100 Pine Street, 10th Floor
San Francisco, CA 94111
(415) 434-9400 • FAX (415) 434-1365

GEOMATRIX

ALCO
HAZMAT
93 DEC -9 AM10: 06

8 December 1993
Project 2530

Ms. Madhulla Logan
Alameda County Health Care Services Agency
80 Swan Way, Room 200
Oakland, CA 94621

Subject: Workplan for Site Characterization
Proposed Encinal Marina Landing
2020 Sherman Avenue
Alameda, California

Dear Ms. Logan:

This workplan has been prepared by Geomatrix Consultants, Inc. (Geomatrix), at the request of Encinal Real Estate, Inc. (Encinal), for additional site characterization of the former warehouse site (originally owned by Alameda Marina Village Associates) located at 2020 Sherman Avenue in Alameda, California. The purpose of conducting the site characterization described in this workplan is to obtain sufficient additional information regarding the presence of chemicals in soil and groundwater to allow Alameda County Health Care Services Agency (ACHCSA) to determine if remediation is required before the site can be developed for residential use. The scope of work presented in this workplan was developed to address ACHCSA concerns and information requirements expressed in a meeting on 23 November 1993.

PREVIOUS INVESTIGATIONS

Previous site investigations completed by others in 1990 include:

- A Phase I Environmental Survey by MSE Environmental, which included a site history, environmental setting discussion, regulatory records review, and aerial photograph review. The records reviewed did not indicate the presence of underground tanks on the property.
- A Phase II Environmental Survey by MSE Environmental, which included a magnetics and radar search for underground tanks which did not locate any evidence suggesting the existence of underground storage tanks, and sampling with analysis of two groundwater grab samples from the site. The groundwater samples were analyzed for petroleum hydrocarbons, benzene, toluene, ethylbenzene, and xylenes. No analyte compounds were detected.

- [redacted] by [redacted]es, which consisted of eight boring locations. One groundwater grab sample was collected and analyzed from each of the borings, and soil samples were collected from all borings at depths of two and six feet. Samples from each depth interval were divided into northern and southern site composites resulting in four samples for laboratory analysis. The groundwater and soil samples were analyzed for petroleum hydrocarbons, volatile and semivolatile organics, pesticides, and CAM 17 metals. In [redacted], [redacted] and 1,1,1-TCA were detected in one sample, and [redacted]ns of [redacted] were detected in both samples. The metals concentrations were below MCL limits with the exception of [redacted]. In [redacted], [redacted], [redacted]es, and low concentrations of [redacted] were detected in certain samples; the metals concentrations were below 10 times the Soluble Threshold Limit Concentration (STLC) except for [redacted] one sample, which was 4 ppb above this criteria.

The results for all samples collected during previous investigations are presented in Tables 1 and 2, and the locations of borings and groundwater grab samples are shown on Figure 2.

OBJECTIVES

Evaluation of previous reports and sampling results does not indicate significant chemical impact to soil at the site, except in the area of EB-1, where elevated concentrations of chlorinated solvents were detected. Additionally, chemically affected groundwater appears to be limited to the same area, assuming that metals detected in groundwater were primarily due to suspended sediment in non-filtered grab groundwater samples.

Based on this evaluation, the sampling program for the site has been designed to accomplish the following:

- Assess the lateral extent of chlorinated VOCs in groundwater in the area of EB-1.
- Confirm that evaluated metal concentrations in groundwater are due to suspended sediment in samples.
- Measure groundwater gradient at the site.

GROUNDWATER CHARACTERIZATION PLAN

To determine the groundwater gradient, four temporary piezometers will be installed near former boring EB-1. Each piezometer will be constructed by placing a clean, 1-inch diameter PVC pipe into a boring and surrounding the pipe with sandpack. The pipe will be screened across the water-bearing zone, and asphalt patch will be used to seal the top of the boring if wet weather is expected. The piezometers will be allowed to equilibrate for at least 24 hours, and water levels will then be measured according to Geomatrix protocols. A registered surveyor will survey the measuring points of the piezometers. These piezometers will be located approximately as shown on Figure 2, and will be destroyed after one set of groundwater elevation data has been collected.

One piezometer and two additional borings will be used to collect groundwater samples which will be analyzed for chromium, arsenic, lead and thallium by EPA Method 6010. These metals are those detected at concentrations higher than MCLs during previous investigations. These samples will be field filtered according to EPA guidance to represent metal concentrations in groundwater. The approximate groundwater sampling locations for metals are shown on Figure 2.

Each boring will be advanced using steam-cleaned split-spoon soil samplers sequentially pushed into the ground with a 70-pound hammer. The sampler will be withdrawn every 1.5 to 2.0 feet, and the soil retained will be observed. Each boring will be logged according to Geomatrix protocols. The boring will be terminated at the base of the fill or the top of the Bay Mud, expected to be a depth of approximately six feet. For additional lithologic information, the four piezometer borings will be advanced beyond the base of the fill until three feet of Bay Mud or soil classified as an aquitard have been observed. To collect groundwater samples, a clean, 1-inch diameter PVC pipe screened across the water-bearing zone will be placed in the boring, and a clean, teflon or stainless-steel bailer will be used to collect groundwater from inside the pipe.

The groundwater samples will be collected in laboratory-prepared bottles and handled according to Geomatrix protocols. The samples for metals analysis will be sent under chain-of-custody procedures to an analytical laboratory certified by the California Department of Health Services for the analyses requested. Samples collected from the piezometers will be retained for one or two days and analyzed during the shallow groundwater survey described below.

One or two days after the work described above has been completed, a one-day shallow groundwater survey will be performed in the vicinity of previous boring EB-1 to determine the lateral extent of 1,1-DCA in the shallow groundwater. In general, sample locations will follow a grid pattern, and will remain within the property boundaries. The survey will be conducted by [redacted]Tracer) under Geomatrix supervision by pushing a 1-inch diameter steel rod into the ground to the expected depth to groundwater, previously found at approximately 6 feet. The rod will be withdrawn one or two feet to allow groundwater to enter the boring, and a clean stainless steel or teflon bailer will be used to collect a groundwater sample. The samples will be analyzed in the field by Tracer using a screening technique for chlorinated halogens including 1,1-DCA. Additional points will be chosen in a progressive manner.

The borings will be backfilled with cement upon completion. Soil cuttings are expected to be minimal due to the boring technique utilized, and will be retained in 55-gallon drums on the site. The drums will be sampled and the cuttings disposed of according to regulatory requirements.

SOIL CHARACTERIZATION

No soil analysis

Soil samples to determine the extent of 1,1-DCA in soil were requested by ACHCSA. We propose to first define the extent of this compound in groundwater before attempting to locate a potential source area in the surficial soils. [redacted] be [redacted].

HEALTH AND SAFETY

A health and safety plan for site characterization work performed by Geomatrix has been developed. It is enclosed as an addendum.

SCHEDULE AND REPORTING

After evaluation of current and previous site data, a report will be prepared summarizing field activities, analytical results, groundwater gradient information, soil types, and our evaluation of environmental site characteristics that could potentially impact residential development. The work is expected to require two days of field time, and is tentatively scheduled for January 1994. The actual schedule will depend on when approval of the workplan is received from the ACHCSA. A report presenting the results of the site characterization work with recommendations will be completed within four weeks of the fieldwork.

Thank you for this opportunity to be of service. If you have any questions, please call either of the undersigned.

Sincerely,

GEOMATRIX CONSULTANTS, INC.

Lisa Rowhe for

Cheri Y. Page, R.G.
Project Geologist

Tom Graf, P.E.
Vice President

CYP/TEG/bab
CONTR\2530-WP.LTR

Attachments:	Table 1 - Summary of Previous Groundwater Sample Results
	Table 2 - Summary of Previous Soil Sample Results
	Figure 1 - Location Map
	Figure 2 - Site Plan
Enclosure:	Health and Safety Plan

cc: Mr. Peter Wang - Encinal Real Estate, Inc.

GEOMATRIX

TABLE 1

SUMMARY OF PREVIOUS GROUNDWATER SAMPLE RESULTS

Encinal Real Estate
2020 Sherman Avenue
Alameda, California

Compounds detected in micrograms per liter (µg/l)

Sample Location I.D.	EPA Method 602	EPA Method 8015	EPA Method 624	EPA Method 625	EPA Method 608	CAM 17 Metals	
TB-1[1]	ND[2]	ND	NA[3]	NA	NA	NA	
TB-2[1]	ND	ND	NA	NA	NA	NA	
EB-1[4]	NA	Motor oil: 300	1,1-DCA: 1500 1,1,1-TCA: 17	ND	ND	Antimony	ND
						Arsenic	170
						Barium	540
						Beryllium	3
						Cadmium	ND
						Cobalt	58
						Chromium	330
						Copper	200
						Mercury	1.3
						Molybdenum	ND
						Nickel	280
						Lead	50
						Selenium	ND
						Silver	ND
						Thallium	280
						Vanadium	320
						Zinc	300

1,1 DCA = 1.5 ppm
1,1,1 TCA .017

TABLE 1

SUMMARY OF PREVIOUS GROUNDWATER SAMPLE RESULTS

Sample Location I.D.	EPA Method 602	EPA Method 8015	EPA Method 624	EPA Method 625	EPA Method 608	CAM 17 Metals	
EB-2[4]	NA	Motor oil: 200	ND	ND	ND	Antimony	ND
						Arsenic	150
						Barium	770
						Beryllium	4
						Cadmium	ND
						Cobalt	50
						Chromium	360
						Copper	200
						Mercury	ND
						Molybdenum	ND
						Nickel	330
						Lead	ND
						Selenium	ND
						Silver	ND
						Thallium	650
						Vanadium	420
						Zinc	310

Notes:

1 Work conducted by MSE Environmental, Inc., in 1990.
2 ND = not detected.
3 NA = not analyzed.
4 Work conducted by Kaldveer Associates in 1990.

TABLE 2

SUMMARY OF PREVIOUS SOIL SAMPLE RESULTS
Encinal Real Estate
2020 Sherman Avenue
Alameda, California

Compounds detected in milligrams per kilogram (mg/kg)

Boring Numbers	Sample Depth (feet)	EPA Method 8240	EPA Method 8270	EPA Method 8080	EPA Method 8015	Metals[1]	
EB-2 EB-3 EB-4 EB-8 (composite)	2	ND[2]	ND	ND	motor oil: 180/110	Antimony	ND
						Arsenic	8
						Barium	59
						Beryllium	0.3
						Cadmium	ND
						Cobalt	7.5
						Chromium	43
						Copper	20
						Cyanide	1.2
						Mercury	ND
						Molybdenum	ND
						Nickel	39
						Lead	31
						Selenium	ND
						Silver	ND
						Thallium	23
						Vanadium	37
						Zinc	42
EB-2 EB-3 EB-4 EB-8 (composite)	6	ND	benzo(b)fluoranthene: 0.35 benzo(a)pyrene: 0.34 pyrene: 0.76	ND	motor oil: 40/70	Antimony	ND
						Arsenic	20
						Barium	52
						Beryllium	0.6
						Cadmium	ND
						Cobalt	15
						Chromium	54
						Copper	26
						Cyanide	0.85
						Mercury	ND
						Molybdenum	0.6
						Nickel	74
						Lead	4
						Selenium	ND
						Silver	ND
						Thallium	39
						Vanadium	50
						Zinc	120

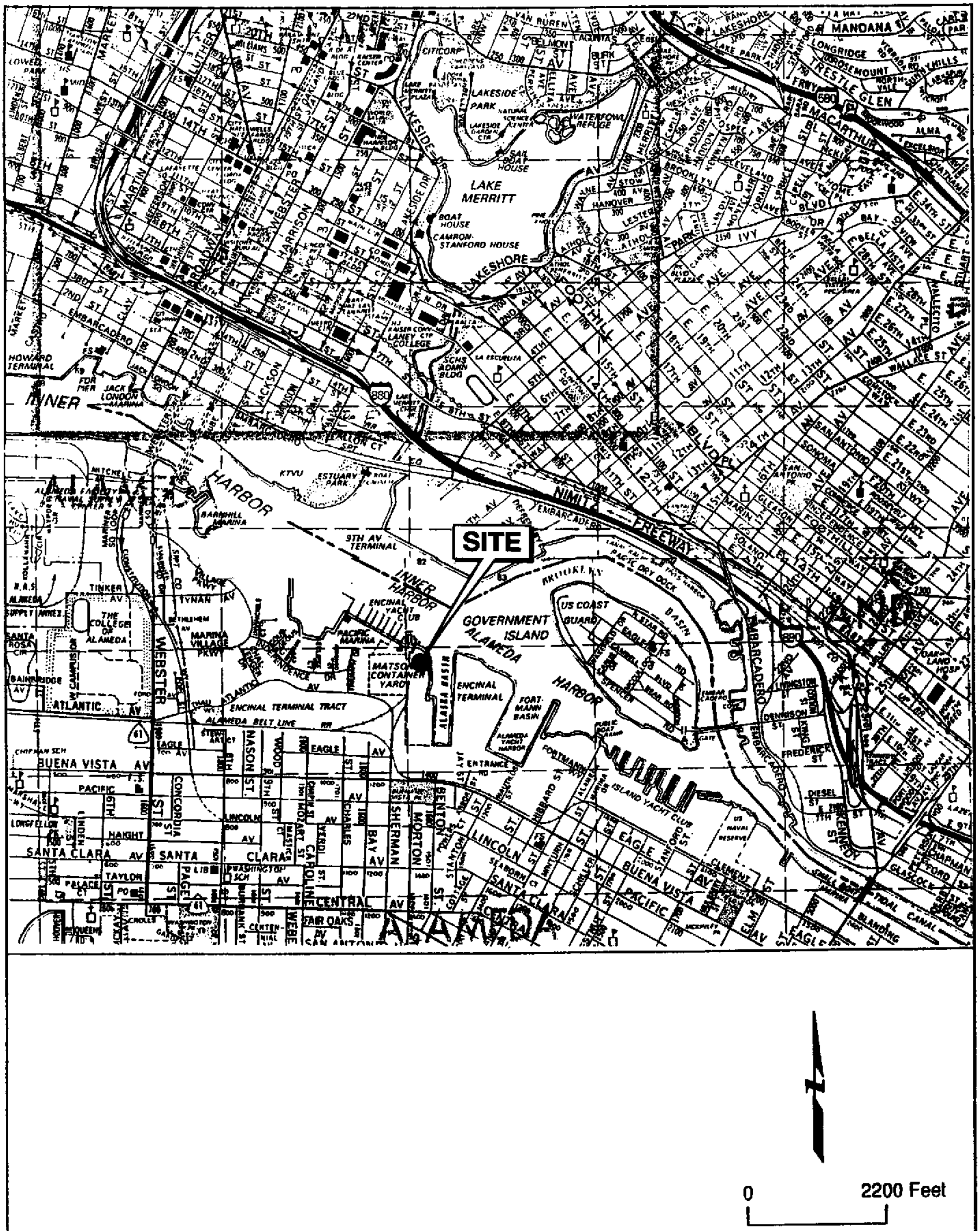
TABLE 2

SUMMARY OF PREVIOUS SOIL SAMPLE RESULTS

Boring Numbers	Sample Depth (feet)	EPA Method 8240	EPA Method 8270	EPA Method 8080	EPA Method 8015	Metals[1]	
EB-1 EB-5 EB-6 EB-7 (composite)	2	ND	ND	ND	motor oil: 180/110	Antimony	ND
						Arsenic	13
						Barium	54
						Beryllium	0.4
						Cadmium	ND
						Cobalt	7.7
						Chromium	43
						Copper	54
						Cyanide	0.5
						Mercury	0.3
						Molybdenum	ND
						Nickel	38
						Lead	9
						Selenium	ND
						Silver	ND
						Thallium	49
						Vanadium	37
						Zinc	51
EB-1 EB-5 EB-6 EB-7 (composite)	6	1,1-DCA: 0.4	ND	ND	motor oil: 40/70	Antimony	ND
						Arsenic	9
						Barium	24
						Beryllium	0.3
						Cadmium	ND
						Cobalt	5.7
						Chromium	35
						Copper	16
						Cyanide	0.4
						Mercury	ND
						Molybdenum	ND
						Nickel	29
						Lead	2
						Selenium	ND
						Silver	ND
						Thallium	20
						Vanadium	33
						Zinc	29

Notes:

1 CAM 17 metals and cyanide
2 ND = not detected

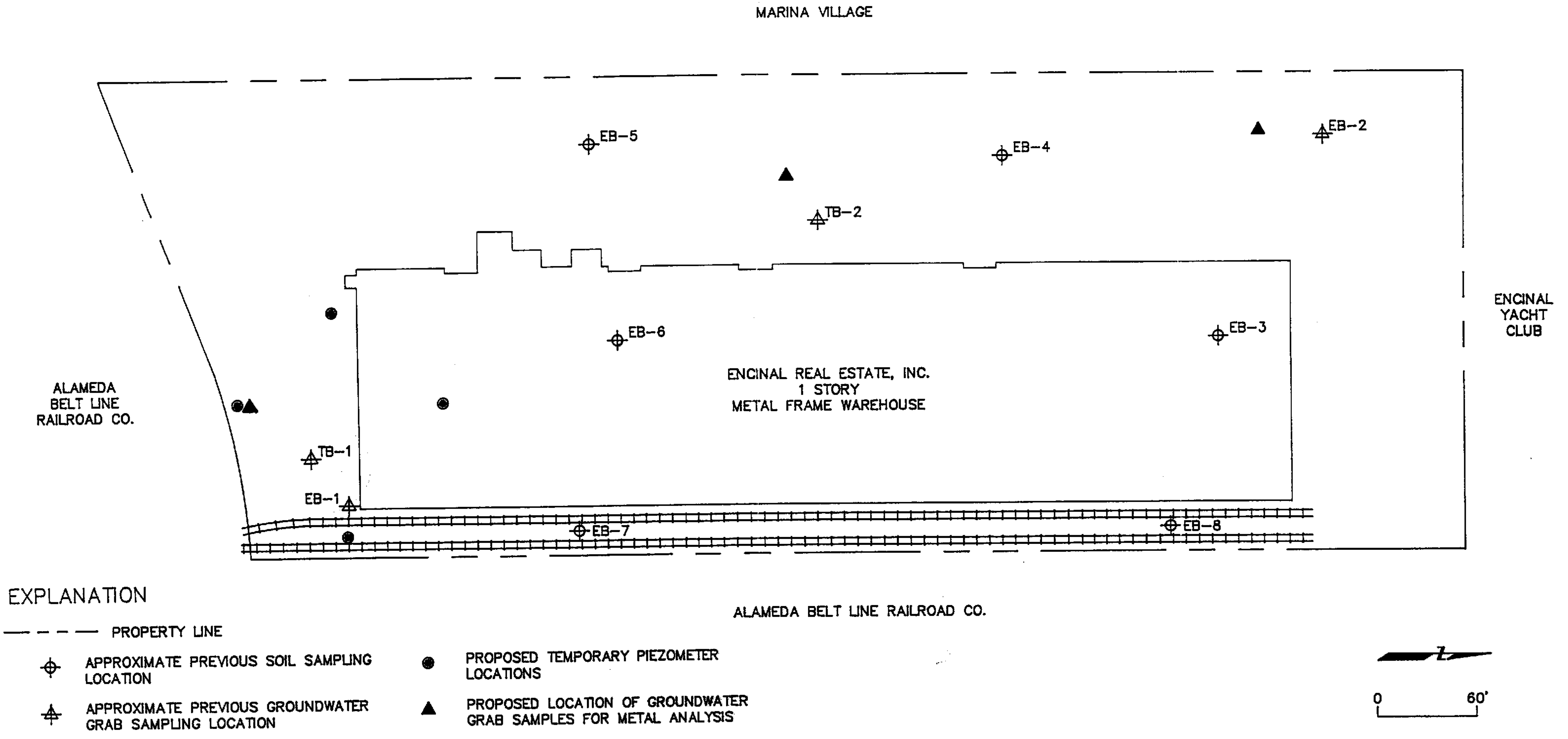


SITE LOCATION MAP
2020 Sherman
Alameda, California

GEOMATRIX

Figure
1

Project No.
2530



GEOMATRIX

Revisions

SOIL AND GROUNDWATER SAMPLE LOCATIONS

ENCINAL REAL ESTATE
2020 SHERMAN AVE.
ALAMEDA, CALIFORNIA

Figure
2

Project No.
2530

SITE HEALTH & SAFETY PLAN

1.0 PURPOSE

This site health and safety plan establishes procedures to address health and safety aspects of fieldwork to be conducted by Geomatrix employees at the site. The observance of procedures in this plan are mandatory for all Geomatrix employees at the site.

This plan shall be used only after the plan has been reviewed by the Project Manager and Project Health and Safety Officer. Prior to entering the site, Geomatrix personnel shall read this plan and sign the attached form verifying that they have read the plan and understand the requirements of the plan.

2.0 ADMINISTRATIVE INFORMATION

Project Name: Encinal Marina Landing

Project Start Date: 18 November 1993 Project Number: 2530

Project Address: 2020 Sherman, Alameda, California

Client: Encinal Real Estate, Inc.

Client Contact: Peter Wang

Telephone No.: (510) 523-8800, Ext. 18

Project Manager: Cheri Page

Telephone No.: (415) 434-9400 (work) (707) 769-8388 (home)

Project Health & Safety Officer: Mary Sue Philp

Telephone No.: (415) 434-9400 (work) (415) 282-3873 (home)

Site Safety Officer: Charlie Crocker

Telephone No.: (415) 434-9400 (work) (415) 921-5082 (home)

SITE HEALTH & SAFETY PLAN (cont.)

3.0 PROJECT DESCRIPTION

3.1 Site History: Inactive shipping terminal with warehouse, soil and groundwater investigation performed in 1990.

3.2 Site Physical Description: Six-acre paved lot with large warehouse

3.3 Type of Investigation: Environmental investigation of soil and groundwater

3.4 Scope of Field Activities (List all field tasks for project):
Soil core sampling without augers, grab groundwater sampling, installing temporary piezometers, on-site laboratory analysis of soil and groundwater, water-level measurements

3.5 Identified Areas of Concern and Media Affected:
1,1-DCA in soil and groundwater in southeast corner of site, possible metals in groundwater above MCLs

3.6 Hazardous Substances Known or Suspected at Site:

CHEMICAL	MEDIA	CONCENTRATION	ROUTES OF EXPOSURE
1,1-DCA	soil	0.4 ppm	Inhalation
1,1-DCA	groundwater	1500 ppb	Inhalation
1,1-TCA	groundwater	1700 ppb	Inhalation
Pyrene	soil	<1 ppm	Inhalation, ingestion
Lead	groundwater	50 ppb	Inhalation, ingestion
Chromium	groundwater	360 ppb	Inhalation, ingestion
Arsenic	groundwater	170 ppb	Inhalation, ingestion
Thallium	groundwater	650 ppb	Inhalation, dermal

(Attach a chemical information sheet for all known or suspected hazardous substances listed.)

SITE HEALTH & SAFETY PLAN (cont.)

3.7 Potential Physical Hazards at Site

SAFETY HAZARDS:
Inactive railroad tracks

UNDERGROUND UTILITY HAZARDS:
An underground utility check shall be performed prior to initiating any subsurface investigation or work. The check will include:

__ USA X Private Locator X Plans Check __ Geophysical

LOCATION OF UNDERGROUND UTILITIES (expand): Parallel edge of building

Other utility hazards: None

OVERHEAD POWER LINES: Not within 100 feet of drilling locations

The following are minimum clearances for overhead high voltage lines.

Normal Voltage (phase to phase				Minimum Required Clearance (feet)
more than	750	-	50,000	10
more than	50,000	-	75,000	11
more than	75,000	-	125,000	13
more than	125,000	-	175,000	15
more than	250,000	-	379,000	21
more than	370,000	-	550,000	27
more than	550,000	-	1,000,000	42

Whenever possible, avoid working under overhead high voltage lines.

NOISE HAZARDS: drill rig

HEAT STRESS HAZARDS: unlikely in winter

SUNBURN HAZARDS: Slight possibility, wear sunscreen on sunny days

SITE HEALTH & SAFETY PLAN (cont.)

TRENCH/EXCAVATION HAZARDS: None

(Attach trenching/excavation operating procedures if hazard exists)

CONFINED SPACE: None

(Attach confined space entry plan)

OTHER HAZARDS: None

4.0 PRIMARY RESPONSIBILITIES

4.1 Project Manager

The project manager (PM) shall:

1. direct all project investigative, monitoring, and remedial activities at the site and vicinity;
2. make the site health and safety officer aware of all pertinent project developments and plans;
3. make available the resources that are necessary for a safe working environment; and
4. maintain communications with client, as necessary.

4.2 Project Health and Safety Officer

The project health and safety officer (PHSO) shall:

1. direct all health and safety aspects of investigative, monitoring, and remedial activities conducted at the site and vicinity;
2. ensure that all personnel have received required training, are aware of the potential hazards associated with site operations, have been instructed in the work practices necessary for personal health and safety, and are familiar with the site health and safety plan's procedures for all scheduled activities and for dealing with emergencies;
3. direct required exposure monitoring to assess site health and safety conditions;
4. prepare any accident/incident reports;
5. modify the site health and safety plan as required based on accidents/incidents and findings regarding personnel exposures and work practices; and
6. report all accidents/incidents and findings regarding personnel exposure and work practices to the project manager.

4.3 Site Safety Officer

The site safety officer (SSO) shall:

1. ensure that appropriate personal protective equipment is available for site personnel and enforce proper utilization of personal protective equipment by on-site personnel and visitors;

2. with guidance from the PHSO, observe subcontractor's procedures with respect to health and safety. If the SSO believes that a subcontractor's personnel are or may be exposed to an immediate health hazard, the SSO shall suspend the subcontractor's site work. If the subcontractor's personnel do not have required protective equipment, the SSO shall consult with the PM or PHSO before proceeding with the work;

3. implement the project health and safety plan and report any observed deviations from anticipated site conditions anticipated in the plan;

4. calibrate monitoring equipment daily and properly record and file results;

5. under direction of the PHSO, perform required exposure monitoring;

6. maintain monitoring equipment or arrange maintenance as necessary;

7. assume other duties as directed by the PM or PHSO; and

8. report observed accidents/incidents or inadequate work practices to the PHSO and the PM.

4.4 Project Personnel

Project personnel involved in on-site investigations and operations shall:

1. take reasonable precautions to prevent injury to themselves and to their fellow employees;

2. perform only those tasks that they can do safely and immediately report accidents and/or unsafe conditions to the SSO or PHSO;

3. follow the procedures set forth in the site health and safety plan and report to the SSO or PHSO any observed deviations from the procedures described in the plan on the part of Geomatrix or subcontractor personnel; and

4. inform the PM and PHSO of any physical conditions that might affect their ability to perform the planned field tasks.

4.5 Training Requirements

All project personnel must be in compliance with OSHA regulations specified in 29 CFR 1910.120. These include completion of a 40-hour health and safety training course and participation in Geomatrix Consultants' medical monitoring program and respiratory protection program.

5.0 SITE CONTROL

The purpose of site control is to minimize the potential exposure to site hazards and to prevent vandalism at the site.

5.1 Site Security

Attach map of site showing hazard areas and areas designated for site work, decontamination, clean areas, and limited access areas. Only authorized personnel shall be permitted access to the site work areas. If possible, work areas will be cordoned with barriers to limit unauthorized access.

Access to work areas will be controlled by means of unoccupied site

5.2 Communications

A field representative should contact the project manager or office at least once a day while in the field.

LOCATION OF CLOSEST TELEPHONE: Field vehicle

6.0 AIR MONITORING

The following air monitoring equipment will be used.

_X_ Photoionization Detector
__ Draeger Pump and Tubes (specify tubes) ______________________________
__ Combustible Gas Meter
__ Oxygen Meter
__ Other (specify) ____

Frequency of sampling: (Specify limits)
__ Continuous ____
__ Intermittent ____
_X_ Screening (Type): Screen borehole after equipment removal and core when exposed

7.0 PERSONAL PROTECTIVE EQUIPMENT

The following personal protective equipment will be used as specified below.

__ Chemical-resistant rubber boots, steel-toed
_X_ Steel-toed boots
_X_ Hard hat
_X_ Ear plugs
_X_ Gloves (specify)
    __ Latex inner liner, nitrile outer glove
    __ Latex inner liner, neoprene outer glove
    _X_ Nitrile outer glove only
    __ Neoprene outer glove only
__ Disposable suit (specify)
    __ Tyvek
    __ Saranex
_X_ Respirator (available)
    __ Disposable dust mask
    _X_ 1/2-face
    __ full-face
__ Cartridges
    _X_ Organic vapor (black)
    __ Dusts, mists, fumes (purple)
    __ Combo organic vapor and dust (purple/black)
    __ Other (specify) ____________
__ Safety glasses/goggles
__ Other (specify) ____________

The following protective equipment/clothing shall be worn during the following activities.

ACTIVITY	EQUIPMENT/CLOTHING
Soil coring and sampling	gloves, steel-toed shoes, ear plugs, hardhat
Water levels	gloves

SITE HEALTH & SAFETY PLAN (cont.)

Listed below are OSHA permissible exposure limits (PELs) and ACGIH recommended threshold limit values (TLVs) for the chemicals of concern at the site.

Respirators shall be worn when air monitoring indicates that concentrations exceed the action levels listed below or conditions arise where the action levels listed below may be exceeded.

CHEMICAL	OSHA PEL	ACGIH TLV
1,1-DCA	100 ppm	100 ppm
1,1,1-TCA	350 ppm	350 ppm
Pyrene	N/A	N/A
Chromium	0.5 mg/m^3	0.5 mg/m^3
Lead	0.15 mg/m^3	0.05 mg/m^3
Arsenic	0.01 mg/m^3	0.01 mg/m^3
Thallium	0.1 mg/m^3	0.1 mg/m^3

Action Levels:

Wear respirator if PID reads $\geq$ 5 ppm in breathing zone

Stop work if PID reads $\geq$ 50 ppm in breathing zone

An explosion hazard shall be assumed to exist where air concentrations of the following exceed their respective lower explosion levels (LEL).

CHEMICAL	LEL

Stop work if Combustible Gas Meter reads $\geq$ 20% LEL

SITE HEALTH & SAFETY PLAN (cont.)

8.0 DECONTAMINATION

8.1 Personnel decontamination procedures (if needed): Wash hands and face prior to eating, drinking, or smoking

8.2 Equipment, sampling gear decontamination procedures: Wash in Alconox and water or steam clean

8.3 Disposal of investigation-derived materials (expendables, decon waste, soil cuttings, groundwater, etc.): 55-gallon barrel for temporary storage on site pending analytical results

9.0 EMERGENCIES

In the event of an accident or emergency condition, the procedure specified below shall be followed immediately.

1. Site safety officer shall take charge of situation.

2. Remove injured or exposed person(s) from immediate danger if possible.

3. Evacuate other on-site personnel to a safe place until it is safe for work to resume.

4. If serious injury or life-threatening condition exists call

   911 - Paramedics, fire department, police
   Hospital emergency room

   Clearly describe location, injury and conditions to dispatcher/hospital. Designate a person to direct emergency equipment to the injured person(s).

5. Provide first aid, if necessary.

6. Call the project manager and/or health and safety officer.

7. Immediately implement steps to prevent reoccurrence of the accident.

8. Attach map of hospital location.

   Hospital Alameda Hospital
   Address 2070 Clinton Avenue
   Alameda, California
   Telephone (510) 522-3700

9. Nearest Poison Control Center Telephone: 1 800 523-2222

10. Other emergency notifications and phone numbers:

SITE HEALTH & SAFETY PLAN (cont.)

10.0 APPROVALS

Peri Y. Page
Project Manager

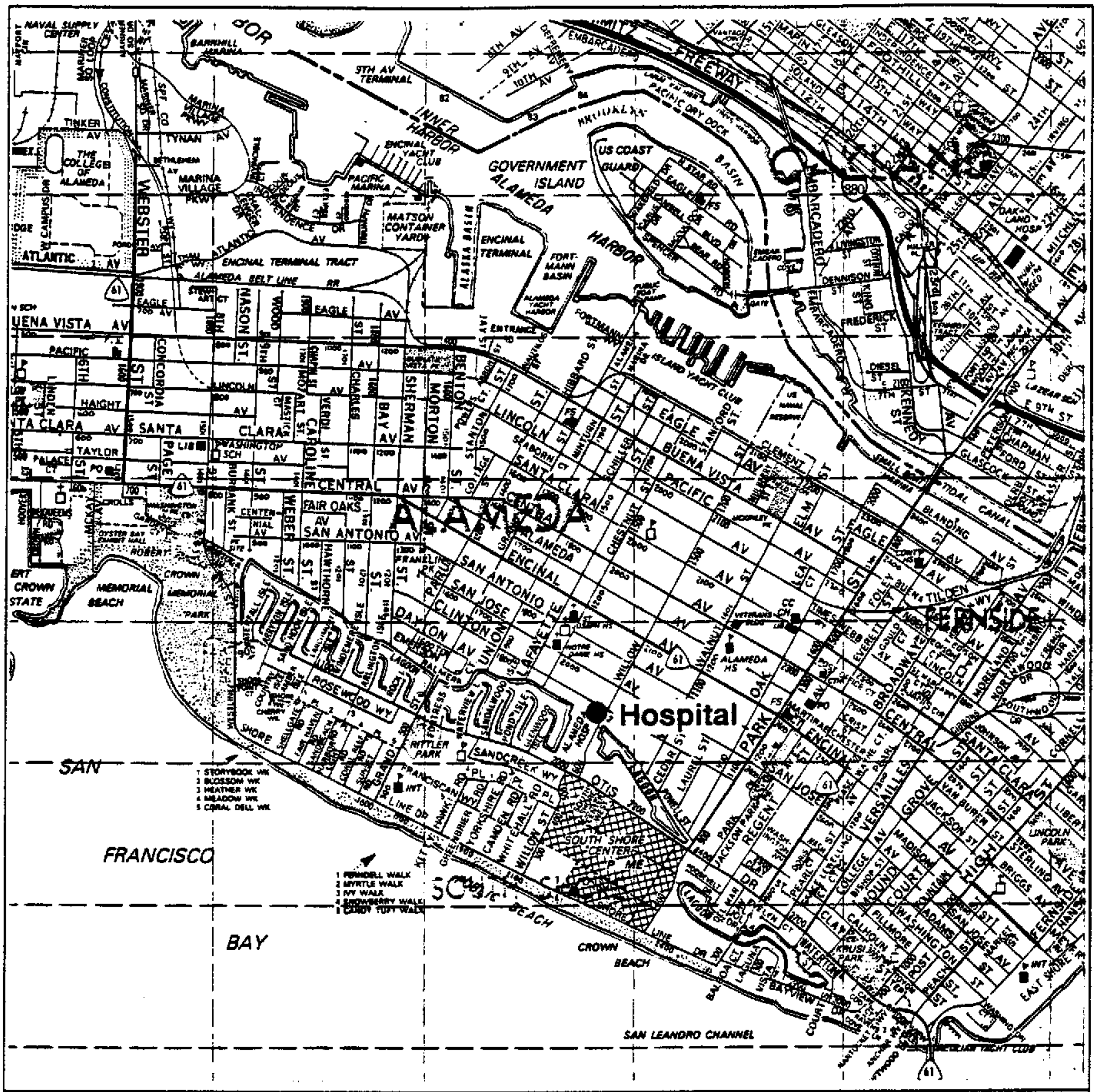
12-8-93
Date

Project Health & Safety Officer

12/8/93
Date

Site Safety Officer

12/8/93
Date



0 2200 Feet

Telephone number: (510) 522-3700

GEOMATRIX

HOSPITAL LOCATION MAP
Alameda Hospital
2070 Clinton Avenue
Alameda, California

Figure

Project No.

METHYL CHLOROFORM

CAS: 71-55-6

1,1,1-Trichloroethane

CH_3CCl_3

TLV-TWA, 350 ppm (≈ 1900 mg/m³)

TLV-STEL, 450 ppm (≈ 2450 mg/m³)

Methyl chloroform is a water-clear, nonflammable liquid. Its physiochemical properties include:

> *Molecular weight: 133.42*
> *Specific gravity: 1.3376 at 20°C*
> *Solidifies: -32.5°C*
> *Boiling point: 74.1°C*
> *Vapor pressure: 100 torr at 20°C*

It burns only in excess oxygen or in air if a strong source of ignition is present. It is almost insoluble in water, but is miscible with most organic solvents.

The major usage of methyl chloroform is as a cleaning solvent. Because of its reactivity with magnesium, aluminum and their alloys, inhibitors are generally added to increase the stability of the solvent.

The oral toxicity of methyl chloroform is low. The LD_{50} for rats, mice, rabbits and guinea pigs was reported to range from 5.7 to 12.3 g/kg.(1) Like many solvents, methyl chloroform will defat the skin, causing redness and scaliness. Absorption through the skin can occur but is not a significant route of toxic exposure; the acute LD_{50} for rabbits is greater than 16 g/kg. When doses of 0.5 g/kg were applied repeatedly for 90 days to rabbits, no effects were caused except for slight reversible irritation of the skin at the site of application.(1)

While comparatively low in systemic toxicity, methyl chloroform is an anesthetic and is capable of causing death when inhaled at concentrations in excess of 14,000-15,000 ppm.(1)

Torkelson and associates(1) described the toxicity of methyl chloroform from repeated exposures of animals. Exposure of animals for three months at concentrations from 1000 to 10,000 ppm caused some pathologic changes in the livers and lungs of some species; the main effect of exposure appeared to be anesthesia. Exposure to the vapor at 500 ppm for seven hours a day, five days a week for six months did not cause any toxic changes of significance in rats, guinea pigs, rabbits or monkeys.

Rowe and associates(2) found that the only effect of repeated exposure of several species at 500 ppm of a mixture containing 75% methyl chloroform and 25% perchloroethylene was a slight degression in the growth of guinea pigs, due to a reduced food intake. At 1000 ppm mild, reversible liver and kidney changes were detected. A time-weighted average limit of 400 ppm was recommended for this mixture.

Other animal studies confirm the low hepatotoxicity of methyl chloroform,(3,4) but indicates that cardiac sensitization can occur if exposures are excessive.(5,6) Studies in dogs given intravenous injections of epinephrine in conjunction with exposure to either 2500, 5000 or 10,000 ppm vapor have been described. Under these exaggerated conditions, no cardiac sensitization was observed at 2500 ppm, but 3 of 18 dogs at 5000 ppm and 12 of 12 at 10,000 ppm were affected.(6) Other studies in rabbits, rats and mice, as well as human experience in anesthesiology, confirm the cardiac effects of methyl chloroform.(7-8) Methyl chloroform is poorly metabolized and is excreted unchanged in the expired air of animals and human test subjects.(9)

Methyl chloroform did not produce teratogenic effects in rats or mice exposed 7 hours per day to 875 ppm during the period of organogenisis.(10) Two lifetime cancer studies have been negative. There were no adverse effects of any kind in rats exposed 6 hours per day for 12 months to either 875 or 1750 ppm vapors.(11) Groups of rats and mice fed methyl chloroform by gavage in the National Cancer Institute (NCI) Bioassay Program showed no increase in tumors over that of the controls.(12) The dosage levels fed were 1500 and 750 g/kg/day.

Industrial experience has been consistent with the findings in laboratory animals.(13) Deaths due to anesthesia and/or cardiac sensitization have been reported to have occurred in poorly ventilated rooms, pits, tanks and other small areas.(1,13) Removal of unconscious individuals has generally resulted in rapid and complete recovery.

In a few test subjects beginning anesthetic effects occur at concentrations approaching 500 ppm.(14) The most extensive study of neurological response has been by Stewart and associates who reported that

> *". . . repetitive vapor exposure to . . . 350 ppm produced no untoward subjective or objective health response . . ."*(15)

Some female test subjects did object slightly to the odor at this concentration. In practice, odor is not a problem until exposure concentrations approach 500 ppm.(13)

The most extensive study of industrially exposed workers has been reported by Kramer and associates who conducted an epidemiological study on 151 men and women exposed for several months to six years to methyl chloroform.(16) During the study period exposures for some workers exceeded 200 ppm. Based on subjective responses and some previous monitoring data, exposure concentrations had been higher prior to study period. When compared to 151 matched pair control subjects by numerous medical and physiological parameters, there were no adverse effects related to exposure.

A time-weighted average TLV for methyl chloroform of 350 ppm is recommended to prevent beginning anesthetic effects and objections to odor. A STEL of 450 ppm is recommended for protection against anesthesia.

Other recommendations: West Germany (1974) 200 ppm; East Germany (1973) 90 ppm; Sweden (197 8) 70 ppm; USSR (1972) 4 ppm; Czechoslovakia (1969) 90 ppm.

References

1. **Torkelson, T.R. et al:** *Am. Ind. Hyg. Assoc. J.* 19:353 (1958).
2. **Rowe, V.K. et al:** *Ibid.* 24:541 (1963).
3. **Gehring, P.J.:** *Tox. Appl. Pharm.* 13:287 (1968).
4. **Plaa, G.L., E.A. Evans and C.H. Hine:** *J. Pharm. Expel. Therap.* 123:224 (1958).
5. **Rennick, B.R. et al:** *Fed. Proc.* 8:327 (1949).
6. **Trochimowicz, H.J. et al:** *JOM* 18:26 (1976).
7. **Aviado, D.M. et al:** *Methyl Chloroform and Trichloroethylene in the Environment.* Clinical Rubber Press, Cleveland, OH (1976).
8. **Dornette, W.H. and J.P. Jones:** *Anesth. Analg.* 39:249 (1966).
9. **Hake, C.L. et al:** *Arch. Env. Health* 1:101 (1966).
10. **Schwetz, B.A. et al:** *TAP* 32:84 (1975).
11. **NIOSH:** *Criteria for a Recommended Standard — Occupational Exposure to 1,1,1-Trichloroethane.* DHEW Pub. No. (NIOSH) 76-184 (1976).
12. **Weisberger, E.:** *Env. Health Perspectives* 21:7 (1977).
13. **Patty, F.A.:** *Industrial Hygiene and Toxicology*, 2nd ed., Vol. II, p. 1288. Interscience, New York (1963).

TIN000 ***HR: 3***

1,1,2-TRICHLOROETHANE

CAS: 79-00-5 NIOSH: KJ 3150000

mf: $C_2H_3Cl_3$ mw: 133.40

PROP: Liquid; pleasant odor. Bp: 114°, fp: −35°, d: 1.4416 @ 20°/4°, vap press: 40 mm @ 35.2°.

SYNS:

ETHANE TRICHLORIDE
NCI-C04579
RCRA WASTE NUMBER U227
β-T
1.1.2-TRICHLORETHANE
β-TRICHLOROETHANE
1.2.2-TRICHLOROETHANE
TROJCHLOROETAN(1.1.2) (POLISH)
VINYL TRICHLORIDE

TOXICITY DATA:	CODEN:
skn-rbt 500 mg open MLD	UCDS** 6/28/72
skn-rbt 810 mg/24H SEV	JETOAS 9,171,76
eye-rbt 162 mg MLD	JETOAS 9,171,76
skn-gpg 1440 mg/15M	APTOA6 41,298,77
otr-mus:emb 25 mg/L	CALEDQ 28,85,85
cyt-gpg-skn 2880 µg/kg	APTOA6 41,298,77
dnd-mam:lym 1 mmol/L	TODED5 11,243,82
orl-mus TDLo:532 mg/kg (14D male):REP	DCTODJ 8,333,85
orl-mus TDLo:76 g/kg/78W-I: CAR	NCITR* NCI-CG-TR-74,78
orl-mus TD :152 g/kg/78W-I: CAR	NCITR* NCI-CG-TR-74,78
orl-rat LD50:580 mg/kg	AIHAAP 30,470,69
ihl-rat LCLo:500 ppm/8H	AIHAAP 30,470,69
orl-mus LD50:378 mg/kg	DCTODJ 8,333,85
ipr-mus LD50:494 mg/kg	TXAPA9 9,139,66
scu-mus LD50:227 mg/kg	JPETAB 123,224,58
orl-dog LDLo:500 mg/kg	AJHYA2 16,325,32
ipr-dog LD50:450 mg/kg	TXAPA9 10,119,67
ivn-dog LDLo:95 mg/kg	QJPPAL 7,205,34
ihl-cat LCLo:13100 mg/m^3/4.5H	AHBAAM 116,131,36
skn-rbt LD50:3730 mg/kg	AIHAAP 30,470,69
scu-rbt LDLo:500 mg/kg	QJPPAL 7,205,34

IARC Cancer Review: Animal Limited Evidence IMEMDT 20,533,79. NCI Carcinogenesis Bioassay (gavage); No Evidence: rat NCITR* NCI-CG-TR-74,78; (gavage); Clear Evidence: mouse NCITR* NCI-CG-TR-74,78. Community Right To Know List. Reported in EPA TSCA Inventory.

OSHA PEL: TWA 10 ppm (skin)
ACGIH TLV: TWA 10 ppm (skin)
DFG MAK: 10 ppm (55 mg/m^3)

THR: Poison by ingestion, intravenous and subcutaneous routes. Moderately toxic by inhalation, skin contact, and intraperitoneal routes. An experimental carcinogen. Experimental reproductive effects. Mutagenic data. An eye and severe skin irritant. Has narcotic properties and acts as a local irritant to the eyes, nose and lungs. It may also be injurious to the liver and kidneys. Incompatible with potassium. When heated to decomposition it emits toxic fumes of Cl^-. A priority pollutant associated with EPA superfund sites. See also CHLORINATED HYDROCARBONS, ALIPHATIC and other trichloroethane entries. For further information, see Vol. 5, No. 3 of *DPIM Report*.

TIM750 **HR: 3**

1,1,1-TRICHLOROETHANE

CAS: 71-55-6 NIOSH: KJ 2975000
DOT: 2831
mf: $C_2H_3Cl_3$ mw: 133.40

PROP: Colorless liquid. Bp: 74.1°, fp: −32.5°, flash p: none, d: 1.3376 @ 20°/4°, vap press: 100 mm @ 20.0°. Insol in water; sol in acetone, benzene, carbon tetrachloride, methanol, ether.

SYNS:

AEROTHENE TT
CHLOROETENE
CHLOROETHENE
CHLOROTHANE NU
CHLOROTHENE
CHLOROTHENE (INHIBITED)
CHLOROTHENE NU
CHLOROTHENE VG
CHLORTEN
INHIBISOL
METHYLCHLOROFORM
METHYL CHLOROFORM (ACGIH, DOT)
METHYLTRICHLOROMETHANE
NCI-C04626
RCRA WASTE NUMBER U226
SOLVENT 111
STROBANE
α-T
1,1,1-TCE
1,1,1-TRICHLOORETHAAN (DUTCH)
1,1,1-TRICHLORAETHAN (GERMAN)
TRICHLORO-1,1,1-ETHANE (FRENCH)
α-TRICHLOROETHANE
1,1,1-TRICLOROETANO (ITALIAN)
TRI-ETHANE

TOXICITY DATA:	CODEN:
eye-man 450 ppm/8H	BJIMAG 28,286,71
skn-rbt 5 g/12D-I MLD	AIHAAP 19,353,58
skn-rbt 500 mg/24H MOD	28ZPAK -,28,72
eye-rbt 100 mg MLD	AIHAAP 19,353,58
eye-rbt 2 mg/24H SEV	28ZPAK -,28,72
dnr-esc 500 mg/L	PMRSDJ 1,195,81
otr-mus:emb 20 mg/L	CALEDQ 28,85,85
orl-rat TDLo:43 mg/kg (1-22D preg/21D post):TER	TJADAB 29(2),25A,84
ihl-rat TCLo:2100 ppm/24H (14D pre/1-20D preg):TER	TOXID9 1,28,81
ihl-man LCLo:27 g/m^3/10M	JOCMA7 8,358,66
ihl-man TCLo:350 ppm:CNS	WEHSAL 10,82,73
orl-hmn TDLo:670 mg/kg:GIT	NTIS** PB257-185
ihl-hmn TCLo:920 ppm/70M: EYE,CNS	AIHAAP 19,353,58
ihl-man TCLo:200 ppm/4H:CNS	ATSUDG 5,96,82
orl-rat LD50:10300 mg/kg	NTIS** PB257-185
ihl-rat LC50:18000 ppm/4H	28ZPAK -,28,72
ipr-rat LD50:5100 mg/kg	NTIS** PB257-185
orl-mus LD50:11240 mg/kg	NTIS** PB257-185
ihl-mus LC50:3911 ppm/2H	SAIGBL 13,226,71
ipr-mus LD50:4700 mg/kg	TXAPA9 13,287,68
orl-dog LD50:750 mg/kg	FMCHA2 -,C242,83
ipr-dog LD50:3100 mg/kg	TXAPA9 10,119,67
ivn-dog LDLo:95 mg/kg	HBTXAC 5,72,59
ihl-cat LCLo:600 mg/m^3/4H	85GMAT -,38,82
orl-rbt LD50:5660 mg/kg	AIHAAP 19,353,58
skn-rbt LDLo:1 g/kg	85GMAT -,38,82
scu-rbt LDLo:500 mg/kg	HBTXAC 5,72,59
orl-gpg LD50:9470 mg/kg	AIHAAP 19,353,58

IARC Cancer Review: Animal Inadequate Evidence IMEMDT 20,515,79. NCI Carcinogenesis Bioassay (gavage); Inadequate Studies: mouse, rat NCITR* NCI-CG-TR-3,77. Community Right To Know List. Reported in EPA TSCA Inventory. EPA Genetic Toxicology Program.

OSHA PEL: TWA 350 ppm
ACGIH TLV: TWA 350 ppm; STEL 450 ppm
DFG MAK: 200 ppm (2080 mg/m^3); BAT: blood 55 μg/dl
NIOSH REL: (1,1,1-Trichloroethane) CL 350 ppm/15M

DOT Classification: ORM-A; Label: None; Poison B; Label: St Andrews Cross

THR: Poison by intravenous route. Moderately toxic by ingestion, inhalation, skin contact, subcutaneous and intraperitoneal routes. An experimental teratogen. Human systemic effects by ingestion and inhalation: conjunctiva irritation, hallucinations or distorted perceptions, motor activity changes, irritability, aggression, hypermotility, diarrhea, nausea or vomiting and other gastrointestinal changes. Experimental reproductive effects. Mutagenic data. A human skin irritant. An experimental skin and severe eye irritant. Narcotic in high concentrations. Causes a proarrhythmic activity which sensitizes the heart to epinephrine-induced arrhythmias. This sometimes will cause cardiac arrest, particularly when this material is massively inhaled as in drug abuse for euphoria.

Under the proper conditions it can undergo hazardous reactions with aluminum oxide + heavy metals; dinitrogen tetraoxide; inhibitors; metals (e.g., magnesium; aluminum; potassium: potassium-sodium alloy); sodium hydroxide; N_2O_4; oxygen. When heated to decomposition it emits toxic fumes of Cl^-. Used as a cleaning solvent, a chemical intermediate to produce vinylidene chloride, and as a propellant in aerosol cans. See also CHLORINATED HYDROCARBONS, ALIPHATIC. For further information see methyl chloroform, Vol. 2, No. 5 of *DPIM Report*.

LCE000 *HR: 3*

LEAD

CAS: 7439-92-1 NIOSH: OF 7525000

af: Pb aw: 207.19

PROP: Bluish-gray, soft metal. Mp: 327.43°, bp: 1740°, d: 11.34 @ 20°/4°. vap press: 1 mm @ 973°.

SYNS:

C.I. 77575
C.I. PIGMENT METAL 4
GLOVER
LEAD FLAKE
LEAD S2
OLOW (POLISH)
OMAHA
OMAHA & GRANT
SI
SO

TOXICITY DATA:	CODEN:
cyt-hmn-unr 50 $\mu g/m^3$	MUREAV 147,301,85
cyt-rat-ihl 23 $\mu g/m^3$/16W	GTPZAB 26(10),38,82
cyt-mky-orl 42 mg/kg/30W	TOLED5 8,165,81
orl-rat TDLo:790 mg/kg (MGN):REP	AEHLAU 23,102,71
orl-rat TDLo:1140 mg/kg (14D pre-21D post):REP	PHMCAA 20,201,78
orl-rat TDLo:1100 mg/kg (1-22D preg):TER	FEPRA7 37,895,78
ihl-rat TCLo:10 mg/m^3/24H (1-21D preg):TER	ZHPMAT 165,294,77
orl-wmn TDLo:450 mg/kg/6Y: PNS:CNS	JAMAAP 237,2627,77
ihl-hmn TCLo:10 $\mu g/m^3$:GIT: LIV	VRDEA5 (5),107,81
ipr-rat LDLo:1000 mg/kg	EQSSDX 1,1,75
orl-pgn LDLo:160 mg/kg	HBAMAK 4,1289,35

IARC Cancer Review: Animal Inadequate Evidence IMEMDT 23,325,80. Lead and its compounds are on the Community Right To Know List. Reported in EPA TSCA Inventory. EPA Genetic Toxicology Program.

OSHA PEL: TWA 0.05 mg(Pb)/m^3
ACGIH TLV: TWA 0.15 mg(Pb)/m^3
NIOSH REL: TWA (Inorganic Lead) 0.10 mg(Pb)/m^3

THR: Poison by ingestion. Moderately toxic by intraperitoneal route. It is a suspected carcinogen of the lungs and kidneys. Human systemic effects by ingestion and inhalation: loss of appetite, anemia, malaise, insomnia, headache, irritability, muscle and joint pains, tremors, flaccid paralysis without anesthesia, hallucinations and distorted perceptions, muscle weakness, gastritis and liver changes. The major organ systems affected are the nervous system, blood system, and kidneys. Lead encephalopathy is accompanied by severe cerebral edema, increase in cerebral spinal fluid pressure, proliferation and swelling of endothelial cells in capillaries and arterioles, proliferation of glial cells, neuronal degeneration and areas of focal cortical necrosis in fatal cases. Experimental evidence now suggests that blood levels of lead below 10 μg/dl can have the effect of diminishing the IQ scores of children. Low levels of lead impair neurotransmission and immune system function and may increase systolic blood pressure. Reversible kidney damage can occur from acute exposure. Chronic exposure can lead to irreversible vascular schlerosis, tubular cell atrophy, interstitial fibrosis, and glomerular sclerosis. Severe toxicity can cause sterility, abortion and neonatal mortality and morbidity. An experimental teratogen. Experimental reproductive effects. Human mutagenic data. Very heavy intoxication can sometimes be detected by formation of a dark line on the gum margins, the so-called "lead line."

When lead is ingested, much of it passes through the body unabsorbed, and is eliminated in the feces. The greater portion of the lead that is absorbed is caught by the liver and excreted, in part, in the bile. For this reason, larger amounts of lead are necessary to cause toxic effects by this route, and a longer period of exposure is usually necessary to produce symptoms. On the other hand, upon inhalation, absorption takes place easily from the respiratory tract and symptoms tend to develop more quickly. For industry, inhalation is much more important than is ingestion. For the general population, exposure to lead occurs from inhaled air, dust of various types, and food and water with an approximate 50/50 division between inhalation and ingestion routes. Lead occurs in water in either dissolved or particulate form. At low pH, lead is more easily dissolved. Chemical treatment to soften water increases the solubility of lead. Adults absorb about 5-15% of ingested lead and retain less than 5%. Children absorb about 50% and retain about 30%.

Lead produces a brittleness of the red blood cells so that they hemolyze with but slight trauma; the hemoglobin is not affected. Due to their increased fragility, the red cells are destroyed more rapidly in the body than is normal, producing an anemia which is rarely severe. The loss of circulating red cells stimulates the production of new young cells which, on entering the blood stream, are acted upon by the circulating lead, with resultant coagulation of their basophilic material. These cells after suitable staining, are recognized as "stippled cells." There is no uniformity of opinion regarding the effect of lead on the white blood cells.

In addition to its effect on the red blood cells, lead produces a damaging effect on the organs or tissues with which it comes in contact. No specific or characteristic lesion is produced. Autopsies in deaths attributed to lead poisoning and experimental work on animals have shown pathological lesions of the kidneys, liver, male gonads, nervous system, blood vessels and other tissues. None of these changes, however, has been found consistently. In cases of severe lead poisoning, the amount of lead found in the blood is frequently in excess of 0.07 mg per 100 cc of whole blood. The urinary lead excretion generally exceeds 0.1 mg per liter of urine.

Flammable in the form of dust when exposed to heat or flame. Moderately explosive in the form of dust when exposed to heat or flame. Mixtures of hydrogen peroxide + trioxane explode on contact with lead. Rubber gloves containing lead may ignite in nitric acid. Violent reaction on ignition with chlorine trifluoride; concentrated hydrogen peroxide; ammonium nitrate (below 200°C with powdered lead); sodium acetylide (with powdered lead). Incompatible with NaN_3; Zr; disodium acetylide; oxidants. Can react vigorously with oxidizing materials. A common air contaminant. When heated to decomposition it emits highly toxic fumes of Pb. See also LEAD COMPOUNDS. For further information, see Vol. 1, No. 1 of *DPIM Report*.

LEAD COMPOUNDS

Lead and its compounds are on the Community Right To Know List.

THR: Lead poisoning is one of the commonest of occupational diseases. The presence of lead-bearing materials or lead compounds in an industrial plant does not necessarily result in exposure on the part of the worker. The lead must be in such form, and so distributed, as to gain entrance into the body or tissues of the worker in measurable quantity, otherwise no exposure can be said to exist. Some lead compounds are carcinogens of the lungs and kidneys. Others are experimental neoplastigens and tumorigens.

Mode of entry into body: 1. By inhalation of the dust, fumes, mists or vapors. (Common air contaminants). 2. By ingestion of lead compounds trapped in the upper respiratory tract or introduced into the mouth on food, tobacco, fingers or other objects. 3. Through the skin; this route is of special importance in the case of organic compounds of lead, as lead tetraethyl. In the case of the inorganic forms of lead, this route is of no practical importance. Significant quantities of lead can be ingested from water that has been sitting in pipes with lead solder. Some water coolers may also have this type of solder.

Lead is a cumulative poison. Increasing amounts build up in the body and eventually reach a point where symptoms and disability occur. See LEAD for symptoms of overexposure.

The toxicity of the various lead compounds appears to depend upon several factors: (1) the solubility of the compound in the body fluids; (2) the fineness of the particles of the compound; solubility is greater in proportion to the fineness of the particles; (3) conditions under which the compound is being used. Where a lead compound is used as a powder, contamination of the atmosphere will be much less if the powder is kept damp. Of the various lead compounds, the carbonate, the monoxide, and the sulfate are considered to be more toxic than metallic lead or other lead compounds. Lead arsenate is very toxic due to the presence of the arsenic radical. Organolead compounds are rapidly absorbed by the respiratory and gastrointestinal systems and through the skin. Tetraethyl lead is converted in the body to triethyl lead which is a more severe neurotoxin than inorganic lead. Diagnostic mobilization of lead with calcium EDTA may be useful in questionable cases. When heated to decomposition they emit toxic fumes of Pb. See also LEAD and specific compounds.

LEAD

CAS: 7439-92-1

Pb

Inorganic Compounds, Dust and Fume

TLV-TWA, 0.15 mg/m^3, as Pb*

Lead, atomic number 82, is metallic element in Group IVB of the periodic table. It is heavy, ductile, and bluish-white in color. Its physiochemical properties include:

Atomic weight: 207.2
Specific gravity: 11.35 at 20°C
Melting point: 327.5°C
Boiling point: 1740°C
Vapor pressure: significant above 500°C (1.77 torr at 1000°C)

Only a few lead compounds are appreciably soluble in water, but many are dissolved by acids and most are sufficiently soluble in body fluids to be toxic, especially when inhaled in finely divided form.

Metallic lead finds wide industrial use where its properties of high density, softness, low melting point, resistance to corrosion and/or opacity to gamma and X-rays are needed. It is a major component of many alloys such as solder, type metal, and many bronzes. Lead compounds have a wide variety of uses, especially as paint pigments, in storage batteries and ceramics.

Despite the tremendous importance of lead as an occupational hazard, only a handful of papers in the voluminous literature on lead poisoning present meaningful data relating to the threshold limit value. The chief reason for this situation is probably the fact that most authorities rely primarily, if not exclusively, on other tests for estimation of the degree of lead hazard. Urinary and blood leads, urinary coproporphyrin and delta aminolevulinic acid, as well as blood examination for stippled cells and other abnormalities, are among the preferred procedures.

A limit of 0.5 mg/m^3 for lead in air was proposed by Legge in 1912, with the comment that, if adhered to, cases of encephalopathy and paralysis would never, and cases of colic would very rarely, occur.(1) The data of Duckering's experiments on the quantities of lead in the air from various industrial processes are given as evidence.(2) This value (0.5 mg/m^3) was quoted by Alice Hamilton in 1925, with a similar comment.(3)

In 1933 Russell *et al*,(4) following a U.S. Public Health Service survey of a lead storage battery plant, proposed a limit of 0.15 mg/ for lead dust and fume in this industry. Eight years later Dreessen et al(5) published results of a follow-up study and considered that their findings confirmed this value. In 1943 Kehoe and other members of the Committee on Lead Poisoning of the American Public Health Association recommended 0.15 mg/m^3, as a time-weighted average, limit.(6)

A number of investigators found the 0.15 mg/m^3 value difficult to achieve in many industries, and observation of workers, combined with lead urinalysis and similar studies convinced them that this limit was unnecessarily stringent. Winn and Shroyer(7) concluded that maintenance of the average concentration of lead dust and fume at or below 0.5 mg/m^3, combined with a medical program, would assure adequate control. Weber(8) considered the 0.15 mg/m^3 too low, but stipulated that 0.3 mg/m^3 should not be exceeded (as time-weighted average). He found that an atmospheric concentration of 0.43 mg/m^3 corresponded to 0.20 mg/L of urine, a level considered by some investigators to represent the upper limit of safety. Elkins(9) assembled the data available on lead in air and lead in urine and concluded that a urinary lead concentration of 0.20 mg/L would, on the average, correspond to an air-lead value of 0.20 mg/m^3.

On the basis of these reports and unpublished data from several sources, the TLV for lead was increased from 0.15 to 0.20 mg/m^3 in 1957. Some authorities continued to use the previous limit, however.(10) Schrenk(11) implied that the 0.15 mg/m^3 value was to be preferred. The preponderance of American opinion, however, seemed to be that the 0.2 mg/m^3 limit was adequate to prevent episodes of lead intoxication. Thus Kehoe,(12) in a discussion of threshold limits for lead, stated that:

> *"Evidence of the validity of the standard (0.2 mg/m^3) has been provided elsewhere and need not be enlarged upon here."*

He went on to warn that this value is adequate only if ingestion of lead is prevented. Johnstone and Miller(13) referred to the 0.2 mg/m^3 limit as generally accepted.

More recent comparisons of atmospheric and urinary lead concentrations have indicated conflicting results. Berg and Zenz,(14) in a foundry study, found that air-lead concentrations between 0.14 and 0.18 mg/m^3 resulted in urinary lead values below 0.15 mg/L; 0.28 mg/m^3 was associated with 0.17 mg/L of urine.

Tsuchiya and Harashima(15) concluded that for a 48- to 60-hour work week, an average air-lead concentration of 0.10 mg/m^3 would bring about an average urinary lead level of 0.15 mg/L; and 0.12 mg/m^3 to 0.20 mg/L. Concentrations of 0.12 to 0.14 mg/m^3 resulted in increased urinary coproporphyrin, some stippling of blood cells and anemia.

Most extensive lead exposure studies have involved lead oxide dust or the fume of metallic lead. Some reports have indicated that the dusts of certain insoluble lead compounds, such as the sulfide(16) and chromate, were less hazardous than more soluble forms of lead. Thus, Harrold and associates(17,18) studied a group of painters exposed to mists of lead chromate in concentrations averaging between 1.2 and 12 mg of lead per cubic meter of air, and found little evidence of lead absorption or intoxication. They also suggested that lead titanate would present relatively little hazard, due to its very low solubility.

On the other hand, Hartogenesis and Zielhuis(19) found blood changes in workers exposed to lead chromate dust at levels above 0.2 mg/m^3 (as lead) and doubtful changes between 0.1 and 0.2 mg/m^3. They consider that the TLV for lead chromate should be the same as that for other inorganic lead compounds.

Curiously, there is evidence that lead fume is less harmful than equal amounts of the dust of relatively soluble lead compounds.(20) This is presumed to be due to a lesser retention of the extremely fine particles present in the fume.

---

* In 1984 the STEL was placed on the Notice of Intended Changes as a deletion with the TWA value retained.

The International Subcommittee for Occupational Health of the Permanent Commission and International Association of Occupational Health, at a meeting in Amsterdam in November 1968, recommended a limit of 0.15 mg/m³ for a 40-hour week. This conclusion represented the concensus of 20 experts from 12 nations.(21,22)

In an extremely thorough study of atmospheric lead exposures and biochemical criteria, Williams et al(23) found among 39 battery workers in England high correlation coefficients between air concentrations and blood lead (r = 0.9): urinary lead (r = 0.82): urinary coproporphyrins (r = 0.82) and urinary dALA (r = 0.68). Lower correlations were found for punctate (stippled) basophilic count (r = 0.45) and percent hemoglobin (r = 0.09). Furthermore, they observed that in every case the upper 95% confidence limit considerably exceeded the safe limits, when the air limit is 0.2 mg/m³, but approximates it when the air limit is 0.15 mg/m³.

In view of these data using improved biochemical indicators of lead exposure, clearly showing that the TLV of 0.2 mg/m³ had little or no margin of safety for some workers, the limit was reduced back to 0.15 mg/m³ in 1971.

In its first criteria document on inorganic lead, published in 1972, NIOSH recommended the 0.15 mg/m³ TLV as a workplace standard,(24) but emphasized that reliance should be placed primarily on biological measurements, especially blood lead, for which the limit of 0.08 mg/100 grams was endorsed. A revised document appeared in 1978, however, in which a lower limit, 0.1 mg/m³, was proposed.(25) The maximum permissible blood lead level was also reduced, to 0.06 from 0.08 mg/100 grams.

Emphasis in the document is placed on findings of adverse effects among workers with blood leads below 0.08 mg/100 grams, but generally above 0.06 mg.

Although the updated document contains 185 additional references (most published since 1971), only five relate directly to atmospheric lead concentrations, and these are all given as support for the amazing statement that "it has been shown that 1 µg lead/m³ in air contributes about 1-2 µg lead/100 grams of blood." Amazing, that is, until examination of the references indicates that four of them deal with continuous exposures of the public, or volunteers, to lead in air levels of the order of 0.01 mg/m³ or less. Only one(26) related to occupational exposure; a mean lead in air concentration in one department of a rubber hose and tire company in Japan of 0.0579 mg/m³ (based on 34 tests) was associated with a mean blood lead level, in 20 workers, of 51.8 µg/100 grams.

In addition, testimony of the Deputy Director of NIOSH at an OSHA hearing refers to an unpublished battery plant study in which average exposures of workers, using personal monitors, were below 0.1 mg/m³ in all departments except pasting and grid casting, where exposures were generally below 0.15 mg/m³.(27) Blood levels in over 90% of the workers were 60 µg/100 grams or less.

The findings of these two reports are hardly adequate to justify the proposed reduction in the limit for lead in workroom air.

The papers on effects associated with blood lead levels below 80 µg/100 grams are also few in number. Findings of changes in urinary ALA and coproporphyrin, erythrocyte protoporphyrin and zinc protoporphyrin in blood, hemoglobin decreases, and altered spermatogenesis are reported in conjunction with likely "excessive absorption," as evidenced by blood leads between 40 and 60 µg/100 grams. The proposed standard apparently would not recognize these effects as inconsistent with a satisfactory state of health. Unacceptable lead absorption, with blood leads in excess of 60 µg/100 grams (mostly, but not entirely, below 80 µg) are associated with CNS effects, peripheral neuropathy, gastrointestinal disturbances and anemia, according to one reference.(28) Another paper(29) cited reported evidence of renal damage in six of thirteen workers, one with a blood lead of 98 µg/100 grams, one with 66 µg, and the remainder below 60 µg/100 grams of blood. An unpublished NIOSH report(30) found renal damage and anemia in similarly exposed (blood leads above 60 µg/100 grams, but presumably not over 80 µg) workers, but no details are given.

Perhaps the strongest case for the reduced limit is presented in a paper on nerve conduction velocities,(31) in which decreases (mostly minimal, but in one system significant) were found in workers with maximal blood leads between 50 and 70 µg/100 grams. The authors felt that these findings were more serious than the alterations in heme synthesis, demonstrated by biochemical measurements, since the regenerative capacity of the nervous system is relatively slow.

The Committee is not convinced that the biochemical changes found due to low level lead absorption are incompatible with good health. It has not adopted, or proposed, a biologic TLV for lead, nor has it accepted the NIOSH hypothesis that an air TLV must be set at a level at which most workers (i.e., 90-95%) do not exceed a specified biologic TLV.

In view of the notation in the title of the consultant's review of the recent literature in the revised NIOSH document(25) that it is to "support the update" of the criteria document, one wonders if the citations are chosen and their contents summarized without bias.

For the present, the time-weighted average TLV of 0.15 mg lead/m³ in air is retained. However, the Committee recommends, at this time, the elimination of the STEL until additional toxicological data and industrial hygiene experience become available to provide a better base for quantifying on a toxicological basis what the STEL should be. The reader is encouraged to review the section on *Excursion Limits* in the Introduction to the Chemical Substances of the current TLV booklet for guidance and control of excursions above the TLV-TWA, even when the 8-hour TWA is within the recommended limits.

• Other recommendations: The American National Standard Institute's Z-37 Committee established 0.2 mg/m³ as its acceptable concentration for lead in 1969. Smyth (1956) suggested that even the 0.15 mg/m³ value was not low enough to prevent mild intoxication. More recent values are: USSR (1977) 0.01 mg/m³; Hungary (1974) 0.02 mg/m³; Czechoslovakia (1976), Poland (1976) and OSHA (1978) 0.05 mg/m³; Romania (1975), Sweden (1975) and West Germany (1978) 0.1 mg/m³; East Germany (1973), Finland (1975) and Yugoslavia (1971) 0.15 mg/m³.

References

1. **Legge, T.M. and K.W. Gadby:** *Lead Poisoning and Lead Absorption*, p. 207. Edward Arnold, London (1962).
2. **Duckering, G.E.:** *J. Hyg.* 7:474 (1908).
3. **Hamilton, A.:** *Industrial Poisons in the U.S.*, p. 57. MacMillan, New York (1925).
4. **Russell, A.E., R.R. Jones, J.J. Bloomfield et al: Britten, R.H., Thompson, L.R.:** *Public Health Bull.* No. 205 (1933).
5. **Dreessen, W.C., T.L. Edwards, W.H. Reinhart et al:** *Public Health Bull.* No. 269 (1941).
6. **American Public Health Assoc.:** Report of Committee on Lead Poisoning, New York (1943).
7. **Winn, G.S. and C. Shroyer:** *J. Ind. Hyg. Tox.* 29:351 (1947).
8. **Weber, H.J.:** *Hygiene Conference*, p. 12. Lead Industries Assoc., New York (1948).
9. **Elkins, H.B.:** *Chemistry of Industrial Toxicology*, p. 56. Wiley & Sons, New York (1959).

10. **Michigan Dept. of Health:** *Occup. Health 7(4)*:3 (1962).
11. **Schrenk, H.H.:** *Proceedings of Lead Hygiene Conference*, p. 19. Lead Industries Assoc., Chicago, IL (1958).
12. **Patty, F.A.:** *Industrial Hygiene and Toxicology*, 2nd ed., Vol. II, p. 953. Interscience, New York (1963).
13. **Johnstone, R.T. and S.E. Miller:** *Occupational Diseases and Industrial Medicine*, p. 297. W.B. Saunders, Philadelphia, PA (1960).
14. **Berg, B.A. and C. Zenz:** *Am. Ind. Hyg. Assoc. J. 29*:175 (1967).
15. **Tsuchiya, K. and S. Harashima:** *Brit. J. Ind. Med. 22*:181 (1965).
16. **Belden, E.A. and L.F. Garber:** *J. Ind. Hyg. Tox. 31*:437 (1949).
17. **Harrold, G.C., S.F. Meek, G.R. Collins and T.F. Merkell:** *Ibid. 26*:47 (1944).
18. **Harrold, G.C. and S.F. Meek:** *Ind. Med. Surg. 18*:407 (1949).
19. **Hartogensis, F. and R.L. Zielhuis:** *Ann. Occup. Hyg. 5*:27 (1962).
20. **Frederick, W.G.:** Meeting for review of TLV for Inorganic Lead, Detroit, MI (May 11, 1970).
21. **Zielhuis, R.L.:** *T. Soc. Geneesk. 47*:743 (1969).
22. **Subcommittee Reports:** *Ind. Med. Surg. 38*:10 (September 1969).
23. **Williams, M.K., E. King and J. Walford:** *Brit. J. Ind. Med.* 26:202 (1969).
24. **NIOSH:** *Criteria for a Recommended Standard — Occupational Exposure to Inorganic Lead.* Pub. No. HSM 73-11010 (1972).
25. **NIOSH:** *Revised Criteria for a Recommended Standard — Occupational Exposure to Inorganic Lead.* DHEW Pub. No. (NIOSH) 78-158 (1978).
26. **Sakurai, H. et al:** *Arch. Env. Health 29*:157 (1974).
27. **Baier, E.J.:** Appendix V, p. XII-1 of reference 25.
28. **Mount Sinai School of Medicine of the City University of New York:** Lead Disease Among Workers in Secondary Lead Smelters. Report by the Environmental Sciences Laboratory to the National Inst. of Environmental Health Sciences (1976). Cited in ref. 25.
29. **Wedeen, R.P. et al:** *Am. J. Med. 59*:630 (1975). *Ibid.*
30. **DHEW, Bureau of Epidemiology & NIOSH:** A report of recent medical studies of five U.S. lead plants (1977). *Ibid.*
31. **Seppaleinen, A.M.:** *Arch. Env. Health 30*:180 (1975).

DFF809 ***HR: 3***

1,1-DICHLOROETHANE

CAS: 75-34-3 NIOSH: KI 0175000
DOT: 2362
mf: $C_2H_4Cl_2$ mw: 98.96

PROP: Lel: 5.6%, uel: 11.4%.

SYNS:

AETHYLIDENCHLORID (GERMAN)
CHLORINATED HYDROCHLORIC ETHER
CHLORURE d' ETHYLIDENE (FRENCH)
CLORURO DI ETILIDENE (ITALIAN)
1,1-DICHLOORETHAAN (DUTCH)
1,1-DICHLORAETHAN (GERMAN)
1,1-DICHLORETHANE
1,1-DICLOROETANO (ITALIAN)
ETHYLIDENE CHLORIDE
ETHYLIDENE DICHLORIDE
NCI-C04535
RCRA WASTE NUMBER U076

TOXICITY DATA:	CODEN:
ihl-rat TCLo:6000 ppm/7H (6-15D preg):TER	TXAPA9 28,452,74
orl-mus TDLo:185 g/kg/78 W-I: ETA	NCITR* NCI-CG-TR-66,78
orl-mus TD :1300 g/kg/78 W-I: ETA	NCITR* NCI-CG-TR-66,78
orl-rat LD50:725 mg/kg	HYSAAV 32,349,67

EPA TSCA Chemical Inventory. NCI Carcinogenesis Bioassay (gavage); Inadequate Studies: mouse, rat NCITR* NCI-CG-TR-66,78.

OSHA PEL: TWA 100 ppm

DOT Classification: Flammable Liquid; Label: Flammable Liquid

THR: Moderately toxic by ingestion. An experimental tumorigen and teratogen. A suspected carcinogen. When heated to decomposition it emits very toxic fumes of Cl^-. See also 1,2-DICHLOROETHANE; and CHLORINATED HYDROCARBONS, ALIPHATIC.

1,1-DICHLOROETHANE

CAS: 75-34-3

Ethylidene chloride

CH_3HCl_2

TLV-TWA, 200 ppm (≈ 810 mg/m³)

TLV-STEL, 250 ppm (≈ 1010 mg/m³)

1,1-Dichloroethane is a colorless, oily liquid which has an odor and taste of chloroform. Its physiochemical properties include:

Molecular weight: 98.97
Specific gravity: 1.1757 at 20°C
Melting point: −96.98
Boiling point: 57.3°C
Vapor pressure: 182 torr at 20°C
Closed cup flash point: 17°F (−8.33°C)
Explosive limits: 6% and 16% by volume in air

It is a fire hazard. Very soluble in alcohol and ether, it is soluble in acetone, benzene, and in 200 parts water.

1,1-Dichloroethane has limited use as a solvent and as a chemical intermediate. Formerly used as an anesthetic, it is of no importance in this field today.

Smyth(1) found that rats survived eight hours at 400 ppm, but were killed by 16,000 ppm. Few published reports are available on the chronic toxicity of this material and industrial usage is not extensive.

> *"However, recent detailed, chronic studies indicate that 1,1-dichloroethane has little capacity for causing liver damage, being similar to methylene chloride and 1,1,1-trichloroethane in this respect. Rats, guinea pigs, rabbits, and dogs were exposed to either 500 or 1000 ppm for seven hours per day, five days per week, for six months. Gross and microscopic pathological and hematological studies showed no evidence of changes attributable to the exposure."(2)*

In a limited study, Hofmann et al(3) have confirmed the low hepatotoxicity of 1,1-dichloroethane.

Based on these data, the suggested TLV for 1,1-dichloroethane of 200 ppm, as a time-weighted average, should provide a wide margin of safety against organic injury from exposure to 1,1-dichloroethane, with a STEL of 250 ppm. The margin of safety against pronounced anesthetic effects is not yet known.

References

1. **Smyth, H.F., Jr.:** *Am. Ind. Hyg. Assoc. Q. 17*:129 (1956).
2. **American Industrial Hygiene Assoc.:** *Hygienic Guide — 1,1-Dichloroethane*, Quoting unpublished data by the Dow Chemical Co., Midland, MI. AIHA, Akron, Ohio (Rev. January 1971).
3. **Hofmann, H.T., H. Birnstiel and P. Jobst:** *Arch. Pharmakol. 266(4/5)*:360-361 (1970).

1,1-DICHLOROETHANE

CAS: 75-34-3
CH_3CHCl_2
1991 TLV-TWA = 200 ppm (810 mg/m^3)
TLV-STEL = 250 ppm (1010 mg/m^3)

Synonym: Ethylidene dichloride

Physical Form. Colorless liquid

Uses. Cleansing agent; degreaser; solvent for plastics, oils, and fats; grain fumigant; chemical intermediate; former anesthetic

Exposure. Inhalation

Toxicology. At high concentrations 1,1-dichloroethane causes central nervous system depression.

There have been no reported cases of human overexposure by inhalation. In the past, 1,1-dichloroethane was used as an anesthetic at levels of approximately 25,000 ppm.[1] This use was discontinued when it was discovered that cardiac arrhythmias might be induced. Cardiovascular toxicity has not been reported in animals following exposure.

Rats exposed to 32,000 ppm for 30 minutes survived but they died after 2.5 hours of exposure.[2] The most consistent findings in animals exposed to concentrations of above 8000 ppm for up to 7 hours were pathological changes in the kidney and the liver, and at much higher concentrations, near 64,000 ppm, damage to the lungs as well. No adverse clinical effects were noted in rats, rabbits, or guinea pigs exposed to 1000 ppm for 13 weeks, which followed a prior 13 week exposure to 500 ppm.[3] Under the same conditions renal injury was apparent in cats, as evidenced by increased serum urea and creatinine levels.

No histopathological alterations were noted in the liver, kidneys, or lungs of male mice that ingested up to 2500 mg/liter 1,1-dichloroethane in drinking water for 52 weeks.[4]

A significant increase in endometrial stromal polyps, a benign neoplasm, occurred in female mice administered up to 3.3 g/kg/day 1,1-dichloroethane by gavage for 78 weeks.[5] There was also a dose-related trend for the incidence of hemangiosarcomas and mammary adenocarcinomas in female rats and hepatocellular carcinoma in male mice. High mortality in all animal groups obscured results. The National Cancer Institute determined that there was no conclusive evidence for carcinogenicity, but 1,1-dichloroethane should be treated with caution by analogy to other chloroethanes shown to be carcinogenic

Applied to the intact or abraded skin of rabbits, the liquid produced slight edema and very slight necrosis after the sixth of ten daily applications. When it was instilled in the eyes of rabbits, there was immediate, moderate conjunctival irritation and swelling, which subsided within a week.[2]

Although the liquid may be absorbed through the skin, it apparently is not absorbed in amounts sufficient to produce systemic injury.

Exposure of rats to 6000 ppm 7 hours/day on days 6 through 15 of gestation was associated with an increased incidence of delayed ossification of sternebrae.[2] Maternal toxicity was limited to decreased weight gain

Odor cannot be relied upon to warn of overexposure.

REFERENCES

1. Browning E: *Toxicity and Metabolism of Industrial Solvents*, pp 247–252. New York, Elsevier Science Pub Co, Inc, 1965
2. Hygienic Guide Series: 1,1-Dichloroethane (ethylidene chloride). *Am Ind Hyg Assoc J* 32:67–71, 1971
3. Hofmann HT, Birnstiel H, Jobst P: [Inhalation toxicity of 1,1 and 1,2 dichloroethane.] *Arch Toxikol* 27:248–265, 1971 (German)
4. Klaunig JE, Ruch RJ, Pereira MA: Carcinogenicity of chlorinated methane and ethane compounds administered in drinking water to mice. *Environ Health Perspect* 69:89–95, 1986
5. National Cancer Institute: Carcinogenesis Technical Report Series No. 66. Bioassay of 1,1-dichloroethane for possible carcinogenicity, NCI-CG-TR-66, p 82. DHEW (NIH) Pub No 78-1316, 1978
6. NIOSH: *Current Intelligence Bulletin 27, Chloroethanes: Review of Toxicity*, p 22. DHEW (NIOSH) Pub No. 78-181, 1978
7. Schwetz BA et al: Embryo and fetoxicity of inhaled carbon tetrachloride, 1,1-dichloroethane and methyl ethyl ketone in rats. *Toxicol Appl Pharmacol* 28:452–464, 1974

1,1-DICHLOROETHANE

CAS: 75-34-3

CH_3CHCl_2

1991 TLV-TWA = 200 ppm (810 mg/m^3)

TLV-STEL = 250 ppm (1010 mg/m^3)

Synonym: Ethylidene dichloride

Physical Form. Colorless liquid

Uses. Cleansing agent; degreaser; solvent for plastics, oils, and fats; grain fumigant; chemical intermediate; former anesthetic

Exposure. Inhalation

Toxicology. At high concentrations 1,1-dichloroethane causes central nervous system depression.

There have been no reported cases of human overexposure by inhalation. In the past, 1,1-dichloroethane was used as an anesthetic at levels of approximately 25,000 ppm.[1] This use was discontinued when it was discovered that cardiac arrhythmias might be induced. Cardiovascular toxicity has not been reported in animals following exposure.

Rats exposed to 32,000 ppm for 30 minutes survived but they died after 2.5 hours of exposure.[2] The most consistent findings in animals exposed to concentrations of above 8000 ppm for up to 7 hours were pathological changes in the kidney and the liver, and at much higher concentrations, near 64,000 ppm, damage to the lungs as well. No adverse clinical effects were noted in rats, rabbits, or guinea pigs exposed to 1000 ppm for 13 weeks, which followed a prior 13 week exposure to 500 ppm.[3] Under the same conditions renal injury was apparent in cats, as evidenced by increased serum urea and creatinine levels.

No histopathological alterations were noted in the liver, kidneys, or lungs of male mice that ingested up to 2500 mg/liter 1,1-dichloroethane in drinking water for 52 weeks.[4]

A significant increase in endometrial stromal polyps, a benign neoplasm, occurred in female mice administered up to 3.3 g/kg/day 1,1-dichloroethane by gavage for 78 weeks.[5] There was also a dose-related trend for the incidence of hemangiosarcomas and mammary adenocarcinomas in female rats and hepatocellular carcinoma in male mice. High mortality in all animal groups obscured results. The National Cancer Institute determined that there was no conclusive evidence for carcinogenicity, but 1,1-dichloroethane should be treated with caution by analogy to other chloroethanes shown to be carcinogenic

Applied to the intact or abraded skin of rabbits, the liquid produced slight edema and very slight necrosis after the sixth of ten daily applications. When it was instilled in the eyes of rabbits, there was immediate, moderate conjunctival irritation and swelling, which subsided within a week.[2]

Although the liquid may be absorbed through the skin, it apparently is not absorbed in amounts sufficient to produce systemic injury.

Exposure of rats to 6000 ppm 7 hours/day on days 6 through 15 of gestation was associated with an increased incidence of delayed ossification of sternebrae.[2] Maternal toxicity was limited to decreased weight gain

Odor cannot be relied upon to warn of overexposure.

REFERENCES

1. Browning E: *Toxicity and Metabolism of Industrial Solvents*, pp 247–252. New York, Elsevier Science Pub Co, Inc, 1965
2. Hygienic Guide Series: 1,1-Dichloroethane (ethylidene chloride). *Am Ind Hyg Assoc J* 32:67–71, 1971
3. Hofmann HT, Birnstiel H, Jobst P: [Inhalation toxicity of 1,1 and 1,2 dichloroethane.] *Arch Toxikol* 27:248–265, 1971 (German)
4. Klaunig JE, Ruch RJ, Pereira MA: Carcinogenicity of chlorinated methane and ethane compounds administered in drinking water to mice. *Environ Health Perspect* 69:89–95, 1986
5. National Cancer Institute: Carcinogenesis Technical Report Series No. 66. Bioassay of 1,1-dichloroethane for possible carcinogenicity, NCI-CG-TR-66, p 82. DHEW (NIH) Pub No 78-1316, 1978
6. NIOSH: *Current Intelligence Bulletin 27, Chloroethanes: Review of Toxicity*, p 22. DHEW (NIOSH) Pub No. 78-181, 1978
7. Schwetz BA et al: Embryo and fetoxicity of inhaled carbon tetrachloride, 1,1-dichloroethane and methyl ethyl ketone in rats. *Toxicol Appl Pharmacol* 28:452–464, 1974

DFF900 *HR: 3*

1,2-DICHLOROETHANE

CAS: 107-06-2 NIOSH: KI 0525000
DOT: 1184
mf: $C_2H_4Cl_2$ mw: 98.96

PROP: Colorless liquid, pleasant odor, sweet taste. Bp: 83.5°, ulc: 60-70, lel: 6.2%, uel: 15.9%, fp: −35.7°, flash p: 56°F, d: 1.257 @ 20°/4°, autoign temp: 775°F, vap press: 100 mm @ 29.4°, vap d: 3.35.

SYNS:

AETHYLENCHLORID (GERMAN)
1,2-BICHLOROETHANE
BICHLORURE D'ETHYLENE (FRENCH)
BORER SOL
BROCIDE
CHLORURE D'ETHYLENE (FRENCH)
CLORURO DI ETHENE (ITALIAN)
1,2-DCE
DESTRUXOL BORER-SOL
1,2-DICHLOORETHAAN (DUTCH)
1,2-DICHLOR-AETHAN (GERMAN)
DICHLOREMULSION
DI-CHLOR-MULSION
DICHLORO-1,2-ETHANE (FRENCH)
α,β-DICHLOROETHANE
sym-DICHLOROETHANE
1,2-DICHLOROETHANE
DICHLOROETHYLENE
1,2-DICLOROETANO (ITALIAN)
DUTCH LIQUID
DUTCH OIL
EDC
ENT 1,656
ETHANE DICHLORIDE
ETHYLEENDICHLORIDE (DUTCH)
ETHYLENE CHLORIDE
ETHYLENE DICHLORIDE (ACGIH, DOT)
1,2-ETHYLENE DICHLORIDE
GLYCOL DICHLORIDE
NCI-C00511
RCRA WASTE NUMBER U077

TOXICITY DATA:	CODEN:
skn-rbt 600 mg open MLD	UCDS** 3/23/70
eye-rbt 63 mg SEV	UCDS** 3/23/70
mmo-sat 40 μmol/plate	CBINA8 20,1,78
msc-hmn:lym 100 mg/L	MUREAV 142,133,85
slt-mus-ipr 300 mg/kg	MUREAV 117,201,83
otr-ham:emb 200 μL/plate	EVSRBT 25,75,82
ihl-rat TCLo:300 ppm/7H (6-15D preg):REP	BANRDU 5,149,80
orl-rat TDLo:5286 mg/kg/69W-I:CAR	BANRDU 5,35,80
ihl-rat TCLo:5 ppm/7H/78W-I:ETA	BANRDU 5,3,80
orl-mus TDLo:3536 mg/kg/78W-I:CAR	BANRDU 5,35,80
ihl-mus TCLo:5 ppm/7H/78W-I:ETA	BANRDU 5,3,80
skn-mus TDLo:1120 g/kg/74W-I:NEO	JJIND8 63,1433,79
orl-rat TD :38 g/kg/78W-I:CAR	NCITR* NCI-CG-TR-55,78
orl-mus TD :76 g/kg/78W-I:CAR,TER	NCITR* NCI-CG-TR-55,78
orl-rat TD :18 g/kg/78W-I:CAR	NCITR* NCI-CG-TR-55,78
orl-mus TD :38 g/kg/78W-I:CAR,TER	NCITR* NCI-CG-TR-55,78
ihl-hmn TCLo:4000 ppm/H:CNS,PNS,GIT	PCOC** -,500,66
orl-hmn TDLo:428 mg/kg:GIT,CNS,PUL	SOMEAU 22,132,58
orl-man TDLo:892 mg/kg:GIT,LIV	WILEAR 28,983,75
orl-hmn LDLo:286 mg/kg:GIT,LIV	CLCEAL 86,203,47
orl-man LDLo:714 mg/kg:CNS,CVS,PUL	KLWOAZ 48,822,70
orl-rat LD50:670 mg/kg	FMCHA2 -,C99,83
ihl-rat LC50:1000 ppm/7H	AMIHBC 4,482,51
scu-rat LDLo:99 mg/kg	AMPLAO 51,346,51
orl-mus LD50:489 mg/kg	TOXID9 1,26,81
ihl-mus LCLo:5000 mg/m³/2H	AEPPAE 141,19,29
scu-mus LDLo:380 mg/kg	JPETAB 84,53,45
orl-dog LDLo:2000 mg/kg	QJPPAL 7,205,34
ivn-dog LDLo:175 mg/kg	QJPPAL 7,205,34
orl-rbt LD50:860 mg/kg	GUCHAZ 6,264,73
ihl-rbt LCLo:3000 ppm/7H	JPETAB 84,53,45
skn-rbt LD50:3890 mg/kg	34ZIAG -,744,69
scu-rbt LDLo:1200 mg/kg	QJPPAL 7,205,34
ihl-pig LCLo:3000 ppm/7H	JPETAB 84,53,45
ihl-gpg LCLo:1500 ppm/7H	JPETAB 84,53,45
ipr-gpg LDLo:600 mg/kg	AIHAAP 35,21,74

IARC Cancer Review: Human Limited Evidence IMEMDT 20,429,79; Animal Sufficient Evidence IMEMDT 20,-429,79. NCI Carcinogenesis Bioassay (gavage); Clear Evidence: mouse-rat NCITR* NCI-CG-TR-55,78. EPA Genetic Toxicology Program. Reported in EPA TSCA Inventory.

OSHA PEL: TWA ~~50~~ 1.0 ppm; ~~CL 100 ppm; Pk 200 ppm/5M/3H~~ STEL = 2.0 ppm
ACGIH TLV: TWA 10 ppm
NIOSH REL: TWA 1 ppm; CL 2 ppm/15M

DOT Classification: Flammable Liquid, Label: Flammable Liquid; IMO: Flammable Liquid, Label: Flammable Liquid, Poison

THR: A human poison by ingestion. Poison experimentally by intravenous and subcutaneous routes. Moderately toxic by inhalation, skin contact, and intraperitoneal routes. An experimental carcinogen, neoplastigen, tumorigen and teratogen. Human systemic effects by ingestion and inhalation: flaccid paralysis without anesthesia (usually neuromuscular blockade), somnolence, cough, jaundice, nausea or vomiting, hypermotility, diarrhea, ulceration or bleeding from the stomach, fatty liver degeneration, change in cardiac rate, cyanosis and coma. An experimental transplacental carcinogen. It may also cause dermatitis, edema of the lungs, toxic effects on the kidneys, and severe corneal effects. A strong narcotic. Experimental reproductive effects. A skin and severe eye irritant, and strong local irritant. Its smell and irritant effects warn of its presence at relatively safe concentrations. Human mutagenic data. A pesticide. A priority pollutant.

A dangerous fire hazard if exposed to heat, flame or oxidizers. Moderately explosive in the form of vapor when exposed to flame. Violent reaction with Al; N_2O_4; NH_3; dimethylaminopropylamine. Can react vigorously with oxidizing materials and emit vinyl chloride and HCl. To fight fire, use water, foam, CO_2, dry chemicals. When heated to decomposition it emits highly toxic fumes of Cl^- and phosgene. See also CHLORINATED HYDROCARBONS, ALIPHATIC.

ARA750 *HR: 3*

ARSENIC

CAS: 7440-38-2 NIOSH: CG 0525000
DOT: 1558
af: As aw: 74.92

PROP: Silvery to black, brittle, crystalline and amorphous metalloid. Mp: 814° @ 36 atm, bp: subl @ 612°, d: black crystals 5.724 @ 14°; black amorphous 4.7, vap press: 1 mm @ 372° (sublimes). Insol in water; sol in HNO_3. See also ARSENIC VAPOR.

SYNS:

ARSEN (GERMAN, POLISH)
ARSENICALS
ARSENIC-75
ARSENIC BLACK
COLLOIDAL ARSENIC
GREY ARSENIC
METALLIC ARSENIC

TOXICITY DATA:	CODEN:
cyt-mus-ipr 4 mg/kg/48H-I	EXPEAM 37,129,81
orl-rat TDLo:605 µg/kg (35 W preg):REP	GISAAA (8)30,77
orl-mus TDLo:120 mg/kg (preg):TER	TJADAB 15,31A,77
ipr-mus TDLo:40 mg/kg (preg): TER	TJADAB 15,31A,77
imp-rbt TDLo:75 mg/kg:ETA	ZEKBAI 52,425,42
orl-man TDLo:7857 mg/kg/55Y: SKN	CMAJAX 120,168,79
orl-man TDLo:7857 mg/kg/55Y: GIT	CMAJAX 120,168,79
ims-rat LDLo:20 mg/kg	NCIUS* PH 43-64-886,SEPT,70
scu-rbt LDLo:300 mg/kg	ASBIAL 24,442,38
ipr-gpg LDLo:10 mg/kg	CRSBAW 81,164,18
scu-gpg LDLo:300 mg/kg	ASBIAL 24,442,38

IARC Cancer Review: Human Sufficient Evidence IMEMDT 23,39,80; Human Inadequate Evidence IMEMDT 2,48,73. Reported in EPA TSCA Inventory. Arsenic and its compounds are on the Community Right To Know List.

OSHA PEL: TWA 0.01 mg(As)/m^3
ACGIH TLV: TWA 0.2 mg(As)/m^3
DFG TRK: 0.2 mg/m^3 calculated as As in that portion of dust that can possibly be inhaled.
NIOSH REL: CL 2 µg(As)/m^3

DOT Classification: Poison B, Label: Poison

THR: A human carcinogen. Poison by subcutaneous, intramuscular, and intraperitoneal routes. Human systemic skin and gastrointestinal effects by ingestion. An experimental teratogen and tumorigen. Mutagenic data. Flammable in the form of dust when exposed to heat or flame or by chemical reaction with powerful oxidizers such as bromates; chlorates; iodates; peroxides; lithium; NCl_3; KNO_3; $KMnO_4$; Rb_2C_2; $AgNO_3$; NOCl; IF_5; CrO_3; ClF_3; ClO: BrF_3; BrF_5; BrN_3; RbC_3BCH; CsC_3BCH. Slightly explosive in the form of dust when exposed to flame. When heated or on contact with acid or acid fumes, emits highly toxic fumes; can react vigorously on contact with oxidizing materials. Incompatible with bromine azide; dirubidium acetylide; halogens; palladium; zinc; platinum; NCl_3; $AgNO_3$; CrO_3; Na_2O_2; hexafluoro isopropylideneamino lithium. For further information, see Vol. 4, No. 1 of *DPIM Report*.

ARF750 *HR: 3*

ARSENIC COMPOUNDS

SYN: ARSENICALS

Arsenic and its compounds are on the Community Right To Know List.

Used as insecticides, herbicides, silvicides, defoliants, desiccants and rodenticides. Poisoning from arsenic compounds may be acute or chronic. Acute poisoning usually results from swallowing arsenic compounds; chronic poisoning from either swallowing or inhalaling. Acute allergic reactions to arsenic compounds used in medical therapy have been fairly common. The type and severity of reaction depending upon the compound of arsenic. Inorganic arsenicals are more toxic than organics. Trivalent is more toxic than pentavalent. Acute arsenic poisoning (from ingestion) results in marked irritation of the stomach and intestines with nausea, vomiting, and diarrhea. In severe cases, the vomitus and stools are bloody and the patient goes into collapse and shock with weak, rapid pulse, cold sweats, coma, and death. Chronic arsenic poisoning, whether through ingestion or inhalation, may manifest itself in many different ways. There may be disturbances of the digestive system such as loss of appetite, cramps, nausea, constipation, or diarrhea. Liver damage may occur, resulting in jaundice. Disturbances of the blood, kidneys, and nervous system are not infrequent. Arsenic can cause a variety of skin abnormalities including itching, pigmentation, and even cancerous changes. A characteristic of arsenic poisoning is the great variety of symptoms that can be produced. A recognized carcinogen of the skin, lungs, liver. An experimental carcinogen of the mouth, esophagus, larynx, bladder and para nasal sinus. Dangerous; when heated to decomposition, or when metallic arsenic contacts acids or acid fumes, or when water solutions of arsenicals are in contact with active metals such as Fe; Al; Zn; they emits highly toxic fumes of arsenic. For further information, see Vol. 1, No. 3 of *DPIM Report*.

In treating acute poisoning from ingestion BAL (dimercaptol) is of questionable effectiveness for acute and chronic poisoning with trivalent arsenicals, such as As trioxide, arsine and arsenites. It is of no value for pentavalent arsenicals, such as cacodylic acid, methanearsonic acid, sodium, cacodylate, MSMA, DSMA, arsanilic acid, arsenic acid, and arsenates. Vomiting and gastric lavage are the preferred emergency treatments for acute arsenical poisoning. Modern medical treatment of arsenical poisoning uses exchange transfusion and dialysis (A. E. De Palma, *J. Occup Med.*, Vol. 11,582-587 (1969). Note: Arsenic compounds are common air contaminants.

ARSENIC and SOLUBLE COMPOUNDS

CAS: 7440-38-2

As

TLV-TWA, 0.2 mg/m^3, as As

Arsenic, an element with atomic number 33, atomic weight 74.92, is in Group VA of the periodic table. The most common form of the element is a gray brittle crystalline solid with a specific gravity of 5.72, which sublimes at 613°C. It also exists in amorphous forms: black, specific gravity of 4.7 and yellow, specific gravity of 2.0, which is relatively volatile. Yellow arsenic is soluble in carbon disulfide; the other forms are insoluble in water or solvents, but dissolved by oxidizing acids.

Elemental or metallic arsenic is employed as an alloying agent for heavy metals, in special solders, and as a doping agent in silicon and germanium solid state products.

In addition to arsenic compounds discussed separately (As_2O_3, AsH_3 and lead arsenate, q.v.) many others find commercial application. The arsenites are important herbicides, calcium and other arsenates are insecticides; sulfides are pigments, rodenticides and used in pyrotechnics; gallium arsenide is in semiconductors; arsenic trichloride, a liquid with a boiling point of 130.5°C, is employed in chemical synthesis; the gaseous tri- and pentafluorides apparently have no important commercial uses. Many organic arsenic compounds, however, have been employed in medicine, or as war gases.

As with other metallic poisons, the toxicities, especially the acute toxicities, of arsenic compounds are related to their solubility in water. Thus, most arsenates and arsenites are acute poisons, while the sulfides are probably less toxic in an acute sense, but may be equally hazardous on prolonged exposure. Elemental arsenic is also less acutely toxic than its oxides, except for the rare yellow arsenic which is highly toxic, possibly similar to yellow phosphorus in some of its properties.

Systemic arsenic poisoning is rarely seen in industry, and still more rarely is it severe in character. According to Hardy,(1) it is hard to explain the difference between industrial and nonindustrial arsenic poisoning, but such variation is recorded in all industrialized countries. The usual effects on workers are local, on skin and mucous membranes, etc. A hoarse voice is characteristic of an arsenic worker, and a perforated nasal septum is a common result of prolonged inhalation of white arsenic dust or fume. A few documented cases of cirrhosis of the liver, however, due to occupational exposure to arsenic, have been recorded.(1)

Although the epidemiologic evidence is not complete, arsenic is considered by some to be a carcinogen, certainly of the skin, and perhaps of the bronchi.(2,3) Cancers from exposure to arsenic have followed: 1) the internal use of Fowler's Solution, an aromatic solution of potassium arsenite;(4) 2) inhalation and skin contact with sheep-dust, a mixture of sodium arsenite and sulfur;(5) 3) the combined inhalation of As_2O_3, SO_2 and other particulates from the smelting of ores containing arsenic (see documentation, arsenic trioxide production). Experimental cancers in animals have not been produced from As_2O_3 despite several attempts(6-8) and the conclusion of Vallee *et al*(8) was that "it is improbable that arsenic (per se) plays a significant role in the generation of cancer." The belief that other occupational factors are necessary for the development of cancer, in addition to arsenic exposure, has been expressed by others.(9)

A search of the world literature reveals no reports of industrial or experimental exposures solely to arsenic compounds which contain both environmental and toxicological criteria from which a TLV can be unequivocally based. Watrous and McCaughey(10) found concentrations of arsenic in a pharmaceutical plant averaging about 0.2 mg/m^3, with no definite evidence of intoxication. Pinto and McGill studies a group of smelter employees and found an average urinary arsenic excretion of 0.8 mg/L.(11) The chief manifestation of toxic exposure was dermatitis, with perforation of the nasal septum, pharyngitis and conjunctivitis noted less frequently. A reasonable interpretation of the urinary arsenic levels would indicate an average exposure of about 0.2 mg/m^3 of arsenic in air. Since individual concentrations as high as 4 mg/L of urine were found, it is probable that many workers were exposed at higher concentrations.

In its criteria document for inorganic arsenic, NIOSH in 1973(12) recommended 0.05 mg As/m^3 (as a TWA) as a workplace air standard. This was changed in 1975 to 0.002 mg/m^3 as a 15-minute ceiling.

The first limit was based primarily on reports of cancer among workers exposed to arsenic, as well as non-occupational cancer resulting from arsenic medications. The only pertinent environmental data cited not already noted consist of an average concentration of 0.56 mg/m^3 computed from the paper by Perry *et al*(13) on an English sheep dip factory study, and a study by Lee and Fraumeni(14) in a smelting plant. Concentrations of 1.47, 1.56 and 1.50 mg/m^3 were reported in "medium and high exposure areas" and 0.65, 0.17 and 0.002 mg/m^3 in "light exposure areas." In both plants an increased incidence of cancer was reportedly found.

The Committee is not aware of any published explanation of the reasons for the reduction of the NIOSH 1973 recommendation of a TWA of 0.05 mg/m^3 as a standard, to a ceiling of 0.002 mg/m^3 in 1975.

Normal values of arsenic in urine, as recorded in the literature, vary from 0.013 to 0.046 mg/L,(1) to 0.13,(11) to 0.25.(15) The urinary excretion, in mg/liter, of elements that are freely eliminated by this route, such as fluorine, mercury and arsenic, is at most 2.5 to 5 times the occupational exposure in mg/cubic meter of air.(16) It is apparent that biological monitoring for arsenic by urinalysis would be of limited value in determining whether or not the NIOSH recommended standard was being met or exceeded.

It is possible that some arsenic compounds, the trichloride for example, might produce certain toxic effects at concentrations below 0.2 mg/m^3 of arsenic. Data to substantiate this speculation are lacking. The contrary situation, that some compounds, or the metal itself, are chronically less toxic than As_2O_3, the form for which most information is available, seems more probable in the light of present knowledge. Therefore, a time-weighted average TLV of 0.2 mg As/m^3 for soluble compounds of arsenic is recommended.

According to the 1980 compilation of occupational exposure limits of the International Labour Office,(17) the following countries had adopted the previous TLV of 0.5 mg/m^3: Australia, Belgium, Finland, Japan, and Holland. Czechoslavakia, East Germany, Hungary and Poland specified the USSR MAC of 0.3 mg/m^3; Romania and Switzerland, 0.2 mg/m^3; Sweden 0.05 mg/m^3; and Italy 0.25 mg/m^3. Only three of 18 countries (West Germany, Italy and Sweden) designated arsenic and compounds as carcinogens, although Belgium and the Netherlands so characterized arsenic trioxide.

References

1. **Hamilton, A. and H.L. Hardy:** *Industrial Toxicology*, 3rd ed., pp. 31-39. Publishing Sciences Group, Acton, England (1974).
2. **Buchanan, W.D.:** *Toxicity of Arsenic Compounds*. Elsevier, Amsterdam (1962) Cited in reference 1.

3. **Snegriff, L.S. and O.M. Lombard:** *Arch. Ind. Hyg. Occup. Med.* 4:199 (1961) Ibid.
4. **Graham, J.H. et al:** *J. Invest. Derm.* 37:317 (1961).
5. **Hill, A.B. and E.L. Faning:** *Brit. J. Ind. Med.* 5:6 (1948).
6. **Heuper, W.C. and W.W. Payne:** *Arch. Env. Health* 5:445 (1962).
7. **Baroni, C. et al:** *Ibid.* 7:668 (1963).
8. **Vallee, E.L. et al:** *Arch. Ind. Health* 21:132 (1960).
9. **Goldblatt, M.W. and J. Goldblatt:** Occupational Carcinogenesis. *Industrial Medicine and Hygiene*, pp. 210-215. E.R.A. Merewether, Ed. Butterworth, London (1956). Cited in reference 1.
10. **Watrous, R.M. and M.B. McCaughey:** *Ind. Med.* 14:639 (1945).
11. **Pinto, S.S. and C.M. McGill:** *Ind. Med. Surg.* 22:281 (1953).
12. **NIOSH:** *Criteria for a Recommended Standard — Occupational Exposure to Inorganic Arsenic* (1973).
13. **Perry, K. et al:** *Brit. J. Ind. Med.* 5:6 (1948). Cited in reference 12.
14. **Lee, A.M. and J.F. Fraumeni, Jr.:** *J. Natl. Cancer Inst.* 42:1045 (1969).
15. **Seifert, P.:** *Deut. Med. Wochschr.* 79:1122 (1954); abst. in *Arch. Ind. Health* 12:665 (1955).
16. **Elkins, H.B.:** *Am. Ind. Hyg. Assoc. J.* 28:305 (1967).
17. *Occupational Exposure Limits for Airborne Toxic Substances*, 2nd (Rev) ed., pp. 46-47. Occupational Safety and Health Series No. 37. International Labour Office, Genveva (1980).

ARSENIC TRIOXIDE PRODUCTION

CAS: 1327-53-3

As_2O_3

TLV-TWA, None
Appendix A2 — Suspected Human Carcinogen

The production of arsenic trioxide (As_2O_3) in the USA results from the smelting of copper sulfide ores of widely varying arsenic content. This process of smelting and refining presents a mixed exposure to arsenic, antimony and sulfur dioxide as well as to copper, cadmium, lead, selenium, silver, tellurium, thallium and mercury, the amount depending upon composition of the ore and the leaks in the furnaces and flues. The crude product contains 95% As_2O_3, from 10,000-20,000 ppm antimony, 300-600 ppm lead and iron, 100-800 ppm copper, 300 ppm zinc, and 15 ppm cadium and selenium, with more or less similar amounts of mercury and tellurium.

Two epidemiologic studies of copper smelting and refining have been reported by Pinto(1,2) in which the health effects from As_2O_3 exposure were statistically presented. In the first study (1953) the deleterious effects of As_2O_3 were principally irritation of exposed body surfaces, skin, conjunctivae and mucous membranes of the nose which in some cases resulted in perforation of the nasal septum. Of 835 urine determinations of 348 workers, arsenic values ranged from 0.1 to 6.44 mg/L, 95% of which were less than 2.1 mg/L, resulting in an average of 0.82 mg As/L urine, compared with 0.13 mg/L of 124 unexposed controls. No relation was found between urinary values and severity of the superficial lesions, and moderate cigarette smoking did not increase the amount of As in the urine.

The second study (1963) focused attention of the possible effects of As_2O_3 exposure on cardiovascular and cancer mortality during 1946 to 1960. Using the same measure of exposure, urinary As, as in the first study, Pinto and Bennett found no evidence that this degree of exposure produced a significant excess of systemic cancer or fatal cardiovascular disease in a total of 229 deaths in active plant employees and pensioners, which averaged 905 and 209, respectively. Pensioners consisted of males over 65 years with a minimum of 15-years exposure. Eighty percent of the cancer deaths occurred among heavy smokers, and 60% of the noncancer deaths were smokers. Relatively more cancer deaths occurred among the cohort control "nonexposed" (19.4% of all deaths) than among those exposed to As_2O_3 (15.8%). No concentrations of sulfur dioxide or other concurrent exposures were reported. Because urinary excretion values of the control cohort were higher than those reported by others,(3-5) relating the pulmonary cancer deaths to those from this cohort was felt(6) to lead to improper conclusions.

Indeed, a review of death certificates for the county in which the smelter was located, revealed 40 respiratory cancer deaths(7) instead of 18 reported by Pinto. Unfortunately the death certificates bore no information on what comprised the exposure or its magnitude, no information on length of employment of workers or where they worked in the operations, no information about previous employment or smoking habits.

In a restudy of mortality experience of 8047 smelter works, by Lee and Fraumeni,(8) which had been exposed during 1938 to 1964, and compared with the male mortality in the same states, a 3-fold over-all excess of respiratory cancer was found. This excess rose to 8-fold among the heaviest exposed smelters who had had 15 or more years of exposure. Although no arsenic, sulfur dioxide or silica levels were reported, respiratory cancer rates were positively correlated with estimated "high," "medium" and "low" levels of As_2O_3, and "high" and "moderate" levels of sulfur dioxide. An inverse correlation was found between observed-to-expected cancer deaths with "heavy," "medium" and "light" silica exposure groups, which was interpreted as "reflecting that work areas with heavy arsenic or heavy SO_2 exposure provided light silica exposure."

The importance of this study is that it may be the first to recognize that respiratory cancer in smelter workers may be promoted by concurrent exposures to respiratory irritants such as sulfur dioxide and silica, and "other metals" (not specified by the authors, but presumably antimony and lead).

In view of the fact that As_2O_3 by itself has never been shown to be a tumorigen in animals, despite several attempts,(9-11) it can only be concluded that if arsenic is to induce respiratory cancer in smelter workers, a promoter (or promoters) is a requisite. Consequently, arsenic trioxide production is given an A2 designation, a chemical substance associated with industrial processes, which are suspect of inducing cancer. No TLV is assigned at this time.

References

1. **Pinto, S.S. and C.M. McGill:** *Ind. Med. Surg.* 22:281 (1953).
2. **Pinto, S.S. and B.M. Bennett:** *Arch. Environ. Health* 7:583 (1963).
3. **Schrenk, H.H. and L. Schreibs, Jr.:** *Am. Ind. Hyg. Assoc. J.* 19:225 (1958).
4. **Perry, K. et al:** *Brit. J. Ind. Med.* 5:6 (1948).
5. **Webster, S.H.:** *U.S. Pub. Hlth. Serv. Rept.* 56:1953 (1941).
6. **NIOSH:** *Criteria for a Recommended Standard — Occupational Exposure to Inorganic Arsenic*, pp. 39-40 (1973).
7. **Milham, S., Jr. and T. Strong:** *Environ. Res.* 7:176 (1974).
8. **Lee, A.M. and J.F. Fraumeni, Jr.:** *J. Natl. Cancer Inst.* 42:1045 (1969).
9. **Hueper, W.C. and W.W. Payne:** *Arch. Env. Health* 5:445 (1962).
10. **Baroni, C. et al:** *Ibid* 7:668 (1963).
11. **Vallee, B.L.:** *Arch. Ind. Health* 21:132 (1960).