methods of analysis available, a reliable range of normal blood chromium concentrations cannot be given with confidence. When using modern methods for analysis, the whole blood concentration is suggested to be within the range of 0.5 to 3 ppb, while the serum level is probably below 0.2 ppb. /Chromium/ [USEPA; Health Assessment Document: Chromium p.2-6 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

FOR ALL PRACTICAL PURPOSES, THERE ARE TWO FORMS OF LEAD. THE FIRST IS INORGANIC LEAD, IN WHICH THE VARIOUS SALTS & OXIDES ARE CONSIDERED TO ACT IDENTICALLY ONCE ABSORBED INTO THE SYSTEMIC CIRCULATION. THE SECOND IS ALKYL LEAD ... CLEARLY DIFFERENT FROM INORGANIC FORMS OF LEAD, AS TO ... ABSORPTION & DISPOSITION IN THE BODY. ... DISTINCTIONS ARE NOT GENERALLY

MADE REGARDING THE DISPOSITION OF THE VARIOUS INORGANIC COMPOUNDS. IT IS ASSUMED THAT LEAD IONS /OF VARIOUS INORG COMPOUNDS/ DISSOCIATE TO SOME DEGREE & ARE ABSORBED & DISTRIBUTED IN THE BODY IN THE SAME MANNER, REGARDLESS OF ENVIRONMENTAL ORIGIN. THE VALIDITY OF THIS ASSUMPTION HAS NOT BEEN TESTED ... /INORGANIC LEAD/ [Doull, J., C.D. Klaassen, and M. D. Amdur (eds.). Casarett and Doull's Toxicology. 2nd ed. New York: Macmillan Publishing Co., 1980. , p. 4151 **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

The absorption rate of deposited lead depends on various factors, particularly on the physiochemical form of lead in particles. There is ... no evidence of lead accunulation in the lung, & any lead compound once deposited is eventually absorbed or transferred to the gastrointestinal tract. /Inorganic lead/ Ifriberg, L., Nordberg, G.F., Kessler, E. and Vouk, V.B. (eds). Handbook of the Toxicology of Metals. 2nd ed. Vols I, 11.: Amsterdam: Elsevier Science Publishers B.V., 1986. V2 3121 **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

Only a very minor fraction of particles over 0.5 um in mean maximal external diameter are retained in the lung but are cleared from the respiratory tract & swallowed. ... The percentage of particles less than 0.5 um retained in the lung increases with reduction in particle size. About 90% of lead particles in ambient air that are deposited in lung are small enough to be retained. Absorption of retained lead through alveoli is relatively efficient & complete. /Inorganic lead/ [Doull, J., C.D.Klassen, and M.D. Amdur (eds.). Casarett and Doull's Toxicology. 3rd ed., New York: Macmillan Co., Inc., 1986. , p. 5991 **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

Studies of the absorption of ingested lead indicate that about 10% is absorbed from the GI tract. However ... balance studies in adults ... /indicate/ that up to 40% of ingested lead was absorbed in some individuals. ... The absorption of radioactive lead in fasting subjects /was found/ to be approx 60% after an oral dose of *4* to *400* ug lead. The absorption of lead decreased from 60 to 10% if calcium & phosphate were added in doses equivalent to the ones found in a normal meal. For children, the fraction absorbed may be much higher ... up to 53% absorption in 8 children ranging in age from 3 mo to 8 yr /have been observed/. These results require verification because of the larger scatter of values & the short period of observation. It should be noted that there is great variation among individuals. /Inorganic Lead/ Ifriberg, L., Nordberg, G.F., Kessler, E. and Vouk, V.B. (eds). Handbook of the Toxicology of Metals. 2nd ed. Vols I, II.: Amsterdam: Elsevier Science Publishers B.V., 1986. V2 3121 **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

THE PROPORTION ABSORBED IS MUCH THE SAME WHETHER THE LEAD IS PRESENT AS A SOLUBLE OR AN INSOLUBLE SALT SUCH AS THE CARBONATE, SULFATE OR SULFIDE ... WHICH, ALTHOUGH INSOLUBLE IN WATER, ARE APPRECIABLY SOLUBLE IN BODY FLUIDS. /INORGANIC LEAD/ [Clarke, M. L., D. G. Harvey and D. J. Humphreys. Veterinary Toxicology. 2nd ed. London: Bailliere Tindall, 1981. , p. 561 **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

INORG LEAD DOES NOT PENETRATE NORMAL SKIN, BUT IT CAN PENETRATE ABRADED SKIN. /INORGANIC LEAD/ [Goodman, L.S., and A. Gilman. (eds.) The Pharmacological Basis of Therapeutics. 5th ed. New York: Macmillan Publishing Co., Inc., 1975. , p. 9381 **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

Little is known about lead transport across the gastrointestinal mucosa. It has been speculated that lead & calcium may compete for a comnon transport mechanism, since there is a reciprocal relationship between the dietary content of calcium & lead absorption. /Inorganic lead/ [Doull, J., C.D.Klassen, and M.D. Amdur (eds.). Casarett and Doull's Toxicology. 3rd ed., New York: Macmillan Co., Inc., 1986. , p. 16061 **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

Absorbed lead is transported by blood & initially distributed in various organs & tissues. It is then gradually redistributed to form an exchangeable compartment (blood & soft tissues) & a storage compartment, essentially bone. In human subjects with low lead exposure, about 90% of the total body burden is found in bone. ... Lead in blood is mainly bound to erythrocytes where its concentration is about 16 times higher than in plasma. The manner in which lead combines with erythrocytes is not clear, but it is probably assoc with hemoglobin. Lead also has an affinity for cell membrane & mitochondria, but not for lysosomes. Lead in bone builds up throughout life; this does not apply to soft tissues where it stabilizes early in adult life; in some tissues it may even decrease with age. /Inorganic lead/ [Friberg, L., Nordberg, G.F., Kessler, E. and Vouk, V.B. (eds). Handbook of the Toxicology of Metals. 2nd ed. Vols I, II.: Amsterdam: Elsevier Science Publishers B.V., 1986. V2 3131 **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

After absorption, inorganic lead is distributed initially in soft tissues, particularly the tubular epithelium of the kidney & in the liver. In time .../it/ is redistributed & deposited in bone, teeth, & hair. ... Only small quantities of inorganic lead accum in the brain, with most of that in gray matter & the basal ganglia. Nearly all circulating inorganic lead is assoc with erythrocytes; only when lead is present in relatively high concn does a significant portion remain in the plasma. /Inorganic lead/ [Gilman, A.G., L.S.Goodman, and A. Gilman. (eds.). Goodman and Gilman's The Pharmacological Basis of Therapeutics. 7th ed. New York: Macmillan Publishing Co., Inc., 1985. , p. 16061 **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

Blood lead levels are a good indicator of recent exposure to lead & are influenced by inhalation & ingestion. A number of recent studies suggest that inhalation of air containing 1 ug/cu m in respirable particles will increase blood lead concentrations by about 1 ug/dl when air lead conc are in the range of 1-5 ug/cu m. Lead in blood varies with age. Children under 7 yr of age have significantly higher blood lead levels than older children, & there is no difference between boys & girls under age 12. Blood lead levels decline during adolescence /& is/ probably related to bone growth & deposition *of* lead in bone with calcium. Blood lead Levels are lower in adult females than adult males. /Inorganic lead/ [Doull, J., C.D.Klassen, and M.D. Amdur (eds.). Casarett and Doull's Toxicology. 3rd ed., New York: Macmillan Co., Inc., 1986. , p. 5991 **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

Urban residents have the highest blood lead levels (20-25 ug/dl); suburban (10-20 ug/dl) & rural populations (10-14 ug/dl) have lower levels. These Levels correlate with air lead levels & perhaps leaded gasoline use. The avg US blood Lead level declined 37% from 1976 to 1980 as leaded gasoline consumption declined 55%. In women of Wales, blood lead levels dropped 30% between 1972 & 1982 despite little change in the total amt of lead used in gasoline. /Inorganic Lead/ [Ellenhorn, M.J. and D.G. Barceloux. Medical Toxicology - Diagnosis and Treatment of Human Poisoning. New York, NY: Elsevier Science Publishing Co., Inc. 1988. , p. 10301 **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

The deposition of lead in bone closely resembles that of calcium, but it is deposited as tertiary lead phosphate. Lead in the bone salts dose not contribute to toxicity. After a recent exposure, the concn of lead is often higher in the flat bones than in the long bones, although, as general rule, the long bones contain more lead. In the early period of deposition, the concentration of lead is highest in the epiphyseal portion of the long bones. This is esp true in growing bones, where deposits may be detected by X-ray examination as rings of increased density in the ossification centers of the epiphyseal cartilage & as a series of tranverse lines in the diaphyses, so called lead lines. Such findings are of diagnostic significance in children. /Inorganic lead/

[Gitman, A.G., L.S.Goodman, and A. Gilman. (eds.). Goodman and Gilman's The Pharmacological Basis of Therapeutics. 7th ed. New York: Macmillan Publishing Co., Inc., 1985. , p. 16061 **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

About 90% of ingested lead is eliminated unabsorbed through feces. Absorbed lead is excreted primarily in urine (about 76%); other ... routes are gastrointestinal secretions (about 16%) & hair, nails & sweat (< 8%). The rate of biliary excretion in man is not known. The mechanism of excretion appears to be essentially glomerular filtration. Lead is also excreted in human milk in concentrations ... up to 12 ug/l. There is considerable species variation for lead excretion in animals. In baboons, the main route of excretion is urine, but in rat & sheep, biliary & transmucosal excretion may be higher than urinary excretion. /Inorganic lead/ [Friberg, L., Nordberg, G.F., Kessler, E. and Vouk, V.B. (eds). Handbook of the Toxicology of Metals. 2nd ed. Vols I, II.: Amsterdam: Elsevier Science Publishers B.V., 1986. V2 3141 **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

Placental transfer of lead has been demonstrated. ... There is fairly good correlation between lead concentrations in the blood of mothers & newborn infants, & the distribution in fetal tissues is similar to the distribution in the adult. ... /Inorganic lead/ [Friberg, L., Nordberg, G.F., Kessler, E. and Vouk, V.B. (eds). Handbook of the Toxicology of Metals. 2nd ed. Vols I, II.: Amsterdam: Elsevier Science Publishers B.V., 1986. V2 3141 **PEER REVIEWED**

ABSORPTION; DISTRIBUTION AND EXCRETION

Maternal blood lead decreases during pregnancy, suggesting that maternal lead is transferred to the fetus or excreted in some way. /Inorganic lead/ [Doull, J., C.D.Klassen, and M.D. Amdur (eds.). Casarett and Doull's Toxicology. 3rd ed., New York: Macmillan Co., Inc., 1986. , p. 5991 **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

In area of high atmospheric lead contamination the mean lead concn in maternal & fetal cord blood in 122 women were 412 & 379 ng/ml, respectively, compared with 147 & 118 ng/ml in 31 controls; birth wt & red cell values of the infants were not adversely affected. /Inorganic lead/ [Reynolds, J.E.F., Prasad, A.B. (eds.) Martindale-The Extra Pharmacopoeia. 28th ed. London: The Pharmaceutical Press, 1982. , p. 9361 **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

Plants take up lead in the ionic form from soils. Lead is absorbed, but not accumulated to any great extent by plants from soil. Lead is either unavailable to plants or "fixed" in the roots with only small amounts being translocated to the above ground portions, even when plants are grown in soil containing substantial amounts of lead. This is due not only to the solubility of lead in the soil, but to some internal factors which govern the mobility of lead within the plant. ... Variations in plant uptake of Lead are due to plant age and species, soil phosphorous, organic matter content, pH, soil texture, climate, topography, pollution, and geological history of the soil. The amount of lead taken up decreases as pH, cation exchange capacity, and available phosphorous of the soil increase. Only about 0.003 to 0.005 percent of total soil lead is available for plant uptake. /Inorganic lead/ [USEPA; The Health and Environmental Impacts of Lead: p.175 (1979) EPA 560/2-79-001] **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

Excretion of lead in /one non-occupationally exposed man/ was: urine (76%); gastrointestinal secretions (16%); hair, nails, sweat, other (8%). /Inorganic lead/ [Rabinowitz MB et al; Science 182: 725-7 (1973) as cited in WHO; Environ Health Criteria: Lead p.84 (1977)] **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

Using radioactive tracers /an estimated/ 70% of the Lead absorbed from the lungs of a human remained present in blood while 30% was almost imnediately absorbed by the soft tissues ... after 24 hr. /Inorganic lead/ [Hursh JB, Mercer TT; J Appl

Physiol 28: 268 (1970) as cited in Nat'l Research Council Canada; Effects of Lead in the Canadian Envir p.271 (1978) NRCC No.16736] **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

... Lead was absorbed /by fresh water field crab, Barytelphusa guerini/ through the gills and distributed by the haemolymph to hepatopancreas, muscle, and exoskeleton. Haemolymph was found to contain the highest amount of lead followed by gill, hepatopancreas, muscle, and exoskeleton. Lead bioaccumulated over the course of the study showed a high degree of organ specificity. /Inorganic Lead/ [Tulasi SJ et al; Bull Environ Contam Toxicol 39 (1): 63-8 (1987)] **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

beta-Aminoisobutyric acid, a normal degradation product of thymine, a constituent of DNA and, to a Lesser extent, of transfer RNA, is excreted in low levels in human urine. /It was/ found that a group of iron workers occupationally exposed to inorganic lead excreted high levels of urinary beta-aminoisobutyric acid. ... The results suggest that increased urinary excretion of beta-aminoisobutyric acid could stem from damage to DNA on exposure to lead. /Inorganic lead/ [Farkas WR et al; Arch Environ Health 42 (2): 96-8 (1987)] **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

IT IS ASSUMED THAT LEAD IONS /OF VARIOUS INORG CMPD/ DISSOCIATE TO SOME DEGREE & ARE ABSORBED & DISTRIBUTED IN THE BODY IN THE SAME MANNER, REGARDLESS OF ENVIRONMENTAL ORIGIN. /INORGANIC LEAD/ [Doull, J., C.D. Klaassen, and M. D. Amdur (eds.). Casarett and Doull's Toxicology. 2nd ed. New York: Macmillan Publishing Co., 1980. , p. 4151 **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

ABSORPTION IS ... INFLUENCED BY CONCURRENT DIETARY LEVELS OF ... CALCIUM, IRON, FATS, & PROTEINS ... /IT/ ... IS ... GREATER IN INFANTS THAN IN ADULTS & DURING FASTING STATE THAN WITH MEALS. /INORGANIC LEAD/ [Doull, J., C.D. Klaassen, and M. D. Amdur (eds.). Casarett and Doull's Toxicology. 2nd ed. New York: Macmillan Publishing Co., 1980. , p. 4161 **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

Plants can absorb and translocate available level lead from nutrient solution, and to a lesser degree from soil. Most of the absorbed Pb remains in the roots, but cessation of growth in late summer and fall is often accompanied by mobilization of Pb from roots into the plant tops. Pasture herbage growing in an abandoned lead mining area (soil pH 4, soil Pb 3600 ppm) accumulated up to 74 ppm Pb (dry weight basis) in their leaves. Corn accumulated 37.8 ppm in tops from a soil treated with 3200 kg Pb/ha, but the kernels did not contain significantly more Pb than controls (0.3-0.5 ppm Pb at 15.5% moisture). Lettuce and radishes were two plants shown to accumulate Pb from soil. Pb chelates were taken up by plants more readily than Pb(2+) and exhibited a high degree of translocation to the plant tops. /Inorganic lead/ [Nat'l Research Council Canada; Effects of Lead in the Canadian Environment p.48 (1978) NRCC No. 16736] **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

... INORGANIC LEAD, IN WHICH VARIOUS SALTS & OXIDES ARE CONSIDERED TO ACT IDENTICALLY ONCE ABSORBED INTO SYSTEMIC CIRCULATION. ... DISTINCTIONS ARE NOT GENERALLY MADE REGARDING DISPOSITION OF VARIOUS INORG CMPD. /INORG LEAD/ [Friberg, L., G.R. Nordberg, and V.B. Vouk. Handbook on the Toxicology of Metals. New York: Elsevier North Holland, 1979. , p. 4151 **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

... POTASSIUM CHROMATE INJECTED INTRATRACHEALLY INTO GUINEA PIGS IS REMOVED RAPIDLY (A FEW DAYS) FROM LUNGS EXCEPT FOR SMALL AMT STILL PRESENT @ 140 DAYS. IN 10 MIN, 20% WAS IN BLOOD & ONLY 5% IN LIVER, KIDNEYS, & SPLEEN TOGETHER. CONSIDERABLE AMT OF CHROMIUM ... ATTACHED TO ERYTHROCYTES. /POTASSIUM CHROMATE/ [Patty, F. (ed.). Industrial Hygiene and Toxicology: Volume II: Toxicology. 2nd ed. New York: Interscience Publishers, 1963. , p. 10201 **PEER REVIEWED**

ABSORPTION, DISTRIBUTION AND EXCRETION

Lead has a strong tendency to localize and accumulate in bone. /Inorganic lead/ [WHO; Environ Health Criteria: Lead p.80 (1977)] **PEER REVIEWED**

METABOLISM/METABOLITES

Chromium(VI) as chromate is potentially an oxidizing agent that may react with reducing (organic) matter to form chromium(III). /Hexavalent chromium/ [NIOSH; Criteria Document: Chromium (VI) p.33 (1976) DHEU Pub. NIOSH 76-1291 **PEER REVIEWED**

METABOLISM/METABOLITES

THE METABOLISM OF LEAD FOLLOWS CLOSELY THAT OF CALCIUM, PARTICULARLY WITH REGARD TO ITS DEPOSITION IN & MOBILIZATION FROM BONE. ... WHEN BONE MARROW ACTIVITY IS INCREASER ... CALCIUM MOBILIZATION MAY TAKE PLACE, CAUSING HYPERCALCEMIA & OSTEOLYSIS. ... LEAD MAY ALSO BE LIBERATED. ... /INORGANIC LEAD/ [Browning, E. Toxicity of Industrial Metals. 2nd ed. New York: Appleton-Century-Crofts, 1969. , p. 173] **PEER REVIEWED**

BIOLOGICAL HALF-LIFE

THE HALF-LIFE OF LEAD IN BLOOD IS ABOUT 1 MO, AND A STEADY STATE IS THUS ACHIEVED IN ABOUT 5 MO. /INORGANIC LEAD/ [Gilman, A.G., L.S.Goodman, and A. Gilman. (eds.). Goodman and Gilman's The Pharmacological Basis of Therapeutics. 7th ed. New York: Macmillan Publishing Co., Inc., 1985. , p. 1606] **PEER REVIEWED**

BIOLOGICAL HALF-LIFE

... /In humans/ with a constant daily oral input of (204) lead a virtually constant concn of the tracer in the blood was achieved after 110 days. Upon withdrawal of the tracer from the diet, the (204)Pb concn in the blood disappeared with a half-time of approx 19 days. ... /Lead cmpd/ [Rabinowitz MB et al; Environ Health Perspect 7: 145 (1974) as cited in WHO; Environ Health Criteria: Lead p.81 (1977)] **PEER REVIEWED**

BIOLOGICAL HALF-LIFE

The half-life of Lead in adult human blood has been measured as 36 days. /Lead cmpd/ [Rabinowitz MB et al; J Lab Clin Med 90: 238-48 (1977) as cited in DHHS/ATSDR; Toxicological Profile for Lead p.56 (1990) TP-88/17] **PEER REVIEWED**

BIOLOGICAL HALF-LIFE

/In another study/, the half-life of lead in adult human blood was 28 days. /Lead cmpd/ [Griffin TB et al; Arh Hig Toksikol 26: 191-208 (1975) as cited in DHHS/ATSDR; Toxicological Profile for Lead p.56 (1990) TP-88/17] **PEER REVIEWED**

BIOLOGICAL HALF-LIFE

The biological half-life of lead in the blood of 2 yr old children was reported to be approx 10 mo. /Lead cmpd/ [Succop PA et al; A Kinetic Model for Estimating Changes in the Concentration of Lead in the Blood of Young Children. In: Lindberg SE, Hutchinson TC (eds). International Conference: Heavy Metals in the Environment Vol 2 (1987) as cited in DHHS/ATSDR; Toxicological Profile for Lead p.56 (1990) TP-88/17] **PEER REVIEWED**

MECHANISM OF ACTION

Inorganic lead causes contraction of the peripheral vascular system and affects the blood and blood-forming tissues (bone marrow). The "normal" cases of lead poisoning in industry always include hemopoietic effects; These occur very early, before the appearance of /other/ symptoms or signs and, therefore, are important for diagnosis. Lead shortens the life of erythrocytes and impairs hemoglobin synthesis. /Inorganic lead/ [International Labour Office. Encyclopedia of Occupational Health and Safety. Vols. 1811. Geneva, Switzerland: International Labour Office, 1983. , p. 12011 **PEER REVIEWED**

MECHANISM OF ACTION

LEAD CLEARLY INHIBITS HEME FORMATION AT SEVERAL POINTS; DELTA-AMINOLEVULINATE DEHYDRATRASE & FERROCHELATASE + FE(2+). INHIBITION OF DELTA-AMINOLEVULINATE (DELTA-ALA) DEHYDRATASE & FERROCHELATASE, WHICH ARE SULFHYDRYL-DEPENDENT ENZYMES, IS WELL DOCUMENTED. LEAD POISONING IN ... MAN & EXPTL ANIMALS IS CHARACTERIZED BY ACCUM OF PROTOPORPHYRIN IX & NONHEME IRON IN RED BLOOD CELLS, BY ACCUMULATION OF DELTA-ALA IN PLASMA, & BY INCR URINARY EXCRETION OF DELTA-ALA. ... THE INCREASE IN DELTA-ALA SYNTHETASE ACTIVITY IS DUE TO THE REDUCTION OF THE

CELLULAR CONCENTRATION OF HEME, WHICH REGULATES THE SYNTHESIS OF DELTA-ALA SYNTHETASE BY FEEDBACK INHIBITION. /INORGANIC LEAD/ [Gilman, A.G., L.S.Goodman, and A. Gilman. (eds.). Goodman and Gilman's The Pharmacological Basis of Therapeutics. 7th ed. New York: Macmillan Publishing Co., Inc., 1985. , p. 16081 **PEER REVIEWED**

MECHANISM OF ACTION

Many toxic effects of inorganic lead can be attributed to the affinity of lead for thiol groups (-SH) and other organic ligands in proteins. Low concn of Pb(2+) inhibit a select group of enzymes, including thiol dependent enzymes involved in heme synthesis and mitochondrial energetics. The toxicologic effects of lead may result from the ability of Pb(2+) to uncouple oxidative phosphorylation and modify mitochondrial ion transport (especially for calcium Ca(2+)). /Lead cmpd/ [Kayser, R., D. Sterling, D. Viviani (eds.). Intermedia Priority Pollutant Guidance Documents. Washington, DC: U.S.Environmental Protection Agency, July 1982. 2-11 **PEER REVIEWED**

MECHANISM OF ACTION

Lead can interfere with the synthesis of heme, thereby altering the urinary or blood concn of enzymes and intermediates in heme synthesis or their derivatives. Thus lead poisoning can lead to accumulation of non-heme iron and protoporphyrin-IX in red cells, an increase in delta-aminolevulinic acid (ALA) in blood and urine, an increase in urinary coproporphyrin, uroporphyrin, and porphobilinogen, inhibition of blood ALA-dehydratase (ALA-D), and an increased proportion of immature red cells in the blood (reticulocytes and basophilic stippled cells). /Inorganic lead/ [NIOSH; Criteria Document: Inorganic Lead p.III-4 (1978) DHEW Pub. NIOSH 78-158] **PEER REVIEWED**

MECHANISM OF ACTION

Many toxic effects of inorganic lead can be attributed to the affinity of lead for thiols and other organic ligands in proteins resulting in an uncoupling of oxidative phosphorylation. /Inorganic lead/ [USEPA; Intermedia Priority Pollutant Guidance Document-Lead (1982)] **PEER REVIEWED**

MECHANISM OF ACTION

... Lead interferes ... with synaptic transmission in the peripheral nervous system. ... /Inorganic lead/ [Kostial K, Vorik VB; Brit J Pharmacol, Chemother 12: 219-22 (1957) as cited in WHO; Environ Health Criteria: Lead p.89 (1977)] **PEER REVIEWED**

MECHANISM OF ACTION

... In humans, lead decreased glucose-6-phosphate dehydrogenase activity and thus decreased the glutathione level in red blood cells. /Lead cmpd/ [Stokinger HE, Mountain JT; J Occup Med 9: 537-42 (1967) as cited in Nat'l Research Council Canada; Effects of Lead in the Canadian Envir p.572 (1978) NRCC No.16736] **PEER REVIEWED**

MECHANISM OF ACTION

Lead inhibits the catabolism of valine and isoleucine which are metabolized via succinyl-CoA to form delta-ALA. ... This is particularly important in the synthesis of heme for intracellular cytochrome synthesis. /Lead cmpd/ [Cavender FL; Biochem Med 5: 515-21 (1971)] **PEER REVIEWED**

MECHANISM OF ACTION

... Lead inhibits several enzymes that participate in heme synthesis, ... heme synthetase, ... coproporphyrin synthetase, ... and coproporphyrin oxidase. /Inorganic lead/ [WHO; Environ Health Criteria: Lead p.86-9 (1977)] **PEER REVIEWED**

MECHANISM OF ACTION

When the blood lead concn is near 80 ug/dl or greater, basophilic stippling (the aggregation of ribonucleic acid) occurs in erythrocytes. This is thought to result from the inhibitory effect of lead on the enzyme, pyrimidine-5'-nucleotidase. Basophilic stippling is not ... pathognomonic of Lead poisoning. A more common hematological result of chronic lead intoxication is a hypochromic microcytic anemia, which is more frequently observed in children. This anemia is morphologically similar to that which results from iron deficiency & is thought to result from two factors: a decreased life span of the erythrocytes & an inhibition of heme synthesis. Very low concn of lead influence the synthesis of

heme. ... Lead clearly inhibits heme formation at several ... /enzymatic steps/. /Inorganic lead/ [Gilman, A.G., L.S.Goodman, and A. Gilman. (eds.). Goodman and Gilman's The Pharmacological Basis of Therapeutics. 7th ed. New York: Macmillan Publishing Co., Inc., 1985. , p. 16081 **PEER REVIEWED**

MECHANISM OF ACTION

Animal studies have ... shown that certain dietary factors, such as milk, fasting, low calcium & vitamin D, & iron deficiency, may enchance lead absorption. /Inorganic lead/ [Friberg, L., Nordberg, G.F., Kessler, E. and Vouk, V.B. (eds). Handbook of the Toxicology of Metals. 2nd ed. Vols I, II.: Amsterdam: Elsevier Science Publishers B.V., 1986. V2 3121 **PEER REVIEWED**

MECHANISM OF ACTION

A distinct relationship has been found between lead poisoning and the metabolism of nicotinic acid. Animals poisoned by lead showed a marked decrease in the nicotinic acid content of blood and urine, indicating an increased utilization of this nutrient by lead, and suggesting that lead exerts serious effects on the pyridine nucleotides, either by blocking their synthesis or by accelerating the degradation of nicotinic acid. /Inorganic lead/ [NIOSH; Criteria Document: Inorganic Lead p.III-12 (1978) DHEW Pub. NIOSH 78-1581 **PEER REVIEWED**

MECHANISM OF ACTION

Four groups of rats received by intratracheal instillation (1) a lead chromate paint particulate suspension, (2) lead tetraoxide suspension, (3) lead acetate solutions, or (4) saline. Lead (Pb) dosed animals received an equivalent dose of 1 mg Pb/kg. The distribution of Pb was monitored through assays of urine, feces, and tissues (lung, bone, muscle, kidney, liver) obtained at postmortem 5 wk after exposure. delta-Aminolevulinic acid dehydratase (ALA-D) activity was measured to determine the effect of Pb on heme biosynthesis. The vast majority of the dosed Pb in the paint matrix remained in the lung. In contrast, in the lead acetate dosed animals, little remained in the lung, but significant elevations were found in bone and kidney. Blood delta-aminolevulinic acid dehydratase was significantly depressed in the lead acetate treated animals, but was not significantly different from control animals in the animals dosed with Pb paint or Pb tetraoxide. [Eaton DL et al; Toxicol Lett 22 (3): 307-13 (1984)] **PEER REVIEWED**

INTERACTIONS

Divalent lead has a strong affinity for inorganic ions containing oxygen (eg carbonate) or sulfur (eg sulfide). Lead can also complex with electron rich Ligands in many organic cmpd such as amino acids, proteins, and humic acid. /Inorganic lead/ [Kayser, R., D. Sterling, D. Viviani (eds.). Intermedia Priority Pollutant Guidance Documents. Washington, DC: U.S.Environmental Protection Agency, July 1982. 1-11 **PEER REVIEWED**

INTERACTIONS

Alcohol ingestion ... may precipitate .../intense periodic abdominal cramping symptoms as a result of its interaction with Lead/. /Inorganic lead/ [Sittig, M. Handbook of Toxic and Hazardous Chemicals and Carcinogens, 1985. 2nd ed. Park Ridge, NJ: Noyes Data Corporation, 1985. , p. 5441 **PEER REVIEWED**

INTERACTIONS

... A HIGH INTAKE OF PHOSPHATE FAVORS SKELETAL STORAGE OF LEAD & A LOWER CONCENTRATION IN SOFT TISSUES. CONVERSELY, A LOW PHOSPHATE INTAKE MOBILIZES LEAD IN BONE & ELEVATES ITS CONTENT IN SOFT TISSUES. HIGH INTAKE OF CALCIUM IN THE ABSENCE OF ELEVATED INTAKE OF PHOSPHATE HAS A SIMILAR EFFECT, OWING TO COMPETITION WITH LEAD FOR AVAIL PHOSPHATE. VITAMIN D TENDS TO PROMOTE THE DEPOSITION OF LEAD IN BONE IF SUFFICIENT AMT OF PHOSPHATE IS AVAIL; OTHERWISE, DEPOSITION OF CALCIUM PREEMPTS THAT OF LEAD. PARATHYROID HORMONE & DIHYDROTACHYSTEROL MOBILIZE LEAD FROM SKELETON & AUGMENT THE CONCN OF LEAD IN BLOOD & ITS RATE OF URINARY EXCRETION. /INORGANIC LEAD/ [Gilman, A.G., L.S.Goodman, and A. Gilman. (eds.). Goodman and Gilman's The Pharmacological Basis of Therapeutics. 7th ed. New York: Macmillan Publishing Co., Inc., 1985. , p. 16071 **PEER REVIEWED**

INTERACTIONS

Corticosteroids should not be given in lead poisoning as severe symptoms may be precipitated. Morphine may excerbate cerebral symptoms. /Inorganic lead/ [Reynolds, J.E.F., Prasad, A.B. (eds.) Martindale-The Extra Pharmacopoeia. 28th ed. London: The Pharmaceutical Press, 1982. , p. 9361 **PEER REVIEWED**

INTERACTIONS

Synergistic effect of the simultaneous exposure of rats to lead & cadmium at 25 ug/day/kg body wt (IM, IP) for 70 days produced the absence of spermatogenesis as observed from histological study. /Inorganic lead; from table/ [National Research Council. Drinking Water and Health, Volume 6. Washington, D.C.: National Academy Press, 1986. , p. 741 **PEER REVIEWED**

INTERACTIONS

Lead exposure during the development of folate deficiency in male Sprague-Dawley rats resulted in alterations in 2 of the parameters diagnostic of the individual conditions. Decreases in erythrocyte mean corpuscular volume (MCV) occur during lead poisoning, while increases occur during the development of folate deficiency. Significant reductions in mean corpuscular volume were observed in both the control + lead and in the low folate + lead groups. The increased mean corpuscular volume characteristic of folate deficiency was prevented by the concomitant lead exposure. Elevations in free erythrocyte protoporphyrin (FEP) levels are characteristic of lead intoxication; free erythrocyte protoporphyrin levels decline during folate deficiency. free erythrocyte protoporphyrin levels were significantly elevated only in the control + lead group. /Inorganic lead/ [Rader JI et al; Drug-Nutr Interact 1 (2): 131-42 (1983)] **PEER REVIEWED**

INTERACTIONS

Administration of ethanol (10% ad libitum in drinking water) had no effect on the toxicity of lead to rats as measured by urinary ALA excretion, renal weight, or lead concn in the kidneys, liver, or bones. /Lead cmpd/ [Mchaffey K; Environ Health Perspect 7: 107-13 (1974) as cited in WHO; Environ Health Criteria: Lead p.97 (1977)] **PEER REVIEWED**

INTERACTIONS

MINERALS SUCH AS CALCIUM, IRON, ZINC, COPPER, AND PHOSPHORUS INTERACT UITH LEAD & INFLUENCE ITS LEAD METABOLISM & TOXICITY IN MAMMALS. ... SUSCEPTIBILITY OF RATS TO LEAD INTOXICATION IS INCR ... BY LOU INTAKE OF CALCIUM ... TRANSFER ... TO NEWBORN & WEANLING PUPS INCR IN CALCIUM DEFICIENT RATS ... IRON DEFICIENCY MARKEDLY INCR ADVERSE EFFECTS OF LEAD. ... /Inorganic lead/ [Venugopal, B. and T.D. Luckey. Metal Toxicity in Mamnals, 2. New York: Plenum Press, 1978. , p. 1941 **PEER REVIEWED**

INTERACTIONS

LACTOSE HYDROLYZED MILK CONSUMPTION BY RATS FED LESS THAN 20 MUG PB/G DIET OVER 8 UK SHOWED TISSUE LEAD CONCN INCR IN RATS FED LOU CONCN OF LEAD & MILK SIMULTANEOUS. LACTOSE WAS APPARENTLY THE FACTOR IN MILK RESPONSIBLE FOR THIS INCR. /Inorganic lead/ [BELL RR ET AL; FOOD COSMET TOXICOL 19 (4): 429-36 (1981)] **PEER REVIEWED**

INTERACTIONS

Zinc given in diet with lead protected horses against toxic effects. ... Probably, this effect was not due to inhibition of lead absorption. Zinc supplementation actually caused an incr in the lead content of liver and kidney, but a decr in the Lead content of brain and bone. /Inorganic lead/ [Willoughby RA et al; Vet Rec 91: 382-383 (1972) as cited in WHO; Environ Health Criteria: Lead p.97 (1977)] **PEER REVIEWED**

INTERACTIONS

... Lead interferes ... with synaptic transmission in the peripheral nervous system and ... the effects can be reversed by calcium. /Lead cmpd/ [Kostial K, Vorik VB; Brit J Phermacol, Chemother 12: 219-22 (1957) as cited in WHO; Environ Health Criteria: Lead p.89 (1977)] **PEER REVIEWED**

NATURAL OCCURRING SWRCES

LEAD CHROMATE OCCURS IN NATURE AS THE MINERALS CROCOITE & PHOENICOCHROITE. IIARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer,

1972-PRESENT. (Multivolume work). V2 109 (1973)] **PEER REVIEWED**

ARTIFICIAL SOURCES

Lead producing industry (est releases to the environment): primary lead: 893 ton/yr (atmosphere), 200-500 ton/yr (waste water), 21,000 ton/yr (land); secondary lead: 750 ton/yr (atmosphere), 3,260 ton/yr (land); secondary brass and bronze: 47 ton/yr (atmosphere), 10 ton/yr (waste water), 460 ton/yr (land). Lead consuming industries (est releases to the environment): battery manufacture: 82 ton/yr (atmosphere), 1-340 ton/yr (waste water), 40 ton/yr (land) lead alkyl manufacture: 1000 ton/yr (atmosphere), 60 ton/yr (waste water); lead oxides and pigments: 112 ton/yr (atmosphere), 200 ton/yr (land); lead stabilizers: 40 ton/yr (atmosphere), 40 ton/yr (land); cable covering: 113 ton/yr (atmosphere); type metal: 435 ton/yr (atmosphere); can soldering: 60 ton/yr (atmosphere); ceramics: 600 ton/yr (atmosphere). Indirect sources (est releases of lead to the environment); gasoline distribution: 420 ton/yr (atmosphere); gasoline combustion: 122,000 ton/yr (atmosphere); waste oil disposal: 3,400 ton/yr (atmosphere), 4,600 ton/yr (land); coal combustion: 225 ton/yr (atmosphere), 4,275 ton/yr (land); oil combustion: 100 ton/yr (atmosphere); cement manufacture: 312 ton/yr (atmosphere); iron and steel manufacture: 605 ton/yr (atmosphere); grey iron products: 1,080 ton/yr (atmosphere); ferro alloy production; 30 ton/yr; solid waste incineration: 1,170 ton/yr (atmosphere); sludge disposal: 5 ton/yr (atmosphere), 2,400 ton/yr (land). /Lead and lead cmpds/ [USEPA; The Health and Environmental Impacts of Lead: p.122 (1979) EPA 560/2-79-001] **PEER REVIEWED**

ARTIFICIAL SOURCES

... Wastes generated by the ... zinc industry which are deposited on the land (slags, dust, sludge, and water treatment residues) resulted in an est of 1,010 metric tons of contained lead. /Inorganic lead/ [USEPA; The Health and Environmental Impacts of Lead: p.127 (1979) EPA 560/2-79-001] **PEER REVIEWED**

ARTIFICIAL SOURCES

Mining, smelting, and refining as well as the manufacture of lead containing cmpd and goods, give rise to lead pollution. /Inorganic lead/ [WHO; Environ Health Criteria: Lead p.34-5 (19771 **PEER REVIEWED**

ARTIFICIAL SOURCES

Coal fired power plants, ceramic manufacturing, and mine seepage. /Lead/ [Nat'l Research Council Canada: Effects of Lead in the Canadian Envir p.76 (1978) NRCC No.0316-01141 **PEER REVIEWED**

ENVIRONMENTAL FATE

Terrestrial Fate: Most soil chromium is in mineral, absorbed, or precipitated form. Chromium probably occurs as the insoluble Cr(III) oxide (Cr203.nH20) in soil, as the organic matter in soil is expected to reduce any soluble chromate to insoluble chromic oxide (Cr203). Chromiun in soil can be transported to the atmosphere by way of aerosol formation. Chromium is also transported from soil through runoff and leaching of water. Runoff could remove both chromiun ions and bulk precipitates of chromium, with final deposition on either a different land area or a water body. In addition, flooding of soils and the subsequent anaerobic decomposition of plant matter may increase dissolution of Cr(III) oxides in the soil. /Chromium/ [USEPA; Health Assessment Document: Chromium p.3-20 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

ENVIRONMENTAL FATE

Atmospheric Fate: Chromiun particles of aerodynamic equivalent diameter < 20 um may remain airborne for long periods and may be transported great distances by wind currents and diffusion forces. Therefore, atmospheric conditions play an important role in determining the chromiun concentration around emission sites. /Chromium/ [USEPA; Health Assessment Document: Chromium p.3-17 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

ENVIRONMENTAL FATE

Aquatic Fate: ... Most of the chromium in surface waters may be present in particulate form as sediment. Some of the particulate chromium would remain as suspended matter and

ultimately be deposited in sediments. ... The exact chemical forms of chromium in surface waters are not well defined. Although most of the soluble chromium in surface waters may be present as Cr(VI), a small amount may be present as Cr(III) organic complexes. Cr(VI) is the major stable form of chromium in seawater; however, Cr(VI) may be reduced to Cr(III) by organic matter present in water, and may eventually deposit in sediments. /Chromium/ [USEPA; Health Assessment Document: Chromiun p.3-18 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

ENVIRONMENTAL FATE

Aquatic Fate: Cr(VI) rarely occurs in nature, apart from anthropogenic sources, because it is readily reduced by oxidizable organic matter. However, after it is introduced into water, Cr(VI) frequently remains unchanged in many natural water sources because of a low concentration of reducing matter. /Hexavalent chromium/ [USEPA; Health Assessment Document: Chromiun p.3-5 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

ENVIRONMENTAL FATE

Atmospheric Fate: Under normal conditions, Cr(III) and Cr(0) are relatively unreactive in the atmosphere. Cr(VI) in air may react with particulate matter or gaseous pollutants to form Cr(III). However, these atmospheric reactions have not been extensively studied. ... Chromium is removed from air through wet and dry depositions. The total yearly deposition of chromium in urban areas may vary from 0.12 ug/sq m to 3 ug/sq m. In general, urban areas have higher total deposition than rural areas. Chromium concentration in a wet deposition may vary from 0.004 to 0.060 ug/ml and 0.0006 to 0.034 ug/l for urban and rural areas, respectively. The precipitated chromium from the air enters surface water or soil. /Chromium/ [USEPA; Health Assessment Document: Chromium p.3-17 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

ENVIRONMENTAL FATE

Aquatic Fate: Cr(VI) exists in solution as hydrochromate, chromate, and dichromate ionic species. The proportion of each ion in solution is dependent on pH. In strongly basic and neutral pHs, the chromate form predominates. As the pH is lowered, the hydrochromate concentration increases. At very low pHs, the dichromate species predominates. In the pH ranges encountered in natural water, the predominant forms are hydrochromate ions (63.6%) at pH 6.0 to 6.2 and chromate ion (95.7%) at pH 7.8 to 8.5. The oxidizing ability of Cr(VI) in aqueous solution is pH dependent. The oxidation potential of Cr(VI) increases at lower pHs. /Hexavalent chromium/ [USEPA; Health Assessment Document: Chromium p.3-1 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

ENVIRONMENTAL FATE

Terrestrial fate: Leaching of lead can be relatively rapid from some soils, especially at highly contaminated sites or landfills. /Inorganic lead/ [Kayser, R., D. Sterling, D. Viviani (eds.). Intermedia Priority Pollutant Guidance Documents. Washington, DC: U.S.Environmental Protection Agency, July 1982. 1-41 **PEER REVIEWED**

ENVIRONMENTAL FATE

Terrestrial Fate: Lead in soil is relatively imnobile and can persist for long periods of time, whether added to the soil as halides, hydroxides, oxides, carbonates or sulfates (from the combustion of automotive fuels or from industrial processes), as lead arsenate, insecticides, or as an impurity in fertilizers or a component of wastewater. /Inorganic lead/ [USEPA; The Health and Environmental Impacts of Lead: p.168 (1979) EPA 560/2-79-001] **PEER REVIEWED**

ENVIRONMENTAL FATE

Terrestrial fate: Soils represent the major sink for pollutant lead. The adsorption or precipitation of lead in soils is promoted by the presence of organic matter, carbonates, and phosphate minerals. Lead usually accumulates in topsoil due to complexation with organic matter and the transformation of soluble lead compounds to relatively insoluble sulfate or phosphate derivatives. The efficient fixation of lead by most soils greatly limits the transfer of lead to aquatic systems and also inhibits absorption of lead by plants. However,

leaching of lead can be relatively rapid from some soils, especially at highly contaminated sites or landfills. /Inorganic lead/ [Kayser, R., D. Sterling, D. Viviani (eds.). Intermedia Priority Pollutant Guidance Documents. Washington, DC: U.S.Environmental Protection Agency, July 1982. 1-41 **PEER REVIEWED**

SOIL ADSORPTION/MOBILITY

Lead in soil is relatively imnobile and can persist for long periods of time, whether added to the soil as halides, hydroxides, oxides, carbonates or sulfates (from the combustion of automotive fuels or from industrial processes), as Lead arsenate, insecticides, or as an impurity in fertilizers or a component of waste water. /Inorganic lead/ CUSEPA; The Health and Enviromental Impacts of Lead: p.168 (1979) EPA 560/2-79-001] **PEER REVIEWED**

SOIL ADSORPTION/MOBILITY

Lead in the soil has a limited mobility except when soluble organic complexes or when the soil lead (Pb) exchange capacity approaches saturation. Three processes; adsorption at mineral surfaces (or on hydrous iron oxides), formation of stable organic complexes, and precipitation of sparingly soluble Pb cmpd (sulfates, phosphates, carbonates), act to limit Pb availability to plants from the soil. Pb is most available from acidic sandy soils which contain little material capable of binding Pb. /Inorganic lead/ [Nat'l Research Council Canada; Effects of Lead in the Canadian Environment p.168 (1978) NRCC No. 16736] **PEER REVIEWED**

SOIL ADSORPTION/MOBILITY

... Lead (Pb) sorption capacity of investigated humus forms decreased in the order mull > moder > mor /soils/ /SRP: ie, increasing organic matter content/. Soil respiration was inhibited after addition of 10 mg Pb/g soil at Pb concn greater than 1 ug Pb/ml of soil soln of mor and moder profiles. Highly significant positive regression coefficients were obtained for decr in soil respiration and decr in dehydrogenase, phosphatase and arylsulfatase activities of 0-horizons. ... After additions of 200 mg Pb/g soil biological activities of investigated humus layers were also affected by a marked incr of acidity of soil soln. /Inorganic lead/ [Wilke B-M; Z Pflanzenernaehr Bodenkd 145(1): 52-65 (1982)] **PEER REVIEWED**

WATER CONCENTRATIONS

Median chromium concentrations in USA drinking waters (ug/l or ppb): tap water, Dallas, TX: 4.0 (1.0 to 20); 100 largest cities, US (1962): 0.4 (0.2 to 35); 380 finished waters, US (1962-1967): 7.5 (1.0 to 29); 3834 tap waters, US (1974-1975): 1.8 (0.4 to 8); 83 Midwestern cities, US: range of < 5.0 to 17.0; and 115 Canadian municipalities (1975-1977): < 2.0 (< 2.0 to 4.1). /Chromium/ CUSEPA; Health Assessment Document: Chromium p.3-27 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

WATER CONCENTRATIONS

Median dissolved chromium levels in Great Lakes waters: Lake Superior, 1.0 (0.2-18) ppb; Lake Huron, 1.6 (0.2-19) ppb; Lake Erie, 1.6 (0.2-14) ppb; and Lake Ontario, 0.7 (0.2-12) ppb. The detection limit for dissolved chromium is approximately 0.2 ppb. /Chromium/ [Nat'l Research Council Canada; Effects of Chromium in the Canadian Environment p.37 (1976) NRCC No. 150171 **PEER REVIEWED**

WATER CONCENTRATIONS

Natural chromium levels (total dissolved and undissolved chromium) in rainwater range from 2.0 to 3.6 ng/cu m. /Chromium/ [Nat'l Research Council Canada; Effects of Chromium in the Canadian Envirorment p.35 (1976) NRCC No. 150171 **PEER REVIEWED**

WATER CONCENTRATIONS

Frequency of dissolved chromium levels in Canadian streams and rivers: < 10 ppb, 4163 samples (95.9%); 10-14 ppb, 92 samples (2.12%); 15-24 ppb, 62 samples (1.43%); 25-49 ppb, 19 samples (0.44%); 50-99 ppb, 4 samples (0.092%); and 100-500 ppb, 2 samples (0.046%). /Chromium/ [Nat'l Research Council Canada; Effects of Chromium in the Canadian Environment p.35 (1976) NRCC No. 150171 **PEER REVIEWED**

EFFLUENTS CONCENTRATIONS

BECAUSE LEAD CHROMATE IS INSOL IN WATER, IT SEEMS UNLIKELY THAT

IT OCCURS IN SIGNIFICANT QUANTITIES IN WASTE STREAMS FROM PLANTS PRODUCING OR USING IT. IIARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-PRESENT. (Multivolume work). V2 109 (1973)] **PEER REVIEWED**

EFFLUENTS CONCENTRATIONS

Solid waste streams containing Cr(VI) constitute the primary problem area involving chromium solid wastes. Wastes resulting from the roasting and leaching steps in the chromate manufacturing process traditionally contain residual Cr(VI). If landfilled, the residual Cr(VI) can slowly leach into surrounding waters via desorption and disproportionation. An estimate of the total amount of chromium released into soil and groundwater as a result of the leaching of chromium-containing solid wastes is not available. /Hexavalent chromium/ CUSEPA; Health Assessment Document: Chromium p.3-17 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

SEDIMENT/SOIL CONCENTRATIONS

Chromium levels in soil vary with soil origin and degree of contamination from anthropogenic sources. Tests on domestic soil have shown avg chromium concentrations ranging from 14-70 ppm. /Chromium/ [USEPA; Health Assessment Document: Chromium p.2-3 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

SEDIMENT/SOIL CONCENTRATIONS

Chromium is present usually as Cr(III) in the soil and is characterized by its lack of mobility, except in cases where Cr(VI) is involved. Chromium(VI) of natural origin is rarely found. /Trivalent and hexavalent chromium/ [Nat'l Research Council Canada; Effects of Chromium in the Canadian Envirorment p.40 (1976) NRCC No. 150171 **PEER REVIEWED**

SEDIMENT/SOIL CONCENTRATIONS

Lead usually accumulates in topsoil due to complexation with organic matter and the transformation of soluble lead cmpd to relatively insoluble sulfate or phosphate derivatives. /Inorganic lead/ [Kayser, R., D. Sterling, D. Viviani (eds.). Intermedia Priority Pollutant Guidance Docunents. Washington, DC: U.S.Environmental Protection Agency, July 1982. 1-41 **PEER REVIEWED**

SEDIMENT/SOIL CONCENTRATIONS

... An avg range in soils from areas remote of human activity is 5-25 mg/kg. /Inorganic lead/ [Swaine DJ; Commonwealth Bur Soic Sci Technol Comnun p.48 (1955) as cited in WHO; Environ Health Criteria: Lead p.30 (1977)] **PEER REVIEWED**

ATMOSPHERIC CONCENTRATIONS

Background ambient air concentrations of total chromium have ranged from as Low as 0.005 ng/cu m (at the South Pole) to 1.1 ng/cu m in other remote areas of the world. In the United States, recent monitoring of the ambient air in many urban and non-urban areas has shown total chromium concentrations averaging in the range of approximately 0.005-0.157 ug/cu m. The maximum 24-hour average concentration found for any one site was 0.684 ug/cu m in the Baltimore, MD area. Because Cr(III) is highly stable and Cr(VI) reacts over time to form Cr(III), it is assumed that most chromium in ambient air occurs in the trivalent state. /Chromium/ [USEPA; Health Assessment Document: Chromium p.2-3 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

ATMOSPHERIC CONCENTRATIONS

Remote-sampling data from a number of investigators point to a world background chromium concn in lower atmosphere aerosols in the range of 0.3 to 2 ng/cu m, with an average near 1 ng/cu m. ... No reports /were found/ which describe the chemical forms of chromium in aerosol particulate matter or rainwater. /Chromium/ [Nat'l Research Council Canada; Effects of Chromium in the Canadian Environment p.33 (1976) NRCC No. 150171 **PEER REVIEWED**

ATMOSPHERIC CONCENTRATIONS

Urban stations concn of Pb: 1.1 ug/cu m, non-urban stations: 0.21 ug/cu m, remote areas: 0.02 ug/cu m. /Inorganic lead/ [McMullen TB et al; J Air Pollut Control Assoc 20 p.269 (1970) as cited in WHO; Environ Health Criteria: Lead p.45 (1977)] **PEER REVIEWED**

FOOD SURVEY VALUES

Background ambient air concentrations of total chromium have ranged from as low as 0.005 ng/cu m (at the South Pole) to 1.1 ng/cu m in other remote areas of the world. In the United States, recent monitoring of the ambient air in many urban and non-urban areas has shown total chromium concentrations averaging in the range of approximately 0.005-0.157 ug/cu m. The maximum 24-hour average concentration found for any one site was 0.684 ug/cu m in the Baltimore, MD area. Because Cr(III) is highly stable and Cr(VI) reacts over time to form Cr(III), it is assumed that most chromium in ambient air occurs in the trivalent state. /Chromium/ [USEPA; Health Assessment Document: Chromium p.2-3 (1984) EPA 600/8-83-014F] **QC REVIEWED**

MILK CONCENTRATIONS

... Lead is also excreted in human milk in concentrations ... up to 12 ug/l. [Friberg, L., Nordberg, G.F., Kessler, E. and Vouk, V.B. (eds). Handbook of the Toxicology of Metals. 2nd ed. Vols I, II.: Amsterdam: Elsevier Science Publishers B.V., 1986. V2 3141 **PEER REVIEWED**

PROBABLE ROUTES OF HUMAN EXPOSURE

The principal route of exposure is food, but it is usually environemtal & presumably controllable sources that produce excess exposure. ... These sources include ... lead in air from combustion of lead containing auto exhausts or industrial emissions, lead based paint, hand to mouth activities of young children living in polluted environments, &, less comnonly, lead dust brought home by industrial workers on their clothes & shoes, & lead glazed earthen ware. /Inorganic lead/ [Doull, J., C.D.Klassen, and M.D. Amdur (eds.). Casarett and Doull's Toxicology. 3rd ed., New York: Macmillan Co., Inc., 1986. , p. 5981 **PEER REVIEWED**

PROBABLE ROUTES OF HUMAN EXPOSURE

LEAD POISONING IN CHILDREN IS A FAIRLY COMMON RESULT OF THEIR INGESTION OF PAINT CHIPS FROM OLD BUILDINGS. PAINTS APPLIED TO DWELLINGS BEFORE WORLD WAR II, WHEN LEAD CARBONATE (WHITE) & LEAD OXIDE (RED) WERE COMMON CONSTITUENTS OF BOTH INTERIOR & EXTERIOR HOUSE PAINT, ARE PRIMARILY RESPONSIBLE IN SUCH PAINT, LEAD MAY CONSTITUTE 5 TO 40% OF DRIED SOLIDS. YOUNG CHILDREN ARE POISONED MOST OFTEN BY NIBBLING LEAD-PAINTED WINDOWSILLS & FRAMES. /INORGANIC LEAD/ [Gilman, A.G., L.S.Goodman, and A. Gilman. (eds.). Goodman and Gilman's The Pharmacological Basis of Therapeutics. 7th ed. New York: Macmillan Publishing Co., Inc., 1985. , p. 16061 **PEER REVIEWED**

PROBABLE ROUTES OF HUMAN EXPOSURE

Eating of lead paint by children and drinking of lead contaminated, distilled (moonshine) whiskey are important sources of non industrial poisoning. .. Exposure to burning battery castings, drinking of liquids from improperly fired, lead glazed containers, and high levels of airborne lead. [NIOSH; Criteria Document: Inorganic Lead p.III-3 (1978) DHEW Pub. NIOSH 78-1581 **PEER REVIEWED**

PROBABLE ROUTES OF HUMAN EXPOSURE

Poor work hygiene, smoking during work (pollution of tobacco; polluted fingers while smoking), poor personal hygiene may considerably increase total exposure mainly by the oral route. /Inorganic lead/ [International Labour Office. Encyclopedia of Occupational Health and Safety. Vols. I&II. Geneva, Switzerland: International Labour Office, 1983. , p. 12013 **PEER REVIEWED**

PROBABLE ROUTES OF HUMAN EXPOSURE

Ingestion of dust, inhalation of dust or fune, skin and eye contact. /Inorganic lead compounds/ [Sittig, M. Handbook of Toxic and Hazardous Chemicals and Carcinogens, 1985. 2nd ed. Park Ridge, NJ: Noyes Data Corporation, 1985. , p. 5441 **PEER REVIEWED**

PROBABLE ROUTES OF HUMAN EXPOSURE

... Man absorbs lead in small amounts, which normally does not cause poisoning, from food, water, and air. /Inorganic Lead/ [NIOSH; Criteria Docunent: Inorganic Lead p.III-3 (1978) DHEW Pub. NIOSH 78-1581 **PEER REVIEWED**

PROBABLE ROUTES OF HUMAN EXPOSURE

The toxicologically important routes of entry are inhalation,

FOOD SURVEY VALUES

Background ambient air concentrations of total chromium have ranged from as low as 0.005 ng/cu m (at the South Pole) to 1.1 ng/cu m in other remote areas of the world. In the United States, recent monitoring of the ambient air in many urban and non-urban areas has shown total chromium concentrations averaging in the range of approximately 0.005-0.157 ug/cu m. The maximum 24-hour average concentration found for any one site was 0.684 ug/cu m in the Baltimore, MD area. Because Cr(III) is highly stable and Cr(VI) reacts over time to form Cr(III), it is assumed that most chromium in ambient air occurs in the trivalent state. /Chromium/ [USEPA; Health Assessment Document: Chromium p.2-3 (1984) EPA 600/8-83-014F] **QC REVIEWED**

MILK CONCENTRATIONS

... Lead is also excreted in hman milk in concentrations ... up to 12 ug/l. [Friberg, L., Nordberg, G.F., Kessler, E. and Vouk, V.B. (eds). Handbook of the Toxicology of Metals. 2nd ed. Vols I, II.: Amsterdam: Elsevier Science Publishers B.V., 1986. V2 3141 **PEER REVIEWED**

PROBABLE ROUTES OF HUMAN EXPOSURE

The principal route of exposure is food, but it is usually environemtal & presumably controllable sources that produce excess exposure. ... These sources include ... lead in air from combustion of lead containing auto exhausts or industrial emissions, lead based paint, hand to mouth activities of young children living in polluted environments, &, less comnonly, lead dust brought home by industrial workers on their clothes & shoes, & lead glazed earthen ware, /Inorganic lead/ [Doull, J., C.D.Klassen, and M.D. Amdur (eds.). Casarett and Doull's Toxicology. 3rd ed., New York: Macmillan Co., Inc., 1986. , p. 5981 **PEER REVIEWED**

PROBABLE ROUTES OF HUMAN EXPOSURE

LEAD POISONING IN CHILDREN IS A FAIRLY COMMON RESULT OF THEIR INGESTION OF PAINT CHIPS FROM OLD BUILDINGS. PAINTS APPLIED TO DWELLINGS BEFORE WORLD WAR 11, WHEN LEAD CARBONATE (WHITE) & LEAD OXIDE (RED) WERE COMMON CONSTITUENTS OF BOTH INTERIOR & EXTERIOR HOUSE PAINT, ARE PRIMARILY RESPONSIBLE IN SUCH PAINT, LEAD MAY CONSTITUTE 5 TO 40% OF DRIED SOLIDS. YOUNG CHILDREN ARE POISONED MOST OFTEN BY NIBBLING LEAD-PAINTED WINDOWSILLS & FRAMES. /INORGANIC LEAD/ [Gilman, A.G., L.S.Goodman, and A. Gilman. (eds.). Goodman and Gilman's The Pharmacological Basis of Therapeutics. 7th ed. New York: Macmillan Publishing Co., Inc., 1985. , p. 16061 **PEER REVIEWED**

PROBABLE ROUTES OF HUMAN EXPOSURE

Eating of lead paint by children and drinking of lead contaminated, distilled (moonshine) whiskey are important sources of non industrial poisoning. ... Exposure to burning battery castings, drinking of liquids from improperly fired, lead glazed containers, and high levels of airborne lead. [NIOSH; Criteria Document: Inorganic Lead p.III-3 (1978) DHEW Pub. NIOSH 78-158] **PEER REVIEWED**

PROBABLE RWTES OF HUMAN EXPOSURE

Poor work hygiene, smoking during work (pollution of tobacco; polluted fingers while smoking), poor personal hygiene may considerably increase total exposure mainly by the oral route. /Inorganic Lead/ [International Labour Office. Encyclopedia of Occupational Health and Safety. Vols. I&II. Geneva, Switzerland: International Labour Office, 1983. , p. 12011 **PEER REVIEWED**

PROBABLE ROUTES OF HUMAN EXPOSURE

Ingestion of dust, inhalation of dust or fune, skin and eye contact. /Inorganic lead compounds/ [Sittig, M. Handbook of Toxic and Hazardous Chemicals and Carcinogens, 1985. 2nd ed. Park Ridge, NJ: Noyes Data Corporation, 1985. , p. 5441 **PEER REVIEWED**

PROBABLE ROUTES OF HUMAN EXPOSURE

... Man absorbs lead in small amounts, which normally does not cause poisoning, from food, water, and air. /Inorganic lead/ CNIOSH; Criteria Document: Inorganic Lead p.III-3 (1978) DHEW Pub. NIOSH 78-158] **PEER REVIEWED**

PROBABLE ROUTES OF HUMAN EXPOSURE

The toxicologically important routes of entry are inhalation,

ingestion and skin and/or eye contact. [Mackison, F. W., R. S. Stricoff, and L. J. Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) Publication No. 81-123 (3 VOLS). Washington, DC: U.S. Government Printing Office, Jan. 1981. , p. 691 **PEER REVIEWED**

PROBABLE EXPOSURES

... Many of the highly colored chromate salts of various metals are used in the pigment, paint, tanning and dyeing industries. /Chromate salts/ IIARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-PRESENT. (Multivolume work). V23 243 (1980)] **PEER REVIEWED**

PROBABLE EXPOSURES

1) Use in metal finishing, in chrome plating, anodizing, conversion coatings, and for corrosion resistance; 2) use in leather finishing for shoe uppers, glove, and garment leathers, and bag leather; 3) use as corrosion inhibitors in radiator coolants, internal combustion and gas turbine engines, refrigerator and air conditioning systems, and water-cooled nuclear reactors; 4) use in photoreproduction processes, as sensitizing agents for photoengraving, photography, lithography, and blueprinting; 5) use as corrosion-inhibiting and coloring pigments, artists' colors, jointing pastes, inks, rubber, ceramics, and color blending; 6) use in dyeing of fur, leather, fabrics, wool, and nylon; oxidizing of dyes; after treating on cotton, and in textile and paper printing; use in manufacture of glue used in shoes, furniture, and packaging; 7) use as fungicides; use in aqueous preservatives and fire retardants for uood; for protection of textiles and seed; 8) use in battery manufacture to incr shelf life; to provide corrosion resistance, and for battery depolarization; 9) use in manufacture of safety matches and explosives; 10) use as a chemical reagent, oxidizing agent, catalyst, indicator, in bleaching of fats, oils, and waxes, in chemical synthesis, and in analytical chemistry; 11) use in manufacture and packing of cement. /Chromic acid and chromates/ [Mackison, F. W., R. S. Stricoff, and L. J. Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) Publication No. 81-123 (3 VOLS). Washington, DC: U.S. Government Printing Office, Jan. 1981. , p. 41 **PEER REVIEWED**

PROBABLE EXPOSURES

Nearly all environmental exposure to lead is to inorganic compounds, even as lead in food. /Inorganic lead/ [Doull, J., C.D.Klassen, and M.D. Amdur (eds.). Casarett and Doull's Toxicology. 3rd ed., New York: Macmillan Co., Inc., 1986. , p. 5991 **PEER REVIEWED**

PROBABLE EXPOSURES

Exposures to lead dust may occur during mining, smelting, and refining and to fume, during high temp (above 500 deg C) operations such as welding or spray coating of metals with molten lead. There are numerous applications for lead compounds, some of the more common being in the plates of electric batteries & accumulators, as compounding agents in rubber mfr, as ingredients in paints, glazes, glass, pigments, & in the chemical industry. It is est that approximately 783,000 industrial workers are potentially exposed to lead products. ... The level of exposure resulting from contact is highly variable. Children with pica for paint chips or for soil may experience elevation in blood lead ranging from marginal to sufficiently great to cause clinical illness. Certain adults may also be exposed to hazardous concn of lead in the workplace, notably in lead smelters and storage battery manufacturing plants. Again the range of exposure is highly variable. /Inorganic lead/ [Sittig, M. Handbook of Toxic and Hazardous Chemicals and Carcinogens, 1985. 2nd ed. Park Ridge, NJ: Noyes Data Corporation, 1985. , p. 5431 **PEER REVIEWED**

PROBABLE EXPOSURES

... OCCUPATIONS IN WHICH LEAD POISONING IS INVOLVED INCLUDE "THE USE OR HANDLING OF OR EXPOSURE TO FUME, DUST OR VAPOR OF LEAD OR COMPD OF LEAD, OR SUBSTANCE CONTAINING LEAD"...

/INORGANIC LEAD/ [Browning, E. Toxicity of Industrial Metals. 2nd ed. New York: Appleton-Century-Crofts, 1969. , p. 1771 **PEER REVIEWED**

PROBABLE EXPOSURES

STORAGE BATTERY MANUFACTURE HEADS THE LIST OF THE DANGEROUS LEAD TRADES IN SOME COUNTRIES &, ALTHOUGH THERE ARE NO RELIABLE STATISTICS, IT IS PROBABLY ONE OF THE MOST PROLIFIC SOURCES OF PLUMBISM IN THE UNITED STATES. /INORGANIC LEAD/ [Hamilton, A., and H. L. Hardy. Industrial Toxicology. 3rd ed. Acton, Mass.: Publishing Sciences Group, Inc., 1974. , p. 901 **PEER REVIEWED**

PROBABLE EXPOSURES

Nonoccupational sources of lead poisoning include battery burning, bullet retention, ceramic making, eating from unfired pottery, cooking in leaden pots, home-distilled wine/whiskey, home abortifacients, target shooting, ingestion of lead-containig herbal medicines, use of lead-containing cosmetics, & soldering. /Inorganic lead; from table/ [Ellenhorn, M.J. and D.G. Barceloux. Medical Toxicology - Diagnosis and Treatment of Human Poisoning. New York, NY: Elsevier Science Publishing Co., Inc. 1988. , p. 10301 **PEER REVIEWED**

PROBABLE EXPOSURES

Sources of lead in a child's environment include house dust (new inner-city homes, 2 to 24 ug/sq ft floor surface; contaminated sources: old inner-city homes, 33 to 486 ug/sqq ft floor surface, mean 11,000 ug/sq g; homes of lead smelter workers, mean 2,687 ug/g; homes within 1.6 km of smelter, mean 22,191 ug/g; & homes located 1.7 to 3.2 km from smelter, mean 2,124 ug/g). /Inorganic lead; from table/ [Ellenhorn, M.J. and D.G. Barceloux. Medical Toxicology - Diagnosis and Treatment of Human Poisoning. New York, NY: Elsevier Science Publishing Co., Inc. 1988. , p. 10311 **PEER REVIEWED**

PROBABLE EXPOSURES

... SEVERE MANIFESTATIONS, ESPECIALLY ENCEPHALOPATHY, ARE PRACTICALLY NEVER ENCOUNTERED IN INDUSTRY ... OCCUPATIONS IN WHICH LEAD POISONING IS INVOLVED INCLUDE "THE USE OR HANDLING OF OR EXPOSURE TO FUME, DUST OR VAPOR OF LEAD OR CMPD OF LEAD, OR SUBSTANCE CONTAINING LEAD"... /INRORGANIC LEAD/ [Browning, E. Toxicity of Industrial Metals. 2nd ed. New York: Appleton-Century-Crofts, 1969. , p. 177] **PEER REVIEWED**

PROBABLE EXPOSURES

EPIDEMIOLOGIC STUDIES INDICATE THAT CHROMATE IS CARCINOGEN WITH BRONCHOGENIC CARCINOMA AS PRINCIPAL LESION. LATENT PERIOD ... 10-15 YR. RELATIVE RISK OF CHROMATE PLANT WORKERS FOR RESP CANCER IS 20 TIMES GREATER THAN THAT OF GENERAL POPULATION. EXPTL STUDIES ... SUGGESTED THAT CALCIUM CHROMATE MAY BE SPECIFIC ... AGENT. [Doull, J., C.D. Klaassen, and M. D. Amdur (eds.). Casarett and Doull's Toxicology. 2nd ed. New York: Macmillan Publishing Co., 1980. , p. 4421 **PEER REVIEWED**

BODY BURDENS

In hman subjects with low level exposure, about 90% of the total body burden is found in bone. .../It was/ found that in the United Kingdom the lead concentration in bones of men & women over 16 yr of age ranged from 9 to 34 mg/kg wet wt. The concn in liver was about 1 mg/kg; & in the kidney, 0.8 mg/kg in the cortex & 0.5 mg/kg in the medulla. Studies in the United States & Japan revealed similar concentrations & distribution patterns. A comparatively high concentration (2.5 mg/kg wet wt) was found in the aorta with atheroma. Also, /it is/ reported that the mean lead concentration in the brain cortex of 50 adult males did not exceed 0.1 mg/kg wet wt with a range of individual values from 0.02 to 0.8 mg/kg; in the basal ganglia the range was 0.04 to 0.2 mg/kg. According to the review ... /of/ literature of accumulation & distribution of lead in human tissues, there are no marked trends of either increase or decrease of avg lead body burden in human adults. /Inorganic lead/ [Friberg, L., Nordberg, G.F., Kessler, E. and Vouk, V.B. (eds). Handbook of the Toxicology of Metals. 2nd ed. Vols I, II.: Amsterdam: Elsevier Science Publishers B.V., 1986. V2 3131 **PEER REVIEWED**

BODY BURDENS

... THERE IS FAIRLY GOOD CORRELATION BETUEEN DEGREE OF LEAD

INTOXICATION & BODY BURDEN OF LEAD, MAIN EXCEPTION BEING WHERE THERE HAS BEEN HIGH EXPOSURE OVER SHORT PERIOD. /INORGANIC LEAD/ [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-PRESENT. (Multivolune work). V1 47 (1972)] **PEER REVIEWED**

BODY BURDENS

Body burden of lead increases from birth to old age. /Inorganic lead/ [Barry PSI; Brit J Ind Med 32: 119-39 (1975) as cited in WHO; Environ Health Criteria: Lead p.81 (1977)] **PEER REVIEWED**

BODY BURDENS

The body burden of the average adult in the USA is reported to be not less than 100 mg and not more than 300 mg. Ninety five percent of the total body burden is stored in bone. /Inorganic lead/ [Committee on Biological Effects of Atmospheric Pollutants, Lead, National Academy of Sciences, washington, DC (1972)] **PEER REVIEWED**

BODY BURDENS

Body burden of lead, as assessed by lead (Pb) excretion 24 hr after Ca-EDTA administration, was increased in 37% of the workers with a mean value of 607 + or - 425 ug. Mean blood Pb was 32 + or - 14 ug/dl. Creatinine clearance was normal in all workers. Maximal urinary concentrating ability was abnormal in a significant fraction (52%) of the men. /Inorganic lead/ [Greenberg A et al; Arch Environ Health 41 (2): 69-76 (1986)] **PEER REVIEWED**

BODY BURDENS

... Lead is also excreted in human milk in concentrations ... up to 12 ug/l. [Friberg, L., Nordberg, G.F., Kessler, E. and Vouk, V.B. (eds). Handbook of the Toxicology of Metals. 2nd ed. Vols I, 11.: Amsterdam: Elsevier Science Publishers B.V., 1986. V2 3141 **PEER REVIEWED**

IMMEDIATELY DANGEROUS TO LIFE OR HEALTH

NIOSH considers all chromic acid and chromates (CrO3) compounds as potential carcinogens. /Chromic acid and chromates (CrO3) compounds/ [NIOSH. NIOSH Pocket Guide to Chemical Hazards. DHHS(NIOSH) Publication No. 90-117. Washington, DC: U.S. Government Printing Office, June 1990 , p. 2331 **QC REVIEWED**

IMMEDIATELY DANGEROUS TO LIFE OR HEALTH

30 mg/cu m /Chromic acid and chromates (CrO3) compounds/ [NIOSH. NIOSH Pocket Guide to Chemical Hazards. DHHS(NIOSH) Publication No. 90-117. Washington, DC: U.S. Government Printing Office, June 1990 , p. 70] **QC REVIEWED**

ACCEPTABLE DAILY INTAKES

Acceptable Daily Intake (ADI) value of 0.175 mg/day/man for Cr(VI). /Hexavalent chromium/ [USEPA; Health Assessment Document: Chromium p.7-161 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

ACCEPTABLE DAILY INTAKES

Estimated Adequate and Safe Intake (EASI) levels for chromium: Infants: 1) age 0.0 to 0.5 yr: 0.01 to 0.04 mg/day, 2) age 0.5 to 1.0 yr: 0.02 to 0.06 mg/day; Children: 1) age 1 to 3 yr: 0.02 to 0.08 mg/day, 2) age 4 to 6 yr: 0.03 to 0.12 mg/day, 3) age 7 to 10 yr: 0.05 to 0.20 mg/day, and 4) age > or = to 11 yr: 0.05 to 0.20 mg/day; and Adults: 0.05 to 0.20 mg/day. /Chromium, from table/ [NAS; Recommended Dietary Allowances (1980) as cited in USEPA; Health Assessment Document: Chromium p.6-2 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

ACCEPTABLE DAILY INTAKES

Tolerable intake of lead for preschool children should be less than the 3 mg/wk recommended provisionally for adults. ... /Inorganic lead/ [WHO; Environ Health Criteria: Lead p.127 (1977)] **PEER REVIEWED**

OSHA STANDARDS

OSHA Permissible Exposure Limit: 50 ug/cu m, 8 hr Time-Weighted Average. **/Fumes** and dusts, as Pb/ [29 CFR 1910.1025 (7/1/87)] **PEER REVIEWED**

OSHA STANDARDS

Meets criteria for OSHA medical records rule. /Inorganic lead/ [29 CFR 1910.20 (7/1/87)] **PEER REVIEWED**

OSHA STANDARDS

OSHA Permissible Exposure Limit: 1 mg/10 cu m (100 ug/cu m

ceiling) /Chromium (VI)/ [NIOSH/CDC. NIOSH Recomnendations for Occupational Safety and Health Standards Sept. 1986. (Supplement to Morbidity and Mortality Weekly Report 35 No. 15, Sept. 26, 1986) 10S] **PEER REVIEWED**

NIOSH RECOMMENDATIONS

NIOSH Recomnended Exposure Limit: <100 ug/cu m Time-Weighted Average; air level to be maintained so that worker blood lead remains less than or equal to 60 ug/100 g. Recomnendations are based on exposures up to 10 hr. /Inorganic lead/ [NIOSH/CDC. NIOSH Recomnendations for Occupational Safety and Health Standards Sept. 1986. (Supplement to Morbidity and Mortality Weekly Report 35 No. 15, Sept. 26, 1986) 21S] **PEER REVIEWED**

NIOSH RECOMMENDATIONS

NIOSH recommends that /chromium (VI) be treated as a potential human carcinogen /Chromium (VI)/ [NIOSH/CDC. NIOSH Recomnendations for Occupational Safety and Health Standards Sept. 1986. (Supplement to Morbidity and Mortality Weekly Report 35 No. 15, Sept. 26, 1986) 10S] **PEER REVIEWED**

THRESHOLD LIMIT VALUES

Time Weighted Avg (TWA) 0.012 mg/cu m, as Cr (1991) [American Conference of Governmental Industrial Hygienists. Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices for 1992-1993. Cincinnati, OH: ACGIH, 1992. , p. 251 **QC REVIEWED**

THRESHOLD LIMIT VALUES

A2, as Cr. A2= Suspected human carcinogen (1991) [American Conference of Governmental Industrial Hygienists. Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices for 1992-1993. Cincinnati, OH: ACGIH, 1992. , p. 251 **QC REVIEWED**

THRESHOLD LIMIT VALUES

A2, as Pb. A2= Suspected human carcinogen. [American Conference of Governmental Industrial Hygienists. Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices for 1992-1993. Cincinnati, OH: ACGIH, 1992. , p. 251 **QC REVIEWED**

THRESHOLD LIMIT VALUES

Time Weighted Avg (TWA) 0.05 mg/cu m as Pb (1991) [American Conference of Governmental Industrial Hygienists. Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices for 1992-1993. Cincinnati, OH: ACGIH, 1992. , p. 251 **QC REVIEWED**

WATER STANDARDS

The procedures described in the "Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses" indicate that, except possibly where a locally important species is very sensitive, 1) freshwater aquatic organisms and their uses should not be affected unacceptably if the four-day average concentration of chromium(VI) does not exceed 11 ug/l more than once every three years, on the average, and if the one-hour average concentration does not exceed 16 ug/l more than once every three years, on the average; 2) saltwater aquatic organisms and their uses should not be affected unacceptably if the four-day average Concentration of chromium(VI) does not exceed 50 ug/l more than once every three years, on the average and if the one-hour average concentration does not exceed 1,100 ug/l more than once every three years, on the average. Data suggest that the acute toxicity of chromium(VI) is salinity-dependent; therefore, the one-hour average concentration might be underprotective at low salinities. /Hexavalent chromium/ CUSEPA; Ambient Water Quality Criteria Doc: Chromium p.19-20 (1984) EPA 440/5-84-029] **PEER REVIEWED**

WATER STANDARDS

Toxic pollutant designated pursuant to section 307(a)(1) of the Clean Water Act and is subject to effluent limitations. /Lead and inorganic and organic lead cmpds/ C40 CFR 401.15 (7/1/87)] **PEER REVIEWED**

WATER STANDARDS

The maximum contaminant level (MCL) of lead is 0.05 mg/l. /Lead as Pb ion/ C40 CFR 141.11 (7/1/87)] **PEER REVIEWED**

ATMOSPHERIC STANDARDS

National primary and secondary ambient air quality standard for

lead and its compounds, measured as elemental lead is: 1.5 ug/cu m, maximum arithmetic mean averaged over a calendar quarter. /Lead and its compounds, as Pb/ [40 CFR 50.12 (7/1/87)] **PEER REVIEWED**

RCRA REQUIREMENTS

A solid waste containing lead chromate (lead ion and/or chromium ion) may become characterized as a hazardous waste when subjected to the Toxicant Extraction Procedure listed in 40 CFR 261.24, and if so characterized, must be managed as a hazardous waste. [40 CFR 261.24 (7/1/87)] **PEER REVIEWED**

RCRA REQUIREMENTS

The Environmental Protection Agency is ... /promulgated/ regulations concerning ground-water monitoring with regard to screening suspected contamination at land based hazardous waste treatment, storage, and disposal facilities. ... /There are/ new requirements to analyze for a specified core list of chemicals plus those chemicals specified by the Regional Administrator on a site-specific basis. ... /Total lead (all species) is included on this list./ /Total lead (all species)/ [52 FR 25942 (7/9/87)] **PEER REVIEWED**

FDA REQUIREMENTS

The FDA action level of lead is 7.0 ug/ml of leaching soln for pottery (ceramics) flatware (avg of 6 units); 5.0 ug/ml of leaching soln for small hollowware (any one of 6 units); 2.5 ug/ml of leaching soln for large hollowware (any one of 6 units); 7.0 ug/ml of leaching soln if product intended for use by adults for silver-plated hollowware (avg of 6 units); and 0.5 ug/ml of leaching soln if product intended for use by infants and children for silver-plated hollowware (one or more of 6 units). /Inorganic lead/ [FDA; Action Levels for Poisonous or Deleterious Substances in Human Food and Animal Feed p.9 (1982)] **PEER REVIEWED**

FDA REQUIREMENTS

Bottled water shall, when a composite of analytical units of equal volume from a sample is examined by the methods described in paragraph (d)(1)(ii) of this section, meet the standards of chemical quality and shall not contain lead in excess of 0.05 mg/l. /Lead as Pb ion/ [21 CFR 103.35 (4/1/88)] **PEER REVIEWED**

SAMPLING PROCEDURES

For expressing aquatic life criteria for chromium ... acid-soluble chromium(III) (operationally defined as the chromium(III) that passes through a 0.45 um membrane filter after the sample is acidified to pH □ 1.5 to 2.0 with nitric acid) and acid-soluble chromium(VI) are probably the best measurements at the present. /Chromium and chromium cmpd/ [USEPA; Ambient Water Quality Criteria Doc: Chromium p.1 (1984) EPA 440/5-84-029] **PEER REVIEWED**

SAMPLING PROCEDURES

Measurements to determine employee ceiling exposure are best taken during periods of maximum expected airborne concentrations of chromic acid or chromates. Each measurement should consist of a fifteen minute sample or series of consecutive samples totalling fifteen minutes in the employee's breathing zone (air that would most nearly represent that inhaled by the employee). A minimum of three measurements should be taken on one work shift with the highest level of all measurements taken as an estimate of the employee's exposure. /Chromic acid and chromates/ [Mackison, F. W., R. S. Stricoff, and L. J. Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) Publication No. 81-123 (3 VOLS). Washington, DC: U.S. Government Printing Office, Jan. 1981. , p. 31 **PEER REVIEWED**

SAMPLING PROCEDURES

Techniques for monitoring hexavalent chromium are subject to considerable error. For example, although the OSHA colorimetric method is the most commonly used analytical tool, particularly in occupational settings, low sample recoveries have been reported in chromium levels of less than 10 ug. /Hexavalent chromium/ [USEPA; Health Assessment Document: Chromium p.2-4 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

SAMPLING PROCEDURES

Analyte: Chromium; Specimen: urine; Vol: 50-200 ml in

polyethylene bottle; Preservative: 5 ml conc HNO3 added after collection; Controls: collect at least 3 urine specimens from unexposed workers; Stability: not established /Metals in urine, Chromium/ [U.S. Department of Health and Hunan Services, Public Health Service. Centers for Disease Control, National Institute for Occupational Safety and Health. NIOSH Manual of Analytical Methods, 3rd ed. Volumes 1 and 2 with 1985 supplement, and revisions. Washington, DC: U.S. Government Printing Office, February 1984. V1 8310-11 **PEER REVIEWED**

SAMPLING PROCEDURES

Analyte: Chromium; Specimen: Blood or tissue; Vol: 10 ml blood, or 1 g tissue; Preservative: Heparin for blood, none for tissue; Controls: collect 3 blood specimens from unexposed workers; Stability: not established /Elements in blood or tissue, Chromium/ [U.S. Department of Health and Human Services, Public Health Service. Centers for Disease Control, National Institute for Occupational Safety and Health. NIOSH Manual of Analytical Methods, 3rd ed. Volumes 1 and 2 with 1985 supplement, and revisions. washington, DC: U.S. Government Printing Office, February 1984. V1 8005-11 **PEER REVIEWED**

SAMPLING PROCEDURES

Analyte: Lead; Matrix: Air; Sampler: Filter (0.8-um cellulose ester membrane); Flow rate: 1 to 4 l/min; Vol: min: 200 l GI 0.05 mg/cu m, max: 1200 l; Stability: stable. /Lead and lead cmpd/ [U.S. Department of Health and Human Services, Public Health Service. Centers for Disease Control, National Institute for Occupational Safety and Health. NIOSH Manual of Analytical Methods, 3rd ed. Volumes 1 and 2 with 1985 supplement, and revisions. Washington, DC: U.S. Government Printing Office, February 1984. V2 7082-11 **PEER REVIEWED**

SAMPLING PROCEDURES

Analyte: Lead; Specimen: Blood or tissue; Vol: 10 ml blood, or 1 g tissue; Preservative: Heparin for blood, none for tissue; Controls: collect 3 blood specimens from unexposed workers; Stability: not established. /Lead and lead cmpd/ [U.S. Department of Health and Human Services, Public Health Service. Centers for Disease Control, National Institute for Occupational Safety and Health. NIOSH Manual of Analytical Methods, 3rd ed. Volumes 1 and 2 with 1985 supplement, and revisions. Washington, DC: U.S. Government Printing Office, February 1984. V1 8005-11 **PEER REVIEWED**

SAMPLING PROCEDURES

Analyte: Lead; Specimen: whole blood, urine; Preservative: (urine) 0.2 ml conc nitric acid; Anticoagulant: (blood) heparin; Controls: commercial urine and blood lead control samples plus pooled urine and blood from non-exposed populations; Stability: (blood) 3 days @ 4 deg C, indefinitely if sonicated and frozen in plastic tubes, (urine) indefinitely if kept acidified with HNO3. /Lead and lead cmpd/ [U.S. Department of Health and Human Services, Public Health Service. Centers for Disease Control, National Institute for Occupational Safety and Health. NIOSH Manual of Analytical Methods, 3rd ed. Volumes 1 and 2 with 1985 supplement, and revisions. Washington, DC: U.S. Government Printing Office, February 1984. V1 8003-11 **PEER REVIEWED**

SAMPLING PROCEDURES

Lead dust or fume is collected on 0.45 micrometer cellulose membrane filters mounted in either 2 or 3 piece filter cassettes. Air is drawn through the filter by means of a pump at a rate of 2 l/min (not less than 1 nor more than 4 l/min). A minimum sample of 100 liters shall be collected. Larger sample volumes are encouraged, provided the filters do not become , loaded with dust to the point that loose material would fall off or the filters would become clogged. With each group of samples, one filter, labeled as a blank, shall be submitted and no air shall be drawn through this filter. /Lead and lead cmpds/ CNIOSH; Criteria Document: Inorganic Lead p.VII-1 (1978) DHEW Pub. NIOSH 78-1581 **PEER REVIEWED**

SAMPLING PROCEDURES

TWO AUTOMATIC SAMPLING APPARATUS BASED ON THE BETA RAY ABSORPTION AND ON THE TYNDALL EFFECTS ARE DESCRIBED. (204)PB DUSTS WERE MEASURED. /LEAD AND LEAD CMPDS/ [CECCHETTI G ET AL; ANN INST SUPER SANITA 13 (1-2): 103 (1977)] **PEER REVIEWED**

ANALYTIC LABORATORY METHODS
ZINC, ZINC OXIDE, LEAD, THE 4 KNOWN ZINC CHROMATES, & RHOMBIC & MONOCLINIC LEAD CHROMATE WERE IDENTIFIED IN 0.3-0.5 MG PAINT SAMPLES BY X-RAY DIFFRACTOMETRY. THE ZINC CHROMATES WERE IDENTIFIABLE AT 7.0-9.3 ANGSTROMS. THE METHOD COULD BE USED TO IDENTIFY, BUT NOT TO QUANTITATE ZINC CHROMATES ON FILTERS OF AIR SAMPLES FROM AREAS USED FOR SPRAY PAINTING. CALTIERI A ET AL; ANN IST SUPER SANITA 13 (1-2): 315 (1977)] **PEER REVIEWED**

ANALYTIC LABORATORY METHODS
AT PRESENT THE 3 MOST SENSITIVE METHODS /FOR DETERMINING CHROMIUM/ ARE: GAS CHROMATOGRAPHY; SPARK SOURCE MASS SPECTROSCOPY; & GRAPHITE FURNACE ATOMIC ABSORPTION METHOD. /TOTAL CHROMIUM/ [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-PRESENT. (Multivolume work). V2 107 (1973)] **PEER REVIEWED**

ANALYTIC LABORATORY METHODS
Particulate filter collection; sulfuric acid and diphenylcarbazide work-up; colorimetric spectroscopy analysis. /Total chromic acid and chromates/ INIOSH. Pocket Guide to Chemical Hazards. 5th Printing/Revision. DHHS (NIOSH) Publ. No. 85-114. Washington, D.C.: U.S. Dept. of Health and Human Services, NIOSH/Supt. of Documents, GPO, Sept. 1985. , p. 821 **PEER REVIEWED**

ANALYTIC LABORATORY METHODS
Several methods are available for measuring elemental chromiun in ... environmental ... samples. These include atomic absorption spectroscopy, instrumental neutron activation analysis, X-ray fluorescence, and particle-induced X-ray emissions (PIXE). While these methods are sensitive to the ppb level, problems in sample collection, preparation and interferences are shared by all. ... In environmental samples, neutron activation analysis data are higher. Generally, a comparison of the results indicates that modified atomic absorption spectroscopy provides relatively reliable analyses. Another problem in chromium determination is the lack of adequate reference materials. Ideally, reference materials should match the samples to be analyzed with respect to chromium levels and each reference composition. Because the materials are not yet standardized, inter-laboratory comparisons are difficult. /Total chromium/ IUSEPA; Health Assessment Document: Chromium p.2-4 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

ANALYTIC LABORATORY METHODS
Air: Analysis by atomic absorption spectroscopy using an air acetylene flame. The method has a typical range of 0.07 - 7.5 ug lead/cu m. Water: atomic absorption spectroscopy, optimum concn range for the 283.3 nm absorption is 1-20 mg/l with an estimated detection limit of 0.1 mg/l. /Total lead/ IUSEPA; Intermedia Priority Pollutant Guidance Docunent: Lead p.9-1 (1982)] **PEER REVIEWED**

ANALYTIC LABORATORY METHODS
Analysis of lead in water is typically accomplished with atomic absorption and emission spectroscopy. ... It is commonly necessary to concentrate the sample by chelating and extracting the lead or by evaporating the water, since the natural lead content of lakes and rivers is in the range of 1 to 10 ug/l. /Total Lead/ [USEPA; Air Quality Criteria for Lead p.4-5 (1977) EPA 600/8-77-017] **PEER REVIEWED**

ANALYTIC LABORATORY METHODS
Water: colorimetric method using dithizone. Analytical range is 1.0-30.D ug Pb. Water: differential pulse anodic stripping voltammetry method. Limit of detection is 0.001 mg/l. Can be used to detect levels up to 0.1 mg/l. /Total lead/ [USEPA; Intermedia Priority Pollutant Guidance Docunent: Lead p.9-2 (1982)] **PEER REVIEWED**

ANALYTIC LABORATORY METHODS
AIR SAMPLES OF LEAD OR INORGANIC LEAD COMPOUNDS ARE DETERMINED BY ATOMIC ABSORPTION SPECTROPHOTOMETRY; PROCEDURE: FILTER COLLECTION, NITRIC ACID DIGESTION. RANGE: 0.128 TO 0.399 MG/CU M WITH COEFFICIENT OF VARIATION OF 0.072 FOR TOTAL ANALYTICAL &

SAMPLING METHOD. THIS VALUE CORRESPONDS TO 0.014 MG/CU M STD DEVIATION AT OSHA STD LEVEL OF 0.2 MG/CU M. /TOTAL LEAD/ [U.S. Department of Health, Education Welfare, Public Health Service. Center for Disease Control, National Institute for Occupational Safety Health. NIOSH Manual of Analytical Methods. 2nd ed. Volumes 1-7. Washington, DC: U.S. Goverrment Printing Office, 1977-present. V3 S341-1] **PEER REVIEWED**

ANALYTIC LABORATORY METHODS

MATRIX: AIR; RANGE: 42-840 UG/CU M; PROCEDURE: FILTER COLLECTION, ACID DIGESTION, ATOMIC ABSORPTION SPECTROPHOTOMETRY. /TOTAL LEAD/ [U.S. Department of Health, Education Welfare, Public Health Service. Center for Disease Control, National Institute for Occupational Safety Health. NIOSH Manual of Analytical Methods. 2nd ed. Volumes 1-7. Washington, DC: U.S. Government Printing Office, 1977-present. V5 173-1] **PEER REVIEWED**

ANALYTIC LABORATORY METHODS

Analyte: Lead. Matrix: air. Procedure: Atomic absorption, flame (air-acetylene, oxidizing). Wavelength: 283.3 nm. Range: 10 to 200 ug/sample. Precision: 0.03. The working range is 0.025 to 0.5 mg/cu m for a 400 l air sample. The method is applicable to elemental lead, including lead fume, & all other aerosols containing lead. This is an elemental analysis, not compound specific. Interferences: Use D2 or H2 continuum background correction to control flame or molecular absorption. High concentrations of calcium, sulfate, carbonate, phosphate, iodide, fluoride, or acetate can be corrected. /Total lead/ [U.S. Department of Health and Human Services, Public Health Service. Centers for Disease Control, National Institute for Occupational Safety and Health. NIOSH Manual of Analytical Methods, 3rd ed. Volumes 1 and 2 with 1985 supplement, and revisions. Washington, DC: U.S. Government Printing Office, February 1984. V2 7082-11 **PEER REVIEWED**

ANALYTIC LABORATORY METHODS

Analyte: lead; matrix: air; procedure: filter collection, acid digestion, inductively coupled plasma-atomic emission spectroscopy; detection limit: 17 ng/l. /Total lead/ [U.S. Department of Health, Education Welfare, Public Health Service. Center for Disease Control, National Institute for Occupational Safety Health. NIOSH Manual of Analytical Methods. 2nd ed. Volumes 1-7. Washington, DC: U.S. Government Printing Office, 1977-present. V7 351-11 **PEER REVIEWED**

ANALYTIC LABORATORY METHODS

METHOD FOR DETERMINATION OF LEAD (PB) IN FOOD PRODUCTS (GRAIN PRODUCTS, FRUIT JUICE, MEAT, & VEGETABLES) INVOLVES CO-PRECIPITATION OF PB WITH STRONTIUM SULFATE, CONVERSION TO CARBONATE, DISSOLUTION IN NITRIC ACID, & DETERMINATION BY ATOMIC ABSORPTION SPECTROTOMETRY. /TOTAL LEAD/ [HOOVER WL; J ASSOC OFF ANAL CHEM 55 (4): 737 (1972)] **PEER REVIEWED**

ANALYTIC LABORATORY METHODS

DETERMINATION OF LEAD IN FOOD SAMPLE BY ANODIC STRIPPING VOLTAMMETRY. /TOTAL LEAD/ [HOLAK W; J ASSOC OFF ANAL CHEM 63 (3): 485 (1980)] **PEER REVIEWED**

ANALYTIC LABORATORY METHODS

DETERMINATION OF LEAD IN CEREAL & GRASS SAMPLES BY FLAMELESS ATOMIC ABSORPTION SPECTROSCOPY. /TOTAL INORGANIC LEAD/ [HORAK O; LANDWIRTSCH FORSCH 29 (3-4): 289 (1976)] **PEER REVIEWED**

ANALYTIC LABORATORY METHODS

ZINC, ZINC OXIDE, LEAD, THE 4 KNOWN ZINC CHROMATES, & RHOMBIC & MONOCLINIC LEAD CHROMATE AS PIGMENTS IN PROTECTIVE PAINTS WERE IDENTIFIED IN 0.3-0.5 MG PAINT SAMPLES BY X-RAY DIFFRACTOMETRY. THE ZINC CHROMATES WERE IDENTIFIABLE AT 7.0-9.3 ANGSTROMS. THE METHOD COULD BE USED TO IDENTIFY, BUT NOT TO QUANTITATE ZINC CHROMATES ON FILTERS OF AIR SAMPLES FROM AREAS USED FOR SPRAY PAINTING. /CHROMIUM, LEAD AND ZINC COMPOUNDS/ [ALTIERI A ET AL; ANN IST SUPER SANITA 13 (1-2): 315 (1977)] **PEER REVIEWED**

ANALYTIC LABORATORY METHODS

EPA Method 7420: Lead (Atomic Absorption, Direct Aspiration) Method 7420 is applicable for the determination of metals in solution by atomic absorption spectrometry. Preliminary treatment of waste water, ground water, EP extracts, and industrial waste is always necessary because of the complexity and variability of sample matrix. After aspiration and

atomization of the sample in a flame, a light beam from a hollow cathode lamp or an electrodeless discharge lamp is directed through the flame into a monochromator and onto a detector that measures the amount of absorbed light. ... The light energy absorbed by the flame is a measure of the concentration of that metal in the sample. The performance characteristics for an aqueous sample free from interferences are: optimum concentration range of 1-20 mg/l at a wavelength of 283.3 nm, sensitivity of 0.5 mg/l, and detection limit of 0.1 mg/l. /Total lead/ [USEPA: Test Methods For Evaluating Solid Waste SW-846 (1986)] **PEER REVIEWED**

ANALYTIC LABORATORY METHODS

EPA Method 7421: Lead (Atomic Absorption, Furnace Technique) Method 7421 is applicable for the determination of metals in solution by atomic absorption spectrometry. For certain samples, lower concentrations may be determined using this technique. To ensure valid data, each matrix must be examined for interference effects and, if detected, treat them accordingly, using either successive dilution, matrix modification, or method of standard additions. If poor recoveries are obtained, a matrix modifier may be necessary. Add 10 ul of phosphoric acid to 1 ml of prepared sample in the furnace sampler cup and mix well. A representative aliquot of a sample is placed in the graphite tube in the furnace, evaporated to dryness, charred, and atomized. ... Radiation from a given excited element is passed through the vapor containing ground state atoms of that element. The metal atoms to be measured are placed in the beam of radiation by increasing the temperature of the furnace, thereby causing the injected specimen to be volatilized. A monochromator isolates the characteristic radiation from the hollow cathode lamp or the electrodeless discharge lamp, and a photo sensitive device measures the attenuated transmitted radiation. The optimum concentration range is 5-100 ug/l and the detection limit is 1 ug/l. /Total lead/ [USEPA: Test Methods for Evaluating Solid Uaste SW-846 (1986)] **PEER REVIEWED**

ANALYTIC LABORATORY METHODS

. Direct Aspiration Atomic Absorption Spectrometry is used for the determination of lead. Using air acetylene as the flame gas at a wavelength of 283.3 nm, the detection limit is 0.05 mg/l, with a sensitivity of 0.5 mg/l, at an optimun concentration range of 1-20 mg/l. /Total lead/ [Franson MA (Ed); Standard Methods for the Examination of Water and Wastewater p.154 (1985)] **PEER REVIEWED**

ANALYTIC LABORATORY METHODS

EPA Method 200.7: An Inductively Coupled Plasma - Atomic Emission Spectrophotmetric method for the determination of dissolved, suspended, or total elements in drinking water, surface water, and domestic and industrial wastewaters, is described. Lead is analyzed at a wavelength of 259.940 nanometers and has an estimated detection limit of 7 ug/l. /Total lead/ [40 CFR 136 (7/1/87)] **PEER REVIEWED**

ANALYTIC LABORATORY METHODS

EPA Method 200.7: An Inductively Coupled Plasma - Atomic Emission Spectrophotmetric method for the determination of dissolved, suspended, or total elements in drinking water, surface water, and domestic and industrial wastewaters, is described. Chromium is analyzed at a wavelength of 267.716 nanometers and has an estimated detection limit of 7 ug/l. /Total chromium/ [40 CFR 136 (7/1/87)] **PEER REVIEWED**

CLINICAL LABORATORY METHODS

Several methods are available for measuring elemental chromium in ... biological samples. These include atomic absorption spectroscopy, instrumental neutron activation analysis, X-ray fluorescence, and particle-induced X-ray emissions (PIXE). While these methods are sensitive to the ppb level, problems in sample collection, preparation and interferences are shared by all. In biological samples, neutron activation analysis data tend to be lower than atomic absorption and X-ray fluorescence data. ... Generally, a comparison of the results indicates that modified atomic absorption spectroscopy provides relatively reliable analyses. Another problem in chromiun determination is the lack of adequate reference materials. Ideally, reference

materials should match the samples to be analyzed with respect to chromium levels and each reference composition. Because the materials are not yet standardized, inter-laboratory comparisons are difficult. /Total chromium/ [USEPA; Health Assessment Document: Chromium p.2-4 (1984) EPA 600/8-83-014F] **PEER REVIEWED**

CLINICAL LABORATORY METHODS

Analyte: Chromium; Matrix: urine; Procedure: Inductively-coupled argon-plasma, atomic emmission spectroscopy; Extraction media: polydithiocarbamate resin; Wavelength: 205.6 nm; Range: 0.25-200 ug/samp; Est LOD: 0.1 ug/samp; Precision: 0.078; Interferences: spectral, minimized by wavelength selection /Metals in urine, Chromium/ [U.S. Department of Health and Human Services, Public Health Service. Centers for Disease Control, National Institute for Occupational Safety and Health. NIOSH Manual of Analytical Methods, 3rd ed. Volumes 1 and 2 with 1985 supplement, and revisions. Washington, DC: U.S. Government Printing Office, February 1984. V1 8310-11 **PEER REVIEWED**

CLINICAL LABORATORY METHODS

Analyte: Chromium; Matrix: blood or tissue; Procedure: Inductively-coupled argon plasma-atomic emission spectroscopy; Wavelength: 205.6 nm; Range: 10 to 10,000 ug/100 g blood, 2 to 2000 ug/g tissue; Est LOD: 1 ug/100 g blood, 0.2 ug/g tissue; Precision: 4.7 (%Sr); Interferences: spectral, minimized by wavelength selection /Elements in blood or tissue, Chromium/ [U.S. Department of Health and Human Services, Public Health Service. Centers for Disease Control, National Institute for Occupational Safety and Health. NIOSH Manual of Analytical Methods, 3rd ed. Volumes 1 and 2 with 1985 supplement, and revisions. Washington, DC: U.S. Government Printing Office, February 1984. V1 8005-11 **PEER REVIEWED**

CLINICAL LABORATORY METHODS

... Zinc protoporphyrin (ZPP) concn /was used/ as an index of blood lead concn. Analysis of zinc protoporphyrin consists of diluting whole blood (1:500), adding dimethyldodecylamioxide and measuring the fluorescence at 594 nm (excitation at 424 nm). A correlation coefficient of 0.87 for zinc protoporphyrin and blood lead concn (by AAS) /was found/. Iron deficiency anemia will also reduce zinc protoporphyrin concn. /Total lead/ [Lamola AA et al; Clin Chem 21 (1): 93-7 (1975) as cited in NIOSH; Criteria Document: Inorganic Lead p.XI-29 (1978) DHEU Pub. NIOSH 78-1581 **PEER REVIEWED**

CLINICAL LABORATORY METHODS

... A simplified method for estimating urinary coproporphyrin as a lead exposure index /was developed/. Samples are acidified with acetic acid and coproporphyrin is extracted into ether. The ether extract is shaken with an iodine-hydrochloric acid solution which oxidizes any coproporphyrinogen to coproporphyrin. Concn is measured by absorbance at the Soret band peak (ca 401 nm). /Total lead/ [NIOSH; Criteria Document: Inorganic Lead p.XI-29 (1978) DHEU Pub. NIOSH 78-1581 **PEER REVIEWED**

CLINICAL LABORATORY METHODS

MICRODETERMINATION OF LEAD IN BLOOD & URINE BY ANODIC STRIPPING VOLTAMMETRY EQUIPPED WITH MERCURY ELECTRODE. NO INTERFERENCE WAS FOUND WITH COEXISTING IONS FOUND IN URINE. /TOTAL LEAD/ CKARAI I ET AL; OSAKA CITY MED J 26 (1): 39-46 (1981)] **PEER REVIEWED**

CLINICAL LABORATORY METHODS

High performance liquid chromatography assay of RBC UMPase activity is a sensitive and rapid assay that appears to meet criteria for a reliable clinical laboratory index of blood lead concentrations. /Total lead/ [Cook LR et al; Br J Ind Med 43: 387-90 (1986)] **PEER REVIEWED**

CLINICAL LABORATORY METHODS

Lead concentrations (ug/g wet weight) in human bone (tibia) were measured noninvasively in vivo employing an x-ray fluoresence technique. Forty-five workers who had been chronically exposed to lead (mean duration of employment 20.9 yr) were found to have a mean bone lead content of 52.9 ug/g wet weight (range 0-198 ug/g). Blood lead, urinary lead excretion after EDTA chelation, zinc protoporphyrin, and

unstimulated urinary lead excretion were also evaluated. The correlation coefficients (r) between bone lead measurements (as assayed by x-ray fluoresence) and blood lead, zinc protoporphyrin, or unstimulated urinary lead were 0.44 (p= 0.004). 0.39 (p= 0.0151, and 0.40 (p= 0.01) respectively. The correlation coefficient between x-ray fluoresence (XRF) findings and lead excretion following Ca-EDTA administration was 0.69 (p< 0.001). /Total lead/ [Wielopolski L et al; Am J Ind Med 9: 221-26 (1986)] **PEER REVIEWED**

CLINICAL LABORATORY METHODS

Biological indicator of exposure to lead or lead compounds. Analyte: Lead. Matrix: blood or tissue. Method: Inductively-coupled argon plasma-atomic emission spectroscopy. Wavelength: 220.4 nm. Precision: 0.85. This method is useful for monitoring the blood of workers exposed to several metals simultaneously. This is a sirmltanous multielemental analysis, but is not compound-specific. /Total lead/ [U.S. Department of Health and Human Services, Public Health Service. Centers for Disease Control, National Institute for Occupational Safety and Health. NIOSH Manual of Analytical Methods, 3rd ed. Volumes 1 and 2 with 1985 supplement, and revisions. Washington, DC: U.S. Government Printing Office, February 1984. V1 8005-11 **PEER REVIEWED**

CLINICAL LABORATORY METHODS

Biological indicator of exposure to lead & lead compounds. Analyte: Lead(II)-APDC (ammonium pyrrolidine dithiocarbamate) complex. Matrix: blood or urine. Technique: atomic absorption, air/acetylene. Quality control: comnercial controls, pooled urine or blood, urine corrected for creatinine. Extraction: APDC-MIBK (methyl isobutyl ketone). Range: 5 to 150 ug/100 g blood; 5 to 150 ug/100 ml urine. Precision: 0.05. This procedure quantitates Pb(2+) in blood or urine to assess body burden, injury to the hematopoietic system, & to comply with Federal regulations. Blood lead is the preferred biological indicator of lead absorption. The optimun working range is 0.1 to 1.5 ug Pb/g or per ml urine. Interferences: Phosphate, EDTA, & oxalate can sequester lead and cause low lead readings. /Total lead/ [U.S. Department of Health and Human Services, Public Health Service. Centers for Disease Control, National Institute for Occupational Safety and Health. NIOSH Manual of Analytical Methods, 3rd ed. Volumes 1 and 2 uith 1985 supplement, and revisions. Washington, DC: U.S. Government Printing Office, February 1984. V1 8003-11 **PEER REVIEWED**

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