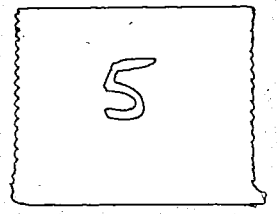
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Work Plan

(5)

Supplemental Site Investigation

Smith Corona Corporation
Cortlandville, New York

November 1988

O'BRIEN & GERE

WORK PLAN

SUPPLEMENTAL SITE INVESTIGATION

SMITH CORONA CORPORATION

CORTLANDVILLE, NEW YORK

MAY 1988

O'BRIEN & GERE ENGINEERS, INC.
1304 BUCKLEY ROAD
SYRACUSE, NEW YORK 13221

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SECTION 1 - INTRODUCTION

1.01 Introduction

A site investigation has evaluated the ground water flow, ground water quality and site soil quality at the Smith Corona Corporation (SCC) site in Cortlandville, New York. The results of the site investigation and associated tramp oil tank and surface soil removal were submitted to the New York State Department of Environmental Conservation (NYSDEC) on October 28, 1987 during settlement negotiations. A draft remedial work plan was submitted to the New York State Department of Environmental Conservation (NYSDEC) on December 14, 1987. This document described in detail the remedial alternatives proposed at the site. The NYSDEC, on December 14, 1987, in their Documents 1 and 2 presented comments on the site investigation to which Smith Corona responded on February 2, 1988.

Settlement negotiations resulted in additional investigatory work being performed and proposed. The agreed to Target Compound List (TCL) sampling of four on-site monitoring wells was performed on March 17 and 18, 1988. This work plan presents the limited supplemental investigatory program as agreed upon with the NYSDEC. The supplemental program includes the following tasks.

Task 1 Installation of Additional Ground Water Monitoring Wells

Task 2 Vertical Plume Delineation

Task 3 Surface Soil Sampling

Task 4 Soil Borings

Task 5 TCL/CLP Sampling

Task 6 Report Preparation

The site-specific Quality Assurance Project Plan (QAPP) which was prepared for field activities is included as Attachment A.

All personnel working on site, as part of this supplemental investigation shall comply with the regulation under the Occupational Safety and Health Regulations for Hazardous Waste Operations 29 CFR 1920.120. For this reason, a Health and Safety Plan which addresses proper safety procedures for various types of activities on site is presented as Attachment B.

SECTION 2 FIELD INVESTIGATION

2.01 Task 1: Additional Monitoring Wells

Six additional ground water monitoring wells will be installed as part of the limited supplemental investigation. Well installation procedures are delineated in Attachment A. Proposed well locations are presented on Figure 1 however actual locations will be determined in the field with concurrence by the NYSDEC field representative. All newly installed monitoring wells will be developed according to the protocols presented in Attachment A. All drilling equipment which comes into contact with soils or ground water will be steamed cleaned between well locations to volatilize any organic compounds according to the protocols presented in Attachment A. In addition, the monitoring wells will be surveyed with reference to offsite well CT-22, which has an assigned top of casing elevation of 1190 ft msl.

Ground water samples and ground water elevations will be collected from all onsite monitoring wells on one occasion after the installation of the new wells. The wells will be sampled according to the protocols presented in Attachment A.

2.01.1 Perimeter Monitoring Wells

A new, deeper, monitoring well (MW-1D) will be installed adjacent to existing well MW-1. The purpose of installing this well is to evaluate ground water chemistry, geology, and hydrogeology at various depths in the aquifer at this location. The addition of this well will complete the downgradient site perimeter monitoring network. The 20 ft. well screen will be installed immediately

above the first encountered confining layer. Split spoon samples will be collected to verify that the confining layer is at least 5 ft. thick.

Monitoring Wells MW-10s, and MW-10d will be installed, as requested by the NYSDEC, between existing monitoring wells MW-1 and MW-2 locations. The purpose of installing these wells is to further evaluate the ground water chemistry and hydrogeology in the area between wells MW-1 and MW-2. The exact location of the well will be decided upon in the field due to the presence of underground utilities, and the steep incline of the area. The supervising hydrogeologist, drilling contractor, and NYSDEC representatives collectively will locate the well. The location of the 20 ft. screen interval for the deep well will be selected based upon the results of the vertical plume delineation, Task 2. The shallow well will be installed to screen the upper portion of the water table.

2.01.2 Interior Monitoring Wells

MW-11 will be installed at the location of the former tramp oil tank to further evaluate deep soil quality and to define the bottom of the aquifer. During the previous investigation, it was not possible to perform a boring at this location, as the trap oil tank was still in the ground. The boring will be completed to the first identified confining layer. Split spoon samples will be collected at 5 foot intervals. Soil samples will be placed in a 40 ml glass vial with a teflon coated screw cap. A portion of each sample will also be placed in a glass jar for field screening. All split spoon

sampling equipment will be cleaned between samples using a clean water rinse, followed by a methanol rinse and a final control water rinse. All split spoon samples in the jars will be field screened using a photoionization detector (PID) and draeger tubes specific to trichloroethene (TCE). Up to fifteen (15) selected split spoon soil samples will be analyzed for volatile organics identified using EPA methods 601 and 602. A 20 ft. length of screen will be installed at the top of the first encountered confining layer.

Monitoring wells MW-12S, and MW-12D will be located between the former tramp oil tank and the proposed location of the recovery well. The shallow well will be set at a depth to correspond to the upper portion of the water table, and the deep well will be installed at the top of the first confining zone. Both wells will use 20 ft. well screens. These wells will serve as an intermediate point for sampling between the site interior and the proposed recovery well location.

2.02 Task 2: Vertical Plume Delineation

The NYSDEC (document 1) expressed the concern that contaminants in the perimeter ground water are isolated in discrete zones within the aquifer rather than being dispersed throughout the aquifer although this concern is not consistent with site data, it will be addressed using packer tests.

Packer tests will be conducted at monitoring wells MW-1S, MW-1D, MW-2S, and MW-2D to determine whether there are any discrete zones of contaminant transport within the aquifer. Ground water samples will be collected at five foot intervals throughout the screened interval of

each monitoring well using a Keck SP-81 submersible pump equipped with an inflatable top packer. Due to the coarse permeable nature of the materials screened in the monitoring well it is likely that the low pumping rate of the submersible pump (about 1 gpm) will not induce any significant vertical flow and should allow for discrete zones within the well to sampled. The samples will be collected directly from the pump and be analyzed for the volatile organics identified using EPA method 601.

Specific details concerning the procedures to be used are presented in Attachment A.

2.03 Task 3: Soil Sampling

Up to fifteen (15) surface soil samples will be randomly collected around the perimeter of the previous excavated areas to confirm the efficiency of soils remediation. In addition, ten (10) surface soil samples will be collected along traverses near the area west of the material handling area. The general areas to be sampled are presented on Figure 1. Actual sample locations will be identified in the field with concurrence with the NYSDEC field representatives.

The soil samples will be collected using a trowel or chisel as a composite sample from a depth of zero to six inches below the ground surface. The samples will be analyzed for volatile organics identified using EPA methods 601 and 602. Portions of the collected soil samples will be placed in two 40 ml vials and placed on ice for transport to the laboratory. Strict chain of custody procedures will be adhered to for the 40 ml vial samples throughout the sampling program.

Another portion of each sample will be placed into a glass jar, covered with tin foil and screw top and allowed to reach ambient temperature. The jarred soil samples will be field screened using an photoionization detector (PID) and draeger tubes specific to trichloroethene (TCE).

2.04 Task 4: Subsurface Soil Sampling

Three test borings will be completed in the vicinity of the stained asphalt pavement around Building 6. The borings will be completed using conventional hollow stem auger drilling methods to a depth of 10 feet below the ground surface. Split spoon soil samples will be collected continuously according to ASTM D Method 1586-84. Soils collected from all boring at a depth of 6 inches to 12 inches will be composited in appropriate containers for one full Target Compound List (TCL) analyses. As agreed to by the NYSDEC, NYS Department of Health and the NYS Attorney General's office any polynuclear aromatic hydrocarbon compounds found during the analyses will be attributed to the overlying pavement and not considered indicative of contamination.

The remaining soil samples will placed in appropriate containers and stored in a refrigerator for a period of 6 months should further laboratory analyses be required. Field screening of the samples for VOCs will be conducted in a similar manner as surface soil samples. Strict chain of custody procedures will be adhered to throughout the sampling and analytical program. Appropriate documentation will be followed.

2.05 Task 5: TCL/CLP SAMPLING

Upon completion of the proposed ground water recovery well one ground water sample will be collected from that well and subjected to for full Target Compound List (TCL) analyses. Contract Laboratory Protocols (CLP) will be followed for the analysis. The purpose of collecting the sample is to verify that contaminants other than volatile organics are not present in the well discharge. A list of all the parameters to be analyzed are presented in Attachment A. The sample will be collected after the installation of the recovery well pump.

2.06 Task 6: Report Preparation

Following the completion of the above work tasks a report addendum will be prepared to include all new data collected in the form of boring logs, analytical data and necessary maps. The results and conclusions presented in the investigation report will be revised and modifications will be made if necessary. In addition, revisions to the Remedial Work Plan will be made if necessary.

Figures

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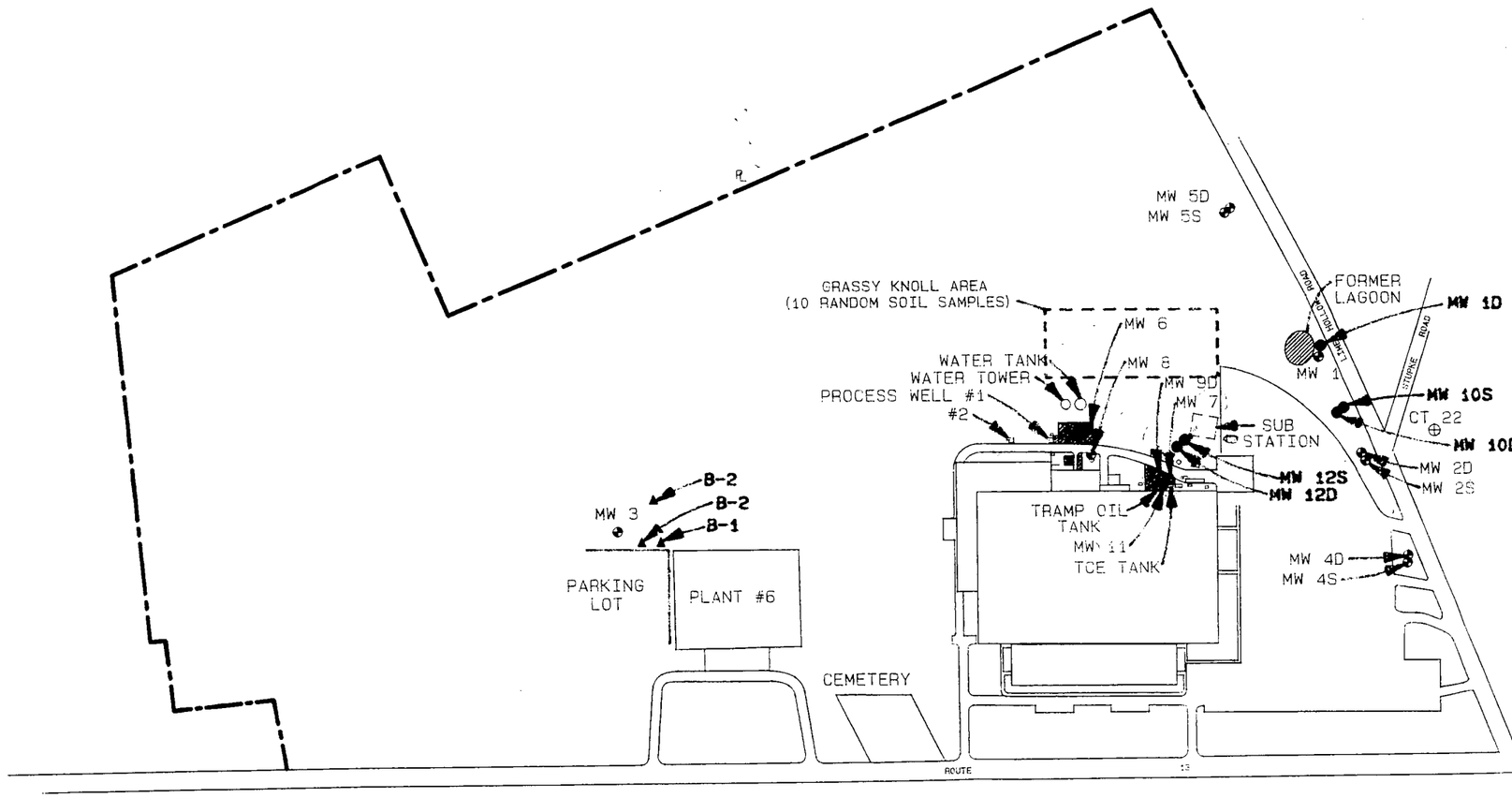
FIGURE 1

SMITH CORONA CORPORATION
CORTLANDVILLE, NEW YORK

PROPOSED MONITORING WELL, TEST BORING AND SURFACE SOIL SAMPLE LOCATIONS



LEGEND

- EXISTING USGS OBSERVATION WELL
- EXISTING MONITORING WELLS
- PROPERTY LINE
- PROPOSED MONITORING WELL LOCATION
- PROPOSED TEST BORING LOCATION
- AREAS OF PREVIOUS EXCAVATION

0 400 800

SCALE IN FEET

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Attachments

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ATTACHMENT A - QUALITY ASSURANCE PROJECT PLAN

The objective of this Quality Assurance Project Plan (QAPP) is to document the specific procedures and practices that will be used in the field investigation of the site.

The QAPP describes the following protocols and documentation:

Task 1:

- Overburden Drilling/Monitoring Well Completion
- Well Development
- Decontamination
- Ground Water Sampling Protocol

Task 2: Vertical Plume Delineation Protocol

Task 3: Surface Soil Sampling Protocol

Task 4: Test Boring Protocol

Task 1: Additional Ground Water Monitoring Well

Task 1 of the Work Plan describes the installation of six (6) monitoring wells. The monitoring wells will be installed and sampled according to the following protocols.

OVERBURDEN DRILLING/MONITORING WELL COMPLETION

I. Drilling Procedures

The drilling method shall be completed using odex or air rotary to a depth specified by the supervising hydrogeologist. The minimum outside diameter of casing to be utilized for 2 inch wells shall be 4 inches.

Samples of the subsurface materials at wells MW-1D, MW-10D MW-11 and MW-12D shall be collected at a minimum of every five (5) feet or a change in the material or at the discretion of the supervising hydrogeologist. The sampling method employed shall be ASTM Method D-1586-84/split barrel sampling using a 2 ft long, 2 inch outside diameter split-spoon sampler. Samples will be collected for the purpose of soil classifications only with the exception of well MW-11.

II. Monitoring Well Completion

All monitoring wells will be constructed of twenty (20) feet of 0.020 inch slot, 2 inch I.D., PVC well screen and riser casing. The riser casing will extend from the screened interval to 2-3 ft above existing grade. Other materials utilized for completion will be bentonite, Portland Cement and a protective steel locking cap with locks.

The well installation method for monitoring wells installed within unconsolidated sediments shall be to place the screen and riser assembly into the casing once the screen interval has been selected. At that time the casing will be withdrawn to allow the native aquifer material to collapse 2-5 ft above the top of the screen. A minimum 2 ft bentonite seal will then be placed above the sand pack. Cement/Bentonite grout

will then be added via tremmie pipe around the drill casing until the entire unsaturated thickness has been sufficiently sealed off from horizontal and/or vertical flow above the screened interval.

A vented protective steel cover shall be placed over the standpipe secured by a lock. Cement shall then be placed laterally at least one foot (1 ft) in all directions from the protective casing and shall slope gently away to drain water away from the well.

The supervising hydrogeologist is responsible for recording the exact well details and measurements. Both the supervising hydrogeologist and drilling contractor are responsible for tabulating all well materials used.

A field survey control program will be conducted using standard instrumentation survey to document well location, ground, inner and outer casing elevations to 0.01 ft with respect to well CT-22.

WELL DEVELOPMENT PROTOCOL

All monitoring wells will be developed or cleared of fine grained materials and sediments that have settled in or around the well screen during installation. The well development will assure that the well screen is transmitting representative portions of ground water. The development will be by one of two methods, pumping or bailing ground water. All water removed from the wells will be allowed to drain onto the ground surface.

Ground water will be pumped from the bottom of the well using a submersible pump, bailer or centrifugal pump. New polypropylene rope will be used for each well. If a submersible pump or centrifugal pump is used clean PVC hose will be used for each well.

All equipment that is placed down the well shall be cleaned using methanol swabbing and a water rinse.

GROUND WATER SAMPLING PROTOCOL

Sampling Procedures (Bailer)

1. Identify the well and record the location on the Ground Water Sampling Field Log.
2. Put on a pair of clean gloves or new disposable gloves. The gloves shall be cleaned using Methanol followed by a clean water rinse.
3. Cut a slit in the center of plastic sheet, and slip it over the well creating a clean surface onto which the sampling equipment can be positioned. In the event that it is not practical to use plastic sheeting (winter), the new polypropylene rope will be kept from touching the ground surface.
4. Using a clean electronic well probe, measure the depth to the water table and the bottom of the well (if not previously measured) from the top of the protective casing or a surveyors mark. The electronic well probe will be cleaned using Methanol.
5. Compute the volume of water in the well, and record this volume on the Ground Water Sampling Field Log.
6. Attach enough new polypropylene rope to a clean bottom loading stainless steel bailer to reach the bottom of the well. The bailer shall be decontaminated using Methanol and organic free or distilled water. (All decontamination fluids will be allowed to drain onto the ground surface).
7. Lower the bailer slowly into the well making certain to submerge it only far enough to fill one-half full. The purpose of this is to recover for observation any oil film, if one is present on the water table.

8. Pull the bailer out of the well keeping the polypropylene rope on the plastic sheeting or off the ground. Empty the ground water from the bailer into a clean glass container and records its appearance on the Ground Water Sampling Field Log.

9. Initiate bailing the well from the top of the water column making certain to keep the polypropylene rope on the plastic sheeting or off the ground surface. All ground water should be poured from the bailer into a graduated pail to measure the quantity of water removed from the well. This water may then be discharged to ground surface.

10. Continue bailing the well from the top of the water column until 3 well volumes have been removed, or until the well is bailed dry. If the well is bailed dry, allow sufficient time for the well to recover before proceeding with the next step. Ph and specific conductance readings shall be taken prior to the collection of the actual sample. Record this information on the Ground Water Sampling Field Log.

11. Remove the sampling bottles from their transport containers, and prepare the bottles for receiving samples. Inspect all labels to insure proper sample identification. Arrange the sampling containers to allow for convenient filling.

12. To minimize agitation of the water in the well, initiate sampling by lowering the bottom loading stainless steel bailer slowly into the well making certain to submerge it only to the middle of the screened interval. The vials labelled "volatiles" should be filled from one bailer then securely capped. The collected samples will be placed in two-40 ml vials and preserved with hydrochloric acid

to pH less than 2. The vial should be turned upside down, and checked for air bubbles. If properly filled there should be no visible air bubbles. Place each container in a cooler and chill to 4°C. Samples must not be allowed to freeze.

13. After the last sample has been collected record the physical appearance of the ground water observed during sampling on the Ground Water Sampling Log.
14. Begin Chain of Custody Record.
15. Replace the well cap, and lock the protection assembly before leaving the well location.
16. Place the polypropylene rope, gloves, etc into a plastic bag for disposal.

Sampling Procedures (Purge by Pump)

1. Identify the well and record the location on the Ground Water Sampling Field Log (copy attached).
2. Put on a pair of clean gloves or new disposable gloves. The gloves shall be cleaned using Methanol followed by a clean water rinse.
3. Cut a slit in the center of a plastic sheet, and slip it over the well creating a clean surface onto which the sampling equipment can be positioned. In the event that it is not practical to use plastic sheeting (winter), the new polypropylene rope will be kept from touching the ground surface.
4. Using a clean electronic well probe, measure the depth to the water table and the bottom of the well, (if not previously

measured) from the top of the protective casing or a surveyor's mark. The electronic well probe will be cleaned with methanol.

5. Compute the volume of water in the well, and record this volume on the Ground Water Sampling Field Log.

6. Attach enough new polypropylene rope to a clean bottom loading stainless steel bailer to reach the bottom of the well. The bailer shall be decontaminated using Methanol and organic free or distilled water (all decontamination fluids will be allowed to drain onto the ground surface).

7. Lower the bailer slowly into the well making certain to submerge it only far enough to fill one-half fill. The purpose of this is to recover for observation any oil film, if one is present on the water table.

8. Pull the bailer out of the well keeping the polypropylene rope on the plastic sheeting or off the ground. Empty the ground water from the bailer into a clean glass container and record its appearance on the Ground Water Sampling log.

9. Prepare the purge pump for operation. Lower the pump to below the water level pump the ground water into a graduated pail. Pumping should continue until sufficient well volumes have been removed or the well is pumped dry. pH and specific conductance readings shall be taken prior to the collection of the actual sample. If the well is pumped dry, allow sufficient time for the well to recover before proceeding with Step 10. Record this information on the Ground Water Sampling Field Log. The pump will be used only to evacuate

the monitoring well. A stainless steel bailer will be used to collect samples.

10. Remove the sampling bottles from their transport containers, and prepare the bottles for receiving samples. Inspect all labels to insure proper sample identification. Arrange the sampling containers to allow for convenient filling.

11. To minimize agitation of the water in the well, initiate sampling by lowering the bottom loading stainless steel bailer slowly into the well making certain to submerge it only to the middle of the screened interval. The vials labelled "volatiles" should be filled from one bailer then securely capped. The collected samples will be placed in two-40 ml vials and preserved with hydrochloric acid to pH less than 2. The vial should be turned upside down, and checked for air bubbles. If properly filled there should be no visible air bubbles. Place each container in a cooler and chill to 4°C. Samples must not be allowed to freeze.

12. After the last sample has been collected record the physical appearance of the ground water observed during sampling on the Ground Water Sampling Log.

13. Begin the Chain of Custody Record.

14. Clean the bailer by rinsing with distilled water, methanol then organic-free water. Store the clean bailer in a fresh plastic bag. Decontaminate the pump with dilute methanol rinse, followed by a clean water rinse using separate wash basins. Clean the pump by pumping potable water. Rinse outer surface with distilled water.

15. Replace the well cap, and lock the well protection assembly before leaving the well location.

16. Place the polypropylene rope, gloves, and plastic sheet into a plastic bag for disposal.

Task 2: Vertical Plume Delineation

Task 2 of the work plan describes the reasoning to test the vertical concentration of contaminants in wells MW-1S, MW-1D, MW-2S, and MW-2D.

The ground water samples for this task will be collected according to following protocol.

The following procedures will be used to obtain representative ground water samples using a Johnson-Keck SP-81 submersible pump equipped with an inflatable top packer.

1. Identify the well and record the location on the Ground Water Sampling Field Log.
2. Put on a pair of clean gloves or new disposable gloves. The gloves shall be cleaned using Methanol followed by a clean water rinse.
3. Cut a slit in the center of the plastic sheet, and slip it over the well creating a clean surface onto which the sampling equipment can be positioned. In the event that it is not practical to use plastic sheeting (Winter), the pump and hose will be kept from touching the ground surface.
4. Using a clean well probe, measure the depth to the water table and the bottom of the well (if not previously measured) from the top of the protective casing or surveyors mark. The electronic well probe will be cleaned with methanol.
5. Prepare the decontaminated submersible pump for operation. Decontamination consists of cleaning the necessary tubing internally and externally with a dilute methanol rinse followed by a clean water rinse using separate wash basins.

6. Lower the pump to five feet above the bottom of the well, inflate the packer, and pump the ground water into a graduated pail.

7. Pumping should continue until approximately 3 volumes have been removed. Ph and specific conductance readings shall be taken prior to the collection of the actual sample. Record this information on the Ground Water Sampling Field Log. All evacuated water will be allowed to drain onto the ground surface.

8. Remove the sampling bottles from their transport containers, and prepare the bottles for receiving samples. Inspect all labels to insure proper sample identification. Arrange the sampling containers to allow for convenient filling.

9. With submersible pump, fill each sample container. The collected sampling will be placed in a two-40 ml vials preserved with hydrochloric acid to a pH less than 2.

   The vials should be turned upside down and checked for air bubbles. If properly filled there should be no visible air bubbles. Place each container in a cooler and chill to 4°C. Samples must not be allowed to freeze.

10. After the sample has been collected, record the physical appearance of the ground water observed during sampling on the Ground Water Sampling Log.

11. Begin the Chain of Custody Record.

12. Deflate the packer; and raise pump an additional 5 feet, making sure that the pump is still below the top of the water column. If the pump is still below the top of the water column re-inflate the pack and go to step 7.

13. Following the completion of packer testing the entire saturated thickness of the well, the pump, packer and associated tools will be decontaminated. Decontamination will consist of a dilute methanol rinse, followed by a clean water rinse using separate wash basins.

14. Replace the well cap, and lock the well protection assembly before leaving the well location.

15. Place the gloves, etc. into a plastic bag for disposal.

Task 3: Surface Soil Samples

Task 3 of the Work Plan describes the approximate locations of 25 surface soils to be collected. The samples will be collected according to the following protocol.

Surface Soil Sampling Protocol:

The soil samples will be collected using a trowel or chisel and composited from a depth of zero to six inches below the ground surface analyzed according to EPA Methods 8010 and 8020. The collected soil samples will be placed in two 40 ml vials and placed on ice for transport to the laboratory. A portion of the sample will be placed into sediment jars, covered with tin foil and allowed to reach room temperature. Soil samples will be field screened with using a photoionization detector HNU Model PI-101 or equivalent and TCE draeger tubes. The chisel or trowel will be decontaminated between locations using methyl alcohol.

Strict chain of custody procedures will be adhered to throughout the sampling program:

Task 4: Subsurface Soil Sampling

Task 4 of the Work Plan describes the installation and approximate location of three (3) test borings to be located near Plant 6. The borings shall be advanced and sampled according to the following protocols.

1. Drilling/Sampling Procedures

Soil borings will be completed using conventional hollow stem auger drilling methods to a depth of ten (10) feet. The minimum I.D. of the augers shall be 3 1/4".

Soil samples shall be collected continuously in accordance with ASTM-D 1586. 1586/split barrel sampling using either a 2' long, 2" outside diameter split spoon sampler with a 140 lb hammer or a 3" outside diameter sampler with a 300 lb hammer.

All samples collected from 6" to 12" will be composited for one full HSL analyses in appropriate containers and immediately placed on ice. All other collected soil samples will be placed in 40 ml vials and placed on ice for storage in a refrigerator for a period of six months, should further laboratory analyses be required. Field screening will be conducted on each sample by placing a portion of the sample in sediment jars. The jars will be capped with aluminum foil and allowed to reach room temperature. The head space of the sediment jars will be monitored using a A-PID and draeger tubes specific to TCE.

To prevent cross contamination of soil samples, the split-spoons will be decontaminated between samples and the drilling equipment will be decontaminated between borings. All decontamination procedures will be in accordance with the decontamination protocols.

The soil borings will be backfilled upon completion. The backfilling procedure will be completed by placing drill cuttings and bentonite powder from the bottom of the boring to the ground surface.

Task 5: TCL/CLP Sampling

Following the installation and development of the proposed recovery well, ground water samples will be collected according to following protocols. ALL TCL analyses will be in accordance with New York State Department of Environmental Conservation Superfund and Contract Laboratory Protocols (January 1985).

Sampling Procedures

a. Turn on pump and allow the system to flush a minimum of three well volumes.

b. Reduce the water flow.

c. Tilt the sample bottles and collect the samples from the gently flowing stream. All samples collected will be placed on I-Chem prepared bottles:

   1. Volatiles: two 40 ml vials preserved with concentrated acid Hydrochloric acid to a pH less than 2.
   2. Semi volatiles: 2-Amber glass approximately 1/2 gallon jugs.
   3. Pesticides/PCB: 1 amber glass approximately 1/2 gallon jug.
   4. Analytes: 1 plastic 500 ml container preserved wtih nitric acid to pH less than 2.
   5. Cyanide: 1 plastic pint preserved with Sodium Hydroxide to a pH less than 12.

d. Properly preserve the sample for the analyses to be done as necessary.

e. Store sample in an insulated ice cooler at 4°C.

f. Record all pertinent information.

   a. Sample site and date.

   b. Sample identification.

   c. Chain of Custody forms.

DECONTAMINATION PROTOCOL

All drilling equipment and associated tools including augers, drill rods, sampling equipment, wrenches and any other equipment or tools that may have come in contact with contaminated materials shall be decontaminated. The decontamination procedure shall be to use a high pressure steam cleaner to remove soils and volatilize organics from the equipment.

The frequency of the decontamination will be determined by the supervising hydrogeologist. At a minimum, the decontamination will be performed between the wells and borings and at the completion of the drilling program prior to removing the equipment from the site. All decontamination fluid will be allowed to drain into the ground surface.

ATTACHMENT B

HEALTH AND SAFETY PLAN

SECTION 1 - INTRODUCTION

1.01 Health and Safety Plan

This document serves as the Health and Safety Plan for all field activities connected with the additional site investigation and remedial activities at the Smith Corona facility in Cortlandville, NY. As the result of previous on-site activities, areas of soil contamination have been excavated and removed from the site. However, residues of potentially hazardous materials remain on-site.

The primary potential work site hazard arises during exposure to exposed soils, due to the potential presence of halogenated solvents and materials potentially derived from them, including trichloroethylene (TCE), trans-1,2-dichloroethylene (t-1,2-DCE), vinyl chloride (VC), and 1,1,1-trichloroethane (TCA), as well as xylene, and ethylbenzene.

All activities involving disturbance of, or contact with, the soil must be conducted using appropriate personnel protection, as detailed in this Plan. This Plan is designed to address the minimum health and safety requirements and general procedures to be met by O'Brien & Gere employees and contractors during the field activities. The Health and Safety (H&S) Officer for any contractor is required to review this plan, and finalize logistical details and considerations prior to commencement of any contractor on-site activities. This plan provides details about the site, the work stages, associated hazards, the required safety equipment and applicable procedures for each stage of the work in order to clearly define the steps necessary to provide adequate protection for all personnel involved with the on-site work. All personnel (O'Brien & Gere Engineers, Inc. employees and

subcontractors, Smith Corona employees, and NYS Department of Environmental Conservation employees, all visitors, media, etc.) are required to adhere to the protocols and procedures in this plan while in the designated project work zones.

1.02 Project Work Plan Summary

The field investigation will generally involve the following activities:

1) soil sampling and installation of ground water monitoring wells and a pilot recovery well.
2) a sampling program to further define the extent of on-site contamination in ground water and soil.

SECTION 2 - SITE DESCRIPTION

2.01 Site Background

The Smith Corona facility is located on Route 13 in Cortlandville, New York, in a predominantly industrial/commercial zone. The facility is engaged in the manufacture of typewriters and related equipment. Several residences are located to the north and west of the facility. Several degreasing solvents have been detected in site soils and groundwater underlying the facility, as delineated in OBG (1987).

2.02 Weather

Prevailing winds are generally westerly. Work on-site is anticipated for all four seasons. Weather in the spring, summer and autumn in southern New York is generally moderate to warm (50-85°), while winter temperatures could be 0° to 30°, or lower, on occasion. The average annual total precipitation in Cortland is 40.66 inches and ranges from 2.81 (Jan) to 4.16 (July) (U.S. Department of Commerce 1974).

2.03 Designation of Project-Specific Areas

2.03.1 Work Zone

Work zones will be defined as actual sites of activity. Each work zone will be surrounded by a fifty foot wide buffer zone. For both administrative and safety reasons, the work zone should be delineated with markers, stakes and flagging tape, or other similar means to designate the area as one of limited access and usage. Access points will be precisely marked.

2.03.2 Decontamination Area

Decontamination areas will be designated on this site. Heavy equipment will be steam-cleaned at the site after use, and wash water will be allowed to drain onto the site surface soil. Disposable protective equipment (i.e. Tyveks, disposable gloves, and outer boots) will remain on-site after use and treated as waste material.

2.03.3 Health/Safety/Emergency

Facilities Materials for health, safety and first aid measures will be available on the site. Provisions will include extra protective clothing and equipment, first aid supplies, fire extinguishers (Type ABC), and emergency phone numbers.

SECTION 3 - WORK SITE HAZARDS

3.01 Hazardous Materials Present

Of the compounds determined to be present in site soil and groundwater (OBG 1987), several have been selected as primary materials of concern. These are vinyl chloride (VC), trichloroethylene (TCE), and tetrachloroethylene (perchloroethylene, PCE). These selections were made based on demonstrated (VC) and potential (TCE and PCE) carcinogenic properties of these materials in humans, their volatile nature which can potentially give rise to vapors from exposed soils, and the concentrations which may be present in the soil. In previous soil investigations, it was determined that VC was much less prevalent than the other materials detected and may have been adequately removed by the soil excavation. Therefore, the protective measures specified below focus on the most prevalent material present, TCE. These measures will be adequately protective for the more toxic VC if it is encountered in exposed soils. The potential for exposure to non-carcinogenic and less chronically toxic materials detected on site such as TCA, t-1,2-DCE, xylene, and ethylbenzene will be minimized by adherence to the appropriate health and safety measures associated with possible contact with potential carcinogens that are detailed in this Plan. Summaries of the toxicological properties of the site-related hazardous substances are provided in Appendix A. For the purposes of establishing appropriate protective monitoring and protective measures, the following are the permissible air exposure limits (PEL) established by the Occupational Safety and Health Administration (OSHA) and the

threshold limit values (TLV) recommended by the American Conference of Governmental Industrial Hygienists:

Vinyl Chloride-	5 ppm (TLV)
	1 ppm (PEL)
Trichloroethylene-	50 ppm (TLV)
	100 ppm (PEL)
Tetrachloroethylene-	50 ppm (TLV)
	100 ppm (PEL)

3.02 Physical Hazards Present

The work will involve the use of heavy equipment such as well-drilling rigs. Use of such equipment, and related activities, presents hazards common to general construction situations including falling, being struck by the equipment, head injuries, and noise. These are most directly addressed with the use of the proper personal protective clothing and equipment such as hard hats, steel-toe boots and coveralls, and common sense, which includes compliance with safety protocols and open and effective communication between all personnel on site.

SECTION 4 - PROTECTIVE MEASURES

Operations on-site not involving exposure or contact with contaminated soils will require the use of D protection. Level C contact protection for all activities involving the movement and/or disturbance of known contaminants site soils. Level C respiratory protection will be also be available in the event that an upgrade in personal protection is required, as indicated by air monitoring. Level C and D protection are as delineated below.

4.01 Contact Protection - Level C

The following protective clothing and equipment are required for all on-site personnel within the designated work zones during soil boring and soil sampling, and well installation within areas of known soil contamination.

- fully hooded Tyvek (or similar brand) suit over long-sleeve, long-pant work clothes
- inner gloves (medical gloves)
- rubber or latex outer gloves
- rubber overboots over steel-toe boots
- tape to seal the joints between the suit, and gloves and boots
- hard hats

4.02 Respiratory Protection - Level C

Full face respirators, equipped with high-efficiency dust/mist/particulate, organic vapor combination cartridges are to be available to all personnel, including equipment operators, during all activities involving movement and/or disturbance of soils. If, as specified below,

air monitoring indicates the presence of unacceptable air contaminant levels, respiratory protection will be required.

A safety station containing a First Aid Kit, Drinking Water and other appropriate equipment will be placed as close as possible to the work zone. A place where employees may break in a clean zone by washing their boots, removing their gloves and respirator for the purpose of receiving water, monitoring vital signs and rest periods will also be established. Employees must wash their hands before eating, drinking, or smoking. No facial hair which could interfere with the proper fit and seal of the respirator is permitted on on-site workers. All personnel must be fit-tested prior to using the respirators for on-site work. Also, all personnel must have medical approval for the use of respiratory protective equipment prior to using such equipment on-site. Personnel must be safety trained in accordance with 29 CFR 1910.120. Those workers who require corrective lenses for accurate vision must be fitted with a full face respirator which can have glasses clipped inside the mask. No one is permitted to wear a standard full face respirator over regular eyeglasses. No one is permitted to wear contact lenses while in the work area.

4.03 Eye Protection

Eye protection will be provided for with the full face respirator. For the work site activities which do not require a respirator, all personnel must wear protective eye-glasses with side shields or goggles while on-site.

4.04 Protection - Level D

Barring the detection of unforeseen hazards during the field investigation, all other field activities will be conducted using level D protection with air monitoring as specified below. Requirements for level D protection include:

- Full-face/half-face air-purifying respirator equipped with appropriate canisters or cartridge must be available for use; and all potential users trained and medically approved for such use.
- Long sleeve work shirt and long pants (work pants or jeans)
- Leather boots
- Safety glasses or chemical splash goggles

Options as required:

1. Work gloves
2. Disposable outer boots
3. Hard hat

SECTION 5 - HEALTH AND SAFETY PROGRAMS AND PROCEDURES

5.01 On-site Organization

Smith-Corona	Eric Cleveland Cortlandville, NY (607) 753-6011
Project Manager (O'Brien & Gere):	Guy Swenson Syracuse, NY (315) 451-4700
Health & Safety Officers (O'Brien & Gere):	Swiatoslav W. Kaczmar, Ph.D., C.I.H. Ruth Wegmann Syracuse, NY (315) 451-4700
New York State Department of Environmental Conservation Cortland Office	(607) 753-3095

5.02 Training Program

As a registered New York site, the Smith-Corona Cortlandville site falls under the jurisdiction of the OSHA standard contained in 29 CFR 1910.120, regarding the health and safety of on-site workers. All project personnel, including employees of Smith-Corona, O'Brien & Gere Engineers, Inc., contractors and subcontractors must demonstrate participation in a 40-hour health and safety training program, which includes use of respiratory protective equipment and protective clothing, decontamination, on-site procedures, and emergency response measures. Any personnel not meeting these requirements, as well as those detailed elsewhere in this plan, are not permitted entry into the Work Zone.

5.03 Monitoring Programs

The previous site activities investigation revealed detectable levels of volatile compounds to which site workers may be exposed, including TCE, as shown in the data in Appendix B. Therefore, air monitoring will be conducted during all site activities with an photoionization organic vapor detector HNU Model (PL-101) or equivalent with a 10.2 eV (122 nm) lamp. During activities requiring only level D respiratory protection, should readings of 5 ppm (chosen on the basis of 10% of the PEL for TCE and in consideration of the 5 ppm TLV for vinyl chloride, potentially present) or greater be observed, personal protective equipment will be upgraded to Level C, including the required use of a respirator equipped with an organic vapor cartridge. The HNU will be calibrated monthly according to manufacturer's instruction. Monitoring for heat stress will be maintained for all personnel dressed in Level C protection. This monitoring will consist of periodic measurements of oral temperature, body weight, and/or heart rate. The following criteria will be used:

- if the heart rate exceeds 110 beats per minute at the beginning of a rest period, shorten the next work cycle by one-third.
- if oral temperature exceeds 99.6°F shorten the next work cycle by one-third.
- if oral temperature exceeds 100.6°F, the worker should not be permitted to continue wearing the protective clothing.
- body water loss (based on weight) should not exceed 1.5% total body weight loss in a work day.

5.04 Decontamination Procedures

5.04.1 Equipment

Equipment decontamination will consist of a steam genny rinse of all pieces of wettable field equipment. Wash water will be disposed of on-site.

5.04.2 Personnel

Decontamination for personnel will take place in two stages. Initial decontamination (washing and disposal of contaminated garments) will take place immediately adjacent to the work site.

5.05 Entry and Exit Procedures

In order to control the potential for the spread of the contaminated materials, entry and exit into the work zone must be controlled. Entry procedures will be as follows:

1. All personnel must dress in required safety clothing specified for each task in Section 4 above.
2. All personnel must notify the H&S Officer of intended operations.
3. The H&S Officer will review team personnel for appropriate personal protective equipment and clothing.
4. Entry time and name must be logged in.
5. Team proceeds through the designated Entry and Exit point. Exit procedures will be as follows:
   a. All personnel must exit through the designated Entry and Exit point.
   b. All personnel must go through appropriate decontamination procedures, as specified in Section 5.04.2.

c. All personnel must log out and record exit time.

d. No one will be allowed on-site alone.

5.06 Off-site Health and Safety Concerns

In addition to providing for the health and safety of all on-site personnel, it is also imperative to consider off-site residents in the immediate area. The study area is in a populated area. Therefore, in the unlikely event that weather conditions are such that any activities create excessive dust, the area will be wetted to suppress dust.

5.07 Medical Program

All personnel working on-site will have passed a general physical exam within 6 months prior to the initiation of on-site activities. A follow-up physical within 12 months following the completion of work activities will also be required. The physicals will include pulmonary and cardiac function tests, and blood test indicative of liver and kidney function.

SECTION 6 - EMERGENCY MEASURES

6.01 Phone Numbers

City of Cortland Police Dept.	(607) 753-3001
City of Cortland Fire Dept.	(607) 756-5611
City of Cortland Memorial Hospital	(607) 756-3500
Ambulance Service	(607) 756-7564

6.02 Hospital Directions

From the Smith Corona facility, turn right (North) on Route 13. Follow Route 13 to Cortland, NY. In Cortland, stay on Route 13 (Tompkins St.) until it intersects Route 11. Take Route 11 North, a left turn, and proceed about 1 mile to Cortland Memorial Hospital. The hospital will be on the left. See Figure 2 for a map showing the location of the hospital.

6.03 Responses to Incidents

Response to major incidents and/or emergency situations will follow the basic steps as explained below.

1. Major Exposure Notify H&S Officer, Project Director and Project Manager. Decontaminate victim to the greatest extent practicable. Remove the victim from the area, using a stretcher if necessary. Administer preliminary first aid, if trained in such. Victim will be transported to treatment at the direction of the H&S Officer and Project Director.
2. Medical Crisis Follow procedures in #1 above.

3. Fire and/or Explosion Evacuate area. Contact Fire Department Follow procedures in #1 above, as necessary.

4. Accident involving equipment Follow procedures in #1 above.

6.04 Follow-Up Procedures

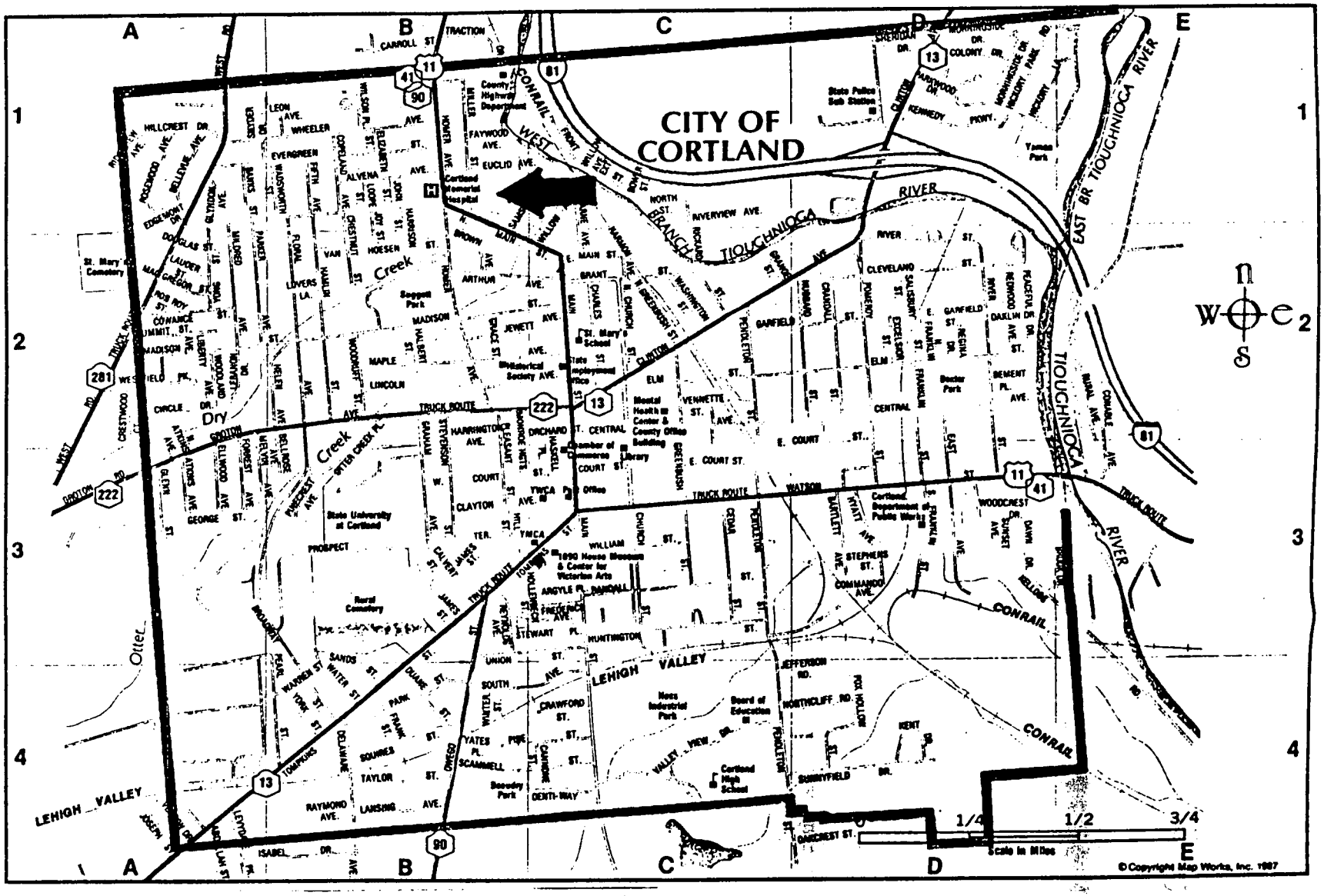
6.04.1 Documentation

Documentation is important in understanding an incident and planning to prevent any similar incidents in the future. A report must be filed with the Project Manager for all incidents of worker illness or injury.

6.04.2 Restore to Order

Work should not be continued following any incident/emergency until all equipment has been restored to readiness, in order to be fully prepared for any future incidents.

Figure 2


CITY OF CORTLAND
TIOUGHNIOGA RIVER
EAST BR TIOUGHNIOGA RIVER
NORTH BRANCH TIOUGHNIOGA RIVER
CONRAIL
WEST
LEHIGH VALLEY
Otter Creek
Dry Creek
Scale in Miles
1/4
1/2
3/4
N
W
E
S
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PERCHLOROETHYLENE

CAS 127-18-4

Tetrachloroethylene

$CCl_2 = CCl_2$

TLV, 50 ppm (≈ 340 mg/m³)†

STEL, 200 ppm (≈ 1340 mg/m³)*

Perchloroethylene is a clear liquid with an etheral odor. Its odor threshold is reported to be as low as 5 ppm[1] for some individuals, but other sources consider 50 ppm to be only barely perceptible.[2] The molecular weight is 165.84 and specific gravity 1.625 (20/4° C). It boils at 121.2° C, solidifies at about -22° C, and has a saturated vapor pressure of 19 torr a 25° C. While quite insoluble in water (0.015 g/mL at 20° C), it is miscible with most organic solvents and oils.

The major uses of perchloroethylene are for commercial dry cleaning and metal degreasing. It also has minor use in products for home use and veterinary anthelmintics.

Excessive exposure to perchloroethylene has resulted in effects on the central nervous system, mucous membranes, eyes, and skin, and to a lesser extent the lungs, liver, and kidneys. The effects most frequently noted have been on the nervous system. Incoordination is generally the first effect observed at low concentrations. Unconsciousness, dizziness, headache, vertigo, or light narcosis have occurred in many instances after occupational exposures. A few deaths have been reported from massive accidental exposure.[2]

A reported oral LD_{50} for mice was 8.850 mg/kg and a lethal concentration in air, also for mice, was 6000 ppm from a 4-hour exposure.[3]

Carpenter[4] exposed rats 8 hr/day, 5 days/week for periods up to 7 months to 70, 230, and 470 ppm. All animals survived with their growth comparable to controls. At 70 ppm, no pathological effects were observed, at 230 and 470 ppm changes were seen in the livers and kidneys.

Rowe *et al*[5] exposed several species 7 hr/day, 5 days/week. Rats survived a concentration of 1600 ppm but drowsiness and depression were observed in the first week with enlarged livers and kidneys apparent after four weeks. Guinea pigs were more affected and had histological changes in their livers which were enlarged. At 400 ppm, 130 seven-hour exposures over six months caused no effect in rats, rabbits, or monkeys, while guinea pigs had increased liver and kidney weights and slight fatty degeneration. Guinea pigs were much more susceptible than the other species; increases in liver weight, total liver lipid, and slight to moderate histopathology occurred at 200 ppm and liver weight increase with no histopathology after exposure to 100 ppm for seven months. In this same laboratory, animals were exposed to a 4:1 mixture of 1,1,1-trichloroethane and perchloroethlene.[6] Repeated 7-hour exposures to 800 ppm 1,1,1-trichloroethane and 200 ppm of perchloroethylene caused the same effects as 200 ppm perchloroethylene alone, while 400 and 100 ppm of each solvent, respectively, had no effect on any species.

Kylin *et al* reported fatty degeneration in the livers of mice exposed 4 hr/day for 1, 2, 4, or 8 weeks to 200 ppm.[7] There was no liver cell necrosis nor were the kidneys affected.

At one time, perchloroethylene had rather extensive use as a vermifuge in animals and man, but better drugs have replaced it. No deaths were reported from dosages of 2.8 and 4 mL (approximately 4.2 and 6 grams) given to humans as a hookworm anthelmintic, but narcotic effects, exhilaration, and inebriation were observed.[8] No variations in liver function tests were seen when perchloroethylene was given in doses of 1 to 8 mL (1.5 and 12 grams) to humans.[9] Unconsciousness was reported by Stewart[10] in a situation where a worker was exposed for 3.5 hours while cleaning steps with a solvent mixture of 1:1 perchloroethylene and Stoddard Solvent. Concentrations were estimated from a similated re-exposure to range up to 1470 ppm perchloroethylene and Stoddard Solvent. Carpenter *et al*[4] indicated that exposures longer than 1.5 minutes at 2000 ppm would cause unconsciousness and subjects exposed at 500 ppm for 50 minutes experienced increased salivation, metallic taste, eye irritation, increased perspiration of hands, and tightnes of the frontal sinuses. Four persons exposed for up to 2 hours at 216 ppm experienced light headedness, burning in the eyes, congestion of the frontal sinus, thickness of the tongue, and difficulty in motor coordination.[4] Slight eye irritation was reported by six individuals exposed 4 hours at concentrations of 100 ppm.[11]

Several studies of the effects of prolonged exposure to perchloroethylene vapors on human volunteers are available.[5,6 11-13] Although there are slight differences due to experimental techniques, failure to consider normal daily variations and inadequate control (sham) exposures, the results are in good agreement with industrial experience. The most comprehensive studies are by Stewart *et al*,[11,12] but others have reached similar conclusions. Prolonged exposure to 200 ppm results in early signs of CNS depression, while there was no response in men or women repeatedly exposed to 100 ppm for 7 hours/day. Clinical chemical studies indicate no liver or kidney effects at these levels but massive exposure to concentrations causing unconsciousness have resulted in proteinuria and hematuria.[10]

Perchloroethylene is eliminated rather slowly via the lungs with only a small amount being metabolized to trichloroethanol and trichloroacetic acid.[13,14]

Skin burns, blistering, and erythema can occur from severe direct contact with liquid perchloroethylene.[15,16] Some dermal absorption takes place but this route of exposure does not appear to be of major significance.[17] Accident victims have survived prolonged exposures to anesthetic vapor concentrations, some found lying in a pool of liquid perchloroethylene.[18] Exfoliation, erthyema, and blistering were severe but recovery was complete. Perchloroethylene was found in the expired air of one subject for several weeks.[10]

Pregnant rats and mice were exposed to 300 ppm perchloroethylene vapors on days 6-15 of gestation without teratological effect on the offspring although fetotoxicity was evident.[19] In a second study no reproductive effects were seen in the offspring of pregnant rats exposed to 100 or 900 ppm.[20] Perchloroethylene does not appear to be mutagenic in most test systems even with activation by liver enzymes.

As with many substances, gavage of massive doses of perchloroethylene has caused hepatocellular carcinomas in mice but not in rats in the NCI Bioassy. The daily doses were 500 or 100 mg/kg for each sex of both species.[21] When given intraperitoneally to the Strain A cancer susceptible mouse, 14 injections of 80 mg/kg, 24 injections of 200 mg/kg, or 24 injections of 400 mg/kg did not increase lung adenomas, the expected

† TWA value adopted in 1982.

* Proposed change in 1982.

positive response in this test system.[22] No cancer was produced on mouse skin with or without a promoter.[23]

Rats were exposed to 300 or 600 ppm vapors 6 hr/day, 5 days/week for one year and were kept for their lifetime. No evidences of a tumorigenic response was shown as a result of their exposure.[24]

Only two limited epidemiological studies of the mortality of workers exposed to perchloroethylene are available.[25,26] Neither study provides a satisfactory basis for drawing definitive conclusions, but both indicated no increase in liver cancer.

Tumors observed following the massive oral doses in mice appear to arise via nongenetic mechanisms related to the greater metabolizing capacity in this species.[27] Tumors are not expected if liver injury does not occur.

A TLV of 50 ppm TWA is recommended to provide a margin of safety in preventing discomfort and subjective complaints which may occur from prolonged exposure to 100-200 ppm. A STEL of 200 ppm is further recommended to prevent anesthetic effects. These levels provide a wide margin of safety in preventing possible liver injury.

Other recommendations: Italy, 90 ppm; Romania, 60 ppm; NIOSH, Finland, and Japan, 50 ppm; East Germany, 44 ppm; Czechoslovakia and the Netherlands, 35 ppm; Sweden, 20 ppm; Poland, 9 ppm; Hungary, 8 ppm; Bulgaria, USSR, and Yugoslavia, 1.5 ppm; Australia, Belgium, Switzerland, and West Germany retain the 100 ppm as of 1978.

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TOLUENE

Toluol

$C_6H_5CH_3$

Skin

TLV, 100 ppm (≈ 375 mg/m³)

STEL, 150 ppm (≈ 560 mg/m³)

Toluene is a colorless liquid with a typical aromatic hydrocarbon odor. Its molecular weight and specific gravity are 92.13 and 0.866, respectively. The boiling point is 110.7° C and solidifies at -95° C. At 25° C, the vapor pressure is 28 mm Hg. The closed cup flash point is 40° F. It is insoluble in water, but miscible with most organic solvents.

Formerly derived solely from coal tar, toluene is now obtained chiefly from petroleum, and is present in gasoline and many petroleum solvents. It is used as a solvent in paints and coatings, for rubber, oils, resins, etc.; as a raw material for the manufacture of benzene and a host of other chemicals, including TNT, TDI, and ingredients of detergents, dyes and drugs.

Because of its wide industrial use and chemical similarity to benzene, the literature of industrial toxicology and industrial medicine, particularly the latter, record numerous investigations of the toxic effects of toluene. According to Patty[1] the concentrations of toluene and benzene required to cause prostration of mice are apparently 3000 ppm and 4700 ppm, respectively. Death from acute poisoning results from 10,000 ppm toluene compared with 14,000 ppm of benzene. Several incidents of workers being overcome by toluene vapor, usually in confined spaces, have been reported. Longley and co-workers[2] describe such an event aboard ship where 26 men were overcome. There were no deaths or serious aftereffects. No irritation of eyes or respiratory passages was observed.

From the standpoint of chronic poisoning, toluene does not cause the severe injury to the bone marrow characteristic of benzene poisoning. Gerarde[3] stated that the myclotoxicity of benzene was completely absent in toluene and other alkyl derivatives of benzene. Von Oettingen *et al*[4] found that exposure of rats at 2500 to 5000 ppm of toluene caused a temporary decrease in the white-cell count, but no evidence of injury to blood-forming organs or liver. Greenburg and co-workers[5] studied a group of painters exposed to toluene in concentrations ranging from 100 to 1100 ppm. Their findings included enlargement of the liver, macrocytosis, moderate decrease in erythocyte count and absolute lymphocytosis, but no leukopenia.

Wilson[6] found that among workers exposed at less than 200 ppm of toluene there were some complaints of headache, lassitude and nausea, but physical findings were essentially negative. At concentrations between 200 and 500 ppm impairment of coordination, momentary loss of memory and anorexia were also present. Between 500 and 1500 ppm palpitation, extreme weakness, pronounced loss of coordination and impairment of reaction time were noted. The red cell count fell in many instances, and there were two cases of aplastic anemia, in which recovery followed intensive hospital treatment. A later comment by Wilson,[7] however, suggests that he did not rule out the possibility that some of the above effects were due to a benzene impurity in the toluene used.

According to Fairhall,[8] severe exposure to toluene may result in a pronounced drop in the red count and partial destruction of the blood-forming elements of the bone marrow. However, Gerarde[9] stated that extensive animal studies clearly indicate that toluene is not a bone marrow poison. While there have been occasional reports of aplastic anemia attributed to toluene,[7] in some instances the presence of benzene was not precluded, and there have been no "epidemics" of this disease among toluene workers comparable to those which have resulted from benzene. Powars[10] reported six cases of aplastic anemia, one of them fatal, among glue sniffers. Although toluene was the solvent chiefly used, no analysis was given of the glue involved in the fatal case. Exposures in these cases are much greater than would normally arise from occupational use of toluene. Thus Knox and Nelson[11] described an instance of permanent encephalopathy involving a man who inhaled toluene regularly for over 14 years.

Von Oettingen and co-workers[4] found that human subjects exposed at 200 ppm suffered slight but definite changes in muscular coordination. They concluded that such concentrations were unlikely to have any discernible untoward effects on health. Gerarde[9] however, believed that von Oettingen's work did not justify the 200 ppm limit. Ogata *et al*[12] found that experimental human subjects exposed at 200 ppm for seven hours showed prolongation of reaction time, decrease in pulse rate and in systolic blood pressure. They consider 200 ppm too high as the MAC. Takeuchi[13] exposed rats at 200 ppm and higher concentrations of toluene for 32 weeks and then to benzene for 39 days. On the basis of differences found between the toluene-exposed animals and controls, e.g., changes in weight of adrenal glands, he suggested that the MAC of 200 ppm for toluene should be reconsidered.

Smyth *et al* reported an oral LD_{50}, administered to rats, to be 7.53 mL/kg.[14]

On the basis of the above data, a reduction in the TLV for toluene from 200 ppm to 100 ppm is recommended, with a STEL of 150 ppm.

Other recommendations: Cook (1945) 200 ppm; Smyth (1956) comments that this limit may permit early signs of narcosis; Elkins (1959) 200 ppm; ANSI (1967) 200 ppm; USSR (1967) 14 ppm; Czechoslovakia (1969) 50 ppm; West Germany (1974) 200 ppm; Sweden (1975) 100 ppm; East Germany (1973) 50 ppm; NIOSH (1973) 100 ppm.

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TOLUENE-2,4-DIISOCYNATE

TDI

$CH_3C_6H_3(NCO)_2$

TLV, 0.005 ppm (≈ 0.04 mg/m³)

STEL, 0.02 ppm (≈ 0.15 mg/m³)

TDI is a liquid at room temperature with a sharp pungent odor. The molecular weight is 174.16. The two commonly used isomers are 2,4-toluene diisocyanate and 2,6-toluene diisocyanate, commercially available in the following three ratios: 1) 100% 2,4; 2) 80% 2,4:20% 2,6; 3) 65% 2,4:35% 2,6. The 80% 2,4:20% 2,6 mixture represents over 95% of the industrial usage and has the following properties: at 25° C, specific gravity 1.22 and vapor pressure 0.5 mm; boiling point 250° C; freezing point 20-22° C; closed cup flash point 270° F. TDI is miscible with alcohol, ether, acetone carbon tetrachloride, benzene and kerosene.

TDI is one of the isocyanates most employed in the manufacture of polyurethane foams, elastomers and coatings. The foams are widely used in furniture, packaging, insulation and boat building, and have many other applications. Polyurethane coatings have many desirable properties, on leather, wire, tank linings and masonry. Elastomers are abrasion and solvent resistant and are used in adhesives, films and linings, and in abrasive wheels and other mechanical items.

Studies in animals by Zapp[1] showed that this isocyanate has a low oral toxicity (approximate lethal dose 5.8 g/kg) but a high toxicity by inhalation. One to two ppm for 30 six-hour exposures resulted in tracheobronchitis. The LC_{50} for three rodent species for a four-hour exposure approximated 12 ppm, according to Duncan *et al.*[2] The animals died of pulmonary edema and hemorrhage. Effects on the liver, kidneys and gastrointestinal tract were also noted, and dermal effects occurred. Repeated daily six-hour exposures at 0.1 ppm were reported to cause chronic inflammation of the tracheo-bronchial mucosa with fibrosa obliterans as the terminal lesions.[3] A fever reaction in animals following intravenous injection of 0.02 mg/kg was reported by Scheel *et al.*[4]

TDI is an irritant causing inflammation and occasional sensitization of the skin, lacrimation, smarting, burning and prickling sensation to the eye; abdominal distress, nausea and vomiting, but the major effect is on the respiratory tract.[1,5]

One type of respiratory response is irritation, indicated by a burning nose and throat, and a choking sensation. With high concentrations this may lead to chemical bronchitis with severe bronchospasm.[6] With sufficient exposure any person will experience these effects even on first exposure.[7] Chemical pneumonitis, pulmonary edema, headache, insomnia have also been reported.

A second respiratory response to TDI is that of sensitization. Some individuals become sensitized on first exposure while others may develop symptoms after exposure over days, months or years. Other workers have had only minimal or no respiratory symptoms for several months of low level exposure, then suddenly develop acute asthmatic reaction to the same level.

The nature of the sensitization process is unknown and many authors have referred to it as allergy, and to the respiratory response in sensitized people as true asthma, comparable to asthma excited by pollens and other exo-allergens. Some TDI sensitized people however, have no history of prior allergic disease.

In a 1963 review Brugsch and Elkins[8] noted reports of 318 cases of TDI intoxication prior to 1961, including two deaths. In most instances data on exposure levels were lacking. Walworth and Virchow,[9] however, reported 83 cases in a plant where the average TDI concentrations ranged from 0.01 to 0.16 ppm. The maximum incidence of cases occurred when the average concentration of vapor was around 0.1 ppm; very little trouble was noted at 0.01 ppm. Hama[10] found TDI vapor levels of 0.03 to 0.07 ppm associated with a high incidence of illness, but no cases were observed from concentrations below 0.03 ppm. Munn[11] considered the 0.1 ppm limit too high.

Elkins *et al*[12] reported 42 accepted or established cases of TDI intoxication, and 73 questionable or disputed cases, among workers in 14 plants in Massachusetts between 1957 and 1962. In 14 of the accepted cases the average TDI vapor concentration found in the workroom was about 0.03 ppm, with very few samples showing more than 0.05 ppm; in 11 cases the average concentration was 0.015 ppm; in 9 cases levels below 0.01 ppm were found; in the remainder measurements representative of worker exposure could not be made. The authors recommended a TLV of 0.01 ppm.

According to Thompson and Scheel,[13] studies with rats support the probability that lung reactivity to TDI is due to chemical damage and not antibody reactions. Markham and Fishburn[14] reported that workers were affected by concentrations generally below 0.02 ppm. Bruckner *et al* described a study of clinical and immunological factors which tended to support the former TLV of 0.02 ppm.[15]

A third type of respiratory response to TDI is that of acute and chronic decrease of ventilatory capacity mea-

sured as FEV_1 with or without overt symptoms of respiratory difficulty.

Gandevia[16] reporting workers exposed to TDI showed an average decrease in mean FEV_1 of 0.18 liters during the course of a single day with some cumulative deficit from Monday to Friday and possible further cumulative deficit over a period of two working weeks. Williamson[6] studied 18 workers exposed toless than 0.02 ppm of TDI over 14 months and showed significant difference in ventilatory measurements for 6 sensitized individuals who showed marked decreased in FEV_1 and FVC. Adams[17] studied 175 workers for five years working in a plant manufacturing TDI where exposures rarely exceeded 0.02 ppm. The group mean annual deterioration of FEV_1 over the five years had significantly exceeded the predicted rate of decline, but furhter analysis showed that only 8 employees accounted for the decrease. A later study by Adams[18] showed no decrease in FEV_1 for symptomatic workers but there was a significant decrease for symptomatic workers. Adams studies are confounded by selection of Tuesday afternoon for collection of pulmonary function measurements.

Peters *et al*[19] studied 38 workers in a polyurethane plant who were exposed to 0.0001 to 0.003 ppm of TDI and showed a decrease of FEV_1 between Monday morning and Monday evening and Friday evening. In a follow-up study 6 months later with TDI exposures of 0-0.012 ppm they showed a significant decrease in mean FEV_1 over the six month interval, which was greater than the predicted decline due to aging alone.[20] These workers were followed for two years, when the decline in FEV_1 continued at a mean annual rate of 0.11 liters, which exceeds the predicted rate of decline to aging alone.[21]

Wegman *et al*[22] studied 112 workers in a polyurethane manufacturing plant. He divided the workers into 4 exposure groups between 0.002 ppm to 0.013 ppm. All four groups demonstrated significant declines in FEV_1, with the magnitude of decrease correlating with levels of exposure indicating a dose response relationship between exposure and acute respiratory effect. The same group of workers were restudied two years later,[23] and divided into three exposures groups. Using FEV_1 as a measure of response, a dose response relationship was observed. Only those in the lowest exposure group (less than 0.0020 ppm) showed normal two year decline. The FEV_1 of those in the highest exposure group (more than 0.0035 ppm) fell 204 mL in two years, which exceeds the expected value by three to fourfold. The decrement in the middle exposure group 0.002-0.003 ppm was borderline (42 mL/yr). A significant association between acute and chronic decrement in FEV_1 was shown.

On the other hand Butcher *et al*[24] followed 166 workers over two years in a new TDI manufacturing plant where 8-hour time-weighted average exposures varied between 0.001 and 0.012 ppm. They state that exposure-related excess decline in pulmonary function does not appear to have occurred during the first two years following initial exposure to TDI. Unexplained increments in FEV_1 and FVC during the study period occur in this population.

The previous TLV of 0.02 ppm, which was unchanged for over 15 years, appears to have been a compromise between the findings of Hama,[10] on the one hand, and Elkins *et al*,[12] on the other. More recent studies, especially those by Peters' group, indicate that even a 0.01 ppm limit is too high.[21,22] NIOSH, in its criteria document for TDI, published in 1973, after a thorough review of the literature available at that time, recommended a workplace environmental standard of 0.005 ppm. as a TWA, with a 20-minute ceiling of 0.02 ppm[5] The most recent report by Wegman *et al*[23] indicates a threshold of response at about 0.002 ppm and, even this low value, may not protect sensitized workers.

It is suggested, after review of all material available, lowering the TLV to 0.005 ppm, with a STEL of 0.02 ppm.

Other recommendations: West Germany (1974) 0.02 ppm; East Germany (1973) 0.015 ppm; Sweden (1975) and Czechoslovakia 0.01 ppm; USSR (1976) 0.007 ppm.

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TRICHLOROETHYLENE

CAS 79-01-6

1,1,2-Trichloroethylene; TCE

$CCl_2 = CHCl$

TLV, 50 ppm (≈ 270 mg/m³)†

STEL, 200 ppm (≈ 1080 mg/m³)*

A colorless liquid with a sweetish odor, trichloroethylene has a molecular weight of 131.3, a specific gravity of 1.4649 (25/4° C), a boiling point of 87° C, a saturated vapor pressure at 20° C of 58 torr, and it solidifies at -84.8° C. It is practically insoluble in water, but highly soluble in lipids. (Partition coefficients at 37°C: blood-air 9, oil-air 940). In the presence of oxygen and heat or short ultraviolet wavelength, trichloroethylene, under certain circumstances, is decomposed to hydrochloric acid and small amounts of phosgene.

Trichloroethylene is used for vapor degreasing and as a solvent. In the past, trichloroethylene was used as an extractant in food-processing, but this was discontinued in 1975, when, on the basis of liver tumors in mice, the National Cancer Institute (NCI) issued an alert warning that trichloroethylene may be a carcinogen. It has limited use as a surgical anesthetic and analgesic.

There is extensive, sometimes conflicting, literature on the toxicology of trichloroethylene. Much of the old literature must be evaluated carefully, since the product then commercially available often contained several percent of hepatotoxic ethane derivatives such as 1,1,2,2-tetrachloroethane. This has been discussed by von Oettengen.[1]

Organ systems reported to be effected by excessive exposure of man and animals to trichloroethylene are: 1) CNS (euphoria, analgesia, anesthesia); 2) liver (degeneration, hepatocellular carcinomas, mice only); 3) kidney (degeneration); 4) lung (tachypnea); 5) heart (arrhythmias); 6) skin (irritation, vesication; paralysis of figers when immersed in liquid trichloroethylene). Of these effects, the limiting factor in decreasing the exposure is depression of the central nervous system.

Trichloroethylene is moderate to low in acute toxicity with an acute oral LD_{50} of 6000-7000 mg/kg reported in rats, dogs, cats, and rabbits.[2,3] It has a typical solvent effect on topical application. Contact with the liquid causes pain but no permanent injury in the eyes and defats the skin causing topical dermatitis. Although it is absorbed through the skin, dermal absorption is not likely to be significant if dermatitis is prevented.[4]

The knowledge of acute human toxicity of trichloroethylene comes mainly from its use as an anesthetic.[5] Tachypnea and ventricular arrhythmias are equated with overexposure (inhaled concentrations greater than 15,000 ppm). Systemic toxicity has been low following anesthesia, but occasionaly hepatotoxicity has been reported, generally attributed to breakdown of the trichloroethylene to dichloroacetylene and phosgene by soda-lime present in some recirculatory anesthesia machines.[1] Little trichloroethylene is used as an anesthetic today.

Vernon and Ferguson[6] found that a 2-hour exposure of a volunteer to 1000 ppm of trichloroethylene resulted in adverse effects on visual perception and motor skill; but 2-hour exposures at 300 ppm and 100 ppm produced no significant effect. In a later report, the same authors[7] noted that low levels of alcohol in the blood (20-30 mg/100 mL) markedly augmented the effect of the 2-hour exposures to trichloroethylene at concentrations of 300 and 1000 ppm.

The effects of trichloroethylene appear to be enhanced in some individuals by simultaneous exposure to caffeine, alcohol, and other drugs. Degreasers Flush, a reddening of the skin, has been observed in some subjects who ingest substantial quantities of alcohol after exposure to trichloroethylene. While disconcerting, this effect did not appear to be injurious, since blood pressure and numerous other parameters studied clinically were not affected.[8]

Hepatic injury was observed in rats[9] exposed for two hours to a trichloroethylene concentration of 10,000 ppm when they were pre-treated with phenobarbital, Aroclor 1254, hexachlorobenzene, 3-methyl cholanthrene, or pregneolone-16-alpha-carbonitrile. Liver pathology appears associated with extensive metabolism of trichloroethylene. The major urinary excretion products in man are trichloroacetic acid and trichloroethanol, the former being present for longer periods of time following exposure.

Death in laboratory animals from acute exposure to trichloroethylene vapor results from respiratory failure or cardiac arrest.[10,11] Although trichloroethylene is reported to have direct action on the bone marrow of rabbits causing myelotoxic anemia after 4-hour repeated exposures, 6 days/week for 45 days to 2800 ppm,[12] other studies have failed to yield evidence of hematological effects in man or animals.

Adams *et al* exposed several species of animals 7 hours/day, 5 day/week for approximately 6 months. At 3000 ppm by volume in air, rats and rabbits showed an increase in liver and kidney weight. At 400 ppm, rats showed an increase in liver and kidney weights and the male rats also showed significantly less growth. Guinea pigs had increased liver weights and the growth of the exposed males was less than controls. Rabbits showed a slight increase in liver weight. An exposed monkey showed no response to 400 ppm. At 200 ppm, the only effect was depressed growth in guinea pigs. Rats, rabbits, and monkeys showed no response. At a concentration of 100 ppm, none of the species showed any significant response. The maximum concentrations without effect for the 6-month period were as follows: monkeys, 400 ppm; rats and rabbits, 200 ppm, and guinea pigs, 100 ppm.[13]

Prendergast *et al*[14] exposed rats, guinea pigs, dogs, rabbits, and monkeys 24 hours/day for 90 days to 35 ppm with no effect except slight growth depression. Repeated 8-hour daily exposures to 700 ppm for 90 days were also without effect.

Ahlmark and Forssman[15] estimated exposure to trichloroethylene by measuring the urinary excretion of trichloroacetic acid. They found the chief symptoms to be abnormal fatigue, irritability, headache, gastric disturbances, and intolerance to alcohol. Ahlmark and Friberg[16] tentatively suggested 30 ppm vapor as a desirable limit for the time-weighted average occupational exposure, based on urinary trichloroacetic acid levels. Although trichloroacetic acid levels are indicative of average exposures, they do not necessarily describe excursions which may have been responsible for the observed symptoms.

Haas[17] and Grandjean *et al*[18] reported a variety of nervous disturbances in a group of 50 workers exposed to trichloroethylene vapor at concentrations ranging from 1.0 to 335 ppm. These disturbances increased with the length

† TWA value adopted in 1982.

* Proposed change in 1982.

of exposure (up to five years or more), and were reported to be distinctly more frequent when average trichloroethylene concentrations exceeded 40 ppm.

Barododej and Vyskocil[19] also recommended a limit of about 40 ppm, finding signs and symptoms of chronic trichloroethylene poisoning including intolerance to alcohol, tremors, giddiness, and anxiety at an exposure above 40 ppm.

Lilis and co-workers[20] reported that workers exposed at concentrations averaging about 10 ppm (12% of the tests showed values about 40 ppm) complained of headache, dizziness, and sleepiness.

The symptoms reported from these industrial exposure situations are highly subjective and are common findings in control individuals with no chemical exposure. Yet, the consistency of these reports suggests the possibility of some subjective complaints as concentrations exceed about 50 ppm. In these industrial environments, exposure concentrations are variable and the effects reported may be associated with transient, high concentrations rather than with the time-weighted average value during the work shift. Furthermore, worker populations are variable with respect to age, health status, nutrition, and intake of alcohol, caffeine, and medicinals, all factors which might influence worker response to TCE.

In contrast, controlled laboratory studies with relatively homogeneous populations of young, healthy subjects have shown less effect of TCE than reported in the industrial setting. The air concentrations of TCE in these studies are, of course, more precisely known and relatively constant throughout the exposure. Stopps and McLaughlin[21] exposed human volunteers to 100 ppm of trichloroethylene and found no changes in various performance tests, but noted some changes at higher concentrations. Stewart and associates[22] reported that in an early study, volunteers exposed to 200 ppm of trichloroethylene for 7 hours/day reported mild responses such as slight fatigue and sleepiness on the fifth day of exposure. There were no measurable objective responses and no control studies were included. Stewart *et al*[23] did not confirm these effects in subsequent, better controlled studies. In the later studies, male subjects were exposed to 20, 100, or 200 ppm and female subjects to 100 ppm 7.5 hours/day for several days. The authors concluded,

> *"the data obtained do not prove that TCE has a well defined deleterious behavioral effect upon humans at either 100 or 200 ppm in the atmosphere for the 7.5 hr per day, 5 days/week... The 100 ppm concentration probably has a three- to four-fold margin of safety for most individuals...."*

The authors stress the importance of controlling for learning by the test subjects, a phenomenon which has not been appreciated adequately in many other studies including the earlier one by Stewart's group.

Triebig *et al*[24] exposed seven healthy volunteers to 100 ppm of trichloroethylene for five days and observed no impairment in mental or psychological capacities. On the other hand, Ertle *et al*,[25] in a similar study, observed fatigue, lassitude, and headache in volunteers.

Trichloroethylene was neither embryotoxic nor teratogenic in Sprague-Dawley rats and Swiss Webster mice inhaling trichloroethylene.[26] These results have been confirmed in two other studies in female rats exposed in one case to 500 ppm and in other to 1800 ppm.[27,28]

Trichloroethylene was found to be weakly mutagenic in *E. coli* in the presence of a metabolizing system,[29] and negative in dominant lethal assays in rats and mice,[30,31] or in extensive studies in Drosophila.[32] Positive effects in some studies may be due to epoxy stabilizers sometimes present in trichloroethylene.

Trichloroethylene came under suspicion as a possible carcinogen as a result of a report from the National Cancer Institute[33] that oral administration of trichloroethylene resulted in hepatocellular carcinomas in mice. In this bioassay, male and female rats (Osborne-Mendel) and mice (B6C3F1) were intubated with trichloroethylene daily for 18 months with an observation period of 3 to 6 months following treatment. Rats were given doses at either 1000 mg/kg or 500 mg/kg, 5 times a week. Male mice were given 2400 or 1200 mg/kg and female mice 1800 mg/kg or 900 mg/kg dose 5 times/week. Hepatocellular carcinomas were not seen in the rats; 30 of the 98 (30.6%) mice given the low dose, and 41 of the 95 (43.2%) mice given the high dose developed hepatocellular carcinomas. Only one (2.5%) of the 40 (25%) control mice developed carcinoma, an unusually low incidence for this tumor in this strain of mice which often has a 40% background incidence.

Maltoni completed a lifetime chronic study[34] in Sprague-Dawley rats which did not indicate any carcinogenic effect. Trichloroethylene was administered in olive oil by gavage 4 to 5 time/week for one year at dose levels of 50 and 250 mg/kg body weight.

When tricholoroethylene was applied to mouse skin by several methods, Van Duuren *et al*[35] found low doses to be inactive. One mg/kg of the material was applied three times weekly to the dorsal skin with or without phorbol myristate acetate as a promotor. It was also injected subcutaneously at weekly intervals at a dosage of 0.5 mg/kg with no significant effect and intragastrically with no increase in tumors of the forestomach.

In an inhalation study, rats, mice, and Syrian hamsters were exposed 6 hours/day, 5 days/week for 18 months to either 0, 100, or 500 ppm of pure trichloroethylene stabilized only with an amine base. The authors concluded,

> *"No significant increase in tumor formation was observed in any species or dosing group, except in malignant lymphomas, which were increased in female mice in the following incidence rates: 9/29 (controls), 17/30 (100 ppm), and 18/28 (500 ppm). Whether or not this high occurrence of lymphomas, which is peculiar to this strain of mice (NMRI) has any relationship to tri-exposure cannot be decided upon by the present experiment. It is concluded that from these findings no indication for a carcinogenic potential of pure trichloroethylene can be deduced."*[36]

A recently completed study[37] investigated the possible differences in metabolism and pharmacokinetics between mice and rats exposed to tricholoroethylene. A comparison of metabolized trichloroethylene on a weight basis indicates that the mouse metabolizes 2.2 times more than the rat at 10 ppm and 3.6 times at 600 ppm. Hepatic macromolecular binding was greater in the mouse than in the rat. The binding data suggest that tumor formation in the mouse exposed to trichloroethylene occurred via a nongenetic mechanism and tumors are not expected if liver injury does not occur.

not sufficient to precipitate arrhythmia in dogs exposed at 5,000-10,000 ppm. With rats,[19] abnormalities in the electrocardiogram appeared during inhalation of 25,000-100,000 ppm. Anesthetized dogs, rats, mice, monkeys, hamsters and rabbits exposed to concentrations ranging from 5,000-150,000 ppm[20-26] have shown various cardiovascular and circulatory abnormalities.

Elimination is relatively rapid. Dogs exposed to 10,000-30,000 ppm[27] for four to five minutes exhaled the major portion within the first two minutes after the end of exposure, but two to seven hours elapsed until concentrations were $\leq$ 5 ppm. Dogs exhaled within one hour[28] essentially all the FC-11 inhaled during a 6 to 20 minute exposure at 5,000 ppm. Inhaled FC-11 was promptly detected[29] in blood, cerebrospinal fluid, bile and urine of anesthetized rabbits and dogs; clearance was slower than with dichlorodifluoromethane. Unanesthetized dogs exposed to 1,000-10,000 ppm for ten minutes[30] showed a rapid rise in blood concentrations of FC-11 during the first five minutes, which was followed by a rapid and then more gradual decline after exposure.

In humans, radiolabeled tests,[31] showed most of the dose (79-100%) exhaled within the first hour after a 7 or 17-minute inhalation at 1,000 ppm. At 30 minutes,[32] retention of the labeled dose inhaled in a single breath was 23% versus 10%, 20% and 12% for comparable doses of FC-12, FC-113 and FC-114, respectively.

A pharmacokinetic model based on analysis in dogs and humans[33] gave an estimate of 77% absorption of the inhaled FC-11 from an eight-hour exposure to 1,000 ppm. This absorption would result in about one-fourth the blood level required to sensitize dogs receiving the additional stress of intravenous epinephrine.

Industrial and commerical experience has been favorable with few reports of injury only at levels in excess of the recommended TLV. Based on the absence of effect in animals exposed 24 hours per day for 90 days to 1,000 ppm this value is recommended as a ceiling. It should provide a substantial margin of safety to prevent organic injury as well as cardiac sensitization.

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TRICHLOROMETHANE

See, **CHLOROFORM**

TRICHLORONAPHTHALENE

Halowax

$C_{10}H_5Cl_3$

Skin

TLV, 5 mg/m³

STEL, 10 mg/m³

A colorless to pale yellow solid with an aromatic odor, trichloronaphthalene has a molecular weight of 231.51. It melts at 92.78° C, boils between 304.44 and 354.44° C and has a vapor pressure of ≤ 1 mm Hg at 20° C. The reported open cup flash point is 392° F. It is insoluble in water.

Trichloronaphthalene is employed in lubricants and as insulation for electrical wire.

Drinker and associates[1] exposed rats at an average concentration of 11 mg/m³ 16 hours a day for a total of 1232 hours. Some tetrachloronaphthalene was present. Liver cells were slightly swollen and over-granular and there were occasional mitotic figures. Very slight swelling of the liver was found in animals exposed at 1.3 mg/m³ for 1896 hours. These authors considered a concentration of 10 mg/m³ permissible for occupational exposure. Bennett et al[2] commented that a mixture of trichloronaphthalenes had toxic properties which were relatively slight in comparison with compounds of higher chlorination. In a later report Drinker[3] reaffirmed the recommendation of 10 mg/m³ as a permissible limit.

Bell[4] found no effects in calves which received 16 and 26 mg/kg of relatively pure trichloronaphthalene orally over periods of seven and ten days. Slightly higher dosage of tetrachloronaphthalene caused mild symptoms associated with hyperkeratosis, from which the animals recovered. Similar or lower dosages of penta- and hexachloronaphthalene caused severe symptoms.

Industrial experience with trichloronaphthalene[5] (usually mixed with some tetrachloronaphthalene) has been favorable, in comparison with the more highly chlorinated naphthalenes. While some cases of chloracne have been reported, the incidence and severity have been less when trichloronaphthalene was the only material used than when penta- or hexachloronaphthalene was present. In addition, there have been no fatal cases of liver injury reported from industrial exposure to trichloronaphthalene. Mayers[6] did record one non-fatal case of toxic hepatitits from a plant where air studies revealed a concentration of about 3 mg trichloronaphthalene per cubic meter of air, but so far as can be determined all the other instances of liver damage from this type of material in industrial workers involved exposure to the more highly chlorinated naphthalenes, or to members of the chlorinated diphenyl series.

The TLV of 5 mg/m³ and a STEL of 10 mg/m³ for trichloronaphthalene are consistent with the TLVs assigned to the more toxic members of the series. When used in conjunction with tetrachloronaphthalene, the lower limit recommended for that compound must be taken into consideration when evaluating the exposure.

Other recommendations: Cook (1945), Smyth (1956), Elkins (1956), Yugoslavia (1971) and Finland (1975) all recommend 5 mg/m³; USSR (1967) 1 mg/m³.

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1,2,3-TRICHLOROPROPANE

Glycerol trichlorohydrin; Allyl trichloride; Trichlorohydrin

$CH_2ClCHClCH_2Cl$

TLV, 50 ppm (≈ 300 mg/m³)

STEL, 75 ppm (≈ 450 mg/m³)

1,2,3-Trichloropropane is a colorless, combustible liquid with an odor described as being quite similar to that of trichloroethylene, i.e., chloroform-like. Its molecular weight is 147.43 and specific gravity is 1.3889. The boiling point is 156.2° C, melting point is -14.7° C and a vapor pressure at 40° C of 8.5 Torr. 164° F is the closed cup flash point. It is insoluble in water, soluble in alcohol and ether.

It is employed as a paint and varnish remover, as a solvent and as a degreasing agent.

Rats and guinea pigs (10 each, 5 of each sex) exposed at 799, 2080 and 5010 ppm of 1,2,3-trichloropropane for 30 minutes resulted in CNS depression, which was minimal at 799 ppm but progressed to narcosis and convulsions at the higher concentrations.[1] Two of 10 rats and 6 of 10 guinea pigs died following exposure at 5010 ppm; the only histopathologic lesions evaluated at 14 days post-exposure were adrenal cortico-medullary necrosis. Mortality in the other two exposure groups was limited to one male rat exposed at 2080 ppm; no irreversible organ lesions were reported.

McOmie and Barnes[2] reported that exposure at 30 mg/L (5000 ppm) for 20 minutes killed several mice, some dying several days later from liver damage. Daily 10-minute exposures at 2500 ppm for 10 days resulted in 7 deaths among 10 mice.

1,2,3-Trichloropropane was tested by Smyth *et al,*[3] who demonstrated that it was more toxic than 1,1,1-trichloropropane. In one trial, 1000 ppm produced death in 5 of 6 rats after 4 hours of exposure; whereas 1,1,1-trichloropropane at 8000 ppm produced similar results. These same authors reported 1,2,3-trichloropropane as nonirritating to the skin but highly irritating to the eyes of rabbits.

Silverman and his associates,[4] in their study of sensory response to a number of industrial solvent vapors, found

In light of the above evidence that neither acute or chronic effects occur from repeated daily exposures after many years, and that irritation may be experiences at around 20 ppm, but not at 10 ppm, the recommended TLV for vinyl acetate is 10 ppm, with a STEL of 20 ppm.

Other recommendations: USSR MAC (1976) 3 ppm; NIOSH (1978) 4 ppm ceiling.

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VINYL BENZENE

See, **STYRENE**

VINYL BROMIDE

Bromoethylene

C_2H_3Br

TLV, 5 ppm (≈ 20 mg/m³), Appendix A2 — Suspected Carcinogen

Vinyl bromide has a molecular weight of 106.96 and the liquid has a specific gravity of 1.4933 at 20° C. It has a melting point of -139.54 and a boiling point of 15.80 at 760 mm Hg. Insoluble in water, it is soluble in alcohol, ether, acetone, benzene or chloroform.

This substance has not been found suitable as an anesthetic, but is useful as a fire-retardant in plastics.

The oral LD_{50} of the 50% solution in corn oil is 500 mg/kg in male rats.[1] Liquid vinyl bromide is slightly to moderately irritating to the eyes, but non-irritating to intact or abraded rabbit skin. Acute inhalation studies show that 100,000 ppm is lethal to rats in 15 minutes; 50,000 ppm renders rats unconscious in 25 minutes and is lethal after 7 hours of exposure. At 25,000 ppm, rats are anesthetized, but recover rapidly even after 7 hours of exposure. Slight to moderate kidney damage was seen in rats surviving exposure to 50,000; but no histopathological changes were seen in rats exposed 7 hours to 25,000 ppm.

A sub-acute inhalation study in rats exposed to 10,000 ppm for 7 hours per day, 5 days per week, revealed significantly depressed body weights after 15 days of exposure, but no compound-related gross or microscopic pathological changes after 20 exposure days.[2]

In a chronic inhalation study, in which groups of rats, rabbits and monkeys were exposed to 250 or 500 ppm for 6 hours/day, 5 days/week for 6 months, no significant changes were detected in any of the following parameters: growth rates, food consumption (rats and rabbits only), hematology, gross pathology, organ to body weight ratios and histopathology.[2] Measurements of blood bromide showed that the levels increased with duration of exposure to all three species and were proportional to the concentration of vinyl bromide in the test atmosphere. Estimated equilibrium values for blood bromide in monkeys exposed to 250 and 500 ppm were well below those levels at which signs of bromism were evident.

Based on interim data obtained after 12 months of a lifetime inhalation study, there appear to be serious toxic effects in Charles River Sprague-Dawley rats exposed to 1250 or 250 ppm 6 hours/day, 5 days/week.[3] The toxic effects include increased mortality, decreased body weight, angiosarcomas of the liver and carcinomas of the zymbal glands of the ears. These responses were dose related and did not occur in groups of male and female rats similarly exposed for 1 year to 50 ppm or 10 ppm.

Based on these data a TLV of 5 ppm, the same as that for vinyl chloride, is recommended, as well as placement on the A2 listing as a suspected human carcinogen.

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VINYL CHLORIDE

Chloroethene

$CH_2 = CHCl$

TLV, 5 ppm (≈ 10 mg/m³) — Appendix A1a — Recognized Carcinogen

A colorless, highly flammable gas with an ethereal odor, vinyl chloride has a molecular weight of 62.50. It boils at -13.9° C and freezes at -159.7° C. Vinyl chloride is usually handled as a liquid under pressure, and containing a polymerization inhibitor (phenol). It is slightly soluble in water, but dissolved by alcohol and ether.

The chief use of vinyl chloride is as a raw material for the manufacture of polyvinyl chloride resins. It is also employed in organic syntheses.

Since vinyl chloride is a gas at room temperature and pressure, the common route of toxic exposure is by inhalation. As with many liquified gases, contact of the skin or

eyes with escaping compressed vinyl chloride can produce freezing frostbite.[1]

Vinyl chloride has long been considered to be very low in toxicity by acute inhalation. Lehmann and Flury[2] summarized the literature and reported work by Schauman who considered vinyl chloride to be a candidate surgical anesthetic. Schauman reported little pathological change even after repeated exposure to anesthetic concentrations. Further work on the anesthetic potential of vinyl chloride indicated that vinyl chloride was unsafe for use as a surgical anesthetic in dogs and that because of its flammability, poor efficacy and its ability to cause cardiac irregularities at anesthetic concentrations vinyl chloride was not suitable for use as an anesthetic in humans.

Despite the early reports ascribing low toxicity to vinyl chloride, injury during the production of polyvinyl chloride (PVC) resins was reported as early as 1949. Significantly this report came from Europe where production of PVC in Europe preceded U.S. production by several years and today the quantity produced in Europe still exceeds U.S. production by about two fold. In 1949, Tribuhk *et al*[3] reported numerous effects in PVC workers in what, by today's standards, must be considered as primitive production facilities. These authors found a *"considerable number of cases of hepatitis among workers"* but were more concerned with other hepatotoxic chemicals such as chlorinated diphenyl and chlorinated naphthylene (Holowax [sic]) than they were with vinyl chloride.

As a result of two deaths in Canada, the acute inhalation toxicity of vinyl chloride was studied by Mastromatteo *et al*[4] who reported that exposure of mice, rats and guinea pigs to 10, 20 and 30 volume percent vinyl chloride caused the following mortality:

NUMBER OF DEATHS IN DIFFERENT GROUPS OF FIVE MICE, RATS AND GUINEA PIGS EXPOSED FOR THIRTY MINUTES TO VARYING CONCENTRATIONS OF VINYL CHLORIDE IN AIR

Vinyl Chloride concentration (percent by volume in air)	Laboratory animal			
	Mice	Rats	Guinea pigs	TOTAL
10	0/5	0/5	0/5	0/15
20	1/5	0/5	0/5	1/15
30	5/5	5/5	1/5*	11/15
40	—	—	2/5*	2/5

*A delayed death occurred within 24 hours following exposure.

Some pulmonary hyperemia and engorgement was observed by these investigators, but liver and kidney injury were remarkably low. Deaths were due to narcosis.

The first report of studies to determine the effect of long-term repeated exposure (6 months) were summarized by Torkelson, Oyen and Rowe[1] as follows:

"Repeated exposures of laboratory animals at several concentrations of vinyl chloride in air were conducted to determine the chronic toxicity of this material towards animals in order to assess the hazard to humans. Vinyl chloride was found to have a slight capacity to cause liver and kidney injury on repeated exposures. Male and female rats showed micropathological changes after repeated daily 7-hour exposures at 500 ppm for 4.5 months. Repeated 7-hour exposured at 200 ppm for six months resulted in micropathological changes in the livers of rabbits and statistically significant increases in the average weight of the livers of male and female rats, but no detectable changes in dogs and guinea pigs. Repeated 7-hour exposures at 100 ppm resulted in slight increases in the average weight of rat livers, the other species were not affected. All species studied tolerated repeated daily 7-hour exposures at 50 ppm for six months with no detectable injury.

Repeated daily 1-hour exposures at 200 and 100 ppm of vinyl chloride were without effect, longer exposures caused a slight increase in liver weight.

The standard for evaluating regular daily 7- or 8-hour exposures may be defined as the concentration below which practically all analytical results must fall. The value of 100 ppm is suggested as this standard for vinyl chloride, with a time-weighted average for all exposures not to exceed 50 ppm."

Lester, Greenberg and Adams[5] took exception to the conclusion of Torkelson *et al* (1961) that 50 ppm should be a maximum time-weighted average exposure for workers. On the basis of 3 months exposure of rats to 2 volume percent and 19 days to 5 volume percent, they concluded that 500 ppm was acceptable as a TLV despite minor changes which they observed in rat livers and which they considered *"were within the normal range and were not pathologic in nature."*

Since 1949 numerous articles describing conditions and problems in PVC production plants have appeared particularly in the Eastern European literature. Filatova and Gronsberg,[6] Gabor *et al*,[7] Suciu *et al*,[8] Gabor *et al*,[9] Grigorescu and Toba,[10] Antonyuzhenko,[11] and Kudryavtseva[12] have all described the effects of apparent gross chronic exposure. These papers and abstracts are difficult to interpret since there are generally inadequate descriptions of the exposure conditions and analysis of the workroom air, so no dose-response relationship can be determined. The injuries and effects described by the authors are not consistent with the levels of exposures claimed by the authors nor are the levels of exposure consistent with past or even present-day chemical technology. Furthermore, mixtures of chemicals are involved making it possible to ascribe the effect to any one of them.

For example, Suciu *et al*[8] (through translation) described nervous disorders including euphoria with whistling and laughing, incoordination and dizziness similar to alcohol intoxication. However, Suciu *et al* ascribes these results to exposure of the order of 5.5 mg/m^3 (2 ppm v/v) which is not consistent with other publications which indicate these effects will be apparent only if concentrations greatly exceed 10,000 to 20,000 ppm v/v. Therefore, the following conclusions by the authors can be construed as being the result of massive and apparently repeated exposures:

1. *Vinyl chloride and the vinyl monomers possess a narcotic action and produce, depending upon concentration, in addition to characteristic neurologic manifestation, a state of euphoria (12%), followed by a state of inebriation similar to that of alcohol intoxication. In certain cases narcosis can appear.*

After leaving the working environment, a state of somnolence (45%) persists, with hypersomnia. Vinyl chloride acts on the skin and produces a sensation of formication and of heat.

2. *After repeated exposure, a neurologic asthenia sets in which somnolence predominates.*
3. *After a variable period of time, dyspeptic disturbances are added to the neurologic manifestations; these are at first not characteristic; they are in the form of epigastric pains (16%), swelling, discomfort, feeling of heaviness in the right hypochrondium (7%) or the left (5%) with anorexia, particularly for fats.*

   In 30.2% of the cases, congestive hepatomegaly appears, which may mimic toxic hepatitis without jaundice; some cases may become chronic.

   In 6% of the cases, the hepatomegaly is accompanied by splenomegaly. The proteinogram and the aldolases are the most sensitive tests and show changes similar to those of acute hepatitis: increase in α-globulins and of the β- and γ-globulins; and thymol test, Greenstedt's reaction and the zinc sulfate test are positive only in few of the cases.
4. *After 3 years of exposure in 9% of the cases a syndrome typical of ulcer without radiologic changes becomes manifest.*
5. *In 6% of the cases the Raynaud syndrome has appeared, particularly among the young men. Plethysmography shows in half of the cases an inhibition of the vasomotor centers.*
6. *In addition, allergic dermatitis in 4.4% of the cases, and scleroderma in 3.6%, has been observed.*
7. *The clinical and laboratory findings are of great importance in occupational pathology because in numerous cases diseases appear in man that cannot be reproduced in the animal (Raynaud's syndrome and scleroderma).*

   The sudden and frequent appearance of these manifestations in the PVC division of several plants, and in certain divisions in normal individuals who are still relatively young, and their disappearance in the majority of the cases after the institution of protective measures and change of work, have shown us decisively that vinyl chloride and the vinyl monomers have played a part in the production of these manifestations. (End of author's summary).

In 1967, reports appeared in the literature describing a condition known as acroosteolysis in workmen engaged in polymerization of vinyl chloride to polyvinyl chloride. Harris and Adams[13] reported on two cases in Europe. Wilson *et al*[14] reported on 37 cases in the B. F. Goodrich Company. Juhe *et al*[15] described a syndrome consisting of (arranged in decreasing order of occurrence) thrombopenia, splenomegaly, liver damage, obstruction of ventilation, circulatory obstruction, and skin and bone alteration.

As a result of this problem, the University of Michigan in 1967 was retained by the Manufacturing Chemists Association to investigate acroosteolysis in sponsoring American companies. The results of a large scale epidemiological study of workers then currently employed in vinyl chloride and polyvinyl chloride production were reported in three publications by this group Dinman *et al*,[16] Cook *et al*[17] and Dodson *et al*.[18]

Dinman *et al*[16] summarized the study as follows:

"An epidemiological study was performed covering 5,011 employees with 21,510 man-years experience in various phases of vinyl chloride (VC) and polyvinyl chloride (PVC) manufacturing in 32 plants throughout the United States and Canada. The total number of definitive cases of acroosteolysis (AOL) was 25; 16 other individuals were under suspicion. This condition is clearly associated with the hand cleaning of polymerizers. Workers engaged in other phases of VC or PVC manufacturing do not appear to be at risk of developing AOL. The importance of Raynaud's phenomenon as a concomitant of AOL us emphasized. Several statistical approaches for rapid medical survey are suggested. Acroosteolysis appears to be a systemic rather than local disease. Presently, neither the etiological agent nor its portal of entry is known."

Cook *et al*[17] describes the polyvinyl chloride production process in considerable detail. They concluded that although no etiological agent could be identified, *"There appeared to be a correlation between the extent of degassing prior to entry into the reactor"* and the incidence of acroosteolysis.

Mutchler and Kramer[19] presented a paper at the 1968 Gordon Research Conference which was subsequently published (1972), which reported on *The Correlation of Clinical and Environmental Measurements for Workers Exposed to Vinyl Chloride.* The authors drew the following conclusion:

"Our findings suggest that repeated exposure to vinyl chloride at TWA levels of 300 ppm or above for a working lifetime together with a very low level of vinylidene chloride may result in slight changes in certain physiologic and clinical laboratory parameters. The possibility of some impairment in liver function tests must be considered, even though no overt clinical disease was evident in any of the individuals studied. We shall continue our study, but suggest that similar studies to help clarify the effects of this material be performed for other worker populations exposed to vinyl chloride alone."

P. L. Viola, in an attempt to produce acroosteolysis in animals, exposed rats 4 hours per day, 5 days per week to 30,000 ppm (3%) vinyl chloride vapor. In his first report on the results of 12 months exposure, he described metaplastic changes in the bones which he considered similar to the human disease acroosteolysis. He made no mention of having observed cancer in these animals until the Tenth International Cancer Congress in May 1970. In the abstracts of this meeting, and subsequently in May 1971, Viola, Bigotti and Caputo[21] reported tumors of the skin, lungs and bones occurring first after 10 months of exposure. The authors summarized this work as follows:

"Rats (Ar/IRE Wistar strain) exposed for 12 months to vapors of vinyl chloride developed tumors of the skin, lungs, and bones. The cutaneous tumors, which always appeared in the area in which submaxillary and parotid glands are located, have been histologically recognized as epidermoid carcinomas, papillomas, and mucoepidermoid carcinomas. The morphological characteristics of lung tumors, which

occurred in a lower percentage, were mainly of the adenocarcinoma type, with the exception of a single epidermoid tumor originating from the epithelial covering cells. In a minor number of rats, a large proliferation of cartilaginous tissue diagnosed as osteochondroma developed in the metacarpal and metatarsal regions of the four limbs."

The report by Viola *et al*[21] is apparently the earliest publication in which carcinogenic activity has been ascribed to vinyl chloride in man or animals. Although there were obvious deficiencies in Viola's study, such as his very impure sample, the presence of food and bedding in the exposure chamber, the excessive exposure concentration as well as in the statistical evaluation and interpretation of the lesions, the report was of serious concern and resulted in additional animal and epidemiological studies which are currently underway in Italy by Maltoni,[22] Maltoni and Lefemine[23,24] and in the U.S., Keplinger *et al.*[25]

On January 22-23, 1974, the B. F. Goodrich Company notified its employees, NIOSH, the Kentucky State Department of Labor, and the public, that three workers had died of angiosarcomas of the liver. The case reports of the first subject has been published by Creech and Johnson.[26] The subject, a 36 year old male, was hospitalized January 5, 1970 and subsequently succumbed September 27, 1971. He had worked in PVC production from November 1955 until his illness. The history, clinical course and pathologic findings are consistent with the others who died of angiosarcoma.

The work of Maltoni and Lefemine[23,24] has been reported publicly at the OSHA hearing, Washington, D.C., February 15, 1974, and included in the 1974 publication of the Second International Symposium on Cancer Detection and Prevention, Bologna, Italy, April 9-12, 1973. In these studies groups of rats as well as mice and hamsters have been exposed at concentrations of 10,000 to 50 ppm vinyl chloride vapor. Maltoni and Lefemine (1974) reported carcinomas of the Zymbal glands, nephroblastoma and angiosarcomas of the livers of rats at concentrations of 250 ppm to 10,000 ppm but not at 50 ppm. Subsequent unpublished information (August 31, 1974) reported, *"1 liver angiosarcoma, 1 extrahepatic angiosarcoma and 1 nephroblastoma, in three animals of the first experiment, exposed at 50 ppm of VC for 1 year, and surviving 135 weeks from the beginning of the treatment."* The authors conclude that,

> *"a dose-response relationship clearly emerges, as far as angiosarcomas and nephroblastomas are concerned, in the lower dose ranges: from 500 ppm to 50 ppm for angiosarcomas, and from 250 ppm to 50 ppm for nephroblastomas. A comparison of the results available at the present moment in rats exposed for 12 months and 4 months (BT1 and BT3 experiments) shows that the neoplastic response, as far as angiosarcomas and nephroblastomas are concerned, is affected by the length of exposure to VC."*

In their experiment BT3 Maltoni and Lefemine reported possible *in utero* production of angiosarcomas in offspring of pregnant rats exposed at 10,000 and 6,000 ppm.

Keplinger *et al*,[25] in a study sponsored by American companies, have confirmed the findings of Maltoni and Lefemine. In this study, groups of 100 rats, mice and hamsters of each sex are being exposed seven hours per day, five days per week to either 2,500, 200 to 500 ppm vinyl chloride monomer. After seven months of exposure angiosarcoma and lung adeaomas have been observed in mice at all exposure levels. Although the data are preliminary in nature and require confirmation, angiosarcomas were apparently also observed in rats at 2,500 and 200 ppm and in a single hamster at 2,500 ppm. This study is still in progress and will not be completed until 1976 or 1977.

Epidemiological studies on U.S. workers have been conducted by Tabershaw-Cooper Associates for the Manufacturing Chemists Association.[27] The summary of this study is as follows:

> This historical prospective mortality study of 8384 men who had at least one year of occupational exposure to vinyl chloride before December 31, 1972, demonstrated that cancers of the digestive system (primarily angiosarcoma), respiratory system, brain, and cancers of unknown site, as well as lymphomas, occurred more often than expected in those members of the study population with the greatest estimated exposure. The mortality from other cancers was lower than that of the general male population, with the exception of cancers of the buccal cavity and pharynx. The explanation for the latter finding is not apparent.

The other major findings of the study are: (1) The overall mortality of the study population was approximately 75% of what would be expected in a comparable population of U.S. males; (2) No cause of death showed a statistically significant excess over what would be expected in a comparable U.S. male population; and (3) No deaths identified as angiosarcoma of the liver were found other than those previously identified.

This is the first epidemiological study which suggests that in humans vinyl chloride may also be associated with cancer of multiple sites.

It is difficult to derive a reasonable TLV from the data presented in the literature summarized above. There is indirect evidence that intermittent exposures to vinyl chloride of the order of thousands of ppm, in this country as well as Russia, have not been infrequent in PVC plants. No data on the past (or even present) concentrations of vinyl chloride in plants where angiosarcoma cases have occurred, or not occurred, appear to be available. One report[29] indicates that 21 of 26 of the early cases of angiosarcoma occurred in former reactor cleaners. Cleaning of reactors was apparently responsible for most of the acroosteolysis cases investigated by Cook and associates,[17] and has resulted in death from acute poisoning by vinyl chloride.[36]

It is the sense of the Committee that, if the average exposure to vinyl chloride does not exceed 5 ppm, there wil be no increase in the incidence of cancer, specifically of angiosarcoma of the liver. It is probable that the cancers reported and attributed to vinyl chloride among PVC workers resulted from exposures many times this level.

NIOSH recommended a limit of 1 ppm as a TWA, with a ceiling of 5 ppm. The 1 ppm value was apparently based on the erroneous belief that this was the lowest concentration that could be readily measured.

Gehring and co-workers,[30] using a probit model, and based on studies with rats, found the predicted incidence of hepatic angiosarcomas from 8 hour/day, 5 days/week, 35 year exposure at 1 ppm to be 1.5 times 10^{-8}.

Recent papers have included a report of 4 cases of respiratory cancer among vinyl chloride workers, but no dose-response relationship.[31]

On the other hand, Fox and Collier,[32] in a study of 7000 men exposed to vinyl chloride in PVC manufacture between 1940 and 1974, found no evidence of cancers due to vinyl chloride at sites other than the liver. There are four liver cancers, two of them angiosarcomas.

Delorme and Theriault[33] described 10 cases of liver angiosarcoma among workers in vinyl chloride polymerizing plant in Quebec, which were accompanied by fibrosis of the liver. Details of 64 cases were presented by Sputas and Kaminski.[34]

Mutufugi, in Japan, noted that in contrast to western countries, in which 70 angiosarcomas cases (associated with vinyl chloride exposure) had been reported, no cancer, but many poisoning cases, have been reported in the USSR.[35]

Based on the above data, an A1a classification as a confirmed carcinogen is given vinyl chloride and a TLV of 5 ppm as a time-weighted average is suggested. If this value is not exceeded, there should be no increase in the incidence of cancer, especially angiosarcoma of the liver.

Limits adopted in other countries, subsequent to the surfacing of vinyl chloride exposure associated cancers, as are follows, according to a 1977 summary: Australia (1973) 25 ppm; Finland (1975), Holland (1973), Poland (1976), Switzerland (1976) and USSR (1977) about 10 ppm; Italy (1975) 5 ppm; Japan (1975) and Sweden (1978) 1 ppm.

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VINYL CYANIDE

See, **ACRYLONITRILE**

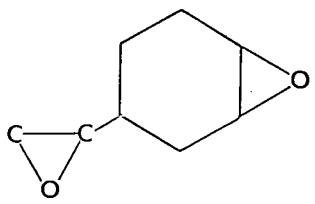
VINYLCYCLOHEXENE DIOXIDE

Vinylhexane dioxide

$C_8H_{12}O_2$

Skin

TLV, 10 ppm (≈ 60 mg/m³), Appendix A2 — Suspected Carcinogen



A colorless liquid which has a specific gravity of 1.0986 at 20° C, vinylcyclohexene dioxide's molecular weight is 140.18. Its freezing point is -108.9° C and boiling point is 228° C. The open cup flash point is 230° F and the viscosity is 7.77 centipoise at 20° C. The vapor pressure is 0.1 mm Hg at 20° C and is very soluble in water.

Vinylcyclohexene dioxide has been used by the plastic industry since the 1950's in the formation of polymers and other types of organic syntheses.[1,2]

In the United States, industrial experience over the past 10 to 20 years has been good.[3] Limited toxicology studies have been conducted. The single oral LD_{50} for rats is 2.8 g/kg of body weight and it is considered to be a mild, acute hazard when ingested.[4] In rabbits, skin penetration following a single application is considered to be a definite hazard. The LD_{50} by this route is reported to be 0.62 mL/kg body weight.[4] Skin irritation is also considered to be a definite hazard. Application of the undiluted compound caused edema and redness comparable to a mild to moderate first degree burn in the rabbit. Human experience indicates that it is a mild to moderate skin irritant[4] though Dernehl[3] states that on occasion, instances of marked skin irritation have been reported. Dernehl further reports one case of severe vesiculation of the skin of both feet when a worker wore shoes previously contaminated with this compound. When tested in the guinea pig, skin sensitization occurred only infrequently. When the compound was applied to the skin of mice as a 30% solution in acetone, many mice survived to a normal life span. *"Skin tumors and some cancers developed in the survivors late in their life span."*[4] Dernehl also states that in 10 years of experience not a single case of systemic illness has been reported and adds, *"We do not know of a single incidence of skin cancer in humans working with vinylcyclohexene dioxide to this date."* (Emphasis added.) Eye injury is considered to be a definite hazard.[4]

A summary of information on various potential occupational hazards includes the following statement:[5]

"Vinyl cyclohexene dioxide is irritating to the eyes and skin of rabbits. It can cause acute respiratory tract irritation and congestion of the lungs. It has been associated with testicular atrophy, leukopenia, and necrosis of the thymus. Several studies have demonstrated the carcinogenicity of vinyl cyclohexene dioxide in rodents. Vinyl cyclohexene dioxide also inhibits the growth of Walker cell carcinomas in rats. No studies have been encountered on the effects of this compound in humans."

The 4-hour LC_{50} for rats is given as 800 ppm.[6] If the reported vapor pressure of 0.1 mm at 20° C is correct, corresponding to 133 ppm, air, even at 30° C, could not contain 800 ppm of vapor except in a state of supersaturation. Hence, the risk by inhalation is considered slight.

Because of very limited toxicological data and because of demonstrated carcinogenicity when the compound is applied to the skin of the mouse, extreme caution should be exercised in the use of this diepoxide. The recommended TLV is 10 ppm and an A2 (experimental carcinogen) designation should be considered until further and adequate data are available.

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VINYLIDENE CHLORIDE

1,1-Dichloroethylene

$CH_2 = CCl_2$

TLV, 10 ppm (≈ 40 mg/m³)

STEL, 20 ppm (≈ 80 mg/m³)

A volatile, colorless liquid which polymerizes readily, vinylidene cholride has a mild sweet odor, a molecular weight of 96.96 and a specific gravity of 1.2129 at 20° C. It boils at 37°C, freezes at -122° C and has a closed cup flash point of 14° F. Insoluble in water, vinylidene chloride is soluble in most organic solvents.

It is used almost exclusively as a co-polymer with vinyl chloride, e.g., as an intermediate in the production of "vinylidene polymer plastics",[1] such as Saran® and Velon®.

Pendergast *et al*[2] exposed rats, rabbits, guinea pigs and monkeys, 8 hours/day, 5 days/week for 6 weeks to 395 mg/m³ (100 ppm). They reported no visible signs of toxicity during exposure but that the rabbits and monkeys lost weight. Serum-urea-nitrogren was normal in guinea pigs and no pathological changes attributable to exposure were observed in any species. They concluded that vinylidene chloride is less toxic than carbon tetrachloride.

This same group exposed these species 23.5 hours/day for 90 days to 189, 101, 61 and 20 mg/m³ (47, 25, 15 and 5 ppm v/v). Mortality appeared to have been increased at the first three exposure levels, however the mortality at 20 mg/m³ did not appear to be elevated. Numerous other effects apparently due to exposure were noted at 189 mg/m³ but were less certain at lower levels. Again these workers concluded that continuous exposure to vinylidene chloride appeared to cause less injury than continuous exposure to carbon tetrachloride, although mortality in animals exposed to vinylidene chloride may have been greater than in those similarly exposed to carbon tetrachloride.

Gage[3] reported that 20 six-hour exposures to 500 ppm caused nasal irritation, retarded weight gain and caused liver cell degeneration, while only nasal irritation was observed in rats exposed to 200 ppm.

The results of 6-month repeated inhalation studies on rats, rabbits, guinea pigs and dogs are reported in Patty.[4] Animals exposed 5 days/week, 8 hours/day for several months showed some injury of the liver and kidneys even at 25 ppm. Vinylidene chloride was considered to be quite comparable in toxicity to carbon tetrachloride.

XYLENE

$C_6H_4(CH_3)_2$

o-, m-, p-isomers

TLV, 100 ppm (≈ 435 mg/m³)
STEL, 150 ppm (≈ 655 mg/m³)

Xylene is a clear, flammable liquid with an aromatic hydrocarbon odor and a molecular weight of 106.16. Commerical xylene is a mixture of three isomers, ortho, meta and para, with the meta form usually the principal component. According to Gerarde,[1] 6 to 15% of ethyl benzene may also be present. The physiochemical properties of the three isomers, meta, ortho, para, respectively, are: specific gravity, 0.8684, 0.08801 and 0.86104; boiling point, 138.8, 144 and 138.5° C; melting point, -47.4, -25 and -13 to -14° C. The vapor pressure at 25° C is between 7 and 9 mm Hg. The boiling ranges and flash points of the commercial product depends on its grade; the 10% grade boils between 135 and 145° C; closed cup flash points are from 81 to over 100° F. Xylene is insoluble in water, but miscible with absolute alcohol, ether and other organic solvents.

Xylene is present in gasoline and many petroleum solvents. It is used extensively as a solvent in paints and other coatings, especially the alkyl resin type, and in rubber cements. Meta-xylene is an intermediate in the preparation of isophthalic acid; ortho-xylene in the manufacture of phthalic anhydride; para-xylene in the synthesis of terephthalic acid. All isomers are used in making drugs, dyes and insecticides.

Fairhall[2] considered the effects of xylene similar to those of toluene, but Gerarde[1,3] stated that the acute toxicity of the xylenes was higher.

Fabre and Truhaut[4] exposed rats and rabbits to a mixture of xylene isomers at a concentration of about 690 ppm for eight hours a day, six days a week. After 130 days no significant deviations from normal in the peripheral blood were found. A decrease in red and white cell counts and an increase in the platelet count in the blood of rabbits followed similar exposures at 1150 ppm for 55 days. Reversible lesions in the cornea of cats exposed to xylene were observed.

Gerarde[1] listed headache, fatigue, lassitude, irritability and gastrointestinal disturbances such as nausea, anorexia and flatulence as the most frequent symptoms among workers exposed to xylene. A report which suggested that xylene might affect the heart and vascular system was cited.

Browning[5] also recorded reports of gastrointestinal as well as neurological disturbances, and injury to heart, liver, kidneys and the nervous system among workers with xylene exposure. In addition, she noted a number of reports of blood dyscrasias, some of them fatal, associated with exposure to xylene. De Oliveira[6] described the death from aplastic anemia of a lithographer who used xylene for several years; and Goldie[7] reported a patient who had an apparent eplieptiform seizure following relatively brief exposure to xylene vapor.

Gerarde,[1] however, considered that industrial experience confirmed the animal experimentation evidence that xylene is not a myelotoxicant. Goldwater[8] was of the opinon that xylene was probably less toxic than toluene to the bone marrow. In most of the cases of blood disease associated with xylene, the presence of benzene as an impurity was not ruled out.

Nelson and associates[9] found 200 ppm of xylene definitely irritating to the eyes, nose and throat of experimental human subjects. Greenburg and Moskowitz[10] suggested a maximum allowable concentration of 200 ppm. Cook,[11] Smyth,[12] Elkins[13] and Gerarde[2] all considered this value too high, and Gerarde suggested 100 ppm as a more acceptable limit.

The NIOSH criteria document on xylene, published in 1975,[14] refers to a report by Morley *et al*[15] in which renal impairment and some evidence of disturbance of liver function were noted in three workers who were overcome by a gross overexposure to xylene (estimated concentration, 10,000 ppm): one worker died, the others suffered from amnesia and did recover, slowly however. A paper by Matthaus describes corneal changes in furniture polishers exposed to xylene in unknown concentrations.[16]

In a study of various hydrocarbon solvents, Carpenter *et al*[17] found the 4-hour LC_{50} for rats to be 6700 ppm. The *no-ill-effect* concentration for rats and dogs, following 65 days (6 hours/day, 5 days/week) was 800 ppm. Sensory response experiments with human subjects indicated a hygienic standard of around 200 ppm.

The TLV of 100 ppm, first adopted in 1967, is retained with a STEL of 150 ppm. It is believed that irritant effects will be minimal, and that no significant degree of narcosis or chronic injuries will result from continued occupational exposure at that level.

NIOSH[14] also recommended a workplace environmental standard of 100 ppm, as a TWA, with a ten minute ceiling of 200 ppm.

Other recommendations: ANSI (1970) 100 ppm; West Germany (1974) 200 ppm; Sweden (1975) 100 ppm; Czechoslovakia (1969) and East Germany (1973) 45 ppm; USSR (1972) 11 ppm.

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m-XYLENE α, α'-DIAMINE

MXDA

$C_6H_4(CH_2NH_2)_2$

Skin

CEILING LIMIT, 0.1 mg/m³

MXDA is a colorless liquid with a molecular weight of 136.19. It boils at 247° C and solidifies at 141.1° C. With a measured vapor pressure of 15 mm at 145° C, the calculated vapor pressure at 25° C is about 0.03 mm Hg. It has a open cup flash point of 273° F and is miscible with water and alcohol, but only partially soluble in paraffin hydrocarbon solvents.

Polyamide fibers and resins made from MXDA have a number of useful properties. It is also used as a curing agent for epoxy resins, and as a source of m-xylylene diisocyanate.

Two studies have indicated it to have a rather low oral acute toxicity to the rat (1500 and 930 mg/kg, respectively,[1] but, to be strongly irritating to the skin. A dermal LD_{50} of 2000 mg/kg was found for rabbits.[2] The undiluted compound was corrosive to the skin of guinea pigs, and a 50% emulsion in an acetone-dioxane mixture was severely irritating, but little effect was produced by a concentration of 10%.[1] A 10% aqueous solution, however, caused severe erythema and irritation, yet repeated application of a 5% concentration was needed to produce swelling and redness.[2]

In one study evidence of mild sensitization was found following repeated application to guinea pig skin,[1] but this finding was not duplicated in the second investigation.[2]

Exposure of rats for one hour to an aerosol of MXDA, at measured concentrations ranging from 1.74 to 6.04 mg/liter, resulted in eye irritation, lacrimation and labored breathing.[2] No deaths occurred during exposure, but several animals died within 48 hours, and a few more later, up to 14 days, the end of the observation period. Of the animals which survived, female rats showed reduced weight gain, while that of males was near normal.

At necropsy macroscopic abnormalities were found chiefly in the lungs, however changes in liver and kidneys were also noted. The LC_{50} for a one hour exposure and 14 day observation period was 3.75 mg/L, or about 700 ppm.

In comparison with the better known phenylene diamine (q.v.), the dermal effects of MXDA seem similar, but the oral toxicity appears less. By analogy, a ceiling limit of 0.1 mg/m³ is recommended for MXDA, until nore information is available. At this concentration, the compound should be largely in the vapor state.

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XYLIDINE

$(CH_3)_2C_6H_3NH_2$

Skin

TLV, 2 ppm ($\approx$ 10 mg/m³)

STEL, 10 ppm ($\approx$ 50 mg/m³)

Xylidine is a pale yellow to brown liquid with a molecular weight of 121.18. Commerical xylidine, a mixture of isomers, has a specific gravity of 0.97 to 0.99 and a boiling range from 213 to 226° C. It has a flash point of 202° F and a reported vapor pressure of $\leq$ 1 mm Hg at 20° C. Sparingly soluble in water, xylidine is miscible with alcohol and ether.

It is a raw material in the manufacture of dyes, pharmaceuticals and other organic compounds.

Von Oettingen and co-workers[1] found that by oral administration to dogs xylidine was less toxic than aniline, and resulted in very much less methemoglobin formation. Similar results were obtained by cutaneous application. With cats, however, xylidine and aniline were approximately equally toxic when applied to the skin, although the former acted much more slowly.

Upon inhalation, the LC_{50} for seven hours for mice was 149 ppm for xylidine and 188 ppm for aniline.

Repeated exposure to xylidine at 45 ppm seven hours a day for 20 to 40 weeks resulted in mortality among dogs, cats and mice, but little or none among rats, rabbits, monkeys and chicks. Liver damage was noted in rats, cats, dogs and mice.

Treon and associates[2] compared the toxicities of xylidine and monomethyl aniline. The minimum lethal oral dose for rabbits was 620 mg/kg; by intravenous injection 240 mg/kg was lethal for rabbits, and 120 mg/kg for cats. In