EA Engineering, Science, and Technology, Inc.

Airport Professional Park
2350 Post Road
Warwick, Rhode Island 02886
Telephone: 401-736-3440
Fax: 401-736-3423
www.eaest.com

26 May 2010

Mr. Joseph T. Martella II, Senior Engineer
RIDEM - Office of Waste Management
Site Remediation Program
235 Promenade Street
Providence, RI 02908

RE: April 2010 Air Sampling Event Comment Letter
Alvarez High School, 333 Adelaide Avenue, Providence, Rhode Island
Case No. 2005-029
EA Project No. 14687.01

Dear Mr. Martella:

On behalf of the City of Providence Department of Public Schools, EA Engineering, Science, and Technology, Inc. (EA) is providing this summary of data collected at the referenced Alvarez High School site (the Site) on 21 April 2010.

In accordance with the Order of Approval and amendments (Amended OA) for this Site, your office was notified via telephone that three compounds, 1,2-Dichloroethane, Chloroform, and Methylene Chloride, were detected within several samples collected from the Alvarez High School at concentrations that exceed the State of Connecticut's Draft Proposed Indoor Residential Targeted Air Concentrations. The detections are detailed below:

- Chloroform
  - Standard: 0.5 $\mu g/m^3$
  - Cafeteria: 0.790 $\mu g/m^3$
  - Elevator Hallway: 0.71 $\mu g/m^3$

- Methylene Chloride
  - Standard: 3.0 $\mu g/m^3$
  - Kitchen Storage Rm: 5.54 $\mu g/m^3$

- 1,2-Dichloroethane
  - Standard: 0.07 $\mu g/m^3$
  - Room 152: 0.162 $\mu g/m^3$

Upon receipt of this detection, EA reviewed monitoring field notes and analytical results of subslab vapor sampling, which was conducted concurrently with the indoor air sampling. Monitoring notes indicate the SSD System continues to operate effectively in accordance with design. Analytical results indicate that 1,2-Dichloroethane was not detected in any samples collected from subslab vapor sampling points. Methylene chloride was detected in one subslab vapor point (IMP-3), but at a lesser concentration (1.74 $\mu g/m^3$) than detected in the indoor air.

Chloroform was detected in two subslab vapor sampling points (IMP-1 at 0.220 $\mu g/m^3$ and IMP-3 at 0.200 $\mu g/m^3$) but also at lesser concentrations than those detected in the indoor air. The absence of these contaminants at concentrations greater than those observed within the school indicates that subslab vapor intrusion is not the source of these detections.

Additionally, EA routinely measures the vacuum at 11 soil vapor monitoring points throughout the school using a Magnahelic vacuum gauge capable of measuring to 0.01 inches of water. The results indicate that a vacuum is being maintained by the SSD system at each sampling point. Therefore, controlled prevention of the soil vapors from entering the school is being maintained (Attachment E).

In accordance with the Amended OA, EA conducted a survey of the school to attempt to determine the source of the detected compounds. No potential sources were identified during the survey. However, after the survey a representative of the Providence School Department brought to our attention the use of a coil cleaner to clean the heating units within the school. The material safety data sheet (MSDS) of the coil cleaner was provided to EA for review. The MSDS indicates the coil cleaner is consists of 90-100% trichloroethylene (TCE). This compound can degrade to all of the chemicals which were detected in excess of the standards in April, as shown in Attachment A. According to the subcontractor responsible for maintenance at the school, the coil cleaner was previously used in June 2009. However, the TCE could have been present at similar concentrations and have not exceeded the TCE standard of 1.0 $\mu g/m^3$. Once the TCE degrades, the resultant 1,2-Dichloroethane exceeds the more stringent 0.07 $\mu g/m^3$.

Based on the above detailed rationale, EA proposes to continue regular quarterly air sampling and analysis and monthly monitoring. If these compounds persist, EA will prepare a response plan to manage the sustained exceedances.

To summarize, 1,2-Dichloroethane, Chloroform, and Methylene Chloride were detected within several areas during April 2010 indoor air sampling conducted at the Alvarez High School. EA will perform quarterly sampling in July 2010 and will review the results and determine if the presence of these compounds persists. However, according to the monitoring data collected in April 2010, the SSD System continues to operate effectively in accordance with design. Copies of the Analytical Reports for Indoor Air and Subslab Vapor sampling for April 2010 are provided in Attachments B and C, respectively. A letter from Alpha Analytical Laboratories attesting to the ability to reach the previously agreed upon reporting limits for the April 2010 analysis is provided as Attachment D.

EA did identify one potential issue relative to the floor slab in the kitchen area. A line of cracked tiles was noted at the entrance to the food service area. EA would recommend removing a tile to determine if the floor slab has been compromised. EA and the School Department are currently determining the logistics of this investigation.

No SSD system modifications or other actions to address current site conditions are warranted or proposed at this time. Your office will be notified if it is determined that this issue persists or if any other issues arise. If you have any questions or require additional information, please contact me at 401-736-3440, Ext. 202.

Sincerely,

EA ENGINEERING, SCIENCE,
AND TECHNOLOGY, INC.

Frank B. Postma, LSP, LEP, PG
Senior Project Manager

FBP/rgm

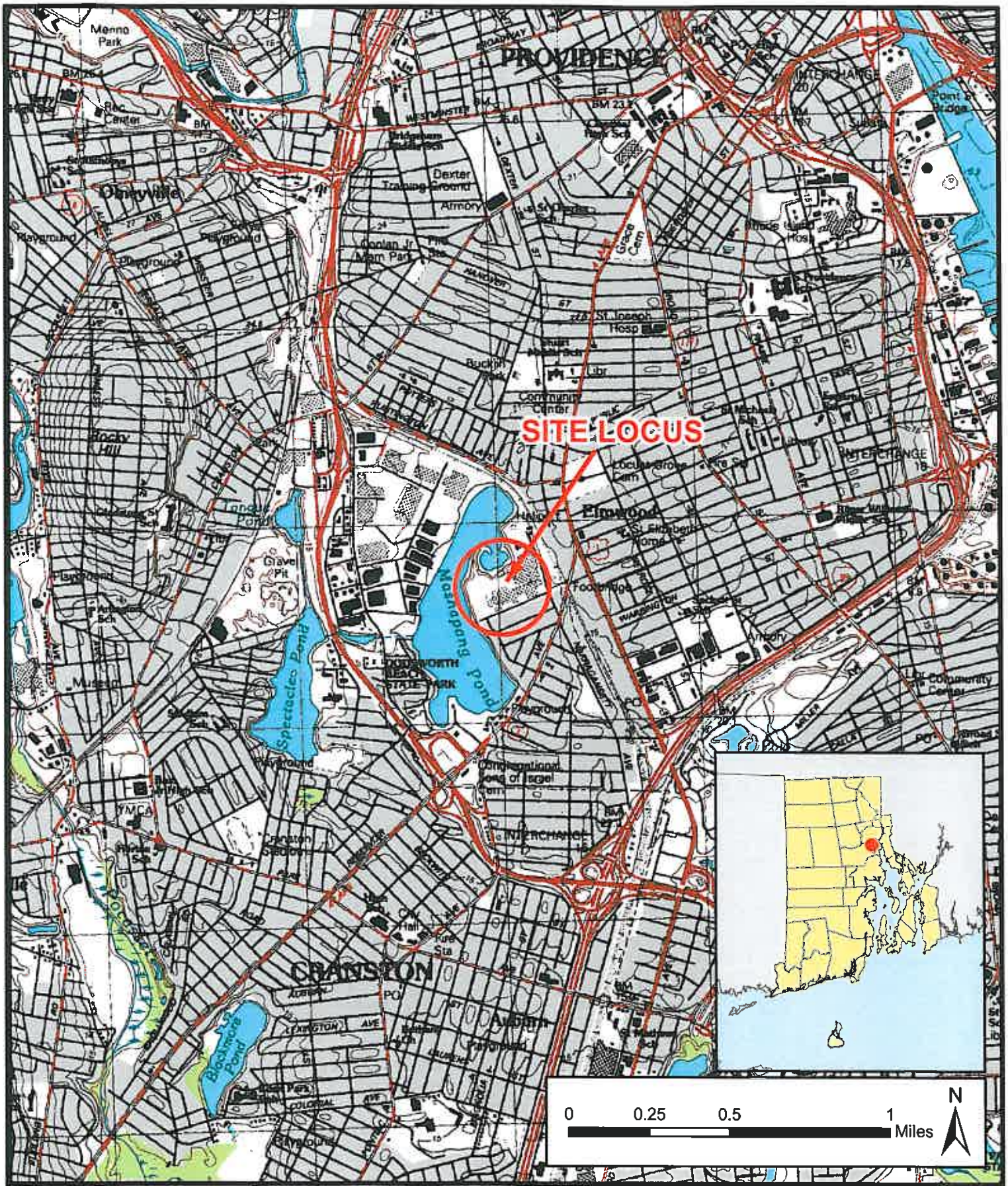
Figures
Figure 1: Site Locus
Figure 2: Indoor Air Sampling and Methane Monitoring Plan
Figure 3: As-Built Subslab Monitoring and Sampling Locations Plan

Attachments
Attachment A: Trichloroethylene Information
Attachment B: Indoor Air Analytical Report, 21 April 2010
Attachment C: Subslab Vapor Analytical Report, 21 April 2010
Attachment D: Alpha Analytical Reporting Limits Letter, 30 April 2010
Attachment E: Operation and Maintenance Form, 21 April 2010

cc: C. Jones, Prov. Dept. of Public Schools
T. Deller, Prov. Redevelopment Agency
J. Fernandez, City of Prov. Law Department
R. Dorr, Neighborhood Resident
Rep. Scott Slater
Knight Memorial Library Repository
A. Sepe, Prov. Dept. of Public Property
S. Fischbach, RI Legal Services
J. Ryan, Partridge, Snow, & Hahn
J. Pichardo, Senator
Principal Torchon, Alvarez High School

Figure 1

Site Locus Map



EA	ALVAREZ HIGH SCHOOL 333 ADELAIDE AVENUE PROVIDENCE, RHODE ISLAND					FIGURE 1 SITE LOCUS	
PROJECT MGR:	DESIGNED BY:	CREATED BY:	CHECKED BY:	SCALE:	DATE:	PROJECT NO:	FILE NO:
FP	PT	PT	FP	1:24,000	FEBRUARY 2010	14687.01	SITE_LOCUS.MXD

Figure 2

Indoor Air Sampling and Methane Monitoring Plan

Methane Sensor Location in West Wing

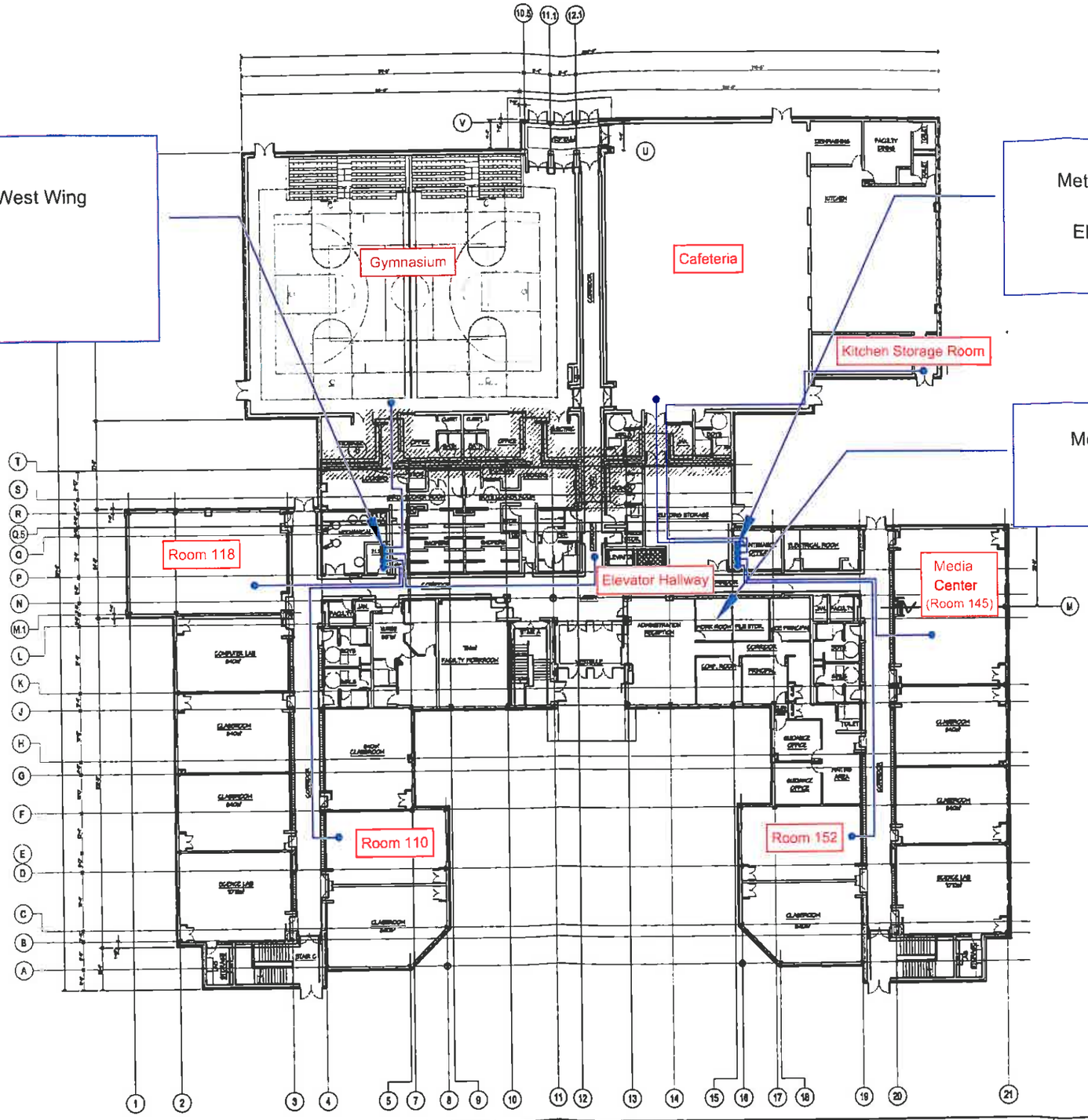
Electrical Room Area

Methane Sensor Location in East Wing

Electrical Room/Maintenance Office Area.

Methane System Controller Location

Adminstration Work Room



NOTE: NOT TO SCALE

PROJECT NORTH

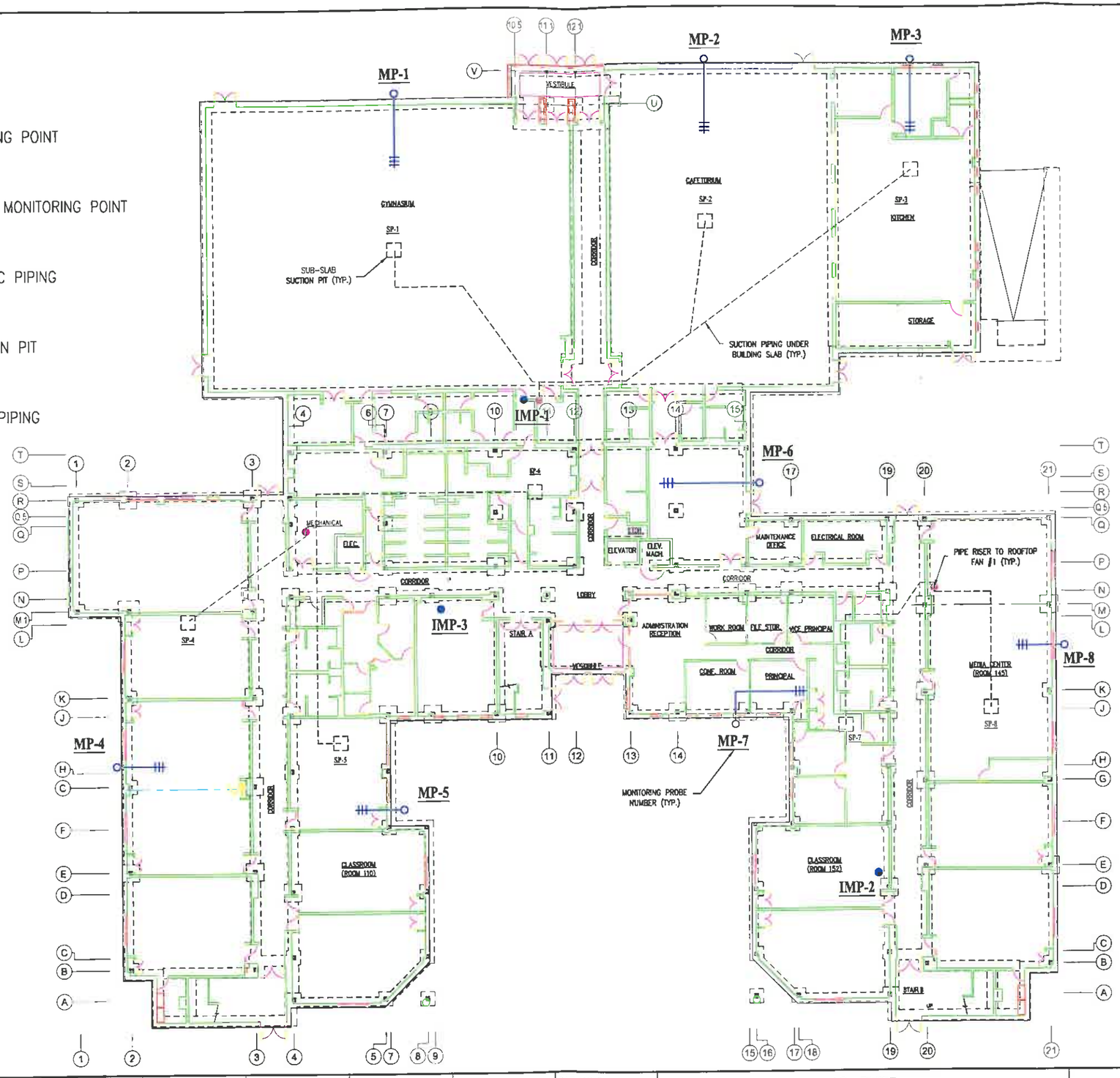
DESIGNED BY PMG	DRAWN BY PMG	DATE 4-3-07	PROJECT NO. 61965.01	FILE NAME Gorham Layout	INDOOR AIR SAMPLING AND METHANE MONITORING SYSTEM DIAGRAM - GORHAM HIGH SCHOOL PROVIDENCE, RHODE ISLAND
CHECKED BY PMG	PROJECT MGR. PMG	SCALE NTS	DRAWING NO. –	FIGURE N/A	QUARTERLY STATUS REPORT FIGURE 2

Figure 3

As-Built Subslab Monitoring and
Sampling Locations Plan

LEGEND :

MP-1	SUB-SLAB MONITORING POINT
IMP-1	INTERIOR SUB-SLAB MONITORING POINT
——||——	SLOTTED 1 INCH PVC PIPING
SP-1 ⬚	SSD SYSTEM SUCTION PIT
--------	SOLID 4 INCH PVC PIPING



EA®	DESIGNED BY PMG	DRAWN BY DMA	DATE AUG 27 2007	PROJECT NO. 14687.01	FILE NAME FIG 3	AS-BUILT SUB SLAB MONITORING AND SAMPLING LOCATIONS ALVAREZ HIGH SCHOOL PROVIDENCE, RHODE ISLAND
	CHECKED BY PMG	PROJECT MGR. PMG	SCALE NTS	DRAWING NO. N/A	FIGURE 3	QUARTERLY STATUS REPORT FIGURE 3

Attachment A

Trichloroethylene Information

INTERNATIONAL PROGRAMME ON CHEMICAL SAFETY

ENVIRONMENTAL HEALTH CRITERIA 50

TRICHLOROETHYLENE

This report contains the collective views of an international group of experts and does not necessarily represent the decisions or the stated policy of the United Nations Environment Programme, the International Labour Organisation, or the World Health Organization.

Published under the joint sponsorship of
the United Nations Environment Programme,
the International Labour Organisation,
and the World Health Organization

World Health Orgnization
Geneva, 1985

The International Programme on Chemical Safety (IPCS) is a joint venture of the United Nations Environment Programme, the International Labour Organisation, and the World Health Organization. The main objective of the IPCS is to carry out and disseminate evaluations of the effects of chemicals on human health and the quality of the environment. Supporting activities include the development of epidemiological, experimental laboratory, and risk-assessment methods that could produce internationally comparable results, and the development of manpower in the field of toxicology. Other activities carried out by the IPCS include the development of know-how for coping with chemical accidents, coordination of laboratory testing and epidemiological studies, and promotion of research on the mechanisms of the biological action of chemicals.

ISBN 92 4 154190 3

city or area or of its authorities, or concerning the delimitation of its frontiers or boundaries.

The mention of specific companies or of certain manufacturers' products does not imply that they are endorsed or recommended by the World Health Organization in preference to others of a similar nature that are not mentioned. Errors and omissions excepted, the names of proprietary products are distinguished by initial capital letters.

CONTENTS

TASK GROUP ON TRICHLOROETHYLENE

Members

Dr R.F. Addison, Department of Fisheries and Oceans, Bedford Institute of Oceanography, Dartmouth, Nova Scotia, Canada

Dr O. Axelson, Department of Occupational Medicine, University Hospital, Linköping, Sweden

Dr A. Di Domenico, High Institute of Health, Rome, Italy

Prof S.D. Gangolli, British Industries Biological Research Association, Carshalton, Surrey, United Kingdom

Prof M. Ikeda, Department of Environmental Health, Tohoku University School of Medicine, Sendai, Japan

Dr S.E. Jaggers, Central Toxicology Laboratory, ICI, Macclesfield, Cheshire, United Kingdom

Prof N. Loprieno, Laboratory of Genetics, University of Pisa, Pisa, Italy

Dr C. Maltoni, Oncology Institute and Tumour Centre "Felice Addari", Bologna, Italy

Dr J.H. Mennear, National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina

Dr A.C. Monster, Coronel Laboratory, University of Amsterdam, Amsterdam, The Netherlands

Prof I.V. Sanotsky, Research Institute of Industrial Hygiene and Occupational Diseases, USSR Academy of Medical Sciences, Moscow, USSR

Representatives from Other Organizations

Mr J. Wilbourn, International Agency for Research on Cancer, Lyons, France

Secretariat

Dr E.M. Smith, International Programme on Chemical Safety, World Health Organization, Geneva, Switzerland

Observers

Prof E. Malizia, Emergency Toxicological Service, Antivenom Center, Umberto the First Polyclinic, La Sapienza University, Rome, Italy

NOTE TO READERS OF THE CRITERIA DOCUMENTS

Every effort has been made to present information in the criteria documents as accurately as possible. In the interest of all users of the environmental health criteria documents, readers are kindly requested to communicate any errors found to the Manager of the International Programme on Chemical Safety, World Health Organization, Geneva, Switzerland, in order that they may be included in corrigenda, which will appear in subsequent volumes.

In addition, experts in any particular field dealt with in the criteria documents are kindly requested to make available to the WHO Secretariat any important published information that may have inadvertently been omitted and which may change the evaluation of health risks from exposure to the environmental agent under examination, so that the information may be considered in the event of updating and re-evaluation of the conclusions contained in the criteria documents.

* * *

A detailed data profile and a legal file can be obtained from the International Register of Potentially Toxic Chemicals, Palais des Nations, 1211 Geneva 10, Switzerland (Telephone no. 988400 - 985850).

ENVIRONMENTAL HEALTH CRITERIA FOR TRICHLOROETHYLENE

Following the recommendations of the United Nations Conference on the Human Environment held in Stockholm in 1972, and in response to a number of resolutions of the World Health Assembly (WHA23.60, WHA24.47, WHA25.58, WHA26.68), and the recommendation of the Governing Council of the United Nations Environment Programme, (UNEP/GC/10, 3 July 1973), a programme on the integrated assessment of the health effects of environmental pollution was initiated in 1973. The programme, known as the WHO Environmental Health Criteria Programme, has been implemented with the support of the Environment Fund of the United Nations Environment Programme. In 1980, the Environmental Health Criteria Programme was incorporated into the International Programme on Chemical Safety (IPCS). The Programme is responsible for a series of criteria documents.

A WHO Task Group on Environmental Health Criteria for Trichloroethylene was held in Rome from 10 to 15 December, 1984. Dr E.M. Smith opened the meeting on behalf of the Director-General. The Task Group reviewed and revised the draft criteria document and made an evaluation of the health risks of exposure to trichloroethylene.

The draft criteria document was developed by the ISTITUTO SUPERIORE DI SANITA, Rome, Director PROFESSOR F. POCCHIARI; the principal author was DR A. DI DOMENICO.

The efforts of all who helped in the preparation and finalization of the document are gratefully acknowledged.

* * *

Partial financial support for the publication of this criteria

document was kindly provided by the United States Department of Health and Human Services, through a contract from the National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina, USA - a WHO Collaborating Centre for Environmental Health Effects. The United Kingdom Department of Health and Social Security generously covered the costs of printing.

1. SUMMARY AND RECOMMENDATIONS FOR FURTHER RESEARCH

1.1. Summary

1.1.1. Properties and analytical methods

Trichloroethylene is a colourless liquid with a characteristic, slightly sweet odour. It is used as a solvent in a variety of applications. There are a number of techniques suitable for the determination of trichloroethylene including colorimetry, infra-red spectroscopy, gas-liquid chromatography (GLC), and gas chromatography/mass spectrometry. In GLC, the use of flame ionization detection gives good sensitivity; however, electron capture detection is markedly more sensitive. Methods are available for the determination of trichloroethylene in blood, fat, other tissues, food, water, etc.

1.1.2. Uses and sources of exposure

A major use of trichloroethylene is in metal degreasing; other significant uses are in textile cleaning, solvent extraction processes, and as a carrier solvent. It is no longer used as a grain fumigant and is now only occasionally used in anaesthesia. For practical use, trichloroethylene requires the addition of stabilizers (up to 2%).

There may be exposure to both the vapour and the liquid in the workplace, the highest atmospheric concentrations occurring in open degreasing processes. Trichloroethylene may be emitted from industrial plants in the form of a vapour and in aqueous effluent.

The major part of the annual world production of trichloroethylene (estimates range from 60 to 90%) is released into the environment.

1.1.3. Industrial exposure

Exposure in the workplace is mainly through inhalation of trichloroethylene vapour, but skin contamination with the liquid also occurs. The highest levels of occupational exposure occur in metal cleaning processes. Atmospheric trichloroethylene concentrations up to several hundred mg/m^3 have been recorded. Exposure during the actual production of trichloroethylene is relatively low because of the nature of the process. Oral intake is insignificant in occupational terms.

1.1.4. Environmental transport and distribution

Contamination of water has been reported but, with the exception of contamination of water supplies through accidental spillage, levels have been very low. Trichloroethylene is probably widely distributed in the environment, but usually only at fairly low levels, i.e., in the µg/kg range in sediments, in the low µg/litre range in natural waters, in the low µg/m^3 range in air, and in the µg/kg range in aquatic biota. The limited toxicity data available show LC_{50} values for aquatic biota in the mg/litre range. Trichloroethylene is degraded in biological and abiotic systems; in air (where most environmental trichloroethylene is expected to occur), its lifetime is about 10 days. It seems unlikely that the present rate of release of trichloroethylene into the environment would contribute significantly to depletion of the stratospheric ozone layer.

1.1.5. Absorption, distribution, biotransformation, and elimination

The most significant uptake of trichloroethylene is through inhalation of the vapour, but uptake can also take place through

the skin or via the gastrointestinal tract. Inhalation exposure is monitored by determining time-weighted average atmospheric concentrations.

Following absorption, trichloroethylene is rapidly distributed and accumulates in the adipose tissue. It easily crosses the placental barrier. Trichloroethylene is eliminated unchanged in exhaled air and, to a lesser extent, in faeces, sweat, and the saliva. It is rapidly metabolized, mainly in the liver.

At least 4 mammalian metabolites of trichloroethylene have been identified: trichoroethanol, trichloroacetic acid, 2-hydroxyacetylethanolamine, and oxalic acid; dichloroacetic acid appears to be specific to mice. The major metabolites in human beings, trichloroethanol and trichloroacetic acid, are excreted in the urine. Estimations of levels of these major urinary metabolites or total trichloro compounds in urine may be used for the biological monitoring of exposure.

There are species differences in the rate of metabolism of trichloroethylene to trichloroacetic acid, the rate in the mouse being more rapid than that in the rat. Isolated hepatocytes obtained from the mouse and the rat accurately reflect the *in vivo* metabolic rates. Isolated human hepatocytes metabolize trichloroethylene to trichloroacetic acid at a slower rate than rat hepatocytes.

In man, the metabolism of trichloroethylene decreases when ethanol has been ingested, and intolerance may occur.

1.1.6. Effects on experimental animals

Trichloroethylene is a moderately toxic substance. In terms of acute toxicity, LC_{50} values in rodent test species range from 45 to 260 mg/m^3, and oral LD_{50} values range from 2400 to 4920 mg/kg body weight. The toxic effects of exposure are related to a depressant action on the central nervous system. Central nervous system depression can lead to coma and death. Liquid trichloroethylene has an irritant effect on the skin and eyes; trichloroethylene vapour is irritant to the respiratory tract. Toxic effects on the kidneys are produced in rats by long-term oral administration. Minimal changes in the kidneys can occur after oral administration of 100 mg/kg body weight per day for 13 weeks and nephrotic changes can be found following oral administration of 500 mg/kg body weight per day, for 2 years. In mice, toxic effects on the kidney occur after oral administration of 3000 mg/kg per day, for 13 weeks, and mild nephrotic changes following 1000 mg/kg per day, for 2 years. Also, in mice, oral administration of 6000 mg/kg body weight per day for 13 weeks produced necrotic changes in the liver. Continuous exposure of mice by inhalation to 810 mg/m^3 trichloroethylene for 2 days resulted in an increased relative liver weight, which decreased following cessation of exposure.

Some immunological changes have been observed in rodents exposed to trichloroethylene by inhalation at concentrations between 10 and 1000 mg/m^3 for several weeks and also in those given trichloroethylene in their drinking-water (0.1 - 5 g/litre) for a similar period.

Trichloroethylene does not cause any biologically significant embryotoxic or teratogenic effects.

The evidence for mutagenic effects is inconclusive.

There is clear evidence that trichloroethylene is carcinogenic in mice with lifetime (2-year) exposures to 1620 mg/m^3 by inhalation or oral adiministration of 700 - 1200 mg/m^3 body weight per day. There is some evidence that trichloroethylene causes tumours in rats; a low incidence of renal tumours occurred in rats exposed for 2 years to levels of 3240 mg/m^3 by inhalation or 500 - 1000 mg/kg per day by the oral route. There are species and strain differences in carcinogenic response and the purity of the trichloroethylene and the nature of any additives affect the

outcome.

1.1.7. Effects on man

The signs and symptoms of over-exposure in human beings are mainly related to the central nervous system; for example, headache, drowsiness, hyperhydrosis, tachycardia, and, in more severe cases, stupor and coma. Trichloroethylene is analgesic and anaesthetic; inhalation of concentrations between 27 000 mg/m^3 (5000 ppm) and 108 000 mg/m^3 (20 000 ppm) have been used in anaesthetic procedures.

Fatalities have been reported through accidental or suicidal over-exposure to trichloroethylene. In general, the lethal oral dose for an adult is of the order of 7000 mg/kg body weight, but a death has occurred following a single dose of 50 ml (75 g). Deaths have been reported following inhalation of trichloroethylene, including a number that have occurred during anaesthetic procedures. While respiratory depression cannot be excluded, it is more likely that cardiac arrest, related to the arrhythmic properties of trichloroethylene, was the cause of death.

In laboratory and work-place studies, demonstrable psychomotor impairment was found following inhalation exposure to 5400 mg/m^3 (1000 ppm) for 2 h, and reaction time was increased by exposure to a concentration of 1320 mg/m^3 (245 ppm), under work-place conditions.

Effects on the respiratory and gastrointestinal tracts are related to the irritant properties of trichloroethylene. Irritation of mucous membranes occurs with exposure to trichloroethylene vapour at concentrations of 810 - 3510 mg/m^3 (150 - 650 ppm). At autopsy, following fatal ingestion, lesions of the gastrointestinal tract have been found.

Liquid trichloroethylene and its vapour at anaesthetic concentrations (27 500 - 108 000 mg/m^3) cause eye irritation and superficial corneal damage, which normally recovers completely. Liquid trichloroethylene is mildly irritating to the skin but, if it is held in contact for any length of time, for example, by clothing or footwear, it can produce marked skin irritation with blistering. Repeated contact produces defatting of the skin and dermatitis.

High oral doses (200 - 300 ml or more), taken suicidally or through misuse, have produced toxic effects on the liver and kidneys. Hepatic necrosis and nephropathy have been found at autopsy. The use of trichloroethylene in a confined unventilated space for 3 - 4 h has also resulted in liver and kidney damage. Addiction to trichloroethylene ("vapour sniffing") has produced liver and kidney damage, and deaths have occurred.

Chronic neurotoxic effects may occur, and a "psychoorganic syndrome", with lassitude and depression, has been described but has not been found consistently in studies on groups of trichloroethylene workers. It is probable that many of the effects described were due to the metabolites of trichloroethylene.

Degeneration of cranial nerves has occurred following short-term exposures to high levels of trichloroethylene, generally in enclosed spaces. However, it is considered that the cranial neuropathy is probably due to breakdown products, mainly dichloroacetylene, rather than to trichloroethylene itself. Polyneuropathies have been reported following long-term exposure.

Data from epidemiological studies on carcinogenicity in occupationally exposed groups are inconclusive.

1.2. Recommendations for Further Research

1. The toxic action and thresholds for toxic effects of trichloroethylene in human beings and experimental animals, at low levels of short- and long-term exposure, need to be defined in more detail.

2. Studies are required for a full evaluation of the genotoxicity of trichloroethylene. Trichloroethylene samples of high purity (with full data on the nature and the amount of any impurities) should be used as well as trichloroethylene samples stabilized with non-mutagenic compounds (e.g., amines).

3. The significance for human beings of the effects seen in rodents with long-term exposures requires further study. The role of metabolism in carcinogenesis, both the rates and the metabolites formed, and the production of biochemical responses that may be the mechanisms of carcinogenic responses in target tissues require further study and interspecies comparison.

4. In view of the equivocal evidence for mutagenicity in bacterial and mammalian cell systems, there is an implication that epigenetic mechanisms may be involved in the carcinogenic effects observed in experimental animals.

   It should be noted that trichloroacetic acid produced by the metabolism of trichloroethylene has induced peroxisome proliferation, with differences in response in isolated hepatocytes from the mouse, rat, and human beings. Peroxisome proliferation has been implicated in the epigenetic induction of hepatocellular carcinoma in mice and rats.

5. The pathological significance of trichloroethylene-induced cytomegaly and karyomegaly of renal tubular cells and the incidence in untreated laboratory rodents of tubular renal carcinoma should be investigated.

6. There should be further epidemiological studies to investigate the possible carcinogenic effects of trichloroethylene exposure. Additional cohort studies should be initiated. Registers of TCA-monitoring data should be organized with epidemiological studies in mind. Case-control studies, particularly of haemolymphatic, pancreatic, and genito-urinary tract cancers, should specifically consider exposure to trichloroethylene in industry, dry-cleaning operations, and via food, such as decaffeinated coffee.

7. Biological monitoring should be extended and more attention paid to interindividual differences in toxicokinetics and to the factors responsible for these, such as anthropometric parameters, sex, genetic make-up, use of drugs and alcohol, and interactions with certain chemicals in the environment.

8. Workers with moderate levels of exposure to trichloroethylene tend to have an increased incidence of subjective symptoms. There should be a systematic approach to the clearer identification, analysis, and evaluation of such symptoms and their correlation with levels of occupational exposure in different industrial environments.

9. Although, at present, trichloroethylene does not appear to be a major environmental problem, this assessment is based on relatively few data describing its distribution in the environment, and its rates and routes of degradation. More comprehensive data should be obtained, and an assessment of geographical or temporal changes in trichloroethylene distribution should be made.

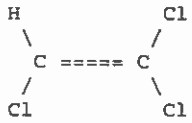
   There should be studies on controlling the input of trichloroethylene into the environment, and the provision of disposal methods other than incineration should also be studied.

2. IDENTITY, PROPERTIES AND ANALYTICAL METHODS

2.1 Identity

Trichloroethylene is an aliphatic substance of the organic halogen and halogen-derivative families.

Chemical structure:



Molecular formula:	C_2HCl_3
IUPAC and CAS name:	trichloroethene
Common synonyms:	acetylene trichloride, ethinyl trichloride, ethylene trichloride, 1-chloro-2,2-dichloroethylene, 1,1-dichloro-2-chloroethylene, 1,1,2-trichloroethylene, TCE, TRI
Common trade names:	Algylen, Anamenth, Benzinol, Blacosolv, Blancosolv, Cecolene, Chlorilen, Chlorylen, Circosolv, Densin-fluat, Dow-Tri, Dukeron, Fleck-Flip, Flock-Flip, Fluate, Gemalgene, Germ-algene, Lanadin, Lethurin, Narcogen, Narkosoid, Nialk, Perm-a-Chlor, Pet-zinol, Philex, Threthylen, Threthylene, Trethylene, Triad, Trial, Triasol, Trichloran, Trichloren, Triclene, Trielene, Trielin, Trielina, Triklone, Trilen, Trilene, Triline, Trimar, Triol, Tri-Plus, Tri Plus M, Vestrol, Vitran
CAS registry number:	79-01-6
Relative molecular mass:	131.40
Conversion factor	1 ppm trichloroethylene = 5.4 mg/m^3

2.2. Physical and Chemical Properties

2.2.1. Pure trichloroethylene

In its pure state, trichloroethylene is a colourless liquid with a characteristic, slightly sweet odour; the odour threshold for human beings is 540 mg/m^3 (100 ppm) (Torkelson & Rowe, 1982).

Some physical and chemical properties of pure trichloroethylene are listed in Table 1.

2.2.1.1. Chemical reactivity

Trichloroethylene oxidizes to yield acids, including hydrochloric acid (Aviado et al., 1976). Its reactivity increases with rise in temperature and with exposure to ultraviolet radiation (UVR). Under pressure, at 150 °C, it reacts with alkalis to produce glycolic acid. With sulfuric acid, it reacts to produce monochloroacetic acid (Kirk & Othmer, 1964). In the presence of alkali, dehydrochlorination may occur in solution as well as in the vapour phase, with the formation of dichloroacetylene, which is highly neurotoxic and carcinogenic for animals and probably for man (Henschler et al., 1970a).

2.2.1.2. Chemical degradation

The chemical degradation of trichloroethylene in water is very slow. In contact with red-hot metals or a direct flame, liquid or vapour-phase trichloroethylene decomposes to form phosgene and hydrogen chloride (Waters et al., 1977).

2.2.1.3. Photochemical degradation

Photochemical reactions initiate the degradation of trichloroethylene in the environment. When exposed to UVR and humidity, the compound decomposes to form acids that have mean half-lives ranging from 6 to 12 weeks (Correia et al., 1977). With

an $OH^{\cdot}$ concentration of the order of 10^6 molecules/cm^3 (accepted mean value), a calculated half-life of trichloroethylene is around 5 days (De More et al., 1983). Trichloroethylene exposure to xenon arc lamp radiation with a wavelength greater than 290 nm, at constant temperature, produces carbon monoxide, carbon dioxide, water, hydrogen chloride, dichloroacetyl chlorides, and phosgene; the phosgene hydrolyses to produce carbon dioxide and hydrogen chloride. Dichloroacetyl chlorides enter the hydrosphere as dichloroacetate anions (McConnell et al., 1975).

Table 1. Physical and chemical properties of trichloroethylene

Melting point (°C)	-84.8 (freeze)		Windholz et al. (1976)
	-87.1		Kirk & Othmer (1979)
	-73.0		CRC (1980)
Boiling point (°C)	86.7	(760 mm Hg)	Windholz et al. (1976)
	-43.8	(1 mm Hg)	Windholz et al. (1976)

Table 1. (contd.)

Specific gravity	1.46	(25/25 °C)	Snell & Hilton (1967)
	1.4904	(4/4 °C)	Windholz et al. (1976)
(vapour density; air = 1)	4.53	(25 °C)	Windholz et al. (1976)
(vapour, g/litre)	4.45	(86.7 °C)	Kirk & Othmer (1979)
Vapour pressure (torr)	5.4	(-20 °C)	Snell & Ettre (1970a)
	20.1	(0 °C)	Snell & Ettre (1970a)
	57.8	(20 °C)	Snell & Ettre (1970a)
	305.7	(60 °C)	Snell & Ettre (1970a)
Other properties:			
Refraction index (n_D)	1.4782	(20 °C)	Kirk & Othmer (1979)
(vapour)	1.001784	(0 °C)	Kirk & Othmer (1979)
Viscosity (cP)	0.58	(20 °C)	Kirk & Othmer (1979)
(vapour)	10 300	(60 °C)	Kirk & Othmer (1979)
Dielectric constant (epsilon)	3.42	(16 °C)	Kirk & Othmer (1979)
Coefficient of cubic expansion	0.00119	(0 - 40 °C)	Kirk & Othmer (1979)
Surface tension (dyn/cm)	26.4	(20 °C)	Kirk & Othmer (1979)
Critical temperature (°C)	271.0		Kirk & Othmer (1979)

Critical pressure (atm)	49.7		Kirk & Othmer (1979)
Dipole moment (debye)	0.90		Kirk & Othmer (1979)

Table 1. (contd.)

Heat of combustion (kcal/g)	1.751		Kirk & Othmer (1979)
Heat of formation (kcal/mole)	0.999		Kirk & Othmer (1979)
(vapour)	-7.00		Kirk & Othmer (1979)
Latent heat of vaporization (cal/g)	57.4	(86.7 °C)	Kirk & Othmer (1979)
Flammability flash point (°C)			
under various conditions	Non-flammable under normal working conditions; Vapours (12.5 - 90% v/v) in poorly-ventilated rooms at temperatures between 30 and 82 °C may ignite if in contact with high-temperature heat sources; Vapour ignites (t>25.5 °C) if mixed with pure oxygen (10.3-64.5% v/v)		Kirk & Othmer (1979) CRC (1967) ASCHIMICI (1980) Aviado et al. (1976)
ignition temp. (°C)	410		ASCHIMICI (1980)
danger of explosion:			
limits (% v/v in air)[a]	8.0 - 10.5 8.0 - 52.0	(25.5 °C) (100 °C)	Kirk & Othmer (1979)
oxidizing properties	none		ASCHIMICI (1980)
Solubility:			
in water (g/litre)	1.07	(20 °C)	Kirk & Othmer (1979)
	1.24	(60 °C)	Kirk & Othmer (1979)
in organic solvents	completely miscible with several organic solvents		Windholz et al. (1976)
in oil	miscible		Windholz et al. (1976)

Table 1. (contd.)

n-octanol/water partition coefficient (log)	Log K^o/w 2.42	Banerjee et al. (1980)
Organic carbon partition coefficient, K_{oc}	K^o/w x 0.6	Karickhoff et al. (1979)
Bioconcentration factor, K_B	K^o/w x 0.048	Mackay (1982)

[a] See section 3.2.1.1.

2.2.2. Commercial trichloroethylene

Trichloroethylene produced for chemical reagent uses has a minimum purity of 99.85%. The commercial product can contain impurities and stabilizers as shown in Table 2.

Table 2. Commercial trichloroethylene: examples of impurities and commonly-used stabilizers

Impurities	Stabilizers
carbon tetrachloride	pentanol-2
chloroform	thymol
1,2-dichloroethane	triethanolamine
trans 1,2-dichloroethylene	triethylamine
cis 1,2-dichloroethylene	2,2,4-trimethylpentene-1
pentachloroethane	cyclohexene oxide
1,1,1,2-tetrachloroethane	*n*-propanol
1,1,2,2-tetrachloroethane	iso-butanol
1,1,1-trichloroethane	*n*-methyl morpholine
1,1,2-trichloroethane	diisopropylamine
1,1-dichloroethylene	*n*-methyl pyrrole
bromodichloroethylene	methyl ethyl ketone
perchloroethylene	epichlorohydrin[a]
bromodichloromethane	
benzene	

[a] Now used to a much lesser extent commercially.

Possible impurities depend on the manufacturing route, the type and quality of feed stock used, the type of distillation equipment, and the technical specification being met. It is uncommon for any individual impurity to be present at a level in excess of 100 mg/kg and for the total impurities to exceed 1000 mg/kg; not all the impurities listed would be detected in any sample.

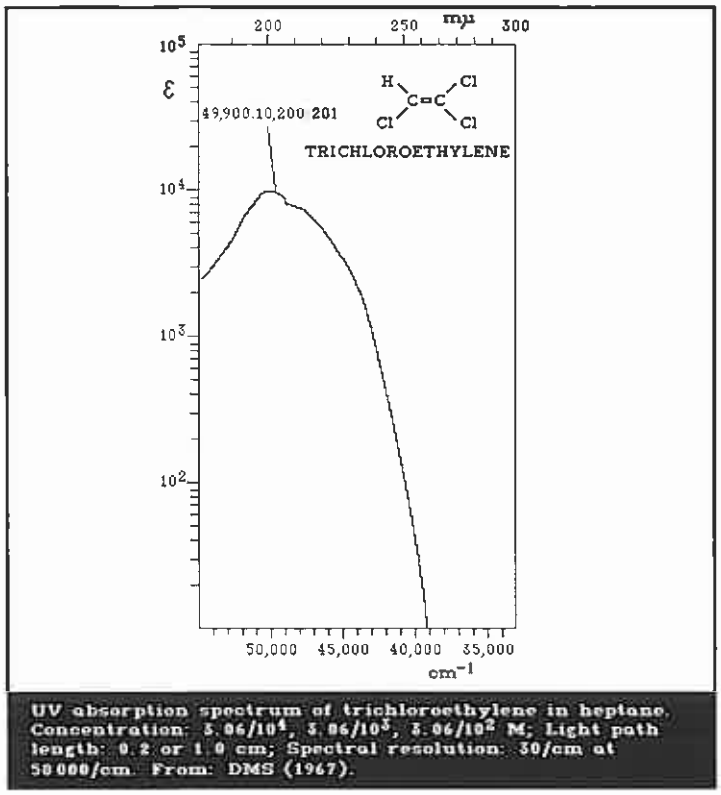
Stabilizers, in the form of antioxidants or acid-receptors (such as phenolic, olephinic, pyrrolic, and/or oxiranic derivatives and aliphatic amines), are usually added in concentrations that normally range from 20 to 600 mg/kg. However, in some cases, for limited quantities and special uses, concentrations as high as 5000 mg/kg are added. The stabilizers used will depend on patent ownership and the technical specification being met.

2.3. Analytical Methods

2.3.1. Identification and purity assessment

The degree of purity of trichloroethylene can be established by a number of methods, described by Snell & Ettre (1970a). Some spectral features of trichloroethylene are shown in Fig. 1 - 3.

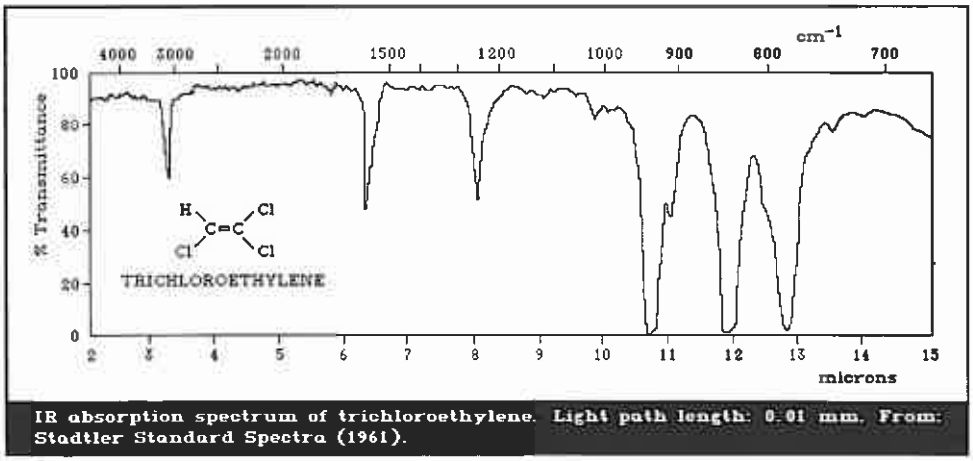
Trichloroethylene can be determined by the following analytical methods.



UV absorption spectrum of trichloroethylene in heptane. Concentration: $5.06/10^4$, $5.06/10^3$, $5.06/10^2$ M; Light path length: 0.2 or 1.0 cm; Spectral resolution: 30/cm at 50 000/cm. From: DMS (1967).

2.3.1.1. Colorimetry tests

In the Fujiwara test, trichloroethylene is treated with pyridine in an alkaline environment. Solution absorbance is then determined at 535 or 470 nm (absorptivity: 18 - 32 litre/g x cm) with a sensitivity of about 1 mg/kg. This test is suitable for other aliphatic halogenated compounds, and so is not substance-specific. Other complementary colorimetric tests that may enable trichloroethylene to be differentiated from other similar compounds have been reported by Snell & Ettre (1970b).



IR absorption spectrum of trichloroethylene. Light path length: 0.01 mm. From: Stadtler Standard Spectra (1961).

2.3.1.2. Infra-red spectroscopy

In the gaseous phase, quantities are determined by measuring the optical density of the mixture at the selected wavelength of 11.8 μm (847/cm). This corresponds to a detection sensitivity of not less than 0.5 μg/litre (Fishbein, 1973).

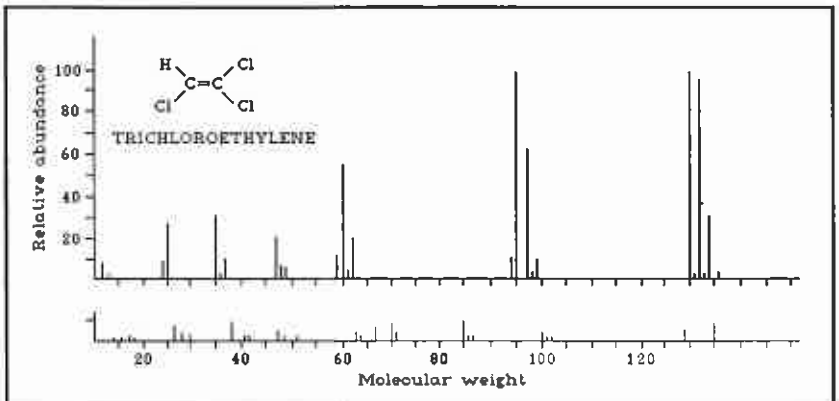
In solutions of carbon disulfide, trichloroethylene can be measured, even in the presence of similar chloro derivatives, using the specific band at 10.8 μm (926/cm) (Snell & Ettre, 1970b; Fishbein, 1973). Detection thresholds of some μg/litre can be

attained (Fishbein, 1973).

2.3.1.3. Gas-liquid chromatography

Generally, either packed or capillary columns (low-resolution or high-resolution chromatography, respectively) are used; the latter are recommended for complex mixtures containing substances similar to trichloroethylene. A number of stationary phases can be used, such as paraffinic hydrocarbons (squalene, hexadecane, paraffin), Apiezon L, Carbowax (600, 4000, 20M), silicones (SE-30, 550, SF-96-350), and arylphosphates. In general, detectors such as argon ionization or flame ionization detectors (sensitivity: ~10 ng) are suitable for several types of analyses; the thermoconductivity detector is little used, because it is less sensitive (sensitivity: ~250 ng). The detection threshold drops considerably (~0.02 ng in air) with electron-capture detectors (ECD). ECD response can be improved slightly by adding small quantities of oxygen to the carrier gas (Miller & Grimsrud, 1979).

Gas chromatography combined with mass spectrometry (GC/MS) is both highly selective and sensitive (Snell & Ettre, 1970b).



Mass spectrum of trichloroethylene. From: Stenhagen et al. (1974).

2.3.2. Determination in environmental media

2.3.2.1. Soil

High-resolution GC (hrGC-ECD) has been used for determining trichloroethylene in soil (De Leon et al., 1980). The hrGC/MS combination has been used as a confirmatory technique, with a detection threshold of ~10 mg/kg (10 ppm).

2.3.2.2. Water

Levels of trichloroethylene in water can be determined by hrGC/MS (Dowty et al., 1975), by GC with an electron-capture detector or an electrolytic conductivity detector (Nicholson et al., 1977; Dietz & Singley, 1979), and by various other techniques such as HPLC, hrGC, GC, and GC/MS (Eklund et al., 1978; Jungclaus et al., 1978). Where specified, detection thresholds are in the region of 1.0 µg/litre, or lower.

2.3.2.3. Air

GC/MS can be used to measure trichloroethylene levels in the urban atmosphere (Ioffe et al., 1977). Herbolsheimer et al. (1972), Sawicki et al. (1975), NIOSH (1977a), Heil et al. (1979), and Makide et al. (1979) describe sampling and sample enrichment techniques. Detection capacity may be as low as some ng/m^3.

Fujiwara's test can be used to measure trichloroethylene levels in air (Rush, 1970).

Gas detector tubes (Kitagawa, 1961), activated carbon tubes (NIOSH, 1977a; Shipman & Whim, 1980), and activated carbon felt badges (Hirayama & Ikeda, 1979) are available for use in work-place

environments. Gas detector tubes are suitable for spot sampling, while activated carbon tubes and felt are suitable for time-weighted average concentration determinations.

2.3.2.4. Foodstuffs

High- and low-resolution GLC can be used for the determination of trichloroethylene and other aliphatic chloro derivatives in various foodstuffs (Entz & Hollifield, 1982). The head-space technique is used in all cases. The sensitivity of the method appears to be higher (< 1 µg/kg) for water-rich samples than for fat-rich foodstuffs (10 -50 µg/kg). The coefficient of variation can be lower than 20%.

2.3.3. Determination in human tissues and fluids

The methods described in this section are those normally used to determine the levels of trichloroethylene or its major metabolites (trichloroacetic acid and trichloroethanol) in blood and urine. These methods can be used to obtain indirect measurements of exposure. There are methods for the determination of trichloroethylene and trichloroethanol in expired air.

2.3.3.1. Trichloroethylene

Blood or urine is distilled or aerated. Any trichloroethylene vapour present is collected in pyridine, which is then subjected to Fujiwara's colour test (Seto & Schultze, 1956; Tada, 1969).

Trichloroethylene detection and determination in blood and urine samples are now generally performed by GC, which has largely replaced colorimetric reactions. Samples are first extracted with solvents, and trichloroethylene concentrations are then determined in the extract. There are various modifications of this technique (Stewart et al., 1962; Stewart & Dodd, 1964; Kylin et al., 1967; Stewart et al., 1970; Ertle et al., 1972). Because of its volatility, trichloroethylene can be sampled using the head-space method (Monster & Boersma, 1975; Triebig et al., 1976; Astrand & Ovrum, 1976). This method has also been successfully coupled with GC/MS (Balkon & Leary, 1979).

GLC alone (Monster & Boersma, 1975; Astrand & Ovrum, 1976) and GC/MS (Barkley et al., 1980) have been used to determine trichloroethylene in exhaled air. The same methods, with suitable sampling techniques, may also be used for the determination of trichloroethylene in alveolar air.

2.3.3.2. Trichloroacetic acid

Fujiwara's colorimetry test is performed on the blood or urine sample extract or, in the case of urine, directly on the sample itself (Abrahamsen, 1960; Soucek & Vlachová, 1960; Bartonícek, 1962; Fawns, 1968; Tanaka & Ikeda, 1968; Tada, 1969; Weichardt & Bardodej, 1970; Ertle et al., 1972; Kimmerle & Eben, 1973; Mantel & Nothmann, 1977).

Trichloroacetic acid can also be determined by gas chromatography of the extract after methylation (Ehrner-Samuel et al., 1973; Ogata & Saeki, 1974; Nomiyama et al., 1978; van der Hoeven et al., 1979), by direct methylation of the specimen followed by head-space sampling and GC (Monster & Boersma, 1975; Triebig et al., 1976), or by inducing trichloroacetic acid decarboxylation and measuring the chloroform thus formed (Müller et al., 1972; Buchet et al., 1974). Ziglio (1979) determined trichloroacetic acid in subjects who had absorbed trichloroethylene in drinking-water. The extraction and methylation method, as well as the method of inducing thermal decarboxylation and then injecting the chloroform thus formed according to the head-space method, were used (sensitivity of extraction and methylation methods is better than 10 µg/litre).

2.3.3.3. Trichloroethanol

Trichloroethanol is found in the free state and as the glucuronide (urochloralic acid) in both blood and urine. For

total trichloroethanol determination, the glucuronide is hydrolysed. The trichloroacetic acid originally present is then removed and the trichloroethanol is oxidized quantitatively to trichloroacetic acid (Vlachov, 1957). The trichloroacetic acid thus formed is then measured by one of the methods previously described. Alternatively, trichloroethanol can be distilled in a vapour stream and measured colorimetrically on the basis of the condensate resulting from the reaction with pyridine and alkalis (Bardodej, 1962). The colour test can also be carried out without prior separation of trichloroethanol from trichloroacetic acid. The two compounds are measured by determining absorbance at 367 or 440 nm, and at 530 nm (Cabana & Gessner, 1967; Ogata et al., 1970; Mantel & Nothmann, 1977). Trichloroethanol can also be measured by determining the difference between the figure obtained for the trichloroacetic acid level and that obtained for all trichloro-derivatives present after quantitative oxidation to trichloroacetic acid (Seto & Schulze, 1956; Tanaka & Ikeda, 1968).

Trichloroethanol can be measured by gas chromatography after quantitative hydrolysis of the glucuronide (Ogata et al., 1970; Ertle et al., 1972; Kimmerle & Eben, 1973; Ogata & Saeki, 1974; Buchet et al., 1974; Nomiyama et al., 1978). The technique of head-space sampling has been used by Monster & Boersma (1975), Triebig et al. (1976), and Balkon & Leary (1979).

Trichloroethanol in exhaled air can be measured directly (Monster & Boersma, 1975).

2.3.3.4. Total trichloro derivatives

In the Imamura & Ikeda (1973) method, urine samples are oxidized with chromium trioxide in heated nitric acid, allowed to cool, and made alkaline; pyridine is added followed by mild heating (Fujiwara test). Solution absorbance is then determined at 530 nm.

2.3.4. Sensitivity

Generally speaking, colorimetric test detection thresholds range between 0.1 and 1 mg/kg. Greater sensitivity is provided by gas chromatography, which has detection thresholds of between 10 and 100 µg/kg for trichloroethylene, trichloroacetic acid, and trichloroethanol.

3. SOURCES IN THE ENVIRONMENT, USES, AND SAFE HANDLING

Trichloroethylene does not occur naturally.

It was first synthesized by Fisher in 1864 and became commercially available for the first time in 1908 in Austria and in the United Kingdom (Kirk & Othmer, 1964).

3.1. Production Processes, Levels, and Uses

3.1.1. Production processes and levels

Trichloroethylene is produced by three processes: the dehydrochlorination of *sym*-tetrachloroethane, the high-temperature oxychlorination of chlorinated products with one or two carbon atoms, or the chlorination of ethylene.

In Western Europe, production was approximately 250 000 tonnes in 1978. The major producing countries are the Federal Republic of Germany, France, whose individual production capacity is of the order of 100 000 tonnes, Italy, and the United Kingdom. Sweden and Spain are smaller producers. In the USA the production of trichloroethylene in 1979 was 130 000 tonnes (US ITC, 1980). In Japan, the annual production was approximately 74 500 tonnes in 1981 and 67 500 tonnes in 1982 (Japanese Yearbook of Chemical Industries Statistics, 1983).

3.1.2. Uses

Trichloroethylene is an industrial solvent mainly (85 - 90%) used for the vapour degreasing and cold cleaning of fabricated metal parts. Trichloroethylene has also been used as a carrier

solvent for the active ingredients of insecticides and fungicides; as a solvent for waxes, fats, resins, and oils; as an anaesthetic for medical and dental use; and as an extractant for spice oleoresins and for caffeine from coffee. Trichloroethylene has been used in printing inks, varnishes, adhesives, paints, lacquers, spot removers, rug cleaners, disinfectants, and cosmetic cleansing fluids. It may also be used as a chain terminator in polyvinyl chloride production and as an intermediate in the production of pentachloroethane (Defalque, 1961; Kirk & Othmer, 1963, 1979; Wetterhahn, 1972; Valle-Riestra, 1974; US CFR, 1976; Waters et al., 1977; IARC, 1979).

3.2. Handling Hazards, and Precautions

3.2.1. Handling hazards

3.2.1.1. Fire, explosion, and thermal decomposition

At normal handling temperatures, trichloroethylene behaves as a non-flammable, non-burnable substance. Under normal conditions, it is virtually impossible to induce an explosion with trichloroethylene. In the presence of air, at temperatures above 400 °C, it produces phosgene, hydrochloric acid, and carbon monoxide. In the vicinity of arc welding, phosgene and hydrogen chloride can be produced from trichloroethylene. In vapour degreasing, using combustion heaters, precautions must be taken to prevent solvent fumes from entering the combustion air. Containers of trichloroethylene exposed to fire should be cooled by sprinkling with water.

3.2.1.2. Chemical reactivity

Trichloroethylene is practically non-reactive with water at room temperature, under normal storage conditions, and the stabilized product does not undergo any changes in the presence of air, humidity, light, or in contact with metals. It is, however, a wise precaution not to expose the product to temperatures exceeding 130 °C.

In the presence of strong alkalis, particularly if heated, trichloroethylene produces dichloroacetylene (Reichert et al., 1980a), which is highly reactive and acutely neurotoxic to both animals and man (Reichert & Henschler, 1978). Dichloroacetylene is also potentially carcinogenic (Reichert et al., 1980b). Under normal circumstances in industrial use, this reaction is unlikely. However, under occasional laboratory conditions and closed-circuit anaesthesia, in the presence of soda lime, some dichloroacetylene may be produced.

Dichloroacetylene may be formed from trichloroethylene by a reaction catalysed by ionic halides in the presence of certain epoxides, including epichlorohydrin (Dobinson & Green, 1972).

Non-stabilized trichloroethylene can react violently with aluminium (especially in the form of dust or filings) giving off hydrogen chloride and hexachlorobutene vapour (McNeill, 1979). Not all stabilizers are effective in preventing the reaction with aluminium; therefore, a suitably-stabilized product should be used when cleaning aluminium, especially ultrasonically or where aluminium particles are present. Suitable products are identified in manufacturers' literature or in specifications.

3.2.2. Handling precautions

3.2.2.1. Personal safeguards

Accidental exposure to trichloroethylene under occupational conditions is more frequently associated with the generation of dense trichloroethylene vapour, e.g., misoperation of vapour-degreasing apparatus (Sagawa et al., 1973) or the use of liquid trichloroethylene for cleaning the inside of a tank.

Individual protective measures should be related to the type and level of exposure. When significant skin contact is likely,

suitable protective clothing should be worn, bearing in mind the limitations of such clothing and the need to maintain it properly and replace it regularly. To control exposure through inhalation, the use of full face masks with filters for organic vapours

(basically for short-term or emergency use), self-contained breathing apparatus, or masks with air-line supply systems may be necessary. Self-contained breathing apparatus should always be available for use in emergencies.

3.2.2.2. Storage

Trichloroethylene can be safely stored in carbon steel or stainless steel containers. It should not be kept in aluminium, aluminium alloy, or galvanized iron containers; plastic containers should not be used unless they are known to be suitable for the storage of trichloroethylene. Storage areas should be cool, well-ventilated, flame-proof, and shielded from direct sunlight, high-temperature surfaces, or sparks. Trichloroethylene should not be stored near food-stuffs, strong acids, alkalis, or oxidizing agents.

3.2.3. Recovery

Used trichloroethylene can readily be recovered by distillation. Trichloroethylene vapours in the aspiration ducts of plants can be recovered by adsorption on activated carbon and subsequent desorption.

3.2.4. Disposal

Where trichloroethylene is not recovered and recycled, it may be disposed of by incineration. Incinerators must be properly operated, at a sufficiently high temperature and for an adequate period of time, to ensure complete combustion and prevent the formation of other toxic chlorinated compounds. The incinerator should incorporate a suitable scrubber to remove the acidic breakdown products.

3.2.5. Emergency measures in case of accidental spills

The spilt liquid should be contained with earth, sand, or other inert adsorbent material to prevent it from spreading.

If possible, remove damaged containers to an isolated and well-ventilated area, preferably outside, or transfer contents to another container by mechanical pumping.

Wash away small leaks with water, taking appropriate measures to avoid creating environmental pollution problems.

When necessary, the contaminated area should be marked off until the risk of dangerous concentrations in the air has been eliminated.

3.2.6. Occupational exposure

Trichloroethylene is a widely-used industrial solvent and degreasing agent. During production, exposure is relatively low and can be controlled, but users of trichloroethylene may be

exposed to higher levels and under relatively uncontrolled conditions depending on the type of operation involved. A WHO Study Group has recommended a time-weighted average exposure not exceeding 135 $\mu g/m^3$ with a ceiling limit value of 1000 mg/m^3 for not more than 15 min (WHO Study Group on Recommended Health-Based Limits in Occupational Exposure to Selected Organic Solvents, 1981). Some national occupational exposure limits are listed in Table 3.

Table 3. Occupational exposure limits used in various countries[a]

Country	Exposure Limit (ppm)	Limit (mg/m^3)	Category of limit

Australia	100	535	TWA[b]
Austria	50	260	TWA
Belgium	100	535	TWA
Bulgaria	2	10	TWA
Czechoslovakia	47	250	TWA
	235	1250	CV[c]
Egypt	50	267	TWA
Finland	50	260	TWA
France	75	405	TWA
	200	1080	CV
German Democratic Republic	47	250	TWA
	141	750	ST[d] (30 min)
Germany, Federal Republic of	50	260[e]	TWA(MAK)
Hungary	10	50	TWA
Italy	75	400	TWA
	200	1000	skin irritation
Japan	50	268	TWA
Netherlands	35	190	TWA
Poland	10	50	CV
Romania	37	200	TWA
	55	300	CV
Spain	100	535	TWA

Table 3. (contd.)

Country	Exposure (ppm)	Limit (mg/m^3)	Category of limit
Sweden	20	110	TWA
	50	250	ST (15 min)
Switzerland	50	260	TWA
United Kingdom	100	535	TWA
USA			
a) OSHA/NIOSH	100	536	TWA
	200	1072	ST (5 min)
	300	1608	CV
	1000	5350	IDLH[f]
b) ACGIH[g]	100	535	TWA
	150	800	ST (15 min)
USSR	2	10	CV
Yugoslavia	50	200	TWA

[a] From: ILO (1980) and IRPTC (1984).

[b] TWA (time-weighted average): a mean exposure limit averaged generally over a working day whereby, within prescribed limits, excursions above the level specified are permitted, provided they are compensated for by excursions below the limit specified.

[c] CV (ceiling value): a maximum allowable concentration that must not be exceeded at any time.
[d] ST (short-term exposure limit): a maximum concentration allowed for a short specified duration.
[e] Suspected carcinogen.
[f] IDLH (immediately dangerous to life and health): a maximum level from which escape is possible within 30 min without escape-impairing symptoms or any irreversible health effects.
[g] Notice of intended change to TWA 270 mg/m^3 (50 ppm) and ST 805 mg/m^3 (150 ppm).

Note: Occupational exposure levels and limits are derived in different ways, possibly using different data and expressed and applied in accordance with national practices. These aspects should be taken into account when making comparisons.

4. ENVIRONMENTAL LEVELS, TRANSPORT AND DISTRIBUTION

4.1. Environmental Levels

4.1.1. Soils and sediments

Trichloroethylene has been found in concentrations exceeding 100 µg/kg in soils and sediments near production sites (IARC, 1979). However, samples taken further away from production sites show lower levels: for example, in Liverpool Bay, United Kingdom, which is near an urban and industrialized area, concentrations in sediments ranged from a few ng/kg to 10 µg/kg (Pearson & McConnell, 1975). An organic-rich anoxic marine sediment from the Pettaquamscutt River in Rhode Island, USA, where there were no obvious local sources of trichloroethylene, contained concentrations ranging from undetectable to 70 µg/kg dry weight (Whelan et al., 1983). Trichloroethylene was concentrated in the upper part of the sediment core, corresponding to the period from about 1940 to the present (determined by ^{210}Pb dating). The authors noted that the compound had been in use only since the mid-1940s.

No relationship between trichloroethylene concentration and particle size or organic matter in sediments, as noted for higher hydrocarbons such as the DDT group (Pierce et al., 1974), has been reported.

4.1.2. Water

Trichloroethylene is widely distributed in surface water, rain-water, well water, and drinking-water from various sources. Chemical industry discharges may contain concentrations up to 200 µg/litre (Eurocop-Cost, 1976); some Milan well waters contain high concentrations (80 µg/litre) because of pollution (Cavallo & Grassi, 1976; Ziglio et al., 1983). However, most reported levels in water are below this, and are usually in the range of 10 - 100 µg/litre (Rook et al., 1975; Ewing et al., 1977). Rain-water has been reported to contain concentrations in the µg/litre range (McConnell et al., 1975) and sea water from Liverpool Bay, United Kingdom contained a mean concentration of 0.3 µg/litre (Pearson & McConnell, 1975). This lower range has also been reported in some Japanese rivers (EAJ, 1983) and in some well water in the USA (Coleman et al., 1976). Even lower concentrations (7 - 11 ng/litre) have been reported for northeast Atlantic surface water (Murray & Riley, 1973).

While trihalomethanes are produced during the chlorination of natural waters containing humic substances, there are no data indicating that trichloroethylene is produced in this way (Bellar et al., 1979; Bauer & Selenka, 1982; Otson et al., 1982). However, treatment of sewage effluent resulted in a small increase in the trichloroethylene level (Bellar et al., 1979). Trichloroethylene was found in drinking-water when the original raw water source was contaminated or when the liquid chlorine used for water treatment

contained trichloroethylene as an impurity. It is also an

intermediate in the breakdown of tetrachloroethylene in some groundwater systems (Parsons et al., 1984).

Because data for carcinogenicity are inadequate for evaluation, a tentative guideline value of 30 µg/litre in drinking-water has been recommended by the World Health Organization (WHO, 1984).

4.1.3. Air

The distribution of trichloroethylene in the atmosphere has been studied intensively, because of its possible contribution to depletion of the ozone layer (Lovelock, 1974) (section 7.3).

Air concentrations are in the µg/m^3 range (Lovelock, 1974; Pearson & McConnell, 1975; Cronn et al., 1977; Singh et al., 1977; Rasmusson et al., 1983). Murray & Riley (1973) reported much lower concentrations, in the ng/m^3 range, in rural areas or from sea stations; one urban sample (Liverpool) contained 0.85 µg/m^3. In general, higher levels are found near industrialized areas (Pearson & McConnell, 1975; Ohta et al., 1976; Correia et al., 1977; Ziglio et al., 1983).

Data describing the partition of trichloroethylene between the gaseous and particulate phases in the atmosphere are not available.

4.1.4. Biota

Pearson & McConnell (1975) have described trichloroethylene concentrations in marine organisms from Liverpool Bay, United Kingdom which is fairly close to an urban and industrialized region. Concentrations ranged from a few ng/g to about 100 ng/g wet weight. There was no obvious correlation between concentration and trophic level. Typical background concentrations are probably around 10 ng/g wet weight.

Other studies have shown the presence of trichloroethylene in marine organisms such as invertebrates (1 µg/kg wet weight), fish muscle (10 µg/kg), sea-bird eggs (50 µg/kg), and seal fat (50 µg/kg) (Pearson & McConnell, 1975).

Pearson & McConnell (1975) analysed samples of marine organisms mainly, but not exclusively, from areas near a region where major organochlorine production plants were situated. Less than 15 µg/kg wet weight was found in fish muscle (plaice, dab, mackerel). Values in sea-bird eggs ranged from 2.4 µg/kg for *Phalacrocorax aristotelis* (shag) to around 30 (23 - 33) µg/kg for *Alca torda* (razorbill), *Uria aalge* (guillemot), and *Rissa tridactyla* (kittiwake). Seal *(Halichaerus grypus)* blubber and liver from the Faroe Islands had values ranging from 2.5 to 7.2 µg/kg.

4.1.5. Food

Trichloroethylene may be present in foodstuffs as a residue from its use as a solvent in food processing or as the result of environmental contamination. A study conducted by McConnell et al. (1975) provided a table of the trichloroethylene contents of some common foodstuffs (Table 4).

Table 4. Trichloroethylene in major foodstuffs[a]

Foodstuff	Concentration (µg/kg)
Dairy foods:	
fresh milk	0.3
Cheshire cheese	3
English butter	10
eggs	0.6
Meat:	
shin of beef	16
adipose tissue of beef	12
pig liver	22

Oils and fats:	
margarine	6
olive oil (Spanish)	9
cod liver oil	19
vegetable oil for frying	7
Drinks:	
fruit juices	5
light beer	0.7
freeze-dried coffee	4
tea in bags	60
wine (Yugoslav)	0.02
Fruit and vegetables:	
potatoes	3
apples	5
pears	5
Cereals:	
fresh bread	7

[a] From: McConnell et al. (1975).

In some cases, upper tolerable limits for trichloroethylene concentrations have been set; for instance, 25 mg/kg dry weight in powdered decaffeinated coffee, 10 mg/kg dry weight in instant coffee, and 30 mg/kg dry weight in spice oleoresins. The US FDA has proposed the prohibition of the use of trichloroethylene in foodstuffs. The progress of this proposal depends on the completion of long-term toxicology and carcinogenicity studies that are being carried out. Before 1976, the US FDA prescribed tolerance level for trichloroethylene in decaffeinated ground coffee was 25 mg/kg dry weight (US CFR, 1976).

Trichloroethylene has been reviewed on a number of occasions by the Joint FAO/WHO Expert Committee on Food Additives, most recently in 1983. An acceptable daily intake (ADI) has not been allocated. The Joint Expert Committee recommended that the use of trichloroethylene as an extraction solvent should be limited, in order to ensure that its residues in food are as low as practicable (Joint FAO/WHO Expert Committee on Food Additives, 1983).

4.2. Environmental Distribution and Transport

4.2.1. Equilibrium distribution

The distribution of trichloroethylene, which is observed in various environmental "compartments", is similar to that which would be expected from a consideration of its physical and chemical properties (cf. Appendix I). The relatively high vapour pressure at normal environmental temperatures should lead to appreciable atmospheric concentrations; this tendency will balance the tendency of relatively high water solubility and low $P_{o/w}$ to lead to high water, biota, or sediment concentrations through either partition or adsorption. The tendency of trichloroethylene to enter the atmosphere is demonstrated further by its rapid evaporation from water; its evaporation half-life is approximately 20 min at 25 °C (Dilling, 1977).

4.2.2. Transformation in the environment

Recent studies on the degradation of trichloroethylene in various environmental compartments are discussed below.

4.2.2.1. Air

The main removal reaction appears to be that of attack by the tropospheric hydroxyl radical (Penkett, 1982), the steady-state concentrations of which are around $4 \times 10^5/cm^3$ (Graedel, 1978).

The decay of trichloroethylene is a function of the rate of its (bimolecular) reaction with the hydroxyl radical (Graedel, 1978), which is about $2.4/10^{12}$ cm^3 per molecule per second at 25 °C (Howard, 1976). This leads to a calculated reaction rate of approximately $4/10^3$ per h, with the calculated lifetime of trichloroethylene in the atmosphere of around 11 days (Graedel, 1978). A half-life of the order of 5 days has been calculated by

(De More et al., 1983). Singh et al. (1977) reported a half-life of less than 2 days in a smog chamber. Pearson & McConnell (1975), using unrealistically high concentrations of trichloroethylene in quartz flasks, estimated its half-life to be 11 weeks.

4.2.2.2. Soils and sediments

When methanogenic bacterial batch cultures were exposed to low concentrations of trichloroethylene (simulating conditions in an organic-rich sediment or in a sewage treatment system), at 35 °C, for 8 weeks, trichloroethylene concentrations were reduced by about 40% (Bouwer & McCarty, 1983). If it is assumed that the reaction rate is halved with every 10 °C drop in temperature, this corresponds to an exponential decay rate (first order with respect to trichloroethylene) of about $2/10^4$ per h at 15 °C.

In a study on a laboratory fresh water-sediment system, it was concluded that trichloroethylene, formed by biotransformation from tetrachloroethylene, was itself biotransformed to chloroethane, cis- and trans-1,2-dichloroethene, and dichloromethane (Parsons et al., 1984).

4.2.2.3. Water

Wakeham et al. (1982) measured a trichloroethylene exponential decay rate in a sea-water mesocosm of approximately $2.5/10^2$ per day at 8 - 16 °C, which is equivalent to a rate of about $1/10^3$ per h. This is similar to the rate described by Bouwer & McCarty (1983) for microbial degradation. Pearson & McConnell (1975) measured a chemical degradation rate, in sealed bottles, which led to a half-life estimate of 2.5 years.

4.2.2.4. Biota

The only data available refer to the degradation of trichloroethylene in a soil-plant system (Klozskowski et al., 1981) in which the rate of trichloroethylene loss was 10% per week. This was accounted for mainly by conversion to carbon dioxide, but with some evaporation of organic compounds. This corresponds to an exponential decay rate of about $6/10^3$ per h, which is about 10 times the microbial decay rates.

5. KINETICS AND METABOLISM

5.1. Absorption

Trichloroethylene absorption in mammals can take place by the respiratory, oral, and/or dermal routes. Intraperitoneal uptake has been demonstrated experimentally.

5.1.1. Inhalation exposure

In all the mammalian species studied, trichloroethylene uptake is high during the first minutes of exposure. It then decreases until equilibrium is reached between uptake by the blood and release from the blood to tissues, and by metabolism. After equilibrium is reached, uptake remains constant for the remainder of exposure (Fernandez et al., 1975; Monster et al., 1976).

In human beings, the blood/air partition coefficient ranges from 9 to 15. Daily body uptake has been estimated to be approximately 6 mg/kg body weight, for an exposure of 4 h at 378 mg/m^3 (70 ppm), and does not seem to be greatly influenced by the quantity of adipose tissue (Monster et al., 1976, 1979; Monster, 1979). Trichloroethylene retention varies according to physical activity. Under laboratory conditions, when human

volunteers at rest were exposed to concentrations of 540 or 1080 mg/m^3 (100 or 200 ppm), for 30 min, 50% of the quantity inhaled was retained. The percentage retained decreased from 50 to 25%, when activity rose from rest to a 150-watt work load, but, because of increased ventilation, the absolute amount absorbed still increased (Astrand & Ovrum, 1976).

5.1.2. Oral exposure

Uptake via the oral route is high because of the ease with which trichloroethylene penetrates the gastrointestinal barrier. In man, oral intake is a frequent cause of acute poisoning (Waters et al., 1977).

5.1.3. Dermal exposure

In the mouse, dermal absorption increases linearly at a constant rate with duration of exposure. For exposure periods of between 15 min and 5 h, absorption rates ranged from 59.8 to 92.4 nmol/min per cm2 (Tsuruta, 1978).

Trichloroethylene applied to the backs of guinea-pigs (glass depot containing at least 1.0 ml) was absorbed and produced blood concentrations of 0.79 mg/litre after 0.5 h and decreased to 0.46 mg/litre after 6 h, in spite of continuing exposure (Jakobson et al., 1982).

When one hand of each of 4 human male volunteers was immersed in trichloroethylene for 30 min, Sato & Nakajima (1978) found blood concentrations of trichloroethylene (samples taken from unexposed arm) of 2 mg/litre, immediately after the end of immersion,

0.34 mg/litre, after 30 min, and 0.22 mg/litre, after 60 min. The trichloroethylene concentration in the expired air was 0.28 mg/litre, 5 min after the end of immersion, 0.06 mg/litre, 30 min after, and 0.024 mg/litre 60 min after.

On the basis of these data and the results of earlier studies by Stewart & Dodd (1964), it is thought unlikely that trichloroethylene would be absorbed in toxic quantities through intact skin during normal industrial use.

5.2. Distribution and Storage

After absorption, trichloroethylene is concentrated in the cellular components but disappears rapidly (Fabre & Truhaut, 1952). This rapid disappearance occurs because substantial amounts of trichloroethylene are metabolized during and after exposure (Monster, 1979). Trichloroethylene reaches the tissues via the blood system and accumulates, particularly in adipose tissue, because of its high liposolubility. The oil/blood partition coefficient is approximately 750 (Droz & Fernandez, 1977; Sato & Nakajima, 1979). Trichloroethylene crosses the placental barrier readily and has been found in fetal blood (Laham, 1970). It was detectable in the fetus in 2 min, and a fetal/maternal concentration equilibrium ratio of 1:1 was reached in 6 min (La Du et al., 1971). Data on the distribution of trichloroethylene in the tissues of some animal species (including human beings) are available but, because of the differences in treatment, comparative conclusions are not possible. Trichloroethylene concentrations found in various organs and tissues from guinea-pigs, rats, and human beings are listed in Table 5.

5.3. Metabolic Transformation

5.3.1. Animals

Trichloroethylene is metabolized primarily in the liver and, to a much lesser extent, in other tissues. Metabolism is by the mixed-function oxidase system and is dependent on cytochrome P 450. Qualitative differences between species do not seem particularly significant with the exception of dichloroacetic acid formation, which appears to be specific for the mouse (Hathway, 1980; Green & Prout, in press). The major mammalian metabolites are free and conjugated trichloroethanol and trichloroacetic acid. Other

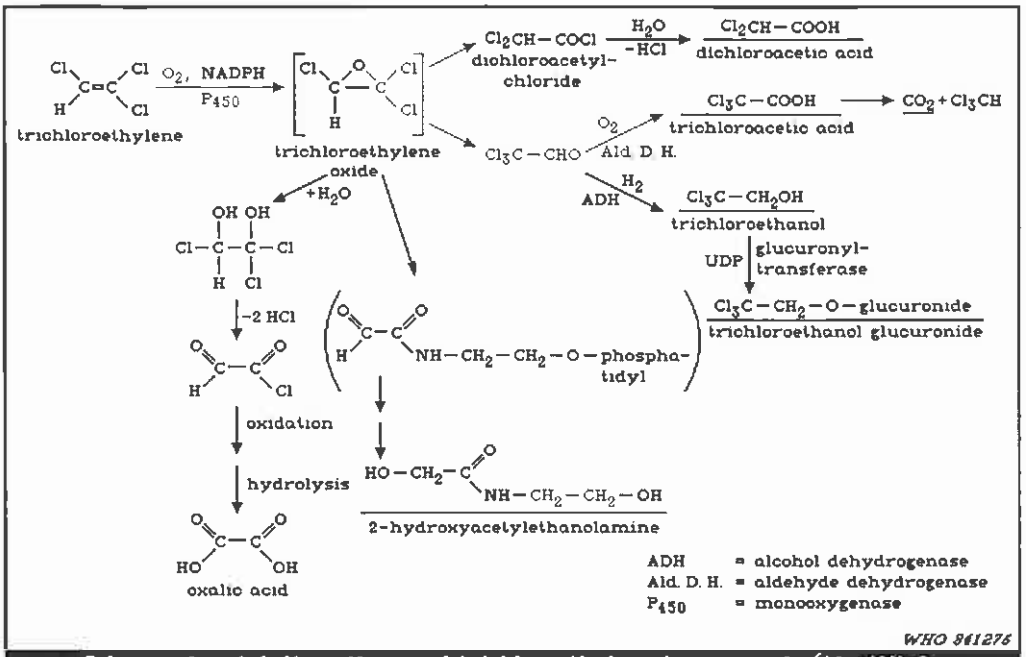
metabolites include 2-hydroxyacetylethanolamine and oxalic acid (Dekant & Henschler, 1982; DeKant et al., 1984). Metabolism is illustrated in Fig. 4.

Quantitative differences in the rates of metabolism in different species are much more significant. Mice metabolize trichloroethylene to a much greater extent than rats (Stott et al., 1982). It is possible to saturate the metabolism of trichloroethylene in the rat, but not in the mouse, at doses up to 2000 mg/kg body weight (Anderson et al., 1980). Mice also produce more reactive tissue-binding metabolites than rats in the liver and the kidney (Stott et al., 1982).

Table 5. Tissue distribution of trichloroethylene in (A) guinea-pigs and rats following exposure to the compound, and (B) in human tissues obtained at autopsy (levels of exposure not specified)

	(A)		(B)	
Organs or tissues	Guinea-pigs[a] (mg/kg)	Rats[b] (mg/kg)	Human beings[d] (μg/kg)	[e] (μg/kg)
adrenals	22	-	-	-
blood	5	0.9	-	-
brain	9	1.0 (1.2)[c]	1	-
fat	39	9.9	8.2 (32)	4.9 (11.7)
	-	-	-	7.8 (42.2)[f]
kidney	14	-	2.0	-
liver	10	0.3	4.1 (5.8)	2.5
lung	7	0.7	-	2.2
muscle	2	-	-	2.4 (156.6)
ovary	23	-	-	-
spleen	13	-	-	-

[a] From: Fabre & Truhaut (1952), 6045 mg/m^3 (1120 ppm) x 5 h per day x 19 days.
[b] From: Savolainen et al. (1977), 1080 mg/m^3 (200 ppm) x 6 h/day x 5 days).
[c] Cerebellum value.
[d] From: McConnell et al. (1975). Mean of 8 subjects aged 48 - 52 years.
[e] From: Bauer (1981). Mean of 15 subjects (Figures in parentheses are maximum values).
[f] Fat from kidney capsule.



Scheme of metabolic pathway of trichloroethylene in mammals (identified metabolites underlined).

Metabolically activated trichloroethylene binds covalently to the hepatic microsomal proteins and DNA, *in vitro*. This finding supports the formation of an epoxide intermediate (Banerjee & Van Duuren, 1978), though this has not been demonstrated *in vivo* (Parchman & Magee, 1982; Stott et al., 1982).

5.3.2. Human beings

As originally suggested by Powell (1945), the formation of the epoxide, an intermediate reactive metabolite that binds covalently with proteins (Bolt & Filher, 1977), has been confirmed by indirect spectral evidence (Uehleke et al., 1977). The epoxide may undergo intramolecular rearrangement in 2 different ways (Henschler & Hoos, 1982; DeKant & Henschler, 1983). One pathway leads to chloral (Henschler, 1977a,b), which is further oxidized to trichloroacetic acid (TCA), or reduced to trichloroethanol (Leibman, 1965). After oral administration, trichloroethanol is also partly metabolized into TCA (Müller et al., 1974). Trichloroethanol is rapidly conjugated with glucuronic acid to form the respective glucuronide. The other pathway leads to the formation of dichloroacetyl chloride which, under *in vitro* conditions, can lead to the formation of dichloroacetic acid. However, under normal *in vivo* conditions, dichloroacetic acid is not found, except in mice following the administration of very high doses of trichloroethylene. Under these conditions, an "overspill" mechanism may operate (Henschler et al., 1979; Hathway, 1980; Henschler et al., 1983). The excretion of chloroform in expired air and monochloroacetic acid in urine have also been proposed as minor routes of metabolism (Ogata & Saeki, 1974; Bartonicek, 1962). Miller & Guenrich (1982) suggested that an epoxide was not an obligatory intermediate step and proposed an alternative model in which chlorine migration occurs in an oxygenated trichloroethylene P 450 transition state.

Trichloroacetic acid binds well with plasma proteins and its concentration in plasma is approximately double that in whole blood (Müller et al., 1972).

5.3.3. Drug and other interactions

A number of commonly-used drugs might be expected to modify the extent of metabolism of trichloroethylene during human exposure. Although largely undocumented in man, the induction of the hepatic microsomal mixed-function oxidase system by drugs, taken for therapeutic reasons, or by exposure to certain environmental

chemicals (e.g., phenobarbital, toluene, PCBs) can bring about an increased rate of trichloroethylene metabolism (Ikeda & Imamura, 1973; Ikeda, 1974).

In human beings, the simultaneous administration of ethanol and trichloroethylene (100 mg/m^3 for 6 h) causes an increase in trichloroethylene levels in both plasma (2.4 times the normal value) and in exhaled air (3.4 times), and a decrease in the levels of trichloroacetic acid and trichloroethanol (Müller et al., 1975).

5.4. Elimination

5.4.1. Studies on animals

The kinetics of the distribution and elimination of trichloroethylene, administered intravenously in Wistar rats at dose levels of 6, 9, 12, or 15 mg/kg body weight show that the blood concentration exhibits a first order, 2-compartment model exponential disappearance, and it has been suggested that a dose of 15 mg trichloroethylene/kg body weight is within the hepatic metabolic capacity in the rat (Withey & Collins, 1980). Daniel (1963) showed that when trichloroethylene was administered orally to rats, the ratio of pulmonary to urinary elimination varied with the dose and that as dose increased, pulmonary excretion increased while urinary elimination decreased. Further evidence showing that the metabolism of trichloroethylene is saturable in Wistar rats was obtained by Filser & Bolt (1979) who showed that the saturation point occurred at 350 mg/m^3 (65 ppm), that the zero order V_{max} was 210 µmol/h per kg body weight and that the first order clearance at a dose of 350 mg/m^3 (65 ppm) was 77 µmol/h per kg body weight. Stott et al. (1982) found that the pulmonary elimination of unchanged trichlorethylene in Osborn Mendel rats was only 2% of the dose at 54 mg/m^3 (10 ppm), but 21% at a dose of 3240 mg/m^3 (600 ppm). In contrast to these findings of a saturable process in the rat, The same authors showed that in the mouse, doses of trichloroethylene up to 3240 mg/m^3 (600 ppm) were completely metabolized.

In dogs, exposed through inhalation, for 1 h, to trichloroethylene at 3780, 8100, and 10 800 mg/m^3 (700, 1500, and 2000 ppm), the excretion of trichloroethylene and trichloroacetic acid was correlated with the trichloroethylene concentration. The rate of trichloroacetic acid excretion was higher than that for trichloroethanol. One hour after exposure ended, the percentage of trichloro compounds in the urine was 0.7% of the total trichloroethylene absorbed (Hobara et al., 1983).

5.4.2. Studies on man

It has been shown that man metabolizes trichloroethylene extensively. Ikeda et al. (1972) showed that the capacity of workers to metabolize trichloroethylene was nonlimiting, at least up to a daily exposure level of 945 mg/m^3 (175 ppm) for 8 h.

In human beings, trichloroethylene is eliminated unchanged through the lungs and is eliminated in the urine in the form of metabolites. Elimination by other routes (e.g., faeces, sweat, and saliva) accounts for less than 10% of the total (Bartonicek, 1962).

After inhalation exposure, about 10% of the amount absorbed is expired unchanged, about 30 - 50% is excreted as trichloroethanol in urine, and about 10 - 30% as trichloroacetic acid in urine (Soucek & Vlachova, 1960; Bartonicek, 1962; Monster et al., 1976, 1979).

The half-life of trichloroethylene in exhaled air and in the blood depends on the length of exposure and on the time of sampling after exposure. The concentration follows a multi-exponential curve, compatible with at least 3 compartments: lungs, blood and most other tissues, and adipose tissue. After a single exposure to trichloroethylene, trichloroethanol reaches its maximum concentration in blood and urine almost directly after exposure. Thereafter, the concentration decreases, with a half-life of about 10 - 15 h (Müller et al., 1974; Monster et al., 1976,

1979; Vesterberg et al., 1976). After a single exposure to trichloroethylene, the concentration of trichloroacetic acid in both the blood and the urine increases for up to 20 - 40 h after exposure. Thereafter, the concentration decreases with a half-life of about 70 - 100 h (Müller et al., 1974; Monster et al., 1979). Trichloroacetic acid, as such, has a shorter half-life of about 50 h.

In a group of workers with long-term exposure to trichloroethylene at a concentration of 270 mg/m^3 (50 ppm), median values of trichloroethanol and trichloroacetic acid of 330 and 319 g/kg creatinine, respectively, were found at the end of a working shift; during the work-free periods, the metabolites of trichloroethylene were eliminated slowly (Triebig et al., 1976).

In a study on factory workers exposed to trichloroethylene, Ikeda & Imamura (1973) observed a half-life of 41 h for the urinary excretion of total trichloro-compounds (i.e., a combination of trichloroacetic acid and trichloroethanol). This half-life is somewhat longer with oral administration of trichloroethylene and of chloral hydrate and is about 85 - 99 h after repeated exposure to trichloroethylene, because of the delayed formation of trichloroacetic acid from trichloroethylene and the trichloroethanol still available from the tissues (Müller et al., 1974). Thus, trichloroacetic acid will be found in the urine, even when trichloroethanol is no longer detectable (Ikeda et al., 1971). Trichloroacetic acid accumulates in the blood and urine during daily exposures to trichlorethylene (Monster et al., 1979; Müller et al., 1975).

5.5. Biological Monitoring of Exposure

Droz & Fernandez (1978) used a mathematical model to study the effects of hourly and daily variations in exposure concentrations on alveolar air trichloroethylene concentrations and on the urinary excretion of trichloroethanol and trichloroacetic acid. The determination of trichloroethanol in urine appeared to be more sensitive than the determination of trichloroethylene in exhaled air. The excretion of trichloroacetic acid can be used for the qualitative evaluation of the preceding day's exposure. In practice, blood analysis would be preferable to analysis of urine, because of the smaller individual variations generally observed with the former. In studies concerning repeated exposure to constant concentrations, the smallest inter-individual variation was found in the concentrations in blood (Monster et al., 1979).

The measurement of total trichloro compounds in urine was described by Takana & Ikeda (1968). The urinary trichloroethanol is oxidized to trichloroacetic acid and the total amount of trichloroacetic acid is then measured with the Fujiwara reaction. In the case of exposure to relatively steady concentrations, this has the advantage of being able to indicate small-scale inter-personal variations. It may be used as an index of exposure intensity, especially when the urine samples are collected at, or close to, the end of the workshift at the end of the work week (Ikeda et al., 1972). However, other studies have demonstrated poor individual correlation between trichloroethylene exposure and the urinary elimination of the major metabolites, trichloroacetic acid and trichloroethanol (Boudène et al., 1983). Separate measurement of urinary metabolites provides more information on exposure to daily fluctuating concentrations, because relatively high concentrations of trichloroethanol indicate recent high exposure, whereas relatively high concentrations of trichloroacetic acid indicate long-term exposure to high concentrations (Monster et al., 1979).

Trichloroethylene concentrations in alveolar air and in blood, shortly after exposure, indicate recent exposure concentrations, while the concentration several hours after exposure indicates the average exposure over the preceding days (Stewart et al., 1974).

6. EFFECTS ON ANIMALS AND CELL SYSTEMS

6.1. Effects on Animals

Data from acute, short-term repeated dose, and long-term toxicity studies on laboratory animals are summarized in Table 6.

6.1.1. Acute toxicity

Acute toxicity data on common laboratory animals are shown in Tables 7 and 8.

Acute toxicity levels following inhalation exposure to trichloroethylene are summarized in Table 7. Table 8 includes data on acute oral, dermal, intraperitoneal (ip), subcutaneous (sc), and intravenous (iv) LD_{50}s for the mouse, rat, rabbit, and dog.

Von Oettingen (1955) reported that oral acutely toxic doses in rats produced gastrointestinal irritation. Moderate increases in aspartate aminotranferase (EC 2.6.1.1) levels were observed in rats 24, 48, and 72 h after a single 6-h exposure to trichloroethylene vapour at concentrations of 54 mg/m^3 (10 ppm) and 540 mg/m^3 (100 ppm) (Deguchi, 1972); the increase at 5400 mg/m^3 (1000 ppm) was small (Deguchi, 1972), presumably due to inactivation of P-450. According to Rigaud et al. (1977), intraperitoneal administration resulted in a significant increase in aspartate aminotransferase (EC 2.6.1.1), alanine aminotransferase (EC 2.6.1.2), and ornithine carbamoyltransferase (OCT) (EC 2.1.3.3) in the rat, whereas Wirtschafter & Cronyn (1964), administering 500 mg/kg body weight, detected only minor hepatic effects over the 12 - 24-h period following administration. No evidence of kidney dysfunction was observed in mice following intraperitoneal administration of trichloroethylene at 0.004M/kg body weight. Application of 2 ml trichloroethylene (7800 mg/kg), under an occlusive dressing, on the skin of 20 guinea-pigs, did not produce any deaths but, during the 35-day observation period, there were reductions in body weight at 1 week ($P < 0.001$), 2 and 3 weeks ($P < 0.01$), and 4 weeks ($P < 0.05$) (Wahlberg & Boman, 1979).

Skin irritation

Trichloroethylene (purity 99.5%), applied (0.5 ml) to the shaved (non-abraded) skin of rabbits, for 24 h, under an occlusive dressing, produced severe skin irritation (Duprat et al., 1976). In another study, trichloroethylene (1.0 ml) was applied, occluded in a "skin depot", to the clipped skin of guinea-pigs. Histological examinations were performed at 15 min, 1, 4, and 16 h. Degenerative changes (pyknotic nuclei) were observed in the epidermis after 15 min and were progressive (pyknosis, karyolysis, junctional separation of the epidermis) up to the end of the study at 16 h (Kronevi et al., 1981).

Table 6. Concentrations of trichloroethylene at which no effects were observed in experimental animals exposed through inhalation

Species	Concentration (mg/m^3) at which no effects were observed	Duration	Biological endpoints being investigated	Reference
rat	3000	7 h	mortality	Adams et al. (1951)
	6400	1.4 h	mortality	Adams et al. (1951)
	12 000	0.6 h	mortality	Adams et al. (1951)
	20 000	0.4 h	mortality	Adams et al. (1951)
	200	7 h per day, 5 days per week, for 26 weeks	mortality	Adams et al. (1951)
	400	8 h per day for 5 days	mortality; body weight; learning capacity	Battig et al. (1963)
	730	8 h per day, 5 days per week for 6 weeks	mortality; body weight; haematology; histology of heart, liver, lung, spleen,	Prendergast et al. (1967)

			and kidneys	
	35[a]	90 days	mortality; body weight; haematology; histology of heart, liver, lung, spleen, and kidneys	Prendergast et al. (1967)
guinea-pig	730	8 h per day, 5 days per week for 6 weeks	mortality; body weight; haematology; histology	Prendergast et al. (1967)
	35	90 days	mortality; body weight; haematology; histology histology	Prendergast et al. (1967)

Table 6. (contd.)

Species	Concentration (mg/m^3) at which no effects were observed	Duration	Biological endpoints being investigated	Reference
guinea-pig (contd.)	100	7 h per day, 5 days per week for 26 weeks	mortality	Adams et al. (1951)
monkey (squirrel)	730	8 h per day, 5 days per week for 6 weeks	mortality; body weight; haematology; histology of heart, liver, lung, spleen, and kidneys	Prendergast et al. (1967)
	35	90 days	mortality; body weight; haematology; histology of heart, liver, lung, spleen, and kidneys	Prendergast et al. (1967)
monkey (Rhesus)	400	7 h per day, 5 days per week for 26 weeks	mortality	Adams et al. (1951)
rabbit	730	8 h per day, 5 days per week for 6 weeks	mortality; body weight; haematology; histology of heart, liver, lung, spleen, and kidneys	Prendergast et al. (1967)
	35	90 days	mortality; body weight; haematology; histology of heart, liver, lung, spleen, and kidneys	Prendergast et al. (1967)
	200	7 h per day, 5 days per week for 6 weeks	mortality	Adams et al. (1951)
dog	730	8 h per day, 5 days per week for 6 weeks	mortality; body weight; haematology; histology of heart, liver, lung, spleen, and kidneys	Prendergast et al. (1967)

Table 6. (contd.)

Species	Concentration (mg/m^3) at which no effects were observed	Duration	Biological endpoints being investigated	Reference
dog (contd.)	35	90 days	mortality; body weight; haematology; histology of heart, liver, lung, spleen, and kidneys	Prendergast et al. (1967)
mouse[b]	5500	20 min	anaesthesia	Gehring (1968)

5500	100 min	liver injury (elevated SGPT)	Gehring (1968)
5500	300 min	mortality	Gehring (1968)

[a] Kalashmikova et al. (1976) reported that rats exposed to 50 mg/m³ for 5 h per day, for 90 days, showed damage to parenchyma in liver and kidney.

[b] Kjellstrand et al. (1983) reported that male NMRI mice continuously exposed to 200 mg/m³ (37 ppm) for 30 days showed increased plasma butyrylcholinesterase (BuChE) (EC 3.1.1.8) activity; female mice did not show any increase in BuChE activity; there was a significant increase in liver weight at 200 mg/m³ (37 ppm) in both sexes.

Table 7. Acute toxicity of trichloroethylene administered via inhalation to laboratory animals

Species	Toxicity index	Exposure level (ppm)	Exposure level (mg/litre)	Exposure duration (h)	Reference
Rat	LC_{100}	20 000	107	0.4	Adams et al. (1951)
	LC_{100}	12 000	64.2	1.4	Adams et al. (1951)
	LC_{100}	2500	13.4	7.0	Adams et al. (1951)
	LC_{50}	26 300	140.7	1	Vernot et al. (1977)
	LC_{50}	12 500	66.9	4	Siegel et al. (1971)
	LC_{L0}[a]	8000	42.8	4	Smyth et al. (1969)
Mouse	LC_{100}	8000	42.7	2	Von Oettingen (1955)
	LC_{100}	5600	30.0	9.75	Gehring (1968)
	LC_{50}	8450	45.1	4	Kylin et al. (1962)
	LC_{50}	41 122	220	0.33	Aviado et al. (1976)
	LC_{50}	49 000	262	0.50	Vernot et al. (1977)
	LC_{L0}[a]	3000	16.0	2	Lazarev (1929)
	LC_{50}		40	4	Lazarev & Gadaskina (1977)
Guinea-pig	LC_{100}	37 000	197.8	0.67	Von Oettingen (1955)
Cat	LC_{L0}[a]	6074	32.5	2	Lehmann & Schmidt-Kehl (1936)
Rabbit	LC	5000	26.75	14.28	McCord (1932)
	LC	10 000	53.5	2.5	McCord (1932)
	LC	20 000	107	2	McCord (1932)

[a] LC_{LO} = lowest published lethal concentration.

Eye irritation

Instillation of 0.1 ml of trichloroethylene (purity 99.5%) into rabbit eyes produced mild to moderate conjunctivitis with superficial epithelial abrasion. At 7 days, there was a resolving keratitis with complete recovery within 2 weeks (Duprat et al., 1976).

6.1.2. Short-term exposures

6.1.2.1. Oral exposures

In a US NTP study (1983), groups of 10 male and 10 female F344/N rats were administered trichloroethylene (in corn oil, by gavage) at doses ranging from 125 to 2000 mg/kg body weight (males) and 625 to 1000 mg/kg (females), 5 times per week, for 13 weeks. All rats survived the 13-week study, but males receiving the 2000 mg/kg dose exhibited a 24% decrease in body-weight gain. At the 1000 mg/kg dose, final body weights for males and for females were similar to those of the controls.

Table 8. Acute oral, dermal, intraperitoneal, subcutaneous, and intravenous LD_{50}s for trichloroethylene in laboratory animals

Species	Oral	Dermal	Intraperitoneal	Subcutaneous	Intravenous

	(mg/kg body weight)	(ml/kg body weight)	(mg/kg body weight)	(mg/kg body weight)	(mg/kg body weight)
Rat	4920[1]		2725[2]		
Dog	5680[3]		2.800[4]		150[5,a]
Mouse	2850[6,b] 2400[12]		3210[7,b] 3150[8,b] 3000[9,c] 1.200[6,b]	1440[10]	34[11]
Rabbit		20[1]			

[1] From: Smyth et al. (1969).
[2] From: Rigaud et al. (1977).
[3] From: Christensen et al. (1974).
[4] From: Klaassen & Plaa (1967).
[5] From: Barsoum & Saad (1934).
[6] From: Aviado et al. (1976).
[7] From: Klaassen & Plaa (1966).
[8] From: Gehring (1968).
[9] From: Gradiski et al. (1974).
[10] From: Plaa et al. (1958).
[11] From: NIOSH (1977b).
[12] From: Tucker et al. (1982).

[a] LD_{Lo}.
[b] In 24 h.
[c] In 14 day.

Histopathological examination of tissues from animals receiving the highest doses showed minimal or mild cytomegaly and karyomegaly of the renal tubular epithelial cells in the inner cortex in 8/9 males dosed with 2000 mg/kg per day, and the same effect, graded as equivocal or mild, was seen in 5/10 females that had received the 1000 mg/kg per day dose.

The results of this 13-week study in F344/N rats were essentially similar to those of an earlier 8-week study conducted on Osborne-Mendel rats (NCI, 1976). In this study, only doses in excess of 5000 mg/kg per day were lethal for rats. Doses of 1000 mg/kg per day had no effect on body-weight gains in males, but depressed weight gains in females by approximately 15%.

Groups of 10 male and 10 female B6C3F1 mice received trichloroethylene (by gavage in corn oil) at doses ranging from 375 to 6000 mg/kg body weight, 5 times per week for 13 weeks. All males and 9/10 females receiving 6000 mg/kg, 7/10 males and 1/10 females receiving 3000 mg/kg, and 2/10 males and 1/10 females receiving 1500 mg/kg died. Mean body weights of male mice dosed with 750, 1500, or 3000 mg/kg were depressed by 11%, 19%, and 17%, respectively, relative to the controls. Mean body weights of control and treated groups of female mice were similar (US NTP, 1983).

Liver weights (both absolute and as percentage body weight) increased in a dose-related fashion. Liver weights were increased by more than 10% relative to the controls for males receiving 750 mg/kg body weight or more and for females receiving 1500 mg/kg or more.

Histopathological examination showed hepatic centrilobular necrosis (6/10 males and 1/10 females administered 6000 mg/kg). This lesion was not seen in either males or females administered 3000 mg/kg, but 2/10 males had multifocal areas of calcification scattered throughout their livers. Multi-focal calcification was also seen in the liver of the single female mouse that survived the 6000 mg/kg dosage regimen. One female in the 3000 mg/kg dose group developed a hepatocellular adenoma, an extremely rare lesion in female mice of this age (20 weeks).

Examination of renal tissues showed the presence of mild to moderate cytomegaly and karyomegaly of the renal tubular epithelial cells of the inner cortex. These changes were found in only 1 of the 23 mice (13 males and 10 females) that died after receiving

doses of 3000 or 6000 mg/kg for up to 6 weeks. However, the changes were found in all 4 of the males that died after receiving the 3000 mg/kg dose for 7 - 13 weeks, and in all animals that survived the 6000 mg/kg (1/10 females) and the 3000 mg/kg doses (3/10 males and 9/10 females). Tissues from mice receiving lower doses of trichloroethylene were not examined.

Trichloroethylene administered orally to mice at doses in excess of 500 mg/kg for 10 days produced proliferation of hepatic peroxisome as demonstrated by increased cyanide-insensitive palmitoyl CoA oxidation (PCO) and electron microscopy. In the rat, trichloroethylene did not have any effect on peroxisome proliferation. Trichloroacetic acid administered for 10 days increased hepatic peroxisome proliferation in both species (Elcombe et al., 1982). It is possible that the rapid rate of trichloroethylene metabolism in the mouse, together with the enterohepatic circulation of trichloroacetic acid, leads to high steady-state blood levels of trichloracetic acid and the concomitant proliferation of peroxisomes.

Primary cultures of isolated mouse and rat hepatocytes have been found to metabolize trichloroethylene to trichloroacetic acid at rates comparable to those in intact animals. Similarly, the isolated hepatocytes respond to exposure to trichloroacetic acid by peroxisome proliferation. Isolated human hepatocytes have been found to produce trichloracetic acid from trichloroethylene at a lower rate than that in the rat. Trichloroacetic acid does not induce peroxisome proliferation in human hepatocytes (Elcombe, 1985).

6.1.2.2. Inhalation exposure

In the rat, exposure to 81 000 mg/m^3 (15 000 ppm) for a period of 2 - 4 min produced complete anaesthesia within 9 min (Schumacher & Grandjean, 1960). In a study on mice, exposure to 36 7000 mg/m^3 (6800 ppm) for 10 - 11 min or 64 800 mg/m^3 (12 000 ppm) for 5 - 6 min produced complete anaesthesia (Friberg et al., 1953). In another study on mice, exposure to 29 700 mg/m^3 (5500 ppm) produced anaesthesia in 46 min (Gehring, 1968).

Studies conducted on rats by Vissarionova et al. (1975) showed that concentrations of 5000 mg/m^3 administered for 5 h a day, for 1 week, resulted in an increase in liver and kidney weight (21.6% and 18%, respectively), and a decrease in alkaline phosphatase and RNA-dependent hepatic dehydrogenase. Histological changes have also been noted in hepatic and renal parenchyma by Kalashnikova et al. (1976).

With doses of 1080 mg/m^3 (200 ppm) administered for 6 h daily for 4 days, Savolainen et al. (1977) found that rats exhibited greater motor activity and less cerebral RNA, 5 days after the last exposure, and an accumulation of trichloroethylene in perirenal fat.

Doses of 250, 500, 800, and 1200 mg/m^3 administered for 15 - 90 h resulted in increases in intracellular lipids (Verne et al., 1959).

No effects were noticed in rats administered 1080 mg/m^3 (200 ppm) for 4 h a day for 4 days (Grandjean, 1960).

Continuous exposure of rats, mice, and gerbils by inhalation to trichloroethylene at 810 mg/m^3 (150 ppm), for periods ranging from 2 to 30 days, produced liver enlargement in all species; the mouse was the most severely affected. After the end of exposure, the liver weights of the mice decreased rapidly. An increased kidney weight was noted in gerbils (Kjellstrand et al., 1981a).

In another study in which 7 different strains of mice (wild, C57BL, DBA, B6CBA, A/su, NZB, and NMRI) were continuously exposed to a trichloroethylene concentration of 810 mg/m^3 (150 ppm), Kjellstrand et al. (1983a) reported large increases in liver weight in all strains with minimal changes in kidney and spleen weights. Plasma butyrylcholinesterase (BuChE) (EC 3.1.1.8) activity

increased in males of all strains and in females of strains A/su and NZB, but to a lesser extent than in the corresponding males. In a further study, with continuous exposure to concentrations of 200 - 1620 mg/m^3 (37 - 300 ppm), plasma BuChE increased in male mice in a time- and concentration-dependent manner. Liver weight increased in a time- and concentration-dependent manner in both sexes.

Exposure of rats, guinea-pigs, rabbits, dogs, and monkeys to 3825 mg/m^3, for 8 h per day, 5 days per week, for 6 weeks, resulted in a loss of overall body weight in dogs and monkeys. There were no changes in haematological or liver enzyme parameters (Prendergast et al., 1967).

Groups of male Swiss Webster mice were exposed to 54 000 mg/m^3 (10 000 ppm) for 1 or 4 h, daily, for 5 consecutive days. In the 4-h group, the NADPH cytochrome c reductase (EC 1.6.99.3) activity in the lung decreased, but that in the liver increased. In the lungs of this group, there were platelet thrombi and vacuolization of bronchial epithelial cells. There were no changes in the liver. It was concluded that the reduced activity of the pulmonary mixed-function oxidase system reflected injury to the lungs (Lewis et al., 1984).

Rabbits exposed to 15 000 mg/m^3 (2790 ppm) for 4 h per day, 6 days a week for 45 days, developed severe normocytic anaemia, leukopenia, and thrombocytopenia due to toxic effects on the bone marrow (Mazza & Brancaccio, 1967).

In rats, urinary levels of trichloro-substituted metabolites and the activity of drug-metabolizing enzymes (cytochrome P-450) were related to the duration of trichloroethylene anaesthesia (Moslen et al., 1977). Trichloroethylene hepatotoxicity in the rat produces increased levels of serum transaminases. Hetaptotoxicity increases the activity of drug-metabolizing enzymes (cytochrome P-450) (correlation coefficient: 0.95) and the urinary excretion of trichloro-metabolites (correlation coefficient: 0.88) (Moslen et al., 1977).

Cytotoxic effects have been observed in the kidney and liver of dogs subjected to 81 000 mg/m^3 (1.5%; 15 000 ppm) trichloroethylene anaesthesia (Kiseleva & Korolenko, 1971).

Studies on rats exposed by inhalation to trichloroethylene concentrations of 2160, 4320, or 8640 mg/m^3 (400, 800, or 1600 ppm) for 6 h revealed no effects at the first concentration, a decrease in swimming activity at 4320 mg/m^3, and a further decrease at 8640 mg/m^3 (Grandjean, 1963). Other rats exposed to 1944 - 2268 mg/m^3 (360 - 420 ppm) for 8 h a day, 5 days a week for 46 weeks, exhibited no effects (Bättig & Grandjean, 1963). After 43 weeks at 2160 mg/m^3 (400 ppm), rats in another set of tests by Bättig (1964) exhibited greater maze skills. Mice exposed intermittently to trichloroethylene showed a decrease in motor activity at 4500 mg/m^3 (900 ppm), but, at 19 440 mg/m^3, it was considerably increased (Kjellstrand et al., 1983b).

In Mongolian gerbils, continuously exposed to trichloroethylene at 1.72 mg/m^3 (320 ppm) for 9 months, there was no effect on spatial memory, but subsequent maze performance test results were interpreted as indicating an irreversible effect on the central nervous system (Kjellstrand et al., 1980). In another study, 2 groups of Mongolian gerbils were continuously exposed to 810 mg trichloroethylene/m^3 (150 ppm) for 71 and 106 days, respectively. In a series of maze tests following the end of exposure, the treated groups performed less well than the unexposed controls (Kjellstrand et al., 1981b).

6.1.2.3. Parenteral exposure

When male Swiss Webster mice were injected ip with 3 doses of 330 mg trichloroethylene/kg body weight (vehicle 0.2 ml of 25% Tween 80 in saline) on alternate days, the activity of hepatic microsomal NADPH cytochrome c reductase was increased. There were no morphological changes in the liver (Lewis et al., 1984).

Studies conducted on rabbits showed that intramuscular administration of 3 ml trichloroethylene, 3 times weekly for 29 days, induced neuronal damage (Bartonécek & Brun, 1970).

6.1.3. Long-term exposure

6.1.3.1. Oral exposure

The US NTP (1983) studied the effects of orally-administered (in corn oil, by gavage) epichlorohydrin-free trichloroethylene in male and female F344/N rats and B6C3F1 mice. Doses (500 and 1000 mg/kg body weight for rats and 1000 mg/kg for mice) were administered 5 days per week for 103 weeks. The survival of treated male rats and male mice was significantly reduced in relation to that of corn-oil control animals. Mean body weights of treated rats (both sexes) were lower than those of corn-oil controls and the reduction in body weight gain was dose-related. The body weights of treated female mice were similar to those of vehicle controls.

Toxic nephrosis was found in 96/98 (98%) of the treated male rats, in 97/97 of the treated female rats, in 45/50 (90%) of the treated male mice, and in 48/49 (98%) of the treated female mice, but was not found in any of the corn-oil control rats or mice. Initially noted in rats that died early, the lesions were diagnosed as frank enlargement of the nucleus and cytoplasm of scattered individual tubular cells with brush borders, located near the cortico-medullary junction. Progression of the lesions was evident. As exposure time increased, affected tubular cells were larger and additional tubules and tubular cells were affected. Some tubules were enlarged or dilated to the extent that they were difficult to identify as tubules. Eventually, there was loss of some enlarged cells. Corresponding tubules became dilated and portions of the basement membrane had a stripped appearance. In the most advanced stage, the lesion had progressed to the sub-capsular cortex, with enlarged tubular cells.

In mice, the pathological development of the renal lesion was basically similar to that observed in rats, but it was relatively less severe and did not develop to a stage where there was extensive loss of cytomegalic epithelial cells and tubular dilation.

When trichloroethylene was given in the drinking-water (0.1, 1.0, 2.5, and 5.0 g/litre) to CD-1 mice for 4 - 6 months, a significant reduction in body weight (males and females at 5.0 g/litre), enlarged liver (males at 1.0, 2.5, and 5.0 g/litre; females at 5.0 g/litre), and increase in kidney weight (males and females at 5.0 g/litre) were observed. However, pathology at 4 and 6 months was unremarkable.

6.1.3.2. Inhalation exposure

Studies on rats showed that exposure to concentrations below 1080 mg/m^3 (200 ppm) for 6 h a day, 5 days a week for 6 months, did not induce any visible effects. At concentrations of 10 800 mg/m^3 (2000 ppm), narcosis and loss of appetite were observed. At 16 200 mg/m^3 (3000 ppm), only 33% of the test animals survived (Taylor, 1936).

When NMRI mice were exposed to trichloroethylene at 700 mg/m^3 (130 ppm) continuously for 30 days, liver weight increased by 1.5 and 1.9 times in males and females, respectively (Kanje et al., 1981).

When rats were exposed to trichloroethylene at 1945 - 2270 mg/m^3 (360 - 420 ppm), for 8 h a day, 5 days a week for 46 weeks, there were no changes in conditioned reflexes or reaction time, but there was an increase in spontaneous climbing activity (Bättig & Grandjean, 1963).

Rats exposed to 50 mg/m^3 (0.05 mg/litre) for 5 h daily, for 3 months, showed damage to hepatic and renal parenchyma

(Kalashnikova et al., 1976).

Long-term exposure to concentrations of 100 - 200 mg/m³ (0.1 - 0.2 mg/litre) reduced the phagocytic activity of rat leukocytes (Pelts, 1962).

In female Mongolian gerbils, exposed continuously to 270 and 810 mg/m³ (50 and 150 ppm) for 12 months, there were small changes in the lipid composition of the cerebral cortex and hippocampus. While protein content and lipid class distribution were virtually unaffected, the cholesterol to phospholipid ratio in the cortex decreased (Kyrklund et al., 1983).

6.1.3.3. Parenteral exposure

Of 6 rabbits given trichloroethylene at 200 mg/kg body weight intramuscularly for 55 - 100 days, 2 died from renal failure (Bartonécek & Brun, 1970). Rabbits given 2 ml (2.92 g) intramuscularly twice a week for 41 - 247 days exhibited neuronal damage (Bartonécek & Sovcek, 1959).

6.1.4. Interactions

Studies on rats showed that trichloroethylene was more toxic in animals on a high-carbohydrate diet than in those on a high-protein diet (Kalashnikova et al., 1974). In rats (Cornish & Adefuin, 1966), rabbits (Desoille et al., 1962), and human beings (Sbertoli & Brambilla, 1962; Ferguson & Vernon, 1970; Pardys & Brotman, 1974), the presence of ethanol increased trichloroethylene

toxicity. Trichloroethylene and carbon tetrachloride acted synergistically in producing hepatotoxicity (Deguchi, 1972; Pessayre et al., 1982).

When trichloroethylene was administered intraperitoneally with toluene, there was a decrease in the side-chain oxidation of the toluene (Ikeda, 1974).

In studies by White & Carlson (1979), trichloroethylene caused spontaneous, and potentiated epinephrine-induced, cardiac arrhythmias in rabbits. A series of studies was conducted to examine the effects of metabolic-inhibitors, enzyme-inducing agents, or pre-treatment with ethanol or caffeine, on the sensitivity of rabbits to epinephrine-induced cardiac arrythmias (White & Carlson, 1979, 1981a, 1982). Rabbits treated with metabolic-inhibiting agents developed more arrhythmias after shorter exposure times in response to lower doses of epinephrine than controls. Phenobarbitone and Aroclor 1254(R), and in a later study benzo(a)pyrene (Carlson & Shite, 1983), were used as enzyme-inducing agents. Phenobarbitone-treated rabbits developed fewer cardiac arrhythmias and had lower blood levels of trichloroethylene. Rabbits treated with benzo(a)pyrene developed more cardiac arrhythmias and at lower doses of epinephrine when exposed by inhalation to a trichloroethylene concentration of 43 500 mg/m³ (8100 ppm). Aroclor 1254(R) did not induce any effects on trichloroethylene metabolism nor on the development of epinephrine-induced cardiac arrhythmias. Rabbits pre-treated with ethanol (1000 mg body weight, intravenously or orally) or caffeine (10 mg/kg body weight, intraperitoneally) 30 min before exposure to trichloroethylene at 32 400 mg/m³ (6000 ppm) by inhalation showed an increased senstivity to epinephrine-induced cardiac arrhythmias.

Male New Zealand rabbits exposed to trichloroethylene at 10 800, 21 600, 32 400, or 43 200 mg/m³ (2000, 4000, 6000, or 8000 ppm) by inhalation for 1 h showed a concentration-related sensitivity to epinephrine-induced cardiac arrhythmias. The blood levels of the trichloroethylene metabolites (trichloroethanol and trichloroacetic acid) were measured. Rabbits given chloral hydrate (50 mg/kg body weight, intravenously), a trichloroethylene intermediate metabolite, had blood levels of trichloroethanol and trichloroacetic acid that were 40 - 100 times higher than those of rabbits exposed to a trichloroethylene level of 43 200 mg/m³ (8000 ppm) but showed no increase in sensitivity to epinephrine-induced cardiac arrhythmias (White & Carlson, 1981b). It is concluded that

trichloroethylene, rather than its metabolites, sensitizes the rabbit myocardium.

6.1.5. Immunotoxicity

Changes in some components of antibody production (7S), cell-mediated immunity (haemagglutination), and bone marrow stem cell colonization have been observed in rodents (female mice were more sensitive than males in one study; chinchillas were used in the other) exposed to low-to-moderate levels (10 - 1000 mg/m^3) by inhalation or to 0.1 - 5 g/litre per day in the drinking-water for periods ranging from a few weeks to 6 - 10 months (Shmuter, 1972; Sanders et al., 1982).

6.1.6. Effects on cell systems

In vitro incubation of nerve-fibre membranes from rat spinal cord with 10 μM trichloroethylene reduced the low relative molecular mass protein fraction (Savolainen & Seppalainen, 1979).

A trichloroethylene concentration of $1.3/10^8$ moles per ml was the LD_{50} for HeLa (human cervix carcinoma) cells on the third day of treatment (Gradiski et al., 1974).

Exposure of rabbit trachea cultures to trichloroethylene vapour at 27 000 mg/m^3 (5000 ppm) for 129 min and 216 000 (40 000 ppm) for 13 min, induced ciliostasis (Tomenius et al., 1979). The isolated guinea-pig heart exposed to trichloroethylene at 530 mg/litre, developed transitory arrhythmia. At a concentration of 1.06%, there was evidence of a reduction in contractile force and coronary flow, and arrhythmia (Bianchi et al., 1963; Matturro et al., 1963).

6.1.7. Carcinogenicity

Groups of 50 male and 50 female Osborne-Mendel rats, 7 weeks old, were administered trichloroethylene (99% trichloroethylene stabilized with 0.19% 1,2-epoxybutane and 0.09% epichlorohydrin) in corn oil, by gavage, 5 days a week for 78 weeks. High-dose animals received varying dose schedules of 1000 - 1500 mg/kg body weight per day, and low-dose animals received 500 - 750 mg/kg body weight per day. All surviving animals were killed 110 weeks after the start of treatment. The time-weighted average doses were 549 and 1097 mg/kg body weight per day. A group of 20 male and 20 female vehicle-treated rats served as controls. Of the males, 17/20 controls, 42/50 low-dose, and 47/50 high-dose animals died before the end of the study; of the females, 12/20 controls, 35/48 low-dose, and 37/50 high-dose animals died early. Median survival times were approximately 60 weeks for high-dose males, 85 weeks for low-dose males, and 70 weeks for high- and low-dose females. Of the males, 5/20 controls, 7/50 low-dose, and 5/50 high-dose rats developed tumours; of the females, 7/20 controls, 12/48 low-dose, and 12/50 high-dose rats developed tumours. There were no liver-cell tumours. Tumours occurred in various other organs in treated and control animals and were mainly reticulum-cell sarcomas, lymphosarcomas or malignant lymphomas, fibroadenomas of the mammary gland, haemangiosarcomas at various sites, follicular adenocarcinomas of the thyroid, chromophobe adenomas of the pituitary, and renal hamartomas. Toxic nephropathy was observed in rats of both sexes treated with high and low doses of trichloroethylene (NCI, 1976).

Groups of 50 male and 50 female B6C3F1 mice, 5 weeks old, were given trichloroethylene (99% trichloroethylene stabilized with 0.19% 1,2-epoxybutane and 0.09% epichlorhydrin) in corn oil by gavage, 5 days a week, for 78 weeks. High-dose males received 2000 - 2400 mg/kg body weight per day, and females 1400 - 1800 mg/kg body weight per day; low-dose males and females received 1000 - 1200 mg/kg body weight per day and 700 - 900 mg/kg body weight per day, respectively. All surviving animals were observed until they reached 95 weeks of age. Time-weighted average doses were 1169 and 869 mg/kg body weight per day, respectively, in low-dose males and females and 2339 and 1739 mg/kg body weight per day, respectively, in high-dose males and females. Groups of 20 male

and 20 female mice served as vehicle-treated matched controls. Survival was reduced in high-dose males and control males. Hepatocellular carcinomas occurred in 1/20 control males and 0/20 control females; in 26/50 low-dose males and 4/50 low-dose females, and in 31/48 high-dose males and 11/47 high-dose females. Metastases of the liver-cell tumours to the lung were found in 7/98 treated males and in 1 control male. The first hepatocellular carcinoma was observed in a mouse which had been treated with the high dose of trichloroethylene and which died during week 27. Lung tumours occurred in treated animals of both sexes: 5/50 (5 adenomas) in males and 4/50 (2 adenomas, 2 carcinomas) in females in the low-dose group, and 2/48 (1 adenoma, 1 carcinoma) in males and 7/47 (5 adenomas, 2 carcinomas) in females treated with the high dose of trichloroethylene. Among controls, only one lung adenoma was reported in a female (NCI, 1976).

In a study by Maltoni & Maioli (1977) and Maltoni et al. (in press), groups of 30 male and 30 female, 13-week-old Sprague Dawley rats were given trichloroethylene (highly purified and epoxide-free, and stabilized with 20 ppm of butyl-hydroxytoluene) in olive oil by gavage, 5 days a week, for 52 weeks. Two doses of trichloroethylene were tested. The high-dose animals received 250 mg/kg body weight, and the low-dose animals 50 mg/kg body weight; 30 males and 30 females received olive oil alone and served as controls. The animals were kept until their spontaneous death (the study lasted 140 weeks). There was no increase in specific tumours. Abnormalities (cytokaryomegaly) in cells of the renal tubules were observed in high-dose male rats. These changes were not observed in females.

Groups of 30 female ICR Swiss mice and a group of 30 male and 30 female mice of the same strain, were treated with trichloroethylene dermally (1 mg in acetone), 3 times weekly, for 83 weeks, subcutaneously (0.5 mg in trioctanoin) once weekly for 89 weeks, and by gavage (0.5 mg in trioctanoin), once weekly, for 89 weeks. Similar-sized groups of animals served as controls. No tumours were observed at the site of application (Van Duuren et al., 1979). Similar negative findings were obtained following thrice-weekly skin applications and once-weekly subcutaneous injections of trichloroethylene oxide for life in a group of 70 female mice of the same strain (Van Duuren et al., 1983)

Groups of 30 male and 30 female Wistar rats, NMRI mice, and Syrian hamsters were exposed by inhalation to trichloroethylene (purified, with no detectable epoxides, and containing 15 ppm triethanolamine as stabilizer) at 540 and 2700 mg/m^3 (100 and 500 ppm). The animals were exposed for 6 h per day, 5 days per week, for 78 weeks. The studies were terminated by killing the surviving mice and hamsters after 130 weeks, and rats after 156 weeks. No carcinogenic effects were observed in rats, hamsters, and male mice. A moderate increase in lymphomas was found in treated female mice (18/28 at 2700 mg/m^3; 17/30 at 540 mg/m^3, and 9/29 in controls). The authors concluded that, on the basis of these findings, there were no indications of carcinogenicity (Henschler et al., 1980).

Groups of 49 - 50 female ICR mice, 4 weeks of age, were exposed by inhalation to trichloroethylene (purity 99.8% with 0.128% carbon tetrachloride, 0.019% benzene, 0.019% epichlorohydrin, and 0.01% 1,1,2-trichloroethane) at 0.270, 810, or 2430 mg/m^3 (0, 50, 150, or 450 ppm), 7 h per day for 5 days per week, for 104 weeks. The surviving animals were killed 107 weeks after the start of the study. Mortality was similar in control and treated groups. Lung adenomas were found in 5, 2, 5, and 4 mice in the control, low-dose, mid-dose, and high-dose groups, respectively. However, it was reported that adenocarcinomas (none of which gave rise to metastases) occurred in 1/49, 3/50, 8/50, and 7/46 mice in the control, low-dose, mid-dose, and high-dose groups, respectively, and that increased incidences in the mid- and high-dose groups, were statistically significant compared with controls. In similar studies on Sprague Dawley rats, no statistically-increased incidence of tumours was detected; one clear-cell carcinoma of the kidney was observed in the high-dose group (Fukuda et al., 1983).

In a large series of inhalation studies, highly purified epoxy-free trichloroethylene (stabilized with 20 ppm butyl-hydroxytoluene) was studied under controlled exposure conditions in male and female Sprague Dawley rats and Swiss and B6C3F1 mice. Treatment was for 7 h per day, 5 days per week for 8, 78, or 104 weeks. After the end of the treatment, all animals were kept under observation until spontaneous death. The following 7 studies were performed (Maltoni & Maioli, 1977; Maltoni et al., in press):

Study 1

Groups of 60 - 90 male and 60 - 90 female Sprague Dawley rats were exposed to 0, 540, or 3240 mg trichloroethylene/m^3 (0, 100, or 600 ppm) for 8 weeks. No increase in tumours related to treatment was observed. Cytomegaly and karyomegaly of tubular cells in the kidney were not observed at either dose.

Study 2

Groups of 60 - 100 male and 60 - 100 female Swiss mice were exposed to trichloroethylene at 0, 540, or 3240 mg/m^3 (0, 100, or 600 ppm) for 8 weeks. No increase in tumours related to treatment was observed.

Studies 3 and 4

Groups of 90 - 95 male and 90 - 105 female Sprague Dawley rats were exposed to trichloroethylene at 0, 540, 1620, 3240 mg/m^3 (0, 100, 300, or 600 ppm) for 104 weeks. Further groups of 40 males and 40 females were started on the same treatment 4 - 5 weeks later (Study 4). The method and results of the 2 studies were similar and the combined data are described. No increase in mortality was observed in the treated animals. Cytomegaly and karyomegaly of renal tubular cells were observed in males in the mid- and high-dose groups and the incidence was dose-related; no such lesions were observed in males in the low-dose group or in females at any of the 3 dose levels, or in the controls. Five tubular adenocarcinomas were observed in 4/130 males and 1/130 females at the high-dose level. Leydig cell tumours of the testis were observed in 6/135 controls, and in 16/130, 30/130, and 31/130 male rats, respectively, in the low-, mid-, and high-dose treated groups. The average time of appearance of the tumours, from the start of the study, was 113 weeks in controls and from 109 to 113 weeks in treated animals. A higher incidence of immunoblastic lymphosarcomas was detected in male and female treated animals: 1/280 in controls, 9/260 at the low dose, 5/260 in the mid-dose group, and 3/260 in the high-dose group.

Study 5

Groups of 90 male and 90 female Swiss mice were exposed to trichloroethylene at 0, 540, 1620, or 3240 mg/m^3 (0, 100, 300, or 600 ppm) for 78 weeks. The incidence of hepatocellular carcinomas in males was 4/90 in controls compared with 2/90 at the low dose, 8/90 at the mid dose, and 13/90 at the high dose. One hepatocellular carcinoma occurred in a female at the high dose. The incidences of lung adenomas and adenocarcinomas were 25/180 in controls, 26/180 in low-dose groups, 36/180 in mid-dose, and 47/180 in the high-dose groups (males and females combined). Two adenocarcinomas were seen in the control groups and 3 in the high-dose group.

Studies 6 and 7

For study 6, groups of 90 male and 90 female B6C3F1 mice were exposed to trichloroethylene at 0, 540, 1620, or 3240 mg/m^3 (0, 100, 300, or 600 ppm) for 78 weeks. Because of excess mortality due to fighting in the male groups, study 7 was started in which trichloroethylene was tested in the same way in equal numbers of male mice. In females, the incidence of pulmonary tumours (all adenomas except one adenocarcinoma in the low-dose group) were 4/90 in controls compared with 6/90, 10/90, and 15/90 in the low-, mid-, and high-dose groups, respectively. Hepatocellular carcinomas were observed in 3/90 controls compared with 4/90, 4/90, and 9/90 in the low-, mid-, and high-dose groups, respectively. In the males in

study 7, the incidence of pulmonary tumours was not affected by the treatment. Hepatocellular carcinomas were observed in 14/90 controls compared with 19/90, 27/90, and 21/90 in the 3 treated groups, respectively. Cytomegaly and karyomegaly were not observed in either study (Maltoni et al., in press).

Groups of 50 male and 50 female rats (F344) and mice (B6C3F1) were treated, by corn-oil gavage, with epichlorohydrin-free trichloroethylene at 500 and 1000 mg/kg body weight (rats), and 1000 mg/kg body weight (mice), 5 times weekly, for 103 weeks. Similar-sized groups of male and female rats and mice treated with corn oil alone, or without any treatment, served as controls. The animals were killed between 103 and 107 weeks from the start of the study. Trichloroethylene significantly reduced the survival of male rats and mice. In male rats treated with trichloroethylene, the incidence of tubular-cell neoplasms was increased (0/49, untreated; 0/48, corn-oil control; 2/49, low dose; 3/49, high dose). There was one tubular-cell tumour in a high-dose female rat. Five low-dose male rats also had malignant peritoneal mesotheliomas, compared with 1/50 untreated control, 1/50 vehicle control, and 0/49 high-dose animals. Trichloroethylene also produced nephrosis in both sexes of both species. In mice, the administration of trichloroethylene increased the incidence of hepatocellular carcinoma (males: 8/48, controls; 30/50, treated; females: 2/48, controls; 13/49, treated). The incidence of hepatocellular adenomas was also increased in male mice (3/48, controls; 8/50, dosed) and in female mice (2/48, controls; 8/49, dosed) (US NTP, 1983).

Groups of 50 male and 50 female Swiss mice were treated by corn-oil gavage with daily doses of different samples of trichloroethylene, initially at 2400 mg/kg body weight (males) and 1800 mg/kg body weight (females), 5 days a week, for 78 weeks. Due to toxicity, dosing and dose levels were reduced during the study. The animals were kept under observation until spontaneous death. The samples included: (a) highly purified trichloroethylene stabilized with 0.0015% triethanolamine, (b) industrial trichloroethylene (99.4% pure), (c) highly purified trichloroethylene stabilized with 0.8% epichlorohydrin, (d) highly purified trichloroethylene, with 0.8% 1,2-epoxybutane, and (e) highly purified trichloroethylene with 0.25% epichlorohydrin and 0.25% epoxybutane. Similar-sized groups of each sex, treated with corn oil alone, served as controls. Mortality was increased in treated males and some treated female groups. No increase in tumour incidence was observed except in groups (c) and (d) where there was an increased incidence of forestomach cancers, attributed to the direct alkylating properties of one of the two stabilizers (Henschler et al., 1984).

6.1.7.1. Conclusions

Trichloroethylene (with or without epoxide stabilizers) caused an increased incidence of hepatocellular carcinomas in 2 different strains of mice, either when given by oral administration or by inhalation.

Trichloroethylene also produced lung tumours in Swiss ICR mice and in female, but not male, B6C3F1 mice.

Trichloroethylene, with added epichlorohydrin, administered by gavage, caused an increased incidence of forestomach cancers in WMRI mice.

Trichloroethylene (epoxide stabilizer-free) produced a low incidence of renal tubular tumours in 2 different strains of mice (mainly in males) following long-term oral or inhalation exposure. These tumours occur very rarely in untreated rats of these strains.

A dose-related increase in Leydig (interstitial) cell tumours of the testis was observed in one study on male Sprague Dawley rats following long-term inhalation exposure.

In one strain of mice and one strain of rats, epoxide stabilizer-free trichloroethylene produced some increases in the incidence of lymphomas. However, these data are insufficient to

draw any conclusions.

Thus, there is clear evidence that trichloroethylene is carcinogenic in mice. There is also some evidence that trichloroethylene causes tumours in rats.

It was noted in the evaluations by IARC (1979, 1982) that it was considered that there was limited evidence for the carcinogenicity of trichloroethylene for experimental animals.

The significance of these findings needs to be evaluated in the context of further studies on the mechanism of action of trichloroethylene.

6.1.8. Mutagenicity

6.1.8.1. Gene mutation

(a) *Bacteria and fungi*

Pure trichloroethylene induced revertants (gene-mutations) in the K-12 strain of *Escherichia coli* in the presence of fortified mouse Arochlor(R)-induced microsomal preparations for only one gene out of four analysed. In the positive case, the induced effect was the doubling of spontaneous background (Greim et al., 1975).

Trichloroethylene (of unspecified purity) in vapour form was slightly mutagenic for the TA-100 strain of *Salmonella typhimurium*, in the presence of S9 mix obtained from mouse liver (control value = 140 revert./plate; treated values: 240 revert./plate) (Simmon et al., 1977).

Technical grade trichloroethylene (containing 1900 ppm of 1,2-epoxybutane and 900 ppm of epichlorohydrin) was mutagenic for the TA-100 strain of *S. typhimurium* in a desiccator, in the absence of a metabolic activation system (untreated 161 - 187 revert./plate; treated: 567 - 651 revert./plate). Trichloroethylene of high purity was not mutagenic for the TA 100 strain of *S. typhimurium* in the presence of 0.5 ml of S9 mix obtained from mouse, rat, and hamster liver; it was, however, mutagenic for this strain of *S. typhimurium* in the presence of 50 - 150 µl of S9 mix from Arochlor-treated rat liver (untreated series: 160 - 180 revert./plate; treated series: 350-379 revert./plate) (Crebelli et al., 1982).

High-purity trichloroethylene was not mutagenic for the TA 100 strain of *S. typhimurium* in a plate assay, with and without addition of S9 mixture (Henschler et al., 1977).

Trichloroethylene containing no traces of 1,2-epoxybutane or epichlorohydrin was tested as a gas in a desiccator on the TA 1535 and TA 100 strains of *S. typhimurium*. Concentrations of 1 - 3% produced a 30% increase (less than a doubling) in revertants in strain TA 100 in the presence of a metabolic activation system from Arochlor(R)-induced rat liver (Baden et al., 1979).

The exposure of *S. typhimurium* strains TA 98 and TA 100 to 0.5 - 10% vapour concentrations of trichloroethylene in sealed vials for 48 h did not produce an increase in revertants either in the presence or absence of rat liver homogenate (Waskell, 1978).

Trichloroethylene of high purity (99.5%) was mutagenic for the TA-100 strain of *S. typhimurium* when used as a gas in a desiccator in the presence of S9 mix (Bartsch et al., 1979). However, it should be noted that the increase in revertants in the treated series was less than twice that of the untreated series, but this slight effect was consistent in repeated experiments.

Trichloroethylene of unspecified purity was reported to double the spontaneous frequency of reverse mutations in the D-7 strain of the yeast *Saccharomyces cerevisiae*, at one concentration, in the presence of an endogenous metabolic activation system (Callen et al., 1980).

Trichloroethylene containing impurities, which were not specified, was mutagenic for *S. cerevisiae*, strain XV185-14C (reverse mutations) in the presence of S9 mix. These results were obtained at a level of yeast cell % survival of < 1 (1 h of treatment) and of < 0.1 (4 h of treatment). These conditions are unusual for the detection of mutagenic effects, since, at these toxic values, selection of different cells (wild type and mutants) is very efficient (Shahin & von Borstel, 1977). One of the authors (von Borstel, personal communication, 1984) stated that, when these studies were repeated with a pure trichloroethylene sample, it was not mutagenic for yeast cells.

Trichloroethylene of high purity (99.8%), as well as trichloroethylene containing stabilizers such as 1,2-epoxy-butane (0.19%) and epichlorohydrin (0.09%), were not mutagenic for the yeast *S. pombe* (forward mutations), with or without a metabolic system obtained from rats and mice treated with phenobarbital and beta-naphthoflavone. Using intraperitoneal or intrasanguineous, host-mediated assay techniques with male mice inoculated with the yeast *S. pombe* and treated orally with 2 g/kg body weight for

4 or 16 h, both pure trichloroethylene and the stabilized trichloroethylene were found not to be mutagenic (Rossi et al., 1983).

A slightly mutagenic action for cells of *S. cerevisiae* strain D7 (reverse mutation at *his* locus) was found for trichloroethylene of unspecified purity in the presence of a metabolic activation system obtained from mice (untreated series: 0.79 - 0.89 *his*, rev/10^6; treated series: 3.00 - 3.60 *his* rev/10^6 with 40 mol). For this experiment, no survival values were reported, nor were the actual number of revertants counted, and therefore the observed effect cannot be definitely assigned to trichloroethylene (Bronzetti et al., 1978). The same authors administered trichloroethylene (unspecified purity) orally to mice (400 mg/kg body weight) that were also inoculated with yeast cells intra-sanguineously; a mutagenic effect was found on yeast cells (reverse mutations at the *his* locus) collected from liver, lung, and kidney but, again, the survival level was not reported nor was the number of revertants counted (Bronzetti et al., 1978).

Trichloroethylene of high purity and containing known contaminants (_80 ppm) was found to be active for the production of reverse mutations (suppressor mutations) on *Aspergillus nidulans sumeth* A1 strain at a dose of 27 000 mg/m^3 (5000 ppm) applied in a desiccator containing the plates inoculated with the fungal conidia (Crebelli et al., in press).

(b) *Mammalian cells (in vitro)*

A pure sample of trichloroethylene, stabilized with thymol, did not induce mutations (forward mutations) at the HPRT locus of Chinese hamster V-79 cell line treated *in vitro* with or without metabolic activation (S9 mix) (Loprieno & Abbondandolo, 1980).

(c) *Mammals (in vivo)*

Trichloroethylene (purity 99.5%) was evaluated for its ability to induce somatic gene mutations *in vivo* in embryonic fibroblasts of mice (spot test). Pregnant mice were injected 12 days after copulation with a dose of 140 mg/kg body weight (50 females), or 350 mg/kg (26 females). The results observed were 2/145 (1.4%) and 2/51 (3.9%) of progeny with "spots", respectively, whereas the untreated series produced 0/146, 3/182 (1.6%), 6/794 (0.75%). These results were considered by the authors to indicate a mutagenic effect (Fahrig, 1977); however, positive data such as those observed for trichloroethylene are among spontaneous values for this test system.

6.1.8.2. Chromosome aberrations

(a) *Mammals (in vivo)*

Trichloroethylene (purity not given), repeatedly administered intraperitoneally to ICR mice for 5 days at a dose equivalent to

half of the LD_{50}, did not induce a significant increase in chromosome aberration frequency in bone marrow cells from animals killed 6, 24, and 48 h after the last treatment (Cerna & Kjpenova, 1977).

A single dose of trichloroethylene of 1000 mg/kg body weight (stabilized with thymol), administered orally to Swiss albino mice, did not induce a significant increase in the frequency of chromosome aberrations in bone marrow, 24 h after treatment (Loprieno & Abbondandolo, 1980).

The exposure of male mice (NMRI-Han/BGA) to trichloroethylene vapour (99.5% purity) for 24 h at concentrations of 272, 1090, and 2430 mg/m^3 (50, 202, and 450 ppm) did not induce dominant lethal mutations (Slacik-Erben et al., 1980).

CDI male mice were treated orally with 3000, 2250, 1500, 1125, 750, or 375 mg/kg body weight (2 treatments in 24 h) with a trichloroethylene sample of high purity (99.5%) and sacrificed 6 h following the second treatment, for the analysis of the frequency of micronucleated erythrocytes in the bone marrow cells. The results indicated a positive dose-related effect of trichloroethylene (Duprat & Gradiski, 1980).

Using high purity (99.8%) trichloroethylene, B6C3F1 mice (10 female and 10 male) were treated by inhalation with 3240 mg/m^3 (600 ppm), for 7 h a day, 5 days a week, for a total of 52 days. The animals were killed 6 h and 24 h after the last treatment. The cytogenetic analysis showed the presence only of gaps without any indication of induced chromosomal aberrations (Loprieno, personal communication, 1984).

When trichloroethylene of high purity (99.8%) was administered to B6C3F1 male mice, by gavage, in a single dose of 1200 mg/kg body weight, an increased frequency (4 times) of micronucleated erythrocytes in the bone marrow cells compared with control animals was observed, 42 h after dosing (Sbrana et al., 1984).

6.1.8.3. DNA damage

Fungi

Trichloroethylene (unspecified purity) was positive for the induction of mitotic gene conversion at the *trp* locus and mitotic recombination in the D7 strain of *S. ceverisiae*, under endogenous metabolic activation conditions (Callen et al., 1980). Trichloroethylene (unspecified purity) was also found to be positive for the induction of mitotic gene conversion at the *trp* locus of *S. cerevisiae* D7 strain treated *in vitro* in the presence of a metabolic activation system obtained from rat liver. Using the host-mediated assay technique, and a single oral dose of 400 mg/kg body weight, given to mice inoculated with yeast cell D4 strain of *S. cerevisiae*, a positive effect on the induction of mitotic gene conversion at the *trp 5* and *ade 2* loci was observed in the yeast cells recovered from the kidney. Repeated oral administration of trichloroethylene to male mice, with a total dose of 3700 mg/kg body weight, induced a positive effect (mitotic recombination) in yeast cells recovered from the liver and kidney. In all these studies, the actual numbers of the convertant or recombinant colonies were not given, and the significance of the results is questionable (Bronzetti et al., 1978).

Trichloroethylene (stabilized with thymol) was considered inactive for the induction of mitotic recombinations in the D4 strain of *S. cerevisiae* following *in vitro* and *in vivo* (host-mediated assay) studies (Loprieno & Abbondandolo, 1980).

Trichloroethylene of high purity (99.9%) induced somatic segregants in *A. nidulans* strain 35 x 17, at concentrations of 40 500 and 81 000 mg/m^3 (7500 and 15 000 ppm), in a desiccator containing the plates inoculated with the fungal conidia; under identical doses and conditions, no mitotic recombinant colonies were induced in this strain of *A. nidulans* by trichloroethylene

(Crebelli et al., in press).

6.1.8.4. Mammalian cells (*in vitro*)

Trichloroethylene of high purity (99.9%) induced morphological transformations, *in vitro*, in rat embryo cell cultures (Price et al., 1978).

Stabilized with thymol, trichloroethylene did not stimulate unscheduled DNA synthesis in an HeLa human cell line grown *in vitro*, in the presence or absence of a metabolic activation system (S9 mix) (Loprieno & Abbondandolo, 1980). Trichloroethylene (unspecified purity) elicited DNA repair synthesis in human lymphocytes in the presence of S9 mix (Perocco & Prodi, 1981).

Exposure of Chinese hamster ovary cells (CHO) to trichloroethylene bubbled into the growth medium for 2 min, corresponding to a dose of 0.17% v/v for 1 h treatment time, did not induce sister-chromatid exchange (White et al., 1979).

Trichloroethylene bound covalently to calf thymus DNA *in vitro* in the presence of a rat liver metabolic activation system (Di Renzo et al., 1982; Bergman, 1983).

When given ip to NMRI mice, twice daily for 5 days (33.6 mg/kg), trichloroethylene bound to RNA and DNA of brain, lung, liver, kidney, spleen, pancreas, and testis (Bergman, 1983).

6.1.8.5. Mutagenic activity of trichloroethylene metabolites

Trichloroethylene oxide was not mutagenic for the TA 1535 strain of *S. typhimurium* or the WP2 UVRA strain of *E. coli* (plate test), but was positive in the DNA repair test on *E. coli* (Kline et al., 1982).

The same compound was found to be a direct mutagen for the yeast *S. pombe* (forward mutation) and for mammalian cells grown *in vitro* (V-79 cell line) (forward mutation) (Loprieno & Abbondandolo, 1980).

Syrian golden hamster embryo cells were exposed to trichloroethylene oxide (1.1, 2.5, and 5.0 μM) at 37 °C for 30 min. After 5 days, the number of transformed colonies was scored. A low but dose-related increased frequency of transformed cells was induced (Di Paolo & Doniger, 1982).

Chloral hydrate has been reported to have direct genotoxic activity in *S. typhimurium* (Waskell, 1978) and in *A. nidulans* (Bignami et al., 1980).

At doses of 82.7, 165.4, or 4135 mg/kg body weight, chloral hydrate, administered intraperitoneally, induced chromosome non-disjunction in the secondary spermatocytes of mice (Russo et al., 1984).

6.1.8.6. Conclusions

Pure trichloroethylene acts as a weak mutagen in *S. typhimurium* in the presence of a metabolic activation system. However, in all cases, the induced effect was never more than twice the spontaneous value. Technical grade trichloroethylene containing stabilizers such as 1,2-epoxybutane epichlorohydrin, is mutagenic in *S. typhimurium* in the absence of a metabolic activation system.

Pure trichloroethylene is not mutagenic for the yeast *S. pombe*, with or without metabolic activation, *in vitro* or *in vivo* (host-mediated assay).

Thymol-stabilized trichloroethylene is not mutagenic for V-75 hamster cells, with or without metabolic activation.

Trichloroethylene (99.5% pure) is a weak mutagen for the induction of somatic mutations in mouse embryo.

In mice, a single oral dose of trichloroethylene (1 g/kg body

weight) or repeated exposure to 3240 mg/m^3 (600 ppm) for 52 days, 7 h/day, through inhalation, did not increase chromosome aberration frequency in bone-marrow cells. A half-LD_{50} dose of trichloroethylene, administered ip to mice, did not increase chromosomal aberration frequency in bone-marrow cells. In a micronucleus test, a single oral treatment with 1200 mg/kg body weight or repeated (twice) oral treatment with a series of doses ranging from 375 to 3000 mg/kg body weight increased the frequency of induced micronucleated erythrocytes in the bone-marrow cells of mice. Trichloroethylene bound to DNA *in vitro* and *in vivo* to mouse tissues.

Dominant lethal mutations were not induced in mice exposed through inhalation to trichloroethylene vapours.

Trichloroethylene induces mitotic gene conversion in yeast cells grown under conditions allowing an endogenous metabolic activation system to function. The same effect reported in yeast in the presence of an exogenous metabolic activation system

(S9 mix), or in the host-mediated assay, is questionable, because of inadequate data and the fact that other studies produced negative data.

A positive effect for the induction of unscheduled DNA synthesis (UDS) in human lymphocytes, treated *in vitro*, was reported by Perocco & Prodi (1981), whereas a negative response for the same biological effect (UDS) in a human fibroblast cell line (HeLa) grown *in vitro* was observed by Loprieno & Abbondandolo (1980).

Trichloroethylene induces morphological transformation in rat embryo cells.

The available results, shown in Table 9, are not adequate for a complete evaluation of the genotoxic potential of trichloroethylene. Only a few mutagenicity studies have indicated the grade and purity of the trichloroethylene sample employed. The confusing results could be due either to the use of trichloroethylene samples stabilized with mutagenic compounds or to the use of pure trichloroethylene samples which, unstablized, can rapidly decompose to chemicals with mutagenic activity.

6.1.9. Reproduction, embryo/fetotoxicity, and teratology

The embryo/fetal toxicity and teratogenic potential of trichloroethylene have been evaluated in the avian embryo system, the mouse, rat, and rabbit. Reproductive function has been examined in male rats.

6.1.9.1. Avian embryo system

Exposing avian embryos to 1% trichloroethylene (10 g/litre) caused a significant increase in embryonic death and a slight increase in anomalies (Fink, 1968). These observations were made on the 3rd day of development, a time at which frank malformations would be difficult to observe or evaluate. Elovaara et al. (1979) reported the results of a comparative study of the teratogenic potential of trichloroethylene and other aliphatic chlorinated hydrocarbons in the chick embryo. Trichloroethylene (reagent grade) in doses ranging from 5 to 1000 µmoles in olive oil (25 µl) was injected into the air space of fertilized eggs on the 2nd and 6th days of incubation and examined on the 14th day. The LD_{50} for trichloroethylene was between 50 and 100 µmoles/egg. Macroscopic embryonic malformations occurred in 9 out of 55 surviving embryos, 5 times the incidence in the vehicle control eggs.

In another study, injection of low doses of trichloroethylene (1, 5, 10, and 25 µmol/egg), on days 1 and 2 of embryogenesis, resulted in embryotoxicity, growth defects, and morphological anomalies (Bross et al., 1983).

Table 9. Summary of the mutagenicity data reported for trichloroethylene

Organism	Genotoxic effects		
	Gene mutation	Chromosome anomalies	DNA damage
prokaryotes	weak positive		
fungi	negative (*in vitro*) negative (hma) positive? (*in vitro*) positive *(A. nidulans)*	positive mitotic segregation *A. nidulans*	negative (*in vitro*) weak (hma) positive (yeast metabolism)
mammalian cells (*in vitro*)	negative		negative (SCE) negative (DNA repair synthesis) positive (covalent binding)
mammals (*in vivo*)	weak	negative (1 dose) (1 g/kg body weight, oral) negative (52 doses) (3240 mg/m^3, inhalation) negative (5 doses) (1/2 LD_{50}, ip) positive (micronuclei) 1200 mg/kg positive (micronuclei) (375 - 3000 mg/kg) negative (dominant lethals) (270, 1090, 2430 mg/m^3, inhalation)	

For human cases, see Table 10.

6.1.9.2. Mouse

Swiss-Webster mice were exposed by inhalation to trichloroethylene at 1620 mg/m^3 (300 ppm), for 7 h daily, on days 6 - 15 of gestation (Day 0 = vaginal plug). The mice were observed daily throughout pregnancy. Maternal body weights were recorded on days 6, 10, and 18 of gestation as an index of maternal toxicity. On day 18, no effect was observed on the average number of implantation sites/litter, litter size, the incidence of fetal resorptions, fetal sex ratios, or fetal body measurements (Schwetz et al., 1975). The incidence of gross anomalies observed by external examination was not significantly greater than that among the control litters. Trichloroethylene exposure did not exert any effect on the incidence of skeletal anomalies in mice, and microscopic examination did not reveal any abnormalities in organs, tissues, or cells (Leong et al., 1975; Schwetz et al., 1975).

6.1.9.3. Rat

In studies similar to the preceding mouse study, exposure to trichloroethylene at 1620 mg/m^3 (300 ppm) did not produce any evidence of maternal toxicity or teratogenicity in Sprague Dawley rats (Leong et al., 1975; Schwetz et al., 1975) or Charles River strain rats (Bell, 1977).

Exposing Sprague Dawley rats to trichloroethylene at 2700 mg/m^3 (500 ppm) for 7 h per day, 5 days per week, during a 3-week pregestational period, and 7 h per day each day for days 0 - 18 and days 6 - 18 of gestation did not cause maternal or embryo/fetal toxicity. Beliles et al. (1980) concluded that the frequency and the character of the macro- or microscopic findings in the treated groups did not indicate either adverse fetal effects or that trichloroethylene, at the dose used, was teratogenic.

Dorfmueller et al. (1979) reported that female Long-Evans rats exposed to trichloroethylene at 9720 mg/m^3 (1800 ppm) before mating, for 6 h per day, 5 days per week, for 2 weeks and/or during pregnancy, for 6 h per day, each day, for 0 - 20 days of gestation) did not exhibit any signs of maternal toxicity. There was no indication of embryotoxicity, and no significant treatment effects or interactions were found in the number of corpora lutea or implantation sites/litter, fetal body weights, resorbed fetuses/litter, or sex ratios. No significant treatment effects

were observed in the analysis of total soft-tissue anomalies. However, prenatal exposure to trichloroethylene at 9720 mg/m^3 (1800 ppm) caused an increase in minor anomalies of the offspring (incomplete ossification of the sternum) indicative of development delays in maturation, but not of major malformation.

While the study did not indicate any treatment-related effects on general post-natal behaviour, there was a small but significant regression in post-natal weight gain in offspring in the pre-mating exposure group.

In groups of male rats dosed by gavage with trichloroethylene at 10, 100, or 1000 mg/kg body weight 5 days per week, for 6 weeks, there was no evidence of spermatotoxic effects. Trichloroethylene-related effects of reduced weight gain and elevated liver/body weight ratios were seen primarily in the 1000 mg/kg group. Copulatory behaviour was initially diminished by the narcotic properties of trichloroethylene, but was normal by the 5th week (Zenick et al., 1984).

6.1.9.4. Rabbit

Female New Zealand white rabbits were exposed by inhalation to trichloroethylene at 2700 mg/m^3 (500 ppm), for 7 h per day, 5 days per week, during a 3-week pregestation period, and daily, for days 0 - 21 and days 7 - 21 of gestation (Day 0 = mating day). There was no evidence of maternal toxicity or embryotoxicity. The occurrence of hydrocephalus in a few fetuses in one of the study groups was reported (4 fetuses in 2 litters), but no definitive conclusion was drawn from these findings (Beliles et al., 1980).

7. EFFECTS ON THE ENVIRONMENT

7.1. Aquatic Organisms

There is little information on the toxicity of trichloroethylene for fish. The US Registry of Toxic Effects of Chemical Substances (Christensen & Lugenbyhl, 1975) reports, for an unidentified species, that exposure to a concentration range of 100 - 1000 mg/litre produced toxic effects in 96 h. Toxicity tests carried out on salt-water flatfish, *Limanda limanda* (sole), 15 - 20 cm long, in a continuous water flow, established a 96-h LC_{50} of 16 mg/litre (Pearson & McConnell, 1975). A 96-h LC_{50} of approximately 40 mg/litre (static) or 67 mg/litre (continuous flow) has been reported for the minnow *Pimephales promelas* (Alexander et al., 1978).

Verschueren (1977) established an LC100 of 600 mg/litre for *Daphnia magna*. The LC_{50} for the balanide salt-water crustacean nauplius (larva) *(Elminius modestus)* was 20 mg/litre after 46 h (Pearson & McConnell, 1975), and the LC_{50} for the protozoon *Entosiphon sulcatum* was established as 1200 mg/litre (Bringmann & Kuhn, 1980).

Various LC_{50} values have been established for algae including 63 mg/litre for *Microcystis aeruginosa*, (Verschueren, 1977); a concentration of 1000 mg/litre did not have any observable effect on *Scenedesmus quadricauda* (Bringmann & Kuhn, 1980). A short-term photosynthesis efficiency test gave an LC_{50} of 8 mg/litre (Pearson & McConnell, 1975) and, finally, in tests carried out on *Thalassiosira pseudonana* and *Dunaliella tertiolecta*, there were observable effects at 50 and 100 µg/litre, in a mixed culture (Biggs et al., 1979).

7.2. Uptake, Distribution, Storage, Metabolism, and Elimination in Plant and Animal Organisms

Bioaccumulation of trichloroethylene in a marine environment has been studied by Pearson & McConnell (1975); concentration levels were determined for a wide variety of marine organisms, mostly in the Bay of Liverpool.

The greatest increase in trichloroethylene concentrations in the tissues of animals that are relatively high up in the food

chain (birds' eggs, fish liver, and seal fat) was nearly 100 times the level in water (from $0.5/10^9$ µg/litre in water to $50/10^9$ µg/kg in tissues).

These data agree with the laboratory findings of Barrows et al. (1980) who, in a 14-day test, noted that trichloroethylene accumulation in the sunfish species, *Lepomis macrochirus* was 17 times that of the aquatic medium with a halving time of less than one day. A low bioconcentration factor (concentration in organism divided by concentration in environment) (Uehleke et al., 1977) has been derived using water solubility and the equation proposed by Kenaga (1980) and Kenaga & Goring (1980).

7.3. Effects on the Stratospheric Ozone Layer

Consideration has been given to the possibility that trichloroethylene, together with other halocarbons in the atmosphere, may contribute to the depletion of the stratospheric ozone layer, which would lead to atmospheric heating and increased exposure of terrestrial biota to ultraviolet radiation (Molina & Rowland, 1974). Atmospheric trichloroethylene concentrations seem to be about one-fifth to one-tenth of those of other chlorocarbons such as CH_3CCl_3, CH_2Cl_2, CCl_4, or C_2Cl_4 or the major chlorofluorocarbons (Cronn et al., 1977; Penkett, 1982). The reason for this is that while trichloroethylene emissions into the atmosphere are of the same order as those of other halocarbons (Jesson, 1980), trichloroethylene is efficiently scavenged by hydroxyl radicals in the troposphere, and the reaction rate for this process is appreciably faster for trichloroethylene than for other halocarbons (Penkett, 1982). Thus, the predicted atmospheric lifetime for trichloroethylene is short (about 10 - 11 days) (Derwent & Eggleton, 1978; Graedel, 1978) compared with those for the chlorofluorocarbons, which may be 10 years or more (Jesson, 1980). It is not clear whether trichloroethylene is even present in the stratosphere (Cronn et al., 1977). However, the data suggest that trichloroethylene is unlikely to be involved in the possible depletion of the ozone layer.

8. EFFECTS ON MAN

8.1. General Symptoms and Signs

8.1.1. Acute effects

Sorgo (1976) reported a lethal oral dose for trichloroethylene to be 7 g/kg body weight, and Lazarev et al. (1977) reported a fatality after the oral ingestion of less than 50 ml.

At exposure concentrations of 270 - 540 mg/m^3 (50 - 100 ppm), the most common symptoms are headache, sluggishness, sleepiness (especially at the end of the workshift), dulling of senses, dizziness, nausea, and vomiting (Rubino et al., 1959; Lilis et al., 1969; Governa, 1981). At narcotic doses, vomiting may occur.

Trichloroethylene has been used as an analgesic and as an anaesthetic on its own or in mixtures. It is now much less used for this purpose, as safer and more effective alternatives are available. Inhalation of 27 000 mg/m^3 (5000 ppm) produces light anaesthesia; concentrations of up to 108 000 mg/m^3 (20 000 ppm) have been used for deeper anaesthesia (Wade, 1977).

Clinical signs are not very specific. There can be enhanced responsiveness to caloric stimulation of the labyrinth and some EEG anomalies may be detected (Chiesura, 1980) (section 8.2). Acute hepatic and renal toxicity has been reported (Sucui & Olinici, 1983).

A particular intolerance to ethanol may occur after ingesting even small amounts of alcoholic beverages. This intolerance is characterized by intense cutaneous vasodilatation, particularly in the face, often called "degreaser's flush" (Stewart et al., 1974).

Non-specific effects on the digestive system (e.g., dyspepsia, gastritis, and diarrhoea) have been reported in cases of suicidal

or accidental ingestion of trichloroethylene (Bozza Marrubini et al., 1978; Governa, 1981).

8.1.2. Chronic effects

The existence of a condition of chronic trichloroethylene poisoning is not clear (Chiesura, 1980; Boudène et al., 1983). However, chronic effects in human beings can occur following prolonged exposure to moderate concentrations of trichloroethylene of about 540 mg/m^3 (100 ppm). The clinical picture is mainly related to the central nervous system (CNS), and consists of asthenia, anorexia, headache, loss of memory, moodiness, depression, insomnia, paraesthesia, and disturbances of the autonomic nervous system such as hyperhydrosis, tachycardia, and dermographism (Ahlmark & Forssman, 1951a,b; Bardodej & Viskocil, 1956; Rubino et al., 1959; Lerza et al., 1963; Castellino, 1969).

The role of trichloroethylene in inducing liver damage in occupationally-exposed human beings is not clear (Parmeggiani, 1956; Capellini & Grisler, 1958; Rubino et al., 1959; Tolot et al., 1964; Schuttmann, 1970; Lachnit, 1971; Thiele, 1982; Sucui & Olinici, 1983; Svabova & Mencik, 1983).

Human subjects with high repeated, but non-occupational, exposure may exhibit toxic effects on the liver (e.g., elevated aspartate (EC 2.6.1.1) and alanine aminotransferase (EC 2.6.1.2)), renal insufficiency, and abnormal EEG patterns (Baerg & Kimberg, 1970). Following accidental or suicidal ingestion (e.g., doses of 100 - 300 ml), hepatic necrosis and nephropathy have been found in autopsy cases (Keinfeld & Tabershaw, 1954; Graovac-Leposavic et al., 1964; Priest & Horn, 1965; Beisland & Wannag, 1970; Clearfield, 1970). A decrease in sexual potency has been reported in industrial workers exposed to trichloroethylene (Bardodej & Vyskocil, 1956; Lazarev & Gadaskiva, 1977).

Trichloroethylene is one of the volatile solvents inhaled ("sniffed") for its euphoric effect (Hayden et al., 1976), and abuse by oral self-administration has been reported (Wells, 1982). Centrilobular hepatic necrosis and renal toxicity have occurred in addicts (Baerg & Kimberg, 1970; Clearfield, 1970), and deaths have been reported (James, 1963; Musclow & Awen, 1971). Some mixtures sold commercially as trichloroethylene may contain other solvents, including carbon tetrachloride, which can contribute to the toxic manifestations (Bouyges et al., 1980; Conso et al., 1980). Dependent patients may show withdrawal symptoms (Ikeda et al., 1971).

8.2. Effects on Organs and Systems

8.2.1. Effects on the nervous system

Acute effects on the central nervous system (CNS) are characterized by two sequential phases (i.e., excitation and depression), and are usually reversible. These symptoms generally prevail over those of other systems (Stewart et al., 1962; Chiesura, 1980; Governa, 1981).

In the early phase of excitation, euphoria and inebriation are present. The subsequent phase of CNS depression is characterized by various degrees of narcosis culminating in coma (Caccuri, 1976; Chiesura, 1980). Muscular hypotony, muscular spasms, reduced tendon reflexes, and loss of co-ordination may occur (Huff, 1971).

Changes in EEG patterns may vary considerably. The EEG may be normal in some cases, while substantial alterations, regressing after a few days, may be observed in others. The most frequently observed change is a decrease in alpha activity, which is often irregular. Widespread rapid rhythms or low theta activity may also be present (Chiesura, 1980).

Exposure to trichloroethylene at a concentration of 540 mg/m^3 (100 ppm) produces various results on the CNS. Though there may not be any evidence of effects on psychomotor capacities (Stewart et al., 1970), reductions in mental performance evidenced by the test for perception, past recall, answering speed, and response

and manual dexterity tests have been reported (Ferguson & Vernon, 1970). Visual and auditory evoked potentials were affected at exposure levels ranging between 270 and 540 mg/m^3 (50 and 100 ppm), for 3 1/2 - 7 1/2 h (Winneke, 1982).

The behavioural effects of exposure to trichloroethylene have been studied under laboratory and work-place conditions. Laboratory exposure to 540 or 1080 mg/m^3 (100 or 200 ppm), for 70 min, had no effect on reaction time or short-term memory (Gamberale et al., 1976). Volunteers exposed to 540, 1620, or 5400 mg/m^3 (100, 300, or 1000 ppm), for 2 h, showed significant impairment in a number of psychomotor tests at 5400 mg/m^3 but not at 1620 or 540 mg/m^3 (Vernon & Ferguson, 1969). In a further study (Ferguson & Vernon, 1970), effects were again found at 5400 mg/m^3 and marginal effects at 1620 mg/m^3. In another study, exposure of volunteers to 810 or 1620 mg/m^3 (150 or 300 ppm), for 2 1/2 h, did not produce any significant impairment in behavioural test results (Ettema & Zielhuis, 1975). With repeated exposure under work-place conditions, no behavioural effects were observed with mean atmospheric levels of 270 mg/m^3 (50 ppm) (Triebig et al., 1977a,b). In a study of complex reaction time in an occupational group with a mean exposure of 1320 mg/m^3 (245 ppm), reaction time was increased in comparison with that in a control group (Gun et al., 1978). Motor nerve conduction velocity was not affected in a group occupationally exposed to mean atmospheric levels of less than 270 mg/m^3 (50 ppm) (Triebig et al., 1982).

A study on 122 exposed workers (Ahlmark & Forssman, 1951a) showed that symptoms of toxicity in human beings were correlated with urinary levels of metabolic trichloroacetic acid. With urinary concentrations of up to 20 mg trichloroacetic acid/litre, no particular symptoms were noted. With urinary levels ranging between 45 and 75 mg/litre, headache, fatigue, increased need for sleep, irritability, and alcohol intolerance were reported in 50% of workers. When urinary levels of trichloroacetic acid exceeded 300 mg/litre, these symptoms were found in 100% of subjects. Initial nervous-system signs of toxicity were reported to be associated with the urinary excretion of trichloroacetic acid at 75 mg/litre and 30 mg/litre (Lazarev & Gadaskiva, 1977).

Two types of irreversible neurotoxic effects have been reported in man. One is the specific degeneration of the cranial nerves, in particular the trigeminal, the olfactory, and the facial nerves (Goldblatt, 1956; James, 1963; Kjellstrand et al., 1980; Barrett et al., 1982). However, it is considered that this effect is not produced by trichloroethylene itself but by its decomposition product, dichloroacetylene (Henschler et al., 1970a). In addition, polyneuropathies involving the whole peripheral nervous system have been described after long-term exposure to trichloroethylene (Szulc-Kuberska, 1972).

8.2.2. Effects on the cardiovascular system

Several cases of sudden death due to cardiac arrest have been reported in subjects exposed to trichloroethylene, either medically or occupationally (Ostelere, 1953; Norris & Stuart, 1957; Meyer, 1966; Wiecko, 1966; Clearfield, 1970; Tomasini, 1976). It has been suggested that these deaths might be due to an increased sensitivity of the myocardium to endogenous and exogenous catecholamines. The event would entail irregular cardiac rhythm due to the onset of ectopic foci with atrioventricular dissociation. A range of alterations in cardiovascular function (e.g., atrial and ventricular extrasystole, tachycardia, and ventricular fibrillation) has been reported (Bardodej & Viskocil, 1956; Anderson, 1957; Lilis et al., 1969; Tomasini, 1976; Governa, 1981).

8.2.3. Effects on the respiratory system

Under normal conditions of exposure, trichloroethylene did not induce any effects on the respiratory tract but, at high exposure levels of 810 - 3510 mg/m^3 (150 - 650 ppm), there may be effects due to local irritation (Bardodej & Viskocil, 1956; Jouglard & Vincent, 1971; Meyer, 1973; Gaultier et al., 1974; Tomasini, 1976).

Breakdown products are of more importance than trichloroethylene with respect to the effects on the respiratory system (Castellino, 1969).

8.2.4. Effects on the urinary tract

Altered renal function has been described in both acute and chronic trichloroethylene poisoning (Nomura, 1962; Graovac-Leposavic et al., 1964; Clearfield, 1970). The nephropathy observed could be due to impurities rather than to the trichloroethylene; technical grade trichloroethylene might have contained nephrotoxic chlorinated hydrocarbons (e.g., 1,1,2,2-tetrachloroethane) as impurities (Chiesura, 1980).

8.2.5. Effects on the skin

When applied to the skin, trichloroethylene causes erythema (Wahlberg, 1984). It is only mildly irritating to human skin providing it is not held in contact (e.g., by clothing or footwear). Chemical burning of the skin has been reported following prolonged contact (Maloof, 1949; Tomasini, 1976). There is a considerable difference in individual susceptibility to the effects of repeated exposure. The reaction to repeated contact (which will defat the skin) may take the form of an erythematous, exudative, vesicular, eczematous, or exfoliative dermatitis (Roche et al., 1958; Stopps & McLaughlin, 1967; Ettema et al., 1975). Secondary infection of the skin may complicate the dermatitis. "Degreaser's flush", a reddening of the skin in some individuals ingesting ethanol after exposure to trichloroethylene, can occur (section 8.1.1).

8.2.6. Effects on the eye

Liquid trichloroethylene in the eye produces pain and discomfort and superficial damage to the cornea, but complete recovery occurs within a few days (Grant, 1974). Exposure to high concentrations of vapour (anaesthetic levels of 27 500 - 108 000 mg/m^3) also causes eye irritation and superficial damage to the cornea, but again with complete recovery (Grant, 1961).

8.2.7. Carcinogenicity

A number of epidemiological studies have been conducted to examine the possible carcinogenic effects of trichloroethylene in occupationally-exposed populations, and the major findings are summarized in Tables 10 and 11. Some of these studies have followed groups with specified exposure to trichloroethylene (Axelson et al., 1978, 1984; Tola et al., 1980) (Table 10), whereas others have involved workers in laundry and dry cleaning with more or less mixed exposures including trichloroethylene (Blair et al., 1979; Malek et al., 1979; Katz & Jowett, 1981) (Table 11). Other studies are of a case-reporting or a case-referent (case-control) type, concerning pancreatic (Lin & Kessler, 1981) and liver tumours (Novotna et al., 1979; Paddle, 1983) or other conditions and, also, involving exposure to other solvents (Lin & Kessler, 1981). Some case-control studies of lymphomas, both of the Hodgkin and non-Hodgkin type (Olsson & Brandt, 1980; Hardell et al., 1981) and of liver cancer (Hernberg et al., 1984) have mentioned exposure to trichloroethylene as a factor in a few cases, but the numbers are too small for any conclusions to be drawn about trichloroethylene as a risk factor.

Table 10. Summary of epidemiological studies on carcinogenicity with specific reference to trichloroethylene exposure

Type of study/cohort	Remarks	Reference
Mortality in a cohort of 518 male workers (7688 person-years)	1. Exposure intensity had been monitored since 1950 in terms of urinary excretion of trichloroacetic acid (TCA). 2. No significant excess of cancer mortality either in the high-dose group (> 100 mg TCA/litre urine) or the low-dose group (< 100 mg TCA/litre urine).	Axelson et al. (1978)

	3. Results of first update, see Axelson (1984) below.	

Table 10. (contd.)

Type of study/cohort	Remarks	Reference
Mortality in a cohort of 1148 male workers and 969 female workers (4269 person-years for women)	1. Exposure information was based on TCA determinations in the urine, permitting latency requirements of 6 - 13 years. Loss to follow-up was 9%. 2. Trichloroethylene was not carcinogenic (given a latency period of 6 to 13 years) among those exposed at low levels (100 mg TCA/litre urine in 91% of the cohort). 3. Cohort to be updated at 5-year intervals.	Tola et al. (1980)
Mortality and incidence in a cohort of 1424 men	1. Extension and update of the first-mentioned study, 65% with exposure from 1970 to 1975 and with more than 90% having TCA levels < 100 mg/litre. 2. Deficit found in mortality from cancers (22 versus 36.9 expected), but a significant excess of urinary tract (11 versus 4.85) and haematolymphatic tumours (5 versus 1.20). 3. Specifically, there were 3 urinary bladder cancers versus 0.83 expected, 4 cancers of the prostrate versus 2.35 expected and 2 lymphomas versus 0.27 expected, requiring 2 years or more of exposure and 10 years of latency.	Axelson et al. (1984)

Table 11. Summary of epidemiological studies and case reports on carcinogenicity of mixed exposures involving trichloroethylene or trichloroethylene production

Type of study/cohort	Remarks	Reference
Cohort of 330 laundry and dry-cleaning workers	1. Significant increase in lung and cervical cancer, and a slight excess of leukaemia and liver cancer detected. 2. Exposed also to other chemicals such as benzene and carbon tetrachloride. 3. To be extended to a prospective study of 2 - 5 x 10^3 workers.	Blair et al. (1979)
Cohort of 37 male dry-cleaning workers	1. Exposure intensity was such that TCA in urine was less than 100 mg/litre in over 60% of the cohort. 2. 6 cases of cancer, none of which were in the liver.	Malek et al. (1979)

Table 11. (contd.)

Type of study/cohort	Remarks	Reference
Cohort of 671 female laundry/dry-cleaning workers (controls: entire female working population and other low-wage occupations)	1. Death certificates were employed. 2. Elevated risks of genital (unspecified) and kidney cancers were detected, along with a smaller excess of bladder and skin cancer and lymphoma.	Katz & Jowett (1981)
109 pancreatic cancer patients, case-control study	1. Hospital records were employed. 2. An association was observed between	Lin & Kessler (1981)

	pancreatic cancer and exposure to dry-cleaning chemicals and gasoline fumes, as well as to drinking of decaffeinated (by means of trichloroethylene) coffee.	
	3. No information was available on extent or type of exposure.	
95 liver cancers, case-study	1. Cancer registry data on liver cancer in the area of a production plant were employed.	Paddle (1983)
	2. No case could be definitely tied to trichloroethylene production.	

While some of these studies are continuing as prospective studies to cover larger populations, they suffer, in general, from one or more of the following factors: (a) small cohort population, (b) young age of the cohort, (c) a rather short follow-up period, (d) relatively few cases, (e) inaccurate or uncertain estimation of intensity and duration of exposure to trichloroethylene, (f) possible exposure to other chemicals, even in the more specific studies, including potential carcinogens, and (g) inadequate information on other general risk factors for cancer, such as smoking, even if the influence of uncontrolled confounding is likely to be limited in this respect.

At present, it is not possible to draw definite conclusions from the existing epidemiological data on the carcinogenic potential of trichloroethylene. However, the appearance of an excess of haematolymphatic malignancies in several of the studies, possibly associated with trichloroethylene exposure (Blair et al., 1979; Hardell et al., 1981; Katz & Jowett, 1981; Axelson et al., 1984) deserves attention. Furthermore, genito-urinary tumours were over-represented in 3 studies (Blair et al., 1979; Katz & Jowett, 1981; Axelson et al., 1984), though the picture is less consistent with regard to the specific sites involved (bladder, kidney, prostrate, and cervix).

It was noted that IARC (1979, 1982) concluded that epidemiological studies on human beings were inadequate to make an evaluation.

9. EVALUATION OF THE HEALTH RISKS FOR MAN

9.1. Levels of Exposure

9.1.1. General population

The general population is exposed to very low levels of trichloroethylene in air, water, and food (Table 12). The move away from the use of trichloroethylene in anaesthesia, the solvent extraction and fumigation of foodstuffs, and the dry-cleaning of textiles has reduced exposure from these sources. Trichloroethylene does not accumulate significantly in the food chain. It is degraded by both biotic and abiotic processes and its persistence in various environmental compartments is relatively short, of the order of days or months rather than years. In ground water, the absence of actinic radiation means that the rate of degradation is slower, and trichloroethylene contamination may be relatively prolonged (e.g., half-life of 2 1/2 years).

Table 12. Maximum observed concentrations of trichloroethylene in environmental media

Media	Maximum observed concentration
Open water reservoir	220 µg/litre
Industrial discharge water	200 µg/litre
Rain water	~ 1 µg/litre
Atmosphere	~ 40 µg/m^3
Dairy foods	10 µg/kg

Meat	22 µg/kg
Fats and oil	19 µg/kg

Accidental or suicidal ingestion by adults occurs, but is influenced by the limited availability of trichloroethylene for domestic use in many countries.

9.1.2. Occupational exposure

Exposure during the actual production of trichloroethylene is relatively low because of the nature of the process. Subsequent uses in, for example, metal degreasing and the dry-cleaning of textiles, can involve higher exposures. The respiratory route is the principal route of exposure with dermal exposure as an additional route. Oral intake is insignificant in occupational terms.

9.2. Evaluation of Human Health Risks

9.2.1. Acute effects

The predominant effect of trichloroethylene in human beings is on the central nervous system; liver and kidney damage can also occur. Central nervous system depression can result in coma and death. A lethal dose as low as 50 ml (75 g) has been reported but, in general, the lethal dose for an adult is approximately 7000 mg/kg body weight. The threshold for demonstrable behavioural effects, resulting from inhalation exposure, is approximately 1350 mg/m^3 (250 ppm); less well-defined subjective complaints have been reported with exposure to concentrations of 270 - 540 mg/m^3 (50 - 100 ppm) for a few hours.

Cardiac arrhythmia has been reported during anaesthesia involving trichloroethylene at inhaled concentrations of 27 000 - 54 000 mg/m^3 (5000 - 10 000 ppm) (blood levels 60 - 120 mg/litre).

It should be noted that ethanol potentiates the central nervous system effects of trichloroethylene. Exposure to trichloroethylene at 1080 mg/m^3 (200 ppm) with blood ethanol levels of 300 - 450 mg/litre produces demonstrable behavioural effects.

Irreversible neurotoxic effects on the trigeminal nerve have been described in anaesthesia with trichloroethylene, using a closed-circuit system with soda lime. This effect is attributed to the generation of dichloroacetylene and not to trichloroethylene.

9.2.2. Chronic effects

The main chronic effects of trichloroethylene in human beings are disturbances in the central nervous system, as well as effects on the kidney and liver.

Nervous system symptoms (headache, fatigue, irritability, and alcohol intolerance) begin to occur when levels of trichloroacetic acid in urine are about 30 - 75 mg/litre. No symptoms have been reported at concentrations of 20 mg/litre in urine, which corresponds to a concentration of trichloroethylene in air of about 50 mg/m^3.

For effects on other organs, there is no clear-cut quantitative information on dose response. There are insufficient data on possible effects on human chromosomes.

Epidemiological data on the carcinogenicity of trichloroethylene are inconclusive, but a slight excess of genito-urinary cancers and lymphomas in some studies on dry cleaners and metal degreasers deserves further attention.

The results of long-term exposure studies on rodents have shown that the liver and kidney are the critical organs. Cytomegaly and karyomegaly of renal tubular cells was observed in male rats

exposed to pure trichloroethylene at 1620 mg/m^3 (300 ppm), for 7 h per day, 5 days per week, for 104 weeks, but not at 540 mg/m^3 (100 ppm).

No embryotoxic or teratogenic effects have been observed in mice and rats with inhalation of trichloroethylene at levels of 1620 mg/m^3 (300 ppm).

Data on the genetic effects of trichloroethylene are inconclusive.

Lifetime exposure of mice to trichloroethylene at 1620 mg/m^3 (300 ppm) through inhalation or to oral doses of 700 - 1200 mg/kg body weight per day produced lung and liver tumours. A low incidence of renal tumours was found in rats exposed to a trichloroethylene concentration of 3240 mg/m^3 (600 ppm) by inhalation or to 500 - 1000 mg/kg body weight per day by gavage.

A dose-related increase in Leydig (interstitial) cell tumours of the testis was observed in one study on male Sprague Dawley rats following long-term inhalation exposure to trichloroethylene concentrations of > 540 mg/m^3 (> 100 ppm).

Thus, there is clear evidence that trichloroethylene is carcinogenic in mice. There is also some evidence that trichloroethylene causes tumours in rats. The significance of these findings needs to be evaluated in the context of further studies on the mechanism of action of trichloroethylene.

9.3. Treatment of Poisoning in Human Beings

9.3.1. Emergency measures

9.3.1.1. General points

In the treatment of trichloroethylene poisoning, pressor amines should not be used because of the risk of producing arrhythmias in the trichloroethylene-sensitized myocardium (Bozza Marrubini et al., 1978). Hypotension may be treated by transfusion. If necessary, anti-arrhythmic and beta-blocking agents can be administered. Haemodialysis, haemoperfusion, or plasmapheresis have been reported to be useful (Zaffiri et al., 1968; Roessler & Morawiec-Borowiac, 1973; Malizia et al., 1984). Medical advice should always be obtained in cases of over-exposure to, or frank poisoning by, trichloroethylene.

9.3.1.2. Ingestion

Vomiting should not be induced, because of the danger of aspiration into the larynx and lungs and consequent risks of vagal inhibition or chemical pneumonitis. Gastric lavage can be effective if performed within 4 h. Adsorbents such as activated charcoal or liquid paraffin (medicinal grade) reduce intestinal absorption; saline laxatives will speed elimination (Bothe et al., 1973). If the patient is in a state of stupor or coma, intubation must be performed before gastric lavage.

9.3.1.3. Inhalation

The victim should be removed from the polluted area and placed in a semi-prone position ensuring that the airway is clear. If the victim is in a state of stupor or coma, oxygen should be administered. If spontaneous respiration is absent or very weak, artifical respiration should be applied.

9.3.1.4. Dermal exposure

All contaminated clothing should be removed and the affected parts of the body washed thoroughly with soap and plenty of water.

9.3.1.5. Eye exposure

The eyes should be thoroughly irrigated with water for at least 15 min, and ophthalmological advice obtained on the possible need for further treatment.

REFERENCES

ABRAHAMSEN, A.M. (1960) Quantitative estimation of trichloroacetic acid in the urine and serum in trichloroethylene poisoning. *Acta pharmacol. toxicol.*, 17: 288-294.

ACGIH (1982) TLVs - *Threshold limit values for chemical substances in workroom air adopted by ACGIH for 1982*, Cincinnati, Ohio, American Conference of Governmental Industrial Hygienists, p. 32.

ADAMS, E.M., SPENCER, H.C., ROWE, V.K., MCCOLLISTER, D.D., & IRISH, D.D. (1951) Vapor toxicity of trichloroethylene determined by experiments on laboratory animals. *Arch. ind. Hyg. occup. Med.*, 4: 469-481.

AHLMARK, A. & FORSSMAN, S. (1951a) Evaluating trichloroethylene exposures by urinalyses for trichloroacetic acid. *Arch. ind. Hyg. occup. Med.*, 3: 386-398.

AHLMARK, A. & FORSSMAN, S. (1951b) The effect of trichloroethylene on the organism. *Acta physiol. scand.*, 22: 326-339.

ALEXANDER, H.C., MCCARTHY, W.M., & BARTLETT, E.A. (1978) Toxicity of perchloroethylene, trichloroethylene, 1,1,1-trichloroethane, and methylene chloride to fathead minnows. *Bull. environ. Contam. Toxicol.*, 20: 344-352.

ANDERSEN, M.E., GARGAS, M.L., JONES, R.A., & JENKINS, L.J., Jr (1980) Determination of the kinetic constants for metabolism of inhaled toxicants *in vivo* using gas uptake measurements. *Toxicol. appl. Pharmacol.*, 54(1): 100-116.

ANDERSSON, A. (1957) [Health hazards in industry on exposure to trichloroethylene: a clinical, experimental, and technical hygienic investigation.] *Acta med. scand.*, 157(Suppl. 323): 1-220 (in German).

ASCHIMICI (1980) *Report for Italian Working Group for trichloroethylene.*

ASTRAND, I. & OVRUM, P. (1976) Exposure to trichloroethylene. I. Uptake and distribution in man. *Scand. J. Work Environ. Health*, 2: 199-211.

AVIADO, D.M., ZAKHARI, S., SIMAAN, J.A., & ULSAMER, A.G. (1976) In: Golberg, L., ed. *Methyl chloroform and trichloroethylene in the environment*, Cleveland, Ohio, CRC Press Inc., pp. 47-89.

AXELSON, O., ANDERSSON, K., HOGSTEDT, C., HOLMBERG, B., MOLINA, G., & DE VERDIER, A. (1978) A cohort study on trichloroethylene exposure and cancer mortality. *J. occup. Med.*, 20: 194-196.

AXELSON, O., ANDERSSON, K., SELDEN, A., & HOGSTEDT, C. (1984) Cancer morbidity and exposure to trichloroethylene. In: *ICOST, International Conference on Organic Solvent Toxicity*, Stockholm, 15-17 November 1984 (Abstract in: *Arbete och Halsa*, 29: 126).

BADEN, J.M., KELLEY, M., MAZZE, R.I., & SIMMON, V.F. (1979) Mutagenicity of inhalation anaesthetics: trichloroethylene, divinyl ether, nitrous oxide and cyclopropane. *J. Anaesth., 51: 417.*

BAERG, R.D. & KIMBERG, D.V. (1970) Centrilobular hepatic necrosis and acute renal failure in "solvent sniffers". *Ann. intern. Med.*, 73: 713-720.

BALKON, J. & LEARY, J.A. (1979) An initial report on a comprehensive, quantitative, screening procedure for volatile compounds of forensic and environmental interest in human biofluids by GC/MS. *J. anal. Toxicol.*, 3: 213-215.

BANERJEE, S. & VAN DUUREN, B.L. (1978) Covalent binding of the carcinogen trichloroethylene to hepatic microsomal proteins and to endogenous DNA *in vitro*. *Cancer Res.*, 38: 776-780.

BANERJEE, S., YALKOWSKY, S.H., & VALVANI, S.C. (1980) Water solubility and octanol/water partition coefficients of organics. Limitations of the solubility-partition coefficient correlation. *Environ. Sci. Technol.*, 14: 1227-1229.

BARDODEJ, Z. (1962) [Value and use of exposure test. VIII. Volatile organic chloride test in urine.] *Cesk. Hyg.*, 7: 234-239 (in Czech).

BARDODEJ, Z. & VISKOCIL, J. (1956) The problem of trichloroethylene metabolism and its effects on the nervous system as a means of hygienic control. *Am. Med. Assoc. Arch. ind. Health*, 13: 581-592.

BARKLEY, J., BUNCH, J., BURSEY, J.T., CASTILLO, N., COOPER, S.D., DAVIS, J.M., ERICKSON, M.D., HARRIS (III), B.S.H., KIRKPATRICK, M., MICHAEL, L.C., PARKS, S.P., PELLIZZARI, E.D., RAY, M., SMITH, D., TOMER, K.B., WAGNER, R., & ZWEIDINGER, R.A. (1980) Gas chromatography mass spectrometry computer analysis of volatile halogenated hydrocarbons in man and his environment: a multimedia environmental study. *Biomed. mass Spectrom.*, 7: 139-147.

BARRETT, L., ARSAC, PH., VINCENT, M., FAURE, J., GARREL, S., & REYMOND, F. (1982) Evoked trigeminal nerve potential in chronic trichloroethylene intoxication. *J. Toxicol. clin. Toxicol.*, 19: 419-423.

BARROWS, M.E., PETROCELLI, S.R., MACEK, K.J., & CARROLL, J.J. (1980) Bioconcentration and elimination of selected water pollutants by bluegill sunfish *(Lepomis macrochirus)*. In: Hague, R., ed. *Dynamics, Exposure and Hazard Assessment of Toxic Chemicals*, Ann Arbor, Michigan, Ann Arbor Science Publishers, pp. 379-392.

BARSOUM, G.S. & SAAD, K. (1934) Relative toxicity of certain chlorine derivatives of the aliphatic series. *Q. J. Pharm. Pharmacol.*, 7: 205-214.

BARTONICEK, V. (1962) Metabolism and excretion of trichloroethylene after inhalation by human subjects. *Br. J. ind. Med., 19: 134-141.*

BARTONICEK, V. & BRUN, A. (1970) Subacute and chronic trichloroethylene poisoning: a neuropathological study in rabbits. *Acta pharmacol. toxicol.*, 28: 359-369.

BARTONICEK, V. & SOUCEK, B. (1959) Metabolism of trichloroethylene in rabbits. *Arch. Gewerbepathol. Gewerbehyg.*, 17: 283-293.

BARTSCH, H., MALAVEILLE, C., BARBIN, A., & PLANCHE, G. (1979) Mutagenic and alkylating metabolites of haloethylenes, chlorobutadienes and dichlorobutenes produced by rodent or human liver tissues. *Arch. Toxicol.*, 41: 249-277.

BATTIG, K. (1964) Comparison of the effects of trichloroethylene with the effects of drugs on exploratory behaviour in the rat. In: *14th International Congress on Occupational Health, Madrid, 1963*, Vol. 2, pp. 887-889.

BATTIG, K. & GRANDJEAN, E. (1963) Chronic effects of trichloroethylene on rat behaviour. *Arch. environ. Health*, 7: 694-699.

BAUER, U. (1981) [Human exposure to environmental chemicals. Investigations on volatile organic halogenated compounds in water, air, food, and human tissues.] *Zbl. Bakt. I. Abt. Orig.*, B174: 200-237 (in German).

BAUER, U. & SELENKA, F. (1982) [Exposure of the population to haloforms and chlorinated solvents in drinking water compared to air and food.] *Von Wasser*, 59: 7-16 (in German).

BEISLAND, H.O. & WANNAG, S.A. (1970) [Trichloroethylene sniffing:

acute liver and kidney damage.] *Tidsskr. Nor. Laegeforen.*, 90: 285-288 (in Norwegian).

BELILES, R.P., BRUSICK, D.J., & MECLER, F.J. (1980) *Teratogenic-mutagenic risk of workplace contaminants: trichloroethylene, perchloroethylene, and carbon disulfide*, Washington DC, US Department of Health, Education and Welfare, pp. 234 (Report for US DHEW Contract No. 210-77-0047) (Govt Rep. Announce. Index (US), 1982: 2728).

BELL, Z. (1977) *Written communication with contractor reports of: (a) Dominant lethal study with trichloroethylene in albino rats exposed via inhalation (February 1977); and (b) Teratogenic study via inhalation with trichlor 132, trichloroethylene in albino rats (March 1977)*, Washington DC, US Environmental Protection Agency (EPA-600/8-82-006).

BELLAR, T.A., BUDDE, W.L., & EICHELBERGER, J.S. (1979) The identification and measurement of volatile organic compounds in aqueous environmental samples. IV. In: Schuetzle, D., ed. *Monitoring toxic substances*, Washington DC, American Chemical Society (ACS Symposium Series No. 94).

BERGMAN, K. (1983) Application and results of whole-body autoradiography in distribution studies of organic solvents. *Crit. Rev. Toxicol.*, 12(1): 59-118.

BIANCHI, A., DE NATALE, G., & MATTURRO, F. (1963) [Comparative research on some pharmacological effects of fluothane and other volatile narcotics.] *G. Ital. Chir.*, 19: 327-349 (in Italian).

BIGGS, D.C., ROWLAND, R.G., & WURSTER, C.F. (1979) Effects of trichloroethylene, hexachlorobenzene, and polychlorinated biphenyls on the growth and cell size of marine phytoplankton. *Bull. environ. Contam. Toxicol.*, 21: 196-201.

BIGNAMI, M., CONTI, G., CONTI, L., CREBELLI, R., MISURACA, F., PUGLIA, A.M., RANDAZZO, R., SCIANDRELLO, G., & CARERE, A. (1980) Mutagenicity of halogenated aliphatic hydrocarbons in *Salmonella typhimurium*, *Streptomyces coelicolor*, and *Aspergillus nidulans*. *Chem.-biol. Interact.*, 30: 9-23.

BLAIR, A., DECOUFLE, P., & GRAUMAN, D. (1979) Causes of death among laundry and dry cleaning workers. *Am. J. public Health*, 69: 508-511.

BOLT, H.M. & FILSER, J.G. (1977) Irreversible binding of chlorinated ethylenes to macromolecules. *Environ. Health Perspect.*, 21: 107-112.

BOTHE, J., BRAUN, W., & DONHARDT, A. (1973) [Effect of mineral oil in hydrocarbon poisoning in mice.] *Arch. Toxikol.*, 30: 243-250 (in German).

BOUDENE, C., TURBIAUX, M., CLUET, J.-L., & TRUHAUT, R. (1983) Contribution à la fixation d'une concentration limite tolérable de trichloroéthylène dans l'atmosphère des locaux de travail. *Arch. Mal. prof. Méd. Trav. Sécur. soc.*, 44(2): 75-91.

BOUWER, E.J. & MCCARTY, P.L. (1983) Transformations of 1- and 2-carbon halogenated aliphatic organic compounds under methanogenic conditions. *Appl. environ. Microbiol.*, 45: 1286-1294.

BOUYGUES, M., DANNE, O., BOUVRY, M., LUCIANI, F., & RABREAU, D. (1980) Hépatite au trichloroéthylène. Action synergique du tétrachlorure de carbone? *Nouv. Presse méd.*, 9(43): 3277.

BOZZA MARRUBINI, M., GHEZZI LAURENZI, R., & UCCELLI, P. (1978) *[Acute intoxication: clinical diagnosis and treatment,] Milan*, Italy, Medico-Farmaceutica Organization Editore, p. 470 (in Italian).

BRINGMANN, G. & KUHN, R. (1980) Comparison of the toxicity thresholds of water pollutants to bacteria, algae, and protozoa in

the cell multiplication inhibition test. *Water Res.*, 14: 231-241.

BRONZETTI, G., ZEIGER, E., & FREZZA, D. (1978) Genetic activity of trichloroethylene in yeast. *J. environ. Pathol. Toxicol.*, 1: 411-418.

BROSS, G., DI FRANCEISCO, D., & DESMOND, M.E. (1983) The effects of low dosages of trichloroethylene on chick development. *Toxicology*, 28: 283-294.

BUCHET, J.-P., LAUWERYS, R., ROELS, H., DEFELD, J.M., & BAUER, H. (1974) Le dosage par chromatographie en phase gazeuse des métabolites urinaires du trichloréthylène: l'acide trichloro-acétique et le trichloroéthanol. *Arch. Mal. prof. Méd. Trav. Sécur. soc.*, 35(3): 395-402.

CABANA, B.E. & GESSNER, P.K. (1967) Determination of chloral hydrate, trichloroacetic acid, trichlorethanol, and urochloralic acid in the presence of each other and in tissue homogenates. *Anal. Chem.*, 39: 1449-1452.

CACCURI, S. (1976) *[Labour medicine,]* 3rd ed., Napoli, Italy, Idelson, pp. 343-352 (in Italian).

CALLEN, D.F., ROLAND WOLF, C., & PHILPOT, R.M. (1980) Cytochrome P-450 mediated genetic activity and cytotoxicity of seven halogenated aliphatic hydrocarbons in *Saccharomyces cerevisiae*. *Mutat. Res.*, 77: 55-63.

CAPELLINI, A. & GRISLER, R. (1958) [Studies on liver function in a group of workers chronically exposed to trichloroethylene.] *Med. Lav.*, 49: 167-172 (in Italian).

CARLSON, G.P. & WHITE, J.F. (1983) Cardiac arrythmogenic action of benzo(a)pyrene in the rabbit. *Toxicol. Lett.*, 15: 43-48.

CASTELLINO, N. (1969) *[Trichloroethylene: technology, pathological manifestations and prevention,]* Milano, Italy, Franco Angeli Editore, p. 117 (in Italian).

CAVALLO, A. & GRASSI, P. (1976) Determination of chlorinated hydrocarbons in potable waters. *Boll. chim. Union Ital. Lab. prov.*, 27: 337-350.

CEFIC (1982) *Chlorinated solvents: trichloroethylene in metal cleaning and other industrial applications*, Brussels, Conseil Européen des Fédérations des Industries Chimiques.

CERNA, M. & KJPENOVA, H. (1977) Mutagenic activity of chloroethylenes analyzed by screening system tests. *Mutat. Res.*, 46: 214-215.

CHIESURA, P. (1980) [Halogenated aliphatic hydrocarbons.] In: Crepet, M., ed. *[Occupational medicine,]* Torino, Italy, Utet (in Italian).

CHIESURA, P. & CORSI, G. (1961) [Acute trichloroethylene poisoning in man followed by liver disease and hyperglycaemic glycosuria.] *Folia med.*, 44: 121-135 (in Italian).

CHRISTENSEN, H.E., LUGINBYHL, T.T., & CARROLL, B.B., ed. (1974) *The toxic substances list 1974*, Washington DC, National Institute of Occupational Safety and Health, p. 353.

CHRISTENSEN, H.E. & LUGINBYHL, T.T. (1975) *Registry of toxic effects of chemical substances 1975*, Washington DC, US Department of Health, Education and Welfare.

CLEARFIELD, H.R. (1970) Hepatorenal toxicity from sniffing spot-remover (trichloroethylene): Report of 2 cases. *Am. J. dig. Dis.*, 15: 851-856.

COLEMAN, W.E., LINGG, R.D., MELTON, R.G., & KOPFLER, F.C. (1976) The occurrence of volatile organics in five drinking water supplies using gas chromatography/mass spectrometry. Identification and

analysis of organisms in polluted water. In: Keith, L.H., ed. *Proceedings of the First Chemical Congress of the North American Continent, 1975*, Ann Arbor, Michigan, Ann Arbor Science, pp. 305-327.

CONSO, F., EFTHYMIOU, M.-L., GARNIER, R., & FOURNIER; E. (1980) Interêt de la toxicovigilance: example de certains trichloréthylènes du commerce. *Arch. Mal. prof. Méd. Trav.*, 41: 198-200.

CORNISH, H.H. & ADEFUIN, J. (1966) Ethanol potentiation of halogenated aliphatic solvent toxicity. *Am. Ind. Hyg. Assoc. J.*, 27: 57-61.

CORREIA, Y., MARTENS, G.J., VAN MENSCH, F.H., & WHIM, B.P. (1977) The occurrence of trichloroethylene, tetrachloroethylene and 1,1,1-trichloroethane in Western Europe in air and water. *Atmos. Environ.*, 11: 1113-1116.

CREBELLI, R., BIGNAMI, M., CONTI, L., & CARERE, A. (1982) Mutagenicity of trichloroethylene in *Salmonella typhimurium* TA 100. *Ann. Ist. Super. Sanità*, 18: 117-122.

CREBELLI, R., CONTI, G., CONTI, L., & CARERE, A. (in press) Mutagenicity of trichloroethylene, trichloroethanol, and chloral hydrate in *Aspergillus nidulans*. *Mutat. Res.*

CRONN, D.R., RASMUSSEN, R.A., ROBINSON, E., & HARSCH, D.E. (1977) Halogenated compound identification and measurement in the troposphere and lower stratosphere. *J. geophys. Res.*, 82: 5935-5944.

DALBEY, W. & BINGHAM, E. (1978) Metabolism of trichloroethylene by the isolated perfused lung. *Toxicol. appl. Pharmacol.*, 43: 267-277.

DANIEL, J.W. (1963) The metabolism of ^{36}Cl-labelled trichloroethylene and tetrachloroethylene in the rat. *Biochem. Pharmacol.*, 12: 795-802.

DEFALQUE, R.J. (1961) Pharmacology and toxicology of trichloroethylene: A critical review of the world literature. *Clin. Pharmacol. Ther.*, 2: 665-688.

DEGUCHI, T. (1972) Threshold limit values for solvent mixtures in the air. Effects of single and mixed chlorinated hydrocarbons upon the level of serum transaminases in rats. *Osaka Shiritsu Daigaku Igaku Zasshi*, 21: 187-209.

DEKANT, W. & HENSCHLER, D. (1982) Dechlorination reactions in the course of the metabolism of trichloroethylene. *Naunyn-Schmiedeb Arch. Pharmacol.*, 319: R16.

DEKANT, W. & HENSCHLER, D. (1983) New pathways of trichloroethylene metabolism. *Dev. Toxicol. environ. Sci.*, 11: 399-402.

DEKANT, W., METZLER, M., & HENSCHLER, D. (1984) Novel metabolites of trichloroethylene through dechlorination reactions in rats, mice, and humans. *Biochem. Pharmacol., 33(13): 2021-2027.*

DE LEON, I.R., MABERRY, M.A., OVERTON, E.B., RASCHKE, C.K., REMELE, P.C., STEELE, C.F., WARREN, V.L., & LASETER, J.L. (1980) Rapid gas chromatographic method for the determination of volatile and semivolatile organochlorine compounds in soil and chemical waste disposal site samples. *J. chromatogr. Sci., 18: 85-88.*

DE MORE, W.B., HAMSON, R.F., MOLINA, M.J., KURYLO, M.J., WATSON, R.T., HOWARD, C.J., GOLDEN, D.M., & RAVISHANKARA, A.R. (1983) *Chemical kinetics and photochemical data for use in stratospheric modeling evaluation number 6*, Washington DC, National Aeronautics and Space Administration (NASA JPL Publication No. 83-62, 15 September, 1983).

DERWENT, R.G. & EGGLETON, A.E.J. (1978) Halocarbon lifetimes and concentration distributions calculated using a two-dimensional tropospheric model. *Atmos. Environ.*, 12: 1261-1268.

DESOILLE, H., PINCHON, R.A., JANS, M., & BOUGUIGNON, A. (1962) Intoxication expérimentale aiguë par le trichloroéthylène. Effets aggravants de l'intoxication éthylique chronique associée. Etude expérimentale électroencéphalographique chez le lapin. *Arch. Mal. prof. Méd. Trav. Sécur. soc.*, 23: 653-664.

DIETZ, E.A., Jr & SINGLEY, K.F. (1979) Determination of chlorinated hydrocarbons in water by headspace gas chromatography. *Anal. Chem.*, 51: 1809-1814.

DILLING, W.L. (1977) Interphase transfer processes. II. Evaporation rates of chloromethanes, ethanes, ethylenes, propanes, and propylenes from dilute aqueous solutions. Comparisons with theoretical predictions. *Environ. Sci. Technol.*, 11: 405.

DI PAOLO, J.A. & DONIGER, J. (1982) Neoplastic transformation of Syrian hamster cells by putative epoxide metabolites of commercially-utilized chloroalkenes. *J. Natl Cancer Inst.*, 69(2): 531-534.

DI RENZO, A.B., GANDOLFI, A.J., & SPIES, I.G. (1982) Microsomal bioactivation and covalent binding of aliphatic halides to DNA. *Toxicol. Lett.*, 11: 243-252.

DMS (1967) *UV atlas of organic compounds*, London, Dortmund, Butterworths-Verlag Chemie, Vol. 3 (Spectrum No. A1/6).

DOBINSON, B. & GREEN, G.E. (1972) Formation of dichloroacetylene from trichloroethylene in the presence of epoxides. *Chem. Ind.*, 5: 214.

DORFMUELLER, M.A., HENNE, S.P., YORK, R.G., BORNSCHEIN, R.L., & MANSON, J.M. (1979) Evaluation of teratogenicity and behavioural toxicity with inhalation exposure of maternal rats to trichloroethylene. *Toxicology*, 14: 153-166.

DOWTY, B.J., CARLISLE, D.R., & LASETER, J.L. (1975) New Orleans drinking water sources tested by gas chromatography-mass spectrometry. *Environ. Sci. Technol.*, 9: 762-765.

DROZ, P.O. & FERNANDEZ, J.G. (1977) Solubility of organic solvents. I. Gas chromatographic determination of olive oil/gas partition coefficient. *Helv. Chim. Acta*, 60: 454.

DROZ, P.O. & FERNANDEZ, J.G. (1978) Trichloroethylene exposure. Biological monitoring by breath and urine analyses. *Br. J. ind. Med.*, 35: 35.

DUPRAT, P. & GRADISKI, D. (1980) Cytogenic effect of trichloroethylene in the mouse as evaluated in the micronucleus test. *IRCS med. Sci. soc. occup. Med.*, 8: 182.

DUPRAT, P., DELSAUT, L., & GRADISKI, D. (1976) Pouvoir irritant des principaux solvants chlorés aliphatiques sur la peau et les muqueuses oculaires du lapin. *Eur. J. Toxicol.*, 9(3): 171-177.

EAJ (1983) *Environmental monitoring of chemicals: environmental survey report of F.Y. 1980 and 1981*, Japan, Office of Health Studies, Department of Environmental Health, Environmental Agency Japan, p. 144.

EHRNER-SAMUEL, H., BALMER, K., & THORSELL, W. (1973) Determination of trichloroacetic acid in urine by a gas chromatographic method. *Am. Ind. Hyg. Assoc. J.*, 34: 93-96.

EKLUND, G., JOSEFFSON, B., & ROOS, C. (1978) Determination of volatile halogenated hydrocarbons in tapwater. *J. High Res. Chrom. & Chrom. Comms.*, July, pp. 34-40.

ELCOMBE, C.R. (in press) Species differences in carcinogenicity and peroxisome proliferation due to trichloroethylene: a biochemical human hazard assessment. *Arch. Toxicol.*

ELCOMBE, C.R., PRATT, I.S., & GREEN, T. (1982) The rate of

trichloroacetic acid (TCA) formation determines two species differences in hepatic peroxisome proliferation due to trichloroethylene (TRI). *Toxicologist*, 2: 173.

ELOVAARA, E., HEMMINKI, K., & VAINIO, H. (1979) Effects of methylene chloride, trichloroethane, trichloroethylene, tetrachloroethylene, and toluene on the development of chick embryos. *Toxicology*, 12: 111-119.

ENTZ, R.C. & HOLLIFIELD, H.C. (1982) Headspace gas chromatographic analysis of foods for volatile hydrocarbons. *J. agric. food Chem.*, 30: 84-88.

ERTLE, T., HENSCHLER, D., JULLER, G., & SPASSOWSKI, M. (1972) Metabolism of trichloroethylene in man. *Arch. Toxikol.*, 29: 171-188.

ETTEMA, J.J., KLEEREKOPER, L., & DUBA, W.C. (1975) Mental stresses during short-term inhalation of trichloroethylene. *Staub-Reinhalt. Luft*, 35: 409-410.

EUROCOP-COST (1976) *A comprehensive list of polluting substances which have been identified in various fresh waters, effluent discharges, aquatic animals and plants, and bottom sediments*, 2nd ed., Luxembourg, Commission of the European Community, p. 57 (EUCO/MDU/73/76, XII/476/76).

EWING, B.B., CHIAN, E.S.K., COOK, J.C., EVANS, C.A., & HOPKE, P.K. (1977) *Monitoring to detect previously unrecognized pollutants in surface waters - appendix: organic analysis data*, Springfield, Virginia, US NTIS, pp. 304 (PB Report PB-273350) (Govt Rep. Announce. Index, 1978: 199).

FABRE, R. & TRUHAUT, R. (1952) Toxicology of trichloroethylene. II. Results of experimental animal studies. *Br. J. ind. Med.*, 9: 39-43.

FAHRIG, R. (1977) The mammalian spot test (Fellfleckenstest) with mice. *Arch. Toxicol.*, 38: 87-98.

FAWNS, H.T. (1968) Trichloroacetic acid in the urine of workers using trichloroethylene. *Proc. Assoc. Clin. Biochem.*, *5: 110-114*.

FERGUSON, R.K. & VERNON, R.J. (1970) Trichloroethylene in combination with CNS drugs: effects on visual-motor tests. *Arch. environ. Health*, 20: 462-467.

FERNANDEZ, J.G., HUMBERT, B.E., DROZ, P.O., & CAPEROS, J.R. (1975) Exposition au trichloroéthylene. Bilan de l'absorption, de l'excrétion, et du métabolisme sur des sujets humains. *Arch. Mal. prof. Méd. Trav. Sécur. soc.*, 36(7-8): 397-407.

FERNANDEZ, J.G., DROZ, P.O., HUMBERT, B.E., & CAPEROS, J.R. (1977) Trichloroethylene exposure: simulation of uptake, excretion, and metabolism using a mathematical model. *Br. J. ind. Med.*, 34: 43-55.

FILSER, J.G. & BOLT, H.M. (1979) Pharmacokinetics of halogenated ethylenes in rats. *Arch. Toxicol.*, 42: 123-136.

FINK, R. (1968) *Toxicity of anaesthetics*, Baltimore, Maryland, Williams & Williams, pp. 270.

FISHBEIN, L. (1973) *Chromatography of environmental hazards*, New York, Elsevier Scientific Publishing Company, Vol. 2, pp. 471-490.

FOOD CHEMICAL NEWS (1978) Preliminary report on TCE study shows no carcinogenicity in rats. *Food chem. News*, 20: 41-42.

FOSSA, A.A., WHITE, J.F., & CARLSON, G.P. (1982) Anti-arrhythmic effects of disulfiram on epinephrine-induced cardiac arrthythmias in rabbits exposed to trichloroethylene. *Toxicol. appl. Pharmacol.*, 66: 109-117.

FRIBERG, G.L., KYLIN, B., & NYSTROM, A. (1953) Toxicities of trichloroethylene and tetrachloroethylene and Fujiwara's pyridine

reaction. *Acta pharmacol. toxicol.*, 9: 303-312.

FUKUDA, K., TAKEMOTO, K., & TSURATA, H. (1983) Inhalation carcinogenicity of trichloroethylene in mice and rats. *Ind. Health*, 21: 243-245.

GAMBERALE, F., ANNWALL, G., & OLSON, B.A. (1976) Exposure to trichloroethylene. III. Psychological functions. *Scand. J. Work environ. Health*, 4: 220-224.

GAULTIER, M. (1974) *[Clinical toxicology,]* Rome, Italy, Società Editrice Demi (in Italian).

GEHRING, P.J. (1968) Hepatoxic potency of various chlorinated hydrocarbon vapours relative to their narcotic and lethal potencies in mice. *Toxicol. appl. Pharmacol.*, 13: 287-298.

GOLDBERG, M.E., JOHNSON, H.E., POZZANI, U.C., & SMITH, H.E., Jr (1964) Effect of repeated inhalation of vapours of industrial solvents on animal behaviour. I. Evaluation of nine solvent vapours on pole-climb performance in rat. *Am. Ind. Hyg. Assoc. J., 25: 369-375.*

GOLDBLATT, M.W. (1956) *Industrial medicine and hygiene*, London, Merewether, Vol. 3.

GOVERNA, M. (1981) [Occupational diseases due to chlorine derivatives of the aliphatic hydrocarbons.] In: Sartorelli, E., ed. *[Textbook of occupational medicine,]* Padova, Italy, Vol. 1, pp. 483-502 (in Italian).

GRADISKI, D., MAGADUR, J.-L., BAILLOT, M., DANIERE, M.C., & SCHUH, M.B. (1974) Toxicité comparée des principaux solvants chlorés aliphatiques. *J. eur. Toxicol.*, 7: 247-254.

GRADISKI, D., BONNET, P., RAOULT, G., MAGADUR, J.-L., & FRANCIN, J.M. (1978) Toxicité aiguë comparée par inhalation des principaux solvants aliphatiques chlorés. *Arch. Mal. prof. Méd. Trav. Sécur. soc.*, 39: 249-257.

GRAEDEL, T.E. (1978) *Chemical compounds in the atmosphere*, New York, Academic Press.

GRANDJEAN, E. (1960) Trichloroethylene effects on animal behaviour. *Arch. environ. Health*, 1: 106-108.

GRANDJEAN, E. (1963) The effects of short exposures to trichloroethylene on swimming performances and motor activity of rats. *Am. Ind. Hyg. Assoc. J.*, 24: 376-379.

GRANDJEAN, E., MUNCHINGER, R., TURRIAN, V., HAAS, P.A., KNOEPFEL, H.K., & ROSENMUND, H. (1955) Investigations into the effects of exposure to trichloroethylene in mechanical engineering. *Br. J. ind. Med.*, 12: 131-142.

GRANT, M.W. (1961) Trichoroethylene in the eye. *J. Am. Med. Assoc.*, 178: 359.

GRANT, M.W. (1974) *Toxicology of the eye*, 2nd ed., Springfield, Illinois, Charles C. Thomas, pp. 1034-1045.

GRAOVAC-LEPOSAVIC, L., MILOSAVLJEVIC, Z., & ILIC, V. (1964) [Liver function in workers exposed to trichloroethylene.] *Arh. Hig. Rad. Toksikol., 15: 93-97 (in Yugoslavian).*

GREEN, T. & PROUT, M.S. (in press) Species differences in response to trichloroethylene. II. Biotransformation in rats and mice. *Arch. Toxicol.*

GREEN, T., PROUT, M.S., & PROVAN, W.M. (in press) Species differences in response to trichloroethylene. I. Pharmacokinetics in rats and mice. *Arch. Toxicol.*

GREIM, H., BONSE, G., RADWAN, Z., REICHERT, D., & HENSCHLER, D. (1975) Mutagenicity *in vitro* and potential carcinogenicity of chlorinated ethylenes as a function of metabolic oxirane formation.

Biochem. Pharmacol., 24: 2013-2017.

GU, Z.W., SELE, B., JALBERT, P., VINCENT, M., VINCENT, F., MARKA, C., CHMARA, D., & FAURE, J. (1981) Induction of sister chromatid exchanges by trichloroethylene and its metabolites. *Toxicol. Eur. Res.*, 3: 63-67.

GUN, R.T., GRYGORCENICZ, D., & NETTELBECK, T.J. (1978) Choice reaction time in workers using trichloroethylene. *Med. J. Aust.*, 1: 535-536.

HARDELL, L., ERIKSSON, M., LENNER, P., & LUNDGREN, E. (1981) Malignant lymphoma and exposure to chemicals, especially organic solvents, chlorophenols, and phenoxy acids: a case-control study. *Br. J. Cancer*, 73: 169-176.

HATHWAY, D.E. (1980) Consideration of the evidence for mechanisms of 1,1,2-trichloroethylene metabolism including new identification of its dichloroacetic acid and trichloroacetic acid metabolites in mice. *Cancer Lett.*, 8: 263-269.

HAYDEN, J.W., COMSTOCK, E.G., & COMSTOCK, B.S. (1976) The chemical toxicology of solvent abuse. *Clin. Toxicol.*, 9: 169-184.

HEIL, E., OESER, H., HATZ, R., & KELKER, H. (1979) [Gas chromatographic determination of traces of C- and C-fluoro- and chlorohydrocarbons in air.] *Fresenius Z. Anal. Chem.*, 297: 357-364 (in German).

HENSCHLER, D. (1977a) Metabolism and mutagenicity of halogenated olefuis: a comparison of structure and activity. *Environ. Health Perspect.*, 21: 61-64.

HENSCHLER, D. (1977b) Metabolism of chlorinated alkenes and alkanes as related to toxicity. *J. environ. Pathol. Toxicol.*, 1: 125-133.

HENSCHLER, D. & HOOS, R. (1982) Metabolic activation and deactivation mechanisms of di-, tri-, and tetrachloroethylenes. In: Snyder, Parke, Kocsis, Jollow, Gibson, & Witmer, ed. *Biological reactive intermediates. II. Part A.*, New York, Plenum Publishing Corporation, pp. 659-666.

HENSCHLER, D., BONSE, G., & GREIM, H. (1970a) [Cranial nerve neuritis due to poisoning with chlorinated acetylenes when handling vinylidene chloride copolymers.] *Arch. Toxikol.*, 26: 62-75 (in German).

HENSCHLER, D., BROSER, F., & HOPF, H.C. (1970b) *Environmental Pollution and Carcinogenic Risks*, Lyons, International Agency for Research on Cancer, pp. 171-175 (IARC Scientific Publications No. 13).

HENSCHLER, D., EDER, E., NEUDECKER, T., & METZLER, M. (1977) Carcinogenicity of trichloroethylene: fact or artifact? *Arch. Toxicol.*, 37: 233-236.

HENSCHLER, D., HOOS, W.R., FETZ, H., DALLMEIER, E., & METZLER, M. (1979) Reactions of trichloroethylene epoxide in aqueous systems. *Biochem. Pharmacol.*, 28: 543-548.

HENSCHLER, D., ROMEN, W., ELSASSER, H.M., REICHERT, D., EDER, E., & RADWAN, Z. (1980) Carcinogenicity study of trichloroethylene by long-term inhalation in three animal species. *Arch. Toxicol.*, 43: 237-248.

HENSCHLER, D., BONSE, G., & DEKANT, W. (1983) Mechanisms and reactions of electroplulic intermediates of halogenated ofefins. In: *Proceedings of the 13th International Cancer Congress. Part B. Biology of Cancer*, New York, Alan R. Liss, Inc., Vol. 1, pp. 175-183.

HENSCHLER, D., ELSASSER, H., ROMEN, W., & EDER, E. (1984) Carcinogenicity study of trichloroethylene, with and without epoxide stabilizers, in mice. *J. Cancer Res. clin. Oncol.*, 107:

149-156.

HERBOLSHEIMER, R., FUNK, L., & DRASCHE, H. (1972) Use of activated carbon as an adsorbent in the determination of trichloroethylene in the air. *Staub-Reinhalt. Luft*, 32: 407-409.

HERNBERG, S., KORKATA, M.-L., ASIKAINEN, V., & RIALA, R. (1984) Primary liver cancer and exposure to solvents. *Int. Arch. occup. environ. Health*, 54: 147-153.

HIRAYAMA, T. & IKEDA, M. (1979) Applicability of carbon felt to the dosimetry of solvent vapour mixture. *Am. Ind. Hyg. Assoc. J.*, 40: 1091-1096.

HOBARA, T., KOBAYASHI, H., HIGASHIHARA, E., KAWAMOTO, T., & SAKAI, T. (1983) [Experimental studies of trichloroethylene toxicity. II. Changes in trichloroethylene in blood serum and in urine during and after exposure to trichloroethylene.] *Nippon Eiseigaku Zasshi*, 38(4): 772-779 (in Japanese with English summary).

HOWARD, C.J. (1976) Rate constants for the gas-phase reactions of OH radicals with ethylene and halogenated ethylene compounds. *J. Chem. Phys.*, 65: 4771.

HUFF, J.E. (1971) New evidence on the old problems of trichloroethylene. *Ind. Med. Surg.*, 40: 25-33.

IARC (1979) *Trichloroethylene*, Lyons, International Agency for Research on Cancer, pp. 545-572 (IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Vol. 20).

IARC (1982) *Chemicals, industrial processes, and industries associated with cancer in humans*, Lyons, International Agency for Research on Cancer, pp. 247-249 (IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Supplement 4).

IKEDA, M. (1974) Reciprocal metabolic inhibition of toluene and trichloroethylene *in vivo* and *in vitro*. *Int. Arch. Arbeitsmed.*, 33: 125-130.

IKEDA, M. & IMAMURA, T. (1973) Biological half-life of trichloroethylene and tetrachloroethylene in human subjects. *Int. Arch. Arbeitsmed.*, 31: 209-224.

IKEDA, M., OHTSUJI, H., KAWAI, H., & KUNIYOSHI, M. (1971) Excretion kinetics of urinary metabolites in patient addicted to trichloroethylene. *Br. J. ind. Med.*, 28: 203-206.

IKEDA, M., OHTSUJI, H., IMAMAURA, T., & KOMOIKE, Y. (1972) Urinary excretion of total trichloro-compounds, trichloroethanol, and trichloroacetic acid as a measure of exposure to trichloroethylene and tetrachloroethylene. *Br. J. ind. Med.*, 29: 328-333.

ILO (1980) *Occupational exposure limits for airborne toxic substances*, 2nd (revised) ed., Geneva, International Labour Office, pp. 206-207 (Occupational Safety and Health Series No. 37).

IMAMURA, T. & IKEDA, M. (1973) A time-saving procedure for the determination of total trichloro-compounds in human urine samples. *Int. Arch. Arbeitsmed.*, 31: 333-338.

IOFFE, B.V., ISIDOROV, V.A., & ZENKEVICH, I.G. (1977) Gas chromatographic-mass spectrometric determination of volatile organic compounds in an urban atmosphere. *J. Chromatogr.*, 142: 787-795.

IRPTC (1984) *IRPTC legal file*, Geneva, International Register of Potentially Toxic Chemicals, United Nations Environment Programme.

JAMES, W.R.L. (1963) Fatal addiction to trichloroethylene. *Br. J. ind. Med.*, 20: 47-49.

JAKOBSON, I., WAHLBERG, J.E., HOLMBERG, B., & JOHANSSON, G. (1982) Uptake via the blood and elimination of 10 organic solvents

following epicutaneous exposure of anaesthetized guinea-pigs. *Toxicol. appl. Pharmacol.*, 63: 181-187.

JAPANESE YEARBOOK OF CHEMICAL INDUSTRIES STATISTICS (1983) Tokyo, Ministry of International Trade and Industry.

JESSON, J.P. (1980) Release of industrial halocarbons and tropospheric budget. In: *Proceedings of the NATO Advanced Study Institute on Atmospheric Ozone: Its Variation and Human Influences*, Washington DC, US Department of Transportation, Federal Aviation Administration.

JOINT FAO/WHO EXPERT COMMITTEE ON FOOD ADDITIVES (1983) *Evaluation of certain food additives: 27th report*, Geneva, World Health Organization (Technical Report Series No. 696).

JOUGLARD, J. & VINCENT, V. (1971) Pulmonary signs of ingestions of solvents. *Marseille Med.*, 108: 696-697.

JUNGCLAUS, G.A., LOPEZ-AVILA, V., & HITES, R.A. (1978) Organic compounds in an industrial waste water: a case study of their environmental impact. *Environ. Sci. Technol.*, 12: 88-96.

KALASHINIKOVA, V.P., VISSARIONOVA, V.Ya., BONDAREV, G.I., SKIRKO, B.K., & KARANTIROVA, O.V. (1974) [Influence of different diets on the course of chronic experimental trichloroethylene poisoning.] *Vopr. Pitan.*, No.6: 43-47 (in Russian).

KALASHINIKOVA, V.P., SKIRKO, B.K., VISSARIONOVA, V.Ya., BONDAREV, G.I., KLEIMENOVA, N.V., POPOVA, N.S., & KARANTIROVA, O.V. (1976) [Evaluation of the toxic effects of trichloroethylene according to morphological and some biochemical indexes.] *Gig. i Sanit.*, No. 2: 107-108 (in Russian).

KANJE, M., KJELLSTRAND, P., FOX, K., & WALLDORF, A. (1981) Neurotransmitter metabolizing enzymes and plasma butyrylcholinesterase in mice exposed to trichloroethylene. *Acta pharmacol. toxicol.*, 49: 205-209.

KARICKHOFF, S.W., BROWN, D.S., & SCOTT, T.A. (1979) Sorption of hydrophobic pollutants on natural sediments. *Water Res., 13: 241-248.*

KATZ, R.M. & JOWETT, D. (1981) Female laundry and dry cleaning workers in Wisconsin: a mortality analysis. *Am. J. public Health*, 71: 305-307.

KEINFELD, M. & TABERSHAW, I.R. (1954) Trichloroethylene toxicity: report of 5 fatal cases. *AMA Arch. ind. Hyg. occup. Med.*, 10: 134-141.

KENAGA, E.E. (1980) Predicted bioconcentration factors and soil sorption coefficients of pesticides and other chemicals. *Ecotoxicol. environ. Saf.*, 4: 26-38.

KENAGA, E.E. & GORING, C.A.I. (in press) Relationship between water solubility, soil sorption, octanol-water partitioning, and bioconcentration of chemicals in biota. In: Eaton, J.C., Parrish, P.R., & Hendricks, A.C., ed. *Aquatic toxicology ASTM STP, 707*, Philadelphia, Pennsylvania, American Society for Testing and Materials.

KIMMERLE, G. & EBEN, A. (1973) Metabolism, excretion, and toxicology of trichloroethylene after inhalation. II. Experimental human exposure. *Arch. Toxicol.*, 30: 127-138.

KIRK, R.E. & OTHMER, D.F., ed. (1963) Anesthetics. In: *Encyclopedia of chemical technology*, 2nd ed., New York, John Wiley and Sons, Vol. 2, pp. 393-410.

KIRK, R.E. & OTHMER, D.F., ed. (1964) Chlorocarbons and chlorohydrocarbons. In: *Encyclopedia of chemical technology*, 2nd ed., New York, John Wiley and Sons, Vol. 5, pp. 183-195.

KIRK, R.E. & OTHMER, D.F., ed. (1979) Chlorocarbons and chlorohydrocarbons. In: *Encyclopedia of chemical technology*, 3rd ed., New York, John Wiley and Sons, Vol. 5, pp. 745-753.

KISELEVA, A.F. & KOROLENKO, A.M. (1971) Histoenzymic changes in liver and kidneys during the experimental effect of anesthetic doses of fluorothan and trylen. *Eksp. Khir. Anesteziol.*, 16: 81-84.

KITAGAWA, T. (1961) The rapid measurement of toxic gases and vapour. In: *Proceedings of the 13th International Congress on Occupational Health*, New York, pp. 506-512.

KJELLSTRAND, P., LANKE, J., BJERKEMO, M., ZETTERQUIST, L., & MANSSON, L. (1980) Irreversible effects of trichloroethylene exposure on the central nervous system. *Scand. J. Work environ. Health*, 6: 40-47.

KJELLSTRAND, P., KANJE, M., MANSSON, L., BJERKEMO, M., MORTENSEN, I., LANKE, J., & HOLMQUIST, B. (1981a) Trichloroethylene: effects on body and organ weights in mice, rats, and gerbils. *Toxicology*, 21: 105-115.

KJELLSTRAND, P., KANJE, M., MANSSON, L., BJERKEMO, M., MORTENSEN, I., LANKE, J., & HOLMQUIST, B. (1981b) Effects of long-term exposure to trichloroethylene on the behaviour of Mongolian gerbils *(Meriones unguiculatus)*. *J. Toxicol. environ. Health*, 8: 787-793.

KJELLSTRAND, P., HOLMQUIST, B., MANDAHL, N., & BJERKEMO, M. (1983a) Effects of continuous trichloroethylene inhalation on different strains of mice. *Acta pharmacol. toxicol.*, 53: 369-374.

KJELLSTRAND, P., HOLMQUIST, B., ALM, P., KANJE, M., ROMARE, S., JONSSON, I., MANSSON, L., & BJERKEMO, M. (1983b) Trichloroethylene: further studies of the effects on body and organ weights and plasma butyrycholinesterase activity in mice. *Acta pharmacol. toxicol.*, 53: 375-384.

KLAASSEN, C.D. & PLAA, G.L. (1966) Relative effects of various chlorinated hydrocarbons on liver and kidney function in mice. *Toxicol. appl. Pharmacol.*, 9: 139-151.

KLAASSEN, C.D. & PLAA, G.L. (1967) Relative effects of various chlorinated hydrocarbons on liver and kidney function in dogs. *Toxicol. appl. Pharmacol.*, 10: 119-131.

KLINE, S.A., MCCOY, E.C., ROSENKRANZ, H.S., & VAN DUUREN, B.L. (1982) Mutagenicity of chloroalkene epoxides in bacterial systems. *Mutat. Res.*, 101: 115-125.

KLOWKOWSKI, R., SCHEUNERT, I., KLEIN, W., & KORTE, F. (1981) Laboratory screening of distribution, conversion, and mineralisation of chemicals in the soil-plant-system and comparison to outdoor experimental data. *Chemosphere*, 10: 1089-1090.

KONIETZKO, H., HABERLANDT, W., HEILBRONNER, H., REILL, G., & WEICHARDT, H. (1978) [Chromosome studies on trichloroethylene workers.] *Arch. Toxicol.*, 40: 201-206 (in German).

KRONEVI, T., WAHLBERG, J.E., & HOLMBERG, B. (1981) Skin pathology following epicutaneous exposure to seven organic solvents. *Int. J. Tiss. React.*, 111(1): 21-30.

KYLIN, B., REICHARD, H., SUMEGI, I., & YLLNER, S. (1962) Heptatoxic effect of tri- and tetrachloroethylene on mice. *Nature (Lond.)*, 193: 395.

KYLIN, B., AXELL, K., EHRNER-SAMUEL, H., & LINDBORG, A. (1967) Effect of inhaled trichloroethylene on the CNS. *Arch. environ. Health*, 15: 48-52.

KYLSZEIJKO, C., LAKOMY, T., & PAPIEROWSKI, A. (1963) [The influence of trichloroethylene during labour process in women in comparison with the results *in vitro*.] *Pol. Tyg. Lek.*, 18: 1333-1338 (in Polish).

KYRKLUND, T., ALLING, C., HAGLID, K., & KJELLSTRAND, P. (1983) Chronic exposure to trichloroethylene: lipid and acyl group composition in gerbil cerebral cortex and hippocampus.

Neurotoxicology, 4(4): 35-42.

LACHNIT, V. (1971) Halogenated hydrocarbons and the liver. *Wein. Klin. Wochenschr.*, 83: 734-737.

LA DU, B.N., MANDEL, H.G., & WAY, E.L., ed. (1971) *Fundamentals of drug metabolism and drug disposition*, Baltimore, Maryland, Williams and Wilkins.

LAHAM, S. (1970) Studies on placental transfer: trichloroethylene. *Ind. Med. Surg.*, 39: 46-49.

LAZAREV, N.V. (1929) [The narcotic efficacy of the vapours of the chlorine derivatives of methane, ethane and ethylene.] *Naunyn-Schmiedeb. Arch. exp. Pathol. Pharmacol.*, 141: 19-24 (in German).

LAZAREV, N.V. & GADASKINA, I.D. (1977) *[Harmful substances in industry,]* Leningrad, Medecine, Vol. 3 (in Russian).

LEHMANN, K.B. & SCHMIDT-KEHL, L. (1936) [Thirteen most important chlorohydrocarbons of the paraffin series from the standpoint of industrial hygiene.] *Arch. Hyg. Bakt.*, 116: 131-268 (in German).

LEIBMAN, K.C. (1965) Metabolism of trichloroethylene in liver microsomes. *Mol. Pharmacol.*, 1: 239-246.

LEONG, B.K.J., SCHWETZ, B.A., & GEHRING, P.J. (1975) Embryo- and fetotoxicity of inhaled trichloroethylene, perchloroethylene, methylchloroform, and methylene chloride in mice and rats. *Toxicol. appl. Pharmacol.*, 33: 136.

LERZA, P., LOMBARDI, F., & VIOTTI, G. (1963) [Health and hygiene considerations regarding work in dry-cleaning establishments using trichloroethylene.] *Lav. Um.*, 15: 459-478 (in Italian).

LEWIS, G.D., REYNOLDS, R.C., & JOHNSON, A.R. (1984) Some effects of trichloroethylene on mouse lungs and livers. *Gen. Pharmacol.*, 15(2): 139-144.

LILIS, R., STANESCU, D., MUICA, N., & ROVENTA, A. (1969) Chronic effects of trichloroethylene exposure. *Med. Lav.*, 60: 595-601.

LIN, R.S. & KESSLER, I.I. (1981) A multifactoral model for pancreatic cancer in man. *J. Am. Med. Assoc.*, 245: 147-152.

LOPRIENO, N. & ABBONDANDOLO, A. (1980) Comparative mutagenic evaluation of some industrial compounds. In: *Proceedings of the Symposium on Short-Term Test Systmes and the Detection of Carcinogenesis*, Berlin, Heidelberg, New York, Springer-Verlag, pp. 333-356.

LOPRIENO, N., SBRANA, I., & LASCIALFARI, D. (in press) [Study on the *in vivo* potential mutagenic effect of trichloroethylene evaluated by cytogenetic analysis in mice treated by inhalation for 52 days.] (in Italian).

LOVELOCK, J.E. (1974) Atmospheric halocarbons and stratospheric ozone. *Nature (Lond.)*, 252: 292-294.

MACKAY, D. (1982) Correlations of bioconcentration factors. *Environ. Sci. Technol.*, 10: 274-278.

MCCONNELL, G., FERGUSON, D.M., & PEARSON, C.R. (1975) Les hydrocarbures chlorés et l'environnement. *Endeavour*, 34: 13-18.

MCCORD, C.P. (1932) Toxicity of trichloroethylene. *J. Am. Med. Assoc.*, 99: 409.

MACKAY, D. & PATERSON, S. (1981) Calculating fugacity. *Environ. Sci. Technol.*, 15: 1006-1014.

MCNEILL, W.C. (1979) Chlorocarbons and chlorohydrocarbons. Trichloroethylene. In: *Kirk-Othmer's encyclopedia of chemical technology*, 3rd ed., New York, Wiley Interscience, Vol. 5, pp. 745-753.

MAKIDE, Y., TOMINAGA, K., & ROWLAND, F.S. (1979) Gas chromatographic analysis of halogenated hydrocarbons in air over Japan. *Chem. Lett.*, 1979: 355-358.

MALEK, B., KRCMAROVA, B., & RODOVA, O. (1979) [Epidemiological study of hepatic tumour incidence in subjects working with trichloroethylene: II. Negative result of retrospective studies in dry cleaners.] *Pracov. Lek.*, 31(4): 124-126 (in Czech).

MALIZIA, E., PELAIA, P., & PIROVINE, C. (1984) Experience in the emergency treatment of acute intoxication by trichloroethylene. *Riv. Toss. Speriment Clin.*, 14: 437.

MALOOF, C.C. (1949) Burns of the skin produced by trichloroethylene vapours at room temperature. *J. ind. Hyg. Toxicol., 31: 295-296.*

MALTONI, I.C. & MAIOLI, P. (1977) [Long-term bioassay of carcinogenicity of trichloroethylene. Preliminary results.] *Osp. Vita*, 4: 108-110 (in Italian).

MALTONI, C., LEFEMINE, G., & COTTI, G. (in press) Experimental research on trichloroethylene carcinogenesis. In: Maltoni, C. & Mehlman, M.A., ed. *Archives of research on industrial carcinogenesis*, Vol. 5.

MANTEL, M. & NOTHMANN, R. (1977) Rapid determination of trichloroethanol and trichloroacetic acid in urine. *Analyst, 102:* 672-677.

MATTURRO, F. (1963) [Effects of some volatile narcotics on isolated guinea-pig heart.] *G. Ital. Chir.*, 19: 95-99 (in Italian).

MAZZA, V. & BRANCACCIO, A. (1967) [Behaviour of the formed elements of the blood and the marrow in experimental intoxications with Tri.] *Folia med.*, 50: 318-324 (in Italian).

MELINO, C., MESSINEO, A., & PACELLI, E. (1979) Risk from trichloroethylene in a factory producing electrical condensers. *Riv. Med. Lab. Ig. Ind.*, 3: 67-81.

MEYER, H.-J. (1966) [Trichloroethylene poisoning by the oral route.] *Arch. Toxikol.*, 21: 225-234 (in German).

MEYER, H.-J. (1973) Bronchopulmonary alterations induced by trichloroethylene and other halogenated hydrocarbons. *Bronches*, 23: 113-124.

MILLER, D.A. & GRIMSRUD, E.P. (1979) Correlation of electron capture response enhancements caused by oxygen with chemical structure for chlorinated hydrocarbons. *Anal. Chem.*, 51: 851-859.

MILLER, R.E. & GUENGERICH, F.P. (1982) Oxidation of trichloroethylene by liver microsomal cytochrome P-450: evidence for chlorine migration in a transition state not involving trichloroethylene oxide. *Biochemistry*, 21: 1090-1097.

MOLINA, M.J. & ROWLAND, F.S. (1974) Stratospheric sink for chlorofluoromethanes: chlorine atom-catalysed destruction of ozone. *Nature (Lond.)*, 294: 810-812.

MONSTER, A.C. (1979) Differences in uptake, elimination, and metabolism in exposure to trichloroethylene, 1,1,1-trichloroethane, and tetrachloroethylene. *Int. Arch. occup. environ. Health*, 42: 311-317.

MONSTER, A.C. & BOERSMA, G. (1975) Simultaneous determination of trichloroethylene and metabolites in blood and exhaled air by gas chromatography. *Int. Arch. occup. environ. Health*, 35: 155-163.

MONSTER, A.C., BOERSMA, G., & DUBA, W.C. (1976) Pharmacokinetics of trichloroethylene in volunteers: influence of workload and exposure concentration. *Int. Arch. occup. environ. Health*, 38: 87-102.

MONSTER, A.C., BOERSMA, G., & DUBA, W.C. (1979) Kinetics of trichloroethylene in repeated exposure of volunteers. *Int. Arch. occup. environ. Health*, 42: 283-292.

MOSLEN, M.T., REYNOLDS, E.S., & SZABO, S. (1977) Enhancement of the metabolism and hepatoxicity of trichloroethylene and perchloroethylene. *Biochem. Pharmacol.*, 26: 369-375.

MULLER, G., SPASSOVSKI, M., & HENSCHLER, D. (1972) Trichloroethylene exposure and trichloroethylene metabolites in urine and blood. *Arch. Toxicol.*, 29: 335-340.

MULLER, G., SPASSOVSKI, M., & HENSCHLER, D. (1974) Metabolism of trichloroethylene in man. II. Pharmacokinetics of metabolites. *Arch. Toxicol.*, 32: 283-295.

MULLER, G., SPASSOVSKI, M., & HENSCHLER, D. (1975) Metabolism of trichloroethylene in man. III. Interaction of trichloroethylene and ethanol. *Arch. Toxicol.*, 33: 173-189.

MURRAY, A.J. & RILEY, J.P. (1973) Occurrence of some chlorinated aliphatic hydrocarbons in the environment. *Nature (Lond.)*, 242: 37-38.

MUSCLOW, C.E. & AWEN, C.F. (1971) Glue sniffing: report of a fatal case. *Can. Med. Assoc. J.*, 104: 315-319.

NCI (1976) *Carcinogenesis bioassay of trichloroethylene.* CAS No. 79-01-6, Washington DC, National Cancer Institute (US DHEW Publication No. (NIH) 76-802).

NEELY, W.B. & MACKAY, D. (1982) In: Dickson, K.L., Maki, A.W., & Cairns, J., ed. *Modelling the fate of chemicals in the aquatic environment*, Ann Arbor, Michigan, Ann Arbor Science Publications.

NICHOLSON, A.A., MERESZ, O., & LEMYK, B. (1977) Determination of free and total potential haloforms in drinking-water. *Anal. Chem.*, 49: 814-819.

NIOSH (1977a) *Manual of analytical methods*, 2nd ed., Cincinnati, Ohio, National Institute of Occupational Safety and Health, Vol. 3, pp. S336-1 to S336-9 (US DHEW Publication No. (NIOSH) 77-157-C).

NIOSH (1977b) *National occupational hazard survey. III. Survey analyses and supplemental tables*, Cincinnati, Ohio, National Institute of Occupational Safety and Health, pp. 2864-2866.

NIOSH (1978) *Special occupational hazard review of trichloroethylene*, Washington DC, National Institute of Occupational Safety and Health (US DHEW Publication No. (NIOSH) 78-130).

NOMIYAMA, H., NOMIYAMA, K., & UCHIKI, H. (1978) Gas-liquid chromatographic determination of trichloroethylene metabolites in urine. *Am. Ind. Hyg. Assoc. J.*, 39: 506-510.

NOMURA, S. (1962) Health hazards in workers exposed to trichloroethylene vapour. I. Trichloroethylene poisoning in an electroplating plant. *Kumamoto med. J.*, 15: 29-37.

NORRIS, W. & STUART, P. (1957) Cardiac arrest during trichloroethylene anaesthesia. *Br. med. J.*, 1: 860-863.

NOVOTNA, E., DAVID, A., & MALEK, B. (1979) [An epidemiological study on hepatic tumour incidence in subjects working with trichloroethylene. I. Negative result of retrospective investigations in subjects with primary liver carcinoma.] *Pracov. Lek.*, 31(4): 121-123 (in Czech).

ODUM, E.P. (1971) *Fundamentals of ecology*, 3rd ed., Philadelphia, Pennsylvania, W.B. Saunders Co.

OGATA, M. & SAEKI, T. (1974) Measurement of chloral hydrate, trichloroethanol, trichloroacetic acid, and monochloroacetic acid in the serum and the urine by gas chromatography. *Int. Arch.*

Arbeitsmed., 33: 49-58.

OGATA, M., TAKATSUKA, Y., TOMUKUNI, K., & MUROI, K. (1970) Simple method for the quantitative analysis of urinary trichloroethanol and trichloroacetic acid as an index of trichloroethylene exposure. *Br. J. ind. Med.*, 27: 378-381.

OHTA, T., MORITA, M., & MIZOGUCHI, I. (1976) Local distribution of chlorinated hydrocarbons in the ambient air in Tokyo. *Atmos. Environ.*, 10: 557-560.

OLSSON, H. & BRANDT, L. (1980) Occupational exposure to organic solvents and Hodgkin's disease in men. *Scand. J. Work environ. Health*, 6: 302-305.

OSTELERE, G. (1953) *Trichloroethylene anaesthesia*, Edinburgh, Scotland, Livingstone.

OTSON, R., WILLIAMS, D.T., & BIGGS, D.C. (1982) Relationships between raw water quality treatment and the occurrence of organics in Canadian potable water. *Bull. environ. Contam. Toxicol.*, 28: 396-403.

PADDLE, G.M. (1983) Incidence of liver cancer and trichloroethylene manufacture: joint study by industry and a cancer registry. *Br. med. J.*, 286: 876.

PARCHMAN, L.G. & MAGEE, P.N. (1982) Metabolism of ^{14}C-trichloroethylene to $^{14}CO_2$ and interaction of a metabolite with liver DNA in rats and mice. *J. Toxicol. environ. Health*, 9: 797-813.

PARDYS, S. & BROTMAN, M. (1974) Trichloroethylene and alcohol: a straight flush. *J. Am. Med. Assoc.*, 229:: 521-522.

PARMEGGIANI, L. (1956) [A case of chronic trichloroethylene poisoning.] *Med. Lav.*, 47: 639-646 (in Italian).

PARSONS, F., WOOD, P.R., & DE MARCO, J. (1984) Transformation of tetrachloroethene and trichloroethene in microcogus and ground water. *J. Am. Water Works Assoc.*, 76(2): 56-59.

PEARSON, C.R. & MCCONNELL, G. (1975) Chlorinated C and C hydrocarbons in the marine environment. *Proc. R. Soc. London Ser. B*, 189: 305-332.

PELTS, D.G. (1962) [Effect of trichloroethylene and tetrachloroethylene on the phagocytic activity of blood leukocytes.] *Gig. i Sanit.*, 27(7): 96-99 (in Russian).

PENKETT, S.A. (1982) Non-methane organics in the remote troposphere. In: Goldberg, E.D., ed. *Atmospheric chemistry*, Berlin, Springer, pp. 329-355.

PEROCCO, P. & PRODI, G. (1981) DNA damage by haloalkanes in human lymphocytes cultered *in vitro*. *Cancer Lett.*, 13: 213-218.

PESSAYRE, D., ALLEMAND, H., WANDSCHEER, J.C., DESCATOIRE, V., ARTIGOU, J-Y., & BENHAMOU, J-P. (1979) Inhibition, activation, destruction, and induction of drug-metabolizing enzymes by trichloroethylene. *Toxicol. appl. Pharmacol.*, 49: 355-363.

PESSAYRE, D., BARTON, C., DESCATOIRE, V., DEGOTT, C., BABANY, G., FUNCK-BRENTANO, C., DELAFORGE, M., & LARREY, D. (1982) Hepatotoxicity of trichloroethylene-carbon tetrachloride mixtures in rats. *Gastroenterology*, 63: 761-772.

PIERCE, R.H., OLNEY, C.E., & FELBECK, G.T. (1974) p-p'-DDT adsorption to suspended particulate matter in sea water. *Geochim. cosmochim. Acta*, 38: 1060-1073.

PLAA, G.L., EVANS, E.A., & HINE, C.H. (1958) Relative hepatoxicity of seven halogenated hydrocarbons. *J. Pharmacol. exp. Ther.*, 123: 224-229.

POWELL, J.F. (1945) Trichloroethylene: absorption, elimination, and metabolism. *Br. J. ind. Med.*, 2: 142-145.

PRENDERGAST, J.A., JONES, R.A., JENKINS, L.J., Jr, & SIEGEL, J. (1967) Effects on experimental animals of long-term inhalation of trichloroethylene, carbon tetrachloride, 1,1,1-trichloroethane, dichlorodifluoromethane, and 1,1-dichloroethylene. *Toxicol. appl. Pharmacol.*, 10: 270-289.

PRICE, P.J., HASSETT, C.M., & MANSFIELD, J.I. (1978) Transforming activities of trichloroethylene and proposed industrial alternatives. *In Vitro*, 14: 290-293.

PRIEST, R.J. & HORN, R.C., Jr (1965) Trichloroethylene intoxication: a case of acute hepatic necrosis possibly due to this agent. *Arch. environ. Health*, 11: 361-365.

RASMUSSEN, R.A. & KHALIL, M.A.K. (1983) Natural and anthropogenic trace gases in the lower troposphere of the Arctic. *Chemosphere*, 12: 371-375.

REICHERT, D. & HENSCHLER, D. (1978) Nephrotoxic and hepatotoxic effects of dichloroacetylene. *Food Cosmet. Toxicol.*, 16: 227-235.

REICHERT, D., METZLER, M., & HENSCHLER, D. (1980a) Decomposition of the neuro- and nephrotoxic compound dichloroacetylene in the presence of oxygen: separation and identification of novel products. *J. environ. Pathol. Toxicol.*, 4: 525-532.

REICHERT, D., SPENGLER, U., ROMEN, W., & HENSCHLER, D. (1980b) Carcinogenic potential of dichloroacetylene. *Dev. Toxicol. environ. Sci.*, 8: 269-272.

RIGAUD, M., CHALABREYSSE, J., PROST, G., & TOLOT, F. (1977) Etude expérimentale de la toxicité hépatique du trichloroéthylène. *Arch. Mal. prof. Méd. Trav. Sécur. soc.*, 38: 263-265.

ROCHE, L., GENEVOIS, M., & MARIN, A. (1958) Trichloréthylène et eczéma de la face. *Arch. Mal. prof. Méd. Trav. Sécur. soc.*, 19: 615-616.

ROESSLER, R. & MORAWIEC-BOROWIAK, D. (1973) [Use of hemodialysis in acute trichloroethylene poisoning.] *Wiad. Lek.*, 26(13): 1271-1275 (in Polish).

ROOK, J.J., MEIJERS, A.P., GRAS, A.A., & NOORDSIJ, A. (1975) [Headspace analysis of volatile trace compounds in the Rhine.] *Vom Wasser*, 44: 23-30 (in German).

ROSSI, A.M., MIGLIORE, L., BARALE, R., & LOPRIENO, N. (1983) *In vivo* and *in vitro* mutagenicity studies of a possible carcinogen, trichloroethylene, and its two stabilizers, epichlorohydrin and 1,2-epoxybutane. *Teratog. Carcinog. Mutagen.*, 3: 75-87.

RUBINO, G.F., SCANSETTI, G., & TROMPEO, G. (1959) [Chronic trichloroethylene poisoning. II. Absorption of trichloroethylene.] *Med. Lav.*, 50(12): 733-742 (in Italian).

RUSH, W.E. (1970) *Quantitative analysis of gaseous pollutants*, London, Ann Arbor-Humphrey Science Publishers, Inc., pp. 229-232.

RUSSO, A., PACCHIELOTTI, F., & METALLI, P. (1984) Nondysfunction induced in mouse spermatogenesis by chloral hydrate, a metabolite of trichloroethylene. *Environ. Mutagen., 6(5): 695-703.*

SADTLER STANDARD SPECTRA (1961) *25 frequently-used spectra for the infrared spectroscopist*, London, Heyden & Son, Ltd.

SAGAWA, K., NISHITANI, Y., KAWAI, H., KUGE, Y., & IKEDA, M. (1973) Transverse lesion of spinal cord after accidental exposure to trichloroethylene. *Int. Arch. Arbeitsmed.*, 31: 257-264.

SANDERS, V.M., TUCKER, A.N., WHITE, K.L., Jr, KAUFFMANN, B.M., HALLETT, P., CARCHMAN, R.A., BORZELLECA, J.F., & MUNSON, A.E. (1982) Humoral and cell-mediated immune status in mice exposed to

trichloroethylene in the drinking water. *Toxicol. appl. Pharmacol.*, 62: 358-368.

SATO, A. & NAKAJIMA, T. (1978) Differences following skin or inhalation exposure in the absorption and excretion kinetics of trichloroethylene and toluene. *Br. J. ind. Med.*, 35: 43-49.

SATO, A. & NAKAJIMA, T. (1979) A structure-activity relationship of some chlorinated hydrocarbons. *Arch. environ. Health*, 34: 69.

SAVOLAINEN, H. & SEPPALAINEN, A.M. (1979) Biochemical and physiological effects of organic solvents on rat axon membranes isolated by a new technique. *Neurotoxicology*, 1: 467-477.

SAVOLAINEN, H., PFAFFLI, P., TENGEN, M., & VAINIO, H. (1977) Trichloroethylene and 1,1,1-trichloroethane: effects on brain and liver after five days intermittent inhalation. *Arch. Toxicol.*, 38: 229-237.

SAWICKI, E., BELSKY, T., FRIEDEL, R.A., HYDE, D.L., MONKMAN, J.L., RASMUSSEN, R.A., RIPPERTON, L.A., & WHITE, L.D. (1975) Organic solvent vapours in air: analytical method. *Health Lab. Sci.*, 12: 394-402.

SBERTOLI, C. & BRAMBILLA, G. (1962) [Three cases of trichloroethylene poisoning presenting with intolerance to alcohol as the only symptoms.] *Med. Lav.*, 53: 353-358 (in Italian).

SBRANA, I., LASCIALFARI, N., & LOPRIENO, N. (1984) [Analysis of the cytogenetic effects of trichloroethylene.] *AHI AGI/ABCD/SIBBM*, 21-25 October, p. 93 (in Italian).

SCHUMACHER, H. & GRANDJEAN, E. (1960) Comparative investigations on the anaesthetic effects and acute toxicity of nine solvents. *Arch. Gewerbepathol. Gewerbehyg.*, 18: 109-119.

SCHUTTMANN, W. (1970) Liver damage after occupational exposure to trichloroethylene. *Dtsch. Z. Verdau Stoffwechselkr.*, 30: 43-45.

SCHWETZ, B.A., LEONG, B.K.J., & GEHRING, P.J. (1975) The effect of maternally-inhaled trichloroethylene, perchloroethylene, methyl chloroform, and methylene chloride on embryonal and fetal development in mice and rats. *Toxicol. appl. Pharmacol.*, 32: 84-96.

SEBA, D.B. & PROSPERO, J.M. (1971) Pesticides in the lower atmosphere of the northern equatorial Atlantic Ocean. *Atmos. Environ.*, 5: 1043-1050.

SELLERS, E.M., CARR, G., BERSTEIN, J.G., SELLERS, S., & KOCK-WESER, J. (1972) Interaction of chloral hydrate and ethanol in man. II. Hemodynamics and performance. *Clin. Pharmacol. Ther.*, 13: 50-58.

SETO, T.A. & SCHULTZE, M.O. (1956) Determination of trichloroethylene, trichloroacetic acid, and trichloroethanol in urine. *Anal. Chem.*, 28: 1625-1629.

SHAHIN, M.M. & VON BORSTEL, R.C. (1977) Mutagenic and letal effects of alpha-benzene hexachloride, dibutyl phthalate, and trichloroethylene in *Saccharomyces cerevisiae*. *Mutat. Res.*, 48: 173-180.

SHANKS, C.A. (1964) The compatibility of octapressin with cyclopropane, trichloroethylene, and halothane. *N.Z. med. J.*, 63: 156-159.

SHIPMAN, A.J. & WHIM, B.P. (1980) Occupational exposure to trichloroethylene in metal cleaning processes and to tetrachloroethylene in the dry-cleaning industry in the U.K. *Ann. occup. Hyg.*, 23: 197-204.

SHMUTER, L.M. (1972) [The effect of the chronic action of low concentrations of chlorinated hydrocarbons on the production of various classes of immunoglobulins.] *Gig. i Sanit.*, 37(2): 32-35 (in Russian).

SIEGEL, J., JONES, R.A., COON, R.A., & LYON, J.P. (1971) Effects on experimental animals of acute, repeated, and continuous inhalation exposures to dichloroacetylene mixtures. *Toxicol. appl. Pharmacol.*, 18: 168-174.

SIMMON, V.F., KAUHANEN, K., & TARDIFF, R.G. (1977) Mutagenic activity of chemicals identified in drinking-water. *Dev. Toxicol. environ. Sci.*, 2: 249-258.

SINGH, H.B., SALAS, L.J., & CAVANAGH, L.A. (1977) Distribution, sources, and sinks of atmospheric halogenated compounds. *J. Air Pollut. Control Assoc.*, 27: 332-336.

SLACIK-ERBEN, R., ROLL, R., FRANKE, G., & VEHLEKE, H. (1980) Tichloroethylene vapours do not produce dominant lethal mutations in mice. *Arch. Toxicol.*, 45: 37.

SMYTH, H.F., Jr, CARPENTER, C., WEIL, C.S., POZZANI, U.C., STRIEGEL, J.A., & NYCUM, J.S. (1969) Range-finding toxicity data. VII. *Am. Ind. Hyg. Assoc. J.*, 30: 470-476.

SNELL, F.D. & ETTRE, L.S., ed. (1970a) Chlorocarbons. In: *Encyclopedia of industrial chemical analysis*, New York, John Wiley and Sons, Vol. 9, pp. 373-410.

SNELL, F.D. & ETTRE, L.S., ed. (1970b) Chlorocarbons. In: *Encyclopedia of industrial chemical analysis*, New York, John Wiley and Sons, Vol. 9, pp. 454-459.

SNELL, F.D. & HILTON, C.L., ed. (1967) Anesthetics. In: *Encyclopedia of industrial chemical analysis*, New York, John Wiley and Sons, Vol. 5, pp. 357, 366-367, 420-421.

SORGO, G. (1976) [Trichloroethylene, carbon tetrachloride, and gasoline intoxication as etiological factors in the developement of arterio- and coronary sclerosis.] *Arch. Toxicol.*, 35: 295-318 (in German).

SOUCEK, B. & VLACHOVA, D. (1960) Excretion of trichloroethylene metabolites in human urine. *Br. J. ind. Med.*, 17: 60-64.

STEERE, N.V., ed. (1967) *Handbook of laboratory safety*, Cleveland, Ohio, The Chemical Rubber Co., pp. 544-545.

STENHAGEN, E., ABRAHAMSSON, S., & MCLAFFERTY, F.W. (1974) *Registry of mass spectral data*, New York, John Wiley and Sons, Vol. 1, p. 246.

STEWART, R.D. & DODD, H.C. (1964) Absorption of carbon tetrachloride, tetrachloroethylene, methylene chloride, and 1,1,1-trichloroethane through the human skin. *Am. Ind. Hyg. Assoc. J.*, 25: 439-446.

STEWART, R.D., GAY, H.H., ERLEY, D.S., HAKE, C.L., & PETERSON, J.E. (1962) Observations on the concentrations of trichloroethylene in blood and expired air following exposures of humans. *Am. Ind. Hyg. Assoc. J.*, 23: 167-170.

STEWART, R.D., DODD, H.C., GAY, H.H., & ERLEY, D.S. (1970) Experimental human exposure to trichloroethylene. *Arch. environ. Health*, 20: 64-71.

STEWART, R.D., HAKE, C.L., & PETERSON, J.E. (1974) Use of breath analysis to monitor trichloroethylene exposures. *Arch. environ. Health*, 29: 6-13.

STOPPS, G.J. & MCLAUGHLIN, M. (1967) Psychophysiological testing of humans exposed to solvent vapors. *Am. Ind. Hyg. Assoc. J.*, 28: 43-50.

STOTT, W.T., QUAST, J.F., & WATANABE, P.G. (1982) The pharmacokinetics and macromolecular interactions of trichloroethylene in mice and rats. *Toxicol. appl. Pharmacol.*, 62: 137-151.

SUCIU, I. & OLINICI, L. (1983) Hepato-renal involvement in acute

occupational trichloroethylene intoxication. *Med. Lav.*, 74(2): 123-128.

SVABOVA, K. & MENCIK, M. (1983) [Changes in the state of health of workers long-term exposed to halogenated hydrocarbons.] *Pracov. Lek.*, 35: 105-111 (in Czech with English abstract).

SZLUC-KUBERSKA, J. (1972) Chronic industrial trichloroethylene poisoning. *Folia med. Lodz.*, 16: 67-90.

TADA, O. (1969) Evaluating the exposure to some chlorinated hydrocarbons. *J. Sci. Labour, Part 2*, 45: 757-765.

TANAKA, S. & IKEDA, M. (1968) A method for determination of trichloroethanol and trichloroacetic acid in urine. *Br. J. ind. Med.*, 25: 214-219.

TAYLOR, H. (1936) Experiments on the physiological properties of trichloroethylene. *J. ind. Hyg. Toxicol.*, 18: 175-193.

THIELE, D.L., EIGENBRODT, E.E., & WARE, A.J. (1982) Cirrhosis after repeated trichloroethylene and 1,1,1-trichloroethane exposure. *Gastroenterology*, 83: 926-929.

TOLA, S., VILHUNEN, R., JARVINEN, E., & KORKALA, M.-L. (1980) A cohort study on workers exposed to trichloroethylene. *J. occup. Med.*, 22: 737-740.

TOLOT, F., VIALLIER, J., ROULLET, A., RIVOIRE, J., & FIGUERES, J.C. (1964) Toxicité hepatique au trichloroéthylène. *Arch. Mal. prof. Méd. Trav.*, 25: 9-15.

TOMASINI, M. (1976) [Cardiac arrythmias due to intoxication by trichloroethylene ("th").] *Med. Lav.*, 67: 163-169 (in Italian).

TOMENIUS, L., HOLMA, B., EHRHER-SAMUEL, H., KYLIN, B., TEBROCK, O., & THOMASON, M. (1979) Effect of trichloroethylene on cilia activity in rabbit trachea. *Acta pharmacol. toxicol.*, 44: 65-70.

TORKELSON, T.R. & ROWE, V.K. (1981) Trichloroethylene. In: *Patty's industrial hygiene and toxicology*, 3rd (revised) ed., New York, John Wiley and Sons, Vol. 2B, pp. 3553-3586.

TRIEBIG, G., GOSSLER, K., & SCHALLER, K.-H. (1976) [Simple and reliable gas-chromatographic determination of trichloroethylene and its metabolites in blood and urine.] *Fresenius Z. Anal. Chem.*, 279: 115-116 (in German).

TRIEBIG, G., LEHRL, S., KINZEL, W., ERZIGHEIT, H., GALSTER, J.V., & SCHALLER, K.H. (1977a) [Psychopathometrical results of follow-up studies of trichloroethylene-exposed persons.] *Zbl. Bakt. Hyg. I. Abt. Orig. B.*, 164: 314-327 (in German with English summary).

TRIEBIG, G., SCHALLER, K.H., ERZIGHEIT, H., & VALENTIN, H. (1977b) Biochemical investigations and psychological studies of persons chronically exposed to trichloroethylene with regard to non-exposure intervals. *Int. Arch. occup. environ. Health*, 38: 149-162.

TRIEBIG, G., TRAUTNER, P., WELTLE, D., SAURE, E., & VALENTIN, H. (1982) Investigations on neurotoxicity of chemical substances at the workplace. III. Determination of the motor and sensory nerve conduction velocity in persons occupationally exposed to trichloroethylene. *Int. Arch. occup. environ. Health*, 51: 25-34.

TSURUTA, H. (1978) Percutaneous absorption of trichloroethylene in mice. *Ind. Health*, 16: 145-148.

TUCKER, A.N., SANDERS, V.M., BARNES, D.W., BRADSHAW, T.J., WHITE, K.L., Jr, SAIN, L.E., BORZELLACA, J.F., & MUNSON, A.E. (1982) Toxicology of trichloroethylene in the mouse. *Toxicol. appl. Pharmacol.*, 62: 351-357.

UEHLEKE, H., TABARELLI-POPLAWSKI, S., BONSE, G., & HENSCHLER, D. (1977) Spectral evidence for 2,2,3-trichloro-oxirane formation during microsomal trichloroethylen oxidation. *Arch. Toxicol.*, 37: 95-105.

US CFR (1976) Code of Federal Regulations. *21 CFR*, 8: 305.

US ITC (1980) *Production statistics, 1979*, Washington DC, International Trade Commisssion.

US NTP (1983) *Technical report on the carcinogenesis studies of trichloroethylene (without epichlorohydrin) in F344/n rats and BCF mice*, Research Triangle Park, NC, National Toxicology Program (NIH Publication No. 83-1979, NTP TR 243).

VALLE-RIESTRA, J.F. (1974) Food processing with chlorinated solvents. *Food Technol.*, 28: 25-32.

VAN DER HOEVEN, R., DROST, R.H., MAES, R.A.A., DOST, F., PLOMP, T.A., & PLOMP, G.J.J. (1979) Improved method for the electron-capture gas chromatographic determination of trichloroacetic acid in human serum. *J. Chromatogr.*, 164: 106-108.

VAN DUUREN, B.L. & BANERJEE, S. (1976) Covalent interaction of metabolites of the carcinogen trichloroethylene in rat hepatic microsomes. *Cancer Res.*, 36: 2419-2422.

VAN DUUREN, B.L., GOLDSCHMIDT, B.M., LOEWENGART, G., SMITH, A.C., MELCHIONNE, S., SELDMAN, I., & ROTH, D. (1979) Carcinogenicity of halogenated olefinic and aliphatic hydrocarbons in mice. *J. Natl Cancer Inst.*, 63: 1433-1439.

VAN DUUREN, B.L., KLINE, S.A., MELCHIONNE, S., & SEIDMAN, I. (1983) Chemical structure and carcinogenicity relationships of some chloroalkene oxides and their parent olefins. *Cancer Res.*, 43: 159-162.

VERNE, J., CECCALDI, P.F., HEBERT, S., & ROUX, J.M. (1959) Hepatic steatosis during intoxication by volatile organic compounds. IV. Biochemistry and histochemistry of fatty livers produced by trichloroethylene poisoning. *Pathol. Biol. (Paris)*, 7: 2311-2316.

VERNON, R.J. & FERGUSON, R.K. (1969) Effects of trichloroethylene on visual-motor performance. *Arch. environ. Health*, 18: 894-900.

VERNOT, E.H., MACEWEN, J.D., HAUN, C.C., & KINKEAD, E.R. (1977) Acute toxicity and skin corrosion data for some organic and inorganic compounds and aqueous solutions. *Toxicol. appl. Pharmacol.*, 42: 417-423.

VERSCHUEREN, K. (1977) *Handbook of environmental data on organic chemicals*, New York, Van Nostrand Reinhold Company, pp. 607-610.

VESTERBERG, O., GORCZAC, J., KRASTS, M. (1976) Exposure to trichloroethylene. II. Metabolites in blood and urine. *Scand. J. Work environ. Health*, 4: 212-219.

VISSARIONOVA, V.Ya., KALASHNIKOVA, V.P., SKIRKO, B.K., KARANTIROVA, O.V., & LAPRUN, I.B. (1975) [Comparative evaluation of indexes of rat liver functions in trichloroethylene poisoning.] *Gig. Tr. Prof. Zabol.*, No.11: 56-58 (in Russian).

VLACHOVA, D. (1957) Determination of trichloroethanol in the urine after exposure to trichloroethylene. *J. Hyg. Epidemiol. Microbiol. Immunol.*, 1-2: 225-229.

VON OETTINGEN, W.F. (1955) *The halogenated aliphatic, olifinic, cyclic, aromatic, and aliphatic-aromatic hydrocarbons including the halogenated insecticides: their toxicity and potential dangers*, Washington DC, US Government Printing Office.

WADE, A., ed. (1977) *Martindale: the extra pharmacopoeia*, 27th ed., London, The Pharmaceutical Press, pp. 714-715.

WAHLBERG, J.E. (1984) Erythema-inducing effects of solvents following epicutaneous administration to man: studied by Laser Doppler flowmetry. *Scand. J. Work environ. Health*, 10: 159-162.

WAHLBERG, J.E. & BOMAN, A. (1979) Comparitive percutaneous toxicity of ten industrial solvents in the guinea-pig. *Scand. J. Work environ. Health*, 5: 345-351.

WAKEHAM, S.G., DAVIS, A.C., & GOODWIN, J.T. (1982) Biogeochemistry of volatile organic compounds in marine experimental ecosystems and the estuarine environment: initial results. In: Grice, G.D. & Reeve, M.R., ed. *Marine Mesocosms*, New York, Springer.

WASKELL, L. (1978) A study of the mutagenicity of anesthetics and their metabolites. *Mutat. Res.*, 57: 141-153.

WATERS, E.M., GERSTNER, H.B., & HUFF, J.E. (1977) Trichloroethylene. I. An overview. *J. Toxicol. environ. Health*, 2: 671-707.

WEAST, R.C., ed. (1980) *Handbook of chemistry and physics*, 61st ed., Boca Raton, Florida, CRC Press, Inc., p. C-317.

WEICHARDT, H. & BARDODEJ, Z. (1970) Determination of trichloroacetic acid and trichloroethanol in the urine of trichloroethylene workers. *Zentralbl. Arbeitsmed. Arbeitsschutz.*, 20: 219-221.

WELLS, J.C.D. (1982) Abuse of trichloroethylene by oral self-administration. *Anaesthesia*, 37: 440-441.

WETTERHAHN, J. (1972) Eye make-up. In: Balsam, M. & Sagarin, E., ed. *Cosmetics science and technology*, New York, John Wiley and Sons, Inc., pp. 419.

WHELAN, J.K., BLANCHETTE, M.A., & HUNT, J.M. (1983) Volatile C_1-C_7 organic compounds in an anoxic sediment core from the Pettaquamscutt River (Rhode Island, USA). *Org. Geochem.*, 5: 29-33.

WHITE, A.E., TAKEHISA, S., EGER, E.I., WOLFF, S., & STEVENS, W.C. (1979) Sister-chromatid exchanges induced by inhaled anaesthetics. *Anaesthesiology*, 50: 426.

WHITE, J.F. & CARLSON, G.P. (1979) Influence of alterations in drug metabolism on spontaneous and epinephrine-induced cardiac arrhythmias in animals exposed to trichloroethylene. *Toxicol. appl. Pharmacol.*, 47: 515-527.

WHITE, J.F. & CARLSON, G.P. (1981a) Epinephrine-induced cardiac arrhythmias in rabbits exposed to trichloroethylene: potentiation by ethanol. *Toxicol. appl. Pharmacol.*, 60: 466-471.

WHITE, J.F. & CARLSON, G.P. (1981b) Epinephrine-induced cardiac arrhythmias in rabbits exposed to trichloroethylene: role of trichloroethylene metabolites. *Toxicol. appl. Pharmacol.*, 60: 458-465.

WHITE, J.F. & CARLSON, G.P. (1982) Epinephrine-induced cardiac arrhythmias in rabbits exposed to trichloroethylene: potentiation by caffeine. *Fundam. appl. Toxicol.*, 2: 125-129.

WHO (1984) *Guidelines for drinking-water quality. I. Recommendations*, Geneva, World Health Organization, 66 pp.

WHO STUDY GROUP ON RECOMMENDED HEALTH-BASED LIMITS IN OCCUPATIONAL EXPOSURE TO SELECTED ORGANIC SOLVENTS (1981) *Recommended health-based limits in occupational exposure to selected organic solvents*, Geneva, World Health Organization, 84 pp (Technical Report Series No. 664).

WIECKO, W. (1966) [Oral poisoning with trichloroethylene.] *Wiad. Lek.*, 19: 1117-1118 (in Polish).

WINDHOLZ, M., BUDAVARI, S., STROUMTSOS, L.Y., & NOETHER FERTIG, M., ed. (1976) *The Merck index*, 9th ed., Rahway, New Jersey, Merck & Co., Inc., p. 1238.

WINNEKE, G. (1982) Acute behavioural effects of exposure to some organic solvents: psychophysiological aspects. *Acta neurol. scand.*,

66(suppl. 92): 117-129.

WITHEY, J.R. & COLLINS, B.T. (1980) Chlorinated aliphatic hydrocarbons used in the foods industry: the comparative pharmaocokinetics of methylene chloride, 1,2-dichloroethane, chloroform, and trichloroethylene after iv administration in the rat. *J. environ. Pathol. Toxicol.*, 3: 313-332.

WIRTSCHAFTER, Z.T. & CRONYN, M.W. (1964) Relative hepatotoxicity: pentane, trichloroethylene, benzene, carbon tetrachloride. *Arch. environ. Health*, 9: 180-185.

ZAFFIRI, O., RUGGERINI, R., & FRANCESCATO, F. (1968) Anesthetics and adrenergic receptors. *Minerva Anestesiol.*, 34: 556-560.

ZENICK, H., BLACKBURN, K., HOPE, E., RICHDALE, N., & SMITH, M.K. (1984) Effects of trichloroethylene exposure on male reproductive function in rats. *Toxicology*, 31: 237-250.

ZIGLIO, G. (1979) [Gas-chromatographic determination of blood trichoroacetic acid concentration in subjects not occupationally exposed to trichloro- and tetrachloroethylene.] *Ig. Mod.*, 72: 876-900 (in Italian).

ZIGLIO, G., FARA, G.M., BELTRAMELLI, G., & PREGLIASCO, F. (1983) Human environmental exposure to trichloro- and tetrachloroethylene from water and air in Milan, Italy. *Arch. environ. contam. Toxicol.*, 12: 57-64.

APPENDIX I

Predicting the Equilibrium Distribution of Trichloroethylene

On the basis of the physical and chemical data summarized in Table 1 of this document, it is possible to predict the approximate equilibrium distribution of trichloroethylene in major environmental "compartments". The models used have been described by Mackay & Paterson (1981). Essentially, these use physical and chemical data and realistic compartment volumes in a "model world" to calculate fugacity capacities and to predict the partitioning of a chemical between various compartments. The following compartment volumes are assumed (Neely & Mackay, 1982) (Table I.1) in an environment, 1 km^2 in area.

Table I.1 Compartment volumes for trichloroethylene

Atmosphere	6×10^9 m^3	1 km^2 area x 6 km height
Water	7×10^6 m^3	70% area x 10 m depth
Soil	4.5×10^4 m^3	30% area x 15 cm depth
Sediment	2.1×10^4 m^3	70% water x 3 cm depth
Suspended aquatic matter	35.0 m^3	water volume x 5 ppm
Aquatic biota	7.0 m^3	water volume x 1 ppm

As an example, the input of trichloroethylene is assumed to be 1000 moles/km^2 per year, based on an annual production capacity of 4×10^5 tonnes per year and an approximate land area of 10^6 km^2. This leads to an actual production of approximately 3000 moles/km^2, but it is assumed that land represents one-third of the total area.

Physical and chemical data for trichloroethylene are given in Table 1 (main text). Environmental temperature is assumed to be 25 °C (atmosphere) or 15 °C (land and water).

Additional compartments at appropriate volumes can be added with appropriate characteristics, e.g., atmospheric particulates at 2 $\mu g/m^3$ (Seba & Prospero, 1971) or terrestrial biota at approximately 1 kg/m^2 (Odum, 1971).

The model predicts the distributions shown in Table I.2. These are remarkably close to the distributions observed in most compartments and, furthermore, the ratios of concentrations predicted to occur between compartments are remarkably similar to those that are observed. The predicted and observed distributions of trichloroethylene are not surprising; as noted in section 4.2.1, its high vapour pressure would be expected to lead to high atmospheric concentrations, and this would offset the tendency of

high water solubility and low partition coefficients (themselves related) to lead to high water, biota, or sediment concentrations, through either partition or adsorption.

Table I.2 Predicted and observed distribution of trichloroethylene (TCE) in the environment using compartment volumes and approaches described in text

Compartment	Predicted % TCE	Predicted TCE Concentration	Observed TCE concentration (see text)
Air	99.7	22 µg/m3	0.01 - 10 µg/m^3
Water	0.3	0.06 µg/litre	0.01 - 100 µg/litre
Sediment	< 0.01	0.4 µg/kg	1 - 10 µg/kg
Aquatic	< 0.01	0.7 ng/g	1 - 100 ng/g

The value of a model like this is not that it predicts the "correct" distribution, but that it illustrates and identifies the compartments that are important as reservoirs or as sites of degradation of chemicals. In the present case, the size of the atmospheric compartment and the high vapour pressure of trichloroethylene are such that the bulk of trichloroethylene released into the environment should be found in the atmosphere. Therefore, atmospheric degradation processes should be important in the eventual degradation of trichloroethylene.

This approach can be refined further by considering not only the distribution at equilibrium (assuming no degradation), but adding rates of degradation. For these purposes, we have assumed the rates of degradation in the appropriate compartment, shown in section 4.2.2. Although absolute trichloroethylene concentrations are predicted by this model to be appreciably lower in each compartment than those shown in Table I.2, the ratios of concentrations between compartments do not change appreciably (Table I.3). However, the important point is that since most of the trichloroethylene is predicted to occur in the atmosphere, and since atmospheric degradation rates are similar to or faster than those in other compartments, most of the degradation should take place in the atmosphere (Table I.3). Washout or fallout of atmospheric particulate material is not likely to be an important process; there seems to be little tendency for trichloroethylene to sorb to particles, because of its low K_{oc}, and residence times of such particles are long compared to the the expected rate of degradation by OH radicals. Total environmental lifetime is controlled by the atmospheric degradation rate, and should, therefore, be about 10 - 11 days. Degradation in sediments and/or biota probably contribute in only a very minor way to overall degradation.

Table I.3 Predicted distribution and compartmental degradation rates for trichloroethylene (TCE) as % of total environmental degradation using compartment volumes and degradation rates in text

Compartment	Predicted TCE concentration	Total degradation (%)
Air	0.6 µg/m^3	99.9
Water	0.002 µg/litre	0.08

Sediment	0.01 µg/kg	< 0.01
Aquatic biota	0.02 ng/g	< 0.01

REFERENCES TO APPENDIX I

MACKAY, D. & PATERSON, S. (1981) Calculating fugacity. *Environ. Sci. Technol.*, 15: 1006-1014.

NEELY, W.B. & MACKAY, D. (1982) In: Dickson, K.L., Maki, A.W., & Cairns, J., *Modelling the fate of chemicals in the aquatic environment*, Ann Arbor, Michigan, Ann Arbor Science Publications.

ODUM, E.P. (1971) *Fundamentals of ecology*, 3rd ed., Philadelphia, Pennsylvania, W.B. Saunders Co.

SEBA, D.B. & PROSPERO, J.M. (1971) Pesticides in the lower atmosphere of the northern equatorial Atlantic Ocean. *Atmos. Environ.*, 5: 1043-1050.

See Also:
Toxicological Abbreviations
Trichloroethylene (ICSC)
Trichloroethylene (WHO Food Additives Series 10)
TRICHLOROETHYLENE (JECFA Evaluation)
Trichloroethylene (FAO/PL:1968/M/9/1)
Trichloroethylene (IARC Summary & Evaluation, Volume 63, 1995)

Attachment B

Indoor Air Analytical Report
21 April 2010

ANALYTICAL REPORT

Lab Number:	L1005822
Client:	EA Engineering, Science and Tech 2350 Post Road Warwick, RI 02886
ATTN:	Frank Postma
Phone:	(401) 736-3440
Project Name:	ALVAREZ HIGH SCHOOL
Project Number:	14687.01
Report Date:	04/29/10

Certifications & Approvals: MA (M-MA030), NY (11627), CT (PH-0141), NH (2206), NJ (MA015), RI (LAO00299), ME (MA0030), PA (Registration #68-02089), LA NELAC (03090), FL NELAC (E87814), US Army Corps of Engineers.

320 Forbes Boulevard, Mansfield, MA 02048-1806
508-822-9300 (Fax) 508-822-3288 800-624-9220 - www.alphalab.com

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01

Lab Number: L1005822
Report Date: 04/29/10

Alpha Sample ID	Client ID	Sample Location	Collection Date/Time
L1005822-01	GYMNASIUM	PROVIDENCE, RI	04/21/10 09:50
L1005822-02	CAFETERIA	PROVIDENCE, RI	04/21/10 09:51
L1005822-03	KITCHEN STORAGE RM	PROVIDENCE, RI	04/21/10 10:13
L1005822-04	ELEVATOR HALLWAY	PROVIDENCE, RI	04/21/10 09:53
L1005822-05	RM 145	PROVIDENCE, RI	04/21/10 09:58
L1005822-06	RM 152	PROVIDENCE, RI	04/21/10 09:58
L1005822-07	RM 118	PROVIDENCE, RI	04/21/10 10:03
L1005822-08	RM 110	PROVIDENCE, RI	04/21/10 10:05
L1005822-09	AMBIENT	PROVIDENCE, RI	04/21/10 11:40

Project Name:	ALVAREZ HIGH SCHOOL	**Lab Number:**	L1005822
Project Number:	14687.01	**Report Date:**	04/29/10

Case Narrative

The samples were received in accordance with the Chain of Custody and no significant deviations were encountered during the preparation or analysis unless otherwise noted. Sample Receipt, Container Information, and the Chain of Custody are located at the back of the report.

Results contained within this report relate only to the samples submitted under this Alpha Lab Number and meet all of the requirements of NELAC, for all NELAC accredited parameters. The data presented in this report is organized by parameter (i.e. VOC, SVOC, etc.). Sample specific Quality Control data (i.e. Surrogate Spike Recovery) is reported at the end of the target analyte list for each individual sample, followed by the Laboratory Batch Quality Control at the end of each parameter. If a sample was re-analyzed or re-extracted due to a required quality control corrective action and if both sets of data are reported, the Laboratory ID of the re-analysis or re-extraction is designated with an "R" or "RE", respectively. When multiple Batch Quality Control elements are reported (e.g. more than one LCS), the associated samples for each element are noted in the grey shaded header line of each data table. Any Laboratory Batch, Sample Specific % recovery or RPD value that is outside the listed Acceptance Criteria is bolded in the report. Definitions of all data qualifiers and acronyms used in this report are provided in the Glossary located at the back of the report.

Please see the associated ADEx data file for a comparison of laboratory reporting limits that were achieved with the regulatory Numerical Standards requested on the Chain of Custody.

For additional information, please contact Client Services at 800-624-9220.

Volatile Organics in Air (SIM)

L1005822-01, -02, -04 through -08: results for Chloromethane should be considered estimated due to co-elution with a non-target peak.

The WG409885-3 LCS recoveries for Benzene (69%) and trans-1,3-Dichloropropene (64%) are outside the 70%-130% acceptance limit. The LCS was within overall method allowances, therefore the analysis proceeded.

I, the undersigned, attest under the pains and penalties of perjury that, to the best of my knowledge and belief and based upon my personal inquiry of those responsible for providing the information contained in this analytical report, such information is accurate and complete. This certificate of analysis is not complete unless this page accompanies any and all pages of this report.

Authorized Signature: Kathleen M. O'Brien Kathleen O'Brien

Title: Technical Director/Representative Date: 04/29/10

AIR

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005822
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005822-01
Client ID: GYMNASIUM
Sample Location: PROVIDENCE, RI
Matrix: Air
Anaytical Method: 48,TO-15-SIM
Analytical Date: 04/27/10 00:18
Analyst: RY

Date Collected: 04/21/10 09:50
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
Dichlorodifluoromethane	0.509	0.050	2.52	0.247		1
Chloromethane	ND	0.500	ND	2.44		1
Vinyl chloride	ND	0.020	ND	0.051		1
Chloroethane	ND	0.020	ND	0.053		1
Acetone	9.76	2.00	23.2	4.75		1
Trichlorofluoromethane	0.453	0.050	2.54	0.281		1
Acrylonitrile	ND	0.500	ND	1.08		1
1,1-Dichloroethene	ND	0.020	ND	0.079		1
Methylene chloride	ND	0.800	ND	1.74		1
trans-1,2-Dichloroethene	ND	0.020	ND	0.079		1
1,1-Dichloroethane	ND	0.020	ND	0.081		1
Methyl tert butyl ether	ND	0.020	ND	0.072		1
2-Butanone	0.941	0.500	2.77	1.47		1
cis-1,2-Dichloroethene	ND	0.020	ND	0.079		1
Chloroform	0.042	0.020	0.205	0.098		1
1,2-Dichloroethane	ND	0.020	ND	0.081		1
1,1,1-Trichloroethane	ND	0.020	ND	0.109		1
Benzene	0.339	0.100	1.08	0.319		1
Carbon tetrachloride	0.089	0.020	0.559	0.126		1
1,2-Dichloropropane	ND	0.020	ND	0.092		1
Bromodichloromethane	ND	0.020	ND	0.134		1
Trichloroethene	0.052	0.020	0.279	0.107		1
cis-1,3-Dichloropropene	ND	0.020	ND	0.091		1
4-Methyl-2-pentanone	ND	0.500	ND	2.05		1
trans-1,3-Dichloropropene	ND	0.020	ND	0.091		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005822
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005822-01
Client ID: GYMNASIUM
Sample Location: PROVIDENCE, RI
Date Collected: 04/21/10 09:50
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
1,1,2-Trichloroethane	ND	0.020	ND	0.109		1
Toluene	1.39	0.020	5.22	0.075		1
Dibromochloromethane	ND	0.020	ND	0.096		1
1,2-Dibromoethane	ND	0.020	ND	0.154		1
Tetrachloroethene	0.065	0.020	0.440	0.136		1
1,1,1,2-Tetrachloroethane	ND	0.020	ND	0.137		1
Chlorobenzene	ND	0.020	ND	0.092		1
Ethylbenzene	0.294	0.020	1.28	0.087		1
p/m-Xylene	1.01	0.040	4.38	0.174		1
Bromoform	ND	0.020	ND	0.206		1
Styrene	0.068	0.020	0.289	0.085		1
1,1,2,2-Tetrachloroethane	ND	0.020	ND	0.137		1
o-Xylene	0.290	0.020	1.26	0.087		1
Isopropylbenzene	ND	0.500	ND	2.46		1
1,3,5-Trimethybenzene	0.427	0.020	2.10	0.098		1
1,2,4-Trimethylbenzene	0.934	0.020	4.59	0.098		1
1,3-Dichlorobenzene	ND	0.020	ND	0.120		1
1,4-Dichlorobenzene	ND	0.020	ND	0.120		1
sec-Butylbenzene	ND	0.500	ND	2.74		1
p-Isopropyltoluene	ND	0.500	ND	2.74		1
1,2-Dichlorobenzene	ND	0.020	ND	0.120		1
n-Butylbenzene	ND	0.500	ND	2.74		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005822
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005822-02
Client ID: CAFETERIA
Sample Location: PROVIDENCE, RI
Matrix: Air
Anaytical Method: 48,TO-15-SIM
Analytical Date: 04/26/10 19:16
Analyst: RY

Date Collected: 04/21/10 09:51
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
Dichlorodifluoromethane	0.470	0.050	2.32	0.247		1
Chloromethane	ND	0.500	ND	2.44		1
Vinyl chloride	ND	0.020	ND	0.051		1
Chloroethane	ND	0.020	ND	0.053		1
Acetone	9.26	2.00	22.0	4.75		1
Trichlorofluoromethane	0.673	0.050	3.78	0.281		1
Acrylonitrile	ND	0.500	ND	1.08		1
1,1-Dichloroethene	ND	0.020	ND	0.079		1
Methylene chloride	ND	0.800	ND	1.74		1
trans-1,2-Dichloroethene	ND	0.020	ND	0.079		1
1,1-Dichloroethane	ND	0.020	ND	0.081		1
Methyl tert butyl ether	ND	0.020	ND	0.072		1
2-Butanone	ND	0.500	ND	1.47		1
cis-1,2-Dichloroethene	0.197	0.020	0.780	0.079		1
Chloroform	0.162	0.020	0.790	0.098		1
1,2-Dichloroethane	ND	0.020	ND	0.081		1
1,1,1-Trichloroethane	ND	0.020	ND	0.109		1
Benzene	0.413	0.100	1 32	0.319		1
Carbon tetrachloride	0.087	0.020	0.547	0.126		1
1,2-Dichloropropane	ND	0.020	ND	0.092		1
Bromodichloromethane	ND	0.020	ND	0.134		1
Trichloroethene	0.108	0.020	0.580	0.107		1
cis-1,3-Dichloropropene	ND	0.020	ND	0.091		1
4-Methyl-2-pentanone	ND	0.500	ND	2.05		1
trans-1,3-Dichloropropene	ND	0.020	ND	0.091		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005822
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005822-02
Client ID: CAFETERIA
Sample Location: PROVIDENCE, RI
Date Collected: 04/21/10 09:51
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
1,1,2-Trichloroethane	ND	0.020	ND	0.109		1
Toluene	2.29	0.020	8.61	0.075		1
Dibromochloromethane	ND	0.020	ND	0.096		1
1,2-Dibromoethane	ND	0.020	ND	0.154		1
Tetrachloroethene	0.111	0.020	0.752	0.136		1
1,1,1,2-Tetrachloroethane	ND	0.020	ND	0.137		1
Chlorobenzene	ND	0.020	ND	0.092		1
Ethylbenzene	0.165	0.020	0.716	0.087		1
p/m-Xylene	0.460	0.040	2.00	0.174		1
Bromoform	ND	0.020	ND	0.206		1
Styrene	0.048	0.020	0.204	0.085		1
1,1,2,2-Tetrachloroethane	ND	0.020	ND	0.137		1
o-Xylene	0.158	0.020	0.686	0.087		1
Isopropylbenzene	ND	0.500	ND	2.46		1
1,3,5-Trimethybenzene	0.075	0.020	0.368	0.098		1
1,2,4-Trimethylbenzene	0.172	0.020	0.845	0.098		1
1,3-Dichlorobenzene	ND	0.020	ND	0.120		1
1,4-Dichlorobenzene	0.030	0.020	0.180	0.120		1
sec-Butylbenzene	ND	0.500	ND	2.74		1
p-Isopropyltoluene	ND	0.500	ND	2.74		1
1,2-Dichlorobenzene	ND	0.020	ND	0.120		1
n-Butylbenzene	ND	0.500	ND	2.74		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005822
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005822-03
Client ID: KITCHEN STORAGE RM
Sample Location: PROVIDENCE, RI
Matrix: Air
Anaytical Method: 48,TO-15-SIM
Analytical Date: 04/26/10 19:54
Analyst: RY

Date Collected: 04/21/10 10:13
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
Dichlorodifluoromethane	0.473	0.050	2.34	0.247		1
Chloromethane	0.523	0.500	2.55	2.44		1
Vinyl chloride	ND	0.020	ND	0.051		1
Chloroethane	ND	0.020	ND	0.053		1
Acetone	11.2	2.00	26.7	4.75		1
Trichlorofluoromethane	0.743	0.050	4.17	0.281		1
Acrylonitrile	ND	0.500	ND	1.08		1
1,1-Dichloroethene	ND	0.020	ND	0.079		1
Methylene chloride	1.56	0.800	5.41	1.74		1
trans-1,2-Dichloroethene	ND	0.020	ND	0.079		1
1,1-Dichloroethane	ND	0.020	ND	0.081		1
Methyl tert butyl ether	ND	0.020	ND	0.072		1
2-Butanone	0.627	0.500	1.85	1.47		1
cis-1,2-Dichloroethene	ND	0.020	ND	0.079		1
Chloroform	0.032	0.020	0.156	0.098		1
1,2-Dichloroethane	ND	0.020	ND	0.081		1
1,1,1-Trichloroethane	ND	0.020	ND	0.109		1
Benzene	0.321	0.100	1.02	0.319		1
Carbon tetrachloride	0.078	0.020	0.490	0.126		1
1,2-Dichloropropane	ND	0.020	ND	0.092		1
Bromodichloromethane	ND	0.020	ND	0.134		1
Trichloroethene	0.046	0.020	0.247	0.107		1
cis-1,3-Dichloropropene	ND	0.020	ND	0.091		1
4-Methyl-2-pentanone	ND	0.500	ND	2.05		1
trans-1,3-Dichloropropene	ND	0.020	ND	0.091		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005822
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005822-03
Client ID: KITCHEN STORAGE RM
Sample Location: PROVIDENCE, RI
Date Collected: 04/21/10 10:13
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
1,1,2-Trichloroethane	ND	0.020	ND	0.109		1
Toluene	1.27	0.020	4.77	0.075		1
Dibromochloromethane	ND	0.020	ND	0.096		1
1,2-Dibromoethane	ND	0.020	ND	0.154		1
Tetrachloroethene	0.094	0.020	0.637	0.136		1
1,1,1,2-Tetrachloroethane	ND	0.020	ND	0.137		1
Chlorobenzene	ND	0.020	ND	0.092		1
Ethylbenzene	0.108	0.020	0.468	0.087		1
p/m-Xylene	0.277	0.040	1.20	0.174		1
Bromoform	ND	0.020	ND	0.206		1
Styrene	0.066	0.020	0.281	0.085		1
1,1,2,2-Tetrachloroethane	ND	0.020	ND	0.137		1
o-Xylene	0.098	0.020	0.425	0.087		1
Isopropylbenzene	ND	0.500	ND	2.46		1
1,3,5-Trimethybenzene	0.024	0.020	0.118	0.098		1
1,2,4-Trimethylbenzene	0.080	0.020	0.393	0.098		1
1,3-Dichlorobenzene	ND	0.020	ND	0.120		1
1,4-Dichlorobenzene	ND	0.020	ND	0.120		1
sec-Butylbenzene	ND	0.500	ND	2.74		1
p-Isopropyltoluene	ND	0.500	ND	2.74		1
1,2-Dichlorobenzene	ND	0.020	ND	0.120		1
n-Butylbenzene	ND	0.500	ND	2.74		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005822
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005822-04
Client ID: ELEVATOR HALLWAY
Sample Location: PROVIDENCE, RI
Matrix: Air
Anaytical Method: 48,TO-15-SIM
Analytical Date: 04/26/10 20:32
Analyst: RY

Date Collected: 04/21/10 09:53
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
Dichlorodifluoromethane	0.472	0.050	2.33	0.247		1
Chloromethane	ND	0.500	ND	2.44		1
Vinyl chloride	ND	0.020	ND	0.051		1
Chloroethane	ND	0.020	ND	0.053		1
Acetone	9.78	2.00	23.2	4.75		1
Trichlorofluoromethane	0.571	0.050	3.20	0.281		1
Acrylonitrile	ND	0.500	ND	1.08		1
1,1-Dichloroethene	ND	0.020	ND	0.079		1
Methylene chloride	ND	0.800	ND	1.74		1
trans-1,2-Dichloroethene	ND	0.020	ND	0.079		1
1,1-Dichloroethane	ND	0.020	ND	0.081		1
Methyl tert butyl ether	ND	0.020	ND	0.072		1
2-Butanone	0.541	0.500	1.59	1.47		1
cis-1,2-Dichloroethene	ND	0.020	ND	0.079		1
Chloroform	0.158	0.020	0.771	0.098		1
1,2-Dichloroethane	ND	0.020	ND	0.081		1
1,1,1-Trichloroethane	ND	0.020	ND	0.109		1
Benzene	0.431	0.100	1.38	0.319		1
Carbon tetrachloride	0.077	0.020	0.484	0.126		1
1,2-Dichloropropane	ND	0.020	ND	0.092		1
Bromodichloromethane	ND	0.020	ND	0.134		1
Trichloroethene	0.094	0.020	0.505	0.107		1
cis-1,3-Dichloropropene	ND	0.020	ND	0.091		1
4-Methyl-2-pentanone	ND	0.500	ND	2.05		1
trans-1,3-Dichloropropene	ND	0.020	ND	0.091		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005822
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005822-04
Client ID: ELEVATOR HALLWAY
Sample Location: PROVIDENCE, RI
Date Collected: 04/21/10 09:53
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
1,1,2-Trichloroethane	ND	0.020	ND	0.109		1
Toluene	1.97	0.020	7.43	0.075		1
Dibromochloromethane	ND	0.020	ND	0.096		1
1,2-Dibromoethane	ND	0.020	ND	0.154		1
Tetrachloroethene	0.096	0.020	0.650	0.136		1
1,1,1,2-Tetrachloroethane	ND	0.020	ND	0.137		1
Chlorobenzene	ND	0.020	ND	0.092		1
Ethylbenzene	0.141	0.020	0.612	0.087		1
p/m-Xylene	0.371	0.040	1.61	0.174		1
Bromoform	ND	0.020	ND	0.206		1
Styrene	0.044	0.020	0.187	0.085		1
1,1,2,2-Tetrachloroethane	ND	0.020	ND	0.137		1
o-Xylene	0.133	0.020	0.577	0.087		1
Isopropylbenzene	ND	0.500	ND	2.46		1
1,3,5-Trimethybenzene	0.053	0.020	0.260	0.098		1
1,2,4-Trimethylbenzene	0.131	0.020	0.643	0.098		1
1,3-Dichlorobenzene	ND	0.020	ND	0.120		1
1,4-Dichlorobenzene	0.026	0.020	0.156	0.120		1
sec-Butylbenzene	ND	0.500	ND	2.74		1
p-Isopropyltoluene	ND	0.500	ND	2.74		1
1,2-Dichlorobenzene	ND	0.020	ND	0.120		1
n-Butylbenzene	ND	0.500	ND	2.74		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005822
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005822-05
Client ID: RM 145
Sample Location: PROVIDENCE, RI
Matrix: Air
Anaytical Method: 48,TO-15-SIM
Analytical Date: 04/26/10 21:46
Analyst: RY

Date Collected: 04/21/10 09:58
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
Dichlorodifluoromethane	0.470	0.050	2.32	0.247		1
Chloromethane	0.516	0.500	2.52	2.44		1
Vinyl chloride	ND	0.020	ND	0.051		1
Chloroethane	ND	0.020	ND	0.053		1
Acetone	9.20	2.00	21.8	4.75		1
Trichlorofluoromethane	0.623	0.050	3.50	0.281		1
Acrylonitrile	ND	0.500	ND	1.08		1
1,1-Dichloroethene	ND	0.020	ND	0.079		1
Methylene chloride	ND	0.800	ND	1.74		1
trans-1,2-Dichloroethene	ND	0.020	ND	0.079		1
1,1-Dichloroethane	ND	0.020	ND	0.081		1
Methyl tert butyl ether	ND	0.020	ND	0.072		1
2-Butanone	ND	0.500	ND	1.47		1
cis-1,2-Dichloroethene	ND	0.020	ND	0.079		1
Chloroform	0.030	0.020	0.146	0.098		1
1,2-Dichloroethane	ND	0.020	ND	0.081		1
1,1,1-Trichloroethane	ND	0.020	ND	0.109		1
Benzene	0.386	0.100	1.23	0.319		1
Carbon tetrachloride	0.080	0.020	0.503	0.126		1
1,2-Dichloropropane	ND	0.020	ND	0.092		1
Bromodichloromethane	ND	0.020	ND	0.134		1
Trichloroethene	0.078	0.020	0.419	0.107		1
cis-1,3-Dichloropropene	ND	0.020	ND	0.091		1
4-Methyl-2-pentanone	ND	0.500	ND	2.05		1
trans-1,3-Dichloropropene	ND	0.020	ND	0.091		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005822
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005822-05
Client ID: RM 145
Sample Location: PROVIDENCE, RI
Date Collected: 04/21/10 09:58
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
1,1,2-Trichloroethane	ND	0.020	ND	0.109		1
Toluene	1.07	0.020	4.03	0.075		1
Dibromochloromethane	ND	0.020	ND	0.096		1
1,2-Dibromoethane	ND	0.020	ND	0.154		1
Tetrachloroethene	0.060	0.020	0.407	0.136		1
1,1,1,2-Tetrachloroethane	ND	0.020	ND	0.137		1
Chlorobenzene	ND	0.020	ND	0.092		1
Ethylbenzene	0.125	0.020	0.542	0.087		1
p/m-Xylene	0.330	0.040	1.43	0.174		1
Bromoform	ND	0.020	ND	0.206		1
Styrene	0.034	0.020	0.145	0.085		1
1,1,2,2-Tetrachloroethane	ND	0.020	ND	0.137		1
o-Xylene	0.130	0.020	0.564	0.087		1
Isopropylbenzene	ND	0.500	ND	2.46		1
1,3,5-Trimethybenzene	0.033	0.020	0.162	0.098		1
1,2,4-Trimethylbenzene	0.087	0.020	0.427	0.098		1
1,3-Dichlorobenzene	ND	0.020	ND	0.120		1
1,4-Dichlorobenzene	0.021	0.020	0.126	0.120		1
sec-Butylbenzene	ND	0.500	ND	2.74		1
p-Isopropyltoluene	ND	0.500	ND	2.74		1
1,2-Dichlorobenzene	ND	0.020	ND	0.120		1
n-Butylbenzene	ND	0.500	ND	2.74		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005822
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005822-06
Client ID: RM 152
Sample Location: PROVIDENCE, RI
Matrix: Air
Anaytical Method: 48,TO-15-SIM
Analytical Date: 04/26/10 22:24
Analyst: RY

Date Collected: 04/21/10 09:58
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
Dichlorodifluoromethane	0.471	0.050	2.33	0.247		1
Chloromethane	ND	0.500	ND	2.44		1
Vinyl chloride	ND	0.020	ND	0.051		1
Chloroethane	ND	0.020	ND	0.053		1
Acetone	8.66	2.00	20.5	4.75		1
Trichlorofluoromethane	0.569	0.050	3.19	0.281		1
Acrylonitrile	ND	0.500	ND	1.08		1
1,1-Dichloroethene	ND	0.020	ND	0.079		1
Methylene chloride	ND	0.800	ND	1.74		1
trans-1,2-Dichloroethene	ND	0.020	ND	0.079		1
1,1-Dichloroethane	ND	0.020	ND	0.081		1
Methyl tert butyl ether	ND	0.020	ND	0.072		1
2-Butanone	ND	0.500	ND	1.47		1
cis-1,2-Dichloroethene	ND	0.020	ND	0.079		1
Chloroform	0.046	0.020	0.224	0.098		1
1,2-Dichloroethane	0.040	0.020	0.162	0.081		1
1,1,1-Trichloroethane	ND	0.020	ND	0.109		1
Benzene	0.389	0.100	1.24	0.319		1
Carbon tetrachloride	0.078	0.020	0.490	0.126		1
1,2-Dichloropropane	ND	0.020	ND	0.092		1
Bromodichloromethane	ND	0.020	ND	0.134		1
Trichloroethene	0.085	0.020	0.456	0.107		1
cis-1,3-Dichloropropene	ND	0.020	ND	0.091		1
4-Methyl-2-pentanone	0.549	0.500	2.25	2.05		1
trans-1,3-Dichloropropene	ND	0.020	ND	0.091		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005822
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005822-06
Client ID: RM 152
Sample Location: PROVIDENCE, RI
Date Collected: 04/21/10 09:58
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
1,1,2-Trichloroethane	ND	0.020	ND	0.109		1
Toluene	1.04	0.020	3.90	0.075		1
Dibromochloromethane	ND	0.020	ND	0.096		1
1,2-Dibromoethane	ND	0.020	ND	0.154		1
Tetrachloroethene	0.070	0.020	0.474	0.136		1
1,1,1,2-Tetrachloroethane	ND	0.020	ND	0.137		1
Chlorobenzene	ND	0.020	ND	0.092		1
Ethylbenzene	0.124	0.020	0.538	0.087		1
p/m-Xylene	0.311	0.040	1.35	0.174		1
Bromoform	ND	0.020	ND	0.206		1
Styrene	0.033	0.020	0.140	0.085		1
1,1,2,2-Tetrachloroethane	ND	0.020	ND	0.137		1
o-Xylene	0.111	0.020	0.482	0.087		1
Isopropylbenzene	ND	0.500	ND	2.46		1
1,3,5-Trimethybenzene	0.036	0.020	0.177	0.098		1
1,2,4-Trimethylbenzene	0.097	0.020	0.476	0.098		1
1,3-Dichlorobenzene	ND	0.020	ND	0.120		1
1,4-Dichlorobenzene	0.021	0.020	0.126	0.120		1
sec-Butylbenzene	ND	0.500	ND	2.74		1
p-Isopropyltoluene	ND	0.500	ND	2.74		1
1,2-Dichlorobenzene	ND	0.020	ND	0.120		1
n-Butylbenzene	ND	0.500	ND	2.74		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005822
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005822-07
Client ID: RM 118
Sample Location: PROVIDENCE, RI
Matrix: Air
Anaytical Method: 48,TO-15-SIM
Analytical Date: 04/26/10 23:02
Analyst: RY

Date Collected: 04/21/10 10:03
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
Dichlorodifluoromethane	0.472	0.050	2.33	0.247		1
Chloromethane	ND	0.500	ND	2.44		1
Vinyl chloride	ND	0.020	ND	0.051		1
Chloroethane	ND	0.020	ND	0.053		1
Acetone	8.13	2.00	19.3	4.75		1
Trichlorofluoromethane	0.623	0.050	3.50	0.281		1
Acrylonitrile	ND	0.500	ND	1.08		1
1,1-Dichloroethene	ND	0.020	ND	0.079		1
Methylene chloride	ND	0.800	ND	1.74		1
trans-1,2-Dichloroethene	ND	0.020	ND	0.079		1
1,1-Dichloroethane	ND	0.020	ND	0.081		1
Methyl tert butyl ether	ND	0.020	ND	0.072		1
2-Butanone	0.504	0.500	1.48	1.47		1
cis-1,2-Dichloroethene	ND	0.020	ND	0.079		1
Chloroform	0.028	0.020	0.136	0.098		1
1,2-Dichloroethane	ND	0.020	ND	0.081		1
1,1,1-Trichloroethane	ND	0.020	ND	0.109		1
Benzene	0.397	0.100	1.27	0.319		1
Carbon tetrachloride	0.078	0.020	0.490	0.126		1
1,2-Dichloropropane	ND	0.020	ND	0.092		1
Bromodichloromethane	ND	0.020	ND	0.134		1
Trichloroethene	0.070	0.020	0.376	0.107		1
cis-1,3-Dichloropropene	ND	0.020	ND	0.091		1
4-Methyl-2-pentanone	ND	0.500	ND	2.05		1
trans-1,3-Dichloropropene	ND	0.020	ND	0.091		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005822
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005822-07
Client ID: RM 118
Sample Location: PROVIDENCE, RI
Date Collected: 04/21/10 10:03
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
1,1,2-Trichloroethane	ND	0.020	ND	0.109		1
Toluene	1.19	0.020	4.49	0.075		1
Dibromochloromethane	ND	0.020	ND	0.096		1
1,2-Dibromoethane	ND	0.020	ND	0.154		1
Tetrachloroethene	0.075	0.020	0.508	0.136		1
1,1,1,2-Tetrachloroethane	ND	0.020	ND	0.137		1
Chlorobenzene	ND	0.020	ND	0.092		1
Ethylbenzene	0.157	0.020	0.681	0.087		1
p/m-Xylene	0.416	0.040	1.80	0.174		1
Bromoform	ND	0.020	ND	0.206		1
Styrene	0.077	0.020	0.328	0.085		1
1,1,2,2-Tetrachloroethane	ND	0.020	ND	0.137		1
o-Xylene	0.145	0.020	0.629	0.087		1
Isopropylbenzene	ND	0.500	ND	2.46		1
1,3,5-Trimethybenzene	0.042	0.020	0.206	0.098		1
1,2,4-Trimethylbenzene	0.116	0.020	0.570	0.098		1
1,3-Dichlorobenzene	ND	0.020	ND	0.120		1
1,4-Dichlorobenzene	0.025	0.020	0.150	0.120		1
sec-Butylbenzene	ND	0.500	ND	2.74		1
p-Isopropyltoluene	ND	0.500	ND	2.74		1
1,2-Dichlorobenzene	ND	0.020	ND	0.120		1
n-Butylbenzene	ND	0.500	ND	2.74		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005822
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005822-08
Client ID: RM 110
Sample Location: PROVIDENCE, RI
Matrix: Air
Anaytical Method: 48,TO-15-SIM
Analytical Date: 04/26/10 23:40
Analyst: RY

Date Collected: 04/21/10 10:05
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
Dichlorodifluoromethane	0.458	0.050	2.26	0.247		1
Chloromethane	ND	0.500	ND	2.44		1
Vinyl chloride	ND	0.020	ND	0.051		1
Chloroethane	ND	0.020	ND	0.053		1
Acetone	8.40	2.00	19.9	4.75		1
Trichlorofluoromethane	0.605	0.050	3.40	0.281		1
Acrylonitrile	ND	0.500	ND	1.08		1
1,1-Dichloroethene	ND	0.020	ND	0.079		1
Methylene chloride	ND	0.800	ND	1.74		1
trans-1,2-Dichloroethene	ND	0.020	ND	0.079		1
1,1-Dichloroethane	ND	0.020	ND	0.081		1
Methyl tert butyl ether	ND	0.020	ND	0.072		1
2-Butanone	ND	0.500	ND	1.47		1
cis-1,2-Dichloroethene	ND	0.020	ND	0.079		1
Chloroform	0.029	0.020	0.141	0.098		1
1,2-Dichloroethane	ND	0.020	ND	0.081		1
1,1,1-Trichloroethane	ND	0.020	ND	0.109		1
Benzene	0.378	0.100	1.21	0.319		1
Carbon tetrachloride	0.073	0.020	0.459	0.126		1
1,2-Dichloropropane	ND	0.020	ND	0.092		1
Bromodichloromethane	ND	0.020	ND	0.134		1
Trichloroethene	0.067	0.020	0.360	0.107		1
cis-1,3-Dichloropropene	ND	0.020	ND	0.091		1
4-Methyl-2-pentanone	ND	0.500	ND	2.05		1
trans-1,3-Dichloropropene	ND	0.020	ND	0.091		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005822
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005822-08
Client ID: RM 110
Sample Location: PROVIDENCE, RI
Date Collected: 04/21/10 10:05
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
1,1,2-Trichloroethane	ND	0.020	ND	0.109		1
Toluene	1.10	0.020	4.14	0.075		1
Dibromochloromethane	ND	0.020	ND	0.096		1
1,2-Dibromoethane	ND	0.020	ND	0.154		1
Tetrachloroethene	0.066	0.020	0.447	0.136		1
1,1,1,2-Tetrachloroethane	ND	0.020	ND	0.137		1
Chlorobenzene	ND	0.020	ND	0.092		1
Ethylbenzene	0.139	0.020	0.603	0.087		1
p/m-Xylene	0.386	0.040	1.67	0.174		1
Bromoform	ND	0.020	ND	0.206		1
Styrene	0.041	0.020	0.174	0.085		1
1,1,2,2-Tetrachloroethane	ND	0.020	ND	0.137		1
o-Xylene	0.139	0.020	0.603	0.087		1
Isopropylbenzene	ND	0.500	ND	2.46		1
1,3,5-Trimethybenzene	0.038	0.020	0.187	0.098		1
1,2,4-Trimethylbenzene	0.111	0.020	0.545	0.098		1
1,3-Dichlorobenzene	ND	0.020	ND	0.120		1
1,4-Dichlorobenzene	0.026	0.020	0.156	0.120		1
sec-Butylbenzene	ND	0.500	ND	2.74		1
p-Isopropyltoluene	ND	0.500	ND	2.74		1
1,2-Dichlorobenzene	ND	0.020	ND	0.120		1
n-Butylbenzene	ND	0.500	ND	2.74		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005822
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005822-09
Client ID: AMBIENT
Sample Location: PROVIDENCE, RI
Matrix: Air
Anaytical Method: 48,TO-15-SIM
Analytical Date: 04/26/10 18:38
Analyst: RY

Date Collected: 04/21/10 11:40
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
Dichlorodifluoromethane	0.453	0.050	2.24	0.247		1
Chloromethane	0.505	0.500	2.46	2.44		1
Vinyl chloride	ND	0.020	ND	0.051		1
Chloroethane	ND	0.020	ND	0.053		1
Acetone	2.09	2.00	4.96	4.75		1
Trichlorofluoromethane	0.224	0.050	1.26	0.281		1
Acrylonitrile	ND	0.500	ND	1.08		1
1,1-Dichloroethene	ND	0.020	ND	0.079		1
Methylene chloride	ND	0.800	ND	1.74		1
trans-1,2-Dichloroethene	ND	0.020	ND	0.079		1
1,1-Dichloroethane	ND	0.020	ND	0.081		1
Methyl tert butyl ether	ND	0.020	ND	0.072		1
2-Butanone	ND	0.500	ND	1.47		1
cis-1,2-Dichloroethene	ND	0.020	ND	0.079		1
Chloroform	ND	0.020	ND	0.098		1
1,2-Dichloroethane	ND	0.020	ND	0.081		1
1,1,1-Trichloroethane	ND	0.020	ND	0.109		1
Benzene	0.105	0 100	0.335	0.319		1
Carbon tetrachloride	0.077	0.020	0.484	0.126		1
1,2-Dichloropropane	ND	0.020	ND	0.092		1
Bromodichloromethane	ND	0.020	ND	0.134		1
Trichloroethene	ND	0.020	ND	0.107		1
cis-1,3-Dichloropropene	ND	0.020	ND	0.091		1
4-Methyl-2-pentanone	ND	0.500	ND	2.05		1
trans-1,3-Dichloropropene	ND	0.020	ND	0.091		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005822
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005822-09
Client ID: AMBIENT
Sample Location: PROVIDENCE, RI
Date Collected: 04/21/10 11:40
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
1,1,2-Trichloroethane	ND	0.020	ND	0.109		1
Toluene	0.110	0.020	0.414	0.075		1
Dibromochloromethane	ND	0.020	ND	0.096		1
1,2-Dibromoethane	ND	0.020	ND	0.154		1
Tetrachloroethene	0.083	0.020	0.562	0.136		1
1,1,1,2-Tetrachloroethane	ND	0.020	ND	0.137		1
Chlorobenzene	ND	0.020	ND	0.092		1
Ethylbenzene	ND	0.020	ND	0.087		1
p/m-Xylene	ND	0.040	ND	0.174		1
Bromoform	ND	0.020	ND	0.206		1
Styrene	ND	0.020	ND	0.085		1
1,1,2,2-Tetrachloroethane	ND	0.020	ND	0.137		1
o-Xylene	ND	0.020	ND	0.087		1
Isopropylbenzene	ND	0.500	ND	2.46		1
1,3,5-Trimethybenzene	ND	0.020	ND	0.098		1
1,2,4-Trimethylbenzene	ND	0.020	ND	0.098		1
1,3-Dichlorobenzene	ND	0.020	ND	0.120		1
1,4-Dichlorobenzene	ND	0.020	ND	0.120		1
sec-Butylbenzene	ND	0.500	ND	2.74		1
p-Isopropyltoluene	ND	0.500	ND	2.74		1
1,2-Dichlorobenzene	ND	0.020	ND	0.120		1
n-Butylbenzene	ND	0.500	ND	2.74		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005822
Report Date: 04/29/10

Method Blank Analysis
Batch Quality Control

Analytical Method: 48,TO-15-SIM
Analytical Date: 04/26/10 15:11

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab for sample(s): 01-09 Batch: WG409885-4						
Dichlorodifluoromethane	ND	0.050	ND	0.247		1
Chloromethane	ND	0.500	ND	2.44		1
Vinyl chloride	ND	0.020	ND	0.051		1
Chloroethane	ND	0.020	ND	0.053		1
Acetone	ND	2.00	ND	4.75		1
Trichlorofluoromethane	ND	0.050	ND	0.281		1
Acrylonitrile	ND	0.500	ND	1.08		1
1,1-Dichloroethene	ND	0.020	ND	0.079		1
Methylene chloride	ND	0.800	ND	1.74		1
trans-1,2-Dichloroethene	ND	0.020	ND	0.079		1
1,1-Dichloroethane	ND	0.020	ND	0.081		1
Methyl tert butyl ether	ND	0.020	ND	0.072		1
2-Butanone	ND	0.500	ND	1.47		1
cis-1,2-Dichloroethene	ND	0.020	ND	0.079		1
Chloroform	ND	0.020	ND	0.098		1
1,2-Dichloroethane	ND	0.020	ND	0.081		1
1,1,1-Trichloroethane	ND	0.020	ND	0.109		1
Benzene	ND	0.100	ND	0.319		1
Carbon tetrachloride	ND	0.020	ND	0.126		1
1,2-Dichloropropane	ND	0.020	ND	0.092		1
Bromodichloromethane	ND	0.020	ND	0.134		1
Trichloroethene	ND	0.020	ND	0.107		1
cis-1,3-Dichloropropene	ND	0.020	ND	0.091		1
4-Methyl-2-pentanone	ND	0.500	ND	2.05		1
trans-1,3-Dichloropropene	ND	0.020	ND	0.091		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005822
Report Date: 04/29/10

Method Blank Analysis
Batch Quality Control

Analytical Method: 48,TO-15-SIM
Analytical Date: 04/26/10 15:11

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab for sample(s): 01-09 Batch: WG409885-4						
1,1,2-Trichloroethane	ND	0.020	ND	0.109		1
Toluene	ND	0.020	ND	0.075		1
Dibromochloromethane	ND	0.020	ND	0.096		1
1,2-Dibromoethane	ND	0.020	ND	0.154		1
Tetrachloroethene	ND	0.020	ND	0.136		1
1,1,1,2-Tetrachloroethane	ND	0.020	ND	0.137		1
Chlorobenzene	ND	0.020	ND	0.092		1
Ethylbenzene	ND	0.020	ND	0.087		1
p/m-Xylene	ND	0.040	ND	0.174		1
Bromoform	ND	0.020	ND	0.206		1
Styrene	ND	0.020	ND	0.085		1
1,1,2,2-Tetrachloroethane	ND	0.020	ND	0.137		1
o-Xylene	ND	0.020	ND	0.087		1
Isopropylbenzene	ND	0.500	ND	2.46		1
1,3,5-Trimethybenzene	ND	0.020	ND	0.098		1
1,2,4-Trimethylbenzene	ND	0.020	ND	0.098		1
1,3-Dichlorobenzene	ND	0.020	ND	0.120		1
1,4-Dichlorobenzene	ND	0.020	ND	0.120		1
sec-Butylbenzene	ND	0.500	ND	2.74		1
p-Isopropyltoluene	ND	0.500	ND	2.74		1
1,2-Dichlorobenzene	ND	0.020	ND	0.120		1
n-Butylbenzene	ND	0.500	ND	2.74		1

Lab Control Sample Analysis
Batch Quality Control

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01

Lab Number: L1005822
Report Date: 04/29/10

Parameter	LCS %Recovery	Qual	LCSD %Recovery	Qual	%Recovery Limits	RPD	Qual	RPD Limits
Volatile Organics in Air by SIM - Mansfield Lab Associated sample(s): 01-09 Batch: WG409885-3								
Dichlorodifluoromethane	96		-		70-130	-		25
Chloromethane	90		-		70-130	-		25
Vinyl chloride	99		-		70-130	-		25
Chloroethane	86		-		70-130	-		25
Acetone	88		-		70-130	-		25
Trichlorofluoromethane	96		-		70-130	-		25
Acrylonitrile	89		-		70-130	-		25
1,1-Dichloroethene	83		-		70-130	-		25
Methylene chloride	80		-		70-130	-		25
trans-1,2-Dichloroethene	75		-		70-130	-		25
1,1-Dichloroethane	78		-		70-130	-		25
Methyl tert butyl ether	73		-		70-130	-		25
2-Butanone	81		-		70-130	-		25
cis-1,2-Dichloroethene	76		-		70-130	-		25
Chloroform	82		-		70-130	-		25
1,2-Dichloroethane	78		-		70-130	-		25
1,1,1-Trichloroethane	82		-		70-130	-		25
Benzene	69	Q	-		70-130	-		25
Carbon tetrachloride	85		-		70-130	-		25
1,2-Dichloropropane	80		-		70-130	-		25
Bromodichloromethane	85		-		70-130	-		25

Lab Control Sample Analysis
Batch Quality Control

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01

Lab Number: L1005822
Report Date: 04/29/10

Parameter	LCS %Recovery	Qual	LCSD %Recovery	Qual	%Recovery Limits	RPD	Qual	RPD Limits
Volatile Organics in Air by SIM - Mansfield Lab Associated sample(s): 01-09 Batch: WG409885-3								
Trichloroethene	83		-		70-130	-		25
cis-1,3-Dichloropropene	79		-		70-130	-		25
4-Methyl-2-pentanone	97		-		70-130	-		25
trans-1,3-Dichloropropene	64	Q	-		70-130	-		25
1,1,2-Trichloroethane	81		-		70-130	-		25
Toluene	70		-		70-130	-		25
Dibromochloromethane	83		-		70-130	-		25
1,2-Dibromoethane	79		-		70-130	-		25
Tetrachloroethene	81		-		70-130	-		25
1,1,1,2-Tetrachloroethane	84		-		70-130	-		25
Chlorobenzene	79		-		70-130	-		25
Ethylbenzene	73		-		70-130	-		25
p/m-Xylene	77		-		70-130	-		25
Bromoform	85		-		70-130	-		25
Styrene	74		-		70-130	-		25
1,1,2,2-Tetrachloroethane	86		-		70-130	-		25
o-Xylene	78		-		70-130	-		25
Isopropylbenzene	82		-		70-130	-		25
1,3,5-Trimethylbenzene	82		-		70-130	-		25
1,2,4-Trimethylbenzene	88		-		70-130	-		25
1,3-Dichlorobenzene	87		-		70-130	-		25

Lab Control Sample Analysis

Batch Quality Control

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01

Lab Number: L1005822
Report Date: 04/29/10

Parameter	LCS %Recovery	Qual	LCSD %Recovery	Qual	%Recovery Limits	RPD	Qual	RPD Limits
Volatile Organics in Air by SIM - Mansfield Lab Associated sample(s): 01-09 Batch: WG409885-3								
1,4-Dichlorobenzene	85		-		70-130	-		25
sec-Butylbenzene	91		-		70-130	-		25
p-Isopropyltoluene	89		-		70-130	-		25
1,2-Dichlorobenzene	86		-		70-130	-		25
n-Butylbenzene	103		-		70-130	-		25

Lab Duplicate Analysis
Batch Quality Control

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01

Lab Number: L1005822
Report Date: 04/29/10

Parameter	Native Sample	Duplicate Sample	Units	RPD	Qual	RPD Limits
Volatile Organics in Air by SIM - Mansfield Lab Associated sample(s): 01-09 QC Batch ID: WG409885-5 QC Sample: L1005820-05 Client ID: DUP Sample						
Dichlorodifluoromethane	0.535	0 479	ppbV	11		25
Chloromethane	ND	ND	ppbV	NC		25
Vinyl chloride	ND	ND	ppbV	NC		25
Chloroethane	ND	ND	ppbV	NC		25
Acetone	30.7	27.4	ppbV	11		25
Trichlorofluoromethane	0.882	0.791	ppbV	11		25
Acrylonitrile	ND	ND	ppbV	NC		25
1,1-Dichloroethene	ND	ND	ppbV	NC		25
Methylene chloride	ND	ND	ppbV	NC		25
trans-1,2-Dichloroethene	ND	ND	ppbV	NC		25
1,1-Dichloroethane	ND	ND	ppbV	NC		25
Methyl tert butyl ether	ND	ND	ppbV	NC		25
2-Butanone	5.72	5.17	ppbV	10		25
cis-1,2-Dichloroethene	ND	ND	ppbV	NC		25
Chloroform	0.045	0.041	ppbV	9		25
1,2-Dichloroethane	ND	ND	ppbV	NC		25
1,1,1-Trichloroethane	0.020	ND	ppbV	NC		25
Benzene	0.199	0.119	ppbV	50	Q	25
Carbon tetrachloride	0.097	0.077	ppbV	23		25

Lab Duplicate Analysis
Batch Quality Control

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01

Lab Number: L1005822
Report Date: 04/29/10

Parameter	Native Sample	Duplicate Sample	Units	RPD		RPD Limits
Volatile Organics in Air by SIM - Mansfield Lab Associated sample(s): 01-09 QC Batch ID: WG409885-5 QC Sample: L1005820-05 Client ID: DUP Sample						
1,2-Dichloropropane	ND	ND	ppbV	NC		25
Bromodichloromethane	ND	ND	ppbV	NC		25
Trichloroethene	0.166	0.136	ppbV	20		25
cis-1,3-Dichloropropene	ND	ND	ppbV	NC		25
4-Methyl-2-pentanone	ND	ND	ppbV	NC		25
trans-1,3-Dichloropropene	ND	ND	ppbV	NC		25
1,1,2-Trichloroethane	ND	ND	ppbV	NC		25
Toluene	0.754	0.610	ppbV	21		25
Dibromochloromethane	ND	ND	ppbV	NC		25
1,2-Dibromoethane	ND	ND	ppbV	NC		25
Tetrachloroethene	9.17	7.50	ppbV	20		25
1,1,1,2-Tetrachloroethane	ND	ND	ppbV	NC		25
Chlorobenzene	ND	ND	ppbV	NC		25
Ethylbenzene	0.490	0.408	ppbV	18		25
p/m-Xylene	1.43	1.18	ppbV	19		25
Bromoform	ND	ND	ppbV	NC		25
Styrene	0.113	0.087	ppbV	23	Q	25
1,1,2,2-Tetrachloroethane	ND	ND	ppbV	NC		25
o-Xylene	0.285	0.235	ppbV	19		25

Lab Duplicate Analysis
Batch Quality Control

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01

Lab Number: L1005822
Report Date: 04/29/10

Parameter	Native Sample	Duplicate Sample	Units	RPD		RPD Limits
Volatile Organics in Air by SIM - Mansfield Lab Associated sample(s): 01-09 QC Batch ID: WG409885-5 QC Sample: L1005820-05 Client ID: DUP Sample						
Isopropylbenzene	ND	ND	ppbV	NC		25
1,3,5-Trimethybenzene	0.036	0.029	ppbV	22		25
1,2,4-Trimethylbenzene	0.133	0.108	ppbV	21		25
1,3-Dichlorobenzene	ND	ND	ppbV	NC		25
1,4-Dichlorobenzene	0.545	0.419	ppbV	24	Q	25
sec-Butylbenzene	ND	ND	ppbV	NC		25
p-Isopropyltoluene	ND	ND	ppbV	NC		25
1,2-Dichlorobenzene	ND	ND	ppbV	NC		25
n-Butylbenzene	ND	ND	ppbV	NC		25

Project Name: ALVAREZ HIGH SCHOOL **Lab Number:** L1005822

Project Number: 14687.01 **Report Date:** 04/29/10

Canister and Flow Controller Information

Samplenum	Client ID	Media ID	Media Type	Cleaning Batch ID	Initial Pressure (In. Hg)	Pressure on Receipt (In. Hg)	Flow Out mL/min	Flow In mL/min	% RSD
L1005822-01	GYMNASIUM	0090	#90 SV		-	-	68	70	3
L1005822-01	GYMNASIUM	424	2.7L Can	L1005024	-29.3	-4.6	-	-	-
L1005822-02	CAFETERIA	0252	#90 SV		-	-	67	80	18
L1005822-02	CAFETERIA	1721	2.7L Can	L1005024	-29.3	0.4	-	-	-
L1005822-03	KITCHEN STORAGE RM	0360	#90 SV		-	-	69	76	10
L1005822-03	KITCHEN STORAGE RM	150B	2.7L Can	L1005024	-29.3	0.6	-	-	-
L1005822-04	ELEVATOR HALLWAY	0042	#90 AMB		-	-	68	68	0
L1005822-04	ELEVATOR HALLWAY	546	2.7L Can	L1005024	-29.3	-3.3	-	-	-
L1005822-05	RM 145	0130	#90 SV		-	-	71	72	1
L1005822-05	RM 145	478	2.7L Can	L1005024	-29.3	-2.0	-	-	-
L1005822-06	RM 152	0451	#90 SV		-	-	72	74	3
L1005822-06	RM 152	372	2.7L Can	L1005024	-29.3	-3.5	-	-	-
L1005822-07	RM 118	0124	#90 SV		-	-	67	67	0
L1005822-07	RM 118	132	2.7L Can	I1005361	-29.3	-4.6	-	-	-
L1005822-08	RM 110	0173	#90 SV		-	-	70	72	3
L1005822-08	RM 110	121	2.7L Can	L1005024	-29.3	-1.6	-	-	-
L1005822-09	AMBIENT	0398	#90 SV		-	-	69	76	10

Project Name: ALVAREZ HIGH SCHOOL **Lab Number:** L1005822

Project Number: 14687.01 **Report Date:** 04/29/10

Canister and Flow Controller Information

Samplenum	Client ID	Media ID	Media Type	Cleaning Batch ID	Initial Pressure (in. Hg)	Pressure on Receipt (in. Hg)	Flow Out mL/min	Flow In mL/min	% RSD
L1005822-09	AMBIENT	1717	2.7L Can	L1005024	-29.3	-0.2	-	-	-

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01

Lab Number: L1005822
Report Date: 04/29/10

Sample Receipt and Container Information

Were project specific reporting limits specified? YES

Reagent H2O Preserved Vials Frozen on: NA

Cooler Information **Custody Seal**
Cooler
N/A Absent

Container Information

Container ID	Container Type	Cooler	pH	Temp deg C	Pres	Seal	Analysis
L1005822-01A	Canister - 2.7 Liter	N/A	N/A		NA	Absent	TO15-SIM(30)
L1005822-02A	Canister - 2.7 Liter	N/A	N/A		NA	Absent	TO15-SIM(30)
L1005822-03A	Canister - 2.7 Liter	N/A	N/A		NA	Absent	TO15-SIM(30)
L1005822-04A	Canister - 2.7 Liter	N/A	N/A		NA	Absent	TO15-SIM(30)
L1005822-05A	Canister - 2.7 Liter	N/A	N/A		NA	Absent	TO15-SIM(30)
L1005822-06A	Canister - 2.7 Liter	N/A	N/A		NA	Absent	TO15-SIM(30)
L1005822-07A	Canister - 2.7 Liter	N/A	N/A		NA	Absent	TO15-SIM(30)
L1005822-08A	Canister - 2.7 Liter	N/A	N/A		NA	Absent	TO15-SIM(30)
L1005822-09A	Canister - 2.7 Liter	N/A	N/A		NA	Absent	TO15-SIM(30)

*Hold days indicated by values in parentheses

Project Name:	ALVAREZ HIGH SCHOOL	**Lab Number:**	L1005822
Project Number:	14687.01	**Report Date:**	04/29/10

GLOSSARY

Acronyms

EPA · Environmental Protection Agency.

LCS · Laboratory Control Sample: A sample matrix, free from the analytes of interest, spiked with verified known amounts of analytes or a material containing known and verified amounts of analytes.

LCSD · Laboratory Control Sample Duplicate: Refer to LCS.

MS · Matrix Spike Sample: A sample prepared by adding a known mass of target analyte to a specified amount of matrix sample for which an independent estimate of target analyte concentration is available.

MSD · Matrix Spike Sample Duplicate: Refer to MS.

NA · Not Applicable.

NC · Not Calculated: Term is utilized when one or more of the results utilized in the calculation are non-detect at the parameter's reporting unit.

NI · Not Ignitable.

RDL · Reported Detection Limit: The value at which an instrument can accurately measure an analyte at a specific concentration. The RDL includes any adjustments from dilutions, concentrations or moisture content, where applicable.

RPD · Relative Percent Difference: The results from matrix and/or matrix spike duplicates are primarily designed to assess the precision of analytical results in a given matrix and are expressed as relative percent difference (RPD). Values which are less than five times the reporting limit for any individual parameter are evaluated by utilizing the absolute difference between the values; although the RPD value will be provided in the report.

Terms

Analytical Method: Both the document from which the method originates and the analytical reference method. (Example: EPA 8260B is shown as 1,8260B.) The codes for the reference method documents are provided in the References section of the Addendum.

Data Qualifiers

A · Spectra identified as "Aldol Condensation Product".

B · The analyte was detected above the reporting limit in the associated method blank. Flag only applies to associated field samples that have detectable concentrations of the analyte at less than five times (5x) the concentration found in the blank. For MCP-related projects, flag only applies to associated field samples that have detectable concentrations of the analyte at less than ten times (10x) the concentration found in the blank. For DOD-related projects, flag only applies to associated field samples that have detectable concentrations of the analyte at less than ten times (10x) the concentration found in the blank AND the analyte was detected above one-half the reporting limit (or above the reporting limit for common lab contaminants) in the associated method blank.

D · Concentration of analyte was quantified from diluted analysis. Flag only applies to field samples that have detectable concentrations of the analyte.

E · Concentration of analyte exceeds the range of the calibration curve and/or linear range of the instrument.

H · The analysis of pH was performed beyond the regulatory-required holding time of 15 minutes from the time of sample collection.

P · The RPD between the results for the two columns exceeds the method-specified criteria.

Q · The quality control sample exceeds the associated acceptance criteria. Note: This flag is not applicable for matrix spike recoveries when the sample concentration is greater than 4x the spike added or for batch duplicate RPD when the sample concentrations are less than 5x the RDL. (Metals only.)

R · Analytical results are from sample re-analysis.

RE · Analytical results are from sample re-extraction.

J · Estimated value. This represents an estimated concentration for Tentatively Identified Compounds (TICs).

ND · Not detected at the reported detection limit (RDL) for the sample.

Report Format: Data Usability Report

Project Name:	ALVAREZ HIGH SCHOOL	**Lab Number:**	L1005822
Project Number:	14687.01	**Report Date:**	04/29/10

REFERENCES

48 Compendium of Methods for the Determination of Toxic Organic Compounds in Ambient Air. Second Edition. EPA/625/R-96/010b, January 1999.

LIMITATION OF LIABILITIES

Alpha Analytical performs services with reasonable care and diligence normal to the analytical testing laboratory industry. In the event of an error, the sole and exclusive responsibility of Alpha Woods Hole Labs shall be to re-perform the work at it's own expense. In no event shall Alpha Analytical be held liable for any incidental, consequential or special damages, including but not limited to, damages in any way connected with the use of, interpretation of, information or analysis provided by Alpha Woods Hole Labs.

We strongly urge our clients to comply with EPA protocol regarding sample volume, preservation, cooling, containers, sampling procedures, holding time and splitting of samples in the field.

Certificate/Approval Program Summary

Last revised December 15, 2009 – Mansfield Facility

The following list includes only those analytes/methods for which certification/approval is currently held.
For a complete listing of analytes for the referenced methods, please contact your Alpha Customer Service Representative.

Connecticut Department of Public Health Certificate/Lab ID: PH-0141.

Wastewater/Non-Potable Water (Inorganic Parameters: pH, Turbidity, Conductivity, Alkalinity, Aluminum, Antimony, Arsenic, Barium, Beryllium, Boron, Cadmium, Calcium, Chromium, Cobalt, Copper, Iron, Lead, Magnesium, Manganese, Mercury, Molybdenum, Nickel, Potassium, Selenium, Silver, Sodium, Strontium, Thallium, Tin, Vanadium, Zinc, Total Residue (Solids), Total Suspended Solids (non-filterable), Total Cyanide. Organic Parameters: PCBs, Organochlorine Pesticides, Technical Chlordane, Toxaphene, Acid Extractables, Benzidines, Phthalate Esters, Nitrosamines, Nitroaromatics & Isophorone, PAHs, Haloethers, Chlorinated Hydrocarbons, Volatile Organics.)

Solid Waste/Soil (Inorganic Parameters: pH, Aluminum, Antimony, Arsenic, Barium, Beryllium, Cadmium, Calcium, Chromium, Hexavalent Chromium, Cobalt, Copper, Iron, Lead, Magnesium, Manganese, Mercury, Molybdenum, Nickel, Potassium, Selenium, Silver, Sodium, Thallium, Vanadium, Zinc, Total Organic Carbon, Total Cyanide, Corrosivity, TCLP 1311. Organic Parameters: PCBs, Organochlorine Pesticides, Technical Chlordane, Toxaphene, Volatile Organics, Acid Extractables, Benzidines, Phthalates, Nitrosamines, Nitroaromatics & Cyclic Ketones, PAHs, Haloethers, Chlorinated Hydrocarbons.)

Florida Department of Health Certificate/Lab ID: E87814. ***NELAP Accredited.***

Non-Potable Water (Inorganic Parameters: SM2320B, EPA 120.1, SM2510B, EPA 245.1, EPA 150.1, EPA 160.2, SM2540D, EPA 335.2, SM2540G, EPA 180.1. Organic Parameters: EPA 625, 608.)

Solid & Chemical Materials (Inorganic Parameters: 6020, 7470, 7471, 9045, 9014. Organic Parameters: EPA 8260, 8270, 8082, 8081.)

Air & Emissions (EPA TO-15.)

Louisiana Department of Environmental Quality Certificate/Lab ID: 03090. ***NELAP Accredited.***

Non-Potable Water (Inorganic Parameters: EPA 120.1, 150.1, 160.2, 180.1, 200.8, 245.1, 310.1, 335.2, 608, 625, 1631, 3010, 3015, 3020, 6020, 9010, 9014, 9040, SM2320B, 2510B, 2540D, 2540G, 4500CN-E, 4500H-B, Organic Parameters: EPA 3510, 3580, 3630, 3640, 3660, 3665, 5030, 8015 (mod), 3570, 8081, 8082, 8260, 8270,)

Solid & Chemical Materials (Inorganic Parameters: 6020, 7196, 7470, 7471, 7474, 9010, 9014, 9040, 9045, 9060. Organic Parameters: EPA 8015 (mod), EPA 3570, 1311, 3050, 3051, 3060, 3580, 3630, 3640, 3660, 3665, 5035, 8081, 8082, 8260, 8270.)

Biological Tissue (Inorganic Parameters: EPA 6020. Organic Parameters: EPA 3570, 3510, 3610, 3630, 3640, 8270.)

Maine Department of Human Services Certificate/Lab ID: MA0030.

Wastewater (Inorganic Parameters: EPA 120.1, 300.0, SM 2320, 2510B, 2540C, 2540D, EPA 245.1. Organic Parameters: 608, 624.)

Massachusetts Department of Environmental Protection Certificate/Lab ID: M-MA030.

Non-Potable Water (Inorganic Parameters: SM4500H+B. Organic Parameters: EPA 624.)

New Hampshire Department of Environmental Services Certificate/Lab ID: 2206. ***NELAP Accredited.***

Non-Potable Water (Inorganic Parameters: EPA 200.8, 245.1, 1631E, 120.1, 150.1, 180.1, 310.1, 335.2, 160.2, SM2540D, 2540G, 4500CN-E, 4500H+B, 2320B, 2510B. Organic Parameters: EPA 625, 608.)

New Jersey Department of Environmental Protection Certificate/Lab ID: MA015. ***NELAP Accredited.***

Non-Potable Water (Inorganic Parameters: SW-846 1312, 3010, 3020A, 3015, 6020, SM2320B, EPA 200.8, SM2540C, 2540D, 2540G, EPA 120.1, SM2510B, EPA 180.1, 245.1, 1631E, SW-846 9040B, 6020, 9010B, 9014 Organic Parameters: EPA 608, 625, SW-846 3510C, 3580A, 5030B, 3035L, 5035H, 3630C, 3640A, 3660B, 3665A, 8081A, 8082 8260B, 8270C)

Solid & Chemical Materials (Inorganic Parameters: SW-846 6020, 9010B, 9014, 1311, 1312, 3050B, 3051, 3060A, 7196A, 7470A, 7471A, 9045C, 9060. Organic Parameters: SW-846 3580A, 5030B, 3035L, 5035H, 3630C, 3640A, 3660B, 3665A, 8081A, 8082, 8260B, 8270C, 3570, 8015B.)

Atmospheric Organic Parameters (EPA TO-15)

Biological Tissue (Inorganic Parameters: SW-846 6020 Organic Parameters: SW-846 8270C, 3510C, 3570, 3610B, 3630C, 3640A)

New York Department of Health Certificate/Lab ID: 11627. ***NELAP Accredited.***

Non-Potable Water (Inorganic Parameters: EPA 310.1, SM2320B, EPA 365.2, 160.1, EPA 160.2, SM2540D, EPA 200.8, 6020, 1631E, 245.1, 335.2, 9014, 150.1, 9040B, 120.1, SM2510B, EPA 376.2, 180.1, 9010B. Organic Parameters: EPA 624, 8260B, 8270C, 608, 8081A, 625, 8082, 3510C, 3511, 5030B.)

Solid & Hazardous Waste (Inorganic Parameters: EPA 9040B, 9045C, SW-846 Ch7 Sec 7.3, EPA 6020, 7196A, 7471A, 7474, 9014, 9040B, 9045C, 9010B. Organic Parameters: EPA 8260B, 8270C, 8081A, DRO 8015B, 8082, 1311, 3050B, 3580, 3050B, 3035, 3570, 3051, 5035, 5030B.)

Air & Emissions (EPA TO-15.)

Pennsylvania Department of Environmental Protection Certificate/Lab ID: 68-02089. ***NELAP Accredited.***

Non-Potable Water (Organic Parameters: EPA 5030B, EPA 8260)

Rhode Island Department of Health Certificate/Lab ID: LAO00299. ***NELAP Accredited via LA-DEQ.***

Refer to MA-DEP Certificate for Non-Potable Water.

Refer to LA-DEQ Certificate for Non-Potable Water.

Texas Commission of Environmental Quality Certificate/Lab ID: T104704419-08-TX. ***NELAP Accredited.***

Solid & Chemical Materials (Inorganic Parameters: EPA 6020, 7470, 7471, 1311, 7196, 9014, 9040, 9045, 9060. Organic Parameters: EPA 8015, 8270, 8260, 8081, 8082.)

U.S. Army Corps of Engineers

Department of Defense Certificate/Lab ID: L2217.01.

Non-Potable Water (Inorganic Parameters: EPA 3005A,3020, 6020, 245.1, 245.7, 1631E, 7470A, 7474, 9014, 120.1, 9050A, 180.1, SM4500H-B, 2320B, 2510B, 2540D,9040. Organic Parameters: EPA 3510C, 5030B, 9010B, 624, 8260B, 8270C, 8270 Alk-PAH, 8082, 8081A, 8015 (SHC), 8015 (DRO).)

Solid & Hazardous Waste (Inorganic Parameters: EPA 1311, 1312,3051, 6020, 747A, 7474, 9045C,9060, SM 2540G, ASTM D422-63. Organic Parameters: EPA 3580, 3570, 3540C, 5035, 8260B, 8270C, 8270 Alk-PAH, 8082, 8081A, 8015 (SHC), 8015 (DRO).

Air & Emissions (EPA TO-15.)

Analytes Not Accredited by NELAP
Certification is not available by NELAP for the following analytes: **8270C:** Biphenyl.

AIR ANALYSIS

PAGE 1 OF 1

ALPHA ANALYTICAL — CHAIN OF CUSTODY

320 Forbes Blvd, Mansfield, MA 02048
TEL: 508-822-9300 FAX: 508-822-3288

Date Rec'd In Lab:

ALPHA Job #: L1005822

04291015:52

Client Information

Client: EA ENGINEERING
Address: 2350 POST ROAD
WARWICK RI 02886
Phone: (401) 736-3440
Fax:
Email: rmack@eaest.com
☐ These samples have been previously analyzed by Alpha

Project Information

Project Name: ALVAREZ SCHOOL
Project Location: PROVIDENCE, RI
Project #: 14687.01
Project Manager: FRANK POSTMA
ALPHA Quote #:

Turn-Around Time

☑ Standard ☐ RUSH (only confirmed if pre-approved)
Date Due: Time:

Report Information - Data Deliverables

☐ FAX
☐ ADEx
Criteria Checker: _____ (Default based on Regulatory Criteria Indicated)
Other Formats: _____
☑ EMAIL (standard pdf report)
☐ Additional Deliverables:
Report to: (if different than Project Manager) RON MACK
rmack@eaest.com

Billing Information

☑ Same as Client info PO #:

Regulatory Requirements/Report Limits

State/Fed	Program	Criteria
CT	TARGET INDOOR AIR CONCENTRATIONS	

Other Project Specific Requirements/Comments:

All Columns Below Must Be Filled Out

ALPHA Lab ID (Lab Use Only)	Sample ID	Collection Date	Start Time	End Time	Initial Vacuum	Final Vacuum	Sample Matrix*	Sampler's Initials	Can Size	ID Cap	ID - Flow Controller	TO-14A by TO-15	TO-15	TO-15 SIM	APH	FIXED GASES	TO-13A	TO-4 / TO-10	Sample Comments (i.e. PID)
5822-1	GYMNASIUM	4/21/10	9:23	9:50	30	6	AA	RM	424	2.7L	0090			X					462 ppb
-2	CAFETERIA	4/21/10	9:21	9:51	29	1		RM	1721		0252								56 ppb
-3	KITCHEN STORAGE RM		9:42	10:13	29	1		RM	1508		0360								0 ppb
-4	ELEVATOR HALLWAY		9:24	9:53	30+	8		RM	546		0042								0 ppb
-5	RM 145		9:28	9:58	30+	5		RM	478		0130								4 ppb
-6	RM 152		9:28	9:58	28	4		RM	372		0451								2 ppb
-7	RM 118		9:30	10:03	30+	10.5		RM	132		0124								11 ppb
-8	RM 110		9:30	10:05	30+	4		RM	121		0173								14 ppb
-9	AMBIENT		11:03	11:40	30+	4		RM	1717		0398								11 ppb

*SAMPLE MATRIX CODES
AA = Ambient Air (Indoor/Outdoor)
SV = Soil Vapor/Landfill Gas/SVE
Other = Please Specify

Container Type

Please print clearly, legibly and completely. Samples can not be logged in and turnaround time clock will not start until any ambiguities are resolved. All samples submitted are subject to Alpha's Terms and Conditions. See reverse side.

Relinquished By:	Date/Time	Received By:	Date/Time:
	4/22/10 1:12		4/22/10 16:30
	4/22/10		4/22/10 16:30

Form No: 101-02 (19-Jun-06)

Attachment C

Subslab Vapor Analytical Report
21 April 2010

ALPHA ANALYTICAL

ANALYTICAL REPORT

Lab Number:	L1005820
Client:	EA Engineering, Science and Tech 2350 Post Road Warwick, RI 02886
ATTN:	Frank Postma
Phone:	(401) 736-3440
Project Name:	ALVAREZ HIGH SCHOOL
Project Number:	14687.01
Report Date:	04/29/10

Certifications & Approvals: MA (M-MA030), NY (11627), CT (PH-0141), NH (2206), NJ (MA015), RI (LAO00299), ME (MA0030), PA (Registration #68-02089), LA NELAC (03090), FL NELAC (E87814), US Army Corps of Engineers.

320 Forbes Boulevard, Mansfield, MA 02048-1806
508-822-9300 (Fax) 508-822-3288 800-624-9220 - www.alphalab.com

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01

Lab Number: L1005820
Report Date: 04/29/10

Alpha Sample ID	Client ID	Sample Location	Collection Date/Time
L1005820-01	MP-2	PROVIDENCE, RI	04/21/10 11:29
L1005820-02	MP-5	PROVIDENCE, RI	04/21/10 11:55
L1005820-03	MP-7	PROVIDENCE, RI	04/21/10 11:50
L1005820-04	MP-8	PROVIDENCE, RI	04/21/10 12:10
L1005820-05	IMP-1	PROVIDENCE, RI	04/21/10 10:18
L1005820-06	IMP-3	PROVIDENCE, RI	04/21/10 10:30

Project Name:	ALVAREZ HIGH SCHOOL	**Lab Number:**	L1005820
Project Number:	14687.01	**Report Date:**	04/29/10

Case Narrative

The samples were received in accordance with the Chain of Custody and no significant deviations were encountered during the preparation or analysis unless otherwise noted. Sample Receipt, Container Information, and the Chain of Custody are located at the back of the report.

Results contained within this report relate only to the samples submitted under this Alpha Lab Number and meet all of the requirements of NELAC, for all NELAC accredited parameters. The data presented in this report is organized by parameter (i.e. VOC, SVOC, etc.). Sample specific Quality Control data (i.e. Surrogate Spike Recovery) is reported at the end of the target analyte list for each individual sample, followed by the Laboratory Batch Quality Control at the end of each parameter. If a sample was re-analyzed or re-extracted due to a required quality control corrective action and if both sets of data are reported, the Laboratory ID of the re-analysis or re-extraction is designated with an "R" or "RE", respectively. When multiple Batch Quality Control elements are reported (e.g. more than one LCS), the associated samples for each element are noted in the grey shaded header line of each data table. Any Laboratory Batch, Sample Specific % recovery or RPD value that is outside the listed Acceptance Criteria is bolded in the report. Definitions of all data qualifiers and acronyms used in this report are provided in the Glossary located at the back of the report.

Please see the associated ADEx data file for a comparison of laboratory reporting limits that were achieved with the regulatory Numerical Standards requested on the Chain of Custody.

For additional information, please contact Client Services at 800-624-9220.

Volatile Organics in Air (SIM)

L1005820-01 through -03, -05, -06, and WG409885-5 duplicate: results for Chloromethane should be considered estimated due to co-elution with a non-target peak.

L1005820-02 and -03 have elevated detection limits due to the dilution required by the elevated concentrations of non-target compounds in the sample.

L1005820-04 has elevated detection limits due to the dilution required by the elevated concentrations of target compounds in the sample. The sample was re-analyzed on dilution in order to quantitate the sample within the calibration range. The result should be considered estimated, and is qualified with an E flag, for any compound that exceeded the calibration on the initial analysis. The re-analysis was performed only for the compound that

Project Name:	ALVAREZ HIGH SCHOOL	**Lab Number:**	L1005820
Project Number:	14687.01	**Report Date:**	04/29/10

Case Narrative (continued)

exceeded the calibration range.

L1005820-05 and -06 were re-analyzed due to over dilution of the original analysis. The results of the re-analysis are reported.

The WG409885-3 LCS recoveries for Benzene (69%) and trans-1,3-Dichloropropene (64%) are outside the 70%-130% acceptance limit. The LCS was within overall method allowances, therefore the analysis proceeded.

WG409885-5: The relative percent difference for Benzene (50%) is above the RPD limit of 25%. This compound represented less than 10% of the compounds detected, therefore no further action was taken.

I, the undersigned, attest under the pains and penalties of perjury that, to the best of my knowledge and belief and based upon my personal inquiry of those responsible for providing the information contained in this analytical report, such information is accurate and complete. This certificate of analysis is not complete unless this page accompanies any and all pages of this report.

Authorized Signature: Kathleen M. O'Brien Kathleen O'Brien

Title: Technical Director/Representative Date: 04/29/10

AIR

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005820
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005820-01
Client ID: MP-2
Sample Location: PROVIDENCE, RI
Matrix: Soil_Vapor
Anaytical Method: 48,TO-15-SIM
Analytical Date: 04/28/10 01:15
Analyst: RY

Date Collected: 04/21/10 11:29
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
Dichlorodifluoromethane	0.115	0.050	0.568	0.247		1
Chloromethane	0.667	0.500	3.25	2.44		1
Vinyl chloride	ND	0.020	ND	0.051		1
Chloroethane	0.028	0.020	0.074	0.053		1
Acetone	9.24	2.00	21.9	4.75		1
Trichlorofluoromethane	0.083	0.050	0.466	0.281		1
Acrylonitrile	ND	0.500	ND	1.08		1
1,1-Dichloroethene	ND	0.020	ND	0.079		1
Methylene chloride	ND	0.500	ND	1.74		1
trans-1,2-Dichloroethene	ND	0.020	ND	0.079		1
1,1-Dichloroethane	ND	0.020	ND	0.081		1
Methyl tert butyl ether	ND	0.020	ND	0.072		1
2-Butanone	2.18	0.500	6.44	1.47		1
cis-1,2-Dichloroethene	ND	0.020	ND	0.079		1
Chloroform	ND	0.020	ND	0.098		1
1,2-Dichloroethane	ND	0.020	ND	0.081		1
1,1,1-Trichloroethane	ND	0.020	ND	0.109		1
Benzene	0.117	0.100	0.373	0.319		1
Carbon tetrachloride	0.020	0.020	0.126	0.126		1
1,2-Dichloropropane	ND	0.020	ND	0.092		1
Bromodichloromethane	ND	0.020	ND	0.134		1
Trichloroethene	ND	0.020	ND	0.107		1
cis-1,3-Dichloropropene	ND	0.020	ND	0.091		1
4-Methyl-2-pentanone	ND	0.500	ND	2.05		1
trans-1,3-Dichloropropene	ND	0.020	ND	0.091		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005820
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005820-01
Client ID: MP-2
Sample Location: PROVIDENCE, RI
Date Collected: 04/21/10 11:29
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
1,1,2-Trichloroethane	ND	0.020	ND	0.109		1
Toluene	0.239	0.020	0.900	0.075		1
Dibromochloromethane	ND	0.020	ND	0.096		1
1,2-Dibromoethane	ND	0.020	ND	0.154		1
Tetrachloroethene	1.06	0.020	7.20	0.136		1
1,1,1,2-Tetrachloroethane	ND	0.020	ND	0.137		1
Chlorobenzene	ND	0.020	ND	0.092		1
Ethylbenzene	0.070	0.020	0.304	0.087		1
p/m-Xylene	0.162	0.040	0.703	0.174		1
Bromoform	ND	0.020	ND	0.206		1
Styrene	ND	0.020	ND	0.085		1
1,1,2,2-Tetrachloroethane	ND	0.020	ND	0.137		1
o-Xylene	0.059	0.020	0.256	0.087		1
Isopropylbenzene	ND	0.500	ND	2.46		1
1,3,5-Trimethybenzene	ND	0.020	ND	0.098		1
1,2,4-Trimethylbenzene	0.043	0.020	0.211	0.098		1
1,3-Dichlorobenzene	ND	0.020	ND	0.120		1
1,4-Dichlorobenzene	ND	0.020	ND	0.120		1
sec-Butylbenzene	ND	0.500	ND	2.74		1
p-Isopropyltoluene	ND	0.500	ND	2.74		1
1,2-Dichlorobenzene	ND	0.020	ND	0.120		1
n-Butylbenzene	ND	0.500	ND	2.74		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005820
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005820-02
Client ID: MP-5
Sample Location: PROVIDENCE, RI
Matrix: Soil_Vapor
Anaytical Method: 48,TO-15-SIM
Analytical Date: 04/27/10 01:35
Analyst: RY

Date Collected: 04/21/10 11:55
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
Dichlorodifluoromethane	0.455	0.250	2.25	1.24		5
Chloromethane	ND	2.50	ND	12.2		5
Vinyl chloride	ND	0.100	ND	0.255		5
Chloroethane	0.100	0.100	0.264	0.264		5
Acetone	86.9	10.0	206	23.7		5
Trichlorofluoromethane	1.80	0.250	10.1	1.40		5
Acrylonitrile	ND	2.50	ND	5.42		5
1,1-Dichloroethene	ND	0.100	ND	0.396		5
Methylene chloride	ND	2.50	ND	8.68		5
trans-1,2-Dichloroethene	ND	0.100	ND	0.396		5
1,1-Dichloroethane	ND	0.100	ND	0.404		5
Methyl tert butyl ether	ND	0.100	ND	0.360		5
2-Butanone	51.1	2.50	150	7.37		5
cis-1,2-Dichloroethene	ND	0.100	ND	0.396		5
Chloroform	ND	0.100	ND	0.488		5
1,2-Dichloroethane	ND	0.100	ND	0.404		5
1,1,1-Trichloroethane	ND	0.100	ND	0.545		5
Benzene	ND	0.500	ND	1.60		5
Carbon tetrachloride	ND	0.100	ND	0.629		5
1,2-Dichloropropane	ND	0.100	ND	0.462		5
Bromodichloromethane	ND	0.100	ND	0.670		5
Trichloroethene	6.32	0.100	34.0	0.537		5
cis-1,3-Dichloropropene	ND	0.100	ND	0.453		5
4-Methyl-2-pentanone	ND	2.50	ND	10.2		5
trans-1,3-Dichloropropene	ND	0.100	ND	0.453		5

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005820
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005820-02
Client ID: MP-5
Sample Location: PROVIDENCE, RI
Date Collected: 04/21/10 11:55
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
1,1,2-Trichloroethane	ND	0.100	ND	0.545		5
Toluene	0.790	0.100	2.97	0.376		5
Dibromochloromethane	ND	0.100	ND	0.460		5
1,2-Dibromoethane	ND	0.100	ND	0.768		5
Tetrachloroethene	4.64	0.100	31.4	0.678		5
1,1,1,2-Tetrachloroethane	ND	0.100	ND	0.686		5
Chlorobenzene	ND	0.100	ND	0.460		5
Ethylbenzene	0.310	0.100	1.34	0.434		5
p/m-Xylene	0.755	0.200	3.28	0.868		5
Bromoform	ND	0.100	ND	1.03		5
Styrene	ND	0.100	ND	0.426		5
1,1,2,2-Tetrachloroethane	ND	0.100	ND	0.686		5
o-Xylene	0.270	0.100	1.17	0.434		5
Isopropylbenzene	ND	2.50	ND	12.3		5
1,3,5-Trimethybenzene	ND	0.100	ND	0.491		5
1,2,4-Trimethylbenzene	0.190	0.100	0.933	0.491		5
1,3-Dichlorobenzene	ND	0.100	ND	0.601		5
1,4-Dichlorobenzene	ND	0.100	ND	0.601		5
sec-Butylbenzene	ND	2.50	ND	13.7		5
p-Isopropyltoluene	ND	2.50	ND	13.7		5
1,2-Dichlorobenzene	ND	0.100	ND	0.601		5
n-Butylbenzene	ND	2.50	ND	13.7		5

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005820
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005820-03
Client ID: MP-7
Sample Location: PROVIDENCE, RI
Matrix: Soil_Vapor
Anaytical Method: 48,TO-15-SIM
Analytical Date: 04/27/10 02:10
Analyst: RY

Date Collected: 04/21/10 11:50
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
Dichlorodifluoromethane	0.525	0.250	2.59	1.24		5
Chloromethane	ND	2.50	ND	12.2		5
Vinyl chloride	ND	0.100	ND	0.255		5
Chloroethane	0.115	0.100	0.303	0.264		5
Acetone	111	10.0	263	23.7		5
Trichlorofluoromethane	0.860	0.250	4.83	1.40		5
Acrylonitrile	ND	2.50	ND	5.42		5
1,1-Dichloroethene	ND	0.100	ND	0.396		5
Methylene chloride	ND	2.50	ND	8.68		5
trans-1,2-Dichloroethene	ND	0.100	ND	0.396		5
1,1-Dichloroethane	ND	0.100	ND	0.404		5
Methyl tert butyl ether	ND	0.100	ND	0.360		5
2-Butanone	11.7	2.50	34.6	7.37		5
cis-1,2-Dichloroethene	ND	0.100	ND	0.396		5
Chloroform	ND	0.100	ND	0.488		5
1,2-Dichloroethane	ND	0.100	ND	0.404		5
1,1,1-Trichloroethane	ND	0.100	ND	0.545		5
Benzene	ND	0.500	ND	1.60		5
Carbon tetrachloride	ND	0.100	ND	0.629		5
1,2-Dichloropropane	ND	0.100	ND	0.462		5
Bromodichloromethane	ND	0.100	ND	0.670		5
Trichloroethene	0.175	0.100	0.940	0.537		5
cis-1,3-Dichloropropene	ND	0.100	ND	0.453		5
4-Methyl-2-pentanone	ND	2.50	ND	10.2		5
trans-1,3-Dichloropropene	ND	0.100	ND	0.453		5

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005820
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005820-03
Client ID: MP-7
Sample Location: PROVIDENCE, RI
Date Collected: 04/21/10 11:50
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
1,1,2-Trichloroethane	ND	0.100	ND	0.545		5
Toluene	0.995	0.100	3.75	0.376		5
Dibromochloromethane	ND	0.100	ND	0.480		5
1,2-Dibromoethane	ND	0.100	ND	0.768		5
Tetrachloroethene	5.24	0.100	35.5	0.678		5
1,1,1,2-Tetrachloroethane	ND	0.100	ND	0.686		5
Chlorobenzene	ND	0.100	ND	0.460		5
Ethylbenzene	0.415	0.100	1.80	0.434		5
p/m-Xylene	1.06	0.200	4.58	0.868		5
Bromoform	ND	0.100	ND	1.03		5
Styrene	ND	0.100	ND	0.426		5
1,1,2,2-Tetrachloroethane	ND	0.100	ND	0.686		5
o-Xylene	0.360	0.100	1.56	0.434		5
Isopropylbenzene	ND	2.50	ND	12.3		5
1,3,5-Trimethybenzene	ND	0.100	ND	0.491		5
1,2,4-Trimethylbenzene	0.290	0.100	1.42	0.491		5
1,3-Dichlorobenzene	ND	0.100	ND	0.601		5
1,4-Dichlorobenzene	ND	0.100	ND	0.601		5
sec-Butylbenzene	ND	2.50	ND	13.7		5
p-Isopropyltoluene	ND	2.50	ND	13.7		5
1,2-Dichlorobenzene	ND	0.100	ND	0.601		5
n-Butylbenzene	ND	2.50	ND	13.7		5

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005820
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005820-04
Client ID: MP-8
Sample Location: PROVIDENCE, RI
Matrix: Soil_Vapor
Anaytical Method: 48,TO-15-SIM
Analytical Date: 04/27/10 02:48
Analyst: RY

Date Collected: 04/21/10 12:10
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
Dichlorodifluoromethane	0.445	0.250	2.20	1.24		5
Chloromethane	ND	2.50	ND	12.2		5
Vinyl chloride	ND	0.100	ND	0.255		5
Chloroethane	0.115	0.100	0.303	0.264		5
Acetone	1210	10.0	2870	23.7	E	5
Trichlorofluoromethane	ND	0.250	ND	1.40		5
Acrylonitrile	ND	2.50	ND	5.42		5
1,1-Dichloroethene	ND	0.100	ND	0.396		5
Methylene chloride	ND	2.50	ND	8.68		5
trans-1,2-Dichloroethene	ND	0.100	ND	0.396		5
1,1-Dichloroethane	ND	0.100	ND	0.404		5
Methyl tert butyl ether	ND	0.100	ND	0.360		5
2-Butanone	623	2.50	1840	7.37	E	5
cis-1,2-Dichloroethene	ND	0.100	ND	0.396		5
Chloroform	ND	0.100	ND	0.488		5
1,2-Dichloroethane	ND	0.100	ND	0.404		5
1,1,1-Trichloroethane	ND	0.100	ND	0.545		5
Benzene	0.505	0.500	1.61	1.60		5
Carbon tetrachloride	ND	0.100	ND	0.629		5
1,2-Dichloropropane	ND	0.100	ND	0.462		5
Bromodichloromethane	ND	0.100	ND	0.670		5
Trichloroethene	ND	0.100	ND	0.537		5
cis-1,3-Dichloropropene	ND	0.100	ND	0.453		5
4-Methyl-2-pentanone	ND	2.50	ND	10.2		5
trans-1,3-Dichloropropene	ND	0.100	ND	0.453		5

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005820
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005820-04
Client ID: MP-8
Sample Location: PROVIDENCE, RI
Date Collected: 04/21/10 12:10
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
1,1,2-Trichloroethane	ND	0.100	ND	0.545		5
Toluene	1.38	0.100	5.20	0.376		5
Dibromochloromethane	ND	0.100	ND	0.480		5
1,2-Dibromoethane	ND	0.100	ND	0.768		5
Tetrachloroethene	5.44	0.100	36.8	0.678		5
1,1,1,2-Tetrachloroethane	ND	0.100	ND	0.686		5
Chlorobenzene	ND	0.100	ND	0.460		5
Ethylbenzene	0.405	0.100	1.76	0.434		5
p/m-Xylene	1.00	0.200	4.34	0.868		5
Bromoform	ND	0.100	ND	1.03		5
Styrene	ND	0.100	ND	0.426		5
1,1,2,2-Tetrachloroethane	ND	0.100	ND	0.686		5
o-Xylene	0.325	0.100	1.41	0.434		5
Isopropylbenzene	ND	2.50	ND	12.3		5
1,3,5-Trimethybenzene	ND	0.100	ND	0.491		5
1,2,4-Trimethylbenzene	0.230	0.100	1.13	0.491		5
1,3-Dichlorobenzene	ND	0.100	ND	0.601		5
1,4-Dichlorobenzene	ND	0.100	ND	0.601		5
sec-Butylbenzene	ND	2.50	ND	13.7		5
p-Isopropyltoluene	ND	2.50	ND	13.7		5
1,2-Dichlorobenzene	ND	0.100	ND	0.601		5
n-Butylbenzene	ND	2.50	ND	13.7		5

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005820
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005820-04 D
Client ID: MP-8
Sample Location: PROVIDENCE, RI
Matrix: Soil_Vapor
Anaytical Method: 48,TO-15-SIM
Analytical Date: 04/28/10 09:38
Analyst: RY

Date Collected: 04/21/10 12:10
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
Acetone	1890	153	4480	362		76.3
2-Butanone	874	38.2	2580	112		76.3

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005820
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005820-05
Client ID: IMP-1
Sample Location: PROVIDENCE, RI
Matrix: Soil_Vapor
Anaytical Method: 48,TO-15-SIM
Analytical Date: 04/28/10 06:48
Analyst: RY

Date Collected: 04/21/10 10:18
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
Dichlorodifluoromethane	0.535	0.050	2.64	0.247		1
Chloromethane	ND	0.500	ND	2.44		1
Vinyl chloride	ND	0.020	ND	0.051		1
Chloroethane	ND	0.020	ND	0.053		1
Acetone	30.7	2.00	72.8	4.75		1
Trichlorofluoromethane	0.882	0.050	4.95	0.281		1
Acrylonitrile	ND	0.500	ND	1.08		1
1,1-Dichloroethene	ND	0.020	ND	0.079		1
Methylene chloride	ND	0.500	ND	1.74		1
trans-1,2-Dichloroethene	ND	0.020	ND	0.079		1
1,1-Dichloroethane	ND	0.020	ND	0.081		1
Methyl tert butyl ether	ND	0.020	ND	0.072		1
2-Butanone	5.72	0.500	16.8	1.47		1
cis-1,2-Dichloroethene	ND	0.020	ND	0.079		1
Chloroform	0.045	0.020	0.220	0.098		1
1,2-Dichloroethane	ND	0.020	ND	0.081		1
1,1,1-Trichloroethane	0.020	0.020	0.109	0.109		1
Benzene	0.199	0.100	0.635	0.319		1
Carbon tetrachloride	0.097	0.020	0.610	0.126		1
1,2-Dichloropropane	ND	0.020	ND	0.092		1
Bromodichloromethane	ND	0.020	ND	0.134		1
Trichloroethene	0.166	0.020	0.891	0.107		1
cis-1,3-Dichloropropene	ND	0.020	ND	0.091		1
4-Methyl-2-pentanone	ND	0.500	ND	2.05		1
trans-1,3-Dichloropropene	ND	0.020	ND	0.091		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005820
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005820-05
Client ID: IMP-1
Sample Location: PROVIDENCE, RI
Date Collected: 04/21/10 10:18
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
1,1,2-Trichloroethane	ND	0.020	ND	0.109		1
Toluene	0.754	0.020	2.84	0.075		1
Dibromochloromethane	ND	0.020	ND	0.096		1
1,2-Dibromoethane	ND	0.020	ND	0.154		1
Tetrachloroethene	9.17	0.020	62.1	0.136		1
1,1,1,2-Tetrachloroethane	ND	0.020	ND	0.137		1
Chlorobenzene	ND	0.020	ND	0.092		1
Ethylbenzene	0.490	0.020	2.12	0.087		1
p/m-Xylene	1.43	0.040	6.22	0.174		1
Bromoform	ND	0.020	ND	0.206		1
Styrene	0.113	0.020	0.481	0.085		1
1,1,2,2-Tetrachloroethane	ND	0.020	ND	0.137		1
o-Xylene	0.285	0.020	1.24	0.087		1
Isopropylbenzene	ND	0.500	ND	2.46		1
1,3,5-Trimethybenzene	0.036	0.020	0.177	0.098		1
1,2,4-Trimethylbenzene	0.133	0.020	0.653	0.098		1
1,3-Dichlorobenzene	ND	0.020	ND	0.120		1
1,4-Dichlorobenzene	0.545	0.020	3.27	0.120		1
sec-Butylbenzene	ND	0.500	ND	2.74		1
p-Isopropyltoluene	ND	0.500	ND	2.74		1
1,2-Dichlorobenzene	ND	0.020	ND	0.120		1
n-Butylbenzene	ND	0.500	ND	2.74		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005820
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005820-06
Client ID: IMP-3
Sample Location: PROVIDENCE, RI
Matrix: Soil_Vapor
Anaytical Method: 48,TO-15-SIM
Analytical Date: 04/27/10 07:33
Analyst: RY

Date Collected: 04/21/10 10:30
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
Dichlorodifluoromethane	0.492	0.050	2.43	0.247		1
Chloromethane	ND	0.500	ND	2.44		1
Vinyl chloride	ND	0.020	ND	0.051		1
Chloroethane	0.044	0.020	0.116	0.053		1
Acetone	30.9	2.00	73.4	4.75		1
Trichlorofluoromethane	0.975	0.050	5.47	0.281		1
Acrylonitrile	ND	0.500	ND	1.08		1
1,1-Dichloroethene	ND	0.020	ND	0.079		1
Methylene chloride	0.501	0.500	1.74	1.74		1
trans-1,2-Dichloroethene	ND	0.020	ND	0.079		1
1,1-Dichloroethane	ND	0.020	ND	0.081		1
Methyl tert butyl ether	ND	0.020	ND	0.072		1
2-Butanone	4.92	0.500	14.5	1.47		1
cis-1,2-Dichloroethene	ND	0.020	ND	0.079		1
Chloroform	0.041	0.020	0.200	0.098		1
1,2-Dichloroethane	ND	0.020	ND	0.081		1
1,1,1-Trichloroethane	ND	0.020	ND	0.109		1
Benzene	0.395	0.100	1.26	0.319		1
Carbon tetrachloride	0.080	0.020	0.503	0.126		1
1,2-Dichloropropane	ND	0.020	ND	0.092		1
Bromodichloromethane	ND	0.020	ND	0.134		1
Trichloroethene	0.374	0.020	2.01	0.107		1
cis-1,3-Dichloropropene	ND	0.020	ND	0.091		1
4-Methyl-2-pentanone	ND	0.500	ND	2.05		1
trans-1,3-Dichloropropene	ND	0.020	ND	0.091		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005820
Report Date: 04/29/10

SAMPLE RESULTS

Lab ID: L1005820-06
Client ID: IMP-3
Sample Location: PROVIDENCE, RI
Date Collected: 04/21/10 10:30
Date Received: 04/22/10
Field Prep: Not Specified

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab						
1,1,2-Trichloroethane	ND	0.020	ND	0.109		1
Toluene	1.35	0.020	5.08	0.075		1
Dibromochloromethane	ND	0.020	ND	0.096		1
1,2-Dibromoethane	ND	0.020	ND	0.154		1
Tetrachloroethene	5.33	0.020	36.1	0.136		1
1,1,1,2-Tetrachloroethane	ND	0.020	ND	0.137		1
Chlorobenzene	ND	0.020	ND	0.092		1
Ethylbenzene	0.360	0.020	1.56	0.087		1
p/m-Xylene	1.10	0.040	4.77	0.174		1
Bromoform	ND	0.020	ND	0.206		1
Styrene	0.136	0.020	0.579	0.085		1
1,1,2,2-Tetrachloroethane	ND	0.020	ND	0.137		1
o-Xylene	0.264	0.020	1.14	0.087		1
Isopropylbenzene	ND	0.500	ND	2.46		1
1,3,5-Trimethybenzene	0.042	0.020	0.206	0.098		1
1,2,4-Trimethylbenzene	0.143	0.020	0.702	0.098		1
1,3-Dichlorobenzene	ND	0.020	ND	0.120		1
1,4-Dichlorobenzene	0.472	0.020	2.84	0.120		1
sec-Butylbenzene	ND	0.500	ND	2.74		1
p-Isopropyltoluene	ND	0.500	ND	2.74		1
1,2-Dichlorobenzene	ND	0.020	ND	0.120		1
n-Butylbenzene	ND	0.500	ND	2 74		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005820
Report Date: 04/29/10

Method Blank Analysis
Batch Quality Control

Analytical Method: 48,TO-15-SIM
Analytical Date: 04/26/10 15:11

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab for sample(s): 02-04,06 Batch: WG409885-4						
Dichlorodifluoromethane	ND	0.050	ND	0.247		1
Chloromethane	ND	0.500	ND	2.44		1
Vinyl chloride	ND	0.020	ND	0.051		1
Chloroethane	ND	0.020	ND	0.053		1
Acetone	ND	2.00	ND	4.75		1
Trichlorofluoromethane	ND	0.050	ND	0.281		1
Acrylonitrile	ND	0.500	ND	1.08		1
1,1-Dichloroethene	ND	0.020	ND	0.079		1
Methylene chloride	ND	0.500	ND	1.74		1
trans-1,2-Dichloroethene	ND	0.020	ND	0.079		1
1,1-Dichloroethane	ND	0.020	ND	0.081		1
Methyl tert butyl ether	ND	0.020	ND	0.072		1
2-Butanone	ND	0.500	ND	1.47		1
cis-1,2-Dichloroethene	ND	0.020	ND	0.079		1
Chloroform	ND	0.020	ND	0.098		1
1,2-Dichloroethane	ND	0.020	ND	0.081		1
1,1,1-Trichloroethane	ND	0.020	ND	0.109		1
Benzene	ND	0.100	ND	0.319		1
Carbon tetrachloride	ND	0.020	ND	0.126		1
1,2-Dichloropropane	ND	0.020	ND	0.092		1
Bromodichloromethane	ND	0.020	ND	0.134		1
Trichloroethene	ND	0.020	ND	0.107		1
cis-1,3-Dichloropropene	ND	0.020	ND	0.091		1
4-Methyl-2-pentanone	ND	0.500	ND	2.05		1
trans-1,3-Dichloropropene	ND	0.020	ND	0.091		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005820
Report Date: 04/29/10

Method Blank Analysis
Batch Quality Control

Analytical Method: 48,TO-15-SIM
Analytical Date: 04/26/10 15:11

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab for sample(s): 02-04,06 Batch: WG409885-4						
1,1,2-Trichloroethane	ND	0.020	ND	0.109		1
Toluene	ND	0.020	ND	0.075		1
Dibromochloromethane	ND	0.020	ND	0.096		1
1,2-Dibromoethane	ND	0.020	ND	0.154		1
Tetrachloroethene	ND	0.020	ND	0.136		1
1,1,1,2-Tetrachloroethane	ND	0.020	ND	0.137		1
Chlorobenzene	ND	0.020	ND	0.092		1
Ethylbenzene	ND	0.020	ND	0.087		1
p/m-Xylene	ND	0.040	ND	0.174		1
Bromoform	ND	0.020	ND	0.206		1
Styrene	ND	0.020	ND	0.085		1
1,1,2,2-Tetrachloroethane	ND	0.020	ND	0.137		1
o-Xylene	ND	0.020	ND	0.087		1
Isopropylbenzene	ND	0.500	ND	2.46		1
1,3,5-Trimethybenzene	ND	0.020	ND	0.098		1
1,2,4-Trimethylbenzene	ND	0.020	ND	0.098		1
1,3-Dichlorobenzene	ND	0.020	ND	0.120		1
1,4-Dichlorobenzene	ND	0.020	ND	0.120		1
sec-Butylbenzene	ND	0.500	ND	2.74		1
p-Isopropyltoluene	ND	0.500	ND	2.74		1
1,2-Dichlorobenzene	ND	0.020	ND	0.120		1
n-Butylbenzene	ND	0.500	ND	2.74		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005820
Report Date: 04/29/10

Method Blank Analysis
Batch Quality Control

Analytical Method: 48,TO-15-SIM
Analytical Date: 04/27/10 17:37

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab for sample(s): 01,04-05 Batch: WG409885-9						
Dichlorodifluoromethane	ND	0.050	ND	0.247		1
Chloromethane	ND	0.500	ND	2.44		1
Vinyl chloride	ND	0.020	ND	0.051		1
Chloroethane	ND	0.020	ND	0.053		1
Acetone	ND	2.00	ND	4.75		1
Trichlorofluoromethane	ND	0.050	ND	0.281		1
Acrylonitrile	ND	0.500	ND	1.08		1
1,1-Dichloroethene	ND	0.020	ND	0.079		1
Methylene chloride	ND	0.500	ND	1.74		1
trans-1,2-Dichloroethene	ND	0.020	ND	0.079		1
1,1-Dichloroethane	ND	0.020	ND	0.081		1
Methyl tert butyl ether	ND	0.020	ND	0.072		1
2-Butanone	ND	0.500	ND	1.47		1
cis-1,2-Dichloroethene	ND	0.020	ND	0.079		1
Chloroform	ND	0.020	ND	0.098		1
1,2-Dichloroethane	ND	0.020	ND	0.081		1
1,1,1-Trichloroethane	ND	0.020	ND	0.109		1
Benzene	ND	0.100	ND	0.319		1
Carbon tetrachloride	ND	0.020	ND	0.126		1
1,2-Dichloropropane	ND	0.020	ND	0.092		1
Bromodichloromethane	ND	0.020	ND	0.134		1
Trichloroethene	ND	0.020	ND	0.107		1
cis-1,3-Dichloropropene	ND	0.020	ND	0.091		1
4-Methyl-2-pentanone	ND	0.500	ND	2.05		1
trans-1,3-Dichloropropene	ND	0.020	ND	0.091		1

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01
Lab Number: L1005820
Report Date: 04/29/10

Method Blank Analysis
Batch Quality Control

Analytical Method: 48,TO-15-SIM
Analytical Date: 04/27/10 17:37

Parameter	ppbV Results	ppbV RDL	ug/m3 Results	ug/m3 RDL	Qualifier	Dilution Factor
Volatile Organics in Air by SIM - Mansfield Lab for sample(s): 01,04-05 Batch: WG409885-9						
1,1,2-Trichloroethane	ND	0.020	ND	0.109		1
Toluene	ND	0.020	ND	0.075		1
Dibromochloromethane	ND	0.020	ND	0.096		1
1,2-Dibromoethane	ND	0.020	ND	0.154		1
Tetrachloroethene	ND	0.020	ND	0.136		1
1,1,1,2-Tetrachloroethane	ND	0.020	ND	0.137		1
Chlorobenzene	ND	0.020	ND	0.092		1
Ethylbenzene	ND	0.020	ND	0.087		1
p/m-Xylene	ND	0.040	ND	0.174		1
Bromoform	ND	0.020	ND	0.206		1
Styrene	ND	0.020	ND	0.085		1
1,1,2,2-Tetrachloroethane	ND	0.020	ND	0.137		1
o-Xylene	ND	0.020	ND	0.087		1
Isopropylbenzene	ND	0.500	ND	2.46		1
1,3,5-Trimethybenzene	ND	0.020	ND	0.098		1
1,2,4-Trimethylbenzene	ND	0.020	ND	0.098		1
1,3-Dichlorobenzene	ND	0.020	ND	0.120		1
1,4-Dichlorobenzene	ND	0.020	ND	0.120		1
sec-Butylbenzene	ND	0.500	ND	2.74		1
p-Isopropyltoluene	ND	0.500	ND	2.74		1
1,2-Dichlorobenzene	ND	0.020	ND	0.120		1
n-Butylbenzene	ND	0.500	ND	2.74		1

Lab Control Sample Analysis
Batch Quality Control

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01

Lab Number: L1005820
Report Date: 04/29/10

Parameter	LCS %Recovery	Qual	LCSD %Recovery	Qual	%Recovery Limits	RPD	Qual	RPD Limits
Volatile Organics in Air by SIM - Mansfield Lab Associated sample(s): 02-04,06 Batch: WG409885-3								
Dichlorodifluoromethane	96		-		70-130	-		25
Chloromethane	90		-		70-130	-		25
Vinyl chloride	99		-		70-130	-		25
Chloroethane	86		-		70-130	-		25
Acetone	88		-		70-130	-		25
Trichlorofluoromethane	96		-		70-130	-		25
Acrylonitrile	89		-		70-130	-		25
1,1-Dichloroethene	83		-		70-130	-		25
Methylene chloride	80		-		70-130	-		25
trans-1,2-Dichloroethene	75		-		70-130	-		25
1,1-Dichloroethane	78		-		70-130	-		25
Methyl tert butyl ether	73		-		70-130	-		25
2-Butanone	81		-		70-130	-		25
cis-1,2-Dichloroethene	76		-		70-130	-		25
Chloroform	82		-		70-130	-		25
1,2-Dichloroethane	78		-		70-130	-		25
1,1,1-Trichloroethane	82		-		70-130	-		25
Benzene	69	Q	-		70-130	-		25
Carbon tetrachloride	85		-		70-130	-		25
1,2-Dichloropropane	80		-		70-130	-		25
Bromodichloromethane	85		-		70-130	-		25

Lab Control Sample Analysis
Batch Quality Control

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01

Lab Number: L1005820
Report Date: 04/29/10

Parameter	LCS %Recovery	Qual	LCSD %Recovery	Qual	%Recovery Limits	RPD	Qual	RPD Limits
Volatile Organics in Air by SIM - Mansfield Lab Associated sample(s): 02-04,06 Batch: WG409885-3								
Trichloroethene	83		-		70-130	-		25
cis-1,3-Dichloropropene	79		-		70-130	-		25
4-Methyl-2-pentanone	97		-		70-130	-		25
trans-1,3-Dichloropropene	64	Q	-		70-130	-		25
1,1,2-Trichloroethane	81		-		70-130	-		25
Toluene	70		-		70-130	-		25
Dibromochloromethane	83		-		70-130	-		25
1,2-Dibromoethane	79		-		70-130	-		25
Tetrachloroethene	81		-		70-130	-		25
1,1,1,2-Tetrachloroethane	84		-		70-130	-		25
Chlorobenzene	79		-		70-130	-		25
Ethylbenzene	73		-		70-130	-		25
p/m-Xylene	77		-		70-130	-		25
Bromoform	85		-		70-130	-		25
Styrene	74		-		70-130	-		25
1,1,2,2-Tetrachloroethane	86		-		70-130	-		25
o-Xylene	78		-		70-130	-		25
Isopropylbenzene	82		-		70-130	-		25
1,3,5-Trimethylbenzene	82		-		70-130	-		25
1,2,4-Trimethylbenzene	88		-		70-130	-		25
1,3-Dichlorobenzene	87		-		70-130	-		25

Lab Control Sample Analysis

Batch Quality Control

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01

Lab Number: L1005820
Report Date: 04/29/10

Parameter	LCS %Recovery	Qual	LCSD %Recovery	Qual	%Recovery Limits	RPD	Qual	RPD Limits
Volatile Organics in Air by SIM - Mansfield Lab Associated sample(s): 02-04,06 Batch: WG409885-3								
1,4-Dichlorobenzene	85		-		70-130	-		25
sec-Butylbenzene	91		-		70-130	-		25
p-Isopropyltoluene	89		-		70-130	-		25
1,2-Dichlorobenzene	86		-		70-130	-		25
n-Butylbenzene	103		-		70-130	-		25
Volatile Organics in Air by SIM - Mansfield Lab Associated sample(s): 01,04-05 Batch: WG409885-8								
Dichlorodifluoromethane	94		-		70-130	-		25
Chloromethane	92		-		70-130	-		25
Vinyl chloride	101		-		70-130	-		25
Chloroethane	101		-		70-130	-		25
Acetone	99		-		70-130	-		25
Trichlorofluoromethane	103		-		70-130	-		25
Acrylonitrile	108		-		70-130	-		25
1,1-Dichloroethene	100		-		70-130	-		25
Methylene chloride	92		-		70-130	-		25

Lab Control Sample Analysis
Batch Quality Control

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01

Lab Number: L1005820
Report Date: 04/29/10

Parameter	LCS %Recovery	Qual	LCSD %Recovery	Qual	%Recovery Limits	RPD	Qual	RPD Limits
Volatile Organics in Air by SIM - Mansfield Lab Associated sample(s): 01,04-05 Batch: WG409885-8								
trans-1,2-Dichloroethene	86		-		70-130	-		25
1,1-Dichloroethane	93		-		70-130	-		25
Methyl tert butyl ether	85		-		70-130	-		25
2-Butanone	91		-		70-130	-		25
cis-1,2-Dichloroethene	90		-		70-130	-		25
Chloroform	96		-		70-130	-		25
1,2-Dichloroethane	96		-		70-130	-		25
1,1,1-Trichloroethane	92		-		70-130	-		25
Benzene	80		-		70-130	-		25
Carbon tetrachloride	94		-		70-130	-		25
1,2-Dichloropropane	96		-		70-130	-		25
Bromodichloromethane	95		-		70-130	-		25
Trichloroethene	93		-		70-130	-		25
cis-1,3-Dichloropropene	96		-		70-130	-		25
4-Methyl-2-pentanone	95		-		70-130	-		25
trans-1,3-Dichloropropene	81		-		70-130	-		25
1,1,2-Trichloroethane	99		-		70-130	-		25
Toluene	90		-		70-130	-		25
Dibromochloromethane	99		-		70-130	-		25
1,2-Dibromoethane	101		-		70-130	-		25
Tetrachloroethene	97		-		70-130	-		25

Lab Control Sample Analysis

Batch Quality Control

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01

Lab Number: L1005820
Report Date: 04/29/10

Parameter	LCS %Recovery	Qual	LCSD %Recovery	Qual	%Recovery Limits	RPD	Qual	RPD Limits
Volatile Organics in Air by SIM - Mansfield Lab Associated sample(s): 01,04-05 Batch: WG409885-8								
1,1,1,2-Tetrachloroethane	101		-		70-130	-		25
Chlorobenzene	101		-		70-130	-		25
Ethylbenzene	98		-		70-130	-		25
p/m-Xylene	103		-		70-130	-		25
Bromoform	104		-		70-130	-		25
Styrene	101		-		70-130	-		25
1,1,2,2-Tetrachloroethane	111		-		70-130	-		25
o-Xylene	103		-		70-130	-		25
Isopropylbenzene	104		-		70-130	-		25
1,3,5-Trimethylbenzene	107		-		70-130	-		25
1,2,4-Trimethylbenzene	114		-		70-130	-		25
1,3-Dichlorobenzene	115		-		70-130	-		25
1,4-Dichlorobenzene	113		-		70-130	-		25
sec-Butylbenzene	112		-		70-130	-		25
p-Isopropyltoluene	105		-		70-130	-		25
1,2-Dichlorobenzene	114		-		70-130	-		25
n-Butylbenzene	119		-		70-130	-		25

Lab Duplicate Analysis
Batch Quality Control

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01

Lab Number: L1005820
Report Date: 04/29/10

Parameter	Native Sample	Duplicate Sample	Units	RPD	Qual	RPD Limits
Volatile Organics in Air by SIM - Mansfield Lab Associated sample(s): 01-06 QC Batch ID: WG409885-5 QC Sample: L1005820-05 Client ID: IMP-1						
Dichlorodifluoromethane	0.535	0.479	ppbV	11		25
Chloromethane	ND	ND	ppbV	NC		25
Vinyl chloride	ND	ND	ppbV	NC		25
Chloroethane	ND	ND	ppbV	NC		25
Acetone	30.7	27.4	ppbV	11		25
Trichlorofluoromethane	0.882	0.791	ppbV	11		25
Acrylonitrile	ND	ND	ppbV	NC		25
1,1-Dichloroethene	ND	ND	ppbV	NC		25
Methylene chloride	ND	ND	ppbV	NC		25
trans-1,2-Dichloroethene	ND	ND	ppbV	NC		25
1,1-Dichloroethane	ND	ND	ppbV	NC		25
Methyl tert butyl ether	ND	ND	ppbV	NC		25
2-Butanone	5.72	5.17	ppbV	10		25
cis-1,2-Dichloroethene	ND	ND	ppbV	NC		25
Chloroform	0.045	0.041	ppbV	9		25
1,2-Dichloroethane	ND	ND	ppbV	NC		25
1,1,1-Trichloroethane	0.020	ND	ppbV	NC		25
Benzene	0.199	0.119	ppbV	50	Q	25
Carbon tetrachloride	0.097	0.077	ppbV	23		25

Lab Duplicate Analysis
Batch Quality Control

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01

Lab Number: L1005820
Report Date: 04/29/10

Parameter	Native Sample	Duplicate Sample	Units	RPD	RPD Limits
Volatile Organics in Air by SIM - Mansfield Lab Associated sample(s): 01-06 QC Batch ID: WG409885-5 QC Sample: L1005820-05 Client ID: IMP-1					
1,2-Dichloropropane	ND	ND	ppbV	NC	25
Bromodichloromethane	ND	ND	ppbV	NC	25
Trichloroethene	0.166	0.136	ppbV	20	25
cis-1,3-Dichloropropene	ND	ND	ppbV	NC	25
4-Methyl-2-pentanone	ND	ND	ppbV	NC	25
trans-1,3-Dichloropropene	ND	ND	ppbV	NC	25
1,1,2-Trichloroethane	ND	ND	ppbV	NC	25
Toluene	0.754	0.610	ppbV	21	25
Dibromochloromethane	ND	ND	ppbV	NC	25
1,2-Dibromoethane	ND	ND	ppbV	NC	25
Tetrachloroethene	9.17	7.50	ppbV	20	25
1,1,1,2-Tetrachloroethane	ND	ND	ppbV	NC	25
Chlorobenzene	ND	ND	ppbV	NC	25
Ethylbenzene	0.490	0.408	ppbV	18	25
p/m-Xylene	1.43	1.18	ppbV	19	25
Bromoform	ND	ND	ppbV	NC	25
Styrene	0.113	0.087	ppbV	23	25
1,1,2,2-Tetrachloroethane	ND	ND	ppbV	NC	25
o-Xylene	0.285	0.235	ppbV	19	25

Lab Duplicate Analysis
Batch Quality Control

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01

Lab Number: L1005820
Report Date: 04/29/10

Parameter	Native Sample	Duplicate Sample	Units	RPD	RPD Limits
Volatile Organics in Air by SIM - Mansfield Lab Associated sample(s): 01-06 QC Batch ID: WG409885-5 QC Sample: L1005820-05 Client ID: IMP-1					
Isopropylbenzene	ND	ND	ppbV	NC	25
1,3,5-Trimethybenzene	0.036	0.029	ppbV	22	25
1,2,4-Trimethylbenzene	0.133	0.108	ppbV	21	25
1,3-Dichlorobenzene	ND	ND	ppbV	NC	25
1,4-Dichlorobenzene	0.545	0.419	ppbV	24	25
sec-Butylbenzene	ND	ND	ppbV	NC	25
p-Isopropyltoluene	ND	ND	ppbV	NC	25
1,2-Dichlorobenzene	ND	ND	ppbV	NC	25
n-Butylbenzene	ND	ND	ppbV	NC	25

Project Name: ALVAREZ HIGH SCHOOL **Lab Number:** L1005820

Project Number: 14687.01 **Report Date:** 04/29/10

Canister and Flow Controller Information

Samplenum	Client ID	Media ID	Media Type	Cleaning Batch ID	Initial Pressure (in. Hg)	Pressure on Receipt (in. Hg)	Flow Out mL/min	Flow In mL/min	% RSD
L1005820-01	MP-2	0463	#90 SV		-	-	67	70	4
L1005820-01	MP-2	131	2.7L Can	L1005024	-29.3	-6.5	-	-	-
L1005820-02	MP-5	0449	#90 SV		-	-	69	72	4
L1005820-02	MP-5	1735	2.7L Can	L1005024	-29.3	-6.8	-	-	-
L1005820-03	MP-7	0062	#90 SV		-	-	67	70	4
L1005820-03	MP-7	360	2.7L Can	L1005024	-29.3	-3.7	-	-	-
L1005820-04	MP-8	0330	#90 SV		-	-	71	72	1
L1005820-04	MP-8	506	2.7L Can	I1005361	-29.3	-6.0	-	-	-
L1005820-05	IMP-1	0466	#90 SV		-	-	70	72	3
L1005820-05	IMP-1	450	2.7L Can	I1005361	-29.3	-5.6	-	-	-
L1005820-06	IMP-3	0368	#90 SV		-	-	69	70	1
L1005820-06	IMP-3	147	2.7L Can	L1005024	-29.3	0	-	-	-

Project Name: ALVAREZ HIGH SCHOOL
Project Number: 14687.01

Lab Number: L1005820
Report Date: 04/29/10

Sample Receipt and Container Information

Were project specific reporting limits specified? YES

Reagent H2O Preserved Vials Frozen on: NA

Cooler Information Custody Seal
Cooler
N/A Absent

Container Information

Container ID	Container Type	Cooler	pH	Temp deg C	Pres	Seal	Analysis
L1005820-01A	Canister - 2.7 Liter	N/A	N/A		NA	Absent	TO15-SIM(30)
L1005820-02A	Canister - 2.7 Liter	N/A	N/A		NA	Absent	TO15-SIM(30)
L1005820-03A	Canister - 2.7 Liter	N/A	N/A		NA	Absent	TO15-SIM(30)
L1005820-04A	Canister - 2.7 Liter	N/A	N/A		NA	Absent	TO15-SIM(30)
L1005820-05A	Canister - 2.7 Liter	N/A	N/A		NA	Absent	TO15-SIM(30)
L1005820-06A	Canister - 2.7 Liter	N/A	N/A		NA	Absent	TO15-SIM(30)

*Hold days indicated by values in parentheses

Project Name:	ALVAREZ HIGH SCHOOL	**Lab Number:**	L1005820
Project Number:	14687.01	**Report Date:**	04/29/10

GLOSSARY

Acronyms

EPA · Environmental Protection Agency.

LCS · Laboratory Control Sample: A sample matrix, free from the analytes of interest, spiked with verified known amounts of analytes or a material containing known and verified amounts of analytes.

LCSD · Laboratory Control Sample Duplicate: Refer to LCS.

MS · Matrix Spike Sample: A sample prepared by adding a known mass of target analyte to a specified amount of matrix sample for which an independent estimate of target analyte concentration is available.

MSD · Matrix Spike Sample Duplicate: Refer to MS.

NA · Not Applicable.

NC · Not Calculated: Term is utilized when one or more of the results utilized in the calculation are non-detect at the parameter's reporting unit.

NI · Not Ignitable.

RDL · Reported Detection Limit: The value at which an instrument can accurately measure an analyte at a specific concentration. The RDL includes any adjustments from dilutions, concentrations or moisture content, where applicable.

RPD · Relative Percent Difference: The results from matrix and/or matrix spike duplicates are primarily designed to assess the precision of analytical results in a given matrix and are expressed as relative percent difference (RPD). Values which are less than five times the reporting limit for any individual parameter are evaluated by utilizing the absolute difference between the values; although the RPD value will be provided in the report.

Terms

Analytical Method: Both the document from which the method originates and the analytical reference method. (Example: EPA 8260B is shown as 1,8260B.) The codes for the reference method documents are provided in the References section of the Addendum.

Data Qualifiers

A · Spectra identified as "Aldol Condensation Product".

B · The analyte was detected above the reporting limit in the associated method blank. Flag only applies to associated field samples that have detectable concentrations of the analyte at less than five times (5x) the concentration found in the blank. For MCP-related projects, flag only applies to associated field samples that have detectable concentrations of the analyte at less than ten times (10x) the concentration found in the blank. For DOD-related projects, flag only applies to associated field samples that have detectable concentrations of the analyte at less than ten times (10x) the concentration found in the blank AND the analyte was detected above one-half the reporting limit (or above the reporting limit for common lab contaminants) in the associated method blank.

D · Concentration of analyte was quantified from diluted analysis. Flag only applies to field samples that have detectable concentrations of the analyte.

E · Concentration of analyte exceeds the range of the calibration curve and/or linear range of the instrument.

H · The analysis of pH was performed beyond the regulatory-required holding time of 15 minutes from the time of sample collection.

P · The RPD between the results for the two columns exceeds the method-specified criteria.

Q · The quality control sample exceeds the associated acceptance criteria. Note: This flag is not applicable for matrix spike recoveries when the sample concentration is greater than 4x the spike added or for batch duplicate RPD when the sample concentrations are less than 5x the RDL. (Metals only.)

R · Analytical results are from sample re-analysis.

RE · Analytical results are from sample re-extraction.

J · Estimated value. This represents an estimated concentration for Tentatively Identified Compounds (TICs).

ND · Not detected at the reported detection limit (RDL) for the sample.

Report Format: Data Usability Report

Project Name:	ALVAREZ HIGH SCHOOL	**Lab Number:**	L1005820
Project Number:	14687.01	**Report Date:**	04/29/10

REFERENCES

48 Compendium of Methods for the Determination of Toxic Organic Compounds in Ambient Air. Second Edition. EPA/625/R-96/010b, January 1999.

LIMITATION OF LIABILITIES

Alpha Analytical performs services with reasonable care and diligence normal to the analytical testing laboratory industry. In the event of an error, the sole and exclusive responsibility of Alpha Woods Hole Labs shall be to re-perform the work at it's own expense. In no event shall Alpha Analytical be held liable for any incidental, consequential or special damages, including but not limited to, damages in any way connected with the use of, interpretation of, information or analysis provided by Alpha Woods Hole Labs.

We strongly urge our clients to comply with EPA protocol regarding sample volume, preservation, cooling, containers, sampling procedures, holding time and splitting of samples in the field.

Certificate/Approval Program Summary

Last revised December 15, 2009 – Mansfield Facility

The following list includes only those analytes/methods for which certification/approval is currently held.
For a complete listing of analytes for the referenced methods, please contact your Alpha Customer Service Representative.

Connecticut Department of Public Health Certificate/Lab ID: PH-0141.

Wastewater/Non-Potable Water (Inorganic Parameters: pH, Turbidity, Conductivity, Alkalinity, Aluminum, Antimony, Arsenic, Barium, Beryllium, Boron, Cadmium, Calcium, Chromium, Cobalt, Copper, Iron, Lead, Magnesium, Manganese, Mercury, Molybdenum, Nickel, Potassium, Selenium, Silver, Sodium, Strontium, Thallium, Tin, Vanadium, Zinc, Total Residue (Solids), Total Suspended Solids (non-filterable), Total Cyanide. Organic Parameters: PCBs, Organochlorine Pesticides, Technical Chlordane, Toxaphene, Acid Extractables, Benzidines, Phthalate Esters, Nitrosamines, Nitroaromatics & Isophorone, PAHs, Haloethers, Chlorinated Hydrocarbons, Volatile Organics.)

Solid Waste/Soil (Inorganic Parameters: pH, Aluminum, Antimony, Arsenic, Barium, Beryllium, Cadmium, Calcium, Chromium, Hexavalent Chromium, Cobalt, Copper, Iron, Lead, Magnesium, Manganese, Mercury, Molybdenum, Nickel, Potassium, Selenium, Silver, Sodium, Thallium, Vanadium, Zinc, Total Organic Carbon, Total Cyanide, Corrosivity, TCLP 1311. Organic Parameters: PCBs, Organochlorine Pesticides, Technical Chlordane, Toxaphene, Volatile Organics, Acid Extractables, Benzidines, Phthalates, Nitrosamines, Nitroaromatics & Cyclic Ketones, PAHs, Haloethers, Chlorinated Hydrocarbons.)

Florida Department of Health Certificate/Lab ID: E87814. ***NELAP Accredited.***

Non-Potable Water (Inorganic Parameters: SM2320B, EPA 120.1, SM2510B, EPA 245.1, EPA 150.1, EPA 160.2, SM2540D, EPA 335.2, SM2540G, EPA 180.1. Organic Parameters: EPA 625, 608.)

Solid & Chemical Materials (Inorganic Parameters: 6020, 7470, 7471, 9045, 9014. Organic Parameters: EPA 8260, 8270, 8082, 8081.)

Air & Emissions (EPA TO-15.)

Louisiana Department of Environmental Quality Certificate/Lab ID: 03090. ***NELAP Accredited.***

Non-Potable Water (Inorganic Parameters: EPA 120.1, 150.1, 160.2, 180.1, 200.8, 245.1, 310.1, 335.2, 608, 625, 1631, 3010, 3015, 3020, 6020, 9010, 9014, 9040, SM2320B, 2510B, 2540D, 2540G, 4500CN-E, 4500H-B, Organic Parameters: EPA 3510, 3580, 3630, 3640, 3660, 3665, 5030, 8015 (mod), 3570, 8081, 8082, 8260, 8270,)

Solid & Chemical Materials (Inorganic Parameters: 6020, 7196, 7470, 7471, 7474, 9010, 9014, 9040, 9045, 9060. Organic Parameters: EPA 8015 (mod), EPA 3570, 1311, 3050, 3051, 3060, 3580, 3630, 3640, 3660, 3665, 5035, 8081, 8082, 8260, 8270.)

Biological Tissue (Inorganic Parameters: EPA 6020. Organic Parameters: EPA 3570, 3510, 3610, 3630, 3640, 8270.)

Maine Department of Human Services Certificate/Lab ID: MA0030.

Wastewater (Inorganic Parameters: EPA 120.1, 300.0, SM 2320, 2510B, 2540C, 2540D, EPA 245.1. Organic Parameters: 608, 624.)

Massachusetts Department of Environmental Protection Certificate/Lab ID: M-MA030.

Non-Potable Water (Inorganic Parameters: SM4500H+B. Organic Parameters: EPA 624.)

New Hampshire Department of Environmental Services Certificate/Lab ID: 2206. ***NELAP Accredited.***

Non-Potable Water (Inorganic Parameters: EPA 200.8, 245.1, 1631E, 120.1, 150.1, 180.1, 310.1, 335.2, 160.2, SM2540D, 2540G, 4500CN-E, 4500H+B, 2320B, 2510B. Organic Parameters: EPA 625, 608.)

New Jersey Department of Environmental Protection Certificate/Lab ID: MA015. ***NELAP Accredited.***

Non-Potable Water (Inorganic Parameters: SW-846 1312, 3010, 3020A, 3015, 6020, SM2320B, EPA 200.8, SM2540C, 2540D, 2540G, EPA 120.1, SM2510B, EPA 180.1, 245.1, 1631E, SW-846 9040B, 6020, 9010B, 9014 Organic Parameters: EPA 608, 625, SW-846 3510C, 3580A, 5030B, 3035L, 5035H, 3630C, 3640A, 3660B, 3665A, 8081A, 8082 8260B, 8270C)

Solid & Chemical Materials (Inorganic Parameters: SW-846 6020, 9010B, 9014, 1311, 1312, 3050B, 3051, 3060A, 7196A, 7470A, 7471A, 9045C, 9060. Organic Parameters: SW-846 3580A, 5030B, 3035L, 5035H, 3630C, 3640A, 3660B, 3665A, 8081A, 8082, 8260B, 8270C, 3570, 8015B.)

Atmospheric Organic Parameters (EPA TO-15)

Biological Tissue (Inorganic Parameters: SW-846 6020 Organic Parameters: SW-846 8270C, 3510C, 3570, 3610B, 3630C, 3640A)

New York Department of Health Certificate/Lab ID: 11627. ***NELAP Accredited.***

Non-Potable Water (Inorganic Parameters: EPA 310.1, SM2320B, EPA 365.2, 160.1, EPA 160.2, SM2540D, EPA 200.8, 6020, 1631E, 245.1, 335.2, 9014, 150.1, 9040B, 120.1, SM2510B, EPA 376.2, 180.1, 9010B. Organic Parameters: EPA 624, 8260B, 8270C, 608, 8081A, 625, 8082, 3510C, 3511, 5030B.)

Solid & Hazardous Waste (Inorganic Parameters: EPA 9040B, 9045C, SW-846 Ch7 Sec 7.3, EPA 6020, 7196A, 7471A, 7474, 9014, 9040B, 9045C, 9010B. Organic Parameters: EPA 8260B, 8270C, 8081A, DRO 8015B, 8082, 1311, 3050B, 3580, 3050B, 3035, 3570, 3051, 5035, 5030B.)

Air & Emissions (EPA TO-15.)

Pennsylvania Department of Environmental Protection Certificate/Lab ID: 68-02089. ***NELAP Accredited.***

Non-Potable Water (Organic Parameters: EPA 5030B, EPA 8260)

Rhode Island Department of Health Certificate/Lab ID: LAO00299. ***NELAP Accredited via LA-DEQ.***

Refer to MA-DEP Certificate for Non-Potable Water.

Refer to LA-DEQ Certificate for Non-Potable Water.

Texas Commission of Environmental Quality Certificate/Lab ID: T104704419-08-TX. ***NELAP Accredited.***

Solid & Chemical Materials (Inorganic Parameters: EPA 6020, 7470, 7471, 1311, 7196, 9014, 9040, 9045, 9060. Organic Parameters: EPA 8015, 8270, 8260, 8081, 8082.)

U.S. Army Corps of Engineers

Department of Defense Certificate/Lab ID: L2217.01.

Non-Potable Water (Inorganic Parameters: EPA 3005A,3020, 6020, 245.1, 245.7, 1631E, 7470A, 7474, 9014, 120.1, 9050A, 180.1, SM4500H-B, 2320B, 2510B, 2540D,9040. Organic Parameters: EPA 3510C, 5030B, 9010B, 624, 8260B, 8270C, 8270 Alk-PAH, 8082, 8081A, 8015 (SHC), 8015 (DRO).)

Solid & Hazardous Waste (Inorganic Parameters: EPA 1311, 1312,3051, 6020, 747A, 7474, 9045C,9060, SM 2540G, ASTM D422-63. Organic Parameters: EPA 3580, 3570, 3540C, 5035, 8260B, 8270C, 8270 Alk-PAH, 8082, 8081A, 8015 (SHC), 8015 (DRO).

Air & Emissions (EPA TO-15.)

Analytes **Not Accredited by NELAP**
Certification is not available by NELAP for the following analytes: **8270C:** Biphenyl.

AIR ANALYSIS

PAGE 1 OF 1

CHAIN OF CUSTODY

ALPHA ANALYTICAL
320 Forbes Blvd, Mansfield, MA 02048
TEL: 508-822-9300 FAX: 508-822-3288

Date Rec'd In Lab:

ALPHA Job #: L1005820

04291016:40

Client Information

Client: EA ENGINEERING
Address: 2350 POST ROAD
WARWICK, RI 02886
Phone: (401) 736-3440
Fax:
Email: rmack@eaest.com
☐ These samples have been previously analyzed by Alpha

Project Information

Project Name: ALVAREZ SCHOOL
Project Location: PROVIDENCE, RI
Project #: 14687.01
Project Manager: FRANK POSTMA
ALPHA Quote #:

Turn-Around Time

☑ Standard ☐ RUSH (only confirmed if pre-approved)
Date Due: Time:

Report Information - Data Deliverables

☐ FAX
☐ ADEx
Criteria Checker: _____
(Default based on Regulatory Criteria Indicated)
Other Formats: _____
☑ EMAIL (standard pdf report)
☐ Additional Deliverables:
Report to: (if different than Project Manager)
RON MACK
rmack@eaest.com

Billing Information

☑ Same as Client info PO #:

Regulatory Requirements/Report Limits

State/Fed	Program	Criteria
CT	TARGET CONCENTRATIONS	AIR

Other Project Specific Requirements/Comments:

All Columns Below Must Be Filled Out

ALPHA Lab ID (Lab Use Only)	Sample ID	Collection Date	Start Time	End Time	Initial Vacuum	Final Vacuum	Sample Matrix*	Sampler's Initials	Can Size	I D Can	I D - Flow Controller	TO-14A by TO-15	TO-15	TO-15 SIM	APH	FIXED GASES	TO-13A	TO-4 / TO-10	Sample Comments (i.e. PID)
5820-1	MP-2	4/21/10	~~10:53~~ 9:53	11:29	30+	9	SV	RM	2.7L	131	0463			X					12 ppm
-2	MP-5		11:26	11:55	28	7		RM		1735	0449								1.2 ppm
-3	MP-7		11:15	11:50	30+	7		RM		360	0062								2.0 ppm
-4	MP-8		11:39	12:10	30+	9		RM		506	0330								11.3 ppm
-5	IMP-1		9:49	10:18	29.5	6		RM		450	0466								53 ppb
-6	IMP-3		9:57	10:30	29	1		RM		147	0368								49 ppb

*SAMPLE MATRIX CODES
AA = Ambient Air (Indoor/Outdoor)
SV = Soil Vapor/Landfill Gas/SVE
Other = Please Specify

Container Type

Please print clearly, legibly and completely. Samples can not be logged in and turnaround time clock will not start until any ambiguities are resolved. All samples submitted are subject to Alpha's Terms and Conditions. See reverse side.

Relinquished By:	Date/Time	Received By:	Date/Time:
	4/22/10 1:12		4/22/10 16:30

Form No: 101-02 (19-Jun-09)

Attachment D

Alpha Analytical
Reporting Limits Letter

April 30, 2010

To: Ron Mack
EA Engineering, Science, & Technology
2350 Post Road
Warwick, RI 02886

From: Katie O'Brien
Alpha Analytical
320 Forbes Blvd
Mansfield, MA 01581

Re: TO15 SIM Reporting Limits

Dear Ron,

As we communicated prior to the TO-15 SIM analyses completed for the Alvarez High School air samples collected on April 21nd; the SIM Reporting Limits achieved for the following compounds are the lowest that we can currently achieve at Alpha. Please note that these reporting limits are above the Draft Proposed CT RSR (Residential) Criteria for these compounds:

1,2-Dichloroethane SIM RL = 0.08 ug/m3
Ethylene Dibromide (a.k.a. 1,2-Dibromoethane) SIM RL = 0.15 ug/m3
1,1,1,2- Tetrachloroethane SIM RL = 0.14 ug/m3
1,1,2,2-Tetrachloroethane SIM RL = 0.14 ug/m3
Bromodichloromethane SIM RL = 0.13 ug/m3

Please don't hesitate to contact me at 508-844-4156 if you have any questions.

Best Regards,

Katie O'Brien

Attachment E

Operation and Maintenance Form

Alvarez High School - SSD & Interior Methane Monitoring System O&M Form

Date of O&M: 4/21/2010 Performed by: RGM/PT

PID/Methane Calibration? US Environmental (yes/no)

Date of Last Methane Sensor Filter Replacement: Feb 2010 Replaced this O&M Visit? No (yes/no)

General Status of SSD System: On-line

General Status of Methane Monitoring System: On-line

Eng. Cap/Fence Inspection Performed/Notes: Buffing and Cleaning Floors

Monitoring/ Sampling Location	Sub-slab or gauge vacuum	Air Velocity (fpm)	VOC Monitoring	Methane Monitoring			Air/Vapor Sample Collection						Comments/Notes (Ambient weather conditions, status of HVAC, possible monitoring/sampling interferences, etc .... continue on separate sheet if needed)
			PID (ppb)	Indoor Sensor (ppm)	(% Gas)	(% LEL)*	Summa Can ID	Controller ID	Start Time	Start Vac (inches Hg)	End Time	End Vac (inches Hg)	
Gymnasium	NA	NA	462.0	0	0	0	424	0090	0923	-30	0950	-6	
Cafeteria	NA	NA	56.0	0	0	0	1721	0262	0921	-29	0951	-1	
Kitchen Storage Room	NA	NA	0.0	0	0	0	1508	0360	0942	-29	1013	-1	
Elevator Hallway	NA	NA	0.0	0	0	0	546	0042	0924	-30+	0953	-8	
Room 145	NA	NA	4.0	0	0	0	478	0130	0928	-30+	0958	-5	
Room 152	NA	NA	2.0	0	0	0	372	0451	0928	-28	0958	-4	
Room 118	NA	NA	11.0	0	0	0	132	0124	0930	-30+	1003	-10.5	
Room 110	NA	NA	14.0	0	0	0	121	0173	0930	-30+	1005	-4	
MP-1	-0.06	NA	1.4 ppm	NA	0.2	4.0	—						
MP-2	-0.05	NA	12.0 ppm	NA	0	0.0	131	0463	1058	-30+	1129	-9	
MP-3	-0.04	NA	974.0	NA	0.3	6.0	—						
MP-4	-0.05	NA	2.6 ppm	NA	0.2	4.0	—						
MP-5	-0.05	NA	1.2	NA	0.1	2.0	1735	0449	1126	-28	1155	-7	
MP-6	-0.09	NA	3.0 ppm	NA	0.2	4.0	—						
MP-7	-0.15	NA	2.0 ppm	NA	0	0.0	360	0062	1115	-30+	1170	-7	
MP-8	-0.14	NA	11.3 ppm	NA	0.1	2.0	506	0330	1139	-30+	1210	-9	
IMP-1	-0.02	NA	53.0	NA	0	0.0	450	0466	949	-29.5	1018	-6	Water in annulus
IMP-2	-0.02	NA	14.0	NA	0	0.0	—						
IMP-3	-0.02	NA	49.0	NA	0	0.0	147	0368	0957	-29	1030	-1	
Roof-Top Fan 1	2.00	1725	308.0	NA	0.3	6.0	—						
Roof-Top Fan 2	1.90	2355	392.0	NA	0.2	4.0	—						
Roof-Top Fan 3	2.20	2375	116.0	NA	0	0.0	—						
Ambient Outdoor Air	NA	NA	11.0	NA	0.2	4.0	1717	0398	1103	-30+	1140	-4	

NA: not applicable.
NM: not monitored on this date
NS: not sampled on this date
* RIDEM Action Level for methane %LEL beneath the building is 10% and within the building is 1%. If these methane levels are exceeded, immediately notify EA Project Manager to initiate response protocol