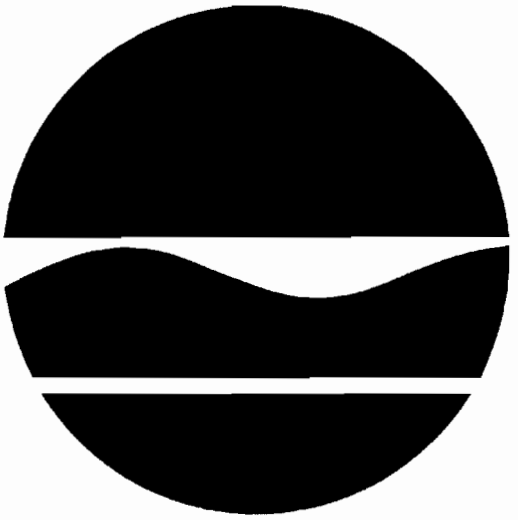
NEW YORK STATE DEPARTMENT OF
ENVIRONMENTAL CONSERVATION

POST-CLOSURE MONITORING AND MAINTENANCE PLAN

LEHIGH INDUSTRIAL PARK SITE
LACKAWANNA (C), ERIE COUNTY

SITE NO. 9-15-145



MAY 1998

New York State Department of Environmental Conservation
GEORGE E. PATAKI, *Governor* **JOHN P. CAHILL**, *Commissioner*

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SECTION 1

INTRODUCTION

This document presents a monitoring and maintenance operations plan for post-closure activities at the Lehigh Industrial Park (LIP) site which complies with the requirements set forth under New York Codes, Rules and Regulations, Title 6 (6 NYCRR), Part 360-2.15(k)(7). The plan describes groundwater monitoring, site cover and drainage system inspection and maintenance, contingency plans, and reporting requirements. Developed in part from a document submittal by Parson's Engineering Science entitled "Post Closure Monitoring and Maintenance Plan for the Lehigh Industrial Park Site", dated June 1995, this plan also contains detailed instructions to be used by site personnel to assure efficient monitoring, groundwater sampling and analysis, and maintenance of facility components for a minimum period of 30 years after site closure.

1.1 PROJECT BACKGROUND

The LIP site is a former automotive scrapping facility, located at 31 South Street in the City of Lackawanna, Erie County, New York. The site occupies 9.1 acres of land bounded by South Street to the north, Buffalo Brake Beam Co. to the south, Conrail and South Buffalo Railway right-of-way to the east, and a residential area on the west. The shore of Lake Erie is approximately one mile to the west and Smokes Creek is approximately 1000 feet south of the southern border.

A Site History Report was prepared by Parsons Engineering Science, Inc. (Parsons ES) in September 1992 and presents detailed information on previous owners and operators, site conditions and occurrences of spills and other mishaps. In summary, a deed search of LIP revealed that in the early 1900's the site was initially separated into four parcels, and that these parcels were utilized independently from one another under different owners. They eventually became consolidated under a single owner in 1973.

Though ownership has changed hands many times, aerial photographs dating back to 1938 have revealed that the site has been used primarily as an automotive and metal scrap yard. The last business to operate at the site was known as Roblin Industries, Inc. (Roblin). Roblin filed for bankruptcy in 1985. Conversations with past Roblin employees and review of documents on file with various public agencies indicate that spills were commonplace, and some drums were received, scrapped, and possibly buried under waste/soil piles. There are, however, no records of drums on file with any of the agencies contacted. The Lehigh Industrial Park purchased the site from the bankruptcy trustee of Roblin in 1988.

Prior to New York State Department of Environmental Conservation

(NYSDEC) involvement, the Erie County Department of Environmental Planning (ECDEP) was involved with environmental compliance issues at the LIP site. In 1979, soil sampling was supervised by the ECDEP as part of a cleanup of a polychlorinated biphenyl (PCB)-laden oil spill from a transformer. After excavation of oil-stained soil was performed, Roblin was advised that no further action was required on its part.

In 1988, after Roblin had gone bankrupt and the site was inactive, another PCB spill occurred (near the location of the previous spill), when hazardous waste disposal workers were removing a transformer. Subsequent sampling confirmed that PCB-contaminated soils were present again at the site.

The LIP site was designated as a Class 2 inactive hazardous waste site (containing hazardous waste that constitutes a significant threat to the environment) in December 1990. For the past several years, the site has been plagued by vandalism, illegal dumping, and suspicious fires.

The LIP site will be redefined to encompass only the portion of the site which contains the waste cell and the infiltration basin. The redefined site will consist of approximately 5.5 acres and it is anticipated the site will be reclassified to a Class 4 (a site that has been remediated but requires continued monitoring and maintenance.)

1.2 POST-CLOSURE SITE CONDITIONS

The Lehigh Industrial Park Site was remediated during the Summer and Fall of 1997. The remediated area included approximately nine acres of land. Approximately half of this area had the existing site soils excavated to one foot and consolidated with non-hazardous waste soil and debris consolidated from the rest of the site into a soil pile approximately 350 feet by 550 feet in size. All soils classified as hazardous were properly disposed of off site. No hazardous waste was left on site. After excavation and consolidation the entire site was covered with a soil cover consisting of 9 inches of low permeability clay and three inches of topsoil. The site was fully re-vegetated to control erosion. An infiltration basin was constructed to intercept runoff from the soil cover area. This basin was fenced off to control access. Specific Details of remedial activities can be found in the report entitled "Remediation Summary Report, Lehigh Industrial Park Site", NYSDEC, datedMay 1998.

1.3 ORGANIZATION OF MONITORING AND MAINTENANCE PLAN

This plan is organized into five sections, including this introduction (Section 1). Section 2 provides a description of groundwater monitoring; Section 3 provides

a description of site cover and drainage system inspections and correction procedures; Section 4 is a contingency plan; and Section 5 provides information on record keeping and the various reports that must be submitted. A site health and safety plan, and post-closure inspection and maintenance report forms for inspection activities are contained in Appendices B, and C, respectively.

SECTION 2

GROUNDWATER MONITORING

2.1 GENERAL

Groundwater monitoring will be a routine part of the LIP site post-closure operations to evaluate groundwater quality and monitor flow direction. The following subsections will describe the procedures for sampling monitoring wells, analysis of samples, and evaluation of sample results.

2.2 MONITORING WELL LOCATIONS

A total of five monitoring wells were originally installed at the Lehigh Industrial Park Site . The wells were installed during the Preliminary Remedial Investigation conducted in 1992. MW-1, MW-3 and MW-5 were subsequently decommissioned during remedial construction in 1997. The wells to be sampled include MW-2 and MW-4. Information regarding the remaining wells, including location and construction details, is provided in Table C.1 and Appendix F.

2.3 GROUNDWATER SAMPLING AND ANALYSIS

2.3.1 Frequency of Sampling

Groundwater sampling and analysis for site specific parameters as detailed in table 2.1 of the two remaining site wells will be conducted quarterly for the first year and annually thereafter for a minimum of thiry years, depending on the analytical results. The two wells will effectively monitor contaminant migration from the site. Should a well yield repeated high contaminant levels or a sudden rise in contamination, the well may be monitored quarterly in lieu of annually upon approval from the NYSDEC project manager. Sampling will be completed by fully qualified subcontractors or Department personnel.

2.3.2 Sample Container Preparation

Sample containers will be properly washed and decontaminated by the laboratory prior to use. The containers will be tagged and Chain of Custody initiated before shipping to the sampling site in coolers. The types of containers and preservation techniques are shown in Table C.2. Since all bottles will contain the necessary preservatives, they need only be filled. Following sample collection, the bottles will be placed on ice in the shipping cooler. The samples will be cooled to

4°C but not frozen.

2.3.3 Field Procedures

The following is a step-by-step sampling procedure to be used to collect the groundwater samples. Well sampling procedures will be recorded on the form shown on Figure C.1.

- Assemble all field equipment necessary for sample collection (Table C.3).
- Inspect equipment to ensure it is working properly.
- Select a well as the initial sampling location.
- Prior to purging and sampling, measure the static water level from the surveyed well elevation mark on the top of the casing with a water level indicator. Water levels will be measured to nearest 0.01 foot and recorded on the Groundwater Sampling Data Sheet (Figure C.1).
- Decontaminate the water level indicator. (See Section 2.3.4 for decontamination procedures.)
- Purge the well by removing a minimum of three well volumes of water. Purging will be conducted with a bailer or a peristaltic pump and dedicated polyethylene tubing. If the well goes dry before the required volumes are removed, the well may be sampled when it recovers sufficiently. The purged water may be disposed on the ground surface a minimum of ten feet away from the well in a downhill location.
- Collect samples from each well with a dedicated bailer lowered by a dedicated nylon line. Temperature, conductivity, dissolved oxygen, pH, and turbidity will be measured, and sample description and location noted on the Groundwater Sampling Data Sheet (Figure C.1). Specific conductance and pH will be measured by pre-calibrated electronic probes. Temperature will be measured by a pre-calibrated probe or thermometer.
- Fill sample containers to be analyzed for volatile organic compounds first. Sample containers to be analyzed for metals and other analytes will then be filled. Sample containers will be labeled with the monitoring well number, date, time, sample type, and SDG number.
- The groundwater samples will be placed in a laboratory cooler, packed on ice

and shipped overnight to the laboratory. Quality assurance blanks will be sent with each sample shipment. Contract Lab Sample Information Sheets (Figure C.3) will be completed. Chain-of-Custody procedures will be strictly followed as outlined in Section 2.3.5.

2.3.4 Equipment Decontamination

Dedicated or disposable equipment will be used to the extent practical. However, prior to sampling equipment use, and between sampling points, all non-dedicated equipment (bailers, water-level indicators, etc.) coming in contact with well water will be properly decontaminated. The general decontamination procedure is as follows: (Water-level indicator is used as an example.)

- Thoroughly clean the water-level indicator with a biodegradable detergent, such as Alconox and tap water.

- Triple rinse the water-level indicator with distilled water.

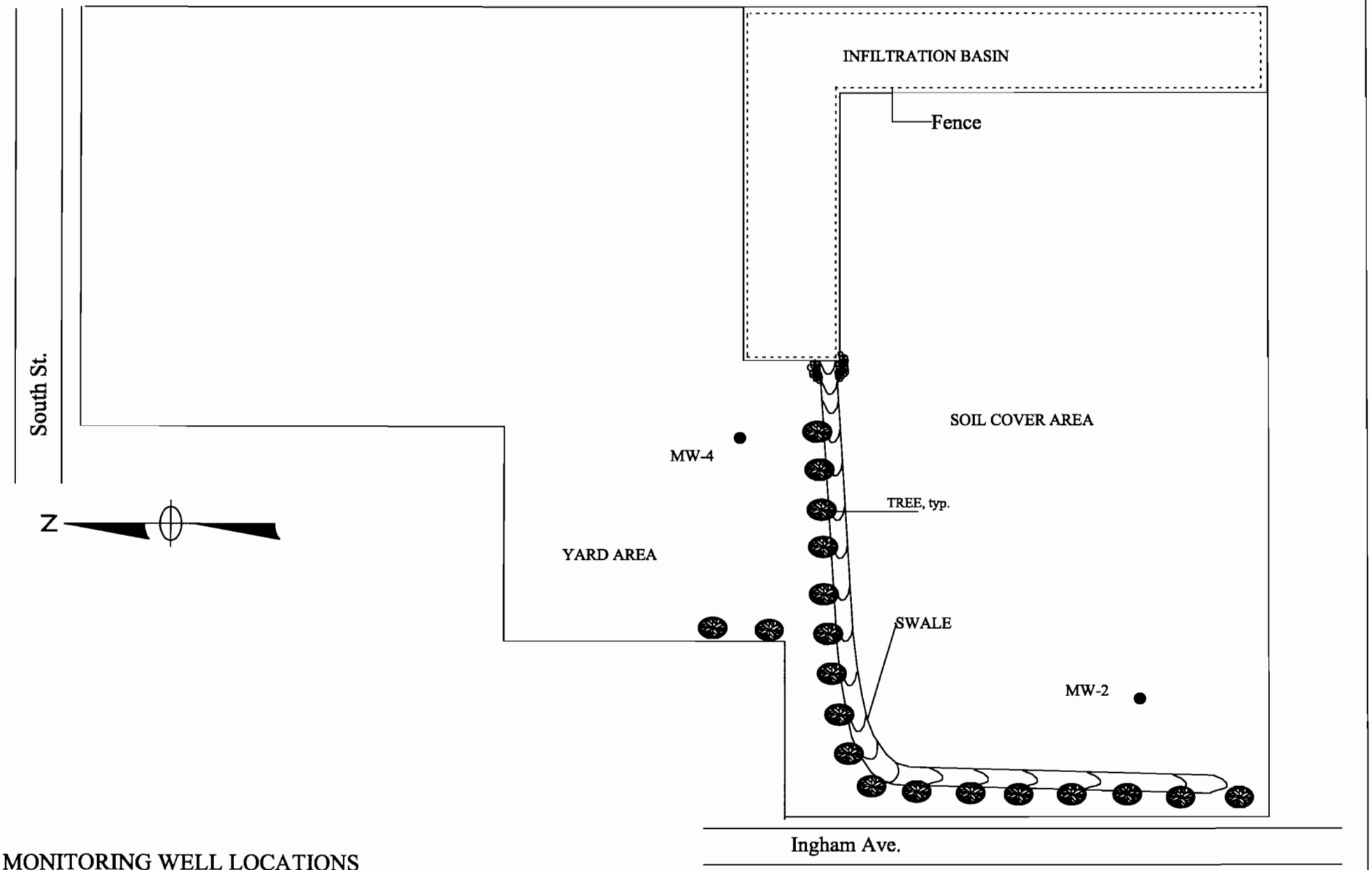
- Allow water-level indicator to air dry or wipe dry using disposable paper towels.

- Wrap water-level indicator in aluminum foil or place in clean plastic bag so that no outside contaminants are introduced.

Between rinses, equipment will be placed on polyethylene sheets or aluminum foil if necessary. At no time will washed equipment be placed directly on the ground.

Figure 2.1

Monitoring Well Location Map



MONITORING WELL LOCATIONS
LEHIGH INDUSTRIAL PARK SITE
SITE NO. 915145, LACKAWANNA (C), ERIE CO.

NOT TO SCALE

MONITORING WELL LOCATIONS	
DIVISION OF ENVIRONMENTAL REMEDIATION	
DATE: 4/15/98	DRAWING: LHP-MW.DWG
SITE: LEHIGH INDUSTRIAL PARK SITE NO. 915145	

Figure 2.1

To prevent cross-contamination between wells, separate bailers and rope will be used for each well.

2.3.5 Field Sample Custody

Evidence of sample traceability and integrity is provided by Chain-of-Custody (COC) procedures. These procedures document the sample traceability from the selection and preparation of the sample containers by the laboratory, to sample collection, to sample shipment, to laboratory receipt and analysis. A sample is considered to be in a person's custody if the sample is:

- In a person's possession;
- Maintained in view after possession is accepted and documented;
- Locked and tagged with Custody Seals so that no one can tamper with it after having been in physical custody; or
- In a secured area which is restricted to authorized personnel only.

A COC record (Figure C.2) accompanies the sample containers from selection and preparation at the laboratory, during shipment to the field for sample containment and preservation, and during return to the laboratory. Triplicate copies of the COC must be completed for each sample set collected.

The COC lists the field personnel responsible for taking samples, the project name and number, the name of the analytical laboratory to which the samples are sent, and the method of sample shipment. The COC also lists a unique description of every sample bottle in the set. If samples are split and sent to different laboratories, a copy of the COC record will be sent with each sample.

The REMARKS space is used to indicate if the sample is a matrix spike, matrix spike duplicate or matrix duplicate. Since they are not specific to any one sample point, trip and field blanks are indicated on separate rows. Once all bottles are properly accounted for on the form, the sampler will write his or her signature and the date and time on the first RELINQUISHED BY space. The sampler will also write the method of shipment, the shipping cooler identification number, and the shipper airbill number on the top of the COC. Mistakes will be crossed out with a single line and initialed by the author.

One copy of the COC is retained by sampling personnel and the other two copies are put into a sealable plastic bag and taped inside the lid of the shipping

cooler. The cooler lid is closed, custody seals provided by the laboratory are affixed to the latch and across the back and front lids of the cooler, and the person relinquishing the sample signs his name across the seal. The seal is taped, and the cooler is wrapped tightly with clear packing tape. It is then relinquished by field personnel to personnel responsible for shipment, typically an overnight carrier. The COC seal must be broken to open the container. Breakage of the seals before receipt at the laboratory may indicate tampering. If tampering is apparent, the laboratory will contact the designated person, and the sample will not be analyzed.

2.3.6 Sample Analysis

Analytical procedures shall conform to the most recent revision of the NYSDEC-ASP. Groundwater will be analyzed for a full baseline sampling the first sampling following remediation. Pending review of the first round of data, it is expected that subsequent sample analysis will consist of the routine parameters of concern found in (Table 2.1.) Routine parameters will be analyzed once per year.

After the first analysis, the need for monitoring for VOCs for subsequent sampling will be evaluated. After five years, the parameter list and monitoring frequency will be reevaluated based on the post-closure sampling results.

Samples will be analyzed by a laboratory which is New York State Department of Health (NYSDOH) Environmental Laboratory Approval Program (ELAP) approved in all categories of solid and hazardous waste. The procedures for the sample preparation and analysis are specified in the NYSDEC-ASP. The laboratory shall expend such an effort and discretion to demonstrate good laboratory practice and demonstrate and attempt to best achieve the method detection limit.

2.3.7 Quality Assurance/Quality Control

In addition to water samples collected from monitoring wells, "blanks" may be collected and submitted to the chemical laboratory for analyzes. The blanks will consist of 40 ml VOA vials, and may be any or all of the following:

a. **Trip Blank** - A Trip Blank will be prepared before the sample bottles are sent by the laboratory. It consists of a sample of distilled, de-ionized water which accompanies the other sample bottles into the field and back to the laboratory. A trip blank will be included with each shipment of water samples, where sampling and analysis for volatile organic compounds is planned. The Trip Blank will be analyzed for volatile organic compounds as a measure of the internal laboratory procedures and their effect on the results.

b. **Field Blank** - Field Blanks are usually collected to indicate possibility of cross-contamination. It is possible to cross-contaminate a sample by introducing contaminants from one well to another. This is usually accomplished by introducing an improperly decontaminated instrument into a well before collection of the sample. This cross-contamination may result from poor or lacking decontamination technique or inadvertent re-contamination after cleaning. Field Blanks may be prepared by pouring High Pressure Liquid Chromatography (HPLC) grade water over any instrument that is consistently used between wells such as water level indicators or bailers. Water poured over the decontaminated item is collected in a bottle appropriate for the analysis requested. This sample will then be handled, submitted and analyzed consistent with the other samples collected.

c. **Atmospheric Blank** - To measure the contribution of atmospheric contaminants, a sample bottle of organic-free distilled, de-ionized water is prepared by the laboratory and sent with the shipment of sample bottles. The blank is opened as sampling takes place. When sampling is completed, the blank is capped. The blank is utilized when sampling and analysis for volatile organic compounds is being performed. In these cases, the blank will be analyzed for volatile organic compounds.

2.3.8 Health and Safety

A Health and Safety Plan is provided in Appendix B which includes information on chemical and physical hazards anticipated during maintenance and monitoring at the site, personnel protection and monitoring equipment, accident prevention and contingency plan, sample handling, monitoring well decommissioning, and decontamination.

2.3.9 Data Evaluation and Reporting

The results of each monitoring event will be summarized in a letter report written for, or by, the NYSDEC. Analytical results will be evaluated with respect to background levels detected in monitoring wells during the Remedial Investigation, and applicable NYSDEC and NYSDOH standards and guidance values. Analytical results showing an increase in contamination must be reported to the NYSDEC project manager within 14 days of such determination.

An annual summary report will be prepared which compares background levels, individual sampling round results and applicable water quality standards. Included in the report will be a table with the following information:

- Sample identification number
- Sample collection date
- Well identification including description of up gradient wells
- Analytical results
- Method Detection Limits (MDL)
- Chemical Abstracts Service (CAS) numbers for all compounds
- Applicable water quality standards
- New York State Department of Health guidance values and statistical triggers
- Delineation of samples which exceed background levels, standards, guidance values, or statistical triggers.

A summary and discussion of all exceedances of background levels, standards, values, or statistical triggers and any proposed modifications to the sampling and analysis schedule will also be included.

The NYSDEC office responsible for the operation, maintenance and mangement of this project is:

New York State Department of Environmental Conservation - Region 9
Division of Environmental Remediation
270 Michigan Avenue
Buffalo, NY 14203-2999
(716) 851-7220

2.3.10 Contingency Monitoring Plan

This Contingency Monitoring Plan provides for increased water quality monitoring should an increase or migration of contaminants be determined. This plan has been developed in accordance with NYCRR Part 360-2.11(c)(5)(iii).If increasing contamination for one or more routine parameters is found, then those affected monitoring wells will be sampled and analyzed for baseline parameters during the next quarter and evaluated. If increased parameters are still indicated a semiannual baseline sampling will continue until the elevated parameter(s) is shown

not to be site-derived, the contaminant release is remediated, or it is determined there is no threat to public health or the environment. If, during analysis for baseline parameters, contamination is found for any parameter(s), quarterly sampling and analysis will be conducted for those baseline parameters. This increased monitoring schedule will continue until the elevated parameter(s) is shown not to be site-derived, the contaminant release is remediated, or it is determined there is no threat to public health or the environment.

TABLE 2.1
LEHIGH INDUSTRIAL PARK SITE, SITE NO. 9-15-145
GROUNDWATER QUALITY ANALYSIS TABLE

	Routine Parameters	Class GA STD's, ug/l	Analysis Method
FIELD PARAMETERS			
Static water level	x	n/a	
Specific Conductance	x	n/a	9050
Temperature	x	n/a	
pH	x	>6.5 - <8.5	9040/9041
Turbidity	x	n/a	
Field Observations [1]	x	n/a	
METALS			
Aluminum	x	n/a	ASP-91
Antimony	x	n/a	ASP-91
Arsenic	x	25	ASP-91
Barium	x	1,000	ASP-91
Beryllium	x	3	ASP-91
Cadmium	x	10	ASP-91
Calcium	x	n/a	ASP-91
Chromium (total and hexavalent)[2]	x	50	ASP-91
Cobalt	x	n/a	ASP-91
Copper	x	200	ASP-91
Iron	x	300*	ASP-91
Lead	x	25	ASP-91
Magnesium	x	35,000	ASP-91
Manganese	x	300*	ASP-91
Mercury	x	2	ASP-91
Nickel	x	n/a	ASP-91
Potassium	x	n/a	ASP-91
Selenium	x	10	ASP-91
Silver	x	50	ASP-91
Sodium	x	20,000	ASP-91
Thallium	x	4	ASP-91
Vanadium	x	n/a	ASP-91
Zinc	x	300	ASP-91
* Total Iron and Manganese = 500 ug/l			
PCBs	x	0.1	ASP-91
CLP-VOAs[3]			ASP-91
BNAEs[3]			ASP-91

This list may be modified as needed.

All samples must be whole and unfiltered except as otherwise specified by the NYSDEC project manager.

1 Any unusual conditions (colors, odors, surface sheens, etc.) noticed during well development, purging, or sampling must be reported.

2 The requirement to analyze Hexavalent Chromium may be waived provided that Total and Hexavalent and Trivalent Chromium values do not exceed 0.05 mg/l.

3 CLP-VOAs and BNAEs will be sampled initially upon completion of remediation for information purposes.

SECTION 3

WASTE CELL CARE AND MAINTENANCE

3.1 INTRODUCTION

This section contains procedures for post-closure care and maintenance of the soil cover, drainage swales, fencing and infiltration basin. It does not include responsibility for the "Flat area" (the area north of the infiltration basin and the "Yard Area". Re-definition of the specific site bounderies will only include that area that encompasses the Waste cell and the Infiltration Basin. It is anticipated that the "Flat Area"maintenance will fall to the responsibility of the land owner in the case of the yard area and to the City of Lackawannain anticipation of beneficial use. Specific procedures include routine inspections, routine maintenance, and contingency actions.

3.2 ROUTINE INSPECTIONS

The site will be inspected annually throughout the post-closure period. The site will also be inspected following particularly heavy storm events, e.g. a 10-minute, 2-year frequency storm. The waste cell will be inspected for:

- integrity of soil cover including:
  - erosion or settling of cover material;
  - animal borrows;
  - woody vegetation.
  - visible debris, litter and waste;
  - loss of vegetative cover;
  - damage due to vandalism;
- integrity of drainage system including:
  - clogging of swales;
  - sediment build-up;
  - pooling or ponding;
  - slope integrity; and

overall adequacy of surface runoff collection system;

damage due to vandalism;

- integrity of the infiltration basin including:

  debris;

  standing water;

  vegetation;

  over flow weir;

- condition of gates and fences;
- integrity of groundwater monitoring wells (to be inspected during sampling);

A site inspection map, post-closure inspection checklist, well inspection checklist, and maintenance schedule are contained in Appendix D. The site plan is to be used to document problems and indicate areas that require attention.

3.3 ROUTINE MAINTENANCE

3.3.1 Soil Cover Maintenance

- Monitor site vegetation progress. Annually confirm that the desired grass species have become established and that the desired ground cover is forming. Reseed and retreat local spots if the vegetation fails to become established by the end of the second growing season.

- Conduct annual ground inspections at the beginning of each summer to determine the status of woody plant species on the site surface.

- Remove unwanted debris and rubbish that may have been blown, or deposited on the site.

- Mow the waste cell cover surface once each year, once in the fall after August, to control woody vegetation and promote short grass species.

- If woody plants are detected, remove the plants using one of the following

methods:

For a small number of isolated individual plants, pull out the plants or cut them off at ground level by hand.

For more extensive areas involving hundreds of individual plants, remove the plants by mowing the area once a year in late summer to early fall. Mowing should be deferred until after the grass cover has become firmly established and will not be damaged by mowing equipment.

Indications of erosion or other site maintenance problems detected during routine site inspections or following particularly heavy storm events will be corrected as soon as possible. Repairs of eroded areas will be made with materials and methods specified herein. If erosion of the topsoil layer is encountered, the repair action may include, but not be limited to, the following:

- Covering repaired areas with topsoil, as specified in the remedial construction (available at NYSDEC), to minimum thickness (min. 3 inches) and design grades; and

- re-seeding and fertilizing in accordance with materials and application rates specified in Section 02990 Finish Grading, Topsoil and Seeding of the Contract Documents.

If erosion is persistent in certain areas, alternate methods for maintaining soil and vegetative cover or erosion protection will be evaluated and corrected as necessary on a case-specific basis.

Spots barren of vegetation on the waste cell cover will be re-seeded and fertilized as necessary. Seed and fertilizer will be of the same type and quality as originally specified. Any undesirable species will be removed if their presence is suspected of deteriorating the integrity of the cover.

The need for cover repairs due to subsidence or settling will be determined based on an evaluation of whether the function of the cap in the affected area has been impaired. Should large areas appear to have settled or drainage is not occurring, a survey of the cover may be conducted to determine the extent and nature of the repairs. Bench marks, such as the monitoring wells, established during construction shall be used for the survey. Those areas where the function has been impaired will be repaired to ensure that the integrity of the cap is maintained. Repair actions may include, but are not limited to:

- stripping and stockpiling topsoil from the affected area;
- regrading the affected area in accordance with the grading plan shown on the record drawings; and
- replacing topsoil, and re-seeding and fertilizing to reestablish vegetative cover as described previously.

For animal control, follow these procedures:

- Conduct an annual site inspection for to look for woodchuck or other animal burrow or den entrances on the waste cell. If den or burrow entrances are found, a program to trap or otherwise remove the burrowing animal(s) will be implemented on a case-specific basis. Following removal of the burrowing animal(s), the entrances will be plugged and the bare areas will be re-seeded and fertilized. Seed and fertilizer will be of the same type and quality as originally specified.

3.3.2 Maintenance of Site Structures

Maintenance activities will be performed as determined necessary based on routine inspections. During all maintenance activities, vehicle traffic crossing over the drainage swales shall be kept to a minimum. Maintenance vehicles shall cross the swale at the break on Ingham Avenue.

Drainage System Management

All elements of the drainage system including, swales, riprap, infiltration basin and overflow weir will be maintained throughout the post-closure period. All elements will be inspected annually or after severe rainfall events to verify the structures are intact and undisturbed, and that channels and discharge areas are free of obstructions which would impair the free flow of surface water run-off. In the event any of the structures are found to be damaged or incapable of conveying the design flows, repairs will be evaluated and made as soon as practical. Any obstructions found in swales will be immediately removed and channels re-graded as necessary. If any areas are found to be damaged such that their function is impaired, they will be repaired or replaced. Accumulated sediment will be removed from drainage channels and/or around outlet structures as required to maintain required capacity and proper operation.

Groundwater Monitoring Wells

Monitoring wells which are damaged such that representative ground water samples cannot be obtained will be repaired or replaced. Repair measures will be based on case-specific evaluation. Any well damaged beyond repair or rendered inoperative will be replaced with a new well of similar depth and construction. Detailed requirements for well installation and decommissioning are specified in Section 02900, Groundwater Monitoring Wells, of the Contract Documents.

Access Control

The access will be maintained in good condition so that routine inspections and required maintenance activities at the site can be carried out. All gates will be kept in good condition and locked to prevent unauthorized access. The condition of the gates, fences and access will be assessed as part of the annual inspections. Repairs will be conducted as needed.

SECTION 4
CONTINGENCY PLAN

4.1 INTRODUCTION

The objective of this contingency plan is to establish procedures for handling events which occur outside the scope of the routine maintenance. The contingency plan should be implemented following the identification of a site condition which is not covered by the routine maintenance plan.

Natural occurrences such as storms, drought and subsidence should be considered "expected occurrences" and are addressed under the routine maintenance program. Certain problems which cannot be reasonably expected to occur, such as earthquakes, are not addressed in this contingency plan.

The following problems are examples of occurrences which are not expected to occur, but may be discovered during a routine post-closure inspection:

- degradation of the soil cover integrity which may be a result of or indicated by:
  - waste/contaminated soil protruding through the topsoil cover;
  - soil erosion or other drainage problems; or
  - uncontrolled burrowing by pests.
- vegetative cover missing despite repeated efforts at revegetation;

The following guidelines are offered to determine when the contingency plan should be implemented and to determine possible corrective actions when responding to a contingency. All corrective actions, where appropriate, will be executed in a timely fashion after notifying the appropriate regulatory agencies.

4.2 FIRE

Fires at the site will be immediately reported to the local fire department.

Appropriate response measures, including personnel safety, will be the responsibility of the fire department. Fires will be quenched according to approved fire department protocol. Damage to the surface drainage system or soil cover will be repaired where these systems have been compromised.

4.3 VANDALISM

Vandalism will be reported to the local law enforcement authorities. If vandals have gained entry to the site, appropriate measures will be taken to eliminate or restrict future access. Vandalism to monitoring wells will be repaired as appropriate on a case-specific basis. Damage caused by off-road vehicles will be repaired where the damage is determined to have compromised the integrity of the soil cover or the function of the surface drainage system.

4.4 SEVERE EROSION AND COMPROMISE OF SOIL COVER INTEGRITY

Severe erosion of the soil cover, as well as the storm water management system will be repaired to original specifications. The cause of severe erosion will be investigated and remedial measures, if needed, will be developed and implemented accordingly.

4.5 UNAUTHORIZED DUMPING OR DISPOSAL

Unauthorized dumping or waste disposal will be reported to the NYSDEC and local enforcement officials. Appropriate measures will be taken to determine the waste characteristics, containment requirements and the necessary removal and disposal techniques. The waste will be removed and disposed of at an approved disposal facility, as appropriate. Efforts will be taken to minimize further dumping and to restrict subsequent entry to the site. Persons found in the act of illegal dumping will be prosecuted according to the law and will be held responsible for all costs incurred in removing the waste.

4.6 QUALITY ASSURANCE/QUALITY CONTROL

To assure the performance of site inspection and maintenance, a reporting procedure has

been established. A site inspection checklist is provided in Appendix C. The site inspection checklist was developed in accordance with the parameters identified in this section. The checklist will be completed after regularly scheduled site inspections and inspections following severe storms. An annual operation and maintenance and review report form (see Figure D.1 and D.2) will be completed each year on the Lehigh Industrial Park Site noting effectiveness and deficiencies of the remediation.

The monitoring and maintenance contractor and any future designated authority responsible for performing site inspections and supervising maintenance operations will be fully qualified (as determined by NYSDEC) to perform the work. Maintenance and repair work shall conform to the requirements set forth in this Plan.

SECTION 5

REPORTING AND RECORD KEEPING

5.1 INTRODUCTION

This section describes the reporting and record keeping that will be followed by the monitoring and maintenance contractor during the 30 year post-closure period. An annual report summarizing monitoring and maintenance activities will be submitted to NYSDEC and NYSDOH. Copies of all reports will be sent to NYSDEC and NYDOH at each of the following locations:

ONE COPY TO:

Chief, Operation, Maintenance & Support Section
New York State Department of Environmental Conservation
50 Wolf Road
Albany, NY 12233-7010
(518) 457-0927

TWO COPIES TO:

Regional Hazardous Waste Engineer
New York State Department of Environmental
Conservation - Region 9
270 Michigan Avenue
Buffalo, New York 14203-2999

ONE COPY TO:

Director, Bureau of Environmental Enforcement Exposure Investigation
New York State Department of Health
2 University Place
Albany, New York 12203

5.2 WELL MONITORING REPORTS

The report will include all groundwater data. The report outline will be based on the data reporting regulations in 6 NYCRR Part 360-2.11(c)(4)(iv), including tables showing collection data, analytical results and applicable NYSDEC and NYSDOH standards and guidance values, a summary of contraventions of water quality standards and a discussion of results.

5.3 ANNUAL REPORT

The annual report will contain a summary of water quality information from the well monitoring reports with special note of any changes in water quality which have occurred throughout the year. The annual report will also summarize the inspection and maintenance activities on the cap and site structures for the year.

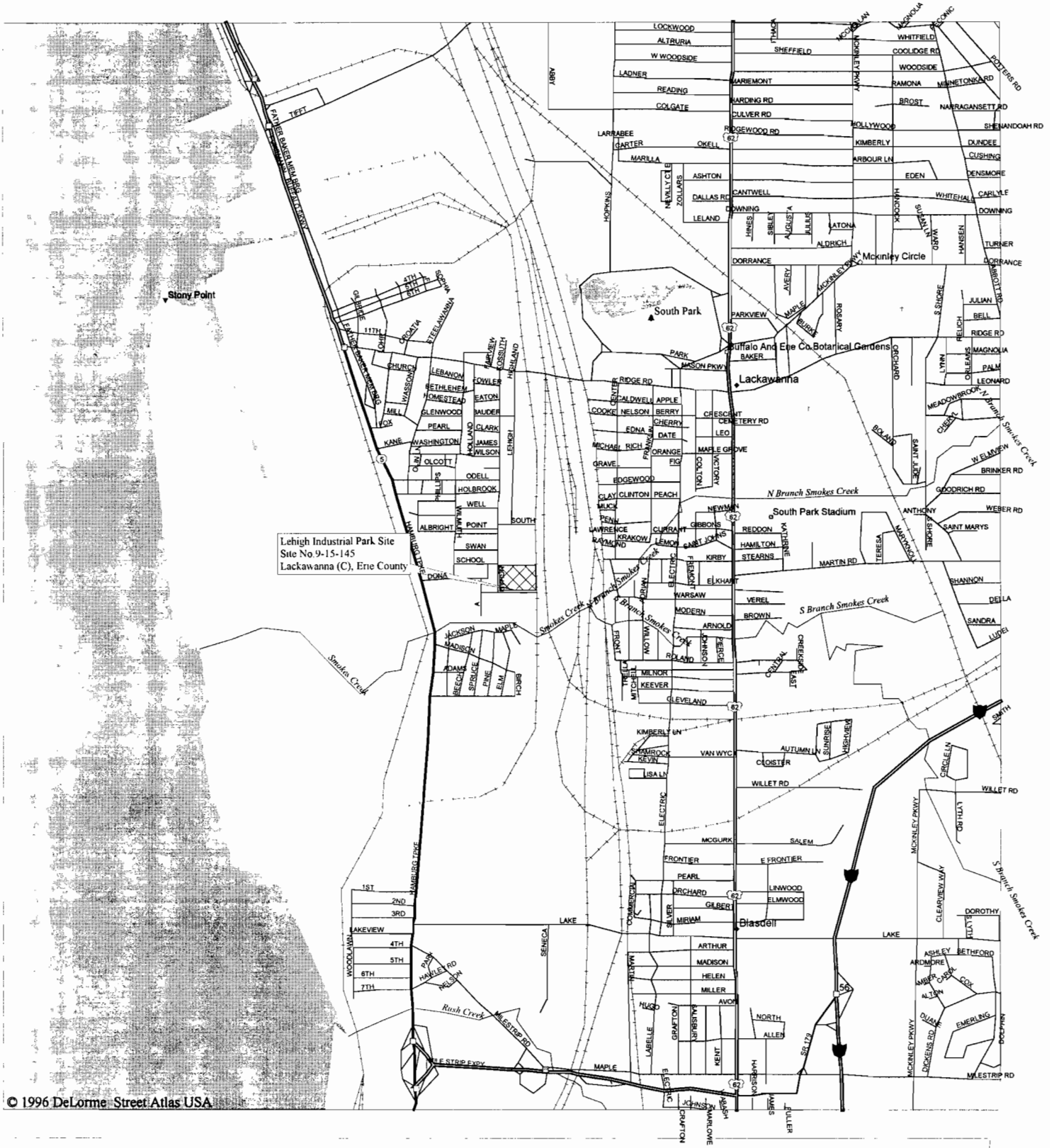
5.4 RECORD KEEPING

Records of data, drawings, and calculations concerning any work proposed or completed at the site will be kept on permanent file by NYSDEC, Albany, New York and Buffalo, New York. In addition, records will be maintained in conjunction with the post-closure monitoring and maintenance at the waste cell. For example, wells will be monitored and data recorded on a data sheet. This and other investigative results will be incorporated into reports that are submitted to or prepared by NYSDEC. Included in the reports will be appendices with copies of data sheets, log books, and laboratory analysis results. The originals will be kept by NYSDEC or its contractor for performing maintenance and monitoring activities for at least five years.

APPENDIX A

SITE LOCATION MAP

Lehigh Industrial Park, Site No. 9-15-145



Mag 14.00
Tue Jun 02 14:07 1998

Scale 1:31,250 (at center)

2000 Feet

1000 Meters

- Secondary SR/Road/Hwy Ramp
- Major Connector
- State Route
- Interstate/Limited Access

APPENDIX B

HEALTH AND SAFETY PLAN

HEALTH AND SAFETY PLAN

For

Post-Closure Monitoring and Maintenance

Field Activities

at the

Lehigh Industrial Park Site

Prepared By:

PARSONS ENGINEERING SCIENCE, INC.
290 Elwood Davis Road, Suite 312
Liverpool, New York 13088

MARCH 1995

Reviewed and Approved By:

	Name	Date
NYSDEC Representative	_____	_____

TABLE OF CONTENTS

LIST OF FIGURES

EMERGENCY CONTACTS

In the event of any situation or unplanned occurrence requiring assistance, the appropriate contact(s) should be made from the list below. For emergency situations, contact should first be made with the site coordinator who will notify emergency personnel who will then contact the appropriate response teams. This emergency contacts list must be in an easily accessible location at the site.

Contacts	Phone Number
Police Department	(716) 822-4900
Fire Department	(716) 823-0212
Poison Control Center	(800) 888-7655
Emergency Dispatch Center	
Emergency Management Office	

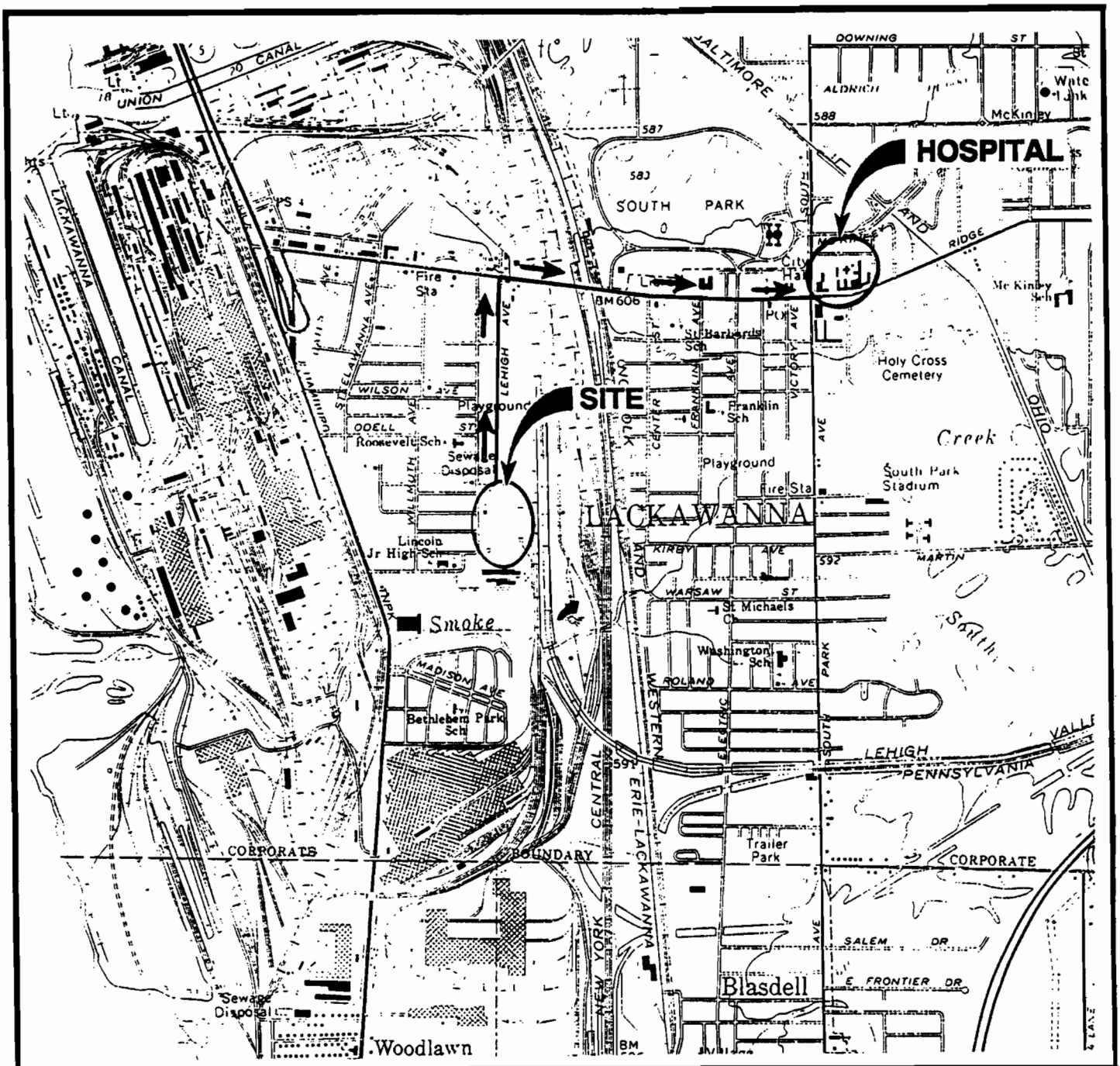
Medical Emergency

Our Lady of Victory Hospital 55 Melroy Ave. Lackawanna	(716) 825-8000

ROUTE TO HOSPITAL:

Head north on Lehigh Ave. to Ridge Rd., and make a right on Ridge Rd. Go about 1.5 miles to South Park Ave. and continue over South Park on Ridge Rd. Hospital Emergency Room entrance will be on the left. SEE MAP, NEXT PAGE.

Travel time approximately 10 minutes.



SOURCE: U.S.G.S. 7.5 MINUTE SERIES TOPOGRAPHIC BUFFALO SE QUADRANGLE. 1965

QUADRANGLE LOCATION

LATITUDE: 42°49'00"
LONGITUDE: 78°50'25"

SCALE

0 2000 4000 FT.

2000 FT.

ENGINEERING-SCIENCE

NEW YORK STATE DEPARTMENT OF ENVIRONMENTAL CONSERVATION

ROUTE TO HOSPITAL

LEHIGH INDUSTRIAL PARK SITE

SECTION B.1

INTRODUCTION

B.1.1 PURPOSE AND REQUIREMENTS

The purpose of this health and safety plan is to establish personnel protection standards and mandatory safety practices and procedures for post-closure field activities at the LIP site.

This plan assigns responsibilities, establishes standard operating procedures, and provides for contingencies that may arise while operations are being conducted at the site. The provisions of the plan are mandatory for all on-site personnel performing groundwater monitoring and site maintenance. All personnel who engage in project activities must be familiar with this plan and sign-off on the Plan Acceptance Form (Attachment A) prior to beginning work on-site. The Plan Acceptance Form must be submitted to the Health and Safety Officer assigned to this site. All personnel should abide by the standard safe work practices presented in Attachment B. Except for regular site maintenance such as mowing and erosion repair, all field personnel must comply with its requirements.

B.1.2 SITE HISTORY AND DESCRIPTION

The LIP site (formerly Roblin Steel) is located at 31 South Street, Lackawanna, New York. The 9.1 acre parcel was operated by Roblin Scrap Products as a materials processing facility processing scrap metals and other materials. At least two incidences of leaking transformers have been documented for the site, one occurring in 1979, the other in 1988. The first spill (1979) was reportedly remediated, at the time. The second spill will be remediated in 1995 and the site is currently listed as a Class 2 site on the State Registry of Inactive Hazardous Waste Sites.

The LIP site will be remediated in the Summer and Fall of 1995. The remediated site will consist of a one foot thick clean soil cover over all contaminated and potentially contaminated areas which include a 350 feet by 570 feet pile of non-hazardous waste soil and debris consolidated from the rest of the site. No hazardous waste will be left on site. The site will be fully revegetated to control erosion and fenced off to control access (Drawing ____, Appendix A).

B.1.3 SCOPE OF WORK

Field tasks to be performed at the LIP site include:

- Site inspection;
- Site maintenance; and
- Groundwater monitoring well sampling.

SECTION B.2

RISK ANALYSIS

B.2.1 CHEMICAL HAZARDS

Contaminants which may be encountered while conducting field tasks at the LIP site include non-hazardous level PCBs, metals such as lead, mercury, chromium, arsenic, and cadmium, and volatile and semi-volatile organic compounds.

In addition to the chemicals detected on-site, some of the solvents used in the processing of samples and for the decontamination of equipment are potentially hazardous to human health if they are not used properly. Material Safety Data Sheets for these compounds are included in Attachment C. Some or all of these compounds may be used in the current tasks to be performed at the site.

B.2.2 PHYSICAL HAZARDS

B.2.2.1 Heat Stress

Heat stress may be present when working during a hot summer day. Proper training and preventive measures will aid in averting loss of worker productivity and serious illness. Heat stress prevention is particularly important because once a person suffers from heat stroke or heat exhaustion, that person may be predisposed to additional heat related illness. To avoid heat stress the following steps should be taken:

- Adjust work schedules.
  - Modify work/rest schedules according to monitoring requirements.
  - Mandate work slowdowns as needed.
  - Perform work during cooler hours of the day if possible or at night if adequate lighting can be provided.
- Provide shelter (air-conditioned, if possible) or shaded areas to protect personnel during rest periods.
- Maintain worker's body fluids at normal levels. This is necessary to ensure that the cardiovascular system functions adequately. Daily fluid intake must approximately equal the amount of water lost in sweat, i.e., eight fluid ounces (0.23 liters) of water must be ingested for approximately every eight ounces (0.23 kg) of weight lost. The normal thirst mechanism is not sensitive enough to ensure that enough water will be drunk to replace lost sweat. When heavy sweating occurs, encourage the worker to drink more. The following strategies may be useful:
  - Maintain water temperature 50° to 60°F (10° to 16.6°C).
  - Provide small disposal cups that hold about four ounces (0.1 liter).
  - Have workers drink 16 ounces (0.5 liters) of fluid (preferably water or dilute drinks) before beginning work.
  - Urge workers to drink a cup or two every 15 to 20 minutes, or at each monitoring break. A total of 1 to 1.6 gallons (4 to 6 liters) of fluid per day are recommended, but more may be necessary to maintain body weight.

- Train workers to recognize the symptoms of heat related illness.

B.2.2.2 Cold-Related Illness

If work on this project begins in the winter months, thermal injury due to cold exposure can become a problem for field personnel. Systemic cold exposure is referred to as hypothermia. Local cold exposure is generally labeled frostbite.

Hypothermia. Hypothermia is defined as a decrease in the patient core temperature below 96°F. The body temperature is normally maintained by a combination of central (brain and spinal cord) and peripheral (skin and muscle) activity. Interferences with any of these mechanisms can result in hypothermia, even in the absence of what normally is considered a "cold" ambient temperature. Symptoms of hypothermia include: shivering, apathy, listlessness, sleepiness, and unconsciousness.

Frostbite. Frostbite is both a general and medical term given to areas of local cold injury. Unlike systemic hypothermia, frostbite rarely occurs unless the ambient temperatures are less than freezing and usually less than 20°F. Symptoms of frostbite are: a sudden blanching or whitening of the skin; the skin has a waxy or white appearance and is firm to the touch; tissues are cold, pale, and solid.

B.2.2.3 Prevention of Cold Related Illness

- Educate workers to recognize the symptoms of frostbite and hypothermia
- Identify and limit known risk factors:
- Assure the availability of enclosed, heated environment on or adjacent to the site.
- Assure the availability of dry changes of clothing.
- Develop the capability for temperature recording at the site.
- Assure the availability of warm drinks.

Monitoring

Start (oral) temperature recording at the job site:

- At the Field Team Leader's discretion when suspicion is based on changes in a worker's performance or mental status.
- At a worker's request.
- As a screening measure, two times per shift, under unusually hazardous conditions (e.g., wind-chill less than 20°F, or wind-chill less than 30°F with precipitation).
- As a screening measure whenever any one worker on the site develops hypothermia.

Any person developing moderate hypothermia (a core temperature of 92°F) cannot return to work for 48 hours.

SECTION B.3

PERSONNEL PROTECTION AND MONITORING

B.3.1 MEDICAL SURVEILLANCE

The sampling and/or monitoring personnel or contractor will utilize the services of a licensed occupational health physician with knowledge and/or experience in the hazards associated with the project to provide the medical examinations and surveillance specified herein.

B.3.1.1 Medical Examination

Personnel involved in the groundwater monitoring operation must have undergone medical evaluation prior to any field work, and thereafter at 12-month intervals. The 12-month medical examination includes a complete medical and work history and a standard occupational physical including examination of all major organ systems, complete blood count with differential (CBC), and a SMAC/23 blood chemistry screen which includes calcium, phosphorous, glucose, uric acid, BUN, creatinine, albumin, SGPT, SGOT, LDH, globulin, A/G ratio, alkaline phosphatase, total protein, total bilirubin, triglyceride, cholesterol, and a creatinine/BUN ratio. Additionally a pulmonary function test will be performed by trained personnel to record Forced Vital Capacity (FVC) and Forced Expiratory Volume in seconds ($FEV_{1.0}$). An audiogram and visual acuity measurement, including color perception, is provided The medical exam is performed under the direction of a licensed Occupational Health Physician. A medical certification as to the fitness or unfitness for employment on hazardous waste projects, or any restrictions on his/her utilization that may be indicated, is provided by the physician. This evaluation will be repeated as indicated by substandard performance or evidence of particular stress that is evident by injury or time loss illness on the part of any worker.

B.3.2 SITE SPECIFIC TRAINING

The site monitoring and maintenance contractor will be responsible for developing a site specific occupational hazard training program and providing training to all personnel that are to work at the site. This training will consist of the following topics:

- Names of personnel responsible for site safety and health.
- Safety, health, and other hazards at the site.
- Proper use of personal protective equipment.
- Work practices by which the employee can minimize risk from hazards.
- Safe use of engineering controls and equipment on the site.
- Acute effects of compounds at the site.
- Decontamination procedures.

B.3.3 PERSONAL PROTECTIVE EQUIPMENT AND ACTION LEVELS

Personnel protection to groundwater monitoring at the LIP site will necessitate the following equipment:

Level D

- Coveralls
- Safety boots
- PVC inner and chemically resistant outer gloves (must be worn during all sampling activities)
- Splash goggles (must be worn if a splash hazard is present)

All personal protective equipment used during the course of any field activities must meet the following OSHA standards:

Type of Protection	Regulation	Source
Eye and Face	29 CFR 1910.133	ANSI Z87.1-1968
Head	29 CFR 1910.135	ANSI Z89.1-1969
Foot	29 CFR 1910.136	ANSI Z41.1-1967

ANSI = American National Standards Institute

B.3.4 AIR MONITORING REQUIREMENTS

Air monitoring is not expected to be necessary during the monitoring and maintenance activities at the LIP site. However, if monitoring and maintenance activities do occur around enclosed spaces or material containers, oxygen deficiency and presence of combustible gases should be monitored.

All monitoring instruments must be calibrated and maintained periodically. The limitations and possible sources of errors for each instrument must be understood by the operator. It is important that the operator ensures that the instrument responds properly to the substances it was designed to monitor. Portable air quality monitoring equipment that measures total ionizables present such as the Photovac-Microtip HL-2000 must be calibrated at least once each day. Combustible gas/oxygen meters (explosimeters) such as the MSA Model 360 must be calibrated at least once each week. The specific instruction for calibration and maintenance provided for each instrument should be followed.

SECTION B.4

ACCIDENT PREVENTION AND CONTINGENCY PLAN

B.4.1 ACCIDENT PREVENTION

On a day-to-day basis, individual personnel should be constantly alert for indicators of potentially hazardous situations and for signs and symptoms in themselves and others that warn of hazardous conditions and exposures. Rapid recognition of dangerous situations can avert an emergency. Before each work assignment, the personnel should be aware of the following:

- Tasks to be performed;
- Time constraints (e.g. rest breaks, cartridge changes);
- Hazards that may be encountered, including their effects, how to recognize symptoms or monitor them, concentration limits, or other danger signals; and
- Emergency procedures.

B.4.2 CONTINGENCY PLAN

In the event that an emergency develops on site, the procedures delineated herein are to be immediately followed. Emergency conditions are considered to exist if:

- Any member of the field crew is involved in an accident or experiences any adverse effects or symptoms of exposure while on site;
- A condition is discovered that suggests the existence of a situation more hazardous than anticipated.

General emergency procedures, and specific procedures for personal injury and chemical exposure, are described in the health and safety plan. As a general precaution, emergency equipment available at the work site should include a first aid kit and eye wash.

B.4.2.1 Chemical Exposure

If a member of the field crew demonstrates symptoms of chemical exposure the procedures outlined below should be followed:

- Another team member (buddy) should remove the individual from the immediate area of contamination. The buddy should communicate to the Field Team Leader (via two-way radio or hand signals) of the chemical exposure. The Field Team Leader should contact the appropriate emergency response agency.
- Precautions should be taken to avoid exposure of other individuals to the chemical.
- If the chemical is on the individual's clothing, the chemical should be neutralized or removed if it is safe to do so.

- If the chemical has contacted the skin, the skin should be washed with copious amounts of water.
- In case of eye contact, an emergency eye wash should be used. Eyes should be washed out for at least 15 minutes.
- All chemical exposure incidents must be reported in writing to the Office Health and Safety Representative. The Site Safety Officer or Field Team Leader is responsible for notifying the Project Manager immediately and completing the accident report (see Attachment A).

B.4.2.2 Air Monitoring Requirements

Air monitoring is not required unless entry into a confined space is needed.

B.4.2.3 Personal Injury

In case of personal injury at the site, the following procedures should be followed:

- Another team member (buddy) should signal the Field Team Leader that an injury has occurred.
- A field team member trained in first aid can administer treatment to an injured worker.
- The victim should then be transported to the nearest hospital or medical center. If necessary, an ambulance should be called to transport the victim.
- The Field Team is responsible for making certain that an accident report form is completed (see Attachment C). This form is to be submitted to the Office Health and Safety Representative. Follow-up action should be taken to correct the situation that caused the accident.

B.4.2.4 Evacuation Procedures

- The Field Team Leader will initiate evacuation procedure by signalling to leave the site.
- All personnel in the work area should evacuate the area and meet in the common designated area.
- All personnel suspected to be in or near the contract work area should be accounted for and the whereabouts of missing persons determined immediately.
- Further instruction will then be given by the Field Team Leader.

B.4.2.5 Procedures Implemented in the Event of a Major Fire, Explosion, or On-Site Health Emergency Crisis

- Notify the paramedics and/or fire department, as necessary;
- Signal the evacuation procedure previously outlined and implement the entire procedure;
- Notify LIP site personnel;
- Isolate the area;

- Stay upwind of any fire;
- Keep the area surrounding the problem source clear after the incident occurs;
- Complete accident report form and distribute to appropriate personnel.
- Smoking, eating and drinking will not be permitted on site (see Attachment B).

HEALTH AND SAFETY PLAN ATTACHMENT A

AIR MONITORING EQUIPMENT CALIBRATION AND MAINTENANCE

AIR MONITORING EQUIPMENT CALIBRATION AND MAINTENANCE

All monitoring instruments must be calibrated and maintained periodically. The limitations and possible sources of errors for each instrument must be understood by the operator. It is important that the operator ensures that the instrument responds properly to the substances it was designed to monitor. Portable air quality monitoring equipment that measures total ionizables present, such as the Photovac MicroTIP HL-2000® must be calibrated at least once each day. Combustible gas/oxygen/% LEL meters (explosimeters) such as the MSA Model 360®, must be zeroed at the beginning of each sampling period. The specific instructions for calibration and maintenance provided for each instrument should be followed.

HEALTH AND SAFETY PLAN ATTACHMENT B

FORMS FOR HEALTH AND SAFETY-RELATED ACTIVITIES

Note: The OSHA Job Safety and Health Protection Poster must be posed prominently during intrusive field activities. The following page is an example of the poster to be used in the field. The actual poster must be a 11 inch by 17 inch size version of this page.

JOB SAFETY & HEALTH PROTECTION

The Occupational Safety and Health Act of 1970 provides job safety and health protection for workers by promoting safe and healthful working conditions throughout the Nation. Provisions of the Act include the following:

Employers

All employers must furnish to employees employment and a place of employment free from recognized hazards that are causing or are likely to cause death or serious harm to employees. Employers must comply with occupational safety and health standards issued under the Act.

Employees

Employees must comply with all occupational safety and health standards, rules, regulations and orders issued under the Act that apply to their own actions and conduct on the job.

The Occupational Safety and Health Administration (OSHA) of the U.S. Department of Labor has the primary responsibility for administering the Act. OSHA issues occupational safety and health standards, and its Compliance Safety and Health Officers conduct jobsite inspections to help ensure compliance with the Act.

Inspection

The Act requires that a representative of the employer and a representative authorized by the employees be given an opportunity to accompany the OSHA inspector for the purpose of aiding the inspection.

Where there is no authorized employee representative, the OSHA Compliance Officer must consult with a reasonable number of employees concerning safety and health conditions in the workplace.

Complaint

Employees or their representatives have the right to file a complaint with the nearest OSHA office requesting an inspection if they believe unsafe or unhealthful conditions exist in their workplace. OSHA will withhold, on request, names of employees complaining.

The Act provides that employees may not be discharged or discriminated against in any way for filing safety and health complaints or for otherwise exercising their rights under the Act.

Employees who believe they have been discriminated against may file a complaint with their nearest OSHA office within 30 days of the alleged discriminatory action.

Citation

If upon inspection OSHA believes an employer has violated the Act, a citation alleging such violations will be issued to the employer. Each citation will specify a time period within which the alleged violation must be corrected.

The OSHA citation must be prominently displayed at or near the place of alleged violation for three days, or until it is corrected, whichever is later, to warn employees of dangers that may exist there.

Proposed Penalty

The Act provides for mandatory civil penalties against employers of up to $7,000 for each serious violation and for optional penalties of up to $7,000 for each nonserious violation. Penalties of up to $7,000 per day may be proposed for failure to correct violations within the proposed time period and for each day the violation continues beyond the prescribed abatement date. Also, any employer who willfully or repeatedly violates the Act may be assessed penalties of up to $70,000 for each such violation. A minimum penalty of $5,000 may be imposed for each willful violation. A violation of posting requirements can bring a penalty of up to $7,000.

There are also provisions for criminal penalties. Any willful violation resulting in the death of any employee, upon conviction, is punishable by a fine of up to $250,000 for $500,000 if the employer is a corporation), or by imprisonment for up to six months, or both. A second conviction of an employer doubles the possible term of imprisonment. Falsifying records, reports, or applications is punishable by a fine of $10,000 or up to six months in jail or both.

Voluntary Activity

While providing penalties for violations, the Act also encourages efforts by labor and management, before an OSHA inspection, to reduce workplace hazards voluntarily and to develop and improve safety and health programs in all workplaces and industries. OSHA's Voluntary Protection Programs recognize outstanding efforts of this nature.

OSHA has published Safety and Health Program Management Guidelines to assist employers in establishing or perfecting programs to prevent or control employee exposure to workplace hazards. There are many public and private organizations that can provide information and assistance in this effort, if requested. Also, your local OSHA office can provide considerable help and advice on solving safety and health problems or can refer you to other sources for help such as training.

Consultation

Free assistance in identifying and correcting hazards and in improving safety and health management is available to employers, without citation or penalty, through OSHA-supported programs in each State. These programs are usually administered by the State Labor or Health department or a State university.

Posting Instructions

Employers in States operating OSHA approved State Plans should obtain and post the State's equivalent poster.

Under provisions of Title 29,Code of Federal Regulations, Part 1903.2(a)(1) employers must post this notice (or facsimile) in a conspicuous place where notices to employees are customarily posted.

More Information

Additional information and copies of the Act, specific OSHA safety and health standards, and other applicable regulations may be obtained from your employer or from the nearest OSHA Regional Office in the following locations:

Atlanta, GA	(404) 347-3573
Boston, MA	(617) 565-7164
Chicago, IL	(312) 353-2220
Dallas, TX	(214) 767-4731
Denver, CO	(303) 844-3061
Kansas City, MO	(816) 426-5861
New York, NY	(212) 337-2378
Philadelphia, PA	(215) 596-1201
San Francisco, CA	(415) 744-6670
Seattle,WA	(206) 553-5930

To report suspected fire hazards, imminent danger safety and health hazards in the workplace, or other job safety and health emergencies, such as toxic waste in the workplace, call OSHA's 24-hour hotline: 1-800-321-OSHA.

Washington, DC
1992 (Reprinted)
OSHA 2203

Robert B. Reich, Secretary of Labor

U.S. Department of Labor

Occupational Safety and Health Administration

This information will be made available to sensory impaired individuals upon request. Voice phone: (202) 219-8615; TDD message referral phone: 1-800-326-2577

PARSONS ENGINEERING SCIENCE COMPANIES

ACCIDENT REPORT FORM

Project Name:_____

INJURED OR ILL EMPLOYEE

1. Name _____ Social Security #_____
   (First) (Middle) (Last)
2. Home Address _____
   (No. and Street) (City or Town) (State and Zip)
3. Age _____ 4. Sex: Male ( ) Female ( )
5. Occupation _____
   (Specific job title, not the specific activity employee was performing at time of injury)
6. Department _____
   (Enter name of department in which injured person is employed, even though they may have been temporarily working in another department at the time of injury)

EMPLOYER

7. Name _____
8. Mailing Address _____
   (No. and Street) (City or Town) (State and Zip)
9. Location (if different from mailing address):_____

THE ACCIDENT OR EXPOSURE TO OCCUPATIONAL ILLNESS

10. Place of accident or exposure _____
    (No. and Street) (City or Town) (State and Zip)
11. Was place of accident or exposure on employer's premises? _____(Yes/No)
12. What was the employee doing when injured?_____

(Be specific - was employee using tools or equipment or handling material?)

13. How did the accident occur? _____
    (Describe fully the events that resulted in the injury or

occupational illness. Tell what happened and how. Name objects and

substances involved. Give details on all factors that led to accident. Use separate sheet if needed)

PARSONS ENGINEERING SCIENCE COMPANIES

ACCIDENT REPORT FORM

14. Time of accident: _____

15. Date of injury or initial diagnosis of occupational illness _____ (Date)

16. ES WITNESS TO ACCIDENT

(Name)	(Affiliation)	(Phone No.)
_____	_____	_____
_____	_____	_____
_____	_____	_____

OCCUPATIONAL INJURY OR OCCUPATIONAL ILLNESS

17. Describe the injury or illness in detail; indicate part of body affected.

_____

_____

18. Name the object or substance which directly injured the employee. (For example, object that struck employee; the vapor or poison inhaled or swallowed; the chemical or radiation that irritated the skin; or in cases of strains, hernias, etc., the object the employee was lifting, pulling, etc.)

_____

_____

19. Did the accident result in employee fatality? _____ (Yes or No)

20. Number of lost workdays ____/restricted workdays _____ resulting from injury or illness?

OTHER

21. Did you see a physician for treatment? _____ (Yes or No) _____ (Date)

22. Name and address of physician _____

_____

(No. and Street) (City or Town) (State and Zip)

23. If hospitalized, name and address of hospital _____

_____

(No. and Street) (City or Town) (State and Zip)

Date of report _____ Prepared by _____

Official position _____

REQUIRED HEALTH AND SAFETY DOCUMENTATION

The subcontractor must provide proof that all employees who will work on the site meet the medical and training requirements of the applicable OSHA regulations. Complete documentation for all employees must be submitted to the Engineering-Science project manager at least one week in advance of the initiation of field activities. Workers without up-to-date documentation will not be allowed to engage in any field work. Proof of the following is required, as indicated (checked) below:

(1) HAZARDOUS WASTE WORK (29 CFR 1910.120)

- ☐ Physical within the last 12 months which meets the requirements of 29 CFR 1910.120.
- ☐ Certified by a physician as fit to wear a respirator.
- ☐ Fit tested for *full-face* air-purifying respirator within the last 6 months.
- ☐ Fit tested for *half-face* air-purifying respirator within the last 6 months.
- ☐ *40-Hour* OSHA Training for hazardous waste operations.
- ☐ *24-Hour* or 40-Hour OSHA Training for hazardous waste operations.
- ☐ 8-Hour Refresher training within the last 12 months.

(2) INDUSTRIAL/TANK WORK
(Only necessary if respirators will be used)

- ☐ Physical within the last 12 months.
- ☐ Certified by a physician to wear a respirator.
- ☐ Fit tested for *full-face* air-purifying respirator within the last 6 months.
- ☐ Fit tested for *half-face* air-purifying respirator within the last 6 months.

Note that any tank work which is the result of a clean-up operation initiated by any governmental body (federal, state, etc.) falls under the purview of the OSHA hazardous waste regulations as specified in 29 CFR 1910.120.

PROJECT HEALTH AND SAFETY DOCUMENT TRACKING FORM

Project Name: _____
Project Number: _____
Project Manager: _____
Site Safety Officer: _____
Update Number: _____

Project Type (Check One): [ ] Hazardous Waste
[ ] Industrial/Tank

Date Form Completed:

ES or Subcontractor (1)	Dates of Field Work	Names of Site Workers	EFFECTIVE DATE OF DOCUMENT (2)						
			Physical	Medical Respirator Fitness	Training		8-Hour Update	HASP Sign-Off	Site-Specific Training Sign-Off
					24-Hour	40-Hour			
ES									
ES									

(1) List each subcontractor below the double line.
(2) NA = not applicable. The requirements vary depending on the type of project and the particular worker involved.

INSTRUCTIONS: THIS FORM MUST BE COMPLETED BY THE PROJECT MANAGER AND SUBMITTED TO THE OFFICE H&S REPRESENTATIVE PRIOR TO THE INITIATION OF SITE ACTIVITIES. IT MUST BE UPDATED REGULARLY BY THE SITE SAFETY OFFICER THEREAFTER UNTIL FIELD WORK IS COMPLETED. COPIES OF ALL UPDATES MUST BE PROVIDED TO THE OFFICE H&S REPRESENTATIVE. ALL DOCUMENTATION LISTED IS TO BE PLACED IN THE H&S FILE FOR EACH PROJECT. FILES WILL BE AUDITED.

PROJECT HEALTH AND SAFETY PLAN

AND WORK PLAN ACCEPTANCE FORM

I have read and agree to abide by the contents of the Work Plan and Health and Safety Plan for the following project:

_____	_____
(Project Title)	(Project Number)

Furthermore, I have read and am familiar with the work plan or proposal which describes the field work to be conducted and the procedures to be utilized in the conduct of this work.

Name (print)	Signature	Date
_____	_____	_____
_____	_____	_____
_____	_____	_____
_____	_____	_____
_____	_____	_____
_____	_____	_____
_____	_____	_____
_____	_____	_____
_____	_____	_____
_____	_____	_____
_____	_____	_____

Place in project Health and Safety File as soon as possible

SITE-SPECIFIC HEALTH AND SAFETY TRAINING

I hereby confirm that site-specific health and safety training has been conducted by the site health and safety officer which included:

- Names of personnel responsible for site safety and health
- Safety, health, and other hazards at the site
- Proper use of personal protective equipment
- Work practices by which the employee can minimize risk from hazards
- Safe use of engineering controls and equipment on the site
- Acute effects of compounds at the site
- Decontamination procedures

For the following project:

_____	_____
(Project Title)	(Project Number)

Name (print)	Signature	Date
_____	_____	_____
_____	_____	_____
_____	_____	_____
_____	_____	_____
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_____	_____	_____

Place in project Health and Safety File as soon as possible

HEALTH AND SAFETY PLAN ATTACHMENT C

MATERIAL SAFETY DATA SHEETS

METHANOL
METHANOL
METHANOL

MATERIAL SAFETY DATA SHEET

FISHER SCIENTIFIC
CHEMICAL DIVISION
1 REAGENT LANE
FAIR LAWN NJ 07410
(201) 796-7100

EMERGENCY NUMBER: (201) 796-7100
CHEMTREC ASSISTANCE (800) 424-9300

SUBSTANCE IDENTIFICATION

CAS-NUMBER 67-56-1

SUBSTANCE: **METHANOL**

TRADE NAMES/SYNONYMS:
METHYL ALCOHOL; WOOD ALCOHOL; METHYL HYDROXIDE, CARBINOL, MONOHYDROXYMETHANE, WOOD SPIRIT, WOOD NAPHTHA, METHYLOL, COLONIAL SPIRIT, COLUMBIAN SPIRIT; PYROXYLIC SPIRIT; COULOMATIC (R) CONDITIONER SOLUTION, STANDARD WATER IN METHANOL; STCC 4909230; UN 1230; RCRA U154, A454; A452; A938; A408; A947; A935, BP1105; A412; A411; A433P; SW2;; SC95; A452SK; A408SK; A412P; A434; A412SK; A450; A433S; CH4O, ACC14280

CHEMICAL FAMILY:
HYDROXYL, ALIPHATIC

MOLECULAR FORMULA: C-H3-O-H

MOLECULAR WEIGHT: 32.04

CERCLA RATINGS (SCALE 0-3): HEALTH=3 FIRE=3 REACTIVITY=0 PERSISTENCE=0
NFPA RATINGS (SCALE 0-4): HEALTH=1 FIRE=3 REACTIVITY=0

COMPONENTS AND CONTAMINANTS

COMPONENT: METHYL ALCOHOL (METHANOL) PERCENT 100
CAS# 67-56-1

OTHER CONTAMINANTS: NONE

EXPOSURE LIMITS:
METHYL ALCOHOL (METHANOL):
200 PPM (262 MG/M3) OSHA TWA (SKIN); 250 PPM (328 MG/M3) OSHA STEL
200 PPM (262 MG/M3) ACGIH TWA (SKIN); 250 PPM (328 MG/M3) ACGIH STEL
200 PPM (262 MG/M3) NIOSH RECOMMENDED TWA (SKIN);
250 PPM (328 MG/M3) NIOSH RECOMMENDED STEL
200 PPM (262 MG/M3) DFG MAK TWA (SKIN);
400 PPM (524 MG/M3) DFG MAK 30 MINUTE PEAK, AVERAGE VALUE 4 TIMES/SHIFT

MEASUREMENT METHOD: SILICA GEL TUBE; WATER; GAS CHROMATOGRAPHY WITH FLAME IONIZATION DETECTION; (NIOSH VOL III # 2000, METHANOL)

5000 POUNDS CERCLA SECTION 103 REPORTABLE QUANTITY
SUBJECT TO SARA SECTION 313 ANNUAL TOXIC CHEMICAL RELEASE REPORTING

**OSHA LIMITS ADOPTED JANUARY 19, 1989 ARE SUBJECT TO THE DECISION OF THE 11TH CIRCUIT COURT OF APPEALS (AFL-CIO V. OSHA) AS OF JULY 7, 1992 **

PHYSICAL DATA

DESCRIPTION: CLEAR, COLORLESS LIQUID WITH A CHARACTERISTIC ALCOHOLIC ODOR

BOILING POINT: 149 F (65 C) MELTING POINT: -137 F (-94 C)

SPECIFIC GRAVITY: 0.7914 VAPOR PRESSURE: 97.25 MMHG @ 20 C

EVAPORATION RATE: (BUTYL ACETATE=1) 4.6 SOLUBILITY IN WATER: VERY SOLUBLE

ODOR THRESHOLD: 100 PPM VAPOR DENSITY: 1.11

SOLVENT SOLUBILITY: ETHER, BENZENE, ALCOHOL, ACETONE, CHLOROFORM, ETHANOL

VISCOSITY: 0.59 CPS @ 20 C

FIRE AND EXPLOSION DATA

FIRE AND EXPLOSION HAZARD

DANGEROUS FIRE HAZARD WHEN EXPOSED TO HEAT, FLAME, OR OXIDIZERS

VAPORS ARE HEAVIER THAN AIR AND MAY TRAVEL A CONSIDERABLE DISTANCE TO A SOURCE OF IGNITION AND FLASH BACK

VAPOR-AIR MIXTURES ARE EXPLOSIVE

FLASH POINT: 52 F (11 C) (CC) UPPER EXPLOSIVE LIMIT: 36.0%

LOWER EXPLOSIVE LIMIT: 6.0% AUTOIGNITION TEMP: 725 F (385 C)

FLAMMABILITY CLASS(OSHA): IB

FIREFIGHTING MEDIA:
DRY CHEMICAL, CARBON DIOXIDE, WATER SPRAY OR ALCOHOL-RESISTANT FOAM (1990 EMERGENCY RESPONSE GUIDEBOOK, DOT P 5800.5).

FOR LARGER FIRES, USE WATER SPRAY, FOG OR ALCOHOL-RESISTANT FOAM (1990 EMERGENCY RESPONSE GUIDEBOOK, DOT P 5800.5).

FIREFIGHTING:
MOVE CONTAINER FROM FIRE AREA IF YOU CAN DO IT WITHOUT RISK. DIKE FIRE-CONTROL WATER FOR LATER DISPOSAL; DO NOT SCATTER THE MATERIAL. APPLY COOLING WATER TO SIDES OF CONTAINERS THAT ARE EXPOSED TO FLAMES UNTIL WELL AFTER FIRE IS OUT. STAY AWAY FROM ENDS OF TANKS. WITHDRAW IMMEDIATELY IN CASE OF RISING SOUND FROM VENTING SAFETY DEVICE OR ANY DISCOLORATION OF TANK DUE TO FIRE. ISOLATE FOR 1/2 MILE IN ALL DIRECTIONS IF TANK, RAIL CAR OR TANK TRUCK IS INVOLVED IN FIRE (1990 EMERGENCY RESPONSE GUIDEBOOK, DOT P 5800.5, GUIDE PAGE 28)

EXTINGUISH ONLY IF FLOW CAN BE STOPPED. USE WATER IN FLOODING AMOUNTS AS FOG. SOLID STREAMS MAY NOT BE EFFECTIVE. COOL CONTAINERS WITH FLOODING QUANTITIES OF WATER. APPLY FROM AS FAR A DISTANCE AS POSSIBLE. AVOID BREATHING TOXIC VAPORS. KEEP UPWIND.

TRANSPORTATION DATA

DEPARTMENT OF TRANSPORTATION HAZARD CLASSIFICATION 49 CFR 172.101.
FLAMMABLE LIQUID

DEPARTMENT OF TRANSPORTATION LABELING REQUIREMENTS 49 CFR 172.101 AND SUBPART E:
FLAMMABLE LIQUID

DEPARTMENT OF TRANSPORTATION PACKAGING REQUIREMENTS: 49 CFR 173.118
EXCEPTIONS: 49 CFR 173.118

FINAL RULE ON HAZARDOUS MATERIALS REGULATIONS (HMR, 49 CFR PARTS 171-180). DOCKET NUMBERS HM-181, HM-181A, HM-181B, HM-181C, HM-181D AND HM-204. EFFECTIVE DATE OCTOBER 1, 1991. HOWEVER, COMPLIANCE WITH THE REGULATIONS IS AUTHORIZED ON AND AFTER JANUARY 1, 1991. (55 FR 52402, 12/21/90)

EXCEPT FOR EXPLOSIVES, INHALATION HAZARDS, AND INFECTIOUS SUBSTANCES, THE EFFECTIVE DATE FOR HAZARD COMMUNICATION REQUIREMENTS IS EXTENDED TO OCTOBER 1, 1993. (56 FR 47158, 09/18/91)

U.S. DEPARTMENT OF TRANSPORTATION SHIPPING NAME ID NUMBER, 49 CFR 172.101:
METHYL ALCOHOL-UN 1230

U.S. DEPARTMENT OF TRANSPORTATION HAZARD CLASS OR DIVISION, 49 CFR 172.101:
3 - FLAMMABLE LIQUID

U.S. DEPARTMENT OF TRANSPORTATION PACKING GROUP, 49 CFR 172.101
PG II

U.S. DEPARTMENT OF TRANSPORTATION LABELING REQUIREMENTS, 49 CFR 172.101 AND SUBPART E
FLAMMABLE LIQUID, POISON

U.S. DEPARTMENT OF TRANSPORTATION PACKAGING AUTHORIZATIONS
EXCEPTIONS: NONE
NON BULK PACKAGING, 49 CFR 173.202
BULK PACKAGING: 49 CFR 173.243

U.S. DEPARTMENT OF TRANSPORTATION QUANTITY LIMITATIONS 49 CFR 172.101.
PASSENGER AIRCRAFT OR RAILCAR: 1 L
CARGO AIRCRAFT ONLY: 60 L

TOXICITY

METHYL ALCOHOL (METHANOL):
IRRITATION DATA: 20 MG/24 HOURS SKIN-RABBIT MODERATE, 40 MG EYE-RABBIT MODERATE; 100 MG/24 HOURS EYE-RABBIT MODERATE.
TOXICITY DATA: 86,000 MG/M3 INHALATION-HUMAN TCLO; 300 PPM INHALATION-HUMAN TCLO; 64,000 PPM/4 HOURS INHALATION-RAT LC50; 1000 PPM INHALATION-MONKEY LCLO; 50 GM/M3/2 HOURS INHALATION-MOUSE LCLO; 44,000 MG/M3/6 HOURS INHALATION-CAT LCLO; 15,800 MG/KG SKIN-RABBIT LD50; 393 MG/KG SKIN-MONKEY LDLO; 428 MG/KG ORAL-HUMAN LDLO, 143 MG/KG ORAL-HUMAN LDLO; 8422 MG/KG ORAL-MAN LDLO; 3429 MG/KG ORAL-MAN TDLO; 4 GM/KG ORAL-WOMAN TDLO; 7 GM/KG

ORAL-MONKEY LD50: 5628 MG/KG ORAL-RAT LD50: 7300 MG/KG ORAL-MOUSE LD50: 14,200 MG/KG ORAL-RABBIT LD50: 7500 MG/KG ORAL-DOG LDLO: 9800 MG/KG SUBCUTANEOUS-MOUSE LD50: 2131 MG/KG INTRAVENOUS-RAT LD50: 4710 MG/KG INTRAVENOUS-MOUSE LD50: 8907 MG/KG INTRAVENOUS-RABBIT LD50: 4641 MG/KG INTRAVENOUS-CAT LDLO: 7529 MG/KG INTRAPERITONEAL-RAT LD50: 10,765 MG/KG INTRAPERITONEAL-MOUSE LD50: 1826 MG/KG INTRAPERITONEAL-RABBIT LD50: 3556 MG/KG INTRAPERITONEAL-GUINEA PIG LD50: 8555 MG/KG INTRAPERITONEAL-HAMSTER LD50: 868 MG/KG UNREPORTED-MAN LDLO: MUTAGENIC DATA (RTECS); REPRODUCTIVE EFFECTS DATA (RTECS)
CARCINOGEN STATUS: NONE
LOCAL EFFECTS: IRRITANT- SKIN, EYE
ACUTE TOXICITY LEVEL: SLIGHTLY TOXIC BY DERMAL ABSORPTION AND INGESTION. RELATIVELY NON-TOXIC BY INHALATION.
TARGET EFFECTS: CENTRAL NERVOUS SYSTEM DEPRESSANT, NEUROTOXIN
AT INCREASED RISK FROM EXPOSURE: PERSONS WITH KIDNEY, EYE OR SKIN DISORDERS

HEALTH EFFECTS AND FIRST AID

INHALATION:
METHYL ALCOHOL (METHANOL)
NARCOTIC/NEUROTOXIN. 25,000 PPM IMMEDIATELY DANGEROUS TO LIFE OR HEALTH
- ACUTE EXPOSURE- MAY CAUSE IRRITATION OF THE MUCOUS MEMBRANES, COUGHING, OPPRESSION IN THE CHEST, TRACHEITIS, BRONCHITIS, TINNITUS, UNSTEADY GAIT, TWITCHING, COLIC, CONSTIPATION, NYSTAGMUS, AND BLEPHAROSPASM. SYMPTOMS FROM OCCUPATIONAL EXPOSURE INCLUDE PARESTHESIAS, NUMBNESS AND SHOOTING PAINS IN THE HANDS AND FOREARMS, METABOLIC ACIDOSIS, AND EFFECTS ON THE EYES AND CENTRAL NERVOUS SYSTEM MAY OCCUR AS DETAILED IN ACUTE INGESTION
- CHRONIC EXPOSURE- REPEATED OR PROLONGED EXPOSURE MAY CAUSE EFFECTS AS IN ACUTE INGESTION. REPEATED EXPOSURE TO 200-375 PPM CAUSED RECURRENT HEADACHES IN WORKERS. EXPOSURE FOR 4 YEARS TO 1200-8000 PPM RESULTED IN MARKED DIMINUTION OF VISION AND ENLARGEMENT OF THE LIVER IN A WORKMAN. REPRODUCTIVE EFFECTS HAVE BEEN REPORTED IN ANIMALS

FIRST AID- REMOVE FROM EXPOSURE AREA TO FRESH AIR IMMEDIATELY. IF BREATHING HAS STOPPED, PERFORM ARTIFICIAL RESPIRATION. KEEP PERSON WARM AND AT REST. TREAT SYMPTOMATICALLY AND SUPPORTIVELY. GET MEDICAL ATTENTION IMMEDIATELY

SKIN CONTACT:
METHYL ALCOHOL (METHANOL).
IRRITANT/NARCOTIC/NEUROTOXIN
- ACUTE EXPOSURE- CONTACT WITH LIQUID MAY CAUSE IRRITATION. SKIN ABSORPTION MAY OCCUR AND CAUSE METABOLIC ACIDOSIS AND EFFECTS ON THE EYES AND CENTRAL NERVOUS SYSTEM AS DETAILED IN ACUTE INGESTION
- CHRONIC EXPOSURE- REPEATED OR PROLONGED CONTACT WITH THE LIQUID MAY CAUSE DEFATTING OF THE SKIN RESULTING IN ERYTHEMA, SCALING, AND ECZEMATOID DERMATITIS. CHRONIC ABSORPTION MAY RESULT IN METABOLIC ACIDOSIS AND EFFECTS AS DETAILED IN ACUTE INGESTION

FIRST AID- REMOVE CONTAMINATED CLOTHING AND SHOES IMMEDIATELY. WASH AFFECTED AREA WITH SOAP OR MILD DETERGENT AND LARGE AMOUNTS OF WATER UNTIL NO EVIDENCE OF CHEMICAL REMAINS (APPROXIMATELY 15-20 MINUTES). GET MEDICAL ATTENTION IMMEDIATELY

EYE CONTACT:
METHYL ALCOHOL (METHANOL)
IRRITANT.
- ACUTE EXPOSURE- VAPORS MAY CAUSE IRRITATION. HIGH CONCENTRATIONS HAVE BEEN REPORTED TO CAUSE VIOLENT INFLAMMATION OF THE CONJUNCTIVA AND EPITHELIAL DEFECTS ON THE CORNEA. MILD IRRITATION MAY OCCUR WITH DILUTE SOLUTIONS; THE UNDILUTED LIQUID HAS PRODUCED MODERATE CORNEAL OPACITY AND CONJUNCTIVAL REDNESS IN RABBITS. APPLICATION OF A DROP OF METHANOL IN RABBIT EYES CAUSED A MILD REVERSIBLE REACTION, GRADED 3 ON A SCALE OF 1-10 AFTER 24 HOURS.
- CHRONIC EXPOSURE- REPEATED OR PROLONGED CONTACT MAY CAUSE CONJUNCTIVITIS

FIRST AID- WASH EYES IMMEDIATELY WITH LARGE AMOUNTS OF WATER OR NORMAL SALINE, OCCASIONALLY LIFTING UPPER AND LOWER LIDS, UNTIL NO EVIDENCE OF CHEMICAL REMAINS (APPROXIMATELY 15-20 MINUTES). GET MEDICAL ATTENTION IMMEDIATELY

INGESTION:
METHYL ALCOHOL (METHANOL).
NARCOTIC/NEUROTOXIN.
- ACUTE EXPOSURE- MAY CAUSE MILD AND TRANSIENT INEBRIATION AND SUBSEQUENT DROWSINESS FOLLOWED BY AN ASYMPTOMATIC PERIOD LASTING 8-48 HOURS. FOLLOWING THE DELAY, COUGHING, DYSPNEA, HEADACHE, DULLNESS, WEAKNESS, VERTIGO OR DIZZINESS, NAUSEA, VOMITING, OCCASIONAL DIARRHEA, ANOREXIA, VIOLENT PAIN IN THE BACK, ABDOMEN, AND EXTREMITIES, RESTLESSNESS, APATHY OR DELIRIUM, AND RARELY, EXCITEMENT AND MANIA MAY OCCUR. RAPID, SHALLOW RESPIRATION DUE TO METABOLIC ACIDOSIS, COLD AND CLAMMY SKIN, HYPOTENSION, CYANOSIS, OPISTHOTONOS, CONVULSIONS, MILD TACHYCARDIA, CARDIAC DEPRESSION, PERIPHERAL NEURITIS, CEREBRAL AND PULMONARY EDEMA, UNCONSCIOUSNESS, AND COMA ARE POSSIBLE. EFFECTS ON THE EYE MAY INCLUDE OPTIC NEURITIS, BLURRED OR DIMMED VISION, DILATED, UNRESPONSIVE PUPILS, PTOSIS, EYE PAIN, CONCENTRIC CONSTRICTION OF VISUAL FIELDS, DIPLOPIA, CHANGE IN COLOR PERCEPTION, PHOTOPHOBIA, AND OPTIC NERVE ATROPHY. PARTIAL BLINDNESS OR POSSIBLY DELAYED TRANSIENT OR PERMANENT BLINDNESS MAY OCCUR. BILATERAL SENSORINEURAL DEAFNESS HAS BEEN REPORTED IN A SINGLE CASE. LIVER, KIDNEY, HEART, STOMACH, INTESTINAL AND PANCREATIC DAMAGE MAY ALSO OCCUR. DEATH

MAY BE DUE TO RESPIRATORY FAILURE OR RARELY FROM CIRCULATORY COLLAPSE. AS LITTLE AS 15 ML HAS CAUSED BLINDNESS; THE USUAL FATAL DOSE IS 60-240 ML. PROLONGED ASTHENIA AND IRREVERSIBLE EFFECTS ON THE NERVOUS SYSTEM INCLUDING DIFFICULTY IN SPEECH, MOTOR DYSFUNCTION WITH RIGIDITY, SPASTICITY, AND HYPOKINESIS HAVE BEEN REPORTED.
- CHRONIC EXPOSURE- REPEATED INGESTION MAY CAUSE VISUAL IMPAIRMENT AND BLINDNESS AND OTHER SYSTEMIC EFFECTS AS DETAILED IN ACUTE INGESTION. REPRODUCTIVE EFFECTS HAVE BEEN REPORTED IN ANIMALS

FIRST AID- IF INGESTION OF METHANOL IS DISCOVERED WITHIN 2 HOURS, GIVE SYRUP OF IPECAC. LAVAGE THOROUGHLY WITH 2-4 L OF TAP WATER WITH SODIUM BICARBONATE (20 G/L) ADDED. GET MEDICAL ATTENTION IMMEDIATELY. LAVAGE SHOULD BE PERFORMED BY QUALIFIED MEDICAL PERSONNEL (DREISBACH, HANDBOOK OF POISONING, 12TH ED.)

ANTIDOTE:
THE FOLLOWING ANTIDOTE(S) HAVE BEEN RECOMMENDED. HOWEVER, THE DECISION AS TO WHETHER THE SEVERITY OF POISONING REQUIRES ADMINISTRATION OF ANY ANTIDOTE AND ACTUAL DOSE REQUIRED SHOULD BE MADE BY QUALIFIED MEDICAL PERSONNEL.

METHANOL POISONING:
GIVE ETHANOL, 50% (100 PROOF), 1.5 ML/KG ORALLY INITIALLY, DILUTED TO NOT MORE THAN 5% SOLUTION, FOLLOWED BY 0.5-1.0 ML/KG EVERY 2 HOURS ORALLY OR INTRAVENOUSLY FOR 4 DAYS IN ORDER TO REDUCE METABOLISM OF METHANOL AND TO ALLOW TIME FOR ITS EXCRETION. BLOOD ETHANOL LEVEL SHOULD BE IN THE RANGE OF 1-1.5 MG/ML (DREISBACH, HANDBOOK OF POISONING, 12TH ED.). ANTIDOTE SHOULD BE ADMINISTERED BY QUALIFIED MEDICAL PERSONNEL

ORAL OR INTRAVENOUS ADMINISTRATION OF 4-METHYLPYRAZOLE INHIBITS ALCOHOL DEHYDROGENASE AND HAS BEEN USED EFFECTIVELY AS AN ANTIDOTE FOR METHANOL OR ETHYLENE GLYCOL POISONING (ELLENHORN AND BARCELOUX, MEDICAL TOXICOLOGY).

REACTIVITY

REACTIVITY:
STABLE UNDER NORMAL TEMPERATURES AND PRESSURES

INCOMPATIBILITIES:
METHYL ALCOHOL (METHANOL)
- ACETYL BROMIDE: VIOLENT REACTION WITH FORMATION OF HYDROGEN BROMIDE.
- ALKYLALUMINUM SOLUTIONS: VIOLENT REACTION
- ALUMINUM: CORRODES
- BARIUM PERCHLORATE: DISTILLATION YIELDS HIGHLY EXPLOSIVE ALKYL PERCHLORATE
- BERYLLIUM HYDRIDE: VIOLENT REACTION, EVEN AT -196 C.
- BROMINE: VIGOROUSLY EXOTHERMIC REACTION
- CALCIUM CARBIDE: VIOLENT REACTION.
- CHLORINE: POSSIBLE IGNITION AND EXPLOSION HAZARD
- CHLOROFORM AND SODIUM HYDROXIDE: EXPLOSIVE REACTION
- CHROMIUM TRIOXIDE (CHROMIC ANHYDRIDE): POSSIBLE IGNITION
- CYANURIC CHLORIDE: VIOLENT REACTION.
- DICHLOROMETHANE: POSSIBLE IGNITION AND EXPLOSION.
- DIETHYL ZINC: POSSIBLE IGNITION AND EXPLOSION.
- HYDROGEN PEROXIDE + WATER: EXPLOSION HAZARD.
- IODINE + ETHANOL + MERCURIC OXIDE: EXPLOSION HAZARD
- LEAD: CORRODES.
- LEAD PERCHLORATE: EXPLOSION HAZARD
- MAGNESIUM: VIOLENT REACTION.
- MAGNESIUM (POWDERED): MIXTURES ARE CAPABLE OF DETONATION
- METALS: INCOMPATIBLE.
- NICKEL: POSSIBLE IGNITION IN THE PRESENCE OF NICKEL CATALYST
- NITRIC ACID (CONCENTRATED): MIXTURES OF GREATER THAN 25% ACID MAY DECOMPOSE VIOLENTLY.
- OXIDIZERS (STRONG): FIRE AND EXPLOSION HAZARD
- PERCHLORIC ACID: EXPLOSION HAZARD.
- PHOSPHOROUS TRIOXIDE: POSSIBLE VIOLENT REACTION AND IGNITION
- PLASTICS, RUBBER, COATINGS: MAY BE ATTACKED.
- POTASSIUM: POSSIBLE DANGEROUS REACTION
- POTASSIUM HYDROXIDE + CHLOROFORM: EXOTHERMIC REACTION
- POTASSIUM TERT-BUTOXIDE: FIRE AND EXPLOSION HAZARD
- SODIUM + CHLOROFORM: POSSIBLE EXPLOSION
- SODIUM HYPOCHLORITE: EXPLOSION HAZARD
- SODIUM METHOXIDE + CHLOROFORM: VIOLENT REACTION.
- SULFURIC ACID: FIRE AND EXPLOSION HAZARD
- ZINC: EXPLOSION HAZARD

DECOMPOSITION:
THERMAL DECOMPOSITION PRODUCTS MAY INCLUDE TOXIC OXIDES OF CARBON

POLYMERIZATION:
HAZARDOUS POLYMERIZATION HAS NOT BEEN REPORTED TO OCCUR UNDER NORMAL TEMPERATURES AND PRESSURES.

STORAGE AND DISPOSAL

OBSERVE ALL FEDERAL, STATE AND LOCAL REGULATIONS WHEN STORING OR DISPOSING OF THIS SUBSTANCE. FOR ASSISTANCE, CONTACT THE DISTRICT DIRECTOR OF THE ENVIRONMENTAL PROTECTION AGENCY

STORAGE

STORE IN ACCORDANCE WITH 29 CFR 1910.106.

STORE AWAY FROM INCOMPATIBLE SUBSTANCES

DISPOSAL

DISPOSAL MUST BE IN ACCORDANCE WITH STANDARDS APPLICABLE TO GENERATORS OF HAZARDOUS WASTE, 40 CFR 262. EPA HAZARDOUS WASTE NUMBER U154.

****

CONDITIONS TO AVOID

AVOID CONTACT WITH HEAT, SPARKS, FLAMES OR OTHER IGNITION SOURCES. VAPORS MAY BE EXPLOSIVE. MATERIAL IS POISONOUS; AVOID INHALATION OF VAPORS OR CONTACT WITH SKIN. DO NOT ALLOW MATERIAL TO CONTAMINATE WATER SOURCES.

****

SPILL AND LEAK PROCEDURES

SOIL SPILL:
DIG HOLDING AREA SUCH AS LAGOON, POND OR PIT FOR CONTAINMENT

DIKE FLOW OF SPILLED MATERIAL USING SOIL OR SANDBAGS OR FOAMED BARRIERS SUCH AS POLYURETHANE OR CONCRETE

AIR SPILL:
APPLY WATER SPRAY TO KNOCK DOWN VAPORS

WATER SPILL:
ALLOW SPILLED MATERIAL TO AERATE

LIMIT SPILL MOTION AND DISPERSION WITH NATURAL BARRIERS OR OIL SPILL CONTROL BOOMS.

USE SUCTION HOSES TO REMOVE TRAPPED SPILL MATERIAL

OCCUPATIONAL SPILL:
SHUT OFF IGNITION SOURCES. DO NOT TOUCH SPILLED MATERIAL. STOP LEAK IF YOU CAN DO IT WITHOUT RISK. USE WATER SPRAY TO REDUCE VAPORS. FOR SMALL SPILLS, TAKE UP WITH SAND OR OTHER ABSORBENT MATERIAL AND PLACE INTO CONTAINERS FOR LATER DISPOSAL. FOR LARGER SPILLS, DIKE FAR AHEAD OF SPILL FOR LATER DISPOSAL. NO SMOKING, FLAMES OR FLARES IN HAZARD AREA! KEEP UNNECESSARY PEOPLE AWAY; ISOLATE HAZARD AREA AND DENY ENTRY

REPORTABLE QUANTITY (RQ): 5000 POUNDS
THE SUPERFUND AMENDMENTS AND REAUTHORIZATION ACT (SARA) SECTION 304 REQUIRES THAT A RELEASE EQUAL TO OR GREATER THAN THE REPORTABLE QUANTITY FOR THIS SUBSTANCE BE IMMEDIATELY REPORTED TO THE LOCAL EMERGENCY PLANNING COMMITTEE AND THE STATE EMERGENCY RESPONSE COMMISSION (40 CFR 355.40). IF THE RELEASE OF THIS SUBSTANCE IS REPORTABLE UNDER CERCLA SECTION 103, THE NATIONAL RESPONSE CENTER MUST BE NOTIFIED IMMEDIATELY AT (800) 424-8802 OR (202) 426-2675 IN THE METROPOLITAN WASHINGTON, D.C. AREA (40 CFR 302.6).

PROTECTIVE EQUIPMENT

VENTILATION:
PROVIDE GENERAL DILUTION VENTILATION TO MEET PUBLISHED EXPOSURE LIMITS. VENTILATION EQUIPMENT MUST BE EXPLOSION-PROOF.

RESPIRATOR:
THE FOLLOWING RESPIRATORS AND MAXIMUM USE CONCENTRATIONS ARE RECOMMENDATIONS BY THE U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES, NIOSH POCKET GUIDE TO CHEMICAL HAZARDS; NIOSH CRITERIA DOCUMENTS OR BY THE U.S. DEPARTMENT OF LABOR, 29 CFR 1910 SUBPART Z.
THE SPECIFIC RESPIRATOR SELECTED MUST BE BASED ON CONTAMINATION LEVELS FOUND IN THE WORK PLACE, MUST NOT EXCEED THE WORKING LIMITS OF THE RESPIRATOR AND BE JOINTLY APPROVED BY THE NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH AND THE MINE SAFETY AND HEALTH ADMINISTRATION (NIOSH-MSHA)

METHYL ALCOHOL (METHANOL):

2000 PPM- ANY SUPPLIED-AIR RESPIRATOR.
ANY SELF-CONTAINED BREATHING APPARATUS

5000 PPM- ANY SUPPLIED-AIR RESPIRATOR OPERATED IN A CONTINUOUS-FLOW MODE

10,000 PPM- ANY SELF-CONTAINED BREATHING APPARATUS WITH A FULL FACEPIECE
ANY SUPPLIED-AIR RESPIRATOR WITH A FULL FACEPIECE
ANY SUPPLIED-AIR RESPIRATOR THAT HAS A TIGHT-FITTING FACEPIECE AND IS OPERATED IN A CONTINUOUS-FLOW MODE.

25,000 PPM- ANY SUPPLIED-AIR RESPIRATOR WITH A FULL FACEPIECE AND OPERATED IN A PRESSURE-DEMAND OR OTHER POSITIVE PRESSURE MODE

ESCAPE- ANY APPROPRIATE ESCAPE-TYPE, SELF-CONTAINED BREATHING APPARATUS.

FOR FIREFIGHTING AND OTHER IMMEDIATELY DANGEROUS TO LIFE OR HEALTH CONDITIONS:

ANY SELF-CONTAINED BREATHING APPARATUS THAT HAS A FULL FACEPIECE AND IS OPERATED IN A PRESSURE-DEMAND OR OTHER POSITIVE-PRESSURE MODE

ANY SUPPLIED-AIR RESPIRATOR THAT HAS A FULL FACEPIECE AND IS OPERATED IN A PRESSURE-DEMAND OR OTHER POSITIVE-PRESSURE MODE IN COMBINATION WITH AN AUXILIARY SELF-CONTAINED BREATHING APPARATUS OPERATED IN PRESSURE-DEMAND OR OTHER POSITIVE-PRESSURE MODE

CLOTHING:
EMPLOYEE MUST WEAR APPROPRIATE PROTECTIVE (IMPERVIOUS) CLOTHING AND EQUIPMENT TO PREVENT REPEATED OR PROLONGED SKIN CONTACT WITH THIS SUBSTANCE.

GLOVES:
EMPLOYEE MUST WEAR APPROPRIATE PROTECTIVE GLOVES TO PREVENT CONTACT WITH THIS SUBSTANCE.

EYE PROTECTION:
EMPLOYEE MUST WEAR SPLASH-PROOF OR DUST-RESISTANT SAFETY GOGGLES TO PREVENT EYE CONTACT WITH THIS SUBSTANCE.

EMERGENCY EYE WASH: WHERE THERE IS ANY POSSIBILITY THAT AN EMPLOYEE'S EYES MAY BE EXPOSED TO THIS SUBSTANCE, THE EMPLOYER SHOULD PROVIDE AN EYE WASH FOUNTAIN WITHIN THE IMMEDIATE WORK AREA FOR EMERGENCY USE

AUTHORIZED - FISHER SCIENTIFIC, INC.
CREATION DATE: 09/25/84 REVISION DATE: 10/12/92

-ADDITIONAL INFORMATION-
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HEALTH AND SAFETY PLAN ATTACHMENT D

STANDARD SAFE WORK PRACTICES

STANDARD SAFE WORK PRACTICES

1) Eating, drinking, chewing tobacco, smoking and carrying matches or lighters is prohibited in a contaminated or potentially contaminated area or where the possibility for the transfer of contamination exists.

2) Avoid contact with potentially contaminated substances. Do not walk through puddles, pools, mud, etc. Avoid, whenever possible, kneeling on the ground, leaning or sitting on equipment or ground. Do not place monitoring equipment on potentially contaminated surfaces (i.e., ground, etc).

3) All field crew members should make use of their senses to alert them to potentially dangerous situations in which they should not become involved; i.e., presence of strong and irritating or nauseating odors.

4) Prevent, to the extent possible, spillages. In the event that a spillage occurs, contain liquid if possible.

5) Field crew members shall be familiar with the physical characteristics of investigations, including:

- Wind direction
- Accessibility to associates, equipment, vehicles
- Communication
- Hot zone (areas of known or suspected contamination)
- Site access
- Nearest water sources

6) All wastes generated during activities on-site should be disposed of as directed by the project manager or his on-site representative.

7) Protective equipment as specified in the section on personnel protection will be utilized by workers during the initial site reconnaissance, and other activities.

APPENDIX C

SAMPLING INFORMATION / FORMS

TABLE C.1

LEHIGH INDUSTRIAL PARK SITE, Site No. 9-15-145

SUMMARY OF MONITORING WELL DATA

Monitoring Well No.2

Date Installed: 7/14/92 - Date modified:5/20/97 (5 foot extension added)

Driller: SJB Drilling - Jim Lamm - Modification by Maxim Technologies

Consultant - Parson's - Engineering Science (Project No. SY 279.02.02)

Northing: 9277.0764 Easting: 4706.5529

Top of Casing: 597.81 ft. Ground Surface Elevation: 596.18

Top of Rock: 16.5 ft. Bottom of Boring: 16.5 ft. Bottom of Well: 15.5 ft.

Modified: T.O.R. - 21.5 ft., Botton of Boring 21.5 ft., Bottom of well 20.5 ft.

Well materials: 2" PVC Riser, 10 slot screen (9 Ft.)

Monitoring Well No.4

Date Installed: 7/15/92

Driller: SJB Drilling - Jim Lamm

Consultant - Parson's -EngineeringScience (Project No. SY 279.02.02)

Northing: 9573.4944 Easting: 4933.7387

Top of Casing: 592.61 ft. Ground Surface Elevation: 590.62

Top of Rock: 15.5 ft. Bottom of Boring: 15.5 ft. Bottom of Well: 15.0 ft.

Well materials: 2" PVC[2] Riser, 10 slot screen (9 Ft.)

NOTES: 1. Depths measured from ground surface

2. PVC = polyvinyl chloride

TABLE C.2

SAMPLE CONTAINER, PRESERVATION AND HOLDING TIMES

NYSDEC-QAQC

PARAMETER	MEDIUM	CONTAINER	VOLUME REQD.	PRESERVATION	HOLDING TIMES
Volatile Organics	Soil/sediment	Glass, Teflon® lined lid or septa	20 g	Cool, 4° C	7 Days
TCL VOC's	Groundwater/Liquid	Glass, Teflon® lined septa	(2) 40 ml	Cool, 4° C	7 Days
Extractable Organics					
TCL, SVOCs, TCL Pesticides/PCBs	Soil/Sediment	Glass, Teflon® lined lid	100 g	Cool, 4° C	5 Days Extract 40 days Analyze
	Groundwater/Liquid	Glass, Teflon® lined cap	(2) 1-L	Cool, 4° C	5 Days Extract 40 days Analyze
Inorganics					
TCL Inorganics	Soil/Sediment	Glass	2 g	Cool, 4° C	6 months
Mercury		Glass	1 g	Cool, 4° C	26 days
Cyanide		Glass	10 g	Cool, 4° C	12 days
Hex. Chromium		Glass	2 g	Cool, 4° C	24 hrs.
TCL Inorganics	Groundwater/Liquid	Glass or Polyethylene	450 ml	HNO_3 to pH<2, 4° C	6 months
Mercury		Glass or Polyethylene	200 ml	HNO_3 to pH<2, 4° C	26 days
Cyanide		Glass or Polyethylene	1 L	NaOH to pH>12, 4° C	12 days
Hex. Chromium		Glass or Polyethylene	500 ml	Cool, 4° C	24 hrs.
Ignitability	Soil/Sediment	Glass, Teflon® lined lid	25 g	Cool, 4° C	28 Days
Reactivity	Soil/Sediment	Glass, Teflon® lined lid	40 g	Cool, 4° C	28 Days
Corrosivity	Soil/Sediment	Glass, Teflon® lined lid	30 g	Cool, 4° C	28 Days

Note: All holding times are from verified time of sample receipt at the laboratory.

TABLE C.3
GROUNDWATER SAMPLING EQUIPMENT

SAMPLING EQUIPMENT

- Photo ionization detector
- Personal safety equipment (hard hats, etc.)
- Sampling and analysis program
- Appropriate number (including spares) of sample bottles
- Water-level indicator (electric drop-line)
- Polyethylene ground cloth
- Aluminum Foil
- Distilled water
- Alconox detergent
- Tap water source
- Disposable surgical gloves
- Disposable towels
- pH meter
- Conductivity meter
- Buckets (small: 5 gallon; large: 25 to 30 gallon)
- Teflon well bailer
- Nylon rope (individual lengths for each well)

SHIPPING AND PACKAGING EQUIPMENT

- Shipping labels
- Sufficient ice chests to hold all sample bottles, packing material and ice

DOCUMENTATION EQUIPMENT

- Well Sampling Record
- Chain-of-Custody Forms
- Waterproof Pens

TABLE C. 4

WELL INSPECTION CHECKLIST

Date:_____

Time:_____

Inspector:_____

Is well accessible ?	Y / N
Is well upright and straight?	Y / N
Does well need painting?	Y / N
Is well secured?	Y / N
Is lock operational?	Y / N
Is seal intact?	Y / N
Does well riser have a cap?	Y / N
Can a bailer be lowered to bottom?	Y / N

FIGURE C.1
GROUNDWATER SAMPLING DATA SHEET

NEW YORK STATE DEPARTMENT OF ENVIRONMENTAL CONSERVATION
GROUND WATER FIELD SAMPLE DATA RECORD

DATE:
PROJECT NAME: LEHIGH INDUSTRIAL PARK
SITE NUMBER: 915145
STATION LOCATION: MONITORING WELL NO. 2 / MONITORING WELL NO. 4
PHOTOGRAPHS: Y / N

FIELD DATA TIME: START_____ AIR TEMP: _____
END_____ WEATHER:_____

WATER DEPTH_____ ☐ TOP WELL WELL DEPTH_____ WELL MATERIAL_____
☐ TOP CASING WELL DIAMETER_____

WELL STICKUP:_____ WELL DIAMETER:_____

VOA LEVELS:_____AMBIENT _____WELL HEAD SPACE

SAMPLE PURGE DATA:

@ INITIAL_____	@_____GAL.	@_____GAL.	@_____GAL
TEMP._____°F	TEMP._____°F	TEMP._____°F	TEMP._____°F
SP.COND._____	SP.COND._____	SP.COND._____	SP.COND._____
pH_____	pH_____	pH_____	pH_____
TURBIDITY_____n.t.u.	TURBIDITY_____n.t.u.	TURBIDITY_____n.t.u	TURB._____n.t.u.
@ INITIAL_____	@_____GAL.	@_____GAL.	@_____GAL
TEMP._____°F	TEMP._____°F	TEMP._____°F	TEMP._____°F
SP.COND._____	SP.COND._____	SP.COND._____	SP.COND._____
pH_____	pH_____	pH_____	pH_____
TURBIDITY_____n.t.u.	TURBIDITY_____n.t.u.	TURBIDITY_____n.t.u	TURB._____n.t.u.

_____

_____

_____Sampler Signature_____

FIGURE C.2
CHAIN OF CUSTODY FORM
NYSDEC -QAQC

Project No.	Site Name:	No. of containers							Remarks:
Sampler(s) Signature									

Station No,	Date	Time	Comp.	Grab	Station location							

Relinquished By:	Date/Time	Received By	Relinquished By:	Date/Time	Received By
Relinquished By:	Date/Time	Received By	Relinquished By:	Date/Time	
Received By Laboratory:			Remarks		

***Triplicate Copies of Form Required**:

FIGURE C.3
CONTRACT LAB SAMPLE INFORMATION SHEET

74-15-1 (9/97)—q

RETURN THIS SHEET TO ROOM 392, 50 WOLF ROAD, ALBANY, NEW YORK 12233-3502

NEW YORK STATE DEPARTMENT OF ENVIRONMENTAL CONSERVATION

CONTRACT LAB SAMPLE INFORMATION SHEET

Print Legibly — Part 1

CAUTION (check if applicable)

☐ Lab personnel are expected to use caution when handling DEC samples, however, please use special caution when handling this sample since it is believed to contain significant concentrations of hazardous and/or toxic materials(s)

CHECK THE BOX PRECEDING THE REQUESTED ANALYSIS

PRIORITY POLLUTANTS (Water Part 136)—SPDES

☐ 2. 13PP Metals	☐ 3. Volatiles—(USEPA 624 GC/MS)	☐ 6. Pesticides/PCBs (USEPA 608-GC)
☐ 4. Acids Base/Neutrals (USEPA 624 GC/MS)	☐ 5. Cyanide	☐ 9. BOD
☐ 7. Halogenated Volatiles (USEPA 601 GC)	☐ 8. Aromatic Volatiles USEPA 602 GC)	☐ 12. TSS
☐ 10. pH	☐ 11. COD	☐ 15. Ammonia
☐ 13. Settleable Solids	☐ 14. TKN	☐ 18. Reactive Phosphorus
☐ 16. Nitrate/Nitrite	☐ 17. Total Phosphorus	☐ 21. Total Phenols
☐ 19. Oil/Grease)	☐ 20. TOC	☐ 60. PCBs congener method (ASP 91-11)
☐ 22. Other _____	☐ 59. PCBs at 0.065 ug/l	☐ 64. Total Solids
	☐ 62. CBOD	☐ 65. Volatiles (USEPA 524.2 GC/MS)

CONTRACT LABORATORY PROTOCOLS

☐ 23 (ALL)—Water—Includes 24-28	☐ 29. (ALL)—Soil/Sediments—Includes 30-34
☐ 24 Base/Neutral/Acid (B/N/A)—Water—GC/MS (ASP #95-2)	☐ 30. (B/N/A)—Soil/Sediments—GC/MS (ASP #95-2)
☐ 25 Volatile Organic Analysis VOA—Water—GC/MS (ASP #95-1)	☐ 31. VOA—Soil/Sediments—GC/MS (ASP #95-1)
☐ 26 Pesticides/PCBs—Water—GC/MS (ASP #95-3)	☐ 32. Pesticides/PCBs—Soil/Sediments—GC (ASP #95-3)
☐ 27 Metals—23 in Water	☐ 33. Metals—23 in Soil/Sediments)
☐ 28 Cyanide—Water	☐ 34. Cyanide—Soil/Sediments)
☐ 66 Dioxin-Water (ASP #91-7)	☐ 67. Dioxin-Soil/Sediments (ASP #91-7)
☐ 35 Other _____	

HAZARDOUS WASTES/RCRA ANALYSIS SW-846

☐ 36. EP Toxicity	☐ 37. EP Toxicity (Metals Only)	☐ 38. Ignitability
☐ 39. Corrosivity	☐ 40. VOA—(USEPA 8260 GC/MS)	☐ 41. BNA—(USEPA 8270 GC/MS)
☐ 42. Pesticides/PCBs (USEPA 8081)	☐ 43. TCLP	☐ 44. TCLP (Metals Only)
☐ 45. Reactivity	☐ 46. Dioxin (USEPA 8280)	☐ 47. Appendix IX
☐ 48. Other _____	☐ 63 Percent Solids	☐ 68. Metals—17 Hazardous

MUNICIPAL SLUDGE

☐ 56. RS-01 ☐ 57. RS-02 ☐ 58. Other _____

COLLECTED BY:	TELEPHONE NUMBER:	REGION NO.:

CONTRACT LABORATORY:	COUNTY:	SAMPLING DATE:	MILITARY TIME:

SAMPLE MATRIX:

☐ Air ☐ Soil/Sediment ☐Groundwater ☐ Surface Water ☐ Wastewater ☐ Other _____

CASE NO.	SDG NO.	SAMPLE NO.	CHECK FOR MS/MD	TYPE OF SAMPLE
| | | | |	| | | | |	| | | | |	☐ This sample	☐ Grab ☐ Composite ☐ Term _____ hours

☐ Check if there will be more samples with this SDG sent in this calendar week.

SAMPLING POINT:

Report via Category B, unless checked ☐

Check if field duplicate ☐ Outfall Number

Check if sampling is part of inspection ☐

FLOW: _____ GPD _____ MGD

SPDES NUMBER/REGISTRY NUMBER

\| \| \| \| \| \| \|

APPENDIX D
INSPECTION AND MAINTENANCE REPORT FORMS

TABLE D.1
SITE INSPECTION CHECKLIST

INSPECTOR:_____	DATE: _____
WEATHER:_____	TEMP._____

AREA	ITEM	ACTION	COMMENTS
COVER SYSTEM	SEEPS	DELINEATE, SAMPLE EVALUATE	
	SUBSIDENCE/PONDING	DELINEATE, FILL, REVEGETATE	
	EROSION/GULLIES	DETERMINE CAUSE, GRADE, AND VEGETATE	
	SLOPE STABILITY	CHECK FOR EROSION, SLIPPAGE, SLOPE FAILURE	
	VEGETATION	MOW ANNUALLY (FALL)	
	VECTORS	CHECK FOR BURROWS, BACKFILL, PERSISTENT PROBLEM REMOVE ENVIRONMENTALLY	
STORM WATER SYSTEM	DITCHES, SWALES	CHECK FOR POOLING, EROSION EXCESSIVE VEGETATION AND WEAK VEGETATION.	
	COVER SYSTEM	CHECK INTEGRITY AND PONDING	
	INFILTRATION BASIN	CHECK FOR DEBRIS AND BOTTOM INTEGRITY, NOTE WATER LEVEL.	
GROUNDWATER MONITORING SYSTEM	MONITORING WELLS	CHECK CONDITION OF CAPS, LOCKS, SURFACE SEALS, AND MARKINGS. LUBRICATE LOCKS	
SECURITY/ACCESS	INFILTRATION BASIN FENCING	CHECK FOR HOLES, GAPS, ILLEGAL DISPOSAL OR BREECHES	
FACILITY ACCESS	GATES	CHECK OPERATION AND LOCKS, LUBRICATE LOCKS	

FIGURE D.1

ANNUAL OPERATION AND MAINTENANCE REVIEW FORM

NEW YORK STATE DEPARTMENT OF ENVIRONMENTAL CONSERVATION

DIVISION OF ENVIRONMENTAL REMEDIATION

INACTIVE HAZARDOUS WASTE SITE OPERATIONS AND MAINTENANCE REVIEW REPORT

FORM DATE 96.10.01

SITE NAME: **CLASS:** **NUMBER:**

O&M FUNDING SOURCE: ☐ STATE SUPERFUND ☐ FEDERAL SUPERFUND ☐ MUNICIPAL ☐ RESPONSIBLE PARTY

O&M INFORMATION: O&M START: END: ANNUAL COST: $☐ ESTIMATED

INTERIM REMEDIAL MEASURES/OPERABLE UNITS IN O&M PHASE:

☐ DRUM REMOVAL ☐ SOIL REMOVAL ☐ TANK REMOVAL

☐ CAP/COVER ☐ CONTAINMENT STRUCTURE ☐ FENCE/SECURITY

☐ GROUNDWATER RECOVERY/TREATMENT ☐ LEACHATE COLLECTION/TREATMENT ☐ VAPOR EXTRACTION/TREATMENT

☐ AIR SPARGING/STRIPPER SYSTEM ☐ TREATMENT/FILTRATION PLANT/SYSTEM ☐ POTABLE WATER SUPPLY/SYSTEM

☐ OTHER:

INSTITUTIONAL CONTROLS: ☐ DEED RESTRICTION ☐ DISCHARGE PERMIT ☐ DEPARTMENT OF HEALTH SAMPLING

☐ OTHER:

O&M REVIEW INFORMATION:

REPORTS:

INSPECTION:

SAMPLING:

OTHER:

CONCLUSIONS:

REMEDY EFFECTIVE? ☐ YES ☐ NO:

ROD COMPLIANCE? ☐ YES ☐ NO:

CONSENT ORDER COMPLIANCE? ☐ YES ☐ NO:

OTHER:

RECOMMENDATIONS:

ROD/CONSENT ORDER MODIFICATIONS? ☐ NO ☐ YES (PER ABOVE) RECLASSIFY THE SITE? ☐ NO ☐ YES → CLASS:

COMMENTS:

PROJECT MANAGER:	**REVIEWER:**
SIGNATURE DATE	SIGNATURE DATE
NAME REGION OR BUREAU TELEPHONE	NAME REGION OR BUREAU TELEPHONE

FIGURE D.2
INSTRUCTIONS TO COMPLETE ANNUAL O&M REVIEW REPORT FORM

GENERAL

IF ADDITIONAL SPACE IS REQUIRED TO EXPLAIN ANY PARTICULAR SECTION, PLEASE CONTINUE THE EXPLANATION IN THE COMMENT AREA WITH A PREFIX REFERENCING THAT SECTION, OR YOU MAY REFER AND ATTACH A SEPARATE SHEET. PLEASE COMPLETE ALL SECTIONS OF THIS REPORT AND SEND ONE SIGNED COPY (WITH ANY ATTACHMENTS) TO THE BUREAU OF HAZARDOUS SITE CONTROL.

SITE INFORMATION

ENTER THE **SITE NAME, CLASS** AND **NUMBER** AS LISTED IN THE *REGISTRY OF INACTIVE HAZARDOUS WASTE DISPOSAL SITES IN NEW YORK STATE*.

O&M FUNDING SOURCE

CHECK THE APPROPRIATE FUNDING SOURCE BOX.

O&M INFORMATION

O&M START: ENTER THE **EARLIEST O&M START DATE** GIVEN IN THE *QUARTERLY STATUS REPORT OF INACTIVE HAZARDOUS WASTE DISPOSAL SITES*. THIS DATE REPRESENTS THE EARLIEST DATE THAT AN IRM OR OPERABLE UNIT AT THE SITE ENTERED INTO ITS O&M PHASE.

O&M END: **IF ALL O&M REQUIREMENTS AND ACTIVITIES HAVE BEEN SATISFIED** AT THIS SITE, ENTER THE **LATEST O&M END DATE** GIVEN IN THE *QUARTERLY STATUS REPORT OF INACTIVE HAZARDOUS WASTE DISPOSAL SITES*. THE O&M END DATE FOR THE SITE REPRESENTS THE CLOSING DATE OF ALL O&M REQUIREMENTS/ACTIVITIES AT THE SITE AND REPRESENTS THE DATE THAT THE SITE IS CLASSIFIED OUT OF O&M INVOLVEMENT.

ANNUAL COST: ENTER THE **TOTAL COST** (ROUNDED TO THOUSANDS) TO OPERATE, MAINTAIN AND MONITOR THE SITE UNDER O&M. INCLUDE COSTS SUCH AS UTILITIES, PERSONNEL, SAMPLE ANALYSIS, TRAVEL, ET CETERA. CHECK THE BOX IF ESTIMATED.

INTERIM REMEDIAL MEASURES/OPERABLE UNITS IN O&M PHASE

IDENTIFY ALL IRM/OPERABLE UNIT CATEGORIES THAT ACTIVELY INVOLVE SOME PHASE OF O&M ACTIVITY AT THE SITE. IF A PARTICULAR CATEGORY IS NOT LISTED, ENTER IT UNDER "OTHER". REFER TO THE *QUARTERLY STATUS REPORT OF INACTIVE HAZARDOUS WASTE DISPOSAL SITES* AS NEEDED.

INSTITUTIONAL CONTROLS

IDENTIFY ALL INSTITUTIONAL CONTROL CATEGORIES THAT MANAGE OR EXERCISE CONTROL OVER ANY ASPECT OF O&M ACTIVITY AT THE SITE. IF A PARTICULAR CATEGORY IS NOT LISTED, ENTER IT UNDER "OTHER". IF THERE ARE NO INSTITUTIONAL CONTROLS, WRITE N/A.

O&M REVIEW INFORMATION

REPORTS: ENTER THE **TITLE, DATE** AND **AUTHOR** OF ANY DISCHARGE MONITORING REPORTS, GROUNDWATER MONITORING REPORTS, TREATABILITY STUDY REPORTS, PLANT CODE EVALUATION REPORTS, ET CETERA USED IN CONNECTION WITH THIS REVIEW.

INSPECTION: FOR ANY INSPECTION RELATED REPORTS OR INFORMATION USED IN CONJUNCTION WITH THIS REVIEW, IDENTIFY THE **AGENCY OR COMPANY** THAT DID THE INSPECTION BY NAME AND ENTER THE CORRESPONDING **INSPECTION** DATE.

SAMPLING: FOR ANY SAMPLING RELATED REPORTS USED IN CONNECTION WITH THIS REVIEW, ENTER THE **TITLE**, DATE AND **AUTHOR** AS APPLICABLE. FOR ANY OTHER SAMPLING RELATED INFORMATION, IDENTIFY THE **TYPE OF SAMPLING,** THE **AGENCY OR COMPANY** THAT DID THE SAMPLING BY NAME, AND THE CORRESPONDING **SAMPLING** DATE.

OTHER: IDENTIFY ANY RELEVANT CONVERSATIONS, LETTERS, MEMORANDA, ET CETERA USED IN CONNECTION WITH THIS REVIEW.

CONCLUSIONS

REMEDY EFFECTIVE? CHECK THE APPROPRIATE RESPONSE BASED ON YOUR REVIEW OF THE AVAILABLE INFORMATION. IN YOUR EVALUATION, CONSIDER ISSUES RELATIVE TO ANY DISCHARGE REQUIREMENTS (ARE THEY BEING MET?), MEASURED HYDRAULIC GRADIENTS (ARE THEY BEING MAINTAINED?), RESULTS OF LONG TERM MONITORING (ARE THERE ANY TRENDS?), ET CETERA AS APPLICABLE. ELABORATE IF THE RESPONSE IS "NO".

ROD COMPLIANCE? IF THERE ARE RECORD OF DECISION STIPULATIONS RELATIVE TO O&M ACTIVITIES, CHECK THE APPROPRIATE RESPONSE, OTHERWISE WRITE N/A. ELABORATE IF THE RESPONSE IS "NO"

CO COMPLIANCE? IF THERE ARE CONSENT ORDER STIPULATIONS RELATIVE TO O&M ACTIVITIES, CHECK THE APPROPRIATE RESPONSE, OTHERWISE WRITE N/A. ELABORATE IF THE RESPONSE IS "NO".

OTHER: IDENTIFY ACTUAL OR POTENTIAL PROBLEMS RELATED TO THE ACTIVE O&M ACTIVITIES AT THE SITE (E.G. WELLS ARE NOT PRODUCING YIELD, EQUIPMENT IS WORN, PIPING IS CLOGGED/BROKEN, THERE ARE CODE VIOLATIONS, ET CETERA).

RECOMMENDATIONS

RECOMMEND ACTIONS TO ADDRESS, SOLVE, OR MITIGATE ANY PROBLEMS OR "NO" RESPONSES GIVEN UNDER THE CONCLUSIONS HEADING ABOVE. CONSIDER **ROD/CONSENT ORDER MODIFICATIONS** AND CHECK THE APPROPRIATE BOX. ELABORATE IF THE RESPONSE IS "YES". CONSIDER **RECLASSIFICATION OF THE SITE** AND CHECK THE APPROPRIATE BOX. INDICATE THE PROPOSED CLASS IF THE RESPONSE IS "YES".

COMMENTS

PLEASE INCLUDE ANY ADDITIONAL INFORMATION RELEVANT TO O&M ACTIVITIES AT THE SITE. IN ADDITION, USE THIS SPACE TO CONTINUE EXPLANATIONS FROM ANY SECTION ABOVE.

APPENDIX F

MONITORING WELL LOGS/CONSTRUCTION DIAGRAMS

ENGINEERING–SCIENCE DRILLING RECORD

BORING MW–2

Contractor: SJB Drilling
Driller: Joe Lamm
Inspector: Anne Zimmerle
Rig Type: Soil Mat HSA

PROJECT NAME Lehigh/NYSDEC
PROJECT NUMBER SY279.02.02

Sheet 1 of 1
Location: Behind fenced area, SW

Site Grid
Northing: 9277.0764
Easting: 4706.3529
TOC Elevation: 592.81 ft.
Ground Surface Elevation: 591.18

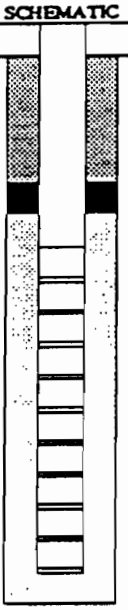
Weather Partly Sunny mid 70's
Date/Time Start 7/14/92 12:35pm
Date/Time Finish 7/14/92 4:45 pm

GROUNDWATER OBSERVATIONS

Water Level				
Date				
Time				
Meas. From				

Miniramp Reading	Head Space	Sample Depth	Blow Counts	Pct Rec.	FIELD IDENTIFICATION OF MATERIAL
		0			Black Sand and Construction Material to .9'
0	0.2		38–24	13"	(FILL)
		2	10–13		Brown fine Sand and Silt, trace fine gravel, dry loose
0	4.1		79–38	8"	Brown fine Sand and Silt, .5' to Limestone
		4	24–40		augered through rock to 4.5'; split spoon to 6' (18")
			–21		Brown Silt and fine Sand, trace clay, limestone chunks
70	32.6	6	40–42	9"	damp, firm
15	53.4		8–14	12"	Green Silt and fine Sand, some (–) fine gravel, rock chips, damp, firm
		8	14–25		(SM)
0.3	29.8		18–26	17"	Gray brown fine Sand, some Silt, some(–) fine gravel to 9.1'
		10	32–70		Black mottling, rocks coated black, damp
0	8.9		24–34	24"	Dark gray fine Sand, some Silt, some (–) fine gravel, firm, moist, wet at 10.5'
		12	38–50		(GC)
0.3	6		21–28	19"	Gray Sand, some coarse to fine gravel, trace silt, wet
		14	31–31		(GM–GP)
0	6.9		15–26	18"	Dark gray medium sand and gravel at 15' change to fine sand and gravel, wet
		16	32–45		
0	0.4		100–	6"	Gray sand and gravel, very damp at 16.5' black shale (SW)
		18	–50/0		at 16.5' SS refusal TOR 16.5'

WELL SCHEMATIC



COMMENTS

stickup = 1.65'
Type I Portland Cement to 3.5'
2" ID Sch40 PVC riser
3/8 Bentonite Pellets from 3.5' to 4.5'
2" ID 10 Slot Sch40 PVC Screen from 4.5' to 15.5'
Sibley 1240 sand
Bottom of Well = 15.5'
Total Depth 16.5'

STANDARD PENETRATION TEST
SS = SPLIT SPOON
A = AUGER CUTTINGS
C = CORED

ENGINEERING–SCIENCE DRILLING RECORD

BORING MW–4

Contractor: SJB Drilling
Driller: Jim Lowe
Inspector: Anne Zerbonia
Rig Type: Soil Mat HSA

PROJECT NAME Lehigh NYSDEC
PROJECT NUMBER SY279.02.02

Sheet 1 of 1
Location: West/Central
near old maintenance bldg

Site Grid
Northing 9573.4944
Easting 4933.7387
TOC Elevation: 592.61
Ground Surface Elevation: 590.62

Weather rain low 70's
Date/Time Start 7/15/92 2:50
Date/Time Finish 7/16/92 9:15 am

GROUNDWATER OBSERVATIONS

Water Level				
Date				
Time				
Meas. From				

Monitor Reading	Head Space	Sample Depth	Blow Counts	PCT Rec	FIELD IDENTIFICATION OF MATERIAL	WELL SCHEMATIC	COMMENTS
		0					stick up = 1.59'
8.3	81.2		11–11	18"	Black Sand, some silt and gravel Brick, Cement, pungent odor		Type I portland cement
		2	26–29				2" Sch40 PVC casing
0.7	6.7		21–31	18"	Brick to 2.8'		Bentonite seal 3' to 4'
		4	21–16		to Black fine sand, some silt, dry Black silt and fill to 4.2' (FILL)		
2	1.1		3–4	22"	Brown fine sand, some silt		
		6	4–2		soft, moist		
0	0.8		1–3	18"	Brown fine sand, some silt grading to		
		8	4–5		Brown fine sand and silt, some clay, more gray color with depth		Silica sand type 12x40 sand
0	8		4–17	6"	Brown fine sand and silt, some clay, grading to dilatant gray brown fine sand		10 Slot
		10	24–37		Drove 2 spoons, little recovery in each		2" ID Sch40 PVC
0	0.7		7–34	24"	Gray till, sand, some silt, some medium–fine gravel, dry, firm		5.5' to 14.5'
		12	35–35		water 2' in hole		
0	2.2		21–62	19"	Gray sand, some silt, some coarse–medium gravel		
		14	65–68		Very dry, firm (SM–SC–SP)		
0	1.2		18–27	14"	Gray sand, some silt and gravel, to 14.6' TOR 15.5'		bottom of well = 15.0'
		16	27–50/1		to Brown sand and silt, some gravel Spoon refusal		Total Depth = 15.5'
		18					

STANDARD PENETRATION TEST
SS = SPLIT SPOON
A = AUGER CUTTINGS
C = CORED

Cutting were paked in 55 gallon drums.

APPENDIX G

CHEMICAL HAZARDS

Patty's Industrial Hygiene and Toxicology

THIRD REVISED EDITION

Volume 2A
TOXICOLOGY

GEORGE D. CLAYTON
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9 CHROMIUM, Cr

9.1 Source and Production (1, 2)

Elemental Cr does not exist naturally, but is found combined in the mineral chromite, $FeOCr_2O_3$, the most important source. Never found pure, conforming to the formula, chromite contains impurities of Mg and Al with interstitial impurities of metal silicates. High grade ore usually contains 48 percent Cr_2O_3, with a Cr/Fe ratio of about 3 : 1.

U.S. domestic mine production of chromite ceased in 1961, when the last Defense Production Act contract was phased out, but the United States continues to be one of the world's leading chromite consumers, producing Cr alloys, refractories, and chemicals. World production of chromite, totaling almost 8 million short tons in 1974, was derived from more than 20 countries worldwide, with the Soviet Union and Republic of South Africa leading all others by large margins. Chromite concentrates are readily produced by flotation with C_{16} or C_{18} amines after comminution of the ore to 120 μm size. Sodium lignin sulfonate is used to suppress the gangue.

Chromium is produced in the form of an iron alloy, ferrochromium, by the reduction of chromite ores with carbon or silicon in an electric furnace. Ferrochromium is also produced from chromite by a silicothermic reaction in the presence of a suitable oxidizing agent, such as calcium chromate, $CaCrO_4$, sodium nitrate, $NaNO_3$, or manganese dioxide, MnO_2. The latter reaction is exothermic. The silicothermic reaction is generally employed to produce ferrochromium of controlled low carbon content (0.03 to 0.1 percent C), although low carbon ferrochromium is also produced in quantity by the reduction and removal of the carbon of normal high carbon ferrochrome in a vacuum furnace by iron oxide, chromic oxide, or silica. The ferrochromium produced by this method is usually of very low carbon content (0.01 to 0.03 percent).

Chromium metal may be produced also by the exothermic reduction of chemically produced Cr_2O_3, using powdered aluminum as the reductant. Since the use of aluminum powder is associated with explosive hazards and with considerable losses of Cr, molten aluminum is poured at a lower temperature into a melt of Cr_2O_3-44 wt. percent CaO. With vigorous stirring nearly 94 percent of the Cr is recovered. The metal quality is good. Chromium metal is also produced on a commercial scale by electrolysis of an ammonium chromium-alum solution, prepared either from Cr ore or from high carbon ferrochromium.

Chromium metal is produced in more limited quantities by the thermal dissociation of CrI_3 in contact with a suitable heated deposition surface under vacuum conditions (the Van Arkel-de Boer process). This is the purest form of Cr presently available. A high purity product is also produced on a commercial scale by the hydrogen reduction of oxide in electrolytic Cr.

Chromium can be plated electrolytically from chromic acid containing a small amount of sulfuric acid. It is also plated by thermal decomposition of the hexacarbonyl, or from a salt bath containing $CrCl_2$, or from gaseous chromous chloride, $CrCl_3$. It is also produced in so-called sponge form by the hydrogen reduction of $CrCl_2$.

Chromium chemicals (chromates) are made by roasting finely ground chromite ore with soda ash and lime at 900 at 1000°C, followed by water leaching and acidification to obtain pure Na_2CrO_4 or $Na_2Cr_2O_7$, from which all other Cr compounds can be derived.

For making $CrCl_2$, Cr ores and coal are ground to 0.074 and 0.25 mm, respectively, pelleted, dried at 200 to 220°C, and chlorinated at 800 to 950°C in an electric shaft furnace. The iron content is 2 wt. percent, maximum, and averages ≤0.2 wt. percent, sometimes 0.03 to 0.08 wt. percent. The Cr loss is 2 to 8 wt. percent.

Chromium carbide that is dissolved in iron or silicon can be purified by distillation from a graphite hearth and enclosure. A row of electron beams distills the metal. The product contains <0.5 wt. percent Fe and <100 wt. ppm each of S, P, O, and C.

9.2 Uses and Industrial Exposures

9.2.1 Uses

One of the principal and most important uses of pure Cr is for plating. Many household appliances are chrome plated, as are decorative parts of other manufactured items, including automobiles. Tools, plug gauges, rolls, drum dryers, chemical equipment, dies, mandrels, electric appliances, gears, food machinery, kettles, pans, packing machinery, and many hundreds of other articles are chrome plated.

Because Cr and other electrodeposits, enamels, and phosphate coatings on metal surfaces to be protected do not always provide uniform and reliable protection at high temperatures, particularly in cases where differences in thermal expansion may cause spalling, recourse is often made to chromizing. In this process a layer of Cr is applied at a temperature causing inward diffusion to form a composition gradient. An important use of chromizing is to protect Ni against a sulfur-containing atmosphere. Chromizing is commonly done by the thermal diffusion of Cr powder, the reduction of Cr oxide or halide in contact, or the thermal decomposition of Cr halide or carbonyl.

Chromium is used extensively for alloying with iron to form stainless steels, special high strength steels (ferritic), and electrical resistance wires; with Ni and Fe to form Ni stainless steels (austenitic); and with Mn and Fe to form Cr–Mn stainless steels. In steel, Cr prevents corrosion caused by atmospheric conditions, corrosive waters, acids, and high temperature conditions.

Chromium is alloyed with nickel in varying quantities to form special heat-resistant alloys. The composition of these alloys is usually 15 to 28 percent Cr and 5 to 78 percent Ni.

Nickel-base alloys of Cr are of particular importance because of their high strength and unique resistance to high temperatures. Other metals can be added to the Cr- or Ni-base alloys, such as Fe, Ti, Cb, Co, Cu, Mo and W, to produce modified alloys for specialty uses.

Chromium forms with Ni the well-known electrical resistance alloy Nichrome, which has made possible the large number of modern home appliances such as electric ranges and water heaters.

Chromium is alloyed with Co for use in producing cutting tools, and some W, V, and C are usually added for these applications. Stellite is probably the best-known commercial form of this type of alloy. These materials also possess desirable high temperature properties.

Chromates and dichromates have many applications in lithography, textile printing, tanning, dyeing, photography, and the manufacture of dyes, pigments, wallpaper, electric cells, explosives, matches, and rubber goods. Zinc chromate is widely used in metal primers. Chromous salts have fewer uses. Chromic salts, particularly the chloride and oxide, are used as a textile mordant, paint pigment, catalyst, and in refractory brick (the oxide). The carbide, Cr_3C_2, is used in gauge blocks, in hot extrusion dies, and in powder form as spray-coating material.

In medicine, gamma-active radioisotope ^{51}Cr is used clinically to label erythrocytes in hematologic studies. In radiotherapy, ^{51}Cr implanted in certain types of tumors has been used when maximal benefit has been achieved from other forms of radiation.

9.2.2 Industrial Exposures

The chief exposure to hazardous Cr substances in American industry is believed to be an acid-soluble, water-insoluble chromate-chromite mixture produced in the preparation of chromate (386). Before recognition and correction of this source of worker exposure, a 29-fold increase in the incidence of bronchogenic carcinoma occurred in workers in the U.S. chromate industry over that among workers in other chemical operations. Perforation of the nasal septa from CrO_3 also occurs. Other sources of exposure are to fume and dust of chromite ore and chrome alloys about electric furnaces and in ore-crushing operations. Chromous and chromic salts provide little industrial hazard. Chromium welding fumes can be hazardous if levels exceed 1 mg Cr/m³.

In the cement industry Cr-containing cements have led to dermatitis (387), as have chromate and chromic acid solutions and mists, such as in Mg foundries where the castings are treated with chromic acid solutions for weather resistance. Contact dermatitis has been reported from handling timber in which Cr salts were impregnated (388). Electrolytic plating baths may carry a mist of Cr into the air; of 233 Cr platers examined by Schwartz and Seike (389) in 1930, 42.6 percent had dermatitis, ulceration, or scars; in 52 percent of these, the nasal membrane was damaged.

9.3 Physical and Chemical Properties

The physical and chemical properties of Cr and some of its compounds are listed in Table 29.9.1.

Table 29.9.1. Physical and Chemical Properties of Cr and Some of Its Compounds

Form of Cr	At. or Mol. Wt.	Sp. Gr.	M.P. (°C)	B.P. (°C)	Solubility
Chromium (Cr)	52.01	7.20 (28°C)	1157 ± 20	2672	Insol. hot or cold H_2O; sol. HCl, dil. H_2SO_4; insol. HNO_3
Chromic oxide (chromium sesquioxide (Cr_2O_3)	152.02	5.21	2435	4000	Insol. hot or cold H_2O; insol. acids, alcohol, alkalies
Chromium trioxide, "chromic acid" (CrO_3)	100.01	2.70	196	Dec.	1.6 kg/liter (15°C), 2.067 kg/liter (100°C); sol. ether, alcohol, H_2SO_4
Chromic chloride ($CrCl_3$)	158.35	2.76 (15°C)	~1150 subl	1300	Insol. cold, sl sol. hot H_2O; insol. alcohol, acetone, MeOH, ether
Chromous chloride ($CrCl_2$)	122.92	2.75	824		V. sol. cold or hot H_2O; sl. sol. alcohol; insol. ether
Chromium carbide (Cr_3C_2)	180.02	6.68	1890	3800	Insol. cold, hot H_2O
Sodium chromate (Na_2CrO_4)	162	2.71	—	—	873 g/liter (30°C); sol. MeOH; sl. sol. EtOH
Potassium dichromate ($K_2Cr_2O_7$)	294.19	2.676 (25°C)	Triclin. monoclin. 241.6–>398	Dec. 500	49 g/liter cold H_2O, 1.02 kg/liter hot H_2O; insol. alcohol

Chromium exhibits a valence of 2+, 3+, and 6+ in its compounds. Chromium is a blue-white hard metal that is not oxidized in moist air and, even when heated, oxidizes to only a slight extent. In an atmosphere of CO_2, it oxidizes to Cr_3O_4; in HCl, to $CrCl_3$. Chromium combines directly with N, C, Si, and B. A passive form of the metal is conferred by oxidizing acids, attributable to a film of Cr_2O_3.

Bivalent Cr compounds are basic, the trivalent compounds amphoteric, and the hexavalent compounds acidic. The chromate ion in acidic solution is a powerful oxidizing agent. Cr^{3+} compounds closely resemble Fe^{2+} compounds; Cr^{3+} compounds resemble those of Al^{3+}. Chromium forms a series of isopoly acids and salts (K_2CrO_4, $K_2Cr_2O_7$, $K_2Cr_2O_{10}$, $K_2Cr_4O_{13}$); a group of chrome alums (e.g., $NH_4Cr(SO_4)_2 \cdot 12H_2O$); and complex ammines (e.g., $[Cr(NH_3)_6]Cl_3 \cdot H_2O$), in which Cr is trivalent.

9.4 Analytic Determination

Samples of air containing Cr compounds may be collected in an impinger using a *N* NaOH solution. Upon addition of KI and acidification, the liberated I_2 is titrated with standardized $Na_2S_2O_3$ for a determination of Cr^{6+}. Microgram quantities may be determined colorimetrically by diphenylcarbazide following permanganate oxidation. The procedure is applicable to samples of air, water, and urine, with good recovery and a sensitivity of 0.03 μg of Cr in 25 ml (390). A modification has been recommended of the Saltzman method of Abell and Carlberg (391). Total particulate Cr in air can also be determined by atomic absorption spectrometry, as recommended by the Physical and Chemical Analysis Branch of NIOSH (63). The method has a sensitivity of about 0.1 μg/ml and the linear range extends to about 4 μg/ml when the nitrous oxide-acetylene flame is used. The range in air, accordingly, is from 0.01 g/m³ to greater than 0.4 μg/m³ when the recommended volumes of 100 liter air and 10 ml of solution are used.

Tissues have been analyzed for water- and acid-soluble Cr by a modified carbazide test (392) and for acid-insoluble Cr (chromite) by the method of Cahnmann (393). Procedures exist for the separation and analysis of soluble Cr^{6+} and soluble total Cr in Portland cement, and for the determination of total Cr (394).

9.5 Physiologic Response

The physiological responses to Cr and its compounds are wide and varied, because associated with each of the three Cr valencies, 2+, 3+, and 6+, are different toxicologic potentials. Further, within each valency group, toxicity varies according to solubility. Thus whereas chromic acid, CrO_3, in which Cr is 6+, is highly corrosive and toxic, Cr^{2+} and Cr^{3+} salts, including Cr_2O_3, are ascribed a far lower order of toxicity; and Na chromate, Na_2CrO_4, a highly soluble Cr compound, in which Cr is also hexavalent, also has a low order of toxicity, but the corresponding insoluble chromates of Ca, Pb, and Zn are suspect, human lung carcinogens. By comparison, the physiological response of univalent elements is more regular and uniform.

The principal toxicologic reaction sites from industrial exposures are the skin (various forms of dermatitis, ulcers of the upper respiratory tract, nasal inflammation, and perforation of the septa), the larynx (inflammation and ulceration), and the lung (inflammation and bronchogenic carcinoma). The liver (enlargement) and the gastrointestinal tract (inflammation and ulceration) have been less commonly involved and only after many years of exposure, as was keratosis of the lips, gingiva, and palate. All responses occurred at Cr levels well above those recommended at the time (0.1 mg/m³ as CrO_3), from Cr^{6+} substances, and after long exposures.

9.5.1 Acute Toxicity

The industrial importance of the chromates, Cr^{6+}, has naturally led to greater amounts of toxicity data than for Cr compounds of lower valency, Cr^{2+} and Cr^{3+}. But even here, strict comparison of data between compounds is impossible because of the piecemeal nature of the investigations, different species and routes of administration having been used on otherwise comparable compounds.

From information compiled in the NIOSH Registry (171) the acute toxicities of K and Na chromates and dichromates obtained variously from six laboratory animal species and by five routes are roughly of the same order of magnitude. These soluble chromates are of very low toxicity by mouth (of the order of 1500 mg/kg) and of intermediate toxicity by cutaneous application (from ca. 200 to 350 mg/kg), but highly toxic by subcutaneous, intraperitoneal, or intramuscular injection (from ca. 10 to 50 mg/kg), depending on the species.

The insoluble chromates of Ca, Pb, and Zn, as determined in the mouse, rat, and guinea pig, varied from a mouse intravenous LD_{LO} of 30 mg/kg for basic Zn chromate, to 400 mg/kg for an intraperitoneal LD_{50} for the guinea pig for Pb chromate (171).

A more detailed study of percutaneous and intraperitoneal toxicity of Na chromate and dichromate in guinea pigs, reported by Wahlberg (395), showed both chromates to be more toxic by intraperitoneal administration (7-day mortality, 100 percent from 140 and 80 mg doses for di- and monochromate, respectively, versus 60 and 20 percent mortality by cutaneous application.

In comparison, chromous (Cr^{2+}) and chromic (Cr^{3+} salts had lower acute toxicities than those of Cr^{6+} (171); again, oral toxicity was far less than that from other parenteral routes; for example, oral rat LD_{50} for $CrCl_3$ of 1870 mg/kg versus 400 mg/kg for intravenous LD_{LO} for the mouse. The acute toxicities, obtained from various species and routes, of Cr^{2+} acetate, chloride, chromic fluoride, nitrate, oxide, and sulfate, Cr hexacarbonyl, and chromic acid (CrO_3) are given in Reference 171. Unfortunately, little acute toxicity for many of the Cr compounds appears to have been determined.

High doses of chromates given subcutaneously to rabbits and guinea pigs damage the kidneys with the production of albumin and casts (396), and inhalation of chromic acid dust by rabbits resulted in pulmonary hyperemia and inflammation (397).

In man, a case of acute K dichromate poisoning was characterized by an enlarged, tender liver with measurable amounts of Cr in the urine (398). The use of chromic acid for the treatment of warts and cauterization of hemorrhoids has resulted in several cases of Cr poisoning. A fatal case of nephritis has been reported (399) following treatment of carcinoma of the face with chromic acid crystals. Anuria rapidly developed, and death occurred in 30 days. The kidneys showed extensive lesions, particularly in the convoluted tubules, and blood chemistry showed very elevated levels of urea, inorganic phosphates, amino acids, and creatinine.

9.5.2 Chronic Toxicity

The chromic Cr^{3+} compounds $Cr_3(PO_4)_3$ and basic Cr carbonate, $Cr_3O(CO_3)_3$, were shown by Akatsuka and Fairhall (400) to have a very low order of toxicity by inhalation and by ingestion compared with Cr^{6+} compounds. Two cats exposed for 30 to 60 min/day for a total of 40.6 and 81.2 hr during a 4-month period to basic Zn chromic carbonate showed no evidence of acute or chronic toxicity, respectively, in spite of lung sections showing pinhead size patches of green Cr discoloration. At an average exposure concentration of 58.3 mg Cr/m³, the estimated Cr inhaled was 115 and 223 mg Cr during the exposure period. The lack of measurable amounts of Cr in tissues other than the lungs tended to substantiate the lack of toxicity, and the 16 and 19 percent of the estimated inhaled dose retained in the lungs was apparently inert.

Ten cats daily ingesting levels of Cr phosphate of from 50 to 1000 mg/cat, amounting to 3.75 to 83 g total intake, also showed no adverse health effects, and of 12 tissues analyzed for Cr, only bone, urine, and blood showed more than the 2 to 17 μg Cr/50 g tissue sample (400). Thus a sharp distinction can be drawn between the toxicity of Cr^{3+} and Cr^{6+} inorganic salts.

In contrast, guinea pigs inhaling chromic acid mist from Cr-plating baths, in which Cr is hexavalent, for 0.5 to 3 hr daily during a 45-day period developed lesions of the mucosa and submucosa of the respiratory tract, as well as changes in the spleen and kidney (401). Also, sintered CrO_3 and $CaCrO_4$ produced cancers, mainly sarcomas, when introduced in pellet form into the thigh muscle and pleural cavity of rats (402). But neither $BaCrO_4$ nor basic KZn chromate, intratracheally injected into rats, resulted in carcinoma of the lungs; nor did a "mixed" chromate dust inhaled by rats and mice 4 hr daily throughout a major part of their lifetime (403), or irradiated ^{51}Cr metal as an implant for 18 months in rats (404).

Tests of oral toxicity of four Pb-containing chrome pigments in worldwide use have been made in 90-day studies in dogs and rats (405). Molybdate orange, medium chrome yellow, primrose chrome yellow, and light chrome yellow (compositions given in Table 29.9.2) were fed for 90 days mixed separately in stock diets at extremely high levels of 2000, 5000, and 20,000 ppm for the purpose of comparing the toxicities with that of a standard basic Pb carbonate, white Pb. All pigments exerted effects at each level that were qualitatively similar, but medium chrome yellow, which had the lowest content of

acid-soluble Pb, was less toxic. The toxicities could not be related quantitatively to their Cr or Pb content, or to acid-soluble Pb. In relative species response, dogs showed greater mortality; only the 2000 ppm level showed none for each of the pigments, whereas rat mortality occurred only at the 20,000 ppm level, and then only in association with the stress of bleeding; female deaths exceeded male by more than tenfold. Mortality response in dogs was probably a reflection of Cr tissue content, for the Cr in liver, kidney, brain, and bone of the dogs for all four pigments was two orders of magnitude greater in liver and kidney and one order of magnitude in brain and bone than in the rat. However, tissue Pb content of kidneys and bone of rats exceeded that of dogs, often by two- to fivefold or more, whereas that of liver was less by ten- to twentyfold; the brain showed comparable values (1 μg/g or less). These varying tissue Cr and Pb contents may explain the general finding of inability to relate toxicity quantitatively to the Cr and Pb content of the pigments. Despite these lesser amounts of tissue Cr, rats showed more marked changes in hematologic variables (mean corpuscular volume, mean corpuscular hemoglobin) for all pigments, and histopathology of the kidney showed some tubular changes in dogs at all levels as did the majority of the rats. Noteworthy is the comparison of the toxicity of the white Pb positive control (for composition, see Table 29.9.2); toxic responses of the chrome pigments were considerably less severe and later in appearance, indicating a capacity of Cr^{6+} to reduce Pb toxicity.

Soluble chromates, as opposed to the insoluble chromates just cited, at approximately 100 to 1000 times lower concentration, however, are well tolerated by both man and animals. Orally, hexavalent (and trivalent) Cr proved nontoxic to rats when given in drinking water for 1 year at levels from 0.45 to 25 ppm (406), and a Long Island, New York family has been reported (407) to have drunk water for several years from a well contaminated with 1 to 25 ppm chromate without known effects (408). On the other hand, an investigator is reported to have become nauseated when drinking 10 ppm of bichromate ($Cr_2O_7^{2-}$), but found 3.5 ppm tolerable (409).

9.5.3 Metabolism

Baetjer et al. studied the metabolism of Na chromate, K dichromate, and chromic chloride hexahydrate in guinea pigs for 140 days in the case of chromates and 60 days for the chromic compound, after intratracheal injection of 200 μg Cr (410). The water-soluble chromates were cleared very rapidly from the lungs; only about 15 percent Cr remained in the lungs 10 min after injection; 20 percent was found in the blood at this time and another 5 percent in the three tissues analyzed (liver, kidneys, spleen), although small amounts were deposited in other soft tissues, and some may have been deposited in the gastrointestinal tract through cilia action. After 24 hr about 13 percent of the dose had been eliminated in the urine; 11 percent remained in the lungs, 8 percent in the red blood cells, 1 percent in the plasma, and about 4 and 3 percent in the liver and kidneys. Small amounts of Cr were deposited in muscle, adrenals, and skin, but essentially none in the bones, even after 90 days. Chromium in all tissues, except lung and spleen, gradually decreased to very low levels or disappeared altogether by 140

Table 29.9.2. Composition of Lead-Containing Chrome Pigments

Molybdate orange (MO)	61.6% Pb; 3.7% Mo, 12.3% Cr; acid-soluble Pb, 4.7%
Medium chrome yellow (MCY)	62.3% Pb, 14.6% Cr; acid-soluble Pb, 2.6%
Primrose chrome yellow (PCY)	64.3% Pb; 10.6% Cr; acid-soluble Pb, 9.3%
Light chrome yellow (LCY)	63.2% Pb; 10.6% Cr; acid-soluble Pb, 5.4%
White lead (WL) (control)	81.9% Pb, acid-soluble Pb, 81%

days. Chromium reached a peak in the spleen in 30 days, after which it declined, owing to uptake of disintegrating Cr-bearing red cells. Chromium was still present in the lungs and spleen even after 140 days, however. The metabolism was the same whether Na or K dichromate was administered.

With $CrCl_3$, however, Cr metabolism was different; 10 min after injection, 69 percent of the dose remained in the lungs, and only 4 percent was found in the blood and the three other tissues that were analyzed. By 24 hr, 45 percent was still in the lungs, 6 percent was excreted in the urine, and only a very small percent was found in the other tissues. The spleen was the only tissue that showed accumulation and that occurred during the first 48 hr. The Cr that reached the blood was found in greater amounts in the plasma than in the cells, thus differing from the distribution of the chromates. Lung retention of Cr^{3+} also differed; at the end of 30 days, 30 percent of Cr^{3+} was still in the lungs compared with 2.4 percent of Cr^{6+}, and at 60 days, the comparable retentions are 12 and 1.6 percent.

Distribution of Cr in rats fed Cr^{6+} in drinking water at various low levels (0.45 to 11 ppm) for 1 year showed highest amounts in spleen, then bone, kidney, and liver. All ppm Cr^{6+} in the drinking water resulted in closely similar values for both males and females in the spleen at 1 year. At 25 ppm Cr^{3+} in the water, the amount retained was about 1/5 to 1/10 that of Cr^{6+} (406).

Mean urinary Cr value of white U.S. chromate workers in 1950, when exposures greatly exceeded the TLV 0.1 μg/m³, was 43 μg/liter, for black workers 71 μg/liter. White workers from this group had a lung cancer rate 14 times the expected number, and blacks had 80 times the expected number. Mean value for blood Cr of white males was 4 μg percent; for black males it was 6 μg percent; 12 percent of the samples showed values of 10 μg percent or greater (386).

As far as can be judged from the environmental data reported, comparable urinary and blood Cr values were found by Mancuso (411) in 90 chromate workers with known exposures to predominantly chromate or chromite, measured as "soluble" and "insoluble" Cr. Urinary Cr values among production and maintenance workers averaged 51 μg/liter, ranging as high as 380 for the production workers and 130 for the maintenance men. Blood Cr values determined in these workers tended to range below 10 percent of the urine values during exposure; 74 days after exposure, blood values tended to exceed 10 percent. Workers mainly exposed to chromate dusts develop higher blood and urine Cr levels than those having contact mainly with chromite dust. The

alloy industry, a single incident of pulmonary fibrosis has been reported, and in the cement industry, allergic hypersensitivity to Cr contributing to "cement dermatitis." All the effects have been from hexavalent Cr forms, except lung cancer, in which evidence for trivalent Cr has also been adduced. Beginning in 1827 by Cummin, in Great Britain, and in 1885 by Pye, who described "chrome holes," literally scores of reports of these industrial injuries have been repeatedly reported, all presumably from exposures in excess of the recommended limits. Because of their voluminous nature, only the more recent or definitive reports on each effect that contain references to previous reports are given.

Skin Effects. Dermatologic lesions, consisting of ulcers from contact with chromic acid or Na and K chromates, contact type dermatitis from the primary irritant, and sensitization from chromic acid and its salts, which are described in detail in the U.S. Public Health Service survey of the early 1950s (386), had already been reported by McCord and others in the 1930s and before. The ulcer, which occurs more readily if there is a break in the skin, is round, nonspreading, and deeply penetrating, with a clean-cut central crater whose base is covered with exudate or a tenacious crust. Once developed it is slow to heal, and if exposure continues it may persist for many months. The healing process usually leads to scar formation. In the six chromate plants surveyed, about 46 percent (300) white workers and 62 percent (151) blacks had ulcers or scars, and the percentage of scars and nearly healed and active lesions was about the same in both groups (84, 5, 12 percent).

Although skin ulceration continued to be reported in chromate and electroplating workers as late as the 1970s (426, 427), in those employed 1 year or less, no incidents of ulceration occurred in one of three plants surveyed in 1973 by Markel and Lucas (428) despite employment for much longer periods of time; 15 were employed 8 years or more, seven between 4 and 8 years, four between 1 and 4 years, and only six less than 1 year. Differences in personal hygiene practices were considered responsible.

Chromate dermatitis has been reported frequently since it was reported in 1944 among workers in the aircraft industry (429), for example, in 1951 among diesel railroad employees who handled chromate solutions as rust inhibitor (430); in 1959 among lithographers (431); in 1963 among automobile assemblers in Great Britain (432); in 1963 and 1964 among welders with Cr hypersensitivity (433, 434); and in 1963 handling chromate paints (435) and in 1954 and 1974 from cement (436, 437) in a few individuals with allergic chromate hypersensitivity. Cement workers who have green tattoo areas (from Cr^{3+}) get a lymphocytic reaction of delayed hypersensitivity along with a simultaneous cement dermatitis, as determined, oddly, by chromate (Cr^{6+}) patch tests (438). Fumes from welding Cr-containing stainless steel, or certain Cr-containing welding rods (E11018, E70S-3, E70T-1) (439) can trigger a severe eczematous eruption on the palms of the hand of a Cr-sensitive individual (434). Dermatologists do not understand the absence of Cr dermatitis among chromate workers who have chrome ulcers (428), and present-day improved work practices have for the most part eliminated Cr dermatitis seen in many of the industries listed above.

Nose, Throat, and Sinuses. At least a dozen reports, mostly from the United States (440), show nasal membrane inflammation and ulceration, nasal septal perforation, and chronic rhinitis, laryngitis, and pharyngitis to be a common finding among chromate workers and those exposed to chromic acid mist alike. Mancuso (411) in an intensive medical study of 97 chromate workers in a plant population of 226, reported in 1951 that 63 percent showed perforations of the nasal septum, 86.6 percent had chronic chemical rhinitis, and 42.3 percent pharyngitis. Clinical changes in the larynx, including polyps and hoarseness, were present in 23 percent of those examined, and sinuses of 89 workers had polyps or cysts in 8 percent. There was a direct relation of sinus involvement to soluble chromates, but is should be noted that it is not possible to relate nasal septal perforation to ambient Cr levels because of the common practice of nose picking to remove encrustations. It should be noted also that there were no instances found of cancer of the affected parts, although carcinoma of the lung was found at this time (1951).

Very similar incidences of these lesions were reported in the 1953 survey made by the U.S. Public Health Service of 897 workers, both white and black, in six chromate plants (386). At this time there was no evidence that chromic acid and its salts led to chrome sensitization (441).

Despite the widespread knowledge of the seriousness of the findings in both these studies, which led to the rebuilding of the chromate producing plants with improved controls, reports of similar Cr injuries appeared as late as 1965 in the United States in the chrome plating of auto parts, (442) and among electroplaters in Brazil in 1974 (443). In both instances, exposures were well above the recommended TLV

Lung Cancer. The Cr lesion that is currently giving the greatest concern to industry and health agencies alike is bronchogenic carcinoma. Attention was first drawn to its appearance in seven chromate producing plants in the United States by Machle and Gregorius (444). These investigators were employed by the chromate industry after it was learned that lung cancers were appearing in the chrome pigment plants in Germany (445). Because this study had some shortcomings, Baetjer (446) brought further attention to the problem when she found from hospital records that the lung cancer rate was higher among chromate workers than in hospital control groups. This, and a more intensive study by Mancuso and Hueper (447) of one of the plants studied by Machle and Gregorius, initiated the comprehensive study by the U.S. Public Health Service of six chromate plants comprising 897 workers (386). This study revealed that there were 28.9 times as many deaths from lung cancer in the male chromate workers between 1940 and 1948 than would have been expected on the basis of average death rate for the United States for that period.

Mancuso has redetermined the lung cancer death rates, after 37 to 43 years, of chromate workers that had been employed from 1931 to 1937 in a plant that began operations in 1931–1932 (448). Of a total of 332 workers, 52 percent or 173 had died by 1974. Of the 173, 41, or 23.7 percent, had died of lung cancer (62.1 percent of all cancers). The latent period was found to cluster around 27 to 36 years, indicating Cr to

be not a very potent carcinogen. Mancuso was able further to ascertain with greater assurance than in the past that (1) both soluble and insoluble Cr forms are involved in eliciting lung cancer. This conclusion was reached on the basis of a clear demonstration of a dose-response relation for both soluble and insoluble forms, made possible by his determination of the relative amounts of the two forms in the workers' exposure in his previous report (411). (2) No cancer deaths occurred from exposures to the insoluble forms at levels less than 0.25 mg/(m³)(year); three deaths resulted from the soluble forms at this level, however; and no deaths resulted from total Cr at levels below 0.50 mg/(m³)(year). In each, the number of deaths increased as the level of Cr increased. Because no smoking histories were available, the first study having been made before the association with lung cancer was realized, it must be assumed that smoking habits were the same in each group.

Attention at long last has been focused on the carcinogenic potential of chrome pigments. First suspected in Germany in 1943 from Pb and $ZnCrO_4$, it was not until the mid 1950s that two reports of very preliminary nature, appeared, one from Norway (449) and one from the United States (450). Four cases of cancer, three of the bronchus and one of the pancreas, were reported from a small cohort of 24 workers employed in three Norwegian plants, and exposed at various times since 1948 to $PbCrO_4$ or $ZnCrO_4$, and sometimes to both (449). Because of the small number in the study cohort (total employment 133 from 1948 to 1972), the relatively short exposure time (six exposed for 4 years, four for 5 years, and 14 for more than 5 years), and the small number and types of cancer, and in light of the long latent period for Cr cancers found by Mancuso, the study reveals no information as to whether Pb or Zn chromates are carcinogenic together or singly. Moreover, one worker with bronchogenic carcinoma was a heavy smoker.

An epidemiologic study of three chrome pigment plants in three states in the United States, sponsored by the Dry Color Manufacturers' Association, was somewhat more revealing (450). The cohort analysis included 548 male workers, among whom 53 deaths had occurred before December 31, 1974. For two plants with only $PbCrO_4$ exposure, a threefold excess of lung cancer was found (SMR 313.7 vs. 100 expected). For workers hired before 1960 and who had worked for at least 10 years, the SMR was 236.4. Employees of the plant with both Pb and Zn chromate exposure, hired before 1960, had an SMR of 237.1. Five deaths due to stomach cancer were also found, apparently from swallowing inhaled chromate dust or from excessive mouth breathing. Because of the small number of cancer deaths, it is still not possible to declare either Pb or Zn chromate carcinogens, and certainly not potent ones, because in nearly one-half of the samples, time-weighted average Cr concentrations exceeded the recommended TLV

Systemic Effects. In addition to the gastric cancers just cited, decrease or loss of taste and smell with gastrointestinal ulcers or hypertrophic gastritis have been reported by several observers, including Mancuso (411). Mancuso also found some blood changes, including leukocytosis or leukopenia, monocytosis, and eosinophilia. Liver injury has been recorded rarely.

A single report of progressive pulmonary fibrosis in a small number of workers in a plant producing chrome alloys, low carbon ferrochrome alloys, and ferrosilicon appeared in 1962 (451). No subsequent report on the subject has since appeared. It is possible, as a result of this finding, that exposures have since been reduced. Air levels of total dust ranged from 0.4 to 445 mg/m³. The fibrosis was believed not to be due to silica because the quartz content of the dust was commonly below 2 percent and no crystalline silica was found in the lung. The disease was characterized by recurring bouts of acute pneumonitis, with cough, wheezing, anorexia, loss of weight, increased sedimentation rate, linear and nodular fibrosis, and ventilatory impairment.

9.6 Hygienic Standards of Permissible Exposure

The TLV Committee of the American Conference of Governmental Industrial Hygienists has recommended a limit of 0.05 mg/m³ as Cr as a time-weighted average for chromic acid (CrO_3) and chromates; of 0.5 mg/m³ as Cr for soluble chromic and chromous salts; of 1 mg/m³ for Cr metal; and a classification of A1a for certain insoluble metal chromates, Pb, Zn, and Ca, which are suspected of carcinogenic potential for man (452).

NIOSH has recommended to OSHA a limit of 0.05 mg/m³ as CrO_3 as a time-weighted average exposure for an 8-hr workday, 40-hr work week, and a ceiling concentration of 0.1 mg/m³ as CrO_3 as determined by a sampling time of 15 min (453); for Cr^{6+} of carcinogenic potential, a limit of 0.001 mg Cr/m³ in the breathing zone; and for noncarcinogenic Cr^{6+}, a limit of 0.025 mg Cr/m³ as a time-weighted average for a 10-hr workday, 40-hr work week, and a ceiling of 0.050 mg/m³ (440).

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18 LEAD, Pb

18.1 Source and Production (1, 2)

Lead rarely occurs in the elemental state, but exists widely throughout the world in a number of ores, the most common of which is the sulfide, galena. The other minerals of commercial importance are the carbonate, cerussite, and the sulfate, anglesite, which are much less common.

Lead also occurs in various U and Th minerals, arising directly from radioactive decay. Because certain isotopes are concentrated in Pb derivatives from such sources, both the atomic weight and density of such samples vary significantly from normal Pb.

Lead ores generally occur in nature associated with Ag and Zn. Other metals commonly occurring with Pb ores are Cu, As, Sb, and Bi. Most of the world production of As, Sb, and Bi arises from their separation from Pb ores.

Commercial Pb ores may contain as little as 3 percent Pb, but a Pb content of about 10 percent is most common. The ores are concentrated to 40 percent or greater Pb content before smelting. A variety of mechanical separation processes may be employed for the concentration of Pb ores, but the sulfide ores are generally concentrated by flotation processes, for which they are particularly suitable.

Domestic (U.S.) production in 1974 amounted to nearly 663,900 tons, representing a rising trend since 1962. Production from Missouri mines constituted nearly 85 percent of the nation's total, with the output from Idaho and Colorado and a declining small production in Utah making up the bulk of the remaining total, although 15 other states had small productions. Production of Pb concentrates accounted for about 250,000 tons.

World production in 1974 including that of the United States amounted to 3,844,687 short tons, with 53 countries other than the United States producing substantial amounts; nine of these, the Soviet Union, Australia, Canada, Peru, Mexico, Yugoslavia, Korea, China, and Morocco, produced more than 100,000 tons each.

U.S. domestic ore sources are Pb ores containing from 1.1 to 11.5 percent Pb with minimal amounts of Zn (essentially none to 1 percent); Zn ores, with 0.25 to 1.25

percent Pb; Pb–Zn ores, with 0.6 to 11 percent Pb; and CuPb, Cu–Zn, and Cu-Pb–Zn ores, with 0.3 to 2.1 percent Pb (1).

More than one-third of the Pb produced in the United States is derived from reclaimed Pb or Pb alloy. The chief source of this secondary Pb is scrapped automobile storage batteries. The secondary Pb is obtained by melting of scrap and is generally not purified but rather reused as Pb alloy. Secondary Pb from storage batteries contains Sb and is reused in battery manufacture. The Pb scrap that contains Sn is reused in the manufacture of solder.

The metallurgy of Pb consists of three separate operations, concentrating, smelting, and refining. Concentrating is a means of ore beneficiation to increase the Pb content, the process involving wet-grinding to a particle size of about 75 percent −325 mesh; conditioning the resulting slurry with certain reagents to establish a proper alkalinity; further mixing with flotation chemicals which collect the lead minerals in a froth; and thickening and filtrating this concentrate. Each individual type of ore is unique and requires some adjustment in the quantities and properties of the reagents added, but those most suitable are sodium carbonate, lime, copper sulfate, pine oil, cresylic acid, xanthate, and sodium cyanide; generally all these are used in amounts ranging from 0.05 to 5.0 lb/ton of ore. Reagents other than those named are also employed. Zn minerals, Fe compounds, and the earthy components of the original ore are depressed instead of floated by this treatment. After further separations, the Zn (and sometimes the Fe) is usually recovered. If Cu, Ag, and Au are present, they normally remain with the Pb and are removed and recovered in the refinery; should the Cu be a major constituent, it may be desirable and economical to separate it from the mixed Pb–Cu concentrates by special flotation procedures.

Minor amounts of Pb ores are concentrated by utilizing their high specific gravity, or by water-leaching of Pb ores under air or oxygen pressure; this treatment converts the sulfur in the ores to H_2SO_4 and the Pb into Pb sulfate which is suitable for feed to a smelter.

For smelting, Pb concentrates, plus occasionally high grade raw ores and returned intermediates such as flue dusts, lime rock, and other fluxes suitable for slag formation, are blended on mixing beds or drawn from proportioning bins to form a smelter feed. This mixture is pelletized to provide a homogeneous and carefully sized material, which is then sintered to eliminate most of the sulfur and to agglomerate the particles into relatively large and hard lumps that will not be blown out of a blast furnace.

The sinter, together with about 9 percent of its weight of carbon in the form of coke, is charged into the top of blast furnaces. The coke supplies both the fuel for smelting the charge and the reducing gas which reacts with the Pb oxide to form metallic Pb. As the charge descends in the furnace, the molten metal flows to the bottom, from where it is withdrawn for further treatment. The remainder of the charge forms slag that floats on the Pb and is removed from the furnace at a higher level. The Pb bullion collects the Ag, Au, Cu, Bi, Sb, As, Sn, and other minor metals; the slag carries the Zn and Fe as well as the silica, lime, and other gangue; the dust contains a variety of readily volatilized elements including Cd and In, plus considerable Pb and Zn

The impure bullion is cooled in kettles to about 350°C, whereupon a dross forms which carries almost all of the Cu along with major amounts of Pb. This material is skimmed and treated with soda ash in a small reverberatory furnace which produces a matte and speiss, high in Cu and low in Pb, which is sent to a Cu smelter. Additions of sulfur remove the final traces of Cu, after which the decopperized Pb bullion proceeds to the refinery.

If Sn is present, the Pb bullion may be reheated to 600°C with the introduction of air, and a second dross containing the Sn is removed prior to refining proper.

Slag from the blast furnace is frequently treated by fuming out the Zn and remaining Pb; the dust from this operation is sent to a Zn plant while the now barren slag is discarded. The dusts from the sinter plant and blast furnace are collected in a baghouse or Cottrell precipitator and returned to the mixing beds or bins.

A refining process is necessary because the decopperized Pb bullion still contains significant amounts of As, Sb, Sn, Bi, Ag, and Au. About 88 percent of the world's output is produced by means of pyrometallurgic techniques; the remainder is electrolytically refined. The latter process is used only when the Bi content of the Pb bullion is relatively high. Further details of the pyro process are to be found in Reference 2.

18.2 Uses and Industrial Exposures

The largest single use of Pb is in the manufacture of storage batteries; the second largest use is said to be for the production of tetraethyl and tetramethyl Pb, but these gasoline additives are being phased out because of reasons of potential adverse effects on community health. Because alloying of Pb with other metals provides greatly improved mechanical and chemical properties compared to pure Pb, alloys chiefly with Sb and Sn confer the properties needed to sheathe power and telephone cables from moisture. Alloys containing 1 percent Sb are used for telephone cable, and Pb–As alloys containing 0.15 percent As, 0.1 percent Sn, and 0.1 percent Bi for power cable. Battery grid alloys for Pb–acid storage batteries, mentioned above, contain 6 to 12 percent Sb for strength, small amounts of Sn to improve castability, and one or more minor additions to retard dimensional change during use. Chemically resistant alloys of Pb find many applications where resistance to chemical corrosion or to attack by water and air is required. Alloys of this type commonly contain 0.06 percent Cu, or 1 to 12 percent Sb when greater strength is needed. Type metals are Pb alloys containing 2.5 to 12 percent Sn and 2.5 to 25 percent Sb; the latter increases hardness and reduces shrinking during solidification, and the former improves fluidity and reproduction of detail; both elements lower the melting temperature of the alloy (460 to 475°F). Lead-bearing metals (babbitt metals) contain 10 to 15 percent Sb, 5 to 10 percent Sn, and for some uses, small amounts of As and Cu. Antimony and Sn combine to provide wear resistance needed in cast sleeve bearings and in freight-care journal bearings. Solders include a large number of Pb-base compositions, most of which contain large amounts of Sn with certain minor constituents added to provide specific benefits such as improved wetting characteristics.

Other uses are for ammunition, sheet, pipe and foil, vibration damping and radiation shielding, and paint pigments.

According to Elkins (719) the most severely hazardous operations in the past from inorganic Pb were spraying molten Pb and Pb paint, grinding or power-sanding Pb or solder, pouring of leaded iron and steel, certain operations in storage battery manufacture, smelting Pb and burning Pb battery boxes, and mixing and weighing Pb powders and pigments. Soldering, Pb-casting, brush painting, linotyping, and steel tempering he regarded as relatively nonhazardous. Today, in the United States at least, many if not about all of these hazardous operations are better controlled, both by engineering practices and medically, because of keener recognition of the hazards, so that frank Pb poisoning, so common in the 1930s, 1940s, and even in the early 1950s, is far less frequently observed (720).

In a U.S. Public Health Service Survey of outdoor exposure to airborne Pb in policemen, firemen, taxi drivers, vehicle tunnel attendants, garage mechanics, and service station attendants few of the samples of blood and urinary Pb even approached the biologic standard (721), tollbooth operators included (721a).

Occasional and unexpected sources of inorganic Pb exposure do occur, however, as in indoor firing ranges (722) and in electric arc welding on materials of unsuspected Pb content. The *Handbook of Chemistry and Physics* (583) lists 124 inorganic and organic acid Pb compounds that offer potentially hazardous exposures if mishandled. According to the *Chemical Buyer's Directory for 1977–1978* (723), this list is incomplete, particularly for the organic acid compounds such as the naphthenates, octoates, linoleates, and tallates, which have relatively large volume use.

Of a listing of 14 organometallic Pb compounds (583), only tetraethyl and tetramethyl Pb are handled in sufficient volume to present a health hazard of serious moment, and even these highly toxic substances cause serious intoxication only accidentally because of the rigorous medical control program initiated by Kehoe (724) and continued throughout the years by Adrian Linch and others. In the future, the possibility of intoxication from these agents should decrease as they are gradually phased out as gasoline additives.

18.3 Physical and Chemical Properties

The physical and chemical properties of some of the more industrially important compounds of Pb are given in Table 29.18.1 representing only 19 of the more than 70 listed as commercially available in Reference 723.

The properties of Pb are such that it enters into combinations, alloys and compounds, that find the widest range of applications of any metal, except possibly Fe, ranging from the many uses of its alloys (Section 18.2) to its use in bullets and shot; its high density provides a maximum of striking power with a minimum of air resistance that makes Pb ideal metal for this purpose. Its capacity to combine with the nonmental N to form the azide makes it the standard detonator for explosives. Because of its great resistance to corrosion, Pb is used extensively in construction, particularly in the chemical industry,

and because of its resistance to attack by many acids, it is widely used in the manufacture and handling of H_2SO_4 and in the cathodic protection of metal structures used in large structures, pipelines, bridges, and ships. Because of its excellent sound attenuation property, Pb is being used in construction to help meet building code requirements of maximal permissible noise levels, and because of its vibration damping property, Pb–asbestos pads are used to isolate heavy machinery. Because of its radiation absorbing property (high density) Pb is used as a protective shielding for X-ray machines, protective aprons, and wherever glass windows are needed in radiation equipment.

Among Pb compounds, its combinations with chromate and molybdate through the chloride intermediate find wide use as "chrome" pigments; its silicate, formed through the Pb acetate intermediate, is used in ceramics and fireproofing fabrics; and the arsenate has long been used as an insecticide.

Newer uses of inorganic Pb compounds include litharge, which is widely used at 2 percent concentration to improve the magnetic properties of Ba ferrite ceramic magnets. High Pb ferrites containing about 20 percent Pb have superior magnetic properties. A calcined mixture of Pb zirconate and titanate is marketed as a piezoelectric material, and PbTe is finding some use as the active component of thermoelectric generators.

18.4 Analytic Determination

Lead in atmospheric dust and fume is determined by conventional aqueous atomic absorption spectroscopy, after collection on cellulose membrane filters and ashing with HNO_3. The detection limit is about 1 μg/ml. For a 100-liter air sample, this represents an air Pb concentration of 0.01 mg/m³, assuming 1 μg Pb per filter dilution to 10 ml. The method has no known interferences.

18.4.1 Biologic Monitoring

In contrast to the straightforward procedures for sampling and analyzing the air for Pb, new methods are continually being proposed and examined in an effort to select those that best reflect worker exposure. The advantages of biologic monitoring over air sampling have been discussed by Stokinger (725). More importantly, it was pointed out that some of the available methods measured *response*, whereas others measured only *exposure*, a fact that is only now becoming recognized. At this time, five tests were available (Table 29.18.2). Three tests, urinary coproporphyrin, urinary δ-aminolevulinic acid (ALA), and erythrocyte δ-aminolevulinic acid (and its related enzyme in the red blood cell, δ-aminolevulinic dehydrase (ALAD)), provide the opportunity to measure response of the lead-exposed worker as well as knowledge of what the overall exposure of the lead worker has been. These tests determine the lead in either blood or urine. To each of these five indexes were assigned definite levels of concern for the industrial hygienist, including the category of biologic threshold limits (BTL). Considerable weight was given these limiting levels because they were arrived at by a

Table 29.18.1. Physical and Chemical Properties of Pb and Some of its Industrially Important Compounds

Material	Chemical Symbol	At. or Mol. Wt	Sp. Gr	M.P. (°C)	B.P. (°C)	Solubility g/liter
Lead, metal	Pb	207.19	11.3437 (16°C)	327.5	1740	Insol. cold, hot H_2O, sol. HNO_3
Lead acetate	$Pb(C_2H_3O_2)_2$	325.28	3.25 (20°C)	280	—	44.3 (20°C), 221 (50°C); v. sl. sol. alcohol; sol. glycerin
Lead arsenate (ortho)	$Pb_3(AsO_4)_2$	899.4	7.80	1042, sl dec. 1000	—	V. sl. sol. cold H_2O; sol. HNO_3
Lead azide	$Pb(N_3)_2$	291.23	—	—	Explodes at 350	0.023 (18°C); 0.09 (70°C)
Lead carbonate (basic white lead)	$2PbCO_3 \cdot Pb(OH)_2$	775.6	6.14	Dec. 400	—	Insol. cold, hot H_2O, sol. HNO_3; insol. alcohol
Lead chloride	$PbCl_2$	278.1	5.85	501	950	0.99 (20°C); 3.34 (100°C); sl. sol. dil. HCl, NH_3; insol. alcohol
Lead chromate (chrome yellow)	$PbCrO_4$	323.18	6.12 (15°C)	844	Dec.	0.0000058 (25°C); insol. hot H_2O; sol. acid, alkalies; insol. acetic acid, NH_3
Lead molybdate (molybdate orange)	$PbMoO_4$	367.13	6.92 (25°C)	1060–1070	—	Insol. hot H_2O; dec. conc. H_2SO_4; sol. acid, KOH; insol alcohol
Lead nitrate	$Pb(NO_3)_2$	331.2	4.53 (20°C)	Dec. 470	—	37.65 (0°C); 127 (100°C); sol. alkalies, NH_3
Lead monoxide (litharge)	PbO	223.19	9.53	888	—	0.0017 (20°C); sol. HNO_3, alkalies
Lead oxide (red)	Pb_3O_4	685.57	9.1	Dec. 500	—	Insol. cold, hot H_2O; sol. HCl, acetic acid; insol. alcohol
Lead sesquioxide	Pb_2O_3	462.38	—	Dec. 370	—	Insol. cold H_2O; dec. hot H_2O, acid
Lead suboxide	Pb_2O	430.38	8.342	Dec.	—	Insol. cold, hot H_2O; sol. acid, alkalies
Lead peroxide	PbO_2	239.19	9.375	Dec. 290	—	Insol. cold, hot H_2O; sol. dil. HCl; sl. sol. acetic acid; insol. alcohol
Lead oxychloride (Nat'l. Matlockite)	$PbCl_2Pb(OH)_2$*	519.29	7.21	Dec. 524	—	0.0095 (18°C); sol. alkalies
Lead silicate (meta)	$PbSiO_3$	283.27	6.49	766	—	Insol. cold H_2O; dec. acid
Lead sulfate (basic)	$PbSO_4PbO$	526.44	6.92	977	—	0.0044 (0°C); v. sl. sol. hot H_2O, sl. sol. H_2SO_4
Lead sulfide (galena)	PbS	239.25	7.5	1114	—	0.000086 (15°C), sol. acid; insol. alcohol
Lead stearate	$Pb(C_{18}H_{36}O_2)_2$	774.15	—	115.7	—	0.05 (35°C); 0.06 (50°C) sl. sol. ether, conc. H_2SO_4; insol. acid
Tetraethyl Pb	$Pb(C_2H_5)_4$	323.44	1.659 (11°C)	−136.8	Dec. 200 91 (19 mm)	Insol. cold H_2O; sol. C_6H_6, pet. alcohol, ether
Tetramethyl Pb	$Pb(CH_3)_4$	267.33	1.995	−27.5	110	Insol. cold H_2O; sol. C_6H_6, pet alcohol, ether

* One of five natural minerals of the same general name.

(730), whose EP tests indicated considerable amounts of Pb remained in the bone marrow even after 3 months of deleading treatment.

One disadvantage of the EP test is that it is the least sensitive in detecting recent, sudden, or mild increases in PbB concentrations. A second shortcoming is that the EP test lacks specificity; Fe deficiency states and erythropoietic porphyria induce elevations in EP which are usually more marked than in Pb poisoning. In these conditions, however, ALAD remains normal (728).

Despite the many published reports on these tests and attempts at standardization (731), at present one still cannot define a standard, universally accepted, "cutoff" value for clinical or subclinical Pb intoxication for ether ALAD or EP, variation in methodology, uses of comparative standards, and reporting units, as well as consideration of hematocrit and sex, all indicate that exact replication of a referenced procedure must be done if comparable results are to be obtained.

Joselow and associates (732) have recommended replacing the EP test, which uses an extraction procedure, with a fluorescent procedure for Zn protoporphyrin (ZPP). The method avoids extraction and determines ZPP directly by fluorometric examination of diluted whole blood with high sensitivity (50 to 80 μg/100 ml, the normal concentrations in blood). A secondary standard of Zn protoporphyrin has been developed by the authors to facilitate standardization in other laboratories; developed also is a "European" standardized method for the determination of ALAD in blood (731). Owing to the relative instability of ALAD in human blood, recommendations have been made for the proper storage time between blood-drawing and assay (733).

From the reports of recommended improved procedures for monitoring response to Pb exposure, such as References 734–736, no conclusion can be drawn presently as to which will ultimately be universally used. For example, attempts are being made to show a correlation between PbB and EP (736), but as expected, correlation coefficients are low, around 0.65 in both unexposed children and in exposed workers. The log of EP gave a slightly better correlation ($\gamma = 0.72$) for Pb workers (737). No better correlation should be expected because one, PbB, measures exposure, whereas the other, EP, measures response to that exposure, which can vary among individuals and with changing degrees of exposure; only in cases when a steady state has been attained over long periods of exposure can a good correlation be expected.

18.4.2 Monitoring for TEL and TML

According to Kehoe (724), who early developed a medical control program for the tetraethyl Pb industry, "the concentration of Pb in the urine provides the only analytical criterion for the estimation of the significance of exposure to, and absorption of, tetraethyl Pb." The same statement can be made for tetramethyl Pb, TML, because both are metabolized similarly by the body, the only difference being the greater volatility of TML (Table 29 18 1), thus presenting a greater exposure hazard.

At the time, unacceptable correlations were found between air and urinary levels. Later work by Linch (738), with improved methods of air sampling and analysis, revealed an approximate linear relationship between Pb concentrations in air and urinary excretion.

Urinary ALA and Pb levels, in all 268 specimens, were measured on 123 male workers of various ages and lengths of service as operators or maintenance men in a production area of alkyl Pb antiknock compounds. Six workers were studied over a 7- to 9-month period (739), despite knowledge that there is no change in the hematologic picture at any stage of TEL intoxication (740). Although some elevated ALA levels were found in those with urinary Pb levels above 150 μg/liter, the author concluded that ALA determinations are of uncertain value in a monitoring program for TEL or TML production, because of the uncertain degree of exposure to inorganic Pb, which does elevate ALA levels. Unfortunately, all but one of the six workers that were followed for extended periods had acceptable urinary Pb levels, that is, less than or equal to 200 μg/liter, and the corresponding ALA levels were well within normal (< 0.6 mg/100 ml).

18.4.3 Analysis of Biologic Tissues

The body tissue analyses that have appeared in the published literature since 1933 (741) have utilized chiefly emission spectroscopy or various modifications of the dithizone extraction procedure. Cholak (742), in a 1964 review of the several methods that had been in use, concluded that whereas both the dithizone and spectrographic methods were capable of detecting microgram amounts of Pb, in biologic specimens the spectrographic method had greater sensitivity (0.01 μg Pb/ml or g), unquestioned specificity, and greater speed. The recommended USPHS dithizone, double-extraction procedure (743) is satisfactory in the absence of Bi and Tl, with recoveries of 95.7 to 98.7 percent for the range of 20 to 150 μg Pb/100 g sample of blood. The polarographic method is satisfactory, but requires several manipulatory steps, with no advantage over the other methods in sensitivity (742). The dithizone procedure, recommended in the NIOSH criteria document on inorganic Pb in 1972, has now been generally replaced by atomic absorption spectrometry, mentioned above, for all types of body tissues including bone, hair, and teeth. Currently there are different kinds or types of procedures of atomic absorption: (1) flameless atomic absorption; (2) delves cup atomic-absorption; (3) chelation, extraction, and aspiration. In addition, anodic stripping voltammetry is becoming increasingly used (744).

18.4.4 Isotopic Analysis of Blood Pb Sources

The various sources of Pb in blood, that is, environmental air, dietary, and skeletal have been determined by measurement of certain stable isotopic Pb ratios by mass spectrometry and plotting the changes in isotope ratios of $^{206}Pb/^{204}Pb$ and $^{206}Pb/^{207}Pb$ with time (745). Working with a Dallas, Texas group of 10 persons, two of whom were South African expatriots, during the period March 1974 to June 1975, Pb isotope ratios of airborne Pb were found to increase by 6 percent. An unexpected finding was seasonal changes in the isotope ratios of blood Pb, which according to present knowledge appear

to be controlled by vitamin D synthesis. Thus the procedure offers an additional means of quantitatively examining the catabolic processes in the skeleton in health and disease, or as artificially induced by chelating agents.

18.5 Physiologic Response

Unlike most metals of industrial concern, relatively few determinations of the toxicity of inorganic Pb compounds have been made, presumably because Pb effects in man have been known since very early times. Of 60 discrete listings of inorganic Pb compounds in the NIOSH Registry of toxic substances (171), only 18 have been tested for acute toxicity, however, all the 13 listed organic Pb compounds bear acute toxicity data, because, unlike inorganic compounds, acute effects are the common manifestation of the organic compounds.

18.5.1 Acute Toxicity to Inorganic Pb

In a review of the data, it was found that strict comparison between oral and parenteral (intraperitoneal, intravenous) routes for any tested species was not possible. The closest comparison, demonstrating the wide differences in toxicity, was the oral LD_{LO} of the soluble nitrate for the guinea pig of 1330 mg/kg versus 50 mg/kg for the hamster and rat intravenous TD_{LO}. Similar wide differences in toxicity were found for the chloride; the oral LD_{LO} for the guinea pig is 2000 mg/kg versus 50 mg/kg for the intravenous TD_{LO} for the hamster. By comparison, the oral LD_{LO} for the guinea pig of the more insoluble fluoride is 4000 mg/kg. When Pb is attached to moieties such as arsenate and chromate, which have contributory toxicities, the acute toxicity for laboratory animals increases; the rat oral LD_{LO} for the arsenate is 100 mg/kg, and the guinea pig intraperitoneal LD_{50} for the chromate is 400 mg/kg Oddly, when Pb is combined with organic acids, as in oleate and naphthenate, acute toxicity is lowered; the guinea pig oral LD_{LO} is 8000 mg/kg for the oleate, and the rat oral LD_{LO} is 5100 mg/kg for the naphthenate. However, the acetate appears to be an exception, the hamster intravenous TD_{LO} being 50 mg/kg; no oral toxicity data are available. Additional data are found in Reference 171.

The early signs of acute intoxication are fatigue, disturbance of sleep, and constipation, with the more severe exposures followed by colic, anemia, and neuritis.

18.5.2 Chronic Toxicity from Inorganic Pb

Because Pb in all its forms is a cumulative poison, chronic responses are the common manifestations seen in industry. An extensive summary to 1973 of the toxicologic effects of Pb exposure, their recognition, and the factors affecting susceptibility has been prepared by Goyer and Rhyne (746) with 257 references, for the most part since 1960, when the second edition of this book was written. Sixteen of the references represent the original work of Goyer

Normal Values Inorganic Pb is a generally ubiquitous element being present in land, water, air, and food; hence measurable amounts exist in all adult body tissues and fluids of individuals in present-day societies. Accordingly, it is essential to determine "normal" ranges of Pb content in body tissues and fluids, if proper evaluation of industrial overexposure is to be made.

Kehoe, in 1961, concluded from extensive investigations (747) that the daily intake of Pb for an individual in the United States with normal Pb exposures was about 330 μg; intake may vary from less than 100 μg to more than 2000 μg/day, and may average as little as 120, or as much as 500 μg/day. Kehoe determined that persons *in approximate Pb balance* of 330 μg/day excreted about 10 percent in the urine (30 μg Pb/day) and the remainder in the feces (ca. 300 μg). These are very general averages; a more detailed grouping of male Philadelphia residents reported in 1965 (748) showed values of urinary Pb ranging from 20 μg/liter for suburban residents to 33 ± 14 μg/liter for police.

Normal blood Pb values in adults, as recorded by Hammond in 1969, are shown in Table 29.18.3. Differences were apparent according to environment and geographic location. Analyses were performed by the dithizone procedure.

Normal levels of Pb in nine tissues of adults are recorded in Table 29.18.4. The data cover a period of some 15 years, the work of seven different investigations. Considering the time span, differences in geographic residence and age (many of the individuals were in the later decades of life), and the different analytic methods (dithizone before 1961, atomic absorption spectrometry and anode stripping voltammetry later), remarkably good agreement among the ranges of values is found (Kehoe, kidney and brain values, versus those of later investigators); see Table 29.18.4.

In agreement with past findings of Schroeder and Tipton (754), accumulation of Pb with age was found by Zaworski and Oyasu (752) for brain tissue (right frontal lobe), and for the lung by Stringer et al. (749). Sex differences were also found for these tissues as well as for hair, more Pb being present in hair from females than in hair from males (Klevay, 1972). The age effect seen in brain tissue amounted to 0.064 mg Pb/100 g for males between 51 and 60 years compared with 0.055 at 21 to 30 years. Females, however, showed little change with age and at corresponding ages showed slightly lower

Table 29.18.3. Blood Pb Levels of Adults

Sampling Group	μg Pb/100 ml Blood
Suburban nonsmokers, Philadelphia	11
Suburban smokers, Philadelphia	15
All policemen, Cincinnati	25
Service station attendants, Cincinnati	28
Traffic policemen, Cincinnati	30
Tunnel employees, Boston	30
Parking lot attendants, Cincinnati	34
Garage mechanics, Cincinnati	38

Table 29.18.4. Pb Content of Adult Human Tissues from "Normal" Unexposed Populations (United States)

Tissue	Pb Content (mg/100 g Wet Tissue)	Investigator	Ref.
Bone	0.67–3.59	Kehoe (1961)	747
Liver	0.04–0.28	Kehoe (1961)	747
Lung	0.03–0.09	Stringer et al. (1974)	749
Lung	8×10^{-4}–0.03[a]	Sweet et al. (1978)	750
Hilar lymph nodes	4×10^{-4}–0.04[a]	Sweet et al. (1978)	750
Kidney	0.02–0.16	Kehoe (1961)	747
Kidney	0.05–0.16[b]	Morgan (1972)	751
Spleen	0.01–0.07	Kehoe (1961)	747
Heart	0.04	Kehoe (1961)	747
Brain	0.01–0.09	Kehoe (1961)	747
Brain	0.01–0.08	Zaworski and Oyasu (1973)	752
Teeth	0.28–31.4	Shapiro et al. (1975)	744
Hair	0.007 ± 1.17[c]	Weiss et al. (1972)	753

[a] Range of 100 urban specimens determined by spark source mass spectrometry and expressed as mg/g *dry* weight.
[b] Lower value refers to mean of 60 whites, higher value to mean of 20 blacks.
[c] Mean of 28 values.

values than males (0.046 to 0.051 mg/100 g). In the lung, Stringer et al. found peak Pb values in the seventh decade for people residing in the Houston, Texas area. Females, however, averaged only 55 percent of the male lung Pb values.

For the kidney, Morgan (751) selected 0.075 mg Pb/100 g wet tissue as a normal average, upper limit, as determined on 80 percent of the autopsies performed on individuals with mean ages between 53 and 65, at the Veterans Administration Hospital in Birmingham, Alabama between October 1967 and March 1968. The author points out that this value is considerably lower than previous values that had been considered normal (755, 756).

Twenty percent of the values did exceed this arbitrarily selected limit, but a previous hospital study suggested that 22 percent of hospital admissions used significant amounts of illicit, Pb-containing alcohol.

Normal Pb values for contemporary teeth of individuals in Philadelphia, Pennsylvania, suggested by Shapiro in 1975 (Table 27.18.4), were 3.7 and 45 times the values found for North Slope Alaskan and Mexican Indians, respectively, in a remote jungle area. The age of the Philadelphia citizens was not given.

In determining normal Pb values for human head hair (Table 29.18.4) Weiss et al. (753) found, surprisingly, a fourteen-fold decrease between values found between 1871 and 1923 and those in 1971, indicating that past environmental sources (water and food) far outweighted present-day increase of Pb from the atmosphere.

The total body burden of Pb in adult populations has been estimated by Schroeder and Tipton (754) from 17 tissues collected between 1952 and 1957 in 10 U.S. cities and cities in Europe, Asia, Africa, and the Far East, by the spectrographic method of Tipton (757). The human body burdens of Pb varied enormously, not only among U.S. cities but between U.S. cities and those elsewhere, as the following data show.

The total body burden as determined on autopsied material from 150 accidental deaths (histories unknown) in nine U.S. cities averaged 121.6 mg (range 9.6 to 435 mg); in 27 cases in San Francisco, with 7 of 10 tissues consistently with greater Pb content, 137.2 mg; in nine cases in Switzerland, with 6 of 10 tissues consistently lower, only 61.9 mg, about the same as estimated from 54 cases from Africa (63.2 mg); body burdens from the Middle East and Far East were 78.4 and 93.7, respectively.

Remarkable increases in cumulated Pb deposits in U.S. subjects were found particularly in the aorta and pancreas between the ages of 21 to 30 and 61 to 70 years amounting to 2.5-fold, after which the Pb content decreased precipitously. Less striking, but similar, were the cumulated increases in livers (1.7-fold) and spleens (threefold, but at lower levels); the increases in values were for lungs, 2.3-fold and for brains, twofold. Mean values for Pb in bone (rib) similarly increased according to age; 16.8 μg/g ash at ages 21 to 30 to 34.2 μg/g at ages 61 to 70, thereafter declining, but individual Pb values varied widely with ranges as great as fiftyfold (754).

Hematologic Effects. Of the many toxicologic effects of Pb on the industrial worker, the effects on the hematopoietic system have been the most prominent and have received the most attention. One of the results of this action is manifested as anemia. Of the microcytic, hypochromic type, the anemia is an early manifestation and may be the only clinical feature of chronic, low grade exposure to Pb. Resembling the anemia of Fe deficiency, it is seldom severe, the total red cell count in cases of moderate severity running between 4 and 4.5 million/cm^3, with a hemoglobin level between 70 and 80 percent and a color index of about 0.8 percent. The most prominent feature of the blood picture is reticulocytosis and basophilic stippling. Previously relied on for overexposure to inorganic Pb, the determination procedure has now been abandoned because of its nonspecificity, its general lack of correlation with degree of exposure, and its replacement by more refined procedures.

The anemia caused by Pb results from two basic red cell defects, shortened life-span, and impairment of heme synthesis (for mechanism, see Section 18.5.4). Red cell shrinkage and distortion and wrinkling of the membrane have been shown by Waldron (758) to result when large Pb concentrations are intravenously introduced into man.

Neurological Effects. Of the several organ systems affected by Pb, the nervous system is particularly sensitive to its effects. Lead encephalopathy, a brain disease with epileptoid convulsions, delirium, hallucination, cerebral edema, and other brain symptoms, used to be seen in Pb workers in the early 1900s. Now, with improved controls, it is rarely seen in the United States except in young children who have ingested Pb paint or been exposed to other Pb sources. Peripheral neuropathy, characterized in

adults by weakness and palsy with wrist drop, similarly is seen with decreasing frequency in the United States.

In spite of the seriousness of the clinical effects of Pb on the central nervous system, less is known about Pb-induced neuropathology than about hematopoietic and renal effects, chiefly because little interest has been shown in the problem by the few experienced neuropathologists. What knowledge exists on the pathology of Pb-induced peripheral neuropathy has evolved from a study of experimental models which showed that animals with chronic Pb intoxication developed a peripheral neuropathy characterized by segmental degeneration of myelin sheaths as well as axons (759). Subsequent investigations by Lampert and Schochet (760) and by Schluepfer (761) have shown segmental demyelination and proliferation, as well as Wallerian degeneration of the posterior roots of the sciatic and fibial nerves, the latter suggesting a cellular basis for Pb-induced paresthesia and sensory nerve loss.

Neurological sequelae of Pb toxicity have included a form of diffuse sclerosis in adults, suggested by the finding of proliferation of astrocytes and microglial cells in experimental Pb encephalopathy (762). Optic atrophy has also occurred in adults with chronic Pb poisoning (763) as well as auditory defects and vertigo (764). Auditory nerve injury was demonstrated in Pb-intoxicated guinea pigs which showed segmental demyelination of the eighth nerve similar to that described in other Pb-poisoned peripheral nerves. The sensory cells of the inner ear and the spiral and vestibular ganglion cells were unaffected, however (765). Night blindness was detected in rhesus monkeys fed diets that produced blood Pb levels of 55 or 85 μg percent compared to 14 μg in untreated controls for the first year of life. Eighteen months later, when blood lead levels were normal, visual discrimination performance of the 85 μg group was impaired in dim light relative both to their own performance under bright light and to that of other groups at all light levels used. The authors, Bushnell et al. (766), interpreted these results as a deleterious, enduring impairment of scotopic visual function as a result of early Pb intoxication. Whether such effects would occur in adults needs further study.

Renal Effects. A very considerable literature exists throughout the industrialized world on the effects of Pb on the kidney, because it is the organ that provides the major route of excretion, and in frank Pb toxicity there is a demonstrated renal pathology. This vast literature has been summarized by Goyer (767) with 101 references, 11 of which represent original research investigations by the author. We now attempt to summarize the chief findings of this report with a view to placing them in perspective for present-day industrial Pb operations in the United States.

A review of the world literature on the renal effects of Pb from the standpoint of its relevance to the U.S. Pb industry today must separate out the numerous old reports on (1) the acute renal effects of Pb from inordinately high levels of past exposures from which individuals died, had Pb-induced anemia (Thomas Oliver, 1914), or had acute, childhood Pb poisoning with encephalopathy (Marsden and Wilson, in England, 1955); (2) chronic forms of end-stage renal disease and renal failure following many years of

excessive Pb exposure (Charcot and Gombault, 1881); (3) childhood Pb intoxication with chronic Pb nephropathy in adulthood (Henderson, Australia, 1954); (4) attempts to show increased incidence of hypertension with increasing degrees of Pb exposure (Cantarow and Trumper, 1944); (5) Pb-contaminated illicit whiskey as the etiologic agent for chronic renal disease (Morgan et al., Southeast United States, 1966); and (6) the association of gout with chronic Pb nephropathy (old reports from England and Central Europe, summarized by Ludwig, 1957). Other studies from Yugoslavia (Radosevic et al.) and Rumania (Lilis et al., 1969) have reported either functional and anatomic changes in the kidneys of workers with prolonged Pb exposure, or renal failure in 17 percent (17 of 102 workers), with 13 having arterial hypertension. All these affected individuals, however, had histories of repeated episodes of Pb-induced abdominal colic.

Although, as Goyer says (767), the evidence from Australia is very suggestive of a cause and effect relationship between childhood Pb intoxication and chronic nephritis in later life, such a correlation has not been confirmed in at least two studies in the United States. Tepper (768), for example, found no evidence of chronic renal disease in 42 persons with a well-documented history of childhood plumbism 20 to 35 years previously at the Boston Children's Hospital. Similarly, Chisolm (769) found no evidence of renal disease in 62 adolescents who had had Pb intoxication 11 to 16 years earlier. One probable reason for the difference according to Chisolm is that the Australian children had a more protracted course of Pb toxicity, were older, ingested a more chalky, smaller particle size paint, and experienced a more chronic form of Pb toxicity.

The continuing improvement in the control of occupational exposure to Pb appears to be responsible for the failure of Lane in England (770) and Belknap in the United States (771) to find an increase in the frequency of hypertension and chronic renal disease. Later, Cramer and Dahlberg (772) were unable to find an excessive incidence of hypertension among Swedish workers in a battery plant, all of which indicates that if chronic renal disease develops from occupational exposure, then exposure must be well above recommended standards of good practice for a protracted period.

In characterizing the renal effects of Pb, Goyer has stated (767) that in the acute form the proximal tubular lining cells are affected, intranuclear inclusion bodies are formed, and there is an associated aminoaciduria. Impairment of mitochondrial function seen in the rat model has not been demonstrated in man, but may be inferred from morphological changes seen in human biopsy material. Reversibility of the acute tubular lesion occurs, however. Excessive aminoaciduria returns to normal a few weeks after chelation therapy but intranuclear inclusion bodies may persist for several years, reflecting a certain residual body burden of Pb. Whether the acute changes progress to a diffuse chronic nephropathy is uncertain because of sparsity of biopsy material, but is consistent with events seen in the experimental model.

A number of dietary factors are believed to enhance the susceptibility to Pb toxicity. Alcohol has been previously mentioned, but few other factors have been given rigorous evaluation. A recent study by Six and Goyer (773) showed that low dietary intake of Ca

increases the soft tissue Pb content, particularly that of the kidneys, thus providing another scientific basis for the long used, worldwide practice of milk drinking for Pb workers (774).

Effect on Other Systems. Research investigations on the toxicologic effects of Pb spanning almost 150 years, (Thackrah, 1831) have continued to reveal new systems affected by Pb-cytogenetic changes, reproductive effects, myocardial, Pb-induced changes in immune mechanisms, and others Unfortunately, space permits only brief mention of the chief findings to date in each of these categories, but a few of the latest references are given for each, from which earlier references can be pursued.

Cytogenetic Effects. Waldron (775), among others, has found nuclear polyploidy and mitotic abnormalities in bone marrow cells; Pb-induced chromosomal aberrations have been reported only since 1969, after the tedious technique of chromosome display had been mastered Muro and Goyer (776) found an increased number of gap-break type aberrations in leukocyte cultures from mice fed a diet high (1 percent) in Pb acetate; usually the breaks involved only single chromatids. In Vigliani's group, Forni and Secchi performed the most extensive studies of chromosome changes in Pb workers. In the first study (777) a statistical evaluation of the data based on 65 males with 65 controls matched for age and sex showed that the rates of chromatid and unstable chromosome changes (again, mostly of the one-break type) were significantly higher in those with preclinical and clinical Pb poisoning than among the matched controls; differences were not significant for those with past poisoning. The finding that the highest mean values of abnormalities occurred in the group with abnormal Pb absorption but without clinical signs of Pb poisoning prompted a second study (777a) of workers during their first exposure to Pb, and to follow their chromosomal changes with time. Comparisons were made with biochemical measurements of Pb absorption. This study confirmed and extended the results of the first study, showing that the rate of abnormal metaphases in the cultured leukocytes approximately doubled in the first month, increased after 2 months, and remained level up to 7 months of exposure; after this, there was a tendency to decrease at months 8, 9, and 11. Concurrently, urinary Pb and coproporphyrin levels increased sharply at 1 month, whereas ALA levels increased moderately and red cell ALAD activity was reduced to almost 50 percent of its initial value, where it remained throughout the study

The mechanism for this effect of Pb is unknown, but Pb does have an affinity for nucleic acids; however, no reports of its combination with chromosomes in vivo have appeared at this writing.

In spite of the many detailed investigations showing chromosomal effects of Pb, their genetic or other significance is unknown. Chromosomal aberrations occur normally, being 2 to 3 percent at age 20 for male workers (Freeport, Texas, 1965) and increasing to 5 to 6 percent at age 60 (778); this was verified in detail in a study of human chromosome changes in senescence (778a).

Reproductive Effects. Severe Pb intoxication has been associated in the past with sterility, abortion, stillbirths, and neonatal deaths, but modern information confirming that Pb poisoning affects birth rate or causes injury to the fetus in man is not conclusive. Stowe and Goyer (779), however, have shown experimentally that if rats are fed 1 percent Pb acetate in the diet, the offspring of Pb-burdened parents have decreased reproductive fitness, both paternal and maternal effects being observed. Paternal effects consisted in a retardation of embryonic growth and reduction in the number of weaned pups per litter; maternal effects consisted of reduced litter size, retardation of fetal development, and impaired postnatal development Because the combined parental effects were greater when both parents were Pb-treated, it was interpreted that the effects were additive.

In an experimental study of the teratogenicity, fetal toxicity, and placental transfer of Pb in rats, McClain and Becker (780) found malformations of the urorectocaudal system when $Pb(NO_3)_2$ was administered intravenously to pregnant rats on day 9 in doses of 25 to 70 mg/kg. $Pb(NO_3)_2$ was increasingly embryo- and fetotoxic when administered on days 10 to 15, but was not teratogenic. Postnatal survival was very poor when exposed on day 9 in utero. Although significant quantities of Pb were transferred to the fetus, the placenta greatly limited the passage, as interpreted from the large maternal–fetal gradients found.

Endocrine Effects. Only a few endocrine functions affected by Pb have been studied in either man or animal. Measurement of urinary excretion of steroids under different conditions of Pb exposure has been made to determine if excretion is related to Pb absorption; adrenal steroid excretion was first found to decrease and then to increase considerably during advanced stages of Pb intoxication (781). Aldosterone secretion rate (and plasma renin activity) were depressed during dietary salt restriction in nine men poisoned by Pb from drinking illicitly distilled alcohol (782).

The only other endocrine function examined in man to date is that of the thyroid, whose function was depressed because of decreased uptake of ^{131}I from abnormal Pb ingestion; thyroid-stimulating hormone can counteract this depression, according to Sandstead et al. (783). A decrease in I_2 uptake by thyroid slices and thyroids of rats had previously been reported by Slingerland (784).

Myocardial Effects. Degenerative changes in heart muscle thus far have only been found in Pb-poisoned children, (785, 786) as shown by abnormal EKG changes. After chelation, the EKG reverted to normal in all but 13 percent of 21 Pb-poisoned children. No reports have been found of such changes in Pb workers. Asokan et al. (787), however, did find diffuse, degenerative changes in the heart muscle of Pb-treated rats by electron microscopy.

Pb Effects on the Immune Mechanism. Little is presently known regarding the effects of abnormal amounts of body Pb on the immune mechanisms related to suscepti-

increase in spontaneous motor activity, a 12 percent decrease in the thickness of hippocampal cortex, with a decrease in midbrain acetylcholine esterase activity at Pb doses that produced no decrement in body weight gain. Male rats were dosed intraperitoneally daily with 7.5 mg/kg from birth to day 10 (794).

Xintaras and associates, in exploiting a then novel approach to evaluating behavioral changes (795), found that variations in the visually evoked response in rats administered Pb acetate to be similar to those evoked in man (795a) and from studying alterations in rapid eye movements during sleep, concluded that Pb may cause impaired neural control in rats

Carcinogenicity. Volume I of the monographs of the International Agency for Research on Cancer (796) summarizes several reports of Pb-induced neoplasms, both benign and malignant, of the kidney and other organs in rodents fed either the soluble Pb acetate or the basic form for prolonged periods. No tumors were elicited, however, when male rats were fed either the insoluble Pb arsenate or the carbonate for the same period (2 years) although renal cellular changes occurred.

Goyer and Rhyne (746), however, in a concluding statement in their summary work on the pathological effects of Pb in 1973, remarked that "there is no evidence that Pb produces cancer in man," presumably from having noted the lack of evidence obtained by Dingwall-Fordyce and Lane (797) for any association between Pb exposure and malignant disease in pensioned battery workers.

But in a later epidemiologic study of more than 7000 male, Pb-production and battery workers in the United States, by Cooper and Gaffey (798), the standardized mortality ratio was slightly elevated for all malignant neoplasms among smelter workers, although the SMR was not significantly elevated for battery workers. A possible reason is the greater solubility of the Pb fume and dust in smelting operations versus the larger Pb oxide particulate used in battery production.

Factors Affecting Susceptibility and Dose Response. Goyer and Rhyne (746) list nine factors affecting the toxicity of inorganic Pb: age, seasonal variation, Ca and P, protein, vitamins, alcohol, Fe deficiency, synergism with other metals, and coexistent disease (sex was omitted), but with the exception of seasonal variation and possibly Fe deficiency, which could be considered a metal synergism, these factors commonly affect the toxicity of most toxic agents. Knowing not only what factors affect toxicity, but also their magnitude, is of value for determining the safety factor to be incorporated in the environmental and biologic standards. Unfortunately, few, if any, have been rigorously investigated to qualify for fulfilling this requirement.

The factor of *seasonal variation* can be dismissed as applying only to childhood Pb poisoning; no reports have been widely circulated indicating that the toxicity in Pb workers is seasonally influenced, except possibly when air concentrations rise in winter months and plant ventilation is reduced. Similarly, the *age* factor is strictly related to young children, who are more susceptible to Pb intoxication than adults.

The demonstration by Six and Goyer previously cited (773) is presently the best experimental evidence of the critical role of Ca and P in influencing Pb toxicity; low Ca and P levels were shown to increase Pb retention in soft tissues (kidney) whereas high levels tend to mobilize Pb from the body.

In common with most toxicants, high levels of dietary *protein* have been found to reduce Pb toxicity, as does supplementation with cysteine or methionine; an 18 percent casein diet was required to reduce Pb toxicity for rats for which a 9 percent casein diet was highly toxic (799).

Vitamins appear to vary in altering susceptibility to Pb, but the evidence is often conflicting, apparently because of failure to recognize that vitamins act in conjunction with other factors present in some studies, absent in others. In one case, a lack of vitamin C increased neurological symptoms in guinea pigs, (800) whereas in another case vitamin C was without effect (801). Again, nicotinic acid was found to reduce porphyrinuria in Pb-poisoned rabbits, (802) but was of no benefit in the Pb-poisoned rat (803). Addition of vitamin D to rat diets actually enhances Pb toxicity (804) presumably by increasing gastrointestinal absorption of Pb, but the mechanism is by no means clear.

Alcohol has long been known to interfere with the coproporphyrin test for response to Pb exposure; however, not only are quantitative data lacking on the amounts of alcohol for significant effect, but the mechanism is far from clear. Several factors suggest themselves, among others the development of nutritional deficiencies and their similar capacity to produce mitochondrial injury.

The significance of *Fe deficiency* for susceptibility to Pb toxicity has been clearly demonstrated by Six and Goyer (746, Table VI). Rats on low dietary Fe and administered Pb had reduced hematocrit (37.8 ± 1.8 vs. 44.2 ± 1.7) to increased toxicity as measured by higher urinary ALA values (356.8 ± 167 vs. 180.2 ± 95); higher kidney Pb (38.7 ± 9.5 vs. 14.5 ± 3.2); and higher femur Pb (225.2 ± 30.3 vs. 75.2 ± 26.2).

In synergism with other (toxic) metals, Pb in some of its actions appears to behave (be metabolized) independently. Clarkson and Kench (805) found that the attachment of Pb to the red blood cell membrane is not influenced in vitro by the presence of other metals, including Cd, Hg, Zn, and Al. However, the previous demonstration of Fe deficiency effects would indicate that with Fe at least, Pb acts synergistically. Further, a synergistic action of Cd and Pb has been shown in their teratogenic effects in experimental animals (806), the intravenous dose levels at which these effects occurred, however, were rather high, 25 and 50 mg/kg.

Coexistent disease in major organs in general enhances the toxicity of industrial substances, and Pb is no exception, particularly individuals with hemoglobin anomalies such as hemoglobin S and C disease and thalassemia, which like Pb affect red blood cell metabolism, as a European report points out (807). Stokinger and Mountain (808) had similarly pointed out that individuals with glucose-6-phosphate dehydrogenase deficiency, chiefly Negroes of African descent, have increased susceptibility to Pb, and should be identified by a screening test before employment in a Pb industry.

Regarding *the sex factor,* it is general opinion that females are more susceptible than males, particularly those of childbearing age who are finding increasing employment even in the heavy industries. In recognition of a certain amount of placental transfer of

Pb and the well-established greater susceptibility of infants and young children to Pb poisoning, a blood Pb limit of 40 μg/100 ml has been recommended for their protection.

18.5.3 Metabolism

The metabolism of Pb has been discussed so widely that only newer aspects are mentioned here, and some misconceptions or little or unrecognized facts are pointed out. References 595, 724, 736, 746, and 809 should be consulted if further information is needed.

One of the less recognized aspects of normal human metabolism of Pb can be derived from an inspection of Table 29.18.5. This table shows that although far less Pb intake occurs from inhalation (by the urban dweller) the amount absorbed into the bloodstream can approximate that by ingestion because of the greater solubility of airborne Pb (810), and that nonindustrial, airborne Pb, even at maximum, represents less than one-third of that of the average industrially exposed.

A determination of particle size to ascertain the relation between dosage and biologic effects was made because previous data were too poorly defined to answer the practical question whether total dust concentration or the respirable fraction should be measured (811). Male baboons exposed to red Pb (Pb_3O_4) dust with mass median diameters of

Table 29.18.5. Environmental Lead Intake and Absorption in Man (Estimated)

Source	Absorption		Total Daily Absorption (μg/day)
	Percent	Daily (μg)	
Food and beverages 10–30 μg/liter Total: ~300 μg/day	5–15[a]	15–45	
City air (U.S.) 1–6 μg/m³ Total: 15–90 μg/day	25–50[b]	4–45	Nonsmoker, urban dweller 20–90
Cigarette smoke 1 μg/cigarette Total: 40 μg/day	10–15	4–6	Smoking, urban dweller 24–96
Industrial air TLV: 200 μg/m³ 10 m³/day Total: 2000 μg/day	~15	300	Industrial, smoking urban dweller 324–396

[a] Kehoe, 1962.
[b] Goldsmith and Hexter, 1967 (810).

2.0, 3.2, and 5.9 μm (0.8 and 1.6 μm mean diameters for "fines" and "coarse") at about 2 mg/m³ for 4 weeks, 4 hr daily, showed peak blood Pb values of 19 μg, 100 ml at 18 days for fines, whereas coarse blood Pb values peaked at 60 μg/100 ml at 26 days, showing that coarser Pb_3O_4 dusts were absorbed to more than a threefold greater extent than fines, and that total dust sampling, not respirable dust, is preferred for Pb_3O_4.

New light has been thrown on the distribution of inorganic Pb in blood (812). Of the total body Pb pool, blood Pb constitutes about 2 percent. Of this, approximately 90 percent of the Pb is bound to red cells, and is not readily diffusible. Of the Pb not so bound, plasma Pb constitutes the remainder in the blood, or approximately 0.2 percent of the total body pool. This plasma-bound Pb is composed of two fractions, a protein-bound fraction, and a diffusible fraction, which latter is believed to be a very small fraction of the plasma Pb.

The clinical significance of this information is that it provides a scientific basis for hematocrit correction of blood Pb in cases of anemia from any cause. Because the greatest part of blood Pb (90 percent) is red cell bound, blood Pb concentration varies as the red cell mass. Consequently anemia, or polycythemia, will affect blood Pb levels and require hematocrit correction. Although plasma-bound Pb constitutes less than 10 percent of blood Pb, this amount can be of considerable importance because of its effect on the ratio of diffusible to protein-bound Pb, analogous to diffusible and plasma-bound Ca.

Extention of knowledge to cellular and subcellular levels includes Pb effects on intranuclear inclusion bodies, on mitochondria, and on protein synthesis according to Goyer (746), who has investigated the pathologic changes accompanying these effects. (Cytogenetic effects, also investigated by Goyer, have already been discussed in Section 18.5.2.)

One of the characteristic cellular metabolic reactions in Pb intoxication is the formation of intranuclear inclusion bodies, a discrete, dense-staining mass found in the liver parenchyma and in the tubular lining cells of the kidney in man and animal. Consisting of a dense, central core and outer fibrillary zone, they can be identified and distinguished from those from Bi intoxication or from those of viral origin by special stains (Hg bromophenol blue and basic fuchsin). Pb has been identified by direct chemical analysis to be in the form of a Pb–protein complex to the extent of about 50 μg Pb/mg protein. The included Pb is 60 to 100 times more concentrated than whole kidney Pb. Goyer has found these inclusion bodies in renal biopsies of two Pb industry workers who had had excess Pb exposure, but only subclinical signs of Pb toxicity in the form of weakness, nausea, some abdominal colic, and blood Pb values of about 100 μg/100 ml. A possible metabolic role of the Pb inclusion bodies, proposed by Goyer (813), is that of an adaptive or protective mechanism during transcellular transport of Pb, whereby, during excretion of Pb from capillary to bile or by renal transtubular flow, a portion of the Pb enters the nucleus, becomes bound in the Pb–protein complex, and is no longer toxicologically active. At the same time, this has the effect of maintaining relatively low cytoplasmic Pb concentrations and of reducing toxicity to sensitive mitochondrial respiration and protein synthesis. In support of Goyer's protection hypothesis is the

finding of inclusion bodies at a lower dose of Pb than that producing signs and symptoms in rats, actually at a dose lower than any other renal effect of Pb. Because of this, it was proposed that inclusion bodies may be useful in diagnosing Pb poisoning from renal biopsy material.

Pb also affects *mitochondria* and *mitochondrial membranes*, for which it shows strong affinity through the several reactive and amino acid groups of the mitochondrial protein. This binding results in ultrastructural alterations consisting of swelling and dilution of matrix granules. Inclusion bodies do not occur in mitochondria, however, an intramitochondrial lamellar type crystalloid has been found in hepatic parenchymal cells of Pb-poisoned swine but the lamellar formations are not specific for Pb injury, occurring in a number of metabolic disturbances, diabetes mellitus, biliary obstruction, and others. A characteristic of Pb-injured mitochondria is impaired respiration and phosphorylative capacity, which is believed by Ulmer and Vallee (814) to result from specific combination of Pb with the dithiol bonds in the pyruvate dehydrogenase system, as determined from in vitro work.

Pb also affects *protein synthesis*, not only specifically as the most potent of metal inhibitors of globin synthesis, but more generally at the level of the ribosome; Ulmer and Vallee (814) showed that Pb disaggregates polyribosomes and prevents the incorporation of leucine in t-RNA in *E. coli*.

Up to this point, new information that has been published since the second edition of this book has included the relative amounts of absorption via inhalation and ingestion, relative amounts absorbed from urban air versus that from industrial exposure, and the effects of particle size on absorption, on body distribution, and at the cellular and subcellular levels. New information on excretion has been almost wholly concerned with the renal route (767, 809); only one definitive report has presented detailed information on fecal excretion, that by Klassen and Shoeman (815).

Using Pb acetate mixed with $^{210}Pb(NO_3)_2$ injected intravenously into rats, the investigators found that more than 20 percent of the dose (3 mg/kg) was excreted into the feces by 24 hr, and 9 percent more by 48 hr; corresponding urinary excretion was less than 2 percent of the dose. Over a 10-day period, 42 percent was excreted, 90 percent of which was in the feces. Because the Pb had been injected intravenously, biliary excretion was considered to be the main route. When measured at the three higher doses (1.0, 3.0, and 10 mg Pb/kg) the maximal route of excretion of Pb into the bile was about 1 μg(min)(kg). At the three lower doses (0.1, 0.3, 1.0 mg/kg), the concentration of Pb in the bile was between 40 and 100 times that in the plasma, owing largely to the higher concentrations (10- to 35-fold) in the liver than in the plasma, and partly to the higher concentration in the bile (three- to tenfold) than in the liver. The great affinity of Pb for these body constituents was shown by the fact that essentially no Pb was dialyzable from the bile, plasma, or liver, although affinity constants varied; Pb exhibited a fivefold greater affinity for liver than bile and 23-fold greater affinity for liver than plasma.

From the observation that a definite overall bile/plasma gradient exists, owing mostly to the large gradient from plasma to liver (20 to 30 for lower doses) and partly to the gradient from liver to bile (approx. 3), it would appear that Pb is excreted into the bile

of rats against a concentration gradient, and that an active transport mechanism exists, a mechanism that may exist for the excretion of metals from the liver.

Marked species differences in biliary excretion of Pb were observed, rabbits excreted Pb into the bile at a rate less than one-half, and dogs, at a rate less than one-fiftieth that of the rat. From the practical standpoint of applying these results to man the report does not make clear from which of the three species the data can best be extrapolated to man. But Haeger-Aronsen (816) quoted Clarkson and Kench [*Biochem. J.*, 69, 432 (1958)], who stated that "as in man, the major part of blood Pb in rabbits is bound to the erythrocytes. The normal urinary 24-hour excretion of ALA, kg is practically equal in rabbit and in man." However, the ZnPBC/ZnP in man is about 10, compared to 3 in rabbits, because of relatively larger amounts of plasma Zn in rabbits.

Of great interest for our understanding of the wide variations in susceptibility to Pb poisoning among workers has been the discovery of a Pb-binding protein in the red blood cells of Pb-exposed workers but not in those of normal, age-matched controls (816a). Because this protein serves as a detoxication mechanism by binding Pb in a nontoxic form, and individuals vary in their ability to synthesize this protein, it can be explained that some Pb workers sustain high blood Pb levels without frank signs of Pb poisoning and that tolerance to Pb occurs because some individuals have a genetically based capacity to induce the synthesis of greater amounts of this protein.

18.5.4 Mechanisms

A very considerable amount of information related to the mechanisms of action of inorganic Pb has appeared since the second edition of this book in 1962. Some of it has already been summarized under the specific topics of inclusion bodies, effects on mitochondria, and protein synthesis. The following represents extensions of our knowledge of previously recognized mechanisms of action of inorganic Pb.

Interference with Heme Synthesis. Research investigations in this area have been directed toward finding mechanistic explanations for increased concentrations of heme intermediates in clinical Pb poisoning and for structural and other functional defects in erythrocytes, as indicated by the altered discoid shape of the red cell and shortened red cell life-span, spherocytosis, decreased osmotic fragility (positive Coombs test), accelerated loss of K^+ from the red cell with altered Na^+/K^+ flux, and decreased Na^+/K^+-dependent ATPase.

The mechanism by which synthesis of the red cell pigment heme is inhibited by Pb is currently known to involve at least two enzymes, a cytoplasmic one, ALAD, active at the beginning of the synthesis, and a mitochondrial one, ferrochelatase, active at the end of the synthesis (817). Oddly, ALAD remains active in the circulating red cell, but the activity of ferrochelatase disappears as soon as the red cells reach circulation, along with the disappearance of the mitochondria. From the usual measurements of ALAD in peripheral blood and the substrate erythroporphyrin (EP) from changes in ferrochelatase activity, the direct action of Pb on circulating blood is evaluated very early,

however, the inhibition of medullary erythroblastic ferrochelatase from the action of Pb in the bone marrow is evidenced only indirectly by a late and gradual increase of EP, corresponding to the gradual maturation and slow release of mature red cells carrying protoporphyrin from the marrow to the circulating blood (816). This mechanistic information explains why EP concentrations may continue to rise after all other biologic measurements of Pb effects have improved or returned to normal, and long after environmental Pb exposure has ceased.

The Na^+/K^+-ATPase system has been studied because normal adenosine triphosphatase activity is indispensable for transporting Na^+ and K^+ across the red cell membrane and assuring its proper functioning. Past studies on Pb workers by Hernberg et al. (818) showed that the activity of the Na^+/K^+-ATPase in red cell membrane fragments was lower on the average than in unexposed controls. A comparison with five other commonly used indicators of excess Pb exposure indicated a sensitivity of response that suggested the system to be a more meaningful basis for health evaluation. The data must be examined further to determine the validity of this conclusion by matching, on an individual basis, the sensitivity of the system.

Secchi et al. (819) further tested the sensitivity of the system in the urban population of Milan, Italy not occupationally exposed to industrial Pb and found progressively lower *mean* values of Na^+/K^+-ATPase activity in red cell membranes and, conversely, higher mean values of blood Pb in traffic policemen and in people living near Pb smelters.

Cardona and Lessler (820) studied the time course of ATPase activity in Pb acetate poisoned rats and found a decrease in ATPase during the entire 24 weeks of study. Red cell catalase, however, rose initially, and then fell. It had been demonstrated previously (1976) that oscillations of mobile Pb in bone marrow can be caused by reversal of the inhibitory effects of Pb on mitochondrial oxidative phosphorylation by exogenous inorganic phosphate, which may be sufficient at times to prevent toxic effects of Pb on red cell precursors in the bone marrow. The investigators proposed this mechanism to explain the clinical findings in certain individuals chronically exposed to Pb who from time to time show a moderate or severe anemia and at other times are not anemic.

Nechay and Saunders (821) probed further into the inhibitory mechanism of Pb on the Na^+/K^+-ATPase system by studying the enzymatic activity in human kidney microsomes. They found that at 50 percent inhibition by Pb (5×10^{-8} *M*) the Pb acted directly at the site activated by Na^+, and indirectly by chelating ATP. The inhibition by Pb was reversible, however; the investigators considered Pb to have a certain specificity for the system, because it was at least tenfold more sensitive to Pb than Mg^{2+}.

Baxter et al. (822) by use of ^{13}C nuclear magnetic resonance spectroscopy, provided evidence for a Pb-ALA complex, with Pb acting most strongly with the carboxyl group and the alpha carbon atoms in ALA. The complexing was felt to be fairly specific for Pb because no complexing was observed with the acetates of Cd, Ag, Hg, or Zn.

Metal-Metal and Other Interactions. Haeger-Aronsen et al. (816), in a paper titled "Antagonistic Effect *in vivo* of Zn on Inhibition of ALAD by Pb," reviewed the

current knowledge of ALAD, pointing out its sulfhydryl (SH) content and its consequent inhibition by "heavy" metals at this site, such as Pb, Cu, Cd, and Hg, its reactivation by SH activators such as glutathione and cysteine, and the effect of Zn, which not only enhances ALAD activity, unlike most metals, but as has been found previously by Haeger-Aronsen, counteracts in vitro inhibition by Pb (and other metals). In extending this finding to the in vivo situation, Zn was found to have a strong activating effect on ALAD in rabbits, with the inhibitory effect of Pb almost eliminated. There was a close correlation between ALAD in red cells and Zn in plasma but none between ALAD and Zn in red cells, for reasons that were not clear at the time. Perhaps the answer lies in that the Zn is locked within the red cells, whereas the activity is on the outer surface of the cell membrane accessible to plasma Zn (author's note). The clinical upshot of the Zn-ALAD antagonism to Pb is that, because Zn often coexists with Pb in some industrial situations, urinary ALA measurements become less reliable.

Finelli et al. (823) had previously (1974) declared ALAD on good experimental evidence to be a Zn-dependent enzyme, at least in the rat red cell, but neither group of investigators was apparently aware of the other's findings, for no mutual references on the subject were listed.

Thomasino et al. (824) confirmed in one human case Haeger-Aronsen's findings in rabbits, which incidentally establishes a point of extrapolation from this animal species to man (see discussion of Klaassen and Shoeman in Section 18.5.3.). A Pb-intoxicated patient with extremely high blood Pb levels (260 μg/100 ml) but unexpectedly mild symptoms, during chelation therapy with CaNa edetate, had decreased red cell ALAD activity as blood Pb levels fell. After oral administration of $ZnSO_4$, a significant increase occurred in ALAD activity, and in vitro additions of $ZnCl_2$ to the patient's red cells resulted in reactivation of ALAD activity. The authors' interpretation of these findings was that "Zn is an important element in the ALAD system in man, and may play a protective role in Pb toxicity, and that Zn supplementation may be a useful adjunct to chelation therapy."

Petering and associates, who have long been investigating interactions of toxic industrial metals with essential trace elements, have found that Pb adversely affects Cu metabolism by (1) depressing ceruloplasmin levels to 44 percent of optimum at 0.5 percent Pb in the diet, (2) depressing plasma Cu levels to 22 percent, and (3) showing conversely that red cell-bound Pb was reduced by increasing plasma Cu levels (825). This is an important finding in that red cell Pb, usually relied on as a measure of Pb exposure, can be altered by changing plasma Cu levels. Rubino et al. (826) had previously found elevated red cell Cu in workers, and suggested that Cu might be a protective mechanism for reducing Pb toxicity.

Pb-Endotoxin Interaction. Of 14 metallic compounds tested for enhancing susceptibility to bacterial endotoxins, only $ScCl_3$ and $Th(NO_3)_4$ approximated Pb in enhancement. Cook et al. (827), after studying rat susceptibility to *S. enteriditis* by measuring several tests of liver function—bromsulfophthalein removal, elevated plasma glutamic-oxaloacetic transaminase, and glutamic-pyruvic transaminase—found that the underly-

ing mechanism was liver parenchymal cell dysfunction as one facet of the pathophysiology manifested by Pb interaction with endotoxin.

Glucose-6-Phosphate Deficiency and Pb Interaction. Because of several reports linking acute hemolytic crises to glucose-6-phosphate (G-6-PD) deficiency, or reduced glutathione, the chief factor in maintaining red cell integrity, McIntyre and Angle (828) investigated the effects of atmospheric Pb on blood Pb levels in black schoolchildren in Omaha, Nebraska. Black children deficient in G-6-PD (829) in both high and low Pb areas had significantly higher concentrations of red cell Pb and of whole blood Pb than nondeficient blacks of comparable age, sex, and census tract. The authors believed this to be the first demonstration of genetic susceptibility to an air pollutant.

Adaptation. A number of reports have been published since 1962 relating to the body's capacity to recover from and adapt to stresses on the hematopoietic system, in particular overexposure to Pb. Outstanding from the standpoint of industrial interest is the report of Haeger-Aronsen et al. on the regeneration of the ALAD enzyme in relation to blood Pb levels in battery workers after termination of Pb exposure (830). From a 16-month follow-up study of 31 male workers who had had from 0.3 to 45 years of exposure ranging from 0.1 to 1.75 mg Pb/m³ (av. 0.39 mg/m³) and who were well controlled with respect to being nonalcoholic and nonfish eating (methyl Hg), regeneration of red cell ALAD activity to about 30 percent of normal was apparent in 4 months, 50 percent in 7 months, about 60 percent in 11 months, and 80 percent in 16 months, the termination of the study. At the same blood Pb level, ALAD activity was slightly higher in those whose exposure had terminated than in those with continuing exposure, and regeneration rates were greater in those with shorter exposure with a lower blood Pb, at cessation of exposure. As far as mechanism is concerned, the most interesting finding was that ALAD regeneration occurred among the terminated workers in the face of increasing blood Pb levels, indicating to me that the Pb mobilized from bone (marrow) is in a toxicologically inactive or less active form than recently inhaled Pb.

From the temporal standpoint of ALAD regeneration from soluble Pb administration in animals, a Du Pont study of dogs fed a level of 100 ppm Pb acetate showed it required 15 or more weeks to return ALAD activity to normal and Pb levels to control group levels (831). Dogs that had received 500 to 1000 ppm Pb in their diet were predicted from regression analysis to require 18 or more months for recovery of ALAD, and years for blood Pb. Again, ALAD recovery during elevated blood Pb levels was noted.

Kniep et al. (832), in attempting to find answers to questions of the activity of the hematopoietic system in response to stresses placed on it by depletion of Fe stores (anemia) in the presence of high blood Pb levels in baboons, found hemoglobin and hematocrit regeneration rates following hemorrhage not to be affected by depressed ALAD activity. An additional significant finding was the increase in free erythroporphyrin following hemorrhage, which lagged the increase in hemoglobin by some 3 to 10 days, and severe anemia, 70 percent or less of normal, which resulted in blood Pb

increases of two- to threefold. The clinical significance of this is that reduction of Pb exposures should be made for those who are likely to be under the stress of Fe deficiency. These include women during the years of menstruation and workers with low hemoglobin or in frank Pb intoxication.

A case of adaptation, probably unique in the annals of human Pb exposure, certainly for its indisputable environmental and medical findings, has been revealed in a broad ecological and epidemiologic study of the inhabitants in the Meza River Valley, Yugoslavia (833). Here Pb has been mined and smelted since the time of Paracelsus (1490–1541), exposing the surrounding valley and its inhabitants to increasing amounts of Pb for centuries. Annual Pb production from one of the smelters using a Pb–Zn sulfide ore was 2500 tons by 1850, 22000 tons in 1970.

An environmental survey directed by Djuric (834) showed gross Pb contamination of the surface waters, ranging from 685 mg/liter in the effluent from the smelter to about 3 mg/liter 22 km away. Lead in the soil surrounding mining and smelting operations ranged from 200 to 25,000 mg/kg, and Pb in vegetables and dairy products ranged from about 2.5 ppm in peeled potatoes, bacon, and milk to 70 ppm in washed greens, and from 120 to 430 ppm in hay. High sulfur-containing vegetables, such as turnips, and onions, contained even larger amounts than low-sulfur vegetables. Bread contained more than 10 ppm compared with 0.5 ppm from uncontaminated areas. Biologic indicators of exposure of the inhabitants showed urinary ALA values in excess of 10 mg/liter with values as high as 44 mg/liter (normal values, less than 6 mg/liter). After filters had been installed at the smelter, ALA values decreased as did ALAD values 1 year after installation; ALAD values were below 70 units/cm³ red cells in 200 of 220 persons examined, and farmers showed decreased activity after Pb mobilization by CaEDTA, although Pb body burdens were estimated to be 10 times greater than for inhabitants elsewhere, not exposed to smelter dusts.

Despite this massive lifetime exposure from air, water, and food, no manifest signs or symptoms of Pb poisoning were seen on medical examination of adults (834a). To my knowledge no questions were raised about fertility or reproductive rates, nor were tests given to determine the effects on learning ability of the children.

Individual resistance (adaptation) to the effects of Pb exposure has been recognized for many years by industrial hygienists in the United States.

Garber and Wei (835) attempted to determine the characteristics of the adaptive mechanism by pretreating mice to small intraperitoneal doses of $Pb(NO_3)_2$, and found that only selected biologic parameters were unaltered. Whereas 4-day pretreatment with 20 mg/kg protected against acute lethal doses of 200 mg/kg, (100 percent survival vs. 30 percent untreated) such pretreatment did not attenuate the subacute toxicity induced in the mice by a 4 percent basic $PbCO_3$ diet. Protection was short-lived (4 to 5 days) and was not due to induction of greater activity of liver ALAD, the only definitive biologic indicator examined; indeed ALAD activity was further inhibited by the treatment.

Although Gerber and Wei were unable to pinpoint the mechanism by which protection developed, they pointed to Goyer's finding (813) of early Pb binding by nuclear inclusion bodies as one of the possible mechanisms. It is possible, however, that the

chronic resistance developed in some Pb workers and in the inhabitants of the Meza Valley, and the lack of such a finding by Garber and Wei, may be the result of their use of fast-acting soluble Pb compounds, rather than the slower-acting, more insoluble Pb oxides which constitute the chronic exposures (author's note).

18.5.5 Recent Occupational Health Experience and Epidemiology

Because several reports bearing on industrial experiences have already been discussed in connection with specific aspects of Pb poisoning, only very recent reports of some new aspect of health threat not before discussed or those extending past information are summarized here. Among the former is the recently recognized, often excessive, Pb exposure of policemen from indoor firing ranges (722); a similar survey of tollbooth operators in the Boston, Massachusetts area, however, failed to show Pb levels of health concern (721a).

During 1973 and 1974, the Industrial Hygiene Services Branch of NIOSH conducted environmental surveys of nine indoor firing ranges. Several sources of Pb dust and fume generation were found; one, from the bullet primer, produced 25 to 30 mg of the detonator, Pb styphenate (trinitroresorcinate, 35 percent), and Pb peroxide. Another source is Pb vaporization, resulting from the 2000°F temperature generated upon firing and fragmentation of the Pb bullet. The 18,000 to 20,000 psi pressure caused by the gas expansion at the high temperature blows dust and fume at right angles to the muzzle, creating turbulence around the breathing zone of the firer.

Of a total of 331 samples for airborne inorganic Pb (187 personal and 144 general air), concentrations of Pb range from 0.10 to 13.17 mg/m³ for general air samples, and from 0.01 to 34.50 mg/m³ for personal samples. From these findings, the investigators concluded that indoor firing ranges constitute "present health hazards from Pb poisoning" (due to improper ventilation control, in addition to noise hazards and danger from carbon monoxide and nitrogen oxides).

Three cases of mild Pb poisoning have been found in instructors at an indoor pistol range (722a).

Two papers have recently appeared providing clinical laboratory evidence that the biologic threshold limit of 80 μg Pb/100 ml blood is too high, at least for secondary Pb-smelting operations (836a, b). In the group of male workers with blood Pb (PbB) values less than 80 μg, only 11 of 45 (25 percent) had acceptable Zn protoporphyrin (ZPP) levels, and of those with PbB levels from 60 to 79 μg, 89 percent had ZPP levels above normal (i.e., > 100 μg/100 ml). Only those with PbB values less than 60 μg had normal ZPPs (24 controls, ZPPs < 100 μg). Measures of nerve conduction velocity, however, were uninformative in those with PbBs less than 80 μg. General symptoms consistent with Pb toxicity, that is, tiredness, fatigue, nervousness, sleeplessness, somnolence, and anxiety, were present to a significant extent for some of the symptoms in the below 80 μg PbB level group, and the correlation found between ZPP levels and these symptoms was felt to be of considerable practical and theoretical interest (836). In a similar study of secondary smelters, positive physical signs of Pb poisoning were found in 17 of 29 workers with PbB values of 60 μg or greater with one exception (836a).

An in-depth epidemiologic study of 7032 male workers in six Pb-production facilities (one primary smelter, two refineries, three recycling plants) with 2352 workers, and 10 battery plants with 4680 workers, who had been employed between 1946 and 1970, has been reported by Cooper and Gaffey (837). Of the smelters, 456 (19.4 percent) had been hired before 1945; of the battery workers, 2588 (55.3 percent), so that the age of the smelter workers was somewhat lower than that of the battery workers. A small number had been employed between 1900 and 1915 (11 smelters, 52 battery) and 138 (1.8 percent) had had 41 to 51 years of employment, but the majority had had between 1 and 10 years (60.4 percent of smelters, 47 percent of battery workers).

Fifteen of the 16 plants had biologic monitoring. The number of workers whose urinary Pb was equal to or greater than 200 μg/liter was relatively small (less than 7 percent for battery workers, but considerably larger for smelters (26 percent of those tested, 1286 and 989, respectively); blood Pb values ≥ 80 μg/100 ml were 9.5 percent for battery workers (126 of 1326 tests) and about 14 percent for smelters (74 of 537 tests). Because the number and type of test performed were considerably influenced by company policy, and thus artificially elevated, workers who showed evidence of overexposure were rechecked for verification or until values returned to satisfactory levels. Thus the biologic values are probably not truly representative of industry averages, and accordingly, correlation between biologic values and mortality was impossible.

Recognizing the nonrandomness and the heterogeneity of the populations under study, the following statistically based conclusions were made.

1. The *overall mortality* of smelter and battery workers combined showed a standardized mortality ratio (SMR) of 101 (107 for smelters, 99 for battery workers). A SMR of 101 (100 is par) was not considered to be indicative of no abnormal mortality risk in the Pb industry, for most employed populations, without adverse health factors at work, enjoy SMRs below 100. It was felt that racial distribution probably contributed to the inflation of the calculated SMR, for 25 percent of the smelters were nonwhite, compared with 11 percent generally; this group experiences a total age-corrected mortality of about 30 percent greater than whites. Certainly racial distribution is of even greater importance in elevating the SMRs of certain cause-specified disease rates.
2. The SMR for *all malignant neoplasms* was slightly elevated at the 5 percent confidence level for smelters (133), but not for battery workers (111), and was slightly higher (140) for smelters hired before 1945, but not for battery workers. Although the SMRs for respiratory, gastrointestinal, and urinary tract tumors were slightly elevated in smelters (148, 150, 178, respectively) they were not considered statistically significant, for in the first two organs, the SMRs for battery workers were below significance, and the number of cases of urinary tract tumors were too small in number to have statistical significance.
3. The overall SMRs for major *cardiovascular* and *renal diseases* were essentially the same as that of the general population, 96 for smelters and 101 for battery workers, but slightly higher for those hired before 1945 (107 and 103, respectively) and for *vascular lesions affecting the central nervous system*, there were no excess deaths from causes under the heading of "stroke." This lack of CNS involvement in U.S. Pb workers

differs from the findings of Dingwall-Fordyce and Lane (797), who found excess CNS deaths, cerebral hemorrhage and thrombosis, and arteriosclerosis, particularly in the heavily exposed group of British Pb battery workers.

4. As with the U.S. cardiovascular findings, the SMRs for *hypertensive heart disease* were not in significant excess (121 for smelters, 97 for battery workers), a finding consistent with clinical studies both in the United States and Europe. But for other hypertensive diseases, nephrosclerosis, uremia, or other, elevated SMRs of 389 for smelters and 223 for battery workers were found and for chronic and unspecified nephritis, the SMR was 264 for smelters and 175 for battery workers.

5. Because of reports that Pb had an adverse effect on immune responses (788), deaths from bacterial and viral diseases in addition to influenza and pneumonia were examined, although this was not a grouping usually employed in mortality analyses. No evidence was found in the group studied of increased risk of death from this group of infectious diseases.

6. It was the conclusion of the authors that, considering the high levels of exposure in the population examined and the small deviations from expected mortality, "one can be optimistic in predicting no detectable effect on the mortality of male adults from occupational exposures to Pb controlled in conformity to currently recommended environmental and biologic standards."

A follow-up study of the mortality of these workers during the period 1971 to 1975 was made by Cooper (837a). The population now consisted of 5490 members of the original group of 7032 who were alive December 31, 1970, of which 1967 were smelters and 3523, battery workers. Of the combined group, 491 deaths were reported, 127 (6.5 percent) smelters and 364 (10.4 percent) battery workers. But as noted in the first study, the battery plant population was an older group with a larger population of 65 years and older, a deficit in the 35 to 44 year olds, and with a below national average of blacks.

From the SMRs for overall mortality and malignant neoplasms there was a noticeable shift in the succeeding 5 years; the observed deaths from all causes combined were more than expected for both groups of workers (108 and 117 for smelters and battery workers), and now a slight deficit of malignant neoplasms in smelters (SMR, 89) and a slight excess in Pb battery plants (SMR, 136). In both groups there was a slight but nonsignificant excess of lung cancer (SMRs, 121 and 128, respectively). Because there was no consistent association between the incidence of cancer deaths and either length of employment or estimated exposures to Pb, and without smoking histories, it was felt that there was insufficient evidence to implicate Pb as a causal agent, although there are unanswered questions.

The reasoning is as follows. The major sites contributing to the slight excesses of malignancies were the respiratory tract and cancers of "other sites" (SMRs, adjusted, 128 and 316, respectively). At other sites, however, no primary sites were conspicuously evident, where among 18 tumors, one was melanoma, two were squamous cell carcinomas of the skin, two involved the brain, two the thyroid, one bone, one connective tissue, and nine cases were carcinomatosis or metastatic carcinomas of unknown origin.

Interesting in connection with the production of renal and central nervous system tumors in experimental animals was the single kidney tumor in the 491 certified deaths, and no tumors of the CNS in smelters, and only two in battery workers. Moreover, the pattern of deaths from malignant neoplasms in those hired before and after January 1, 1946, in those with more than 10 and 20 years of employment, and in those with high, medium, or low exposures, showed no pattern suggestive of an association with Pb.

Of the other major disease categories examined in the first report, the only significant difference found in the 1971 to 1975 study was a favorable shift from excess deaths in "other hypertensive diseases, chronic nephritis and renal sclerosis" to a reduction to only 3 of 491 deaths from these causes (2.93 expected).

18.5.6 Deleading Therapy

Chisolm has discussed in great detail the use of chelating agents in the treatment of adult Pb poisoning (838). He recommends a combination of BAL and CaEDTA (BAL, 2.5, CaEDTA, 8 mg/kg intramuscularly) in a 3- to 5-day course for symptomatic cases followed by oral D-penicillamine until urinary Pb goes below 500 μg/24 hr, or 2 months, whichever is less; for asymptomatic cases, where blood Pb is above 100 μg/100 ml, a similar 3- to 5-day treatment course is recommended with penicillamine. For asymptomatic cases, where blood Pb is between 80 and 100 μg, oral penicillamine is recommended for 1 or 2 months depending on clinical response. Oral penicillamine therapy, for which good response alone has been reported, has the advantage of home administration and avoids painful and oftentimes incompatible injections with BAL.

Westerman and Pfitzer et al. (839) had previously (1965) reported a comparative evaluation of the capacity of CaEDTA and D,L-penicillamine (1 to 2 g doses, respectively) to delead four asymptomatic patients and found that whereas pencillamine induced a twofold or greater excretion of Pb, CaEDTA caused a nearly tenfold excretion.

Molina-Ballesteros et al. (840) after trials in 42 confirmed cases of plumbism, have suggested D-penicillamine therapy in doses of 0.75 and 1.5 g/day to be an excellent procedure to confirm the diagnosis of Pb poisoning, and that urinary ALA is a good biologic indicator to evaluate the effectiveness of penicillamine therapy and to detect Pb poisoning.

18.5.7 Organic Pb

Quite a different order of toxicity is associated with organically bound Pb. As mentioned earlier, the acute response is the common form as contrasted with inorganic Pb. Of the 13 organic Pb compounds for which acute animal toxicity data are listed, "TEL and TML are of main industrial interest. However, owing to the phasing out of these fuel additives in the United States by 1980, and with no presently foreseen large industrial use, interest in their toxicology will be confined to those countries where its use will be continued. Accordingly, the toxicology of TEL and TML will be limited largely to readily available literature references where such information can be found.

Kehoe has discussed the health hazards associated with the handling of TEL, and pointed out that the measurement of urinary Pb is the best indicator of its absorption and hence its environmental control, in Volume 2 of the second edition of this book (724). TML at that time had not come into general use. When TML was later used as a new fuel additive in the United States, Schepers made a detailed study of the relative toxicities, absorption, signs and symptoms distribution, and comparative pathologies of both additives in the experimental rat (841). The details of the numerous findings in the 18-page report are not given here; only salient findings are mentioned.

1. The approximate acute, oral rat LD_{50} of TEL was 17 mg/kg and of for TML, 108, on the basis of Pb content, the values are 84 and 11.3 mg/kg, respectively, making TML 7.4 times less toxic than TEL. In view of the greater volatility of TML (Table 29.18.1) and hence greater hazard, the potential health hazards of the two are approximately the same. All rats survived multiple doses one-tenth the lethal administered for 21 days, however, suggesting a certain metabolic capacity for handling small, repeated doses of these highly toxic compounds, in the rat at least, where some tolerance was observed.

2. Toxic signs and symptoms differed somewhat between TEL and TML, although the same toxic manifestations of each occurred whether administered by oral, cutaneous, or inhalative routes. All rats on TEL displayed increasing irritability, hyperactivity, tremors, spasticity, arched backs, and prostration; those on TML showed marked hyperactivity, tremors, convulsions, and moderate weight loss beginning on the third day. Females were less affected. All symptoms had abated within 2 weeks for both compounds.

3. Although undesirable gaps in data on early tissue distribution sites occurred, data at 21 weeks after both single and multiple doses of TEL showed either no Pb, or slight traces in the brain, lungs, or spleen, trace amounts oddly being found at the lowest single dose administered (0.17 mg/kg) (data tabulation in error?). On the other hand, the livers contained large amounts (120 and 80 times that in the brains of the males and females, respectively) and the kidneys, appreciable quantities. In contrast, all six organs, including the testes, from TML-treated animals still had demonstrable Pb content, with the livers again showing 112 and 107 times as much as the brains of the males and females, respectively. On a concentration basis, more Pb was stored in the carcass, blood, muscle, and pancreas from TML than from TEL, with the muscle of female rats containing more Pb than that of males.

4. Damage to the nervous system from both delayed and cumulative action of TML was greater to all sections of the CNS, cerebral cortex, central ganglia (corpus striatum, thalamus, midbrain) cerebellum and pons, medulla oblongata and spinalis, nerves and appendages, than from TEL at all dosage regimens, with cellular reactions of all types being more severe. When no neuronal damage occurred from TEL at any level, damage from TML was seen at all levels. In general, the nervous damage from TML at the lower dose (1.08 mg/kg) was about the same as the higher dose (17 mg/kg of TEL).

For details of histological changes in other organs, the original article (841) should be consulted.

The mechanism of action of tetraalkyl Pb compounds had been elucidated earlier by Cremer and Callaway (842) in studying the toxicologic actions of tetra, trimethyl, and propyl Pb compounds by inhalation in experimental animals. By this route they found that (1) TML had approximately one-tenth the acute toxicity for rats as TEL, by the oral route, TML was about 4.3 times less toxic than TriML. (2) The primary site of action for both tetra and trialkyl Pb compounds was the CNS. (3) Tetra derivatives are converted in vivo by the liver to the trialkyl forms. (4) From the demonstration that trialkyl derivatives inhibited the oxidation of glucose in rat brain cortex 100 times faster than the tetraalkyl derivatives and that the trialkyl forms were more toxic than the tetra, the probable mode of action is the reduction of the tetra to the tri form, at the same time explaining the low toxicity of the tetra forms.

Further elucidation of the mechanism of alkyl Pb on the CNS has been revealed by Niklowitz (843) through electron microscopy in rabbits acutely poisoned with TEL. TEL produced neurofibrillary changes in hydropic degenerating pyramidal cells of the frontal cortex and hippocampus, which ultrastructurally are tangled bundles of straight, smooth tubules of great length, 200 Å in diameter, which on occasion were shorter segments of 800 Å tubules. The significance of these findings is their similarity to Alzheimer's disease, a presenile dementia, which resembled the CNS changes seen 42 years after childhood encephalopathy.

Finally, there is therapy of alkyl Pb intoxication, which carries a mortality rate of about 20 percent. For this condition chelating agents are not used, but heavy and prolonged sedation with short-acting barbiturates under hospital supervision, according to Chisolm (838). Fluid and electrolyte balance must be maintained, which often proves difficult because of the patient's hyperactivity. Convalescence can be prolonged and punctuated by recurrent, irrational behavior.

18.6 Hygienic Standards

Exposure to inorganic and organic Pb has been controlled over the years by engineering (environmental) and medical (biologic) means, with the latter being solely used for organic Pb control and recently the method of choice for inorganic Pb, for the reason that biologic measurements permit evaluation on an individual basis of not only their exposure but of response to that exposure.

The Threshold Limits Committee of the American Conference of Governmental Industrial Hygienists has documented the many changes that have occurred in the last 50 years in the industrial air standard for inorganic Pb (844). An environmental limit of 0.15 mg Pb/m³ of air was considered to be appropriate value in the 1930s and 1940s as a result of a recommendation of a 1928 U.S. Public Health Service survey of storage battery workers published in 1933. This value was generally accepted until 1957, when the TLV Committee raised the TLV to 0.20 mg/m³, based in part on Elkins' data showing that exposures at 0.20 mg/m³ would result in a urinary excretion value of 0.20 mg Pb/liter, a value considered to be the upper limit of safety. In 1971, the Committee recommended reducing the limit to its former value of 0.15 mg/m³ on the basis of the recommendations of the 1968 Amsterdam meeting of the International Subcommittee on

Occupational Health of the Permanent Commission, and a study by Williams et al. of British battery workers in whom a high correlation was found between air levels and blood and urine Pb values, and urinary coproporphyrins and ALA. For each correlated parameter, however, the upper 95 percent confidence limit exceeded the safe limits of 0.20 mg m^3 but approximates it when the limit is 0.15 mg/m^3.

Nevertheless the current workroom air standard established under the OSHA act of 1970 published in the *Federal Register*, August 13, 1971, is 0.2 mg/m^3. This is a time-weighted average for an 8-hr day and 40-hr work week, and was based on the inorganic Pb limit recommended by the American National Standards Institute, which in turn based it on the TLV Committee's recommendation in 1968.

On October 3, 1975 OSHA published in the *Federal Register* of that date a proposal to lower the occupational standard for inorganic Pb to 0.10 mg Pb/m^3. The reduction was proposed "to provide significant protection against the effects, clinical or subclinical, and the mild symptoms which may occur at blood Pb levels below 80 μg/100 g." Formal adoption of this proposal has yet to be made, but it has the sponsorship of NIOSH.

Regarding biologic standards, no biologic limit of any of the several parameters has been made officially, but industrial hygienists have generally agreed on 70 or 80 μg Pb/100 ml blood as a satisfactory control limit. Reduction to 70 μg from the long-standing Kehoe limit of 80 μg was recommended at the international Amsterdam conference on the basis that blood Pb determinations vary by at least ±10 percent, and hence 80 μg, which is a borderline limit, could exceed safe limits. Since the OSHA issuance, NIOSH has proposed a blood Pb limit of 40 μg/100 ml for women of childbearing age, who are increasingly entering the work force.

As previously mentioned, biologic standards have been found to offer the best means of controlling exposures to the alkyl Pb compounds, TEL and TML. Here urinary Pb levels have proved best. The limit for TEL recommended by the TLV Committee is 0.1 mg Pb/m^3 and for TML, 0.15 mg Pb/m^3. Both limits have been documented (844).

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