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EPA

Staten Island/New Jersey Urban Air Toxics Assessment Project Report

Volume VI
Part B

Appendices

ACKNOWLEDGEMENTS

This report is a collaborative effort of the staffs of the Region II Office of the U.S. Environmental Protection Agency (EPA), the New Jersey Department of Environmental Protection and Energy, the New York State Department of Environmental Conservation, the New York State Department of Health, the University of Medicine and Dentistry of New Jersey and the College of Staten Island. The project was undertaken at the request of elected officials and other representatives of Staten Island concerned that emissions from neighboring industrial sources might be responsible for suspected excess cancer incidences in the area.

Other EPA offices that provided assistance included the Office of Air Quality Planning and Standards, which provided contract support and advice; and particularly the Atmospheric Research and Exposure Assessment Laboratory, which provided contract support, quality assurance materials, and sampling and analysis guidance, and participated in the quality assurance testing that provided a common basis of comparison for the volatile organic compound analyses. The Region II Office of Policy and Management and its counterparts in the States of New York and New Jersey processed the many grants and procurements, and assisted in routing funding to the project where it was needed.

The project was conceived and directed by Conrad Simon, Director of the Air and Waste Management Division, who organized and obtained the necessary federal funding.

Oversight of the overall project was provided by a Management Steering Committee and oversight of specific activities, by a Project Work Group. The members of these groups are listed in Volume II of the report. The Project Coordinators for EPA, Robert Kelly, Rudolph K. Kapichak, and Carol Bellizzi, were responsible for the final preparation of this document and for editing the materials provided by the project subcommittee chairs. William Baker facilitated the coordinators' work.

Drs. Edward Ferrand and, later, Dr. Theo. J. Kneip, working under contract for EPA, wrote several sections, coordinated others, and provided a technical review of the work.

The project was made possible by the strong commitment it received from its inception by Christopher Daggett as Regional Administrator (RA) for EPA Region II, and by the continuing support it received from William Muszynski as Acting RA and as Deputy RA, and from Constantine Sidamon-Eristoff, the current RA. The project has received considerable support from the other

project organizations via the Management Steering Committee, whose members are listed in Volume II.

PREFACE - DESCRIPTION OF THE STATEN ISLAND/NEW JERSEY URBAN AIR TOXICS ASSESSMENT PROJECT REPORT

This report describes a project undertaken by the States of New York and New Jersey and the United States Environmental Protection Agency with the assistance of the College of Staten Island, the University of Medicine and Dentistry of New Jersey and, as a contractor, the New Jersey Institute of Technology.

Volume I contains the historical basis for the project and a summary of Volumes II, III, IV, and V of the project report.

Volume II of the report lists the objectives necessary for achieving the overall purpose of the project, the organizational structure of the project, and the tasks and responsibilities assigned to the participants.

Volume III of the report presents the results and discussion of each portion of the project for ambient air. It includes monitoring data, the emission inventory, the results of the source identification analyses, and comparisons of the monitoring results with the results of other studies. Volume III is divided into Part A for volatile organic compounds, and Part B for metals, benzo[α]pyrene (BaP), and formaldehyde. Part B includes the quality assurance (QA) reports for the metals, BaP, and formaldehyde.

Volume IV presents the results and discussion for the indoor air study performed in this project. It contains the QA reports for the indoor air study, and a paper on the method for sampling formaldehyde.

Volume V presents the results of the detailed statistical analysis of the VOCs data, and the exposure and health risk analyses for the project.

Volume VI, in two parts, consists of information on air quality in the project area prior to the SI/NJ UATAP; quality assurance (QA) reports that supplement the QA information in Volume III, Parts A and B; the detailed workplans and QA plans of each of the technical subcommittees; the QA reports prepared by the organizations that analyzed the VOC samples; descriptions of the sampling sites; assessment of the meteorological sites; and a paper on emissions inventory development for publicly-owned treatment works.

The AIRS database is the resource for recovery of the daily data for the project. The quarterly summary reports from the sampling organizations are available on a computer diskette from the National Technical Information Service.

STATEN ISLAND/NEW JERSEY
URBAN AIR TOXICS ASSESSMENT PROJECT

VOLUME VI. APPENDICES

PART A, EPA/902/R-93-001g
PART B, EPA/902/R-93-001h

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New York State Department of Environmental Conservation

Division of Air Resources

Staten Island Toxics Study 1987 - 1989

Quality Assurance Summary

Prepared by
Bureau of Toxic Air Sampling
Bureau of Air Quality Surveillance
Bureau of Technical Services

April 1990

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INTRODUCTION

STATEN ISLAND TOXICS STUDY

The goal of the Staten Island Toxics Study was to provide an assessment of the magnitude and distribution of toxic air pollutants on Staten Island and nearby New Jersey counties and to develop the methodology to accomplish this task. This project, begun in 1987, was a cooperative effort involving various State and Federal agencies as well as academia, EPA, NYSDEC and NJDEP provided the funding for this project. EPA provided project coordination.

An integral element of this project has been the quality control/quality assurance procedures that were established from project inception. These procedures were performed for both sample collection and sample analysis. The Quality Assurance Section, in addition, performed independent flow audits on the sampling equipment and arranged, with assistance from EPA, to perform audits on the gas chromatograph used to analyze the samples collected.

The Staten Island Project allowed DEC to develop a VOC monitoring network that will be used throughout the State.

The developmental work completed during the 2 year project progressed from manual equipment and a basic plan to a fully automated laboratory analytical system and fully operational field sampling program.

The data obtained is generally very good with the first 2 quarters (4th '87 and 1st '88) being the weakest as we progressed on the learning curve. The 17 analytes monitored have proved to be an effective choice for statewide monitoring.

The only portion of our VOC plan that did not develop as planned was the realtime automated gas chromatograph station. Staff transfers and shortages ended any possibility of testing and developing a system for deployment in this project. Review of one year of auto GC data revealed it to be invalid because of valve and software malfunctions.

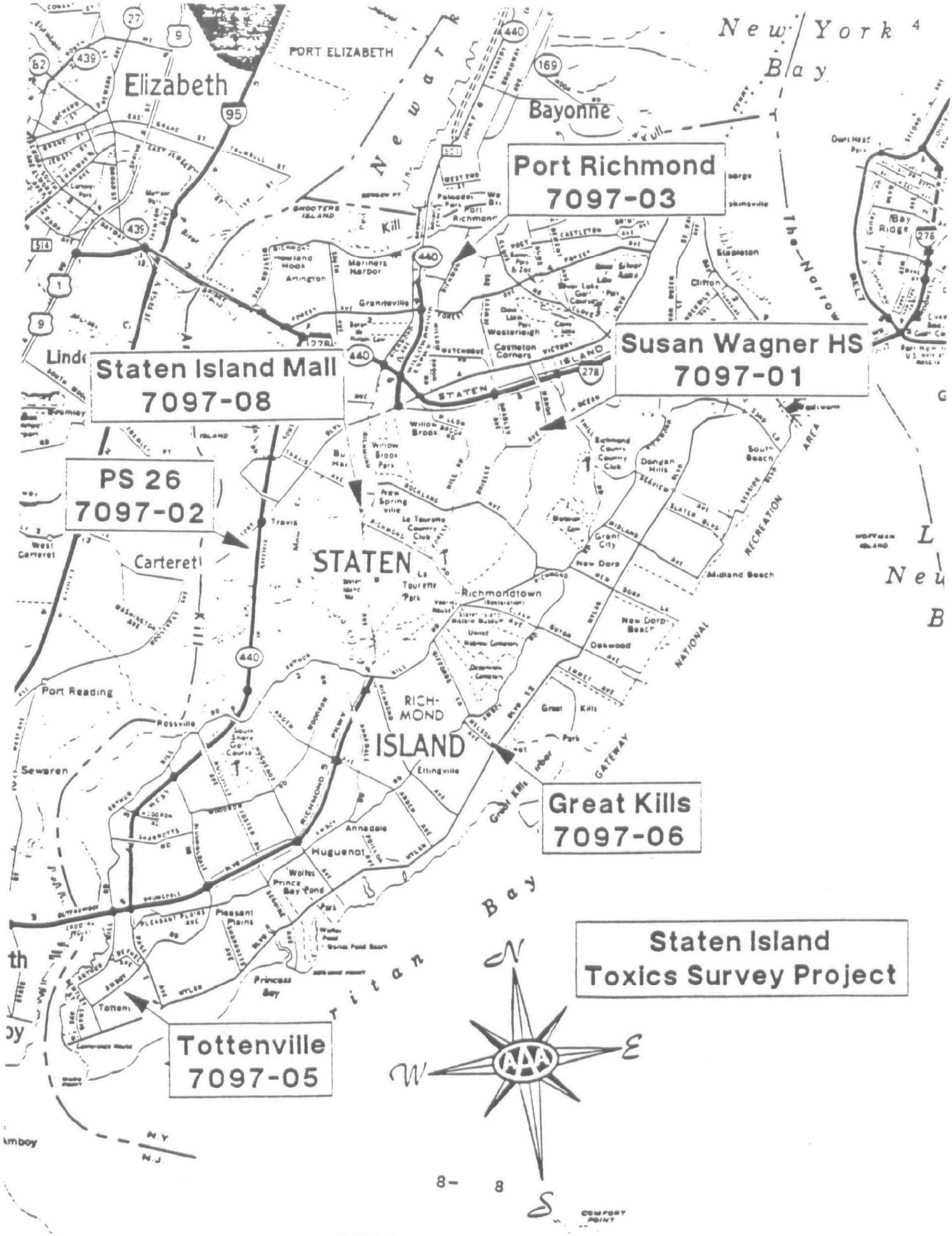
The NYSDEC VOC monitoring network by the end of 1989 encompasses 3 Bureaus and Region 2 for field sampling, analysis, calibration and quality assurance of the systems. General VOC accuracy is +/- 30% with some analytes better and some worse. A realistic goal of +/- 20% is possible as we correct individual analyte problems. A major accomplishment was the use of three sample tubes (2 hi, 1 lo flow) for each sample run. Comparison of the hi flow duplicates allows us to determine the 95% confidence limits of the measurement precision which ranged from 20% to 80%. Graphical presentation of the precision information is in the text.

Precision and accuracy numbers which range as high as +/- 50% may seem large while actually being very respectable for this low level ambient toxic air monitoring program. In many cases these

percentages reflect data differences of only fractions of a part per billion.

Following is a summary of the essential elements of the Quality Assurance, sample analysis and monitoring effort.

90-3-16


Port Richmond
7097-03
Susan Wagner HS
7097-01
Staten Island Mall
7097-08
PS 26
7097-02
Great Kills
7097-06
Tottenville
7097-05
Staten Island
Toxics Survey Project
Elizabeth
Bayonne
STATEN
ISLAND
RICH-
MOND
New York
Bay
Kill
Newark
Port Reading
Carteret
Sewaren
Tottenham
Princess Bay
Titan Bay
Great Kills
Midland Beach
Richmondtown
Port Richmond
Staten Island
Mariners Harbor
Arlington
Graniteville
Castleton Corners
Clifton
Stapleton
Bay Ridge
Dongan Hills
South Beach
Oakwood
Annadale
Huguenot
Pleasant Plains
Woodrow
Travis
Rossville
Elting ville
Willowbrook
Westerleigh
Grant City
New Dorp
New Dorp Beach
Midland Beach
Port Elizabeth
Shooters Island
Howland Hook
Linden
Perth Amboy
N.Y.
N.J.
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8- 8

Chapter 1

Summary of Field Blank Data

N.Y.S. DEC STATEN ISLAND PROJECT

SUMMARY OF FIELD BLANK DATA

Table 1-1 is a listing of the average blank values obtained during the S.I.N.J. monitoring project. The 2 analytical methods are separate as the adsorption tubes and desorbtion systems were very different.

During the 2nd quarter of 1988 the average of the last 5 blanks were subtracted from the sample prior to the calculations of the ppb concentration value. This process was then used for the duration of the project.

The ENV data shows a gradual increase in the benzene and toluene blanks. This is probably due to the aging of the tenax in the adsorbtion tubes used during the first year. The Envirochem System tubes were made of glass and during the study the tenax portion of the tubes became noticeably more brown with repeated cleaning and use.

The changeover to the ATD (Automatic Thermal Desportion) system brought brand new tubes and a storage system that was not as effective as the Envirochem's.

The O-ring sealed caps supplied by the manufacturer did not seal the samples as well or the teflon capped vials used by Envirochem.

TABLE 1-1 SUMMARY OF BLANKS IN ng (average)(std dev)

QTR & (#)	DICHLOROMETHANE	1,1,1 TRICHLOROETHANE	BENZENE	TOLUENE	ETHYLBENZENE	M,P-XYLENE	O-XYLENE	1,3 DICHLOBENZ
1987 DATA(23)	NF	NF	[65](16)	[29](10)	[21](17)	NF	[2](2)	NF
1ST QTR 88 ENV(14)	NF	NF	[64](18)	[16](6)	[7](4)	NF	NF	NF
2ND QTR 88 ENV(15)	NF	NF	[81](16)	[19](3)	[5](4)	[17](14)	NF	[2](1)
3RD QTR 88 ENV(7)	NF	NF	[87](30)	[23](8)	[4](2)	[3](5)	NF	[2](.2)
4TH QTR 88 ENV(13)	NF	NF	[92](23)	[26](10)	[5](2)	NF	NF	[3](1)
3RD+4TH 88 ATD(19)	[18](14)	[21](20)	[24](8)	[63](49)	[5](3)	[15](8)	[4](2)	[4](5)
1ST QTR 89 ATD(24)	[7](6)	[9](10)	[23](7)	[72](61)	[4](3)	[12](11)	[3](3)	[6](4)
2ND QTR 89 ATD(24)	[16](24)	[16](13)	[31](11)	[76](40)	[5](3)	[14](9)	[4](3)	[15](5)
3RD QTR 89 ATD(32)	[3](5)	[5](5)	[36](18)	[42](47)	[4](4)	[6](9)	[2](4)	[9](12)

Chapter 2

Duplicate Samples

SAMPLE PRECISION ANALYSIS

Duplicate samples were analyzed on the gas chromatograph routinely to evaluate sample analysis precision.

Every sampling event was done in triplicate. Two adsorption tubes at normal sample volume and one at a low sample volume for distributed volume statistics.

In reviewing the following data the following should be noted:

1. Even though the 95% confidence limits appear wide, the means are generally very low and the data will be used based upon computed annual averages.

2. These 95% confidence limits appear wide, the means are generally very low and the data will be used based upon computed annual averages.

3. Even errors of 50% represent fractions of a part per billion at the concentrations being measured.

Following is a summary of these precision results by quarter both in tabular and graphical form. Appendix C contains the details of this sample precision analysis.

Staten Island Toxics Study

4th Quarter 1987
Sample Precision Analysis

Envirochem

Compound	No.	Mean	Std. Dev.	Lover Limit	Upper Limit
Dichloromethane	15	5.4	24.4	-42	+53
Chloroform	3*				
1,2 Dichloroethane	2*				
1,1,1 Trichloroethane	22	2.0	16.7	-31	+35
Benzene	23	3.4	27.5	-51	+57
Carbon Tetrachloride	0*				
Trichloroethylene	4	8.3	15.3	-22	+38
1,1,2 Trichloroethane	0*				
Toluene	21	1.1	19.1	-36	+39
Tetrachloroethylene	20	0.4	18.2	-35	+36
Chlorobenzene	0*				
Ethylbenzene	21	-0.2	23.0	-45	+45
M,P-Xylene	23	3.0	17.2	-31	+37
O-Xylene	23	2.6	16.4	-30	+35
1,3 Dichlorobenzene	10	13.2	15.3	-17	+43
1,4 Dichlorobenzene	*0				
1,2 Dichlorobenzene	*0				

* Insufficient data pairs to evaluate statistically

Staten Island Toxics Study

2nd Quarter 1988
Sample Precision Analysis

Envirochem

Compound	No.	Mean	Std. Dev.	Lover Limit	Upper Limit
Dichloromethane	16	-2.7	13.9	-30	+24
Chloroform	14	-4.7	13.5	-31	+22
1,2 Dichloroethane	4	-0.5	11.7	-24	+23
1,1,1 Trichloroethane	35	1.7	13.2	-24	+28
Benzene	24	2.5	27.2	-51	+56
Carbon Tetrachloride	14	10.5	24.7	-38	+59
Trichloroethylene	17	-6.4	20.9	-47	+35
1,1,2 Trichloroethane	0*				
Toluene	34	-2.0	11.7	-25	+21
Tetrachloroethylene	28	1.5	5.6	-9	+12
Chlorobenzene	5	-10.2	14.1	-38	+17
Ethylbenzene	28	-0.2	10.5	-21	+20
M,P-Xylene	36	4.9	30.9	-56	+65
O-Xylene	33	1.0	6.0	-11	+13
1,3 Dichlorobenzene	19	0.5	7.5	-14	+15
1,4 Dichlorobenzene	0*				
1,2 Dichlorobenzene	0*				

* Insufficient data pairs to evaluate statistically

Staten Island Toxics Study

3rd Quarter 1988
Sample Precision Analysis

ATD

Compound	No.	Mean	Std. Dev.	Lower Limit	Upper Limit
Dichloromethane	6	13.7	18.8	-23	+51
Chloroform	6	2.7	38.1	-72	+77
1,2 Dichloroethane	1*				
1,1,1 Trichloroethane	6	12.2	38.0	-63	+87
Benzene	8	-9.5	47.7	-103	+84
Carbon Tetrachloride	0*				
Trichloroethylene	4	12.5	59.9	-105	+130
1,1,2 Trichloroethane	0*				
Toluene	9	11.1	20.0	-28	+50
Tetrachloroethylene	9	6.8	10.5	-14	+27
Chlorobenzene	0*				
Ethylbenzene	9	10.6	19.3	-27	+48
M,P-Xylene	9	7.4	14.5	-21	+35
O-Xylene	9	8.4	17.1	-25	+42
1,3 Dichlorobenzene	0*				
1,4 Dichlorobenzene	5	22.5	16.4	-10	+55
1,2 Dichlorobenzene	2*				

* Insufficient data pairs to evaluate statistically

Staten Island Toxics Study

4th Quarter 1988
Sample Precision Analysis

ATD

Compound	No.	Mean	Std. Dev.	Lower Limit	Upper Limit
Dichloromethane	33	1.4	18.3	-34	+37
Chloroform	27	-4.3	21.3	-46	+37
1,2 Dichloroethane	1*				
1,1,1 Trichloroethane	44	-8.4	42.0	-91	+74
Benzene	48	-0.8	36.8	-73	+71
Carbon Tetrachloride	9	-3.0	31.2	-64	+58
Trichloroethylene	31	1.5	36.7	-71	+73
1,1,2 Trichloroethane	0*				
Toluene	52	1.9	23.8	-45	+49
Tetrachloroethylene	46	1.1	13.6	-26	+28
Chlorobenzene	4*				
Ethylbenzene	50	-0.7	11.7	-24	+22
M,P-Xylene	52	2.4	16.0	-29	+34
O-Xylene	37	0.3	10.8	-21	+21
1,3 Dichlorobenzene	15	-3.8	22.3	-47	+40
1,4 Dichlorobenzene	5	-29	41.0	-108	+51
1,2 Dichlorobenzene	0*				

* Insufficient data pairs to evaluate statistically

Staten Island Toxics Study

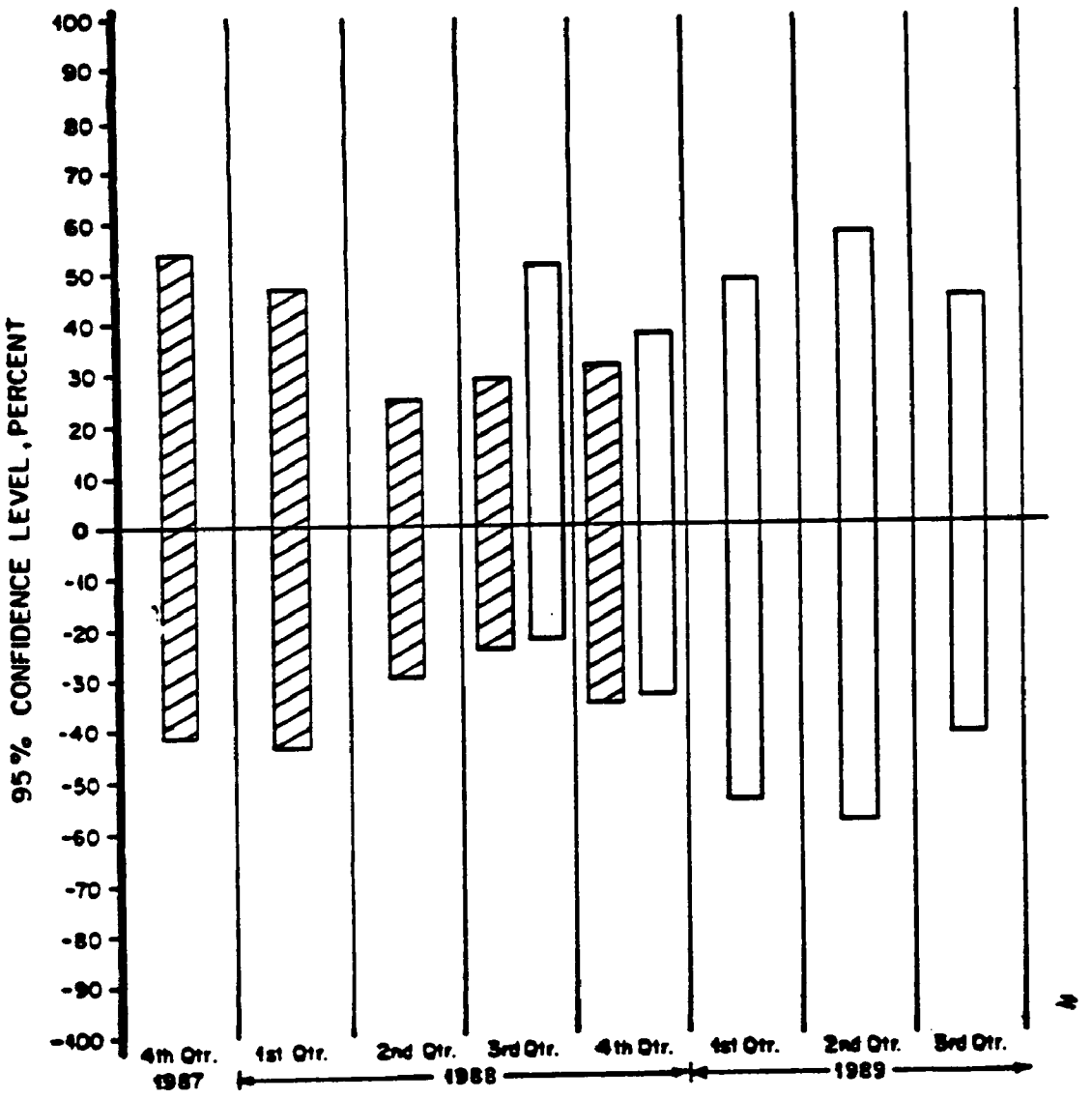
2nd Quarter 1989
Sample Precision Analysis

ATD

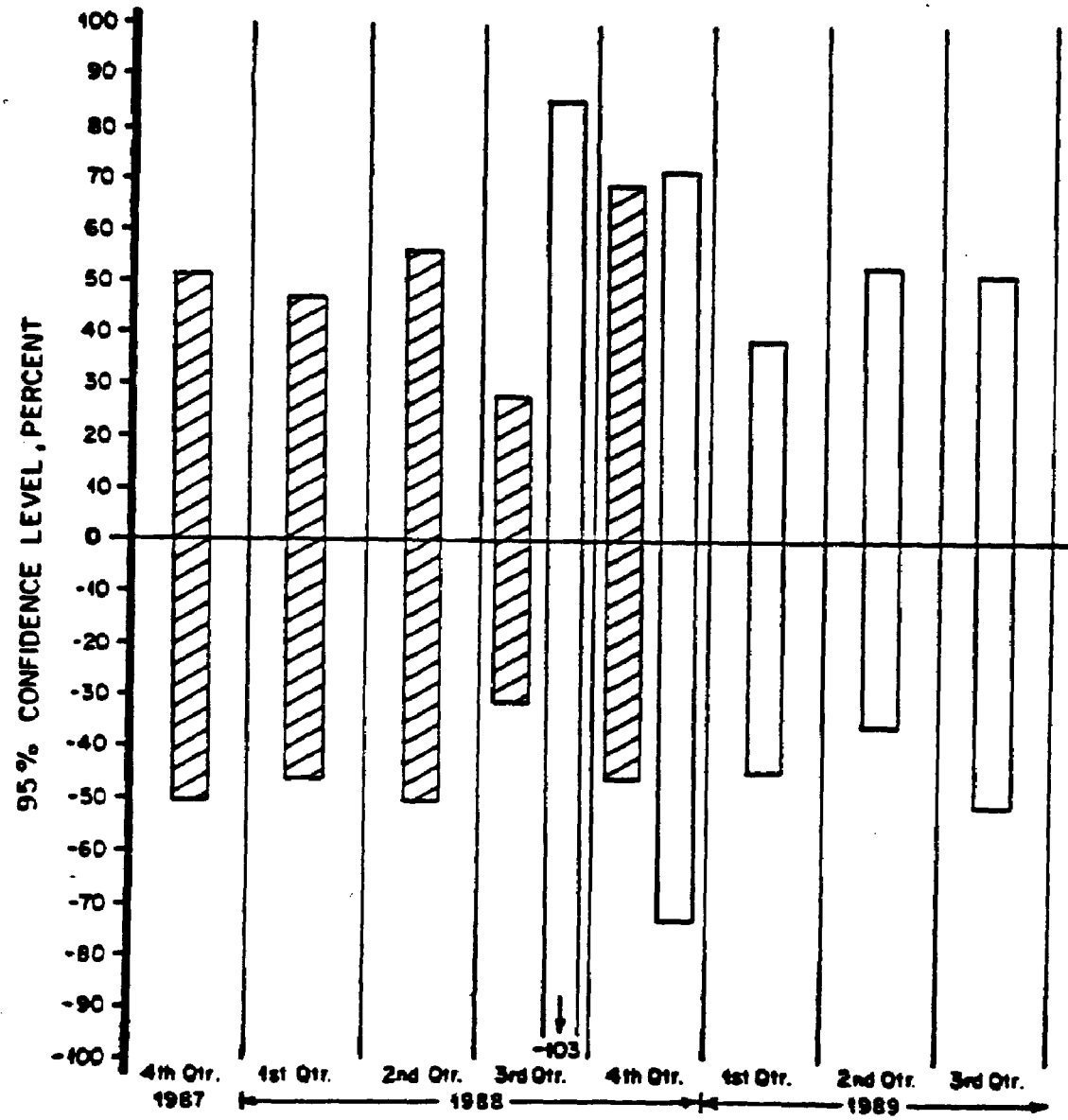
Compound	No.	Mean	Std. Dev.	Lower Limit	Upper Limit
Dichloromethane	36	-1.5	29.3	-59	+56
Chloroform	23	4.0	13.8	-23	+31
1,2 Dichloroethane	12	0.5	25.5	-51	+50
1,1,1 Trichloroethane	68	-1.0	25.3	-51	+48
Benzene	64	7.5	22.9	-37	+52
Carbon Tetrachloride	24	6.2	27.4	-47	+60
Trichloroethylene	60	-7.0	36.6	-79	+65
1,1,2 Trichloroethane	0*				
Toluene	73	5.8	33.3	-60	+71
Tetrachloroethylene	62	0.9	14.2	-27	+29
Chlorobenzene	8	301	21.4	-39	+45
Ethylbenzene	71	5.7	23.9	-41	+53
M,P-Xylene	73	-1.6	21.2	-43	+40
O-Xylene	73	2.2	13.4	-24	+28
1,3 Dichlorobenzene	39	-5.8	33.3	-71	+60
1,4 Dichlorobenzene	0*				
1,2 Dichlorobenzene	0*				

* Insufficient data pairs to evaluate statistically

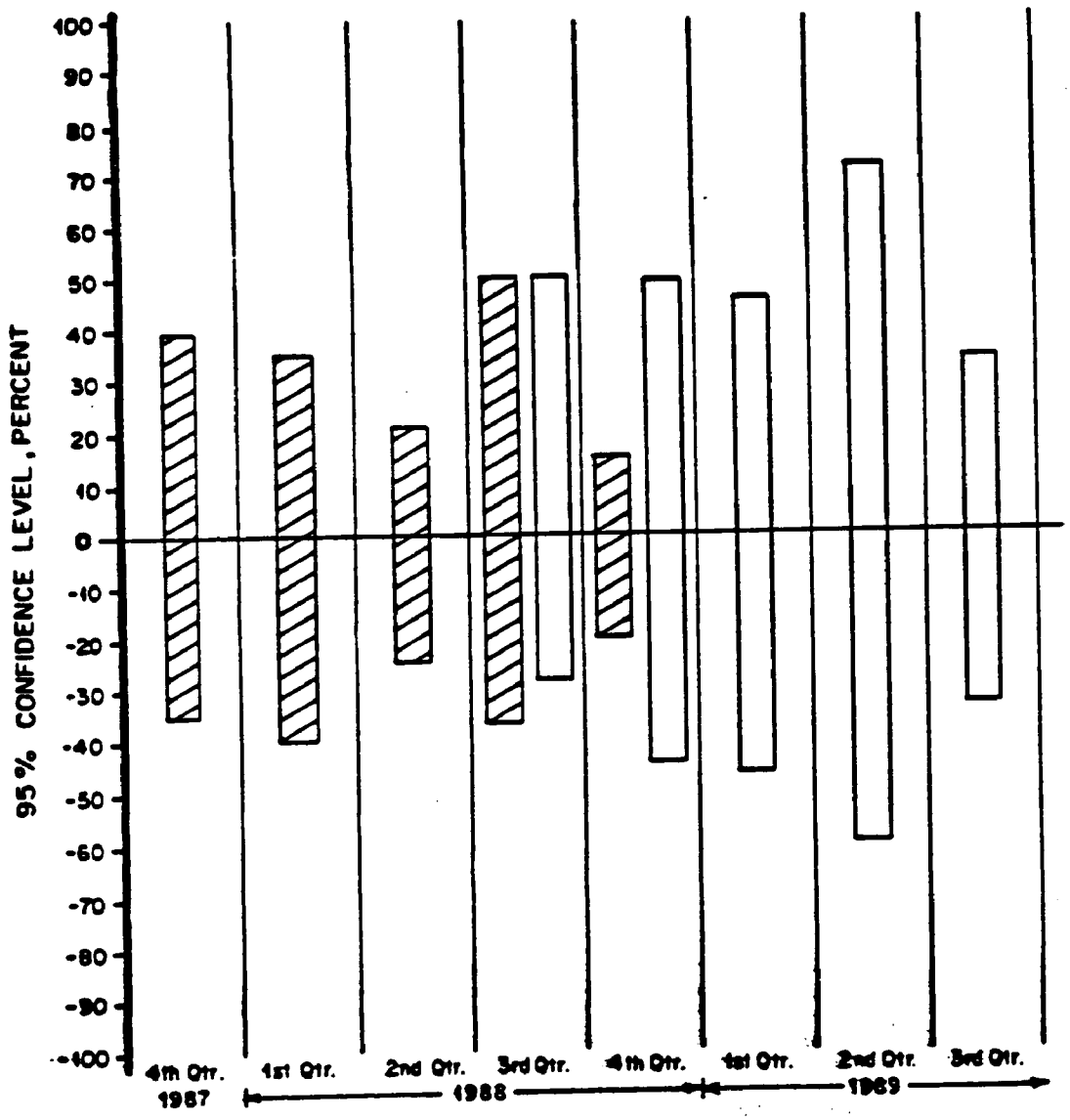
1987 – 1989
STATEN ISLAND TOXICS STUDY
SAMPLE PRECISION ANALYSIS
DICHLOROMETHANE



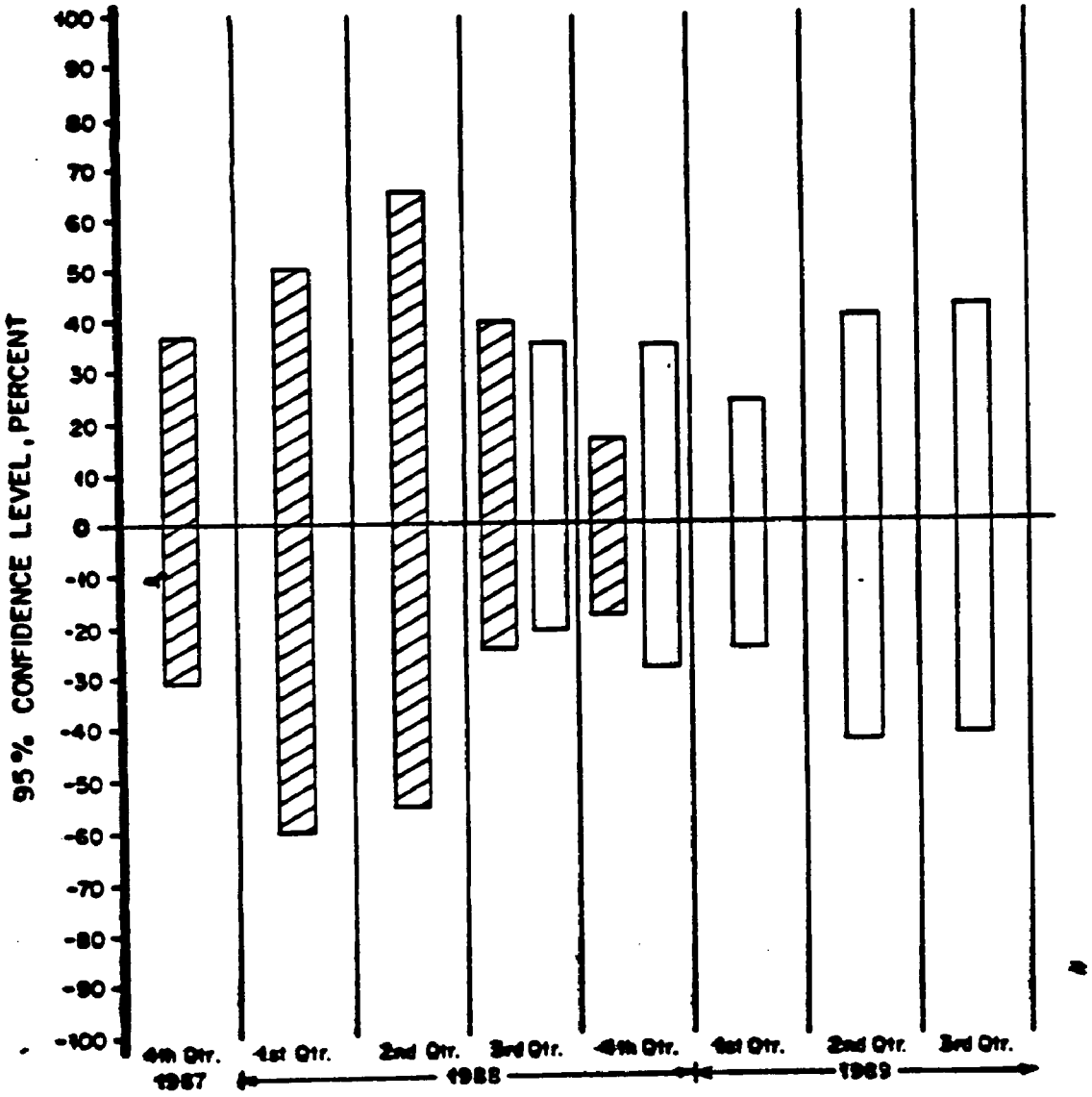
1987 - 1989
STATEN ISLAND TOXICS STUDY
SAMPLE PRECISION ANALYSIS
BENZENE



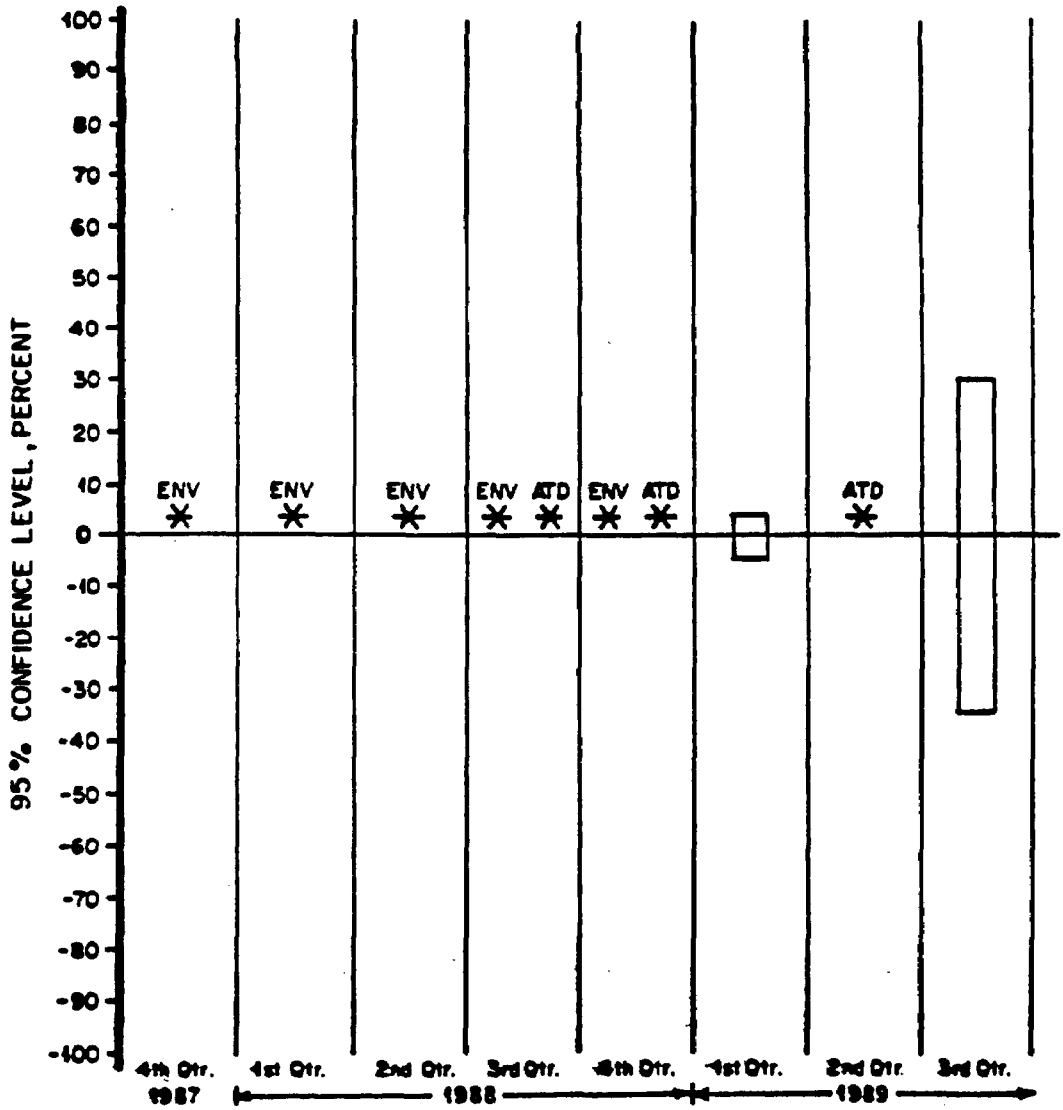
1987 - 1989
STATEN ISLAND TOXICS STUDY
SAMPLE PRECISION ANALYSIS
TOLUENE



1987 - 1989
STATEN ISLAND TOXICS STUDY
SAMPLE PRECISION ANALYSIS
M_1P-XYLENE



1987 - 1989
STATEN ISLAND TOXICS STUDY
SAMPLE PRECISION ANALYSIS
1,2 DICHLOROBENZENE



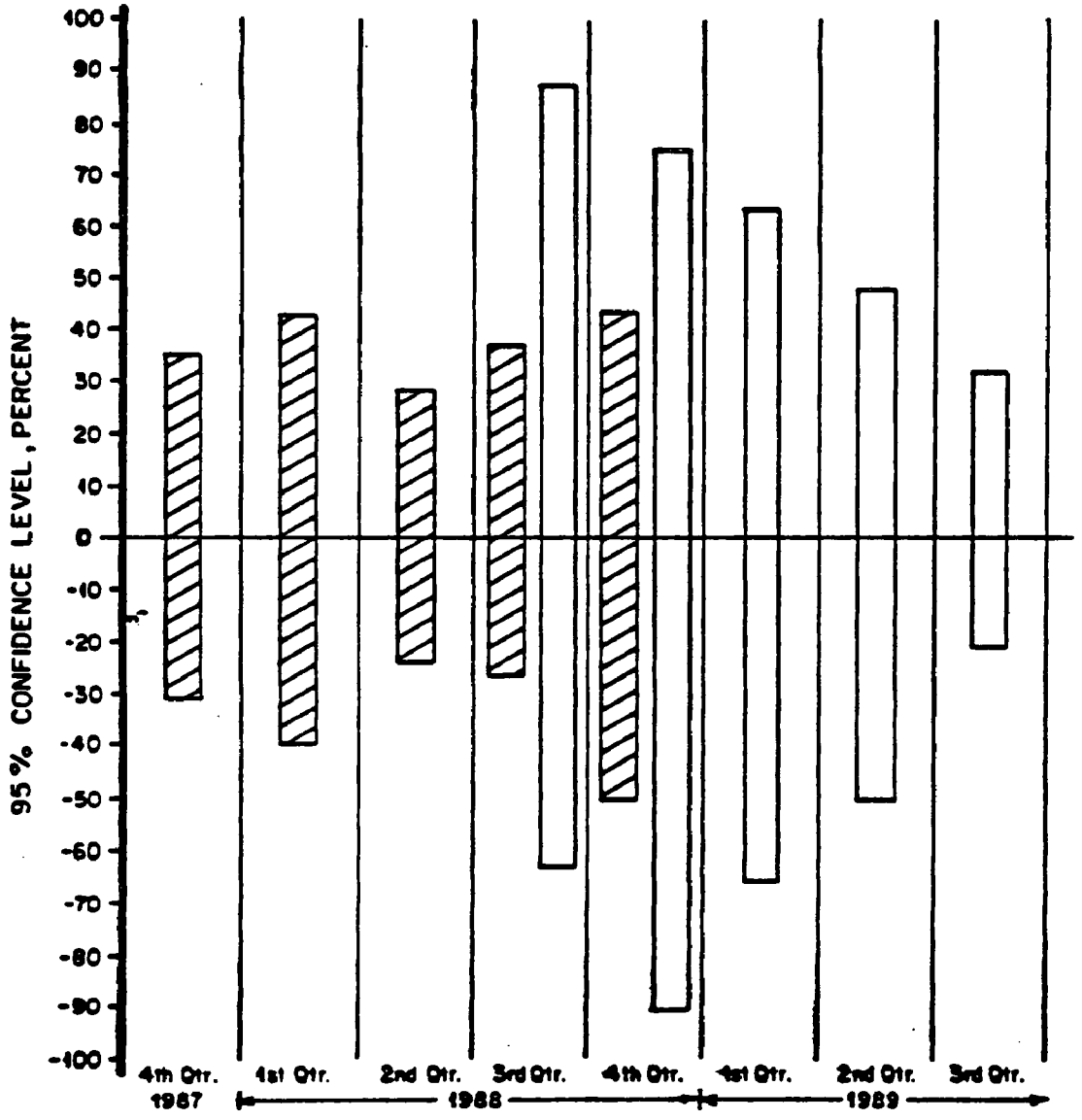
* INSUFFICIENT DATA PAIRS TO EVALUATE STATISTICALLY

SAMPLING METHOD

ENVIROCHEM

ATD

1987 – 1989
STATEN ISLAND TOXICS STUDY
SAMPLE PRECISION ANALYSIS
1,1,1 TRICHLOROETHANE



SAMPLING METHOD

ENVIROCHEM

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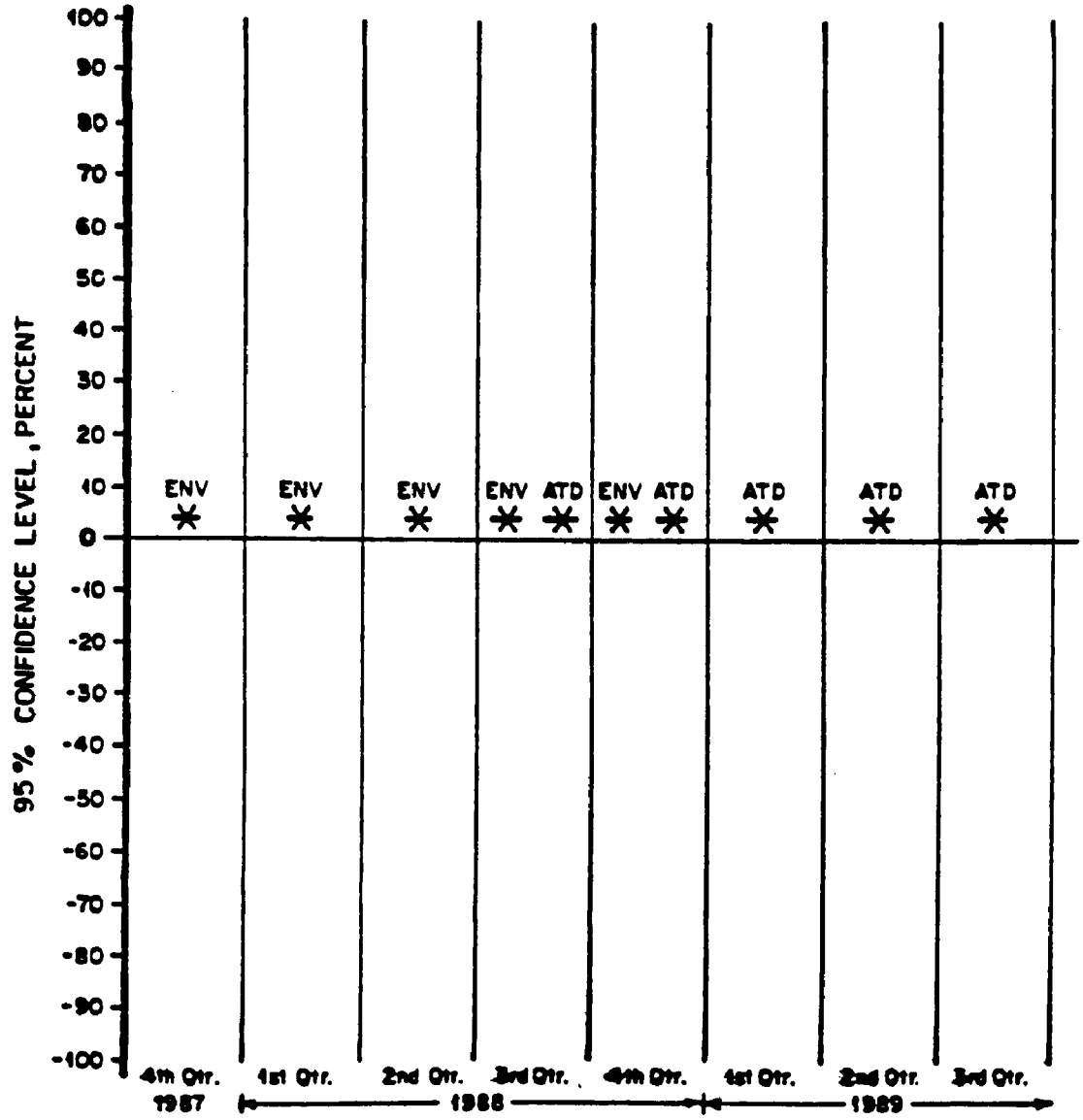
1987 - 1989
STATEN ISLAND TOXICS STUDY
SAMPLE PRECISION ANALYSIS
1,1,2 TRICHLOROETHANE



* INSUFFICIENT DATA PAIRS TO EVALUATE STATISTICALLY

SAMPLING METHOD

ENVIROCHEM

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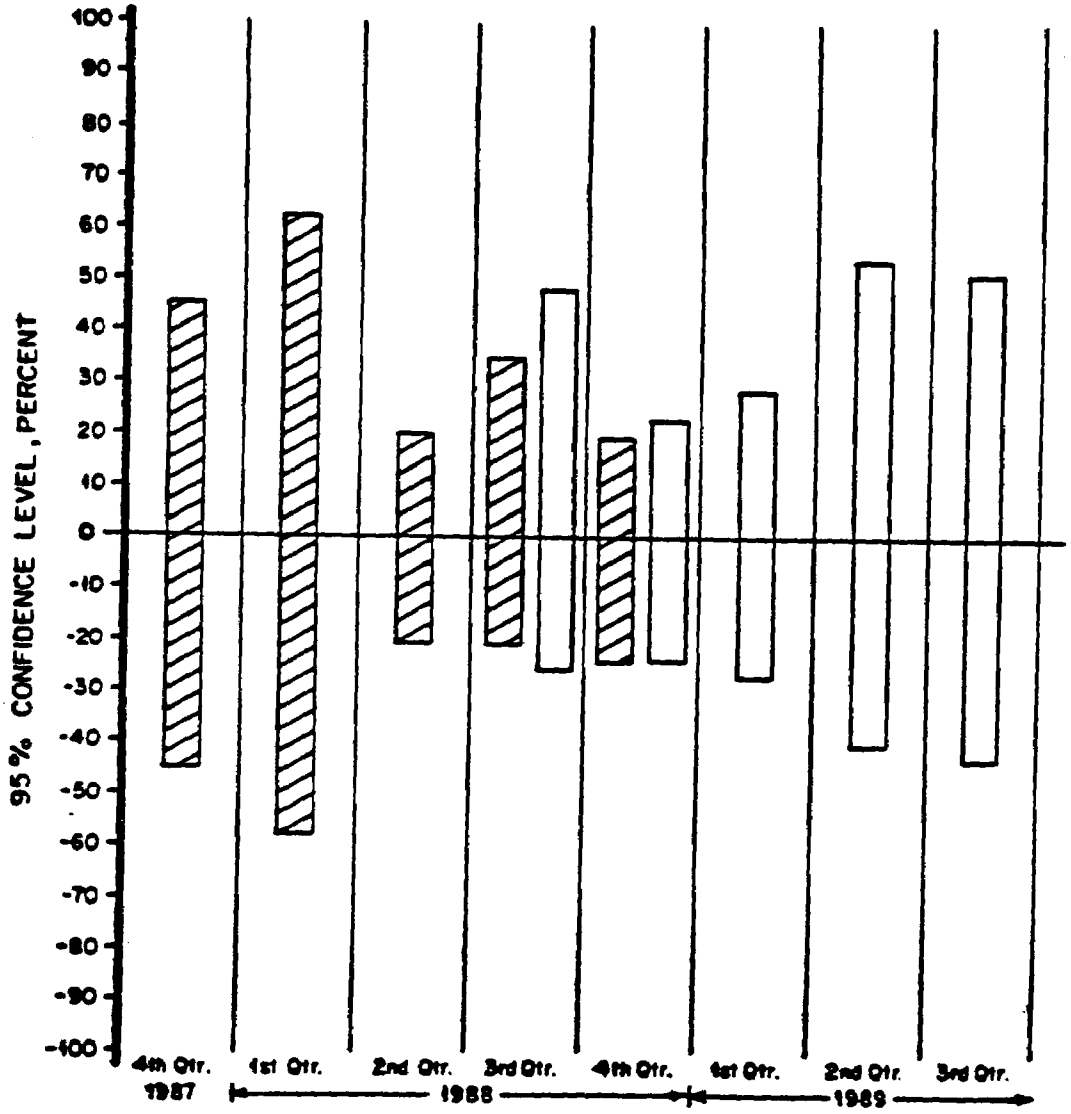
1987 - 1989
STATEN ISLAND TOXICS STUDY
SAMPLE PRECISION ANALYSIS
ETHYLBENZENE

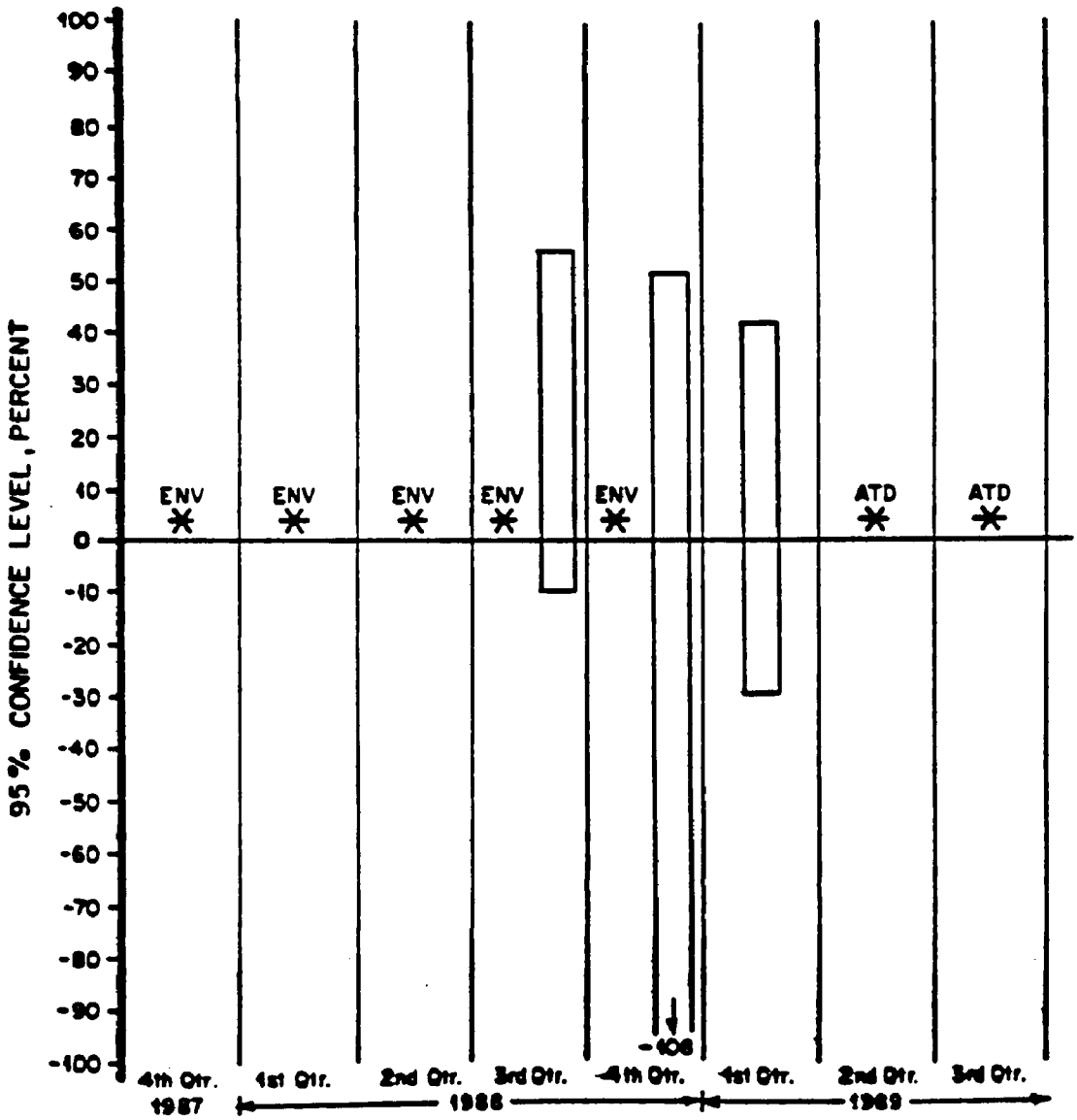


SAMPLING METHOD

ENVIROCHEM

ATD

1987 - 1989
STATEN ISLAND TOXICS STUDY
SAMPLE PRECISION ANALYSIS
1,4 DICHLOROBENZENE



* INSUFFICIENT DATA PAIRS TO EVALUATE STATISTICALLY

SAMPLING METHOD

ENVIROCHEM

ATD

Chapter 3

Distributive Volumes

ORGANIZATION: NEW YORK STATE ENCON SURGENTS ENVIRONMENT

QUARTER OF OCTOBER TO DECEMBER , 1987

DISTRIBUTED VOLUMES

COMPOUND	# PAIRS RUN	AVERAGE DIFFERENCE (PPB)	STD. DEV. AVG. DIFF. (PPB)	95% CL INTERVAL (PPB) LL	UL	T > 0.05 (Y/N)
TOLUENE	32	0.51	0.19	0.12	0.90	Y
BENZENE	32	0.77	0.12	0.53	1.01	Y
M,P XYLENE	33	0.49	0.13	0.23	0.75	Y
1,1,1 TRICHLOROETHANE	32	0.05	0.03	0.00	0.10	N
TETRACHLOROETHYLENE	30	0.01	0.02	-0.04	0.06	N
DICHLOROMETHANE	28	0.09	0.06	-0.03	0.21	N
CARBON TETRACHLORIDE	NO DATA AVAILABLE					
ETHYLBENZENE	29	0.02	0.03	-0.04	0.08	N
CHLOROFORM	3	0.01	0.02	-0.06	0.08	N
O XYLENE	32	0.08	0.04	0.00	0.16	Y

NOTES ATTACHED (Y/N):___

QUARTER OF JULY TO SEPTEMBER, 1988

DISTRIBUTED VOLUMES

COMPOUND	# PAIRS RUN	AVERAGE DIFFERENCE (PPB)	STD. DEV. AVG. DIFF. (PPB)	95% CL INTERVAL (PPB) LL	UL	T > 0.05 (Y/N)
TOLUENE	16	0.13	0.13	-0.15	0.41	N
BENZENE	13	0.24	0.13	-0.04	0.52	N
M,P XYLENE	15	0.08	0.05	-0.04	0.20	N
1,1,1 TRICHLOROETHANE	16	0.02	0.02	-0.03	0.07	N
TETRACHLOROETHYLENE	14	0.01	0.00	0.00	0.02	Y
DICHLOROMETHANE	15	0.13	0.05	0.02	0.24	Y
CARBON TETRACHLORIDE	NO DATA AVAILABLE					
ETHYLBENZENE	16	-0.02	0.01	-0.05	0.01	N
CHLOROFORM	11	0.02	0.02	-0.02	0.06	N
O XYLENE	16	0.00	0.01	-0.03	0.03	N

NOTES ATTACHED (Y/N):___

QUARTER OF OCTOBER TO DECEMBER, 1988

DISTRIBUTED VOLUMES

COMPOUND	# PAIRS RUN	AVERAGE DIFFERENCE (PPB)	STD. DEV. AVG. DIFF. (PPB)	95% CL INTERVAL (PPB) LL	UL	T > 0.05 (Y/N)
TOLUENE	35	0.04	0.05	-0.07	0.15	N
BENZENE	33	0.00	0.07	-0.14	0.14	N
M/P XYLENE	32	0.05	0.02	0.01	0.09	Y
1,1,1 TRICHLOROETHANE	32	0.01	0.02	-0.03	0.05	N
TETRACHLOROETHYLENE	32	0.00	0.00	-0.01	0.01	N
DICHLOROMETHANE	32	0.06	0.02	0.01	0.11	Y
CARBON TETRACHLORIDE	NO DATA AVAILABLE					
ETHYLBENZENE	34	0.00	0.02	-0.04	0.04	N
CHLOROFORM	22	0.00	0.01	-0.01	0.01	N
O XYLENE	32	0.00	0.01	-0.01	0.01	N

NOTES ATTACHED (Y/N):___

QUARTER OF JANUARY TO MARCH, 1989

DISTRIBUTED VOLUMES

COMPOUND	# PAIRS RUN	AVERAGE DIFFERENCE (PPB)	STD. DEV. AVG. DIFF. (PPB)	95% CL INTERVAL (PPB) LL	UL	T > 0.05 (Y/N)
TOLUENE	74	0.39	0.10	0.19	0.59	Y
BENZENE	73	-0.06	0.04	-0.14	0.02	N
M,P XYLENE	74	0.07	0.03	0.01	0.13	Y
1,1,1 TRICHLOROETHANE	71	0.02	0.02	-0.02	0.06	N
TETRACHLOROETHYLENE	68	0.01	0.01	-0.01	0.03	N
DICHLOROMETHANE	66	-0.02	0.05	-0.11	0.07	N
CARBON TETRACHLORIDE	68	0.00	0.00	-0.01	0.01	N
ETHYLBENZENE	72	0.02	0.01	0.00	0.04	Y
CHLOROFORM	58	-0.01	0.01	-0.03	0.01	N
O XYLENE	74	0.02	0.01	0.00	0.04	Y

NOTES ATTACHED (Y/N):___

QUARTER OF APRIL TO JUNE, 1989

DISTRIBUTED VOLUMES

COMPOUND	# PAIRS RUN	AVERAGE DIFFERENCE (PPB)	STD. DEV. AVG. DIFF. (PPB)	95% CL INTERVAL (PPB) LL	UL	T > 0.05 (Y/N)
TOLUENE	68	0.10	0.15	-0.21	0.41	N
BENZENE	65	-0.04	0.04	-0.11	0.03	N
M/P XYLENE	70	-0.01	0.04	-0.09	0.07	N
1,1,1 TRICHLOROETHANE	67	0.01	0.02	-0.03	0.05	N
TETRACHLOROETHYLENE	67	0.08	0.04	0.00	0.16	Y
DICHLOROMETHANE	53	0.02	0.07	-0.12	0.16	N
CARBON TETRACHLORIDE	59	0.01	0.00	0.00	0.02	Y
ETHYLBENZENE	70	0.03	0.02	0.00	0.06	N
CHLOROFORM	45	0.01	0.00	0.00	0.02	Y
O XYLENE	70	0.00	0.01	-0.03	0.03	N

NOTES ATTACHED (Y/N):___

Chapter 4

Sorbent Tube vs. Canister

ORGANIZATION: NEW YORK STATE ENCON

SORBENT: ENVIROCHEM

QUARTER OF APRIL TO JUNE, 1988

SORBENT TUBE VS. CANISTER

COMPOUND	# PAIRS RUN	AVERAGE DIFFERENCE (PPB)	STD. DEV. AVG. DIFF. (PPB)	95% CL INTERVAL (PPB) LL	UL	T > 0.05 (Y/N)	AVERAGE % DIFFERENCE
TOLUENE	11	-0.03	0.24	-0.56	0.50	N	29.5
BENZENE	9	0.32	0.27	-0.30	0.94	N	-17.6
M,P XYLENE	11	-0.32	0.21	-0.78	0.14	N	41.1
O XYLENE	8	-0.30	0.12	-0.58	-0.02	N	137.5
1,1,1 TRICHLOROETHANE	5	-0.16	0.19	-0.68	0.36	N	85.9
TETRACHLOROETHYLENE	6	-0.31	0.22	-0.88	0.26	N	184.3
DICHLOROMETHANE	5	-0.13	0.15	-0.54	0.28	N	80.2
CARBON TETRACHLORIDE			NO DATA PAIRS				

NOTES ATTACHED (Y/N):___

ORGANIZATION: NEW YORK STATE ENCON SORBENT: RTD

QUARTER OF OCTOBER TO DECEMBER, 1988

SORBENT TUBE VS. CANISTER

COMPOUND	# PAIRS RUN	AVERAGE DIFFERENCE (PPB)	STD. DEV. AVG. DIFF. (PPB)	95% CL INTERVAL (PPB) LL	UL	T > 0.05 (Y/N)	AVERAGE % DIFFERENCE
TOLUENE	20	0.71	0.24	0.20	1.22	Y	-13.15
BENZENE	20	0	0.06	-0.12	0.12	N	0.15
M,P XYLENE	19	0.15	0.07	0.00	0.30	Y	-6.89
O XYLENE	14	-0.05	0.05	-0.15	0.05	N	23.2
1,1,1 TRICHLOROETHANE	10	-0.16	0.13	-0.46	0.14	N	46.6
TETRACHLOROETHYLENE	10	-0.05	0.03	-0.13	0.03	N	23.4
DICHLOROMETHANE	16	-0.11	0.11	-0.34	0.12	N	52.8
CARBON TETRACHLORIDE			NO DATA PAIRS				

NOTES ATTACHED (Y/N):___

ORGANIZATION: NEW YORK STATE ENCON

SORBENT: ATD

QUARTER OF JULY TO SEPTEMBER, 1989

SORBENT TUBE VS. CANISTER

COMPOUND	# PAIRS RUN	AVERAGE DIFFERENCE (PPB)	STD. DEV. AVG. DIFF. (PPB)	95% CL INTERVAL (PPB) LL	UL	T > 0.05 (Y/N)	AVERAGE % DIFFERENCE
TOLUENE	42	0.59	0.15	0.30	0.88	Y	-14
BENZENE	42	-0.40	0.04	-0.48	-0.32	N	14
M,P XYLENE	42	-0.39	0.18	-0.75	-0.03	N	30
O XYLENE	41	-0.80	0.23	-1.26	-0.34	N	227
1,1,1 TRICHLOROETHANE	39	-0.24	0.03	-0.30	-0.18	N	109
TETRACHLOROETHYLENE	14	-0.16	0.08	-0.33	0.01	N	82
DICHLOROMETHANE	42	-0.25	0.03	-0.31	-0.19	N	101
CARBON TETRACHLORIDE	2	-0.12	0.02	-0.23	-0.01	N	17

NOTES ATTACHED (Y/N):___

Chapter 5

Minimum Analytical Detection Limits

MINIMUM ANALYTICAL DETECTION LIMITS

Chemical	D.L. (NG)	PPB(15 L. Samples)
Dichloromethane	10	.2
Chloroform	3	.04
1,2 Dichloroethane	3	.04
1,1,1 Trichloroethane	3	.06
Benzene	10	.2
Carbon Tetra Chloride	6	.06
Trichloroethylene	2	.02
1,1,2 Trichloroethylene	4	.04
Toluene	6	.1
Tetra Chloroethylene	5	.04
Chlorobenzene	2	.02
Ethylbenzene	3	.04
M,P xylene	3	.04
O-xylene	3	.04
Dichlorobenzenes	2	.02

Samples in the range from 1-3 times the detection limit are not as precise as values above these limits. A 50% variation is expected in the range close to the detection limit.

Chapter 6

Instrument Accuracy

1988 GAS CHROMATOGRAPH PERFORMANCE AUDITS

During the first full year of this study (1988), blank sample tubes were provided to EPA which spike them with various organic compounds of interest to this study. These sample tubes were then routinely analyzed on the gas chromatograph and the results reported to EPA who then reported back on the audit results. The actual results of the audits can be found in Appendix D. Following is a summary of these results for both audits.

Compound	No. of Spiked Tubes	Mean	Std Dev.	UL Percent	LL Percent
Chloroform	22	2.1	19.2	41	-36
1,1,1-Trichloroethane	22	-34.3	61.6	89	-158
Carbon Tetrachloride	22	-27.0	31.0	35	-89
Benzene	22	26.9	17.4	62	-8
1,2-Dichloroethane	22	0.0	14.4	29	-29
Trichloroethylene	20	-35.6	10.7	-14	-57
Toulene	22	11.8	22.8	+35	-11
Tetrachloroethylene	22	-19.5	21.5	24	-63
Chlorobenzene	22	-5.3	15.3	25	-36
Ethylbenzene	22	-6.4	11.8	17	-30
O-xylene	22	-3.0	25.1	47	-53

UL - Upper 95% confidence level
LL - Lower 95% confidence level

1989 PARTICIPATION IN EPA GC PERFORMANCE AUDITS

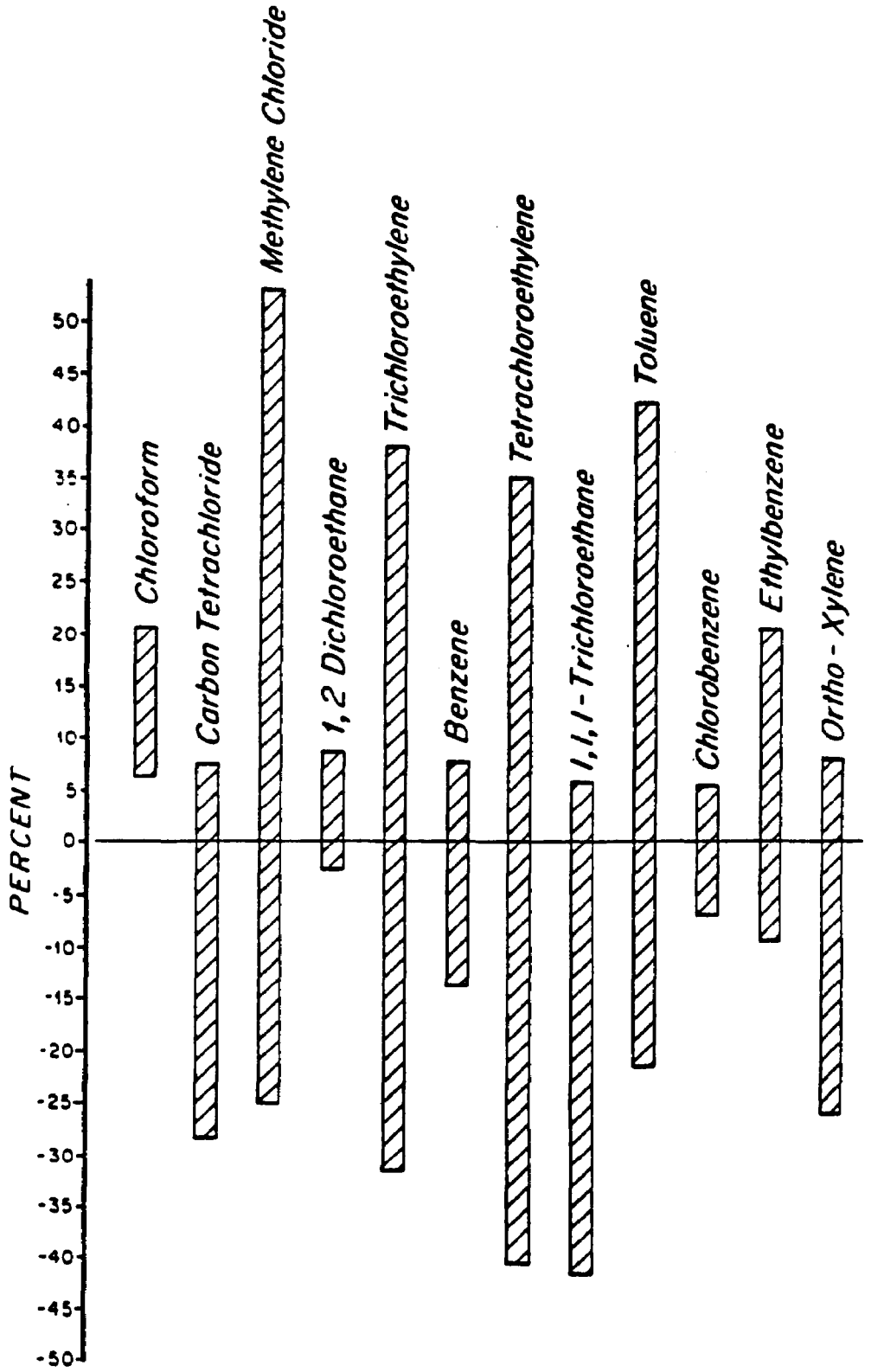
Twice during the term of this study, in the 1st quarter and 3rd quarter 1989, an audit of the gas chromatograph used to analyze the ATD-50 and Envirochem samples was performed.

The audit gas utilized was loaned to us by Research Triangle Institute, Center for Environmental Measurements. This gas was used to spike 4 sample tubes which were then analyzed on the gas chromatograph and reported to RTI. The audit results were then reported back to us. The actual results of the audits can be found in Appendix A. The following is a statistical summary of the results.

Summary of Independent Hazardous Organic Gases Audit Results

Compound	n	$\bar{\mu}$	σ	UL	LL
Chloroform	3	+13.3	3.4	20.1	6.5
Carbon Tetrachloride	3	-10.3	9.0	7.7	-28.3
Methylene Chloride	3	38.7	7.0	53	25
1,2 Dichloroethane	3	3.0	2.8	8.6	-2.6
Trichloroethylene	2	34.5	1.5	37.5	31.5
Benzene	3	-3.0	5.4	7.8	-13.8
Tetrachloroethytene	3	-2.7	18.9	35.1	-40.5
1,1,1-Trichloroethane	3	-17.7	11.8	5.9	-41.3
Toluene	3	10.3	16.0	42.3	-21.7
Chlorobenzene	3	-0.7	3.1	5.5	-6.9
Ethylbenzene	3	15.0	2.8	20.6	9.4
Ortho-Xylene	3	-9.0	8.5	8.0	-26.0

n- Number of samples
$\bar{u}$- Mean value
σ- Standard deviation
UL- Upper 95% confidence limit
LL- Lower 95% confidence limit



Hazardous Organic Gases Audit Results 8- 42
95% Confidence Limits

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
ENVIRONMENTAL MONITORING SYSTEMS LABORATORY
RESEARCH TRIANGLE PARK
NORTH CAROLINA 27711

January 28, 1988

Mr. Gary Boynton
NY State Department of
Environmental Conservation
50 Wolf Road, Room 138
Albany, NY 12233

Dear Mr. Boynton:

Your analytical results on the spiked Tenax samples provided by us for the methods comparison at Susan Wagner High School are in the accompanying table.

Please call me at 919/541-2723 if you want to discuss these data.

Sincerely yours,

Howard Crist

Howard L. Crist
Performance Evaluation Branch
Quality Assurance Division (MD-77B)

Enclosure

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UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
ENVIRONMENTAL MONITORING SYSTEMS LABORATORY
RESEARCH TRIANGLE PARK
NORTH CAROLINA 27711

MEMORANDUM

DATE: September 1, 1988

SUBJ: Audit Results for Staten Island Project

FROM: Howard L. Crist
PEB/QAD/EMSL (MD-77B)

TO: Paul Brown
Air and Water Section (2ES-MM)
Region II

The results of the audit sample analyses from the New York State Department of Environmental Conservation for the Staten Island Project are in the enclosed tables.

If you have any questions, please call me at FTS 629-2723.

Enclosures

cc: Garry Boynton, NYS/DEC
Dick Means, NSI

Table 3. Percent Bias of Audit Results
(NYS/DEC)

	Bias								
	T-1	T-2	T-3	T-4	T-5	T-6	T-7	T-8	T-9
chloroform	-16	-22	-15	-20	-24	-8.3	-11	-24	-7.4
1,1,1-trichloroethane	-12	-23	-8.2	-13	-13	-4.9	-23	-18	-2.1
carbon tetrachloride	-39	-41	-24	-38	-33	-15	-27	-32	-16
benzene	38	14	6.0	28	-3.1	12	36	19	26
1,2-dichloroethane	11	-15	-9.1	-0.7	-8.2	-1.1	-19	-16	-0.4
trichloroethylene	-28	-32	-29	-27	-27	-26	-21	-28	-50
toluene	3.2	-14	-6.1	-16	-5.8	-0.2	4.1	-0.3	0.9
tetrachloroethylene	-39	-33	-26	-36	-29	-28	-29	-35	-30
chlorobenzene	-9.1	-20	-20	-11	-15	-7.8	-8.7	-20	-18
ethylbenzene	-20	-18	-6.9	-13	-18	-9.8	-3.2	-11	-3.5
o-xylene	-20	-22	-7.0	-23	-21	-13	-10	-14	-5.4

	Bias, %								
	A002	A006	A009	A013	A015	A021	A028	A043	A046
chloroform	19	11	4.2	31	10	-4.6	24	17	-15
1,1,1-trichloroethane	-99	-99	-98	-98	-98	-98	-98	-98	-98
carbon tetrachloride	-50	-46	-55	-66	-53	-38	-64	-46	-35
benzene	51	43	40	57	42	2.6	45	39	36
1,2-dichloroethane	10	-7.1	-12	11	-3.3	-19	5.8	-3.3	-13
trichloroethylene	-41	-49	-53	-45	-39	-39	-45	-42	-49
toluene	7.7	76	6.8	43	42	5.6	43	14	-4.1
tetrachloroethylene	-24	-26	-29	-22	-24	-28	-25	-29	-34
chlorobenzene	-4.6	-11	-13	-2.9	25	-10	-8.3	-9.9	-18
ethylbenzene	-5.1	-9.9	-12	-3.2	-9.0	-9.5	-8.9	-11	-20
o-xylene	-8.3	-14	-16	-6.7	-12	-12	-12	-13	-23

$$\% \text{ Bias} = \frac{\text{reported} - \text{spiked}}{\text{spiked}} \times 100$$

No corrections made for blank background

Chapter 7

Narrative Reports by Quarter

Gas Chromotography Operation and Sample Collection

Prepared by

Bureau of Toxic Air Sampling

DATA REPORT

4th Quarter - 1987

Envirochem System is running satisfactorily. The performance evaluation and shoot out samples indicated problem areas that allowed us to fine tune the system.

This quarter's data has several sampling days missing as logistics and analysis deadlines are being firmed up.

Data prior to the shoot out is not included, as integration parameters were changed significantly to increase reliability and accuracy.

The analysis time has been shortened to allow all three sites to be analyzed in one day. The room must be kept cool to decrease oven cooling time and integration and data transfer speeded up.

This was accomplished by the end of the year, so in 1988 each day's samples will be analyzed in one day.

DATA REPORT

2nd Quarter - 1988

Data completeness is increasing.

A variation between the lower volume and higher volume samples was noted prior to March. The first stage absorption time was increased by 25% to insure complete desorbtion of all samples. Data variability decreased markedly. This shows that breakthrough during sampling is not the problem, but rather, incomplete desorbtion. Our gradient tube with tenax, ambersorb and carbon will help increase our minimum detection level by increasing sample volume, but we must insure that the sample is completely desorbed.

The ATD system is being prepared for the July shoot out. Third quarter data will be compared so a complete changeover can occur this fall. The ATD system allows for overnight automatic analysis.

DATA REPORT

4th Quarter - 1988

Comparison of the colocated samplers at Port Richmond show a good correlation between the ATD and Envirochem systems. The conversion to all ATD tubes will be completed for the January 1989 sampling runs.

The data completeness for this quarter was excellent. Workload was reduced with the loss of the Texas and UMDNJ contract sites. The 6 sites for ATD sampling and the new field log forms created by Mike Steiniger have helped immensely in decreasing sampling errors and decreasing sample turnaround time.

In December, we changed the calibration level for toluene. Our calibration range now extends from 0 to 7 ppb. This level will encompass the vast majority of samples, yet not interfere with the surrounding peaks because of peak size.

DATA REPORT

2nd Quarter - 1989

Mailing problems cropped up again. The April 22 and June 2 sample runs was lost in the mail. Discussions with the postal service have prompted us to change to first class mail only. Business rate mail is easily lost with no way to trace. At $1200 per sample run, we can not afford to lose 20 ATD tubes at a time. We will be using next day mail to Staten Island and certified on the return. This has worked successfully since we began this method of shipping.

Field sampling errors eliminated samples on 4/28 at sites 1 and 6 and 6/9 at site 3.

Chapter 8

Assessment of Variability of Data

CHAPTER VIII

PROJECT SUMMARY

The SI/NJ Toxics Air Monitoring Program was a major success. Toxic air monitoring developed from 2 or 3 day studies with analysis contracted out, to capabilities for a 12 Station Statewide network on a 6 day schedule with GC/MS, rapid turnaround, in house analysis. The QA procedures work, and accuracy and precision for most chemicals exceeds EPA Contract Laboratory Program protocols. The development stage has been completed and routine monitoring is now a capability.

The following narrative collects the most pertinent observations for the Staten Island Project in one chapter. It is done in a bulletin form to be concise.

1) The 4th quarter 87 data while acceptable is the least accurate of the study. The desorbtion system was modified. The data quantification program was modified extensively prior to 1988 and sample analysis was spread out over 2 days. Analysis times were trimmed to allow a days' samples to be analyzed in a single MSD run. This reduced the probability of instrument sensitivity drift.

2) MDL's were determined by the method listed in the EPA audit report. These values are conservative and for a few chemicals, levels below MDL's were routinely reported. These values are not

as precise or accurate as values 3 times the detection limit. They are reported because it was our feeling that the data represents real detects. All the data handling was done by computer and scanned by staff for obvious irregularities.

3) Initial calibration procedures included weighing the permeation tubes each quarter to verify the certified permeation rate. Humidity, static and other unknown problems prevented us from continuing this verification method. Tubes were checked twice a year to insure enough chemical to permeate. We now use the certified rate and replace tubes when they reach 3/4 empty as permeation rates begin to change significantly at this point.

4) The Dichloromethane values for 1989 are biased approximately 25% high. The permeation tube was not replaced on time and both data and QA checks for 1989 indicated a positive bias. The data has not been corrected for measurement bias.

5) The trichloroethylene values for the 3rd quarter 89 are NOT VALID. Calibration problems existed throughout the year resulting in a +25% bias for the first quarter 89 and +50% bias for the second quarter 89. The data has not been corrected for this bias.

6) Blank data usage changed in early 1988 from subtracting that sampling days' blank from the samples averaging the last 5 blanks and subtracting the average from the samples. The latter worked

very well at smoothing the blank subtraction, and allowed us to watch sorbent tube aging throughout the study.

7) The sample tubes storage caps were changed during the 2nd quarter of 89. The o-ring sealed storage caps were replaced with swagelock caps using teflon ferrules. These sealed much better and our blank valves were immediately reduced as passive sampling during storage and transport was halted.

8) Special ball valves were used at the inlet end of the sample tube through the majority of the study to reduce the passive sampling occurring prior to sampling and after sampling before staff could collect and ship the samples for analysis. A special study was conducted to prove the value of using the ball valves in our network. The conclusion was that valves reduced the passive sampling by sample tubes but were not as effective as capped blanks. Extended passive sampling times up to 140 hours yielded a slight but measurable difference from 0 to 10% from capped blanks. Our methods of capping with ball valves continued as planned and the pickup of weekend samples were delayed until the next regular workday.

All other VOC data from NYS DEC can be used at a +/- 30% accuracy level and precision of +/- 40 to 50%. Averaging the 3 samples (two hi and one low flow) per site allows us much more confidence in the values. The use of true duplicates and distributed volume sample insures our ability to track precision

and guard against breakthrough or desorption problems in an on going program. We will continue to use the 3 sample system for the Statewide network.

90-3-17

-- ORGANIZATION: NEW YORK STATE ENCON COMPOUND: CHLOROFORM

DATA VARIABILITY FOR STUDY

METHOD	# SAMPLES	MEAN SAMPLE CONCENTRATION PPB	MEAN DIFFERENCE BETWEEN PAIRED SAMPLES (PPB)	STANDARD DEVIATION OF THE MEAN DIFFERENCE BETWEEN PAIRED SAMPLES (PPB)
ENVIROCHEM (10/1/87 - 12/31/87)	3		0.01	0.02
ENVIROCHEM (1/1/88 - 12/31/88)	51		-0.04	0.04
ATD-50 (6/1/88 - 9/30/89)	199		0.00	0.01

NOTES ATTACHED (Y/N):

ORGANIZATION: NEW YORK STATE ENCON COMPOUND: DICHLOROMETHANE

DATA VARIABILITY FOR STUDY

METHOD	# SAMPLES	MEAN SAMPLE CONCENTRATION PPB	MEAN DIFFERENCE BETWEEN PAIRED SAMPLES (PPB)	STANDARD DEVIATION OF THE MEAN DIFFERENCE BETWEEN PAIRED SAMPLES (PPB)
ENVIROCHEM (10/1/87 - 12/31/87)	28		0.09	0.06
ENVIROCHEM (1/1/88 - 12/31/88)	97		0.05	0.02
ATD-50 (6/1/88 - 9/30/89)	237		0.05	0.04

NOTES ATTACHED (Y/N):

ORGANIZATION: NEW YORK STATE ENCON

COMPOUND: M,P XYLENE

DATA VARIABILITY FOR STUDY

METHOD	# SAMPLES	MEAN SAMPLE CONCENTRATION PPB	MEAN DIFFERENCE BETWEEN PAIRED SAMPLES (PPB)	STANDARD DEVIATION OF THE MEAN DIFFERENCE BETWEEN PAIRED SAMPLES (PPB)
ENVIROCHEM (10/1/87 - 12/31/87)	33		0.49	0.13
ENVIROCHEM (1/1/88 - 12/31/88)	119		0.08	0.03
ATD-50 (6/1/88 - 9/30/89)	281		-0.07	0.02

NOTES ATTACHED (Y/N):

ORGANIZATION: NEW YORK STATE ENCON

COMPOUND: ETHYLBENZENE

DATA VARIABILITY FOR STUDY

METHOD	# SAMPLES	MEAN SAMPLE CONCENTRATION PPB	MEAN DIFFERENCE BETWEEN PAIRED SAMPLES (PPB)	STANDARD DEVIATION OF THE MEAN DIFFERENCE BETWEEN PAIRED SAMPLES (PPB)
ENVIROCHEM (10/1/87 - 12/31/87)	29		0.02	0.03
ENVIROCHEM (1/1/88 - 12/31/88)	120		-0.01	0.01
ATD-50 (6/1/88 - 9/30/89)	282		0.03	0.01

NOTES ATTACHED (Y/N):

ORGANIZATION: NEW YORK STATE ENCON

COMPOUND: 1,1,1 TRICHLOROETHANE

DATA VARIABILITY FOR STUDY

METHOD	# SAMPLES	MEAN SAMPLE CONCENTRATION PPB	MEAN DIFFERENCE BETWEEN PAIRED SAMPLES (PPB)	STANDARD DEVIATION OF THE MEAN DIFFERENCE BETWEEN PAIRED SAMPLES (PPB)
ENVIROCHEM (10/1/87 - 12/31/87)	32		0.05	0.03
ENVIROCHEM (1/1/88 - 12/31/88)	121		0.01	0.02
ATD-50 (6/1/88 - 9/30/89)	264		0.00	0.01

NOTES ATTACHED (Y/N):

ORGANIZATION: NEW YORK STATE ENCON

COMPOUND: O-XYLENE

DATA VARIABILITY FOR STUDY

METHOD	# SAMPLES	MEAN SAMPLE CONCENTRATION PPB	MEAN DIFFERENCE BETWEEN PAIRED SAMPLES (PPB)	STANDARD DEVIATION OF THE MEAN DIFFERENCE BETWEEN PAIRED SAMPLES (PPB)
ENVIROCHEM (10/1/87 - 12/31/87)	32		0.08	0.04
ENVIROCHEM (1/1/88 - 12/31/88)	120		-0.01	0.01
ATD-50 (6/1/88 - 9/30/89)	275		0.02	0.01

NOTES ATTACHED (Y/N):

Chapter 9a

Miscellaneous

Summary of Data Availability

STATEN ISLAND TOXICS STUDY

DATA AVAILABILITY SUMMARY

	% Data Availability	
Quarter	Envirochem Samplers	ATD Samplers
4th 1987	75	*
1st 1988	91	*
2nd 1988	85	*
3rd 1988	34	*
4th 1988	82	93
1st 1989	*	84
2nd 1989	*	82
3rd 1989	*	100

*Not in operation during this quarter.

Overall study data availability - 81%

Chapter 9b

Miscellaneous

Permeation Tube Calibration System

Flow Audits and Calibration

New York State Department of Environmental Conservation

MEMORANDUM

TO: G. Boynton
FROM: G. Froehlich
SUBJECT: BTAS Permeation Tube Calibration System
DATE: February 17, 1989

On February 2, 1989 I certified the BTAS Permeation Tube Calibration System at the Hemlock Street facility. Due to back pressure problems I didn't use the Hastings Mass Flow Controller, instead I used our NBS Standard bubble meter. The Hastings Mass Flow Controllers in the Permeation Tube Calibration System were calibrated as per their respective instruction manual procedures.

The results are listed below:

Tube Flow Controllers

Range		Set at	B.O.M. Measured at	Adj. to	%d	Setpoint Measured at
#1*	0-500 sccm	4.90V	493.6	Not adj.	+0.7	252.3
#2	0-500 sccm	4.90V	587.1	488.0	-0.4	251.9
#3	0-500 sccm	4.90	587.7	498.1	+1.7	254.9
#4	0-500 sccm	4.90	606.7	488.3	-1.4	246.5

*transducer #1 replaced with new transducer before calibration/certification.

Dilution Flow Controllers

#1	Blank				
#2	Not done by request				
#3	0-5 SLPM	2.00V	2047.3	Not Adj.	+2.4
#4	0-10 SLPM	4.75V	4931.1	4813.3	+1.3

This certification is valid from 2/2/89 to the week of 8/7/89. Please notify me at least two weeks in advance so I can schedule the recertification. If you have any questions, please don't hesitate to call.

cc: Mr. Webster
Mr. Coon

GF/bc

15 (12-75)

New York State Department of Environmental Conservation

MEMORANDUM

TO: Garry Boynton
FROM: George Proehlich
SUBJECT: Sorbent Tube Calibration System
DATE: October 31, 1989

On August 15, 1989 Quality Assurance Standard's Lab staff audited and re-calibrated for certification the Hastings mass flow controllers in the BTAS Sorbent Tube Calibration System at the Hemlock Street facility.

The Hastings mass flow controller audits were conducted with the Standard's Lab NBS traceable Bubble-meter (Hastings Model HBM-1, SN-64) and the flows were corrected for temperature and pressure. The results were as follows:

Dilution Flows:

Unit #2 (500 sccm total flow)

Audit	Calibration Before	Calibration After
Set 250 sccm	zero: 0.005	+0.00
B.O.M. 289.9 sccm (+16.5%)	span: 5750	4900
	Set point: 2500	2505

Unit #3 (5 SLPM Total Flow)

Audit	Calibration Before	Calibration After
Set 2000 sccm	zero: 0.07	+0.00
B.O.M. 2059.6 sccm (+3.0%)	span: 5270	4940
	Set point: 2500	2510

Unit #4 10 SLPM Total Flow)

Audit	Calibration Before	Calibration After
Set 4750.0 sccm	zero: 0.06	+0.00
B.O.M. 4781.1 sccm (+0.9%)	span: 10425.4	9867
	Set point: 4750	4624

RECEIVED
NOV - 2 1989
BUREAU OF TOXIC AIR SAMPLING

Tube Flows:

Unit #1 (500 sccm Total Flow)

Audit	Calibration	
	Before	After
Set 250.0 sccm	zero: 0.01	+0.00
B.O.M. 254.7 sccm (+2.0%)	span: 487	493
	Set point: 250	253

Unit #2 (500 sccm Total Flow

Audit	Calibration	
	Before	After
Set 250 sccm	zero: 0.03	±0.00
B.O.M. 246.0 sccm (-1.6%)	span: 481	492
	Set point: 250	251.5

Unit #3 (500 sccm Total Flow)

Audit	Calibration	
	Before	After
Set 250 sccm	zero: 0.06	+0.00
B.O.M. 255.6 sccm (+2.4%)	span: 499.2	492.0
	Set point: 250	251.0

Unit #4 (500 sccm Total Flow)

Audit	Calibration	
	Before	After
Set 250 sccm	zero: 0.05	±0.00
B.O.M. 245.6 sccm (-1.6%)	span: 486.0	490.0
	Set point: 250	251.5

The sorbent tube calibration system is due for a flow controller audit the week of January 15, 1990, and a new certification the week of August 13, 1990. If you have any questions, please don't hesitate to let me know.

cc: Randy Coon

GF/bc

Chapter 9c

Miscellaneous

Quality Assurance Laboratory

Certification Procedures

A. VOTS Flow Channel and Flow Measuring Module Certification Procedures

To assure flow accuracy in the VOTS monitoring instrumentation, the following procedures were adopted for flow channel and measuring module certification.

1. VOTS flow channel certification procedure.

   a. Each flow channel was built, adjusted and locked to a preset flow.

   b. Each flow channel was tested, at constant vacuum (20"Hg) on at least 10 different days and until it showed no more than ±.3 cc/min. variation over 5 days. Flow graphs are kept for each channel to monitor its stability. Unstable flow channels were reset if possible or eliminated.

   c. Each flow channel was marked with a brass identification tag to assure proper record keeping both in the laboratory and in the field.

   d. Each flow channel was used until a field flow audit indicated its certified value had changed more than ±10% at 20" Hg vacuum at which time it was returned to the laboratory for recertification.

2. VOTS flow measurement module certification procedure.

   a. Each flow measurement module was built, adjusted to 8 cc/min nominal flow and locked in position.

   b. Each flow measurement module was run at the following nominal flows 8, 15, 22, 29 and 36 cc/min with the total manometer deflection recorded at each flow.

   c. A linear regression was then calculated by entering deflection as the X value and the corresponding flows as the y value.

   d. The correlation factor, slope and intercept was then recorded on the modules certification sheet as well as on the module itself.

   e. Each flow measurement module was used until a field flow audit indicated its certified value had changed at which time it was returned to the laboratory for recertification.

B. VOTS Audit Flow Box Certification Procedure.

   a. Several VOTS audit devices were built and maintained in the laboratory. These audit devices have a 0-50 cc/min range and are internally heated and insulated to assure stable operation.

   b. Each VOTS audit device is certified at six month intervals.

   c. The following checks are made prior to certification.

      i. visual inspection performed.
      ii. electrical and safety inspection performed.
      iii. heater circuitry checked

iiii. mass flow meter calibrated following manufacturers procedure.
iiiii certification sheet and lab documentation completed.

C. Canister Sampler Modifications and Certification Procedures

a. Modifications performed prior to operation

i. Leak check performed and internal plumbing leaks eliminated.
ii. New pumps procured from manufacturer and interchanged with existing leaky pumps.
iii. Formaldehyde inlet removed from rear of instrument and teed into the stainless steel sampling manifold as per EPA recommendation.

b. Flow Certification

i. Internal plumbing leak checked under pressure before installing evacuated cylinder.

ii. Visual, electrical and safety inspection performed.

iii. Canister mass flow controller is calibrated with bubble-O-meter following manufacturers procedure.

iiii. Certification sheet and lab documentation completed.

D. VOTS Monitoring Shelter Modifications

a. Installation of mud-dauber fittings on the vacuum reflief valves to eliminate the plugging of the orifice by insects and airborne debris.

b. A 12" extension of the above vacuum relief valve to facilitate checking, cleaning and replacement when necessary.

c. Purchase and certification of 4" vacuum test gauges for each site. This insures on a weekly basis that the manifold vacuum is 20" Hg ± 1.5" Hg.

Chapter 9d

Miscellaneous

Flow Performance Audits

PERFORMANCE FLOW AUDITS

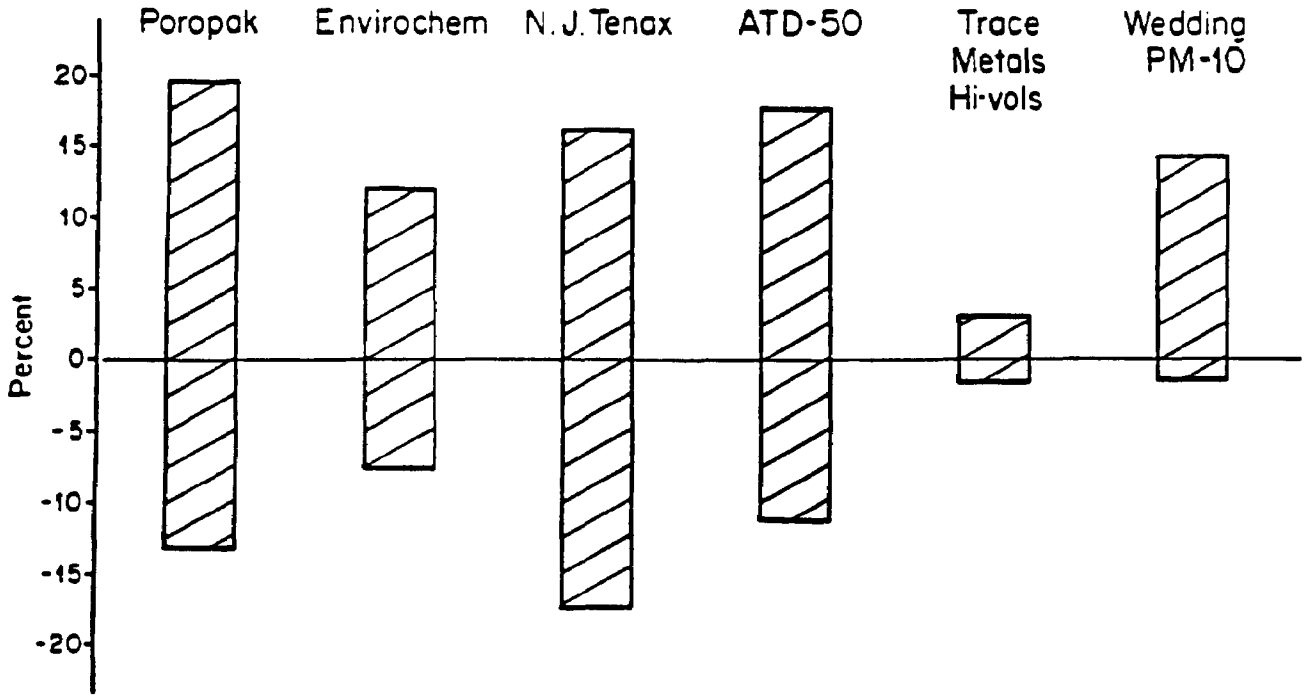
Routine performance flow audits were performed by the operations group. In the fall of 1988, the Quality Assurance Section of the Bureau of Technical Services assumed responsibility for biannual oversight performance flow audits in addition to the routine operational audits. These Quality Assurance audits were performed in the fall of 1988, spring and fall of 1989. The summary statistics that follow represent the combined results of both operational and Quality Assurance Section audits. Appendix B is a sample of the flow performance audits performed during the 3rd quarter 1989.

Summary Statistics
Flow Performance Audits

	n	$\bar{u}$	σ	UL	LL
Poropak	17	3.02	8.2	19.4	-13.4
Envirochem	19	2.2	4.9	12.0	-7.6
N.J. Tenax	13	-0.7	8.4	16.1	-17.5
ATD-50	66	3.1	7.2	17.5	-11.3
Trace Metal Hi-vols	6	0.7	1.2	3.1	-1.7
Wedding PM-10	6	8.0	3.2	14.4	1.6

n- Number of samplers
$\bar{u}$- Mean value
σ- Standard deviation

UL- Upper 95% confidence limit
LL- Lower 95% confidence limit



Flow Performance Audits
95% Confidence Limits

Chapter 9e

Miscellaneous

Report on

Effectiveness of Gravity Valve

In Prevention of Passive Sampling

of Sorbent Tubes

Effectiveness of Gravity Valve in Prevention of Passive Sampling of Sorbent Tubes

May 1989

Garry Boynton, Dr. William Webster, Brian Lay

N.Y.S. D.E.C. Div. of Air Resources

This short study was set up to show the effectiveness of a gravity ball valve in reducing passive sampling by sorbent tubes before and after the active sampling period. This capability would reduce the overtime and scheduling problems of a sorbent tube network run on a 6 day schedule by allowing the field staff to work normal hours as degradation of samples would not occur over a weekend.

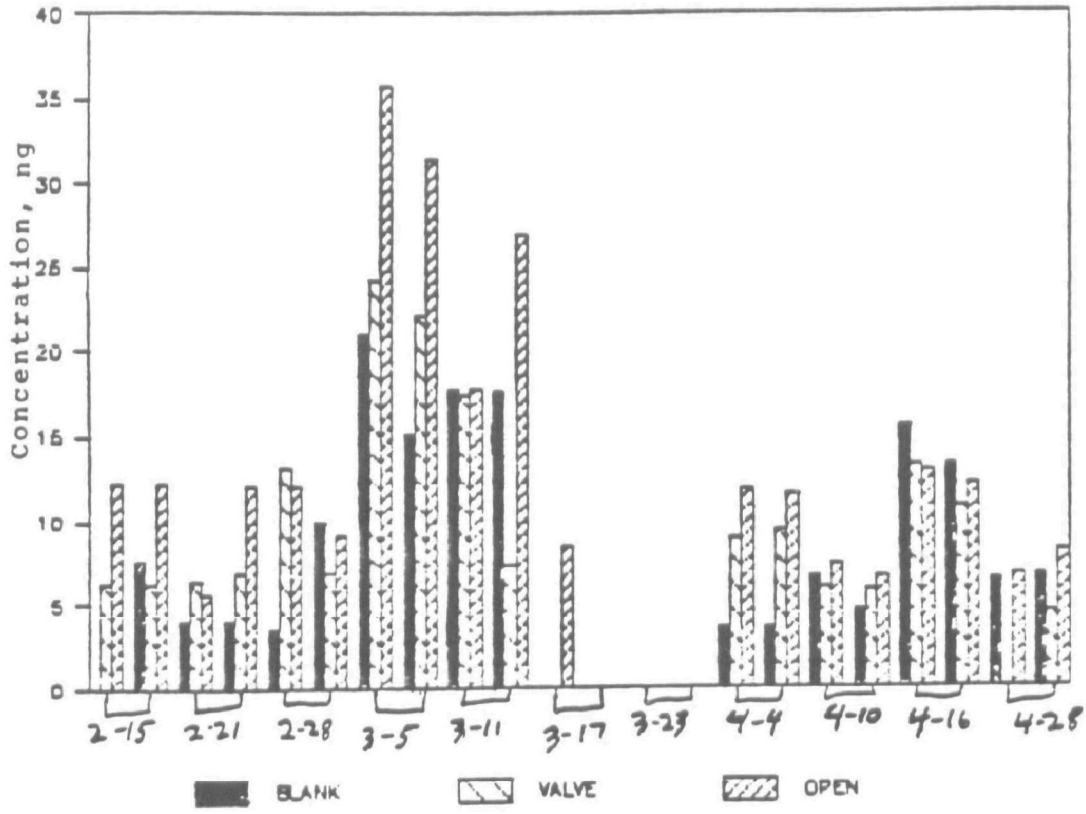
The study consisted of 11 sampling events from Feb. to April 1989. Two tubes were left capped with the standard storage caps from the time they were cleaned until they were analyzed. Two tubes were placed in an unplugged sampler with the gravity valves attached. Parallel to these two were two more tubes attached to the sampler and their ends left open.

These tubes were analyzed with the routine samples taken on the six day schedule for the Staten Island/New Jersey Monitoring project.

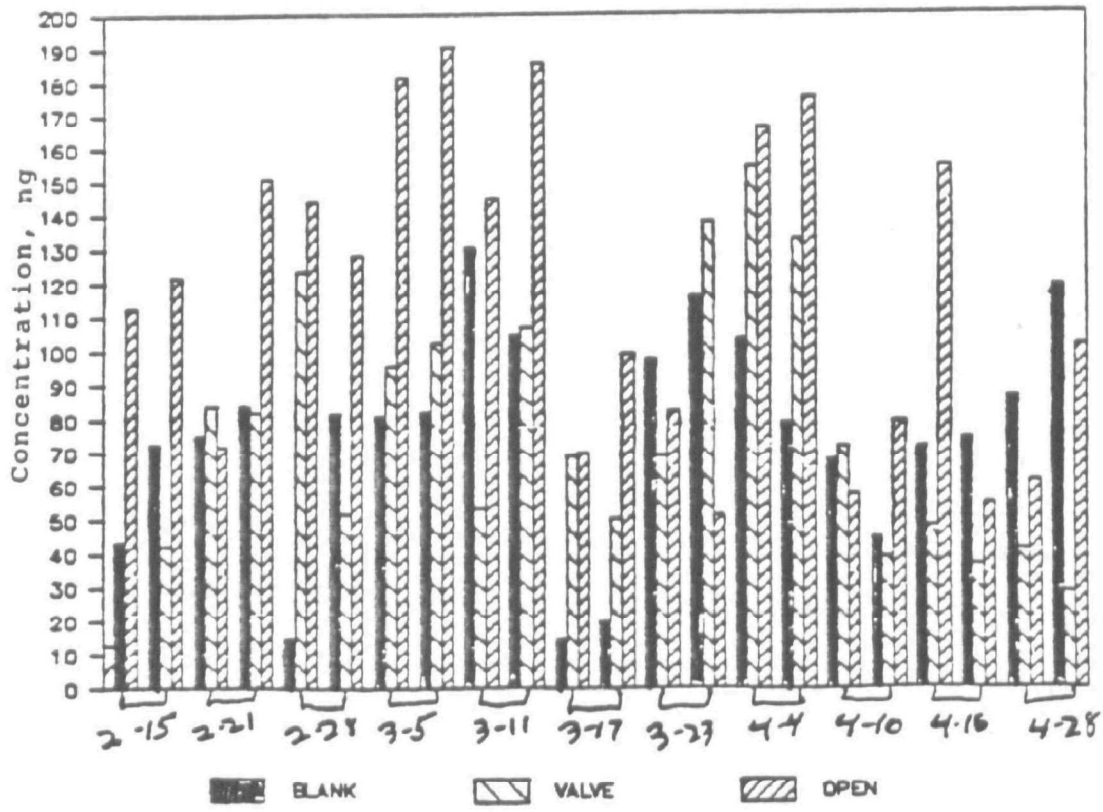
Bar charts for Dichloromethane, 111 Trichloroethane, Benzene, and Toluene are attached as well as the raw data. The first 5 columns in the raw data are sample information from the analytical system. The fourth column is the key to the sample. The AT# refers to the capped blanks, the PASOPE refers to a passive test with open end and PASVAL, refers to a passive test with a valved end. The fifth column is the number of hours the tubes were exposed.

A rough comparison was performed. Each passive test was compared to the average of the 2 blanks for that day. The results are tabulated in two groups, less than the Avg. blank or greater than. These results were then used in the Binomial Test (same as used for breakthrough). Failing this test means that passive sampling occurred enough to show a statistical difference from the average blank values.

DICHLOROMETHANE



TOLUENE



7097-06	03/15/89	BLANK	AT3211	0.000	[illegible]	[illegible]	[illegible]	[illegible]
	03-24-1989	CALIBRATION	PASVAL01	-81.000	6.380	NG	5.[illegible]	21.000	43.100
	03-27-1989	CALIBRATION	PASVAL02	-81.000	6.510	NG	6.[illegible]	22.000	41.900
	03-27-1989	CALIBRATION	PASOPE01	-81.000	12.20	NG	12.00	NG	47.40	NG	117.9	NG
	03-27-1989	CALIBRATION	PASOPE02	-81.000	11.20	NG	10.[illegible]	NG	38.[illegible]	NG	101.9	NG
7097-06	03/21/89	BLANK	AT3001	0.000	4.001	NG	7.[illegible]	NG	32.[illegible]	NG	74.02	NG
7097-06	03/21/89	BLANK	AT3011	0.000	3.[illegible]	NG	7.01	NG	26.[illegible]	NG	81.03	NG
	03-28-1989	CALIBRATION	PASVAL03	144.000	6.[illegible]	NG	10.01	NG	23.[illegible]	81.43	NG
	03-28-1989	CALIBRATION	PASVAL04	144.000	6.[illegible]	NG	8.444	NG	31.16	NG	81.53	NG
	03-28-1989	CALIBRATION	PASOPE03	144.000	5.[illegible]	NG	6.[illegible]	30.41	NG	71.47	NG
	03-28-1989	CALIBRATION	PASOPE04	144.000	13.[illegible]	NG	14.10	NG	54.[illegible]	NG	150.7	NG
7097-06	03/27/89	BLANK	AT3901	0.000	3.001	NG	1.561	NG	20.21	NG	14.60	NG
7097-06	03/27/89	BLANK	AT3911	0.000	3.060	NG	8.233	NG	29.92	NG	81.22	NG
	03-10-1989	CALIBRATION	PASVAL05	121.000	13.09	NG	10.67	NG	42.39	NG	123.6	NG
	03-10-1989	CALIBRATION	PASVAL06	121.000	6.[illegible]	NG	8.774	NG	29.400	51.500
	03-10-1989	CALIBRATION	PASOPE05	121.000	12.10	NG	12.11	NG	40.13	NG	144.3	NG
	03-10-1989	CALIBRATION	PASOPE06	121.000	9.[illegible]	NG	10.40	NG	40.10	NG	118.3	NG
7097-01	3/5/89	BLANK	AT3001	0.000	20.[illegible]	NG	9.715	NG	19.90	NG	80.64	NG
7097-05	3/5/89	BLANK	AT3010	0.000	15.20	NG	8.[illegible]	NG	22.90	NG	81.73	NG
	03-20-1989	CALIBRATION	PASOPE07	145.000	35.7[illegible]	NG	21.22	NG	30.30	NG	101.6	NG
	03-20-1989	CALIBRATION	PASVAL07	145.000	24.20	NG	11.100	NG	30.60	NG	95.42	NG
	03-20-1989	CALIBRATION	PASOPE08	145.000	31.70	NG	20.[illegible]	NG	44.05	NG	190.2	NG
	03-20-1989	CALIBRATION	PASVAL08	145.000	[illegible]	NG	11.[illegible]	NG	31.[illegible]	NG	101.9	NG
7097-03	3/11/89	BLANK	AT3101	0.000	17.[illegible]	NG	10.60	NG	27.61	NG	130.4	NG
7097-03	3/11/89	BLANK	AT3111	0.000	17.50	NG	9.400	NG	20.60	NG	104.2	NG
	03-27-1989	CALIBRATION	PASOPE09	170.000	17.700		11.95	NG	40.85	NG	145.2	NG
	03-27-1989	CALIBRATION	PASVAL09	170.000	17.500		9.[illegible]	NG	29.01	NG	52.600
	03-27-1989	CALIBRATION	PASOPE10	170.000	26.9[illegible]	NG	21.75	NG	30.95	NG	105.5	NG
	03-27-1989	CALIBRATION	PASVAL10	170.000	[illegible]		[illegible]		[illegible]	NG	106.2	NG
7097-03	3/17/89	BLANK	AT3201	0.000	Not Found		5.033	NG	10.56	NG	14.11	NG
7097-03	3/17/89	BLANK	AT3211	0.000	Not Found		7.610	NG	20.41	NG	19.67	NG
	03-31-1989	CALIBRATION	PASOPE11	72.000		0.300	56.19	NG		17.100	69.05	NG
	03-31-1989	CALIBRATION	PASOPE12	72.000	Not Found		24.35	NG	26.39	NG	90.51	NG
	03-31-1989	CALIBRATION	PASVAL11	72.000	Not Found		10.64	NG	30.45	NG	60.30	NG
	03-31-1989	CALIBRATION	PASVAL12	72.000	Not Found		21.00	NG	30.33	NG	49.60	NG
7097-01	3/23/89	BLANK	AT3301	0.000	Not Found		11.60	NG	20.01	NG	97.17	NG
7097-03	3/23/89	BLANK	AT3311	0.000	Not Found		12.[illegible]	NG	22.19	NG	115.9	NG
	04-07-1989	CALIBRATION	PASOPE13	45.000	Not Found		0.[illegible]		32.55	NG	81.96	NG
	04-07-1989	CALIBRATION	PASOPE14	45.000	Not Found		6.[illegible]		25.79	NG	50.700
	04-07-1989	CALIBRATION	PASVAL13	45.000	Not Found		0.[illegible]		22.84	NG	68.44	NG
	04-07-1989	CALIBRATION	PASVAL14	45.000	Not Found		15.74	NG	25.57	NG	137.9	NG

STATEN ISLAND H2O DESORPTION (ATD) BLANK DATA SECOND QTR:

SITE #	SAMPLE DATE	SAMPLE TYPE	ANALYSIS #	Hours	DICHLOROMETHANE PPB	1,1,1 TRICHLOROETHANE PPB	BENZENE PPB	TOLUENE PPB
7097-01	04/04/89	BLANK	AT3401	0.000	3.577	NG	14.05	NG	21.10	NG	102.7	NG
7097-03	04/04/89	BLANK	AT3411	0.000	7.461	NG	8.[illegible]	NG	11.02	NG	78.35	NG
	04-24-1989	CALIBRATION	PASOPE15	96.000	11.7[illegible]	NG	23.37	NG	30.09	NG	165.9	NG
	04-24-1989	CALIBRATION	PASOPE16	96.000	11.40	NG	24.57	NG	30.79	NG	175.4	NG
	04-24-1989	CALIBRATION	PASVAL15	96.000	8.81[illegible]	NG	21.19	NG	34.09	NG	154.0	NG
	04-24-1989	CALIBRATION	PASVAL16	96.000	[illegible]		18.[illegible]	NG	26.00	NG	132.6	NG
7097-01	4/10/89	BLANK	AT3501	0.000	6.4[illegible]	NG	45.50	NG	16.00	NG	67.27	NG
7097-05	4/10/89	BLANK	AT3511	0.000	4.544	NG	36.[illegible]	NG	10.30	NG	44.14	NG
	04-24-1989	CALIBRATION	PASOPE17	95.000	7.351	NG	41.03	NG	20.45	NG	56.600
	04-24-1989	CALIBRATION	PASOPE18	95.000	6.510	NG	27.84	NG	20.30	NG	70.700
	04-24-1989	CALIBRATION	PASVAL17	95.000	5.880	NG	42.09	NG	16.400	71.200
	04-24-1989	CALIBRATION	PASVAL18	95.000	[illegible]		30.65	NG	[illegible]	NG	30.300
7097-01	04/16/89	BLANK	AT3601	0.000	15.39	NG	17.50	NG	30.21	NG	71.00	NG
7097-03	04/16/89	BLANK	AT3611	0.000	13.13	NG	10.50	NG	24.90	NG	73.01	NG
	05-04-1989	CALIBRATION	PASOPE19	72.000	12.[illegible]	NG	34.31	NG	34.15	NG	154.6	NG
	05-04-1989	CALIBRATION	PASOPE20	72.000	12.11	NG	4.000	21.00	NG	54.000
	05-04-1989	CALIBRATION	PASVAL19	72.000	13.10	NG	12.000	24.07	NG	47.300
	05-04-1989	CALIBRATION	PASVAL20	72.000	10.[illegible]	NG	6.110	25.[illegible]	NG	36.700
7097-02	04/28/89	BLANK	AT3701	0.000	6.[illegible]	NG	15.86	NG	40.[illegible]	NG	85.06	NG
7097-03	04/28/89	BLANK	AT3708	0.000	6.780	NG	21.91	NG	33.03	NG	110.8	NG
	05-12-1989	CALIBRATION	PASOPE21	73.000		6.[illegible]		10.400	32.500	60.600
	05-12-1989	CALIBRATION	PASOPE22	73.000		0.000	19.56	NG	9.[illegible]	NG	101.1	NG
	05-12-1989	CALIBRATION	PASVAL21	73.000	Not Found		7.000	10.00	NG	40.500
	05-12-1989	CALIBRATION	PASVAL22	73.000		4.400	5.[illegible]	31.[illegible]	27.900

Chapter 9f

Miscellaneous

Standard Operating Procedure for

Envirochem Thermal Desorbtion

Hewlett Packard 5970 GC/MS

Volatile Organic Analytical System

STANDARD OPERATING PROCEDURE FOR
ENVIROCHEM THERMAL DESORBTION
HEWLETT PACKARD 5970 GC/MS
VOLATILE ORGANIC ANALYTICAL SYSTEM
11/88

NYS DEC
DIVISION OF AIR RESOURCES
BUREAU OF TOXIC AIR SAMPLING
AMBIENT TOXICS INVESTIGATIONS
GARRY A. BOYNTON

1. CLEAN UP

A. ENVIROCHEM (TENAX, AMBERSORB, CARBON)

Tubes in storage longest are chosen for cleaning and logged on a shipping list. Tube cleaner is preset for temperature, (290°C) and purge Flow (50-70^{cc}/min. (UPC N_2)). Time cycle is programed into Gralab electric timer - N_2 purge 20 min.

Heat cycle 10 min.

After cleaning cycle, tubes are immediately placed in teflon capped glass storage tubes and placed in foam lined shipping container.

2. SHIPPING

The foam lined container is sealed in a cardboard box for Parcel Post.

Extra foam is placed over the glass sample tubes to hold them in place. Business return mail placard is used to eliminate need for postage from field.

3. SAMPLING

See Toxics QA Manual Section 30 Sorbent Tube Vapor Collection

4. RETURN SHIPPING

All tubes from sampling event are shipped same method as to the field.

5. LOG IN

Once samples are received by ATIS staff they are logged into a computer to calculate flows and initiate the analysis process. The log in also creates a unique analyses number for future

tubes are cleaned up the same way as sample tubes. Calibration tubes are then generated at 200 ng per analyte and cycled the same as the internal ref. in the sample tubes.

7. ANALYSIS See Appendix for Parameters List

A. Timer controlled flush cycle - Timer to start Envirochem system should be at 5:15 AM to allow 3 cycles to flush the internal traps prior to an analysis run.

B. Turn MSD from standby to on and turn heater on evening before analysis run.

C. Turn timer controller off and unplug unit to prevent continued cycling and allow traps to cool below 50°C. Turn on both transfer lines, power and bring helium flow up to 4 (top of ball on rotameter) switch rear transfer valve to H.P. to allow carrier gas flow to equilibrate. Turn desorber control from off to auto.

D. Autotune - daily full autotune with P.F.T.B.A. as prescribed by Hewlett Packard - no modifications from standard H.P. method.

E. Turn on IBM PC and call up HP transfer program. This program accepts the final report transmitted by RS 232 from the chemstation and stores it in lotus compatible files for validation and printout. (see appendix for basic program)

F. Specify and load method (Auto.M or Calib.M) appropriate for analysis. Calibration is first analysis of day then blank

```
S I M   A C Q U I S I T I O N     10 Nov 88   9:13 am  DATA:THERM51.A

solvent delaY  8.00    eM volts     0 relAtive       resulting voltage 1400
-------------------------------------------------------------------------
      Group      1     2     3     4     5     6     7     8     9    10
  # of Ions     13    20    20    10    20    20    20    20    20    20
  start Time   8.00 11.00 14.00 19.00 ----- ----- ----- ----- ----- -----
low mass Resolution   NO                              cycles per second 0.9
-------------------------------------------------------------------------
ion #     1     2     3     4     5     6     7     8     9    10
  m/Z 47.00 49.00 61.00 62.00 63.00 64.00 83.00 84.00 85.00 86.00
Dwell    75    75    75    75    75    75    75    75    75    75

ion #    11    12    13
  m/Z 87.00 97.00 99.00
Dwell    75    75    75
-------------------------------------------------------------------------
   nUmber of plot traces 1        initiaLly ON         time Window 25.0

 Plot # 1  m/Z TOTAL   sCale 1000000


S I M   A C Q U I S I T I O N     10 Nov 88   9:13 am  DATA:THERM51.A

solvent delaY  8.00    eM volts     0 relAtive       resulting voltage 1400
-------------------------------------------------------------------------
      Group      1     2     3     4     5     6     7     8     9    10
  # of Ions     13    20    20    10    20    20    20    20    20    20
  start Time   8.00 11.00 14.00 19.00 ----- ----- ----- ----- ----- -----
low mass Resolution   NO                              cycles per second 0.6
-------------------------------------------------------------------------
ion #     1     2     3     4     5     6     7     8     9    10
  m/Z 49.00 60.00 61.00 62.00 63.00 64.00 77.00 78.00 83.00 85.00
Dwell    75    75    75    75    75    75    75    75    75    75

ion #    11    12    13    14    15    16    17    18    19    20
  m/Z 95.00 96.00 97.00 99.00 117.00 119.00 121.00 130.00 132.00 134.00
Dwell    75    75    75    75    75    75    75    75    75    75
-------------------------------------------------------------------------
   nUmber of plot traces 1        initiaLly ON         time Window 25.0

 Plot # 1  m/Z TOTAL   sCale 1000000


S I M   A C Q U I S I T I O N     10 Nov 88   9:13 am  DATA:THERM51.A

solvent delaY  8.00    eM volts     0 relAtive       resulting voltage 1400
-------------------------------------------------------------------------
      Group      1     2     3     4     5     6     7     8     9    10
  # of Ions     13    20    20    10    20    20    20    20    20    20
  start Time   8.00 11.00 14.00 19.00 ----- ----- ----- ----- ----- -----
low mass Resolution   NO                              cycles per second 0.6
-------------------------------------------------------------------------
ion #     1     2     3     4     5     6     7     8     9    10
  m/Z 61.00 77.00 83.00 85.00 91.00 92.00 95.00 96.00 97.00 99.00
Dwell    75    75    75    75    75    75    75    75    75    75

ion #    11    12    13    14    15    16    17    18    19    20
  m/Z 105.00 106.00 112.00 114.00 129.00 131.00 133.00 164.00 166.00 168.00
Dwell    75    75    75    75    75    75    75    75    75    75
-------------------------------------------------------------------------
   nUmber of plot traces 1        initiaLly ON         time Window 25.0
```

8- 80

```
REPORT GENERATOR                                  Rev 3.1.1 31-Mar-86

  Integration
 Results File: DATA:GORPT.1

  Report Type: ESTD          Format : Summary
               Cal File name :  DATA:CALEXSTD.0

  Destination: Disc File
               Dest File name : DATA:GORPT.ASC

    Sequence
   Macro File: DATA:AUTORPT
CALIBRATION                                       Rev 3.1.1 31-Mar-86

 Integration Results File: DATA:GORPT.1

   Calibration Table File: DATA:CALEXSTD.0
             Last Update:  4 Nov 86  10:05 am

       Calibrate By: Area          Level Number:  1    Replace Level

     Level number exists, recalibration of this level assumed

   ---------------- Calibration Table Header Information ---------------

 Title: THERMAL DESORBTION CALIBRATION WITH PERMEATION TUBES

  Multiplier: 1.000       Sample Amount: 0.000       Uncal Peak RF: 0.000
              Signal Correlation Window: 0.03010    min

                    Recognition Windows, as Percent of RT
      Reference Peaks: 5.000                Non-Reference Peaks: 5.000
```

*** CARBON-TETRAC ***

Peak Num	Type	Int Type	Ret Time	Signal Description	Compound Name	Area	Amount
6		1BB	12.159	117.00- 119.00 amu	CARBON-TETRA	138853	12.44 NG

*** No Additional Qualifiers ***

Counts * Response Factor = Corrected Amt
138853 * 95.87e-6 = 12.44

*** TRICHLETHENE ***

Peak Num	Type	Int Type	Ret Time	Signal Description	Compound Name	Area	Amount
7		1BBA	12.949	130.00- 134.00 amu	TRICHLETHENE	132854	5.985 NG

*** No Additional Qualifiers ***

Counts * Response Factor = Corrected Amt
132854 * 45.05e-6 = 5.985

*** 112TRICHETHAN ***

Peak Num	Type	Int Type	Ret Time	Signal Description	Compound Name	Area	Amount
8		1	-------	95.00- 99.00 amu	112TRICHETHA	*** Not Found ***	

*** TOLUENE ***

Peak Num	Type	Int Type	Ret Time	Signal Description	Compound Name	Area	Amount
9		1PBA	14.797	Mass 91.00 amu	TOLUENE	29410070	424.4 NG

*** Qualifiers ***

Channel	Description	Exp Resp	Tolerance	Actual Resp
Mass	92.00 amu	60.00	* 90/-30%	59.67

Counts * Response Factor = Corrected Amt
29410070 * 20.79e-6 = 424.4

*** TETRACHLETHEN ***

Peak Num	Type	Int Type	Ret Time	Signal Description	Compound Name	Area	Amount
10		1BBA	16.988	164.00- 170.00 amu	TETRACHLETHE	1359475	39.71 NG

*** Qualifiers ***

Channel	Description	Exp Resp	Tolerance	Actual Resp
129.00-	133.00 amu	50.00	* 85/-40%	81.65

Counts * Response Factor = Corrected Amt
1359475 * 29.21e-6 = 39.71

Counts * Response Factor * Corrected Amt
409335 * 28.66e-6 * 11.73

*** 14-DICHLBENZ ***

Peak Num	Type	Int Type	Ret Time	Signal Description	Compound Name	Area	Amount
16		1	-------	146.00- 148.00 amu	14-DICHLBENZ	*** Not Found ***	

*** 12-DICHLBENZ ***

Peak Num	Type	Int Type	Ret Time	Signal Description	Compound Name	Area	Amount
17		1BB	21.585	146.00- 148.00 amu	12-DICHLBENZ	129535	3.623 NG

*** Qualifiers ***

Channel	Description	Exp Resp	Tolerance	Actual Resp
74.00-	76.00 amu	30.00	* 50/-15%	*** Not Found ***
Mass	111.00 amu	25.00	* 50/-10%	*** Not Found ***

Counts * Response Factor * Corrected Amt
129535 * 27.97e-6 * 3.623

*** FLUO-21000-BENZENE ***

Peak Num	Type	Int Type	Ret Time	Signal Description	Compound Name	Area	Amount
18 *		1BV	22.674	Mass 222.00 amu	FLUO-21000-B	7786038	503.9 NG

*** Qualifiers ***

Channel	Description	Exp Resp	Tolerance	Actual Resp
74.00-	76.00 amu	30.00	* 45/-15%	39.57
Mass	95.00 amu	20.00	* 45/-15%	47.30

Counts * Response Factor * Corrected Amt
7786038 * 64.72e-6 * 503.9
Calibration Peak #2's Qualifiers were not satisfied.
Error : Could not find Calibration Peak

Chapter 9g

Miscellaneous

Staten Island/Northern New Jersey Urban Air Toxic Assessment Project Quality Assurance Subcommittee Audit Report

STATEN ISLAND/NORTHERN NEW JERSEY
URBAN AIR TOXICS ASSESSMENT PROJECT
QUALITY ASSURANCE SUBCOMMITTEE
AUDIT REPORT
OF THE
NEW YORK STATE DEPARTMENT OF ENVIRONMENTAL CONSERVATION

Auditors:

Avraham Teitz
Monitoring Management Branch, USEPA - Region II

Joseph Soroka, Ph.D
Technical Support Branch, USEPA - Region II

Paul Brown
Monitoring Management Branch, USEPA - Region II

Approved by:

Marcus Kantz, QA Subcommittee Chairman
Monitoring Management Branch, USEPA -Region II

BACKGROUND

This Audit report contains information on the performance of the New York State Department of Environmental Conservation (NYSDEC), in carrying out their duties and responsibilities for the Staten Island/New Jersey Urban Air Toxics Assessment Project (SI/NJ UATAP). Specific areas evaluated were the implementation of field and laboratory procedures used by NYSDEC. The findings reported are the result of data submitted by NYSDEC, conversations and meetings with NYSDEC researchers and an onsite audit. Conclusions and recommendations are reported at the end of this document. This report has been prepared by the United States Environmental Protection Agency - Region II for the Quality Assurance Subcommittee of the SI/NJ UATAP.

Agency audited: New York State
Department of Environmental Conservation
Air Resources
50 Wolf Road
Albany, New York 12233-3527
(518) 457-7454

On site portion of Audit: September 15, 1988

Personnel present at audit:

Joseph Soroka, Ph.D	- USEPA - Region II
Paul Brown	- USEPA - Region II
Avraham Teitz	- USEPA - Region II
Garry Boynton	- NYSDEC
Brian Lay	- NYSDEC

Organization responsibilities for the SI/NJ UATAP:

1. Collect samples of Tenax, sorbent traps,canisters formaldehyde, and metals at appropriate sites.

2. Prepare and analyze sorbent traps from NYSDEC sites in Staten Island.

3. Maintain all samplers used in the study.

Project Director:	Don Gower
Monitoring Network Manager:	Will Smith
Quality Assurance Officer:	Ray McDermot
Field Operations Supervisor:	Mike Steineger
Laboratory Supervisor:	Garry A. Boynton
Data Management Supervisor:	Brian Lay

points of each method are demonstrated. This includes special handling to avoid contamination and hands on experience. Training culminates with the trainee demonstrating proficiency with the use of the equipment while being monitored by the Field Operations Supervisor.

4. The samplers for all sorbent trap media, and the high volume metal samplers, function on the same principles, and are calibrated in the same fashion. The principle of operation is a vacuum drawing air through a critical orifice. After every sampling run, a manometer is used to measure the pressure drop across the orifice. This number is recorded, and using the calibration equation for the orifice and the time that the sampler was run, the volume of air sampled is computed. The critical orifice is audited quarterly by the Field Operations Supervisor using a mass flow controller whose calibration is traceable to a primary standard. Audits are conducted by NYSDEC Quality Assurance personnel to monitor field performance of the samplers. The criterion for acceptable performance of any sampler is ±10% of the actual level.

The formaldehyde and canister samplers used by NYSDEC are housed together, but use different sampling lines. Flows for both are regulated by mass flow controllers. The flow rates were measured at the start of the study, but QA/QC measures have not yet been implemented for insuring the calibration of the mass flow controllers. This is more important with formaldehyde samples because canister performance can be judged by the final pressure after sampling. Also, measurements of flow rate are not needed to determine concentrations of pollutants in a canister sample, as the whole air sampled is contained in the canister; this is not the case with formaldehyde samples.

5. Travel blanks for Tenax, Envirochem, and ATD-50 samples are handled as follows. Samples are received by the field office in bulk, with generally 10-30 tubes per shipment. One sealed tube of each type is taken to a site where that particular tube will be sampled that day. After sampling, the sealed travel blank is sent to the appropriate laboratory together with all the sorbent tubes of the same type that were run that day. In the case that not enough sampling tubes are available, travel blanks can not be sent, although this has been the exception rather than the rule.

Travel blanks for the Poropak samples are handled differently. Upon receipt of a shipment of Poropak tubes, two tubes are selected as travel blanks. These blanks are taken to a site and left sealed on site for four to five days. After this time they are sent to the laboratory for analysis. Poropak tubes from regular sampling runs are not accompanied by a travel blank on the return trip from the field to the laboratory. Travel blanks are not used for canister and formaldehyde samples. Duplicate samples for sorbent media are taken at each sampling run as follows: Two tubes are run at different flow rates, as are all sorbent tubes throughout the study; and a third tube is run at the higher of the two flow rates. Formaldehyde and canister samples are not duplicated.

sorbent cleaning and the packing of sorbent into the tubes is carried out by a contractor to the state. When new tubes are received by the NYSDEC, they twice undergo the standard cleaning procedure used to clean sampled tubes.

After tubes have been sampled and analyzed, they are cleaned by heating them for 10 minutes at 290C and are then purged for 10 minutes with an nitrogen. Six tubes are cleaned at one time and 10% of all cleaned tubes are treated as oven blanks. This cleaning has been found by NYSDEC to be sufficient, with background levels of cleaned blanks having < 60 ng of benzene and < 12-20 ng toluene. Tubes are replaced when background levels of organic compounds begin to rise in the oven blanks. This has not been observed to date, and this has been attributed to the fact that each tube has only been recycled 15-16 times.

3. Analysis of all sorbent tubes is by GC/MS. Compounds used as standards are obtained as NBS certified liquid permeation tubes and placed in permeation ovens. Calibration of the tubes is taken per the manufacturers specifications. Direct weighing of permeation tubes, to give direct gravimetric determinations of permeation efficiency was attempted, but fluctuations in the weight of the tube, due to heating effects, static electricity and humidity rendered this approach impractical.

4. The NYSDEC calibration system is set up as follows: Permeation tubes are first grouped according to the temperature that is specified in their calibration. Each group of tubes is then placed in an oven set at the appropriate temperature. Currently there are four Metronix ovens in 18 tubes in use. All the ovens outputs are ganged together by copper tubing that was cleaned with hexane and methanol and air then dried. A pump is in constant operation which sucks zero air through the permeation ovens. Zero air is obtained by cooling air to a dew point of -100C and then passing it through two canisters of carbon, one canister of molecular sieve, and one canister of Drierite. Air flow through the system is controlled with mass flow controllers. This zero air is checked daily through the calibration process. When a tube is to be calibrated, it is connected to a 1/4" Swagelok fitting and the output of the permeation ovens are diverted through the tube. The calibration system is set-up in such a manner that the output of any of the four permeation ovens in any combination may be diverted through the tube to be calibrated. Up to four sorbent tubes may be spiked at one time. Spike concentration is a function of the time that the oven outputs flow through the sorbent tube and the quantity of dilution air.

Minimum detection limits (MDL) are determined as follows. Injections of 25 ng of each compound are made into the GC/MS system. Then data are examined from previous runs where lower levels (0-20 ng) of compounds were observed. These two sets of data were compared for their relationship and consistency and determinations of MDL made. System linearity has also been checked and found to hold for levels up to 300 ng for the Envirochem tubes and at least 200 ng with the ATD-50 tubes.

samples. Oven blanks are examined to insure that their levels do not exceed criteria that were established during a cleaning study at the beginning of the project. Contamination of tubes is determined at the discretion of the laboratory supervisor.

9. Laboratory equipment is maintained by Garry Boynton. Repairs of equipment is done by the appropriate manufacturer although no service contracts are in place. Criteria for column replacement are that peak shapes and retention times differ from the norm. Parameters for repair of the MS are evident from the results of the daily autotune procedure. To date, no columns have been replaced and neither source has yet had to be cleaned.

10. Documentation of samples received by the laboratory includes a unique sample number and the field data sheet. Logbooks are kept for most laboratory instruments. Data transfer from the GC/MS to a personal computer for data manipulation is done automatically. Raw data is stored on a Hewlett Packard minicomputer for a period of two years and is then archived on tape.

ATTACHMENT #1

NYS Department of Environmental Conservation

Staten Island

Toxic Air Monitoring Status

Site	Porapak	2 Tube Tenax	Grad. Adsorbent	EPA CAM.	Auto GC's	Hi-Vol	Formaldehyde	Wind Spd Wind Dir	Met. Data Reporting	Sorbent Tube Data Reporting	Trace Metals Porapak Data Reporting
#1 S. Wagner H.S.	4/85	10/87 (EMI/UMDNJ)	7/87 (Envirochem)	4/27/88 (Every 6 days)	3-Oper 7/87-7/88 (HOLD)	Ongoing (Trace Metals)	7/88	Ongoing	Through 7/88	10/87 6/1/88-EPA Format 2nd Qtr. 1988	7/88 All Data
#2 P.S. #26 Travis	4/85	5/27/88 (UMDNJ)	7/87 (Envirochem)	8/88 (Every 18 days)	N.A.	Ongoing (Trace Metals)	N.A.	N.A.	N.A.	10/87 6/1/88-EPA Format 2nd Qtr. 1988	7/88 All Data
#4 Fire Station Great Kills	N.A.	N.A.	8/88 START 8/31 (ATD-50)	8/88 (Every 18 days)	N.A.	N.A.	N.A.	N.A.	N.A.	10/1/88 EPA Format 3rd Qtr. 1988	N.A.
#5 Post Office Port Richmond	N.A.	10/87 (EMI/ Texas A&M)	7/87 (Envirochem/ ATD-50)	6/88 (Every 6 days)	HOLD	TSP only	7/88	Optional	Optional	10/87 6/1/88 EPA Format	N.A.
#7 Pump Station S.I. Mall	N.A.	N.A.	9/88 START 9/30 ATD-50	9/88 (Every 18 days)	HOLD	N.A.	N.A.	9/30/88	9/30/88	10/1/88 EPA format 3rd Qtr. 1988	N.A.
#9 Fire Station Tottenville	N.A.	5/27/88 (EMI/ Texas A&M)	7/88 START (ATD-50)	6/88 (Every 18 days)	N.A.	N.A.	N.A.	4/27/88 (also R.N.)	Through 7/88	10/1/88 EPA Format 3rd Qtr. 1988	N.A.

8/23/88
88-3-141

APPENDIX A

RESULTS OF

GAS CHROMATOGRAPH FIELD AUDITS

PPB FIELD AUDIT REPORT AUDIT # 312 page 1

PART A - To be filled out by organization supplying audit cylinders

1. Individual requesting Audit, organization, and phone number:
   Ray McDermolt, NY State Dept. of Env. Consevation

2. Audit supervisor, organization, and phone number:
   Dr. R.K.M. Jayanty, Research Triangle Institute (919) 541-6483

3. Shipping instructions:
   Ray McDermolt
   NY State Dept. of Env. Conservation
   QA Section
   50 Wolf Road
   Albany, NY 12233-3253
4. Cylinder shipping date: 1-24-1989
5. Expected arrival date: 2-1-1989

6. Audit cylinder status:
   a. Cylinder ID: AAL 19655
   b. Pressure, psig: 1120
   c. Construction: aluminum
   d. Balance gas: nitrogen

7. Organic chemical manufacturing process:
   none

8. Name of individual or organization being audited:
   in house

9. Location of audit:
   Albany, NY

10. Use of audit material:
    Ascertain accuracy of adsorption tube sampling and GC/FID analysis procedures used at Staten Island toxic ambient air monitoring study.
    a. Sampling method: adsorption tube (charcoal)
    b. Analytical method: GC/FID
    c. Accuracy assessment of:

    [ ] VOST method prior to RCRA trial burn
    [ ] bag method prior to RCRA trial burn
    [ ] VOST method during actual RCRA trial burn
    [ ] bag method during actual RCRA trial burn
    [XX] measurement method used for routine ambient air measurements
    [ ] measurement method used at hazardous waste landfill
    [ ] other (explain):_____

cc: Darryl von Lehmden, EPA/EMSL, RTP
    Robert Lampe, EPA/EMSL, RTP
    Gary Boynton, Bureau of Toxic Air Sampling, NY

PPB FIELD AUDIT REPORT AUDIT # 370 page 1

PART A - To be filled out by organization supplying audit cylinders

1. Individual requesting Audit, organization, and phone number:
   Ray McDermolt, NY State Dept. of Env. Conservation

2. Audit supervisor, organization, and phone number:
   Dr. R.K.M. Jayanty, Research Triangle Institute (919) 541-6483

3. Shipping instructions:
   Ray McDermolt
   NY State Dept. of Env. Conservation
   QA Section
   50 Wolf Road
   Albany, NY 12233-3253
4. Cylinder shipping date: 08/8/89
5. Expected arrival date: 08/8/89

6. Audit cylinder status:
   a. Cylinder ID: 370
   b. Pressure, psig: 1300
   c. Construction: aluminum
   d. Balance gas: nitrogen

7. Organic chemical manufacturing process:
   none

8. Name of individual or organization being audited:
   no contractor

9. Location of audit:
   NY State Lab

10. Use of audit material:
    Ascertain accuracy of charcoal tube sampling and GC/FID analysis procedures prior to ambient air sampling at Staten Island
    a. Sampling method: Adsorption Tube(charcoal)
    b. Analytical method: GC/FID
    c. Accuracy assessment of:

    [ ] VOST method prior to RCRA trial burn
    [ ] bag method prior to RCRA trial burn
    [ ] VOST method during actual RCRA trial burn
    [ ] bag method during actual RCRA trial burn
    [XX] measurement method used for routine ambient air measurements
    [ ] measurement method used at hazardous waste landfill
    [ ] other (explain):_____

cc: Darryl von Lehmden, EPA/AREAL, RTP
    Robert Lampe, EPA/AREAL, RTP

APPENDIX B

A Sample of a Field

Flow Performance Audit

3rd Quarter 1989

27-Sep-89 TABLE 1

NEW YORK STATE DEPT. OF ENVIRONMENTAL CONSERVATION
DIVISION OF AIR RESOURCES BUREAU OF TECHNICIAL SERVICES
QUALITY ASSURANCE SECTION

ATD-50 AUDIT RESULTS
3rd QUARTER 1989

Site	Sampler Type	Flow Module#	Cert.Flow/VOTS %d	Calc.Flow/VOTS %d	Comments
S. WAGNER 7097-01	ATD-50	L010	6.7	-9.7	Changed vacuum relief valve
		L082	-8.2	-0.2	
		L023	-3.4	-3.9	
PUMP STA. 7097-08	ATD-50	L0103	-5.9	-0.2	
		L0107	-8.8	-2.4	
		L0033	2.3	-11.4	
GREAT KILLS 7097-06	ATD-50	L070	-11.1	4.8	
		L015	-7.6	-0.9	
		L054	-3.7	-3.5	
TOTTENVILLE 7097-05	ATD-50	L0057	-11.4	-1.7	
		L0044	-10.6	-1.0	
		L0041	-6.6	-2.0	
P.S. 26 7097-02	ATD-50	L0036	-13.6	-3.9	
		L0022	-8.6	-2.9	
		L0003	-1.5	-3.4	
P. RICHMOND 7093-07	ATD-50	L010	-8.2	-4.1	
		L0040	-17.6	-1.6	
		L0002	-6.0	-3.2	

28-Sep-89

TABLE 2

NEW YORK STATE DEPT. OF ENVIRONMENTAL CONSERVATION
DIVISION OF AIR RESOURCES BUREAU OF TECHNICIAL SERVICES
QUALITY ASSURANCE SECTION

CANISTER, HI-VOL, AND PM-10 SAMPLER AUDIT RESULTS
3rd QUARTER 1989

Site	Sampler Type	Audit Device Flow	Sampler Flow	%d
S. WAGNER 7097-01	CANISTER	8.95 cc/min	N/A	N/A
	WEDDING	36.08 CFM	40.92 CFM	13.4
	TRACE METALS	40.43 CFM	40.99 CFM	1.4
PUMP STA. 7097-08	CANISTER	8.66 cc/min	N/A	N/A
GREAT KILLS 7097-06	CANISTER	8.31 cc/min	N/A	N/A
TOTTENVILLE 7097-05	CANISTER	9.63 cc/min	N/A	N/A
P.S. 26 7097-02	CANISTER	8.59 cc/min	N/A	N/A
	WEDDING	38.12 CFM	40.21 CFM	5.5
	TRACE METALS	40.81 CFM	40.39 CFM	-1.0
P. RICHMOND 7097-03	CANISTER	8.91 cc/min	N/A	N/A
	WEDDING	36.91 CFM	40.31 CFM	9.2
	TRACE METALS	38.52 CFM	39.17 CFM	1.7

New York State Department of Environmental Conservation
Division of Air

Bureau of Technical Services
Quality Assurance Section

Volatile Organic Sampler Performance Audit

Date 9/20/89

Site Name Susan Wagner Site Number 7097-01

Type of Sampler ATD 50 Performed by McDermott/White

VOTS Box No. 2 Cert. Date 4/23/89 Vac. Test Gauge No. A020

Meas. Mod. No. 5026 Slope 1.07 Inter 4.689 Cert. Date 12/9/88

Temp. 81.4 °F 27.7 °C Vacuum Relief Valve Vac. 21.5 "Hg.

Atmospheric Pressure 29.74 "Hg. 755 mm Hg.

Correction Factors:

$$A = \left[\frac{298}{T + 273}\right] \times \left[\frac{P}{29.92}\right] = 0.985$$

T= Actual Temperature °C P= Actual Pressure "Hg

Flow Module Number 1

Module No. L010 Cert. Date 3/28/89 Cert. Flow 8.43 cc/min

VOTS Flow Box 7.9 cc/min Percent d (Cert./VOTS)= +6.7 %

Manometer Reading R 0.75 L 0.75 Total 1.5

Calc. Flow 7.23 cc/min X (Corr. Fact. A)= 7.13 cc/min

Percent d (Calc./VOTS)= -9.7 %

Flow Module Number 2

Module No. L082 Cert. Date 12/27/88 Cert. Flow 20.74 cc/min

VOTS Flow Box 22.6 cc/min Percent d (Cert./VOTS)= -8.23 %

Manometer Reading R 2.2 L 2.2 Total 4.4

Calc. Flow 22.9 cc/min X (Corr. Fact. A)= 22.5 cc/min

Percent d (Calc./VOTS)= -0.25 %

After replacing vacuum relief value

New York State Department of Environmental Conservation
Division of Air

Bureau of Technical Services
Quality Assurance Section

Volatile Organic Sampler Performance Audit

Date 9/20/89

Site Name Susan Wagner Site Number 7097-01

Type of Sampler ATD 50 Performed by McDermett/White

VOTS Box No. 2 Cert. Date 4/23/89 Vac. Test Gauge No. HG20

Meas. Mod. No. 5026 Slope 1.07 Inter 4.689 Cert. Date 12/9/88

Temp. 81.4 °F 27.7 °C Vacuum Relief Valve Vac. 20.5 "Hg.

Atmospheric Pressure 29.74 "Hg. 755 mm Hg.

Correction Factors:

$$A = \left[\frac{298}{T + 273}\right] \times \left[\frac{P}{29.92}\right] = 0.985$$

T= Actual Temperature °C P= Actual Pressure "Hg

Flow Module Number 1

Module No. L010 Cert. Date 3/28/89 Cert. Flow 8.43 cc/min

VOTS Flow Box 7.4 cc/min Percent d (Cert./VOTS)= +13.92 %

Manometer Reading R 0.7 L 0.7 Total 1.4

Calc. Flow 6.72 cc/min X (Corr. Fact. A)= 6.62 cc/min

Percent d (Calc./VOTS)= -10.5 %

Flow Module Number 2

Module No. L082 Cert. Date 12/27/88 Cert. Flow 20.74 cc/min

VOTS Flow Box 21.7 cc/min Percent d (Cert./VOTS)= -4.4 %

Manometer Reading R 2.0 L 2.0 Total 4.0

Calc. Flow 20.67 cc/min X (Corr. Fact. A)= 20.36 cc/min

Percent d (Calc./VOTS)= -6.17 %

Canister Sampler

New York State Department of Environmental Conservation
Division of Air

Bureau of Technical Services
Quality Assurance Section

See comments section

Volatile Organic Sampler Performance Audit

Date 9/26/89

Site Name Susan Wagner Site Number 7097-01

Type of Sampler Canister Performed by McDermett/White

VOTS Box No.____ Cert. Date _____ Vac. Test Gauge No. ____

Meas. Mod. No._____Slope_____Inter_____Cert. Date_____

Temp. 81.4 °F 27.7 °C Vacuum Relief Valve Vac. _____"Hg.

Atmospheric Pressure 29.74 "Hg. 755 mm Hg.

Correction Factors:

$$A=\left[\frac{298}{T+273}\right] X \left[\frac{P}{29.92}\right] = \text{_____}$$

T= Actual Temperature °C P= Actual Pressure "Hg

Flow Module Number 1

Module No. _____ Cert. Date _____ Cert. Flow _____cc/min

VOTS Flow Box _____cc/min Percent d (Cert./VOTS)=_____%

Manometer Reading R _____ L _____ Total _____

Calc. Flow _____cc/min X (Corr. Fact. A)= _____cc/min

Percent d (Calc./VOTS)=_____%

Flow Module Number 2

Module No. _____ Cert. Date _____ Cert. Flow _____cc/min

VOTS Flow Box _____cc/min Percent d (Cert./VOTS)=_____%

Manometer Reading R _____ L _____ Total _____

Calc. Flow _____cc/min X (Corr. Fact. A)=_____cc/min

Percent d (Calc./VOTS)=_____%

NEW YORK STATE DEPARTMENT OF ENVIRONMENTAL CONSERVATION
DIVISION OF AIR

BUREAU OF TECHNICAL SERVICES
QUALITY ASSURANCE SECTION

PARTICULATE SAMPLER FLOW AUDIT

DATE 9/20/89 AUDITOR McDermott/White

SITE NAME Susan Wagner SITE NUMBER 7097-01

TEMP. 81.4 °F 27.7 °C ATM. PRES. 29.74 "HG 755 mm Hg

SAMPLER TYPE: TSP_____ TRACE METALS_____ PM10-WEDDING ✓

COLLOCATED____ LEAD____ PM10- DICOT_____. PM10-ANDERSEN_____

SLOPE
Q(FLOW CFM)= INTERCEPT X (ΔH)

AUDIT DEVICE DATA: CHK. INT. USED
30 °F___
40 °F___
60 °F___

ID# _____ PRI. CAL._____ SLOPE_____INT._____

LEFT _____" RIGHT_____" TOTAL_____" Q_____CFM

SAMPLER DATA: CHK. INT. USED
30 °F___
40 °F___
60 °F___

ID# _____ PRI. CAL._____SLOPE_____ INT._____

LEFT _____" RIGHT_____" TOTAL_____" Q_____CFM

PERCENT DIFF. _____%

DICOT ONLY:	ROTOMETER COB	ROTOMETER LPM	ORIFICE DEVICE SN	ORIFICE DEVICE H	ORIFICE DEVICE LPM	PERCENT DIFF.
TOTAL FLOW:	_____	_____	_____	____	_____	_____
COARSE FLOW:	_____	_____	_____	____	_____	_____

AUDITOR CHECKS (INDICATE EQUIPMENT CONDITION):

SHELTER	OK	GASKET	OK
MOTOR	OK	TIMER	OK
ORIFICE	N/A	ELECTRICAL WIRING	OK
MANOMETER & TUBING	OK	CHECK ORIFICE NO.	N/A

COMMENTS: _____

_____

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NEW YORK STATE DEPARTMENT OF ENVIRONMENTAL CONSERVATION
DIVISION OF AIR

BUREAU OF TECHNICAL SERVICES
QUALITY ASSURANCE SECTION

PARTICULATE SAMPLER FLOW AUDIT

DATE 9/20/89 AUDITOR White/McDermott

SITE NAME Susan Wagner SITE NUMBER 7097-01

TEMP. 81.4 °F 27.7 °C ATM. PRES. 29.74 "HG 755 mm Hg

SAMPLER TYPE: TSP ✓ TRACE METALS ✓ PM10-WEDDING ____

COLLOCATED ____ LEAD ____ PM10- DICOT ____ . PM10-ANDERSEN ____

Q(FLOW CFM) = INTERCEPT X (ΔH) SLOPE

AUDIT DEVICE DATA: CHK. INT. USED 30°F ___ 40°F ___ 60°F ✓

ID# 15438 PRI. CAL. 9/29/88 SLOPE 0.50 INT. 17.4

LEFT 1.8 " RIGHT 3.6 " TOTAL 5.4 " Q 40.43 CFM

SAMPLER DATA: CHK. INT. USED 30°F ___ 40°F ___ 60°F ✓

ID# 90437 PRI. CAL. 1/30/89 SLOPE 0.52 INT. 24.0

LEFT 1.4 " RIGHT 1.4 " TOTAL 2.8 " Q 40.99 CFM

PERCENT DIFF. +1.4 %

DICOT ONLY:

	ROTOMETER COB	ROTOMETER LPM	ORIFICE DEVICE SN	ORIFICE DEVICE H	ORIFICE DEVICE LPM	PERCENT DIFF.
TOTAL FLOW:	____	____	____	____	____	____
COARSE FLOW:	____	____	____	____	____	____

AUDITOR CHECKS (INDICATE EQUIPMENT CONDITION):

SHELTER	OK	BASKET	OK
MOTOR	OK	TIMER	OK
ORIFICE	OK	ELECTRICAL WIRING	OK
MANOMETER & TUBING	OK	CHECK ORIFICE NO.	OK

COMMENTS: ____

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New York State Department of Environmental Conservation
Division of Air

Bureau of Technical Services
Quality Assurance Section

Volatile Organic Sampler Performance Audit

Date 9/20/89

Site Name Mall Pump Station Site Number 7097-08

Type of Sampler ATD 50 Performed by McDermott/White

VOTS Box No. 2 Cert. Date 4/23/89 Vac. Test Gauge No. A020

Meas. Mod. No. 5014 Slope 0.997 Inter 4.097 Cert. Date 5/15/89

Temp. 78.6 °F 26.2 °C Vacuum Relief Valve Vac. 21.0 "Hg.

Atmospheric Pressure 30.03 "Hg. 763 mm Hg.

Correction Factors:

$$A = \left[\frac{298}{T + 273}\right] \times \left[\frac{P}{29.92}\right] = 0.9996$$

T= Actual Temperature °C P= Actual Pressure "Hg

Flow Module Number 1

Module No. L0103 Cert. Date 5/9/89 Cert. Flow 8.09 cc/min

VOTS Flow Box 8.6 cc/min Percent d (Cert./VOTS)= -5.9 %

Manometer Reading R 1.1 L 1.0 Total 2.1

Calc. Flow 8.58 cc/min X (Corr. Fact. A)= 8.58 cc/min

Percent d (Calc./VOTS)= -0.2 %

Flow Module Number 2

Module No. L0107 Cert. Date 6/2/89 Cert. Flow 17.15 cc/min

VOTS Flow Box 18.8 cc/min Percent d (Cert./VOTS)= -8.8 %

Manometer Reading R 2.3 L 2.2 Total 4.5

Calc. Flow 18.35 cc/min X (Corr. Fact. A)= 18.35 cc/min

Percent d (Calc./VOTS)= -2.4 %

Canister Sampler 147

New York State Department of Environmental Conservation
Division of Air

Bureau of Technical Services
Quality Assurance Section

See comments section

Volatile Organic Sampler Performance Audit

Date 9/20/89

Site Name Mall Pump Station Site Number 7097-08

Type of Sampler Canister Performed by McDermott/White

VOTS Box No.____ Cert. Date _____ Vac. Test Gauge No. ____

Meas. Mod. No._____Slope_____Inter_____Cert. Date_____

Temp. 79.6 °F 26.2 °C Vacuum Relief Valve Vac. _____"Hg.

Atmospheric Pressure 30.03 "Hg. 763 mm Hg.

Correction Factors:

$$A=\left[\frac{298}{T+273}\right] X \left[\frac{P}{29.92}\right] = \text{-------}$$

T= Actual Temperature °C P= Actual Pressure "Hg

Flow Module Number 1

Module No. _____ Cert. Date _____ Cert. Flow _____cc/min

VOTS Flow Box _____cc/min Percent d (Cert./VOTS)=_____%

Manometer Reading R _____ L _____ Total _____

Calc. Flow _____cc/min X (Corr. Fact. A)= _____cc/min

Percent d (Calc./VOTS)=_____%

Flow Module Number 2

Module No. _____ Cert. Date _____ Cert. Flow _____cc/min

VOTS Flow Box _____cc/min Percent d (Cert./VOTS)=_____%

Manometer Reading R _____ L _____ Total _____

Calc. Flow _____cc/min X (Corr. Fact. A)=_____cc/min

Percent d (Calc./VOTS)=_____%

New York State Department of Environmental Conservation
Division of Air

Bureau of Technical Services
Quality Assurance Section

Volatile Organic Sampler Performance Audit

Date 9/21/89

Site Name Great Kills Site Number 7097-06

Type of Sampler ATD-50 Performed by McDermott/White

VOTS Box No. 2 Cert. Date 4/23/89 Vac. Test Gauge No. A020

Meas. Mod. No. 5019 Slope 0.993 Inter 7.45 Cert. Date 8/26/88

Temp. 78 °F 25.5 °C Vacuum Relief Valve Vac. 22.5 "Hg.

Atmospheric Pressure 29.81 "Hg. 757 mm Hg.

Correction Factors:

$$A = \left[\frac{298}{T + 273}\right] \times \left[\frac{P}{29.92}\right] = 0.9946$$

T= Actual Temperature °C P= Actual Pressure "Hg

Flow Module Number 1

Module No. L070 Cert. Date 8/26/88 Cert. Flow 7.82 cc/min

VOTS Flow Box 8.8 cc/min Percent d (Cert./VOTS)= -11.1 %

Manometer Reading R 0.6 L 0.7 Total 1.3

Calc. Flow 9.27 cc/min X (Corr. Fact. A)= 9.22 cc/min

Percent d (Calc./VOTS)= +4.8 %

Flow Module Number 2

Module No. L015 Cert. Date 8/26/88 Cert. Flow 16.17 cc/min

VOTS Flow Box 17.5 cc/min Percent d (Cert./VOTS)= -7.6 %

Manometer Reading R 1.2 L 1.3 Total 2.5

Calc. Flow 17.75 cc/min X (Corr. Fact. A)= 17.65 cc/min

Percent d (Calc./VOTS)= +0.88 %

Canister Sampler

New York State Department of Environmental Conservation
Division of Air

See comment section

Bureau of Technical Services
Quality Assurance Section

Volatile Organic Sampler Performance Audit

Date 9/21/89

Site Name Great Kills Site Number 7097-06

Type of Sampler Canister Performed by McDermott/White

VOTS Box No.____ Cert. Date _____ Vac. Test Gauge No. ____

Meas. Mod. No._____Slope_____Inter_____Cert. Date_____

Temp. 78 °F 25.5 °C Vacuum Relief Valve Vac. _____"Hg.

Atmospheric Pressure 29.81 "Hg. 757 mm Hg.

Correction Factors:

$$A=\left[\frac{298}{T+273}\right]\times\left[\frac{P}{29.92}\right]=$$ --------

T= Actual Temperature °C P= Actual Pressure "Hg

Flow Module Number 1

Module No. _____ Cert. Date _____ Cert. Flow _____cc/min

VOTS Flow Box _____cc/min Percent d (Cert./VOTS)=_____%

Manometer Reading R _____ L _____ Total _____

Calc. Flow _____cc/min X (Corr. Fact. A)= _____cc/min

Percent d (Calc./VOTS)=_____%

Flow Module Number 2

Module No. _____ Cert. Date _____ Cert. Flow _____cc/min

VOTS Flow Box _____cc/min Percent d (Cert./VOTS)=_____%

Manometer Reading R _____ L _____ Total _____

Calc. Flow _____cc/min X (Corr. Fact. A)=_____cc/min

Percent d (Calc./VOTS)=_____%

New York State Department of Environmental Conservation
Division of Air

Bureau of Technical Services
Quality Assurance Section

Volatile Organic Sampler Performance Audit

Date 9/20/89

Site Name Tottenville Site Number 7097-05

Type of Sampler ATD 50 Performed by McDermott/White

VOTS Box No. 2 Cert. Date 4/23/89 Vac. Test Gauge No. A020

Meas. Mod. No 5027 Slope 1.05 Inter 3.465 Cert. Date 3/8/89

Temp. 79.5 °F 26.2 °C Vacuum Relief Valve Vac. 21.3 "Hg.

Atmospheric Pressure 29.96 "Hg. 761 mm Hg.

Correction Factors:

$$A = \left[\frac{298}{T + 273}\right] \times \left[\frac{P}{29.92}\right] = 0.997$$

T= Actual Temperature °C P= Actual Pressure "Hg

Flow Module Number 1

Module No. L0057 Cert. Date 6/2/89 Cert. Flow 7.8 cc/min

VOTS Flow Box 8.8 cc/min Percent d (Cert./VOTS)= -11.4 %

Manometer Reading R 1.2 L 1.2 Total 2.4

Calc. Flow 8.69 cc/min X (Corr. Fact. A)= 8.66 cc/min

Percent d (Calc./VOTS)= -1.70 %

Flow Module Number 2

Module No. L0044 Cert. Date 6/2/89 Cert. Flow 17.61 cc/min

VOTS Flow Box 19.7 cc/min Percent d (Cert./VOTS)= -10.61 %

Manometer Reading R 2.6 L 2.6 Total 5.2

Calc. Flow 19.56 cc/min X (Corr. Fact. A)= 19.51 cc/min

Percent d (Calc./VOTS)= -0.97 %

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See comments section

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Volatile Organic Sampler Performance Audit

Date 9/20/89

Site Name Tottenville Site Number 7097-05

Type of Sampler Canister Performed by McDermott/White

VOTS Box No.____ Cert. Date _____ Vac. Test Gauge No. ____

Meas. Mod. No._____Slope_____Inter_____Cert. Date_____

Temp. 74.5 °F 26.2°C Vacuum Relief Valve Vac. _____"Hg.

Atmospheric Pressure 29.96 "Hg. 761 mm Hg.

Correction Factors:

$$A=\left[\frac{298}{T+273}\right]\times\left[\frac{P}{29.92}\right]=$$ _____

T= Actual Temperature °C P= Actual Pressure "Hg

Flow Module Number 1

Module No. _____ Cert. Date _____ Cert. Flow _____cc/min

VOTS Flow Box _____cc/min Percent d (Cert./VOTS)=_____%

Manometer Reading R _____ L _____ Total _____

Calc. Flow _____cc/min X (Corr. Fact. A)= _____cc/min

Percent d (Calc./VOTS)=_____%

Flow Module Number 2

Module No. _____ Cert. Date _____ Cert. Flow _____cc/min

VOTS Flow Box _____cc/min Percent d (Cert./VOTS)=_____%

Manometer Reading R _____ L _____ Total _____

Calc. Flow _____cc/min X (Corr. Fact. A)=_____cc/min

Percent d (Calc./VOTS)=_____%

New York State Department of Environmental Conservation
Division of Air

Bureau of Technical Services
Quality Assurance Section

Volatile Organic Sampler Performance Audit

Date 9/20/89

Site Name PS 26 Site Number 7097-02

Type of Sampler ATD 50 Performed by McDermott/White

VOTS Box No. 2 Cert. Date 4/23/89 Vac. Test Gauge No. A020

Meas. Mod. No. 5021 Slope 0.991 Inter 4.120 Cert. Date 5/15/89

Temp. 82.4 °F 27.9 °C Vacuum Relief Valve Vac. 22.5 "Hg.

Atmospheric Pressure 30.01 "Hg. 762 mm Hg.

Correction Factors:

$$A = \left[\frac{298}{T + 273}\right] \times \left[\frac{P}{29.92}\right] = 0.9933$$

T= Actual Temperature °C P= Actual Pressure "Hg

Flow Module Number 1

Module No. LCC36 Cert. Date 6/2/89 Cert. Flow 8.04 cc/min

VOTS Flow Box 9.3 cc/min Percent d (Cert./VOTS)= -13.6 %

Manometer Reading R 1.1 L 1.1 Total 2.2

Calc. Flow 9.00 cc/min X (Corr. Fact. A)= 8.94 cc/min

Percent d (Calc./VOTS)= -3.9 %

Flow Module Number 2

Module No. L022 Cert. Date 6/2/89 Cert. Flow 16.73 cc/min

VOTS Flow Box 18.3 cc/min Percent d (Cert./VOTS)= -8.6 %

Manometer Reading R 2.2 L 2.2 Total 4.4

Calc. Flow 17.9 cc/min X (Corr. Fact. A)= 17.77 cc/min

Percent d (Calc./VOTS)= -2.9 %

Canister Sampler

New York State Department of Environmental Conservation
Division of Air

See comments section

Bureau of Technical Services
Quality Assurance Section

Volatile Organic Sampler Performance Audit

Date 9/20/89

Site Name PS 26 Site Number 7097-02

Type of Sampler Canister Performed by McDermott/White

VOTS Box No.____ Cert. Date _____ Vac. Test Gauge No. ____

Meas. Mod. No._____Slope_____Inter_____Cert. Date_____

Temp. 82.4 °F 27.9 °C Vacuum Relief Valve Vac. _____"Hg.

Atmospheric Pressure 30.01 "Hg. 762 mm Hg.

Correction Factors:

$$A=\left[\frac{298}{T+273}\right]\times\left[\frac{P}{29.92}\right]=\text{--------}$$

T= Actual Temperature °C P= Actual Pressure "Hg

Flow Module Number 1

Module No. _____ Cert. Date _____ Cert. Flow _____cc/min

VOTS Flow Box _____cc/min Percent d (Cert./VOTS)=_____%

Manometer Reading R _____ L _____ Total _____

Calc. Flow _____cc/min X (Corr. Fact. A)= _____cc/min

Percent d (Calc./VOTS)=_____%

Flow Module Number 2

Module No. _____ Cert. Date _____ Cert. Flow _____cc/min

VOTS Flow Box _____cc/min Percent d (Cert./VOTS)=_____%

Manometer Reading R _____ L _____ Total _____

Calc. Flow _____cc/min X (Corr. Fact. A)=_____cc/min

Percent d (Calc./VOTS)=_____%

NEW YORK STATE DEPARTMENT OF ENVIRONMENTAL CONSERVATION
DIVISION OF AIR

BUREAU OF TECHNICAL SERVICES
QUALITY ASSURANCE SECTION

PARTICULATE SAMPLER FLOW AUDIT

DATE 9/20/09 AUDITOR McDERMOTT/WHITE

SITE NAME P.S. 26 SITE NUMBER 7097-02

TEMP. 82.4 °F 27.9 °C ATM. PRES. 30.01 "HG 762 mm Hg

SAMPLER TYPE: TSP_____ TRACE METALS_____ PM10-WEDDING ✓

COLLOCATED____ LEAD____ PM10- DICOT_____. PM10-ANDERSEN_____

Q(FLOW CFM)= INTERCEPT X (ΔH) SLOPE

AUDIT DEVICE DATA: CHK. INT. USED
30 °F___
40 °F___
60 °F___

ID# _____ PRI. CAL._____ SLOPE_____INT._____

LEFT _____" RIGHT_____" TOTAL_____" Q_____CFM

SAMPLER DATA: CHK. INT. USED
30 °F___
40 °F___
60 °F___

ID# _____ PRI. CAL._____SLOPE_____ INT._____

LEFT _____" RIGHT_____" TOTAL_____" Q_____CFM

PERCENT DIFF. _____%

DICOT ONLY:	ROTOMETER COB	ROTOMETER LPM	ORIFICE DEVICE SN	ORIFICE DEVICE H	ORIFICE DEVICE LPM	PERCENT DIFF.
TOTAL FLOW:	_____	_____	_____	____	_____	_____
COARSE FLOW:	_____	_____	_____	____	_____	_____

AUDITOR CHECKS (INDICATE EQUIPMENT CONDITION):

SHELTER	OK	GASKET	Needs replacement
MOTOR	OK	TIMER	OK
ORIFICE	N/A	ELECTRICAL WIRING	OK
MANOMETER & TUBING	OK	CHECK ORIFICE NO.	N/A

COMMENTS: _____

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NEW YORK STATE DEPARTMENT OF ENVIRONMENTAL CONSERVATION
DIVISION OF AIR

BUREAU OF TECHNICAL SERVICES
QUALITY ASSURANCE SECTION

PARTICULATE SAMPLER FLOW AUDIT

DATE 9/20/89 AUDITOR McDermott/White

SITE NAME P.S. 26 SITE NUMBER 7097-02

TEMP. 82.4 °F 27.9 °C ATM. PRES. 30.01 "HG 762 mm Hg

SAMPLER TYPE: TSP ✓ TRACE METALS ✓ PM10-WEDDING ____

COLLOCATED ____ LEAD ____ PM10- DICOT ____ . PM10-ANDERSEN ____

Q(FLOW CFM) = INTERCEPT X (ΔH) SLOPE

AUDIT DEVICE DATA: CHK. INT. USED
30 °F ___
40 °F ___
60 °F ✓

ID# 1543B PRI. CAL. 9/29/88 SLOPE 0.50 INT. 17.4

LEFT 2.5 " RIGHT 3.0 " TOTAL 5.5 " Q 40.81 CFM

SAMPLER DATA: CHK. INT. USED
30 °F ___
40 °F ___
60 °F ✓

ID# 90092 PRI. CAL. 1/30/89 SLOPE 052 INT. 24.1

LEFT 1.3 " RIGHT 1.4 " TOTAL 2.7 " Q 40.39 CFM

PERCENT DIFF. -1.0 %

DICOT ONLY:

	ROTOMETER COB	ROTOMETER LPM	ORIFICE DEVICE SN	ORIFICE DEVICE H	ORIFICE DEVICE LPM	PERCENT DIFF.
TOTAL FLOW:	____	____	____	____	____	____
COARSE FLOW:	____	____	____	____	____	____

AUDITOR CHECKS (INDICATE EQUIPMENT CONDITION):

SHELTER	OK	GASKET	OK
MOTOR	OK	TIMER	OK
ORIFICE	~~N/A~~ OK	ELECTRICAL WIRING	OK
MANOMETER & TUBING	OK	CHECK ORIFICE NO.	~~N/A~~ OK

COMMENTS: ____

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New York State Department of Environmental Conservation
Division of Air

Bureau of Technical Services
Quality Assurance Section

Volatile Organic Sampler Performance Audit

Date 9/21/89

Site Name Port Richmond Site Number 7097-03

Type of Sampler ATD 50 Performed by McDermott/Lawhite

VOTS Box No. 2 Cert. Date 4/23/89 Vac. Test Gauge No. A020

Meas. Mod. No. 5009 Slope .985 Inter 4.136 Cert. Date 5/15/89

Temp. 71 °F 25.5 °C Vacuum Relief Valve Vac. 22.0 "Hg.

Atmospheric Pressure 29.84 "Hg. 758 mm Hg.

Correction Factors:

$$A = \left[\frac{298}{T + 273}\right] \times \left[\frac{P}{29.92}\right] = 0.9956$$

T= Actual Temperature °C P= Actual Pressure "Hg

Flow Module Number 1

Module No. 20010 Cert. Date ? Cert. Flow 7.8 cc/min

VOTS Flow Box 8.5 cc/min Percent d (Cert./VOTS)= -8.2 %

Manometer Reading R 1.0 L 1.0 Total 2.0

Calc. Flow 8.2 cc/min X (Corr. Fact. A)= 8.15 cc/min

Percent d (Calc./VOTS)= -4.1 %

Flow Module Number 2

Module No. 20040 Cert. Date ? Cert. Flow 7.5 cc/min

VOTS Flow Box 9.1 cc/min Percent d (Cert./VOTS)= -17.6 %

Manometer Reading R 1.1 L 1.1 Total 2.2

Calc. Flow 9.00 cc/min X (Corr. Fact. A)= 8.95 cc/min

Percent d (Calc./VOTS)= -1.6 %

Canister Sampler

New York State Department of Environmental Conservation
Division of Air

See comments section

Bureau of Technical Services
Quality Assurance Section

Volatile Organic Sampler Performance Audit

Date 9/21/89

Site Name Fort Richmond Site Number 7097-03

Type of Sampler Canister Performed by McDermott/White

VOTS Box No.____ Cert. Date _____ Vac. Test Gauge No. ____

Meas. Mod. No._____Slope_____Inter_____Cert. Date_____

Temp. 78 °F 25.5 °C Vacuum Relief Valve Vac. _____"Hg.

Atmospheric Pressure 29.84 "Hg. 758 mm Hg.

Correction Factors:

$$A=\left[\frac{298}{T+273}\right]\times\left[\frac{P}{29.92}\right]=$$ --------

T= Actual Temperature °C P= Actual Pressure "Hg

Flow Module Number 1

Module No. _____ Cert. Date _____ Cert. Flow _____cc/min

VOTS Flow Box _____cc/min Percent d (Cert./VOTS)=_____%

Manometer Reading R _____ L _____ Total _____

Calc. Flow _____cc/min X (Corr. Fact. A)= _____cc/min

Percent d (Calc./VOTS)=_____%

Flow Module Number 2

Module No. _____ Cert. Date _____ Cert. Flow _____cc/min

VOTS Flow Box _____cc/min Percent d (Cert./VOTS)=_____%

Manometer Reading R _____ L _____ Total _____

Calc. Flow _____cc/min X (Corr. Fact. A)=_____cc/min

Percent d (Calc./VOTS)=_____%

NEW YORK STATE DEPARTMENT OF ENVIRONMENTAL CONSERVATION
DIVISION OF AIR

BUREAU OF TECHNICAL SERVICES
QUALITY ASSURANCE SECTION

PARTICULATE SAMPLER FLOW AUDIT

DATE 9/21/89 AUDITOR McDermott/White

SITE NAME Port Richmond SITE NUMBER 7097-03

TEMP. 78 °F 25.5 °C ATM. PRES. 29.84 "HG 758 mm Hg

SAMPLER TYPE: TSP_____ TRACE METALS_____ PM10-WEDDING ✓

COLLOCATED____ LEAD____ PM10- DICOT_____. PM10-ANDERSEN_____

Q(FLOW CFM)= INTERCEPT X (ΔH) SLOPE

AUDIT DEVICE DATA:

CHK. INT. USED
30 °F___
40 °F___
60 °F___

ID# _____ PRI. CAL._____ SLOPE_____INT._____

LEFT _____" RIGHT_____" TOTAL_____" Q_____CFM

SAMPLER DATA:

CHK. INT. USED
30 °F___
40 °F___
60 °F___

ID# _____ PRI. CAL._____SLOPE_____ INT._____

LEFT _____" RIGHT_____" TOTAL_____" Q_____CFM

PERCENT DIFF. _____%

DICOT ONLY:	ROTOMETER COB	ROTOMETER LPM	ORIFICE DEVICE SN	ORIFICE DEVICE H	ORIFICE DEVICE LPM	PERCENT DIFF.
TOTAL FLOW:	_____	_____	_____	____	_____	_____
COARSE FLOW:	_____	_____	_____	____	_____	_____

AUDITOR CHECKS (INDICATE EQUIPMENT CONDITION):

SHELTER	OK	GASKET	OK
MOTOR	OK	TIMER	OK
ORIFICE	N/A	ELECTRICAL WIRING	OK
MANOMETER & TUBING	OK	CHECK ORIFICE NO.	N/A

COMMENTS: _____

NEW YORK STATE DEPARTMENT OF ENVIRONMENTAL CONSERVATION
DIVISION OF AIR

BUREAU OF TECHNICAL SERVICES
QUALITY ASSURANCE SECTION

PARTICULATE SAMPLER FLOW AUDIT

DATE 9/21/89 AUDITOR McDermott/White

SITE NAME Port Richmond SITE NUMBER 7097-03

TEMP. 78 °F 25.5 °C ATM. PRES. 29.84 "HG 758 mm Hg

SAMPLER TYPE: TSP ✓ TRACE METALS ✓ PM10-WEDDING ____

COLLOCATED ____ LEAD ____ PM10- DICOT ____ . PM10-ANDERSEN ____

Q(FLOW CFM)= INTERCEPT X (ΔH) SLOPE

AUDIT DEVICE DATA:

CHK. INT. USED
30 °F ___
40 °F ___
60 °F ✓

ID# 15438 PRI. CAL. 9/29/89 SLOPE 0.50 INT. 17.4

LEFT 2.5 " RIGHT 2.4 " TOTAL 4.9 " Q 38.52 CFM

SAMPLER DATA:

CHK. INT. USED
30 °F ___
40 °F ___
60 °F ✓

ID# 90163 PRI. CAL. 9/15/89 SLOPE 0.52 INT. 25.4

LEFT 1.1 " RIGHT 1.2 " TOTAL 2.3 " Q 39.17 CFM

PERCENT DIFF. 1.7 %

DICOT ONLY:

	ROTOMETER COB	ROTOMETER LPM	ORIFICE DEVICE SN	ORIFICE DEVICE H	ORIFICE DEVICE LPM	PERCENT DIFF.
TOTAL FLOW:	____	____	____	____	____	____
COARSE FLOW:	____	____	____	____	____	____

AUDITOR CHECKS (INDICATE EQUIPMENT CONDITION):

SHELTER	OK	GASKET	OK
MOTOR	OK	TIMER	OK
ORIFICE	OK	ELECTRICAL WIRING	OK
MANOMETER & TUBING	OK	CHECK ORIFICE NO.	OK

COMMENTS: ____

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APPENDIX C

SAMPLE PRECISION ANALYSIS RESULTS

ECISION ANALYSIS
UTRH QUARTER 19 87

ALYTE: DICHLOROMETHANE
MBER OF PAIRS: 15
ERAGE % DIFFERENCE: 5.35
ANDARD DEVIATION OF % DIFFERENCE: 24.38
WER 95% CONFIDENCE LIMIT: -42.44
PER 95% CONFIDENCE LIMIT: 53.15

ALYTE: CHLOROFORM
MBER OF PAIRS: 3
T ENOUGH PAIRS TO EVALUATE STATISTICS

ALYTE: 1,2DICHLOROETHANE
MBER OF PAIRS: 2
T ENOUGH PAIRS TO EVALUATE STATISTICS

ALYTE: 1,1,1TRICHLOROETHANE
MBER OF PAIRS: 22
ERAGE % DIFFERENCE: 1.95
ANDARD DEVIATION OF % DIFFERENCE: 16.70
WER 95% CONFIDENCE LIMIT: -30.78
PER 95% CONFIDENCE LIMIT: 34.68

ALYTE: BENZENE
MBER OF PAIRS: 23
ERAGE % DIFFERENCE: 3.36
ANDARD DEVIATION OF % DIFFERENCE: 27.49
WER 95% CONFIDENCE LIMIT: -50.52
PER 95% CONFIDENCE LIMIT: 57.23

ALYTE: CARBON TETRACHLORIDE
MBER OF PAIRS: 0
T ENOUGH PAIRS TO EVALUATE STATISTICS

ALYTE: TRICHLOROETHYLENE
MBER OF PAIRS: 4
ERAGE % DIFFERENCE: 8.28
ANDARD DEVIATION OF % DIFFERENCE: 15.30
WER 95% CONFIDENCE LIMIT: -21.70
PER 95% CONFIDENCE LIMIT: 38.27

ALYTE: 1,1,2TRICHLOROETHANE
MBER OF PAIRS: 0
T ENOUGH PAIRS TO EVALUATE STATISTICS

ALYTE: TOLUENE
MBER OF PAIRS: 21

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'ALYTE: DICHLOROMETHANE
'MBER OF PAIRS: 21
'ERAGE % DIFFERENCE: 0.73
'ANDARD DEVIATION OF % DIFFERENCE: 22.99
'WER 95% CONFIDENCE LIMIT: -44.34
'PER 95% CONFIDENCE LIMIT: 45.80

IALYTE: CHLOROFORM
IMBER OF PAIRS: 13
'ERAGE % DIFFERENCE: -6.72
:ANDARD DEVIATION OF % DIFFERENCE: 26.18
)WER 95% CONFIDENCE LIMIT: -58.03
'PER 95% CONFIDENCE LIMIT: 44.59

IALYTE: 1,2DICHLOROETHANE
)MBER OF PAIRS: 3
)T ENOUGH PAIRS TO EVALUATE STATISTICS

)ALYTE: 1,1,1TRICHLOROETHANE
)MBER OF PAIRS: 25
/ERAGE % DIFFERENCE: 1.02
TANDARD DEVIATION OF % DIFFERENCE: 20.70
)WER 95% CONFIDENCE LIMIT: -39.56
PPER 95% CONFIDENCE LIMIT: 41.60

NALYTE: BENZENE
UMBER OF PAIRS: 25
VERAGE % DIFFERENCE: -0.18
TANDARD DEVIATION OF % DIFFERENCE: 23.83
OWER 95% CONFIDENCE LIMIT: -46.89
PPER 95% CONFIDENCE LIMIT: 46.52

NALYTE: CARBON TETRACHLORIDE
UMBER OF PAIRS: 2
'OT ENOUGH PAIRS TO EVALUATE STATISTICS

)NALYTE: TRICHLOROETHYLENE
IUMBER OF PAIRS: 19
,VERAGE % DIFFERENCE: -1.52
:TANDARD DEVIATION OF % DIFFERENCE: 22.11
.OWER 95% CONFIDENCE LIMIT: -44.85
IPPER 95% CONFIDENCE LIMIT: 41.81

)NALYTE: 1,1,2TRICHLOROETHANE
)UMBER OF PAIRS: 0
)OT ENOUGH PAIRS TO EVALUATE STATISTICS

NALYTE: 1,4DICHLOROBENZENE
UMBER OF PAIRS: 0
OT ENOUGH PAIRS TO EVALUATE STATISTICS

NALYTE: 1,2DICHLOROBENZENE
UMBER OF PAIRS: 2
OT ENOUGH PAIRS TO EVALUATE STATISTICS

ALYTE: 1,1,2TRICHLOROETHANE
MBER OF PAIRS: 0
T ENOUGH PAIRS TO EVALUATE STATISTICS

ALYTE: TOLUENE
MBER OF PAIRS: 34
ERAGE % DIFFERENCE: -1.96
ANDARD DEVIATION OF % DIFFERENCE: 11.74
WER 95% CONFIDENCE LIMIT: -24.96
PER 95% CONFIDENCE LIMIT: 21.05

ALYTE: TETRACHLOROEHTYLENE
MBER OF PAIRS: 28
ERAGE % DIFFERENCE: 1.53
ANDARD DEVIATION OF % DIFFERENCE: 5.58
WER 95% CONFIDENCE LIMIT: -9.41
PER 95% CONFIDENCE LIMIT: 12.46

ALYTE: CHLOROBENZENE
MBER OF PAIRS: 5
ERAGE % DIFFERENCE: -10.16
ANDARD DEVIATION OF % DIFFERENCE: 14.09
WER 95% CONFIDENCE LIMIT: -37.78
PER 95% CONFIDENCE LIMIT: 17.46

ALYTE: ETHYLBENZENE
MBER OF PAIRS: 28
ERAGE % DIFFERENCE: -0.22
ANDARD DEVIATION OF % DIFFERENCE: 10.48
WER 95% CONFIDENCE LIMIT: -20.76
PER 95% CONFIDENCE LIMIT: 20.33

ALYTE: M,P-XYLENE
MBER OF PAIRS: 36
ERAGE % DIFFERENCE: 4.90
ANDARD DEVIATION OF % DIFFERENCE: 30.89
WER 95% CONFIDENCE LIMIT: -55.66
PER 95% CONFIDENCE LIMIT: 65.45

ALYTE: O-XYLENE
MBER OF PAIRS: 33
ERAGE % DIFFERENCE: 0.97
ANDARD DEVIATION OF % DIFFERENCE: 5.95
WER 95% CONFIDENCE LIMIT: -10.69
PER 95% CONFIDENCE LIMIT: 12.64

ALYTE: 1,3DICHLOROBENZENE
MBER OF PAIRS: 19

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IRD QUARTER 19 88

ALYTE: DICHLOROMETHANE
MBER OF PAIRS: 12
ERAGE % DIFFERENCE: 1.30
ANDARD DEVIATION OF % DIFFERENCE: 13.47
WER 95% CONFIDENCE LIMIT: -25.09
PER 95% CONFIDENCE LIMIT: 27.69

ALYTE: CHLOROFORM
MBER OF PAIRS: 13
ERAGE % DIFFERENCE: -4.13
ANDARD DEVIATION OF % DIFFERENCE: 12.89
WER 95% CONFIDENCE LIMIT: -29.40
PER 95% CONFIDENCE LIMIT: 21.14

ALYTE: 1,2DICHLOROETHANE
MBER OF PAIRS: 2
T ENOUGH PAIRS TO EVALUATE STATISTICS

ALYTE: 1,1,1TRICHLOROETHANE
MBER OF PAIRS: 15
ERAGE % DIFFERENCE: 5.04
ANDARD DEVIATION OF % DIFFERENCE: 16.40
WER 95% CONFIDENCE LIMIT: -27.10
PER 95% CONFIDENCE LIMIT: 37.18

ALYTE: BENZENE
MBER OF PAIRS: 12
ERAGE % DIFFERENCE: -2.78
ANDARD DEVIATION OF % DIFFERENCE: 15.00
WER 95% CONFIDENCE LIMIT: -32.17
PER 95% CONFIDENCE LIMIT: 26.61

ALYTE: CARBON TETRACHLORIDE
MBER OF PAIRS: 3
T ENOUGH PAIRS TO EVALUATE STATISTICS

ALYTE: TRICHLOROETHYLENE
MBER OF PAIRS: 8
ERAGE % DIFFERENCE: -3.57
ANDARD DEVIATION OF % DIFFERENCE: 10.10
WER 95% CONFIDENCE LIMIT: -23.37
PER 95% CONFIDENCE LIMIT: 16.23

ALYTE: 1,1,2TRICHLOROETHANE
MBER OF PAIRS: 0
T ENOUGH PAIRS TO EVALUATE STATISTICS

LYTE: 1,2DICHLOROBENZENE
BER OF PAIRS: 1
ENOUGH PAIRS TO EVALUATE STATISTICS

ALYTE: TOLUENE
MBER OF PAIRS: 9
ERAGE % DIFFERENCE: 11.10
ANDARD DEVIATION OF % DIFFERENCE: 19.96
WER 95% CONFIDENCE LIMIT: -28.01
PER 95% CONFIDENCE LIMIT: 50.22

ALYTE: TETRACHLOROEHTYLENE
MBER OF PAIRS: 9
ERAGE % DIFFERENCE: 6.80
ANDARD DEVIATION OF % DIFFERENCE: 10.52
WER 95% CONFIDENCE LIMIT: -13.81
PER 95% CONFIDENCE LIMIT: 27.42

ALYTE: CHLOROBENZENE
MBER OF PAIRS: 0
T ENOUGH PAIRS TO EVALUATE STATISTICS

ALYTE: ETHYLBENZENE
MBER OF PAIRS: 9
ERAGE % DIFFERENCE: 10.62
ANDARD DEVIATION OF % DIFFERENCE: 19.27
WER 95% CONFIDENCE LIMIT: -27.14
PER 95% CONFIDENCE LIMIT: 48.38

ALYTE: M,P-XYLENE
MBER OF PAIRS: 9
ERAGE % DIFFERENCE: 7.42
ANDARD DEVIATION OF % DIFFERENCE: 14.46
WER 95% CONFIDENCE LIMIT: -20.92
PER 95% CONFIDENCE LIMIT: 35.76

ALYTE: O-XYLENE
MBER OF PAIRS: 9
ERAGE % DIFFERENCE: 8.38
ANDARD DEVIATION OF % DIFFERENCE: 17.09
WER 95% CONFIDENCE LIMIT: -25.13
PER 95% CONFIDENCE LIMIT: 41.88

ALYTE: 1,3DICHLOROBENZENE
MBER OF PAIRS: 0
T ENOUGH PAIRS TO EVALUATE STATISTICS

ALYTE: 1,4DICHLOROBENZENE
MBER OF PAIRS: 5
ERAGE % DIFFERENCE: 22.53
ANDARD DEVIATION OF % DIFFERENCE: 16.43
WER 95% CONFIDENCE LIMIT: -9.67
PER 95% CONFIDENCE LIMIT: 54.73

ECISION ANALYSIS ENVIROCHEM SYSTEM
JTRH QUARTER 19 88

ALYTE: DICHLOROMETHANE
MBER OF PAIRS: 29
ERAGE % DIFFERENCE: -2.36
ANDARD DEVIATION OF % DIFFERENCE: 17.18
WER 95% CONFIDENCE LIMIT: -36.03
PER 95% CONFIDENCE LIMIT: 31.31

ALYTE: CHLOROFORM
MBER OF PAIRS: 17
ERAGE % DIFFERENCE: -8.24
ANDARD DEVIATION OF % DIFFERENCE: 29.84
WER 95% CONFIDENCE LIMIT: -66.73
PER 95% CONFIDENCE LIMIT: 50.25

ALYTE: 1,2DICHLOROETHANE
MBER OF PAIRS: 1
T ENOUGH PAIRS TO EVALUATE STATISTICS

ALYTE: 1,1,1TRICHLOROETHANE
MBER OF PAIRS: 34
ERAGE % DIFFERENCE: -4.90
ANDARD DEVIATION OF % DIFFERENCE: 23.75
WER 95% CONFIDENCE LIMIT: -51.46
PER 95% CONFIDENCE LIMIT: 41.65

ALYTE: BENZENE
MBER OF PAIRS: 34
ERAGE % DIFFERENCE: 10.21
ANDARD DEVIATION OF % DIFFERENCE: 29.30
WER 95% CONFIDENCE LIMIT: -47.23
PER 95% CONFIDENCE LIMIT: 67.64

ALYTE: CARBON TETRACHLORIDE
MBER OF PAIRS: 7
ERAGE % DIFFERENCE: -6.51
ANDARD DEVIATION OF % DIFFERENCE: 8.34
WER 95% CONFIDENCE LIMIT: -22.86
PER 95% CONFIDENCE LIMIT: 9.84

ALYTE: TRICHLOROETHYLENE
MBER OF PAIRS: 22
ERAGE % DIFFERENCE: -7.58
ANDARD DEVIATION OF % DIFFERENCE: 23.64
WER 95% CONFIDENCE LIMIT: -53.92
PER 95% CONFIDENCE LIMIT: 38.76

ALYTE: 1,1,2TRICHLOROETHANE

ER 95% CONFIDENCE LIMIT: 34.16

LYTE: 1,4DICHLOROBENZENE
BER OF PAIRS: 0
T ENOUGH PAIRS TO EVALUATE STATISTICS

LYTE: 1,2DICHLOROBENZENE
BER OF PAIRS: 2
T ENOUGH PAIRS TO EVALUATE STATISTICS

MBER OF PAIRS: 0
T ENOUGH PAIRS TO EVALUATE STATISTICS

ALYTE: TOLUENE
MBER OF PAIRS: 52
ERAGE % DIFFERENCE: 1.90
ANDARD DEVIATION OF % DIFFERENCE: 23.82
WER 95% CONFIDENCE LIMIT: -44.79
PER 95% CONFIDENCE LIMIT: 48.58

ALYTE: TETRACHLOROEHTYLENE
MBER OF PAIRS: 46
ERAGE % DIFFERENCE: 1.06
ANDARD DEVIATION OF % DIFFERENCE: 13.57
WER 95% CONFIDENCE LIMIT: -25.54
PER 95% CONFIDENCE LIMIT: 27.65

ALYTE: CHLOROBENZENE
MBER OF PAIRS: 4
ERAGE % DIFFERENCE: 0.00
ANDARD DEVIATION OF % DIFFERENCE: 0.00
WER 95% CONFIDENCE LIMIT: 0.00
PER 95% CONFIDENCE LIMIT: 0.00

ALYTE: ETHYLBENZENE
MBER OF PAIRS: 50
ERAGE % DIFFERENCE: -0.66
ANDARD DEVIATION OF % DIFFERENCE: 11.70
WER 95% CONFIDENCE LIMIT: -23.60
PER 95% CONFIDENCE LIMIT: 22.28

ALYTE: M,P-XYLENE
MBER OF PAIRS: 52
ERAGE % DIFFERENCE: 2.35
ANDARD DEVIATION OF % DIFFERENCE: 16.04
WER 95% CONFIDENCE LIMIT: -29.09
PER 95% CONFIDENCE LIMIT: 33.79

ALYTE: O-XYLENE
MBER OF PAIRS: 37
ERAGE % DIFFERENCE: 0.29
ANDARD DEVIATION OF % DIFFERENCE: 10.80
WER 95% CONFIDENCE LIMIT: -20.87
PER 95% CONFIDENCE LIMIT: 21.45

ALYTE: 1,3DICHLOROBENZENE
MBER OF PAIRS: 15
ERAGE % DIFFERENCE: -3.79
ANDARD DEVIATION OF % DIFFERENCE: 22.28
WER 95% CONFIDENCE LIMIT: -47.46

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ST QUARTER 19 89

ALYTE: DICHLOROMETHANE
MBER OF PAIRS: 48
ERAGE % DIFFERENCE: -4.04
ANDARD DEVIATION OF % DIFFERENCE: 25.86
WER 95% CONFIDENCE LIMIT: -54.74
PER 95% CONFIDENCE LIMIT: 46.65

ALYTE: CHLOROFORM
MBER OF PAIRS: 34
ERAGE % DIFFERENCE: -1.54
ANDARD DEVIATION OF % DIFFERENCE: 20.27
WER 95% CONFIDENCE LIMIT: -41.26
PER 95% CONFIDENCE LIMIT: 38.18

ALYTE: 1,2DICHLOROETHANE
MBER OF PAIRS: 2
T ENOUGH PAIRS TO EVALUATE STATISTICS

ALYTE: 1,1,1TRICHLOROETHANE
MBER OF PAIRS: 74
ERAGE % DIFFERENCE: -2.44
ANDARD DEVIATION OF % DIFFERENCE: 33.12
WER 95% CONFIDENCE LIMIT: -67.35
PER 95% CONFIDENCE LIMIT: 62.47

ALYTE: BENZENE
MBER OF PAIRS: 72
ERAGE % DIFFERENCE: -4.23
ANDARD DEVIATION OF % DIFFERENCE: 21.38
WER 95% CONFIDENCE LIMIT: -46.13
PER 95% CONFIDENCE LIMIT: 37.67

ALYTE: CARBON TETRACHLORIDE
MBER OF PAIRS: 33
ERAGE % DIFFERENCE: -0.19
ANDARD DEVIATION OF % DIFFERENCE: 17.99
WER 95% CONFIDENCE LIMIT: -35.46
PER 95% CONFIDENCE LIMIT: 35.07

ALYTE: TRICHLOROETHYLENE
MBER OF PAIRS: 62
ERAGE % DIFFERENCE: -5.54
ANDARD DEVIATION OF % DIFFERENCE: 21.96
WER 95% CONFIDENCE LIMIT: -48.58
PER 95% CONFIDENCE LIMIT: 37.50

ALYTE: 1,1,2TRICHLOROETHANE

R 95% CONFIDENCE LIMIT: 59.72

YTE: 1,4DICHLOROBENZENE
ER OF PAIRS: 7
AGE % DIFFERENCE: 5.51
DARD DEVIATION OF % DIFFERENCE: 18.31
R 95% CONFIDENCE LIMIT: -30.39
R 95% CONFIDENCE LIMIT: 41.40

YTE: 1,2DICHLOROBENZENE
ER OF PAIRS: 5
.GE % DIFFERENCE: -0.82
DARD DEVIATION OF % DIFFERENCE: 1.83
R 95% CONFIDENCE LIMIT: -4.39
R 95% CONFIDENCE LIMIT: 2.76

ALYTE: 1,1,2TRICHLOROETHANE
MBER OF PAIRS: 0
T ENOUGH PAIRS TO EVALUATE STATISTICS

ALYTE: TOLUENE
MBER OF PAIRS: 73
ERAGE % DIFFERENCE: 5.77
ANDARD DEVIATION OF % DIFFERENCE: 33.34
WER 95% CONFIDENCE LIMIT: -59.59
PER 95% CONFIDENCE LIMIT: 71.12

ALYTE: TETRACHLOROEHTYLENE
MBER OF PAIRS: 62
ERAGE % DIFFERENCE: 0.93
ANDARD DEVIATION OF % DIFFERENCE: 14.17
WER 95% CONFIDENCE LIMIT: -26.84
PER 95% CONFIDENCE LIMIT: 28.70

ALYTE: CHLOROBENZENE
MBER OF PAIRS: 8
ERAGE % DIFFERENCE: 3.07
ANDARD DEVIATION OF % DIFFERENCE: 21.37
WER 95% CONFIDENCE LIMIT: -38.83
PER 95% CONFIDENCE LIMIT: 44.96

ALYTE: ETHYLBENZENE
MBER OF PAIRS: 71
ERAGE % DIFFERENCE: 5.72
ANDARD DEVIATION OF % DIFFERENCE: 23.93
WER 95% CONFIDENCE LIMIT: -41.18
PER 95% CONFIDENCE LIMIT: 52.62

ALYTE: M,P-XYLENE
MBER OF PAIRS: 73
ERAGE % DIFFERENCE: -1.62
ANDARD DEVIATION OF % DIFFERENCE: 21.23
WER 95% CONFIDENCE LIMIT: -43.23
PER 95% CONFIDENCE LIMIT: 40.00

ALYTE: O-XYLENE
MBER OF PAIRS: 73
ERAGE % DIFFERENCE: 2.15
ANDARD DEVIATION OF % DIFFERENCE: 13.39
WER 95% CONFIDENCE LIMIT: -24.10
PER 95% CONFIDENCE LIMIT: 28.40

ALYTE: 1,3DICHLOROBENZENE
MBER OF PAIRS: 39

:CISION ANALYSIS ATD
RD QUARTER 19 89

LYTE: DICHLOROMETHANE
BER OF PAIRS: 38
RAGE % DIFFERENCE: 0.41
NDARD DEVIATION OF % DIFFERENCE: 21.74
ER 95% CONFIDENCE LIMIT: -42.19
ER 95% CONFIDENCE LIMIT: 43.01

LYTE: CHLOROFORM
BER OF PAIRS: 41
RAGE % DIFFERENCE: 6.86
NDARD DEVIATION OF % DIFFERENCE: 20.57
ER 95% CONFIDENCE LIMIT: -33.47
ER 95% CONFIDENCE LIMIT: 47.18

LYTE: 1,2DICHLOROETHANE
BER OF PAIRS: 3
ENOUGH PAIRS TO EVALUATE STATISTICS

LYTE: 1,1,1TRICHLOROETHANE
BER OF PAIRS: 82
RAGE % DIFFERENCE: 4.60
NDARD DEVIATION OF % DIFFERENCE: 13.52
ER 95% CONFIDENCE LIMIT: -21.91
ER 95% CONFIDENCE LIMIT: 31.10

LYTE: BENZENE
BER OF PAIRS: 82
RAGE % DIFFERENCE: -0.46
NDARD DEVIATION OF % DIFFERENCE: 26.24
ER 95% CONFIDENCE LIMIT: -51.89
ER 95% CONFIDENCE LIMIT: 50.97

LYTE: CARBON TETRACHLORIDE
BER OF PAIRS: 35
RAGE % DIFFERENCE: 0.55
NDARD DEVIATION OF % DIFFERENCE: 17.97
ER 95% CONFIDENCE LIMIT: -34.68
ER 95% CONFIDENCE LIMIT: 35.77

LYTE: TRICHLOROETHYLENE
BER OF PAIRS: 78
RAGE % DIFFERENCE: 0.82
NDARD DEVIATION OF % DIFFERENCE: 33.67
ER 95% CONFIDENCE LIMIT: -65.18
ER 95% CONFIDENCE LIMIT: 66.81

LYTE: 1,1,2TRICHLOROETHANE

ER 95% CONFIDENCE LIMIT: 41.14

LYTE: 1,4DICHLOROBENZENE
BER OF PAIRS: 0
ENOUGH PAIRS TO EVALUATE STATISTICS

LYTE: 1,2DICHLOROBENZENE
BER OF PAIRS: 6
RAGE % DIFFERENCE: -3.06
NDARD DEVIATION OF % DIFFERENCE: 16.63
ER 95% CONFIDENCE LIMIT: -35.65
ER 95% CONFIDENCE LIMIT: 29.53

APPENDIX D

Sample Tabulation and Calculations for Distributed Volumes

Sample Distributed Volume Calculations
4th Quarter 1987 Benzene Envirochem

High	Low	Diff. High-Low	High	Low	Diff. High-Low	High	Low	Diff. High-Low	
1.16	3.07	1.91	0.66	1.50	0.84	1.30	2.83	1.53	n = 32
1.79	1.09	-0.70	1.02	1.35	0.33	1.90	2.61	0.71	Mean = 0.78
1.20	2.15	0.95	0.66	1.46	0.80	1.37	2.16	0.79	Std. dev. = 0.67
1.11	1.45	0.34	1.90	1.96	0.06	0.86	1.14	0.28	
0.98	1.44	0.46	0.64	1.06	0.42	1.85	2.26	0.41	
0.91	1.83	0.92	0.96	1.47	0.51	3.91	5.10	1.19	
1.22	1.03	-0.19	0.83	1.52	0.69	1.16	2.24	1.08	
1.04	1.77	0.73	1.06	1.96	0.90	0.90	1.62	0.72	
1.07	1.58	0.51	1.09	2.49	1.40				
0.81	1.05	0.24	1.00	2.96	1.96				
0.55	1.27	0.96	1.22	4.22	3.00				
0.87	1.27	0.40	1.01	1.78	0.77				

n = no. of sample
μ = mean
σ = std. deviation

$$t\ (\text{calc}) = \text{mean/std dev.} = \frac{0.78}{0.67} = 1.16$$

$$t\ (\text{table})_{n-1,0.05} = t\ (\text{table})_{31,0.05} = 2.042$$

$t > 0.05$, No

95% Confidence Interval

$$\bar{\mu} - \frac{(\sigma)(t_{n-1,\sigma})}{\sqrt{n}} \leq \mu \leq \bar{\mu} + \frac{(\sigma)(t_{n-1,\sigma})}{\sqrt{n}}$$

$$0.78 - \frac{(0.67)(2.042)}{\sqrt{32}} \leq \mu \leq 0.78 + \frac{(0.67)(2.042)}{\sqrt{32}}$$

$$0.78 - 0.24 \leq \mu \leq 0.78 + 0.24$$

$$0.54 \leq \mu \leq 1.02$$

9. CSI VOCS QUALITY ASSURANCE REPORT

Staten Island/New Jersey Urban Air Toxics Assessment Program

QUALITY ASSURANCE REPORT

for

CENTER FOR ENVIRONMENTAL SCIENCE
THE COLLEGE OF STATEN ISLAND
THE CITY UNIVERSITY OF NEW YORK
50 Bay Street
Staten Island, New York 10301

John R. Oppenheimer, Ph. D.
Director

for the period of
July 1987 to September 1989

Submitted to
U. S. Environmental Protection Agency, Region II
Quality Assurance Branch
Edison, New Jersey

Draft 20 July 1990
Final 10 November 1990

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QUALITY ASSURANCE REPORT FOR THE

COLLEGE OF STATEN ISLAND

INTRODUCTORY COMMENTS

Since the guidance document for preparation of this report included extensive analysis by use of the paired-sample t-test, it is important to understand its interpretation and its limitations.

The underlying hypothesis of the paired-sample t-test is that "mean1 - mean2 = 0". If the two means are different, the t- value will be large and this will be indicated by a "Yes" in the tables as formatted by the instructions given. However, the paired-sample t-test is extremely sensitive to consistent difference or bias. This will be seen in the figures in the various chapters that show data in relation to time on the X-axis, where data for a quarter are significantly different if one set of values (such as tenax values) are consistently above or consistently below the other (such as canister values). Data for a quarter are <u>not</u> different, if during the quarter, the values of one set vary above and below those of the other set. Thus a quarter's data, where one set of values is consistently 0.001 ppb above the other set, will be different even though the means are very similar, whereas if the data pair differ by 10 ppb, but shift back and forth, in terms of which one is higher, the means will be statistically the same, even though quantitatively different. Because of this, little comment will be made about the results of the paired-sample t test, but the results will be given in the tables. More emphasis will be given to whether there is visual agreement between the two sets of measurements as seen in these figures and as looked at below.

The real question in terms of quality assurance is whether the two sets of data are in agreement with one another. This will be addressed below in terms of regression analysis and figures showing a scattergram with either one or more regression lines or a 45 degree lines plotted through the data points. If the regression line runs through the origin and its slope (beta) is equal to 1, then the values of the two data sets are equal; if the slope is < 1, the values on the x-axis are larger than those on the y-axis and vice versa. This analysis will be done for the entire time span of the study, as opposed to quarterly. Quarterly analysis would be more helpful, but the time required to carry this out is currently not available because of deadlines which need to be met. A further step, which may be attempted at a later date would be to plot a 95% confidence belt parallel to the regression line. For now note will simply be made of data points which are clear outliers.

Means will be reported with plus or minus ($\pm$) one standard deviation (S). Unless otherwise indicated, the 95% confidence

limits reported in the tables are the limits for the mean difference, i.e., that there is a 95% probability that the true mean lies within the stated limits.

ACKNOWLEDGMENTS. I wish to thank Qingmei Zha, Susmita Biswas, and Donna Daly for their assistance in the preparation of this report, and particularly Wa Hing Chan for doing much of the data processing and sniffing out of errors. An earlier draft of this document was reviewed by Avraham Teitz of the Monitoring Management Branch, USEPA, and by Clifford P. Weisel, formerly at CSI, but now at UMDNJ. Their comments were greatly appreciated.

CHAPTER I

BLANKS

PROJECT SUMMARY

The following method was used in this report to assess contamination of blanks and consequently of samples. The mean monthly concentration for an individual compound was determined for ambient samples. If the blank trap had a concentration which was greater than 30% of the monthly mean level, the blank was considered contaminated. However, what was recorded when this was done was the number of samples that were consequently determined to represent contamination values. Thus in the following table the information is reported as % of samples contaminated (# of contaminated sample traps/# of sample traps processed).

However, for duration of the study, the working procedure was to take a rolling average of the blank value and ambient values over 5 days; if the mean blank value exceeded 50% of the of the mean ambient value, contamination was assumed to have occurred (C. P. Weisel, pers. com., 5 November 1990). Five day mean blank values were subtracted from ambient sample values.

The average percent contamination of samples (or blanks) was 3.4% over the whole study for all compounds (Table 1.1). Six compounds had more than 5% of their samples and blanks contaminated: methylene chloride (8%), chloroform (6%), ethylbenzene (7%), m/p xylene (7%), o-xylene (6%), and p-dichlorobenzene (17%). Without these six compounds, the % contamination was 1.5 $\pm$ 1.3. The average % contamination for the eight compounds that were usually at or below their MDLs was 4.3 $\pm$ 5.4, with methylene chloride and p-dichlorobenzene having the highest contamination levels of more than 7%.

Over the quarters, the % contamination ranged from 1.4 in 4th quarter '88 to 7.5 in 2nd quarter '89. The high levels of contamination in 2nd quarter '89 were probably due to the extended use of the tenax absorbent in the traps. This was corrected in July when newly cleaned tenax was loaded into the traps. The high levels of contamination with 1,1-dichloroethane and trichloroethene in 3rd quarter '89 were probably due to the new tenax, as the levels of these compounds dropped after the traps had been used a couple of times.

The lowest number of compounds contaminating the traps was in 3rd quarter '89, after new tenax had been cleaned by soxlet extraction with cyclohexane, acetone, and methanol each for 48 hours. The stainless steel traps were also cleaned with full strength chromic sulfuric acid; this had not been done before.

CHAPTER II

DUPLICATE SAMPLES

Duplicate sampling for low and high flows started at the end of July 1988 and continued until the end of the study. This was done by using a second set of flowmeters in the field (C-low and D-high). The second of each pair of duplicates (C & D) was processed along with the first (A & B) in terms of GC/MS analysis, but the data did not go through blank value substraction and temperature-pressure correction. Consequently only data from the A and B samples were included in the data set sent to the USEPA and thus appear in the report this is appended to. During preparation of this report, it seemed unnecessary to do this for the second of each pair, since data uncorrected for blank values and uncorrected for temperature-pressure already existed for both, and the differences between them would have remained the same. However, some compounds appear to have values above their MDLs in this presentation because subtraction of blank values was not done, when in fact they were at or below their MDL (see below).

Duplicate low flows (A & C) and duplicate high flows (B & D) were similar over the entire study, i.e., no paired-t values were significant (Tables 2.1 and 2.2).

There were minor differences in precision by compound in terms of % difference, and this tended to be greatest for those compounds which were measurable only on a third or fewer of the days. **Methylene chloride** was the most volatile of the compounds looked at and is held weakly by tenax, the absorbent; it had measurable levels on 22.1% of the days, and for low flow had a 6.19% difference. **1,1-Dichloroethane** was the second most volatile compound looked at and had measurable levels on only 6.6% of the days; it had the greatest % difference for low (46%) and high (269%) flows; however, the sample sizes were small and the concentrations were only 4 times greater than the MDL. **Chlorobenzene** was seen on only 31% of the days, but more frequently on the high flow traps and only slightly above its MDL; thus at low flow it had a 12.8% difference. **Bromoform** occurred on only 6 of 180 days (3.3%) at only one of the three sites. **Meta-dichlorobenzene** occurred above its MDL on 2 of 180 days (1.1%) and only at one of the three sites; thus what is measured here is variation in trap contamination levels. The same is true for **o- and p-dichlorobenzene**, though they were seen above their MDLs on 28 (15.6%) and 23 (12.3%) of 180 sampling days, respectively. **1,1,2-trichloroethane** was never seen above its MDL in the 180 days for which measurement was attempted; thus the low % difference for both low and high flow rates.

Hereafter attention will only be given to those compounds which were measurable, and the low and high flows will be treated separately.

Low flows

With the above eight compounds omitted, the average % difference was 2.97 ± 2.69. There were only four compounds with % differences above 5%: hexane, 1,2-dichloroethane, trichloroethene and o-xylene (Table 2.1). With the exclusion of these four compounds, the average % difference for the remaining nine compounds was 1.45 ± 1.31.

In terms of regression analysis, the variation in the second of each pair (C) of the low flow set was almost totally accounted for by variation in the value of the first of each pair (A) (mean R^2 = 0.93 ± 0.05; Table 2.3). The regression coefficient fell below 0.92 for only three of the measurable compounds: trichloroethene, styrene and o-xylene. For many compounds (benzene, trichloroethene, toluene, tetrachloroethene, ethylbenzene, styrene, and o-xylene) the deviation from the regression line was greatest at the higher concentration values.

High flows

With the compounds which occurred infrequently at measurable levels omitted, the average % difference was 3.15 ± 2.64 (N = 13). Again there were only four compounds with a % difference above 5%, but they were different compounds from those at low flow: chloroform, 1,1,1-trichloroethane, 1,2-dichloroethane, and benzene (Table 2.2). Without these four compounds the average % difference for the remaining nine compounds was 1.68 ± 1.18.

In terms of regression analysis, the variation in the second of each pair (D) of the high flow set was again almost totally accounted for by variation in the value of the first of each pair (B) (mean R^2 = 0.93 ± 0.07; Table 2.3). The regression coefficient fell below 0.93 for only two of the thirteen measurable compounds: chloroform and 1,1,1-trichloroethane. For most compounds (hexane, 1,2-dichloroethane, carbon tetrachloride, toluene, and ethylbenzene) the deviation from the regression line was greatest at the higher concentration values.

The most deviant data pair for most compounds (Figures 2.1 to 2.26) had a low value for the first high flow sample (it occurred at Eltingville on 7 September 1989)(see benzene, mid-point); it represents a total desorption failure on the cold trap as confirmed by both internal standards. The first internal standard for sample B was only 38% of the first internal standard for sample D, and the 2nd internal standard for sample B was only 29% of that for sample D. This represents the type of sample that should be and normally is removed from the overall data set for failing QA standards. (See quarterly treatment below.)

DUPLICATE SAMPLES

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Attention will be focused on the 13 compounds which had measurable concentrations most of the time (Tables 2.4 to 2.13). At low flow five had significantly different values in one quarter each: one in 4th quarter '88 (1,2-Dichloroethane) and four in 3rd quarter '89 (ethylbenzene, m/p-xylene, o-xylene and styrene). At high flow, two measurable compounds had significantly different values in one quarter each: styrene in 4th quarter '88, and chloroform in 1st quarter '89. With the small number of samples per compound per quarter, it is to be expected that occasionally the values for the first of each pair would be slightly, but consistently above or below that of the other.

The average % difference for the thirteen frequently measurable compounds varied across quarters: low flow varied from 5.69% in 3rd quarter '88 to 47.73% in 2nd quarter '89, and high flow varied from 4.48% in 1st quarter '89 to 8.30% in 3rd quarter '89 (Tables 2.4 to 2.13).

Mean average % difference by quarter for 13 frequent compounds

	1988		1989			
	3rd Q	4th Q	1st Q	2nd Q	3rd Q	Mean
Low flow						
Mean	5.69	9.45	8.09	47.73	11.98	2.97
S	4.76	6.43	3.09	39.47	9.96	2.69
High flow						
Mean	6.85	4.70	4.48	7.04	8.30	3.15
S	5.94	3.19	3.37	7.10	13.02	2.64

These variations were related to variation in desorption from the cold trap as seen in the quantification of the internal standards that were placed on the traps in the laboratory just prior to analysis on the GC/MS. For instance, in 2nd quarter '89 there were 4 days with duplicate low flow values; two of these days had already been eliminated from the data base because, in the "A" sample of each A:C pair, there was a failure of desorption of the second internal standard relative to the first (2nd/1st should have been 46%, but it was < 27%). The remaining two daily pairs were entered into the database, but one of them had a failure in the opposite direction, i.e., the second internal standard was larger than it should have been (66% instead of 46%); however, both failed for another reason. The "C" sample, which was not included in the data base, had an 83% greater desorption of the 1st internal standard as compared to that in the "A" sample

of each pair, and the 2nd internal standard had more than a 54% greater desorption. During 2nd quarter '89 there were similar problems with the high flow duplicates, but to a lesser extent. Third quarter '89 had similar desorption problems. Consequently, the data for these two quarters show larger average differences and larger standard deviations than in previous quarters (see table on page 10).

CHAPTER III

DISTRIBUTED VOLUMES

Of the 21 compounds where quantification was attempted, only thirteen occurred above their minimum detection limits (MDLs) most or all of the time, and will be called "measurable." Eight other compounds were measurable at one or more sites on less than 31% of the days: chlorobenzene (30.5%), methylene chloride (22.1%), o-dichlorobenzene (15.6%), p-dichlorobenzene (12.8%), 1,1-dichloroethane (6.6%), bromoform (3.3%), and m-dichlorobenzene (1.1%); and one compound was apparently always below its MDL - 1,1,2-trichloroethane (0%). These compounds will be called "unmeasurable."

Analysis based on paired-sample t tests

Measurable compounds. Since data for the thirteen measurable compounds were obtained for different numbers of quarters, some for 9 and others for only 8 or 7, the easiest way to summarize is on the basis of the % of "compound-quarters" where the paired-sample t tests were insignificant (see table on next page). There were 107 compound-quarters, and in 38% of these the low and high flows were the same. There appeared to be a strong seasonal effect, with 62% agreement (no significant difference) in 2nd quarter and only 22% agreement in 3rd quarter.

Percent agreement by "compound-quarters"

Year	1st Q	2nd Q	3rd Q	4th Q	Combined
1987	-	-	17	56	40
1988	38	54	8	15	29
1989	38	69	38	-	49
Combined	38	62	22	32	38

In terms of measurable compounds, the best agreement was for tetrachloroethene (75% of the quarters), and for m/p xylene (71% of the quarters) (Table 3.1). The worst agreement was for 1,1,1-trichloroethane (0%) and carbon tetrachloride (0%). However, a

further pattern can be seen. The first seven measurable compounds had a higher percentage difference (mean = 78.9% ± 15.6) than did the last six (mean = 41.2 ± 12.2)(two sample t test = 4.78, df = 11, $p < 0.001$). [The compounds are listed in terms of increasing retention time, or decreasing volatility.]

Unmeasurable compounds. The eight unmeasurable compounds had significantly different means for low and high flow rates 72% of the time (Table 3.1). Since 70 to 100% of the values for these compounds were recorded at one-half their minimum detection limits, and the low flow mdl was twice the high flow mdl, this is reasonable. In those few quarters where the all the values for a compound were one-half the MDLs, the standard error of the average difference was zero and the t-value was insignificant.

Table 3.1. Comparison of low and high flow values on basis of paired sample t test

Measurable most of time			Measurable < 33% of time		
Compound	Quarters #	% diff*	Compound	Quarters #	% diff*
Hexane	9	78	Methylene chloride	8	75
Chloroform	9	67	1,1-Dichloroethane	7	71
111-Trichloroethane	9	100	112-Trichloroethane	4	75
12-Dichloroethane	9	78	Chlorobenzene	9	100
Benzene	9	67	Bromoform	4	25
Carbon tetrachloride	9	100	m Dichlorobenzene	3	33
Trichloroethene	8	62	o Dichlorobenzene	4	100
Toluene	8	50	p Dichlorobenzene	4	100
Tetrachloroethene	8	25			
Ethylbenzene	7	57			
m/p Xylene	7	29			
Styrene	7	43			
o Xylene	7	43			

Mean % quarters different	61.5	72.4
S	23.8	29.5

* difference based paired t test

Analysis based on means and ratios of low to high flow rates

The ratios of low flow values to high flow values show that there were three groups of compounds (Table 3.2): A1) measurable compounds with large standard deviations (0.11 to 0.32), A2) those with small standard deviations (0.04 to 0.07), and B) unmeasurable compounds with low flow values almost twice as high as high flow values (1.73 to 1.98).

A1) These seven measurable compounds were the more volatile ones with retention times ranging from 9.7 minutes for hexane to 13.9 minutes for trichloroethene. Their low flow values on the average were 16% higher than the high flow values (Table 3.2). This difference had a strong seasonal component, which was what was shown in the paired-sample t test above. Low and high flow values were similar in the cold months of October to March (4th and 1st quarters), but in the warm months of April to June (2nd quarter) low flow values were 15 to 19% higher than were high flow values, and in the hot months of July to September (3rd quarter) were 42% higher (Figure 3.1). [Temperature data currently are only partially complete for the months of April 1988 to March 1989 (see Fig.3.1), and are not yet available for prior or following time periods.] Consequently, low flow values would appear to be the most accurate measure of ambient air concentrations in the second and third quarters of each year for these compounds, or they could be used for all quarters.

Comments on compounds. 1,1,1-Trichloroethane (RT = 11.6 minutes) and carbon tetrachloride (RT = 12.4 minutes) both showed breakthrough in all quarters, though it was greatest in the hot quarter as above. For both compounds the deviation between low and high values increased with increasing ambient concentrations. Low flow values are probably the best estimates of ambient concentrations of these two compounds.

1,2-Dichloroethane (RT = 11.7 minutes) followed the above group pattern, but its mean ratio across all quarters was close to 1.0 because it also had quarters where the average low flow value fell 14 to 25% below that of the high flow value (1Q88, 4Q88, and 1Q89). This occurred because there was a large number of days in these quarters when the ambient air concentration occurred close to the low flow MDL (0.02) and was reported by convention to be at 1/2 the MDL (0.01). However, the high flow values for these days ranged from 0.005 (1/2 the MDL) to to as high as 0.05. All other values above the MDL showed good agreement in these quarters.

Benzene had a retention time of 12.3 minutes, which would suggest a potential problem with breakthrough, but its mean ratio across quarters was close to 1.0. Because it is an aromatic compound, it is held more strongly by Tenax GC (C. P. Weisel,

pers. com. 11 September 1990). It none-the-less did have low flow values 17 to 21% higher than high flow values in the hot quarter. The reason for the mean ratio close to 1.0 was because in one quarter (1Q89) almost all high flow values were higher than low flow at all concentrations (see trichloroethene below).

Trichloroethene (RT = 13.9 minutes) showed what appears to be breakthrough in 3rd quarter 1988, but not in 3rd quarter 1989. In addition, in 4th quarter 1987 its low flow values were lower than the high flow values. In both quarters where there was a difference between low and high flow values, it occurred at all concentrations; this does not appear to fit the breakthrough pattern. Without these two deviant quarters, trichloroethene would have been included with the group A2 compounds.

A2) These six measurable compounds had lower volatility with retention times of 17.0 minutes for toluene to 23.2 minutes for ortho xylene. Their low and high flow values were essentially the same in all seasons (Table 3.2, Fig. 3.1).

Comments on compounds: Toluene, the xylenes, and ethylbenzene are aromatics and should be held tightly to Tenax GC (C. P. Weisel, pers. com. 11 September 1990). However, in 3rd and 4th quarters of 1988 toluene's low flow values were 9-10% higher than the high flow values. This difference increased progressively with increasing ambient concentrations. In other quarters the low and high flow values were the same. The xylenes (ortho and meta/para) and ethylbenzene, showed the same phenomenon in these two quarters.

Tetrachloroethene values were almost the same for low and high flow, with low flow slightly lower. For most quarters, values under 1 ppb were the same for low and high flow, whereas values above 1 ppb tended to be higher for high flow. An exception was first quarter 1989 when intermediate values (0.4 to 0.8 ppb) were 30 to 50% higher for high flow, and high values (> 1.4 ppb) were up to 21% lower for high flow; this might be related to the shape of the calibration curves.

B) The unmeasurable compounds showed little variation with season (Table 3.2, Fig. 3.1), but did have low flow values almost twice as high as high flow values because most of the concentrations were below the MDLs, and low flow MDLs were twice as high as high flow MDLs.

Some of this problem is eliminated for these compounds if only those days, which had low and high flow values at or above their MDLs, are used (Table 3.3). As indicated at the start, 1,1,2-trichloroethane (RT = 18.0 min) had ambient concentrations which apparently were below the low and high flow MDLs.

The difference was eliminated for five compounds: 1,1-dichloroethane, chlorobenzene, bromoform, and m- and o-dichlorobenzene. 1,1-Dichloroethane had good agreement, with half of the values at the low flow MDL (0.02). The slight difference for bromoform is an artifact; on 5 of the 6 days the high flow value was above the high flow MDL of 0.01, but below the low flow MDL of 0.02. Thus the low flow values were reported at one-half their MDL or at 0.01. This resulted in the high flow values being higher by 10%.

However, the high flow means were still lower for two compounds: methylene chloride (RT = 7.5 min), and p-dichlorobenzene (RT = 28.2 min). For methylene chloride, this probably represents breakthrough, as this was the most volatile of the compounds and Tenax was known to hold this compound poorly. One suspects that the low flow values for methylene chloride also suffered from breakthrough, but to a lesser extent. (This is confirmed by comparison with canister values for methylene chloride - Chapter IV.) It is not clear why p- dichlorobenzene should show a difference between low and high flow values, as it had next to the lowest volatility of the compounds looked at and it is an aromatic, which should be held tightly by Tenax (C. P. Weisel, pers. com, 1 Sept. 1990). O- dichlorobenzene, which follows it by 0.1 minutes showed similar values for low and high flows based on the variability in the data.

Table 3.3. Means of lo-hi values > MDL for 7 compounds seen on < 31% of days

Compound (N=)		Flow Low	High	Diff	% Diff	t-value
Methylene chloride 58*	Mean	1.40	0.77	0.63	81.8	6.43
	S					
1,1-Dichloroethane 10	Mean	0.032	0.032	0.000	0.0	0.00
	S	1.874	1.874	0.005		
Chlorobenzene 112	Mean	0.036	0.032	0.004	13.1	
	S	0.033	0.026			
Bromoform 6	Mean	0.022	0.024	0.0025	10.3	
	S	0.029	0.025			
m-Dichlorobenzene 1	Mean	0.100	0.140	0.040	28.6	
	S					
o-Dichlorobenzene 20	Mean	0.037	0.031	0.006	19.4	
	S	0.032	0.033			
p-Dichlorobenzene 14	Mean	0.592	0.292	0.300	102.7	
	S	0.183	0.086			

* values through December 1988

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The quarterly tables are attached (Tables 3.4 to 3.12). See above for comments.

CHAPTER IV

TENAX vs. CANISTER

Matching tenax and canister data were obtained for eleven compounds during the study. Eight compounds had regression coefficients (R^2) that ranged from 0.1 (N = 43) to 0.9 (N = 26) with all data points included, and 0.4 (N = 40) to 0.9 (N = 3) with outlier points removed from the regression analysis. The slopes of the regression lines for these eight compounds differed significantly from zero at $p < 0.001$ (Table 4.1; Figs. 4.1 to 4.10). Two compounds, carbon tetrachloride and trichloroethene, had five or fewer data points, with all values at or near the canister MDL; the variability of the complete set of tenax values for these two compounds had no relation to variability in canister values ($R^2 = 0.07$ and 0.27, $p > 0.05$). One compound, methylene chloride (dichloromethane), had fifty-one pairs, but only in seven of these was the tenax value above its MDL.

One additional compound, chloroform, was reported for canisters only in the last quarter. This was because the canister MDL was higher than the ambient chloroform concentrations as seen with tenax throughout the study.

Data on the paired t test are given in Table 4.2 for the study as a whole. The individual compounds will be treated below in the order of their retention times, and consequently their volatility.

Methylene chloride (RT = 7.5 min): Fifty-one data pairs were obtained, with a mean canister value of 1.51 $\pm$ 3.37 and a mean tenax value of 0.26 $\pm$ 0.17. However, only eight of the tenax values were above the tenax MDL. Since all values at or below the MDL are reported as one-half the MDL there was an even larger difference due to this. It was known at the start of the study that methylene chloride would be poorly held by tenax, and that the values reported for our three sites would be well below the actual ambient concentrations.

The $R^2 = 0.01$ as most tenax values were reported at the MDL and thus did not track the canister values.

Hexane (RT = 9.7 min): Forty-five data pairs were obtained. The mean canister value was 9.61 ppb $\pm$ 25.35 and the mean tenax value was 0.86 ppb $\pm$ 0.62. However, during 1st quarter '88 the canister values ranged from 8 to 120 ppb (tenax values for 1st quarter ranged from 0.4 to 3.5 ppb), whereas canister values for the rest of the study ranged from 0.1 to 1.9 ppb (Fig. 4.1a). It is assumed that these high canister values were due to an error in laboratory analysis and/or reporting.

Without 1st quarter '88 (37 data pairs), the mean canister value was 0.87 ppb ± 0.71 and the mean tenax value was 0.81 ± 0.50. However, removal of one additional data pair (C:T = 4.84:0.49) seems warranted, as it appears to be a typographical error, i.e., it probably should have been 0.48 (the canister values preceding and following ranged from 0.4 to 0.9 with a mean of 0.67, N = 5). Thus with 36 data pairs (Fig. 4.1b), the means for canisters and tenax were the same (C: mean = 0.820 ± 0.32; T: mean = 0.827 ± 0.50); the paired-t for these 36 data pairs was 0.8023, which was not significant.

If 1st quarter 88 is used (45 data pairs), R^2 = 0.11, and the slope was significant at p = 0.05. Without 1st quarter '88 and the additional data set (36 data pairs), R^2 = 0.52, and the slope (1.12) was significant at 0.001. Figures including and excluding 1st quarter '88 follow. It may be that removal of one or more additional data points, such as 0.6:1.7 would further improve the relationship, but at this point there is no information available for excluding them as above.

Chloroform (RT = 10.5 min): This compound was reported for canisters only in 3rd quarter 1989, when tenax showed the concentrations as below its MDL of 0.015 ppb. During the earlier quarters tenax showed chloroform at values up to 0.1 ppb, but this was below the canister MDL of 0.2 ppb. The five canister values in 3rd quarter 89 ranged between 0.3 and 0.5 ppb (mean = 0.39 ± 0.09), whereas tenax showed it below its MDL of 0.015 ppb; this may indicate a problem with tenax data during 3rd quarter '89. (With tenax, breakthrough occurred, at least at high flow, in 2nd and 3rd quarters of both years - Chapter III.)

1,1,1-Trichloroethane (RT = 11.6 min): Canister values on the average were higher than tenax values (C: mean = 0.764 ± 0.511; T: mean = 0.417 ± 0.314). However, one canister value for 3rd quarter '89 was higher than any of the other values reported in the study (C = 2.7, T = 0.23) and probably was a typographical error (Fig. 4.2). Without this data pair, the mean difference decreased from 152% to 130% (C: mean = 0.719 ± 0.390; T: mean = 0.421 ± 0.359), but was still significantly different at $p < 0.001$. (With tenax, breakthrough caused high flow values to be 24% lower than low flow values - Chapter III. Thus if low flow values were used instead of means, the difference with canisters would be reduced to about 6%, which could be due to error or to slight breakthrough at low flow.)

There were 43 data pairs with an R^2 = 0.308 and the slope of the regression line was significantly greater than zero. With the above data pair omitted (N = 42), R^2 = 0.57, and the slope of the regression line increased from 0.34 to 0.57 and more

closely fit the rest of the data points. The low slope indicates that canister values were higher than tenax (see above comments).

Benzene (RT = 12.3 min): Canister values were the same as tenax values (C: mean = 1.288 ± 0.592; T: mean = 1.246 ± 0.879; paired-t = 0.555, $p > 0.5$).

With 65 data pairs $R^2 = 0.529$ and the slope of the regression line = 1.08, which was significantly greater than 0 at $p < 0.001$ (Fig. 4.3).

Carbon tetrachloride (RT = 12.35 min): There were only five data pairs* for this compound, but the means for canister and tenax were similar (C: mean = 0.28 ± 0.09; T: mean = 0.29 ± 0.23; paired-t = 0.144, $p > 0.5$). *[The low number of data pairs was due to the high canister MDL of 0.2 ppb relative to the concentration in the air, which was mostly below 0.16 ppb, except for 1st quarter '89 when it rose to 0.25 ppb.]

Three of the canister values were at or below the canister MDL of 0.20 (tenax MDL = 0.03). On three days the tenax values were above the canister MDL, but the canister saw only two of them. Consequently, $R^2 = 0.07$ and the slope of the regression line did not differ from zero (Fig. 4.4).

Trichloroethene (RT = 13.9): There were only four data pairs for this compound, but the means were the same (C: mean = 0.29 ± 0.05; T: mean = 0.23 ± 0.20; paired-t = 0.736, $p > 0.5$).

The MDL for canisters was 0.2 ppb and for tenax was 0.01. On two of the days tenax and canister values were above the canister MDL. On one day the tenax value was 0.1 and the canister value was reported as the MDL. On the fourth day the tenax value was 0.01 (the tenax MDL) and the canister was 0.3, 0.1 above its MDL. Because of this $R^2 = 0.266$ and the slope was not significantly different from zero. With this one data pair omitted (N = 3), $R^2 = 0.994$ and the slope was significantly greater than zero (Fig. 4.5). A larger sample size would be needed to determine the actual level of agreement between canister and tenax.

Toluene (RT = 16.97): There were 66 data pairs and the means were similar (C: mean = 3.18 ± 2.05; T: mean = 3.51 ± 1.97; paired-t = 1.517, $p > 0.1$).

For 66 data pairs $R^2 = 0.36$ with a slope of 0.58, which was significantly different from zero at $p < 0.001$. One data pair was

an extreme outlier and pulled the regression line down (C:T - 13.0:3.6); with this data pair removed, R^2 = 0.59 with a regression line that went more closely through the data points (Fig. 4.6). Removal of two other outliers (1.926:7.47 and 6.42:1.64, N = 63) further improves the regression coefficient (R^2 = 0.78, df = 61, slope = 1.08, t = 14.9, p < 0.001).

Tetrachloroethene (RT = 18.84): There were 26 data pairs and the means were similar (C: mean = 0.71 ± 0.64; T: mean = 0.76 ± 0.76; paired-t = 1.092, p > 0.2). The R^2 = 0.91 with a slope of 1.134, which was significant at p < 0.001 (Figure 4.7).

Ethylbenzene (RT = 21.06): There were 41 data pairs and the means were significantly different because most tenax values were close to but slightly higher than canister values (C: mean = 0.49 ± 0.23; T: mean = 0.69 ± 0.51; paired-t = 2.950, p < 0.01).

For 41 data pairs, R^2 = 0.242 with a slope of 1.09, which differed significantly from zero at p < 0.002. However, the first two data pairs, which were taken in January 1988, have problems; the canister values were higher (above 1.0 ppb) than for the rest of the study (less than 0.9 ppb), and the first tenax value was more than twice as high (3.16 ppb) as the highest tenax values reported later (1.46 ppb)(this appears to be a problem with the calibration curve, which was changed at the beginning of February). With these two data pairs removed (N = 39), R^2 = 0.44 with a slope of 1.24 (Fig. 4.8).

Meta/para-Xylene (RT = 21.4): There were 61 data pairs and the means were significantly different because most tenax values were close to but slightly higher than canister values (C: mean = 1.42 ± 0.85; T: mean = 1.78 ± 1.06; paired-t = 3.034, p < 0.005).

For 61 data pairs R^2 = 0.28 with a slope of 0.66, which differed significantly from zero at p < 0.001. However, one data pair from 1st quarter 88 is a clear outlier (C:T - 5.3:0.89), with the canister value twice as high as the highest canister value reported later in the study and more than five times higher than other canister values for 1st quarter '88. With this data pair removed, R^2 = 0.68 with a slope of 1.39, which differed significantly from zero at p < 0.001 (Fig. 4.9).

Ortho-Xylene (RT = 22.42): There were 48 data pairs and the means were significantly different because the canister values were almost twice as high as the tenax values (C: mean = 0.62 ± 0.28; T: mean = 0.39 ± 0.24; paired-t = 9.029, p < 0.001).

For 48 data pairs R^2 = 0.613 with a slope of 0.680, which differed significantly from zero at p < 0.001 (Fig. 4.10). During

1st quarter '88 and the first sampling day for 2nd quarter '88 the canister MDL was 0.5 ppb and two of the sampling days had values at the MDL; tenax values on these two days and on two others were below this level. Removal of the two days with canister values at the MDL gave an R^2 = 0.624, and consequently only a slight improvement. The canister MDL for most of the study thereafter was 0.2 ppb and tenax was 0.075 ppb.

To summarize, four compounds had similar means on canister and tenax (mean tenax/mean canister: hexane - 1.01, benzene - 0.97, toluene - 1.10, and tetrachloroethene - 1.07), and had regression coefficients ranging from 0.52 to 0.91 with slopes of 1.08 to 1.13. Two compounds (mt/mc: 1,1,1-trichloroethane - 0.59, o-xylene - 0.63) had higher values on canisters, but the canister and tenax values changed together over time (R^2's = 0.57 and 0.61) with slopes less than one (0.57 and 0.68). Two compounds (mt/mc: ethylbenzene - 1.41, and m/p xylene - 1.25) had lower values on canisters, but again the values changed together over time (R^2's = 0.44 and 0.68) with slopes greater than one (1.24 and 1.39).

So to answer the questions posed, the difference between canister and tenax values did differ among the various compounds. The reasons for this may be clear to others. However, one would need to look at whether there was any consistency in these differences across the different laboratories; if so, the problem would be with the canister values. If other labs showed no difference, then the problem(s) is (are) here. If other labs show a different pattern of differences, perhaps a meeting would be in order.

To partially assess laboratory bias, we can look at the results of the proficiency tests done for the New York State Department of Health's Environmental Laboratory Approval Program (Table 4.3). These tests were done once every 6 months; a mean was taken for each concentration of each compound for all the values reported by the participating laboratories and the 95 and 99% confidence limits were calculated (for additional details see Chapter VI). Table 4.3 shows our reported values divided by the mean value. For sixteen compounds with two to eight values each, our mean was 1.095 $\pm$ 0.099, or 9.5% greater. For comparison with the eight compounds where we had canister and tenax matches as reported above see the list below:

	Tenax/Canister	ELAP	(N=)
Hexane	1.01	-	(0)
Benzene	0.97	1.04	(2)
Toluene	1.10	1.14	(6)
Tetrachloroethene	1.07	1.12	(4)
Ethylbenzene	1.41	1.11	(6)
m/p-Xylene	1.25	1.12	(8)
1,1,1-Trichloroethane	0.59	-	(0)
o-Xylene	0.63	-	(0).

Based on this comparison, it would seem that for the first four compounds where canister and tenax values agree, that the values are close to the ambient concentrations. For ethylbenzene and m/p-xylene, it would appear that the canister values were low. For 1,1,1-trichloroethane and o-xylene, additional points of reference are needed, perhaps from the other two laboratories. However, for 1,1,1-trichloroethane breakthrough did occur on high flow tenax (Chapter III).

In conclusion, at least for the compounds where there were matches for tenax and canisters, it seems that the tenax values were close to ambient, with a tendency to be 10% above.

TENAX vs. CANISTER

QUARTERLY REPORTS

Quarterly data are presented for the paired t test (Tables 4.4 to 4.10). Data are also provided for each compound across quarters (Figures 4.11 to 4.20).

Methylene chloride

Tenax and canister values differed in all quarters, except the 1st quarter of 1988, when the tenax MDL was highest. The large percent difference in 1st quarter was due to a single canister value of 24, which was way above all other canister values.

Hexane

With the exclusion of 1st quarter '88, canister and tenax values were the same in all quarters (Figs. 4.11a and b)

Chloroform

The only comparison data for this compound occurred in 3rd quarter '89. See above for details.

1,1,1-Trichloroethane

The canister and tenax values were similar in the 1st, and 4th quarters of 1988, and 2nd quarter of '89, but differed in 2nd and 3rd quarter '88 and in the 3rd quarter of '89 Fig. 4.12). In 1st quarter '88 all four canister values were at its MDL (0.5 ppb), whereas one tenax value was above this and three were below. In the remaining quarters the canister MDL was 0.2 ppb and all canister values were reported above this level. The greatest differences occurred in 2nd and 3rd quarters of '88 and 3rd quarter of '89 when ambient concentrations were at their lowest, close to the canister MDL. (It was also in these three quarters when the highest levels of breakthrough were seen in the high flow tenax traps - Chapter III).

Benzene

The canister and tenax values were similar in the first three quarters of '88, but differed thereafter (Fig. 4.13). Tenax values were usually slightly higher than canister in 4th quarter '88 and 1st quarter '89; however, canister values were higher in 2nd and 3rd quarters of '89. This same phenomenon occurred in latter half of 2nd quarter and early half of 3rd quarter '88. The

second and third quarters of both years had the lowest ambient concentrations of benzene as seen on tenax.

Carbon tetrachloride

Ambient concentrations tended to be below the canister MDL, so little comparison can be made (Fig. 4.14).

Trichloroethene

Same as for carbon tetrachloride (Fig. 4.15).

Toluene

Tenax was slightly but consistently higher in 1st and 3rd quarters of '88 and thus significantly different from canister, but the means were similar (C = 2.82 and 3.62, T = 3.39 and 4.86). In all other quarters, tenax values tended to be slightly higher, but there were some days when canister values were the higher ones, and thus there were no significant differences (Fig. 4.16).

Tetrachloroethene

During 1st quarter '88 the canister MDL was 0.5 ppb and most values fell below that, but one data pair was obtained with similar values (C = 0.66, T = 0.62). Thereafter the canister MDL was 0.2 ppb and there was good agreement in all quarters, except 3rd quarter '88 when tenax values were slightly, but consistently higher (C = 0.59, T = 0.82) (Fig. 4.17).

Ethylbenzene

The agreement in 1st quarter '88 should be ignored (see above). In the 2nd to 4th quarters of '88 tenax values were slightly and consistently higher than canister. In the three quarters of '89 there was no significant difference between tenax and canister; however, the tenax mean was higher in 1st quarter (0.53:0.73), the same in 2nd quarter (0.29:0.30), and lower in 3rd quarter (0.40:0.27) (Fig. 4.18).

m/p-Xylene

Tenax and canister means were the same in 1st and 2nd quarters of '88, but differed thereafter (Fig. 4.19). In 3rd quarter '88 through 2nd quarter '89 tenax values were slightly

CHAPTER V

MINIMUM ANALYTICAL DETECTION

(Written in consultation with Dr. Weisel.)

Minimum detection limits (MDL) were determined for twenty-one compounds. These compounds fell into two groups: those that had consistent measurable blank values and those that did not.

There were seven compounds with consistently measurable blank values: methylene chloride, benzene, toluene, ethylbenzene, m/p-xylene, o-xylene, and p-dichlorobenzene. Since the blank values for these compounds were much higher than the instrument detection limits, "method detection limits" were determined. This was done by taking twice the average oven blank value for the compound for the month.

For the remaining 14 compounds, which did not have blank problems, a series of standard samples with different concentrations was analyzed. A concentration that could just be detected by the instrument was the suspected limit of detection. Five replicates were run close to the suspected limit of detection, and the standard deviation was calculated. The standard deviation was multiplied by the Student t-value at the 99% confidence limit at 4 degrees of freedom to determine the MDL.

The values of the detection limits were determined relative to the instrumental response in nL of gas . These MDL values were divided by the nominal volumes expected for 8 and 16 cc/min for a 24 hour period, or 12 and 24 liters, to convert to ppb.

The following tables show the monthly "ppb" values for each compound as determined above (Table 5.1a and b), and "ng" values as converted from the ppb table for this report (Table 5.2a and b).

For most compounds, precision seemed

higher than canister, and in 3rd quarter '89 they were slightly lower. The biggest differences occurred in 3rd quarter '88 and 2nd quarter '89.

o-Xylene

Tenax and canister values differed in all quarters, with canister values slightly higher on all but two days in 1st quarter '89 (Fig. 4.20).

To summarize the quarterly data, the absolute percent difference between the tenax and canister values were calculated. This was done by dividing the compound's mean tenax value for the quarter by the mean canister value (Table 4.11), and then the absolute difference was determined (Table 4.12). The largest differences occurred in 2nd and 3rd quarters of '88, and in the 3rd quarter of '89, periods of high temperatures. Differences tended to be greatest for benzene when its ambient concentrations were low, but this did not hold for toluene, tetrachloroethene, or ethylbenzene. Further tenax values were lower in these quarters for benzene, but higher for toluene and ethylbenzene. In 3rd quarter '88 tenax values tended to be higher than canister, whereas in 3rd quarter '89 they tended to be lower.

Table 1.1. Percentage of samples judged contaminated based on blank level

Quarter	3Q87	4Q87	1Q88	2Q88	3Q88	4Q88	1Q89	2Q89	3Q89	Mean	S
# handling blanks run	87	86	97	85	68	74	87	7	20		
Methylene chloride		13.3	11.6	11.6	9.2	5.0	2.9	10.7	0.0	8.0	4.5
1,1-Dichloroethane			0.0	0.0	0.0	0.0	0.0	0.0	19.5	2.8	6.8
Hexane	2.7	0.2	0.8	1.4	0.2	0.0	0.4	0.0	0.0	0.6	0.9
Chloroform	22.0	0.4	0.8	0.0	2.4	0.0	2.2	14.3	7.3	5.5	7.3
1,1,1-Trichloroethane	8.2	1.0	0.0	0.2	0.9	0.2	0.7	0.0	0.0	1.3	2.5
1,2-Dichloroethane	0.0	0.4	0.0	0.2	0.2	0.0	0.0	0.0	0.0	0.1	0.1
Benzene	14.3	0.0	0.0	0.6	0.0	0.0	0.2	3.6	0.0	2.1	4.5
Carbon tetrachloride	0.5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.2
Trichloroethene	0.0	0.0	0.0	0.2	0.0	0.0	0.2	3.6	12.2	1.8	3.8
Toluene	10.4	0.0	0.0	1.6	0.7	0.2	0.7	10.7	0.0	2.7	4.2
1,1,2-Trichloroethane						0.0	0.0	0.0	0.0	0.0	0.0
Tetrachloroethene	0.0	0.0	0.0	1.0	0.2	0.0	0.0	0.0	0.0	0.1	0.3
Chlorobenzene	4.9	0.0	0.3	1.8	0.5	0.0	0.0	10.7	1.2	2.2	3.4
Ethylbenzene			3.8	6.2	12.0	1.1	2.0	25.0	0.0	7.1	8.2
m/p-Xylene			5.9	9.6	12.7	1.1	2.4	14.3	0.0	6.6	5.3
Bromoform					0.0	0.0	0.0	0.0	0.0	0.0	0.0
Styrene			0.0	2.2	0.9	0.6	2.4	10.7	2.4	2.8	3.4
o-Xylene	19.2	1.8	3.8	6.8	8.7	0.9	1.5	10.7	0.0	5.9	5.9
m-Dichlorobenzene						1.7	0.4	7.1	0.0	2.3	2.9
p-Dichlorobenzene						15.4	17.5	25.0	8.5	16.6	5.9
o-Dichlorobenzene						3.2	0.4	10.7	0.0	3.6	4.3
Mean	7.5	1.4	1.7	2.7	2.9	1.4	1.6	7.5	2.4	3.4	2.2
S	7.7	3.6	3.1	3.6	4.4	3.4	3.7	7.7	5.1	3.8	

Table 2.1

Organization: College of Staten Island Sorbent: Tenax

SUMMARY REPORT (JULY 88 TO SEPTEMBER 89)

DUPLICATE LOW FLOW

Compound name	# Pairs run	Average Low Flow 1	Average Low Flow 2	Average Diff. (ppb)	% Diff. (ppb)	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	T Test	T > 0.0 (Y/N)
Methylene Chloride	25	0.211	0.224	-0.013	6.19%	0.016	(-0.047 - 0.021)	0.802	N
1,1 Dichloroethane	6	0.146	0.079	0.067	45.91%	0.036	(-0.026 - 0.159)	1.857	N
Hexane	27	0.987	0.937	0.050	5.09%	0.035	(-0.022 - 0.122)	1.435	N
Chloroform	20	0.050	0.050	.000	0.05%	0.002	(-0.005 - 0.005)	0.010	N
1,1,1 Trichloroethane	27	0.464	0.453	0.011	2.33%	0.014	(-0.018 - 0.040)	0.768	N
1,2 Dichloroethane	15	0.069	0.065	0.004	5.91%	0.004	(-0.005 - 0.013)	0.999	N
Benzene	27	1.354	1.345	0.009	0.64%	0.052	(-0.099 - 0.116)	0.167	N
Carbon Tetrachloride	27	0.109	0.107	0.002	1.75%	0.004	(-0.006 - 0.010)	0.503	N
Trichloroethene	26	0.069	0.065	0.004	6.42%	0.004	(-0.005 - 0.014)	0.991	N
Toluene	27	3.777	3.727	0.050	1.33%	0.111	(-0.179 - 0.279)	0.450	N
1,1,2 Trichloroethane	2	0.048	0.048	.000	0.76%	0.001	(-0.009 - 0.008)	0.532	N
Tetrachloroethene	26	0.524	0.548	-0.023	4.45%	0.036	(-0.098 - 0.052)	0.641	N
Chlorobenzene	7	0.012	0.014	-0.002	12.82%	0.002	(-0.007 - 0.004)	0.757	N
Ethyl Benzene	26	0.641	0.637	0.004	0.58%	0.024	(-0.045 - 0.052)	0.158	N
m/p Xylene	26	2.307	2.282	0.026	1.12%	0.076	(-0.131 - 0.182)	0.339	N
Bromoform	0								
Styrene	26	0.086	0.085	0.001	0.78%	0.005	(-0.010 - 0.011)	0.132	N
o Xylene	26	0.459	0.421	0.037	8.16%	0.025	(-0.014 - 0.089)	1.492	N
m Dichlorobenzene	0								
p Dichlorobenzene	22	0.176	0.172	0.004	2.36%	0.025	(-0.047 - 0.055)	0.166	N
o Dichlorobenzene	3	0.023	0.021	0.002	6.81%	0.004	(-0.008 - 0.011)	0.385	N

Table 2.2

Organization: College of Staten Island Sorbent: Tenax

SUMMARY REPORT (JULY 88 TO SEPTEMBER 89)

DUPLICATE HIGH FLOW

compound name	# Pairs run	Average High Flow 1	Average High Flow 2	Average Diff. (ppb)	% Diff.	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	T Test	T > 0.0 (Y/N)
Methylene Chloride	24	0.123	0.120	0.002	1.75%	0.009	(-0.017 - 0.021)	0.230	N
1,1 Dichloroethane	4	0.012	0.046	-0.033	269.38%	0.021	(-0.084 - 0.017)	1.613	N
Hexane	25	0.663	0.675	-0.012	1.81%	0.022	(-0.058 - 0.034)	0.539	N
Chloroform	23	0.038	0.036	0.002	6.09%	0.003	(-0.003 - 0.008)	0.841	N
1,1,1 Trichloroethane	25	0.354	0.322	0.032	8.98%	0.027	(-0.024 - 0.087)	1.173	N
1,2 Dichloroethane	24	0.050	0.048	0.003	5.04%	0.002	(-0.002 - 0.007)	1.205	N
Benzene	25	1.190	1.122	0.068	5.72%	0.038	(-0.009 - 0.146)	1.801	N
Carbon Tetrachloride	25	0.095	0.094	.000	0.47%	0.003	(-0.007 - 0.008)	0.130	N
Trichloroethene	25	0.064	0.062	0.002	3.84%	0.004	(-0.005 - 0.010)	0.646	N
Toluene	25	3.098	3.128	-0.030	0.95%	0.074	(-0.182 - 0.122)	0.398	N
1,1,2 Trichloroethane	2	0.047	0.047	.000	0.63%	0.001	(-0.003 - 0.003)	0.303	N
Tetrachloroethene	24	0.532	0.515	0.017	3.16%	0.017	(-0.018 - 0.052)	0.992	N
Chlorobenzene	13	0.009	0.009	.000	2.32%	0.001	(-0.001 - 0.002)	0.318	N
Ethyl Benzene	24	0.546	0.555	-0.010	1.76%	0.019	(-0.049 - 0.030)	0.497	N
m/p Xylene	24	1.909	1.924	-0.015	0.81%	0.059	(-0.136 - 0.105)	0.265	N
Bromoform	0								
Styrene	23	0.079	0.081	-0.001	1.87%	0.003	(-0.007 - 0.004)	0.588	N
o Xylene	24	0.383	0.385	-0.002	0.47%	0.011	(-0.024 - 0.020)	0.166	N
m Dichlorobenzene	0								
p Dichlorobenzene	20	0.121	0.140	-0.018	15.19%	0.020	(-0.059 - 0.022)	0.929	N
o Dichlorobenzene	9	0.010	0.012	-0.002	16.68%	0.001	(-0.004 - .000)	1.806	N

Table 2.3

Organization: College of Staten Island **Sorbent: Tenax**

Regression summary for July 1988 to September 1989

Compound name	Low Flow Duplicate Samples				High Flow Duplicate Samples			
	df	Regression coefficient	Slope	t-Test on slope	Significance	df	Regression coefficient	Slope	t-Test on slope	Significance
Methylene Chloride	23	0.81	0.84	9.82	P < 0.05	22	0.82	0.84	9.96	P < 0.05
1,1 Dichloroethane	4	0.83	0.42	4.35	P < 0.05	2	0.67	11.00	1.99	P > 0.05
Hexane	25	0.97	0.89	29.21	P < 0.05	23	0.96	0.99	24.83	P < 0.05
Chloroform	18	0.93	0.84	15.25	P < 0.05	21	0.81	0.71	9.51	P < 0.05
1,1,1 Trichloroethane	25	0.95	0.99	21.14	P < 0.05	23	0.76	0.75	8.62	P < 0.05
1,2 Dichloroethane	13	0.99	0.89	29.39	P < 0.05	22	1.00	0.90	74.75	P < 0.05
Benzene	25	0.95	0.91	21.10	P < 0.05	23	0.98	0.95	30.58	P < 0.05
Carbon Tetrachloride	25	0.95	0.91	21.87	P < 0.05	23	0.95	1.04	21.18	P < 0.05
Trichloroethene	24	0.89	0.85	14.18	P < 0.05	23	0.92	0.87	16.38	P < 0.05
Toluene	25	0.95	0.93	20.79	P < 0.05	23	0.97	0.99	25.38	P < 0.05
1,1,2 Trichloroethane										
Tetrachloroethene	24	0.96	1.10	25.45	P < 0.05	22	1.00	0.91	76.17	P < 0.05
Chlorobenzene	5	0.60	1.01	2.72	P < 0.05	11	0.67	0.64	4.77	P < 0.05
Ethyl Benzene	24	0.92	0.94	16.79	P < 0.05	22	0.93	0.99	17.28	P < 0.05
m/p Xylene	24	0.94	0.96	18.80	P < 0.05	22	0.95	0.96	20.87	P < 0.05
Bromoform										
Styrene	24	0.86	0.90	12.27	P < 0.05	21	0.96	0.98	23.40	P < 0.05
o Xylene	24	0.83	0.85	10.65	P < 0.05	22	0.96	0.95	23.65	P < 0.05
m Dichlorobenzene										
p Dichlorobenzene	20	0.46	0.67	4.16	P < 0.05	18	0.71	1.47	6.61	P < 0.05
o Dichlorobenzene	1	0.98	1.28	6.28	P > 0.05	7	0.96	1.15	13.24	P < 0.05

Table 2.4

Organization: College of Staten Island Sorbent: Tenax

Quarter of July to September , 1988

DUPLICATE LOW FLOW

compound name	# Pairs run	Average Low Flow 1	Average Low Flow 2	Average Diff. (ppb)	% Diff. (ppb)	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	T Test	T > 0.0 (Y/N)
Methylene Chloride	3	0.281	0.337	-0.057	20.22%	0.112	(-0.539 - 0.425)	0.506	N
1,1 Dichloroethane	0								
Hexane	5	1.013	0.921	0.093	9.17%	0.051	(-0.049 - 0.234)	1.822	N
Chloroform	3	0.102	0.094	0.008	7.59%	0.009	(-0.033 - 0.048)	0.826	N
1,1,1 Trichloroethane	5	0.532	0.454	0.078	14.65%	0.043	(-0.042 - 0.198)	1.798	N
1,2 Dichloroethane	3	0.221	0.198	0.022	10.15%	0.017	(-0.052 - 0.097)	1.294	N
Benzene	5	1.239	1.202	0.037	2.99%	0.073	(-0.165 - 0.239)	0.509	N
Carbon Tetrachloride	5	0.093	0.092	0.001	0.92%	0.011	(-0.029 - 0.030)	0.081	N
Trichloroethene	5	0.131	0.124	0.008	5.84%	0.008	(-0.014 - 0.029)	0.988	N
Toluene	5	4.727	4.650	0.078	1.64%	0.184	(-0.434 - 0.589)	0.421	N
1,1,2 Trichloroethane	0								
Tetrachloroethene	5	0.459	0.450	0.008	1.80%	0.017	(-0.040 - 0.056)	0.477	N
Chlorobenzene	0								
Ethyl Benzene	5	0.969	0.947	0.022	2.29%	0.056	(-0.132 - 0.177)	0.399	N
m/p Xylene	5	3.547	3.484	0.062	1.76%	0.151	(-0.357 - 0.482)	0.413	N
Bromoform	0								
Styrene	5	0.093	0.080	0.012	13.07%	0.012	(-0.021 - 0.045)	1.026	N
o Xylene	5	0.474	0.484	-0.010	2.06%	0.021	(-0.067 - 0.047)	0.476	N
p Dichlorobenzene	1	0.380	0.421	-0.041	10.81%				
m Dichlorobenzene	0								
o Dichlorobenzene	0								

Table 2.5

Organization: College of Staten Island Sorbent: Tenax

Quarter of October to December , 1988

DUPLICATE LOW FLOW

compound name	# Pairs run	Average Low Flow 1	Average Low Flow 2	Average Diff. (ppb)	% Diff.	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	T Test	T > 0.0 (Y/N)
Methylene Chloride	8	0.299	0.346	-0.047	15.60%	0.028	(-0.112 - 0.019)	1.682	N
1,1 Dichloroethane	0								
Hexane	8	0.856	0.886	-0.031	3.61%	0.037	(-0.118 - 0.056)	0.844	N
Chloroform	4	0.025	0.031	-0.006	25.03%	0.003	(-0.017 - 0.005)	1.773	N
1,1,1 Trichloroethane	8	0.401	0.432	-0.031	7.78%	0.015	(-0.066 - 0.004)	2.098	N
1,2 Dichloroethane	2	0.057	0.048	0.010	16.79%	.000	(0.008 - 0.011)	68.615	Y
Benzene	8	1.599	1.677	-0.078	4.90%	0.082	(-0.273 - 0.116)	0.953	N
Carbon Tetrachloride	8	0.126	0.131	-0.005	3.66%	0.005	(-0.017 - 0.008)	0.865	N
Trichloroethene	7	0.053	0.054	-0.001	2.34%	0.004	(-0.010 - 0.008)	0.329	N
Toluene	8	3.358	3.582	-0.224	6.67%	0.171	(-0.628 - 0.180)	1.310	N
1,1,2 Trichloroethane	1	0.085	0.086	-0.001	1.22%				
Tetrachloroethene	8	0.695	0.805	-0.110	15.89%	0.089	(-0.322 - 0.101)	1.234	N
Chlorobenzene	0								
Ethyl Benzene	8	0.579	0.630	-0.051	8.77%	0.038	(-0.140 - 0.038)	1.349	N
m/p Xylene	8	2.228	2.412	-0.184	8.25%	0.124	(-0.477 - 0.109)	1.485	N
Bromoform	0								
Styrene	8	0.085	0.095	-0.010	11.46%	0.005	(-0.022 - 0.003)	1.856	N
o Xylene	8	0.446	0.481	-0.034	7.67%	0.023	(-0.089 - 0.021)	1.466	N
p Dichlorobenzene	8	0.244	0.215	0.029	11.78%	0.055	(-0.102 - 0.160)	0.520	N
m Dichlorobenzene	0								
o Dichlorobenzene	1	0.013	0.005	0.008	62.96%				

Table 2.6

Organization: College of Staten Island Sorbent: Tenax

Quarter of January to March , 1989

DUPLICATE LOW FLOW

compound name	# Pairs run	Average Low Flow 1	Average Low Flow 2	Average Diff. (ppb)	% Diff.	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	T Test	T > 0.((Y/N)
Methylene Chloride	5	0.294	0.259	0.036	12.08%	0.018	(-0.014 - 0.085)	1.986	N
1,1 Dichloroethane	0								
Hexane	5	0.779	0.689	0.090	11.58%	0.059	(-0.074 - 0.255)	1.520	N
Chloroform	4	0.039	0.036	0.003	6.57%	0.003	(-0.006 - 0.011)	0.926	N
1,1,1 Trichloroethane	5	0.549	0.535	0.014	2.53%	0.036	(-0.087 - 0.115)	0.382	N
1,2 Dichloroethane	1	0.055	0.059	-0.004	7.19%				
Benzene	5	1.788	1.547	0.241	13.46%	0.156	(-0.191 - 0.673)	1.546	N
Carbon Tetrachloride	5	0.187	0.176	0.011	5.75%	0.008	(-0.011 - 0.032)	1.388	N
Trichloroethene	5	0.055	0.052	0.002	4.18%	0.004	(-0.010 - 0.014)	0.530	N
Toluene	5	3.913	3.532	0.381	9.75%	0.302	(-0.457 - 1.220)	1.262	N
1,1,2 Trichloroethane	0								
Tetrachloroethene	4	0.407	0.369	0.038	9.38%	0.029	(-0.054 - 0.130)	1.317	N
Chlorobenzene	0								
Ethyl Benzene	4	0.778	0.699	0.079	10.19%	0.074	(-0.157 - 0.315)	1.070	N
m/p Xylene	4	2.730	2.466	0.264	9.68%	0.222	(-0.441 - 0.970)	1.192	N
Bromoform	0								
Styrene	4	0.122	0.115	0.007	5.64%	0.014	(-0.037 - 0.051)	0.496	N
o Xylene	4	0.632	0.574	0.058	9.21%	0.056	(-0.121 - 0.238)	1.031	N
p Dichlorobenzene	4	0.190	0.198	-0.008	4.05%	0.019	(-0.068 - 0.053)	0.404	N
m Dichlorobenzene	0								
o Dichlorobenzene	0								

Table 2.7

Organization: College of Staten Island Sorbent: Tenax

Quarter of April to June , 1989

DUPLICATE LOW FLOW

compound name	# Pairs run	Average Low Flow 1	Average Low Flow 2	Average Diff. (ppb)	% Diff. (ppb)	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	T Test	T > 0.0 (Y/N)
Methylene Chloride	2	0.077	0.086	-0.009	11.89%	0.016	(-0.216 - 0.198)	0.564	N
1,1 Dichloroethane	1	0.007	0.018	-0.011	143.96%				
Hexane	2	0.489	0.589	-0.100	20.51%	0.287	(-3.750 - 3.550)	0.349	N
Chloroform	2	0.100	0.103	-0.003	2.83%	0.011	(-0.143 - 0.137)	0.257	N
1,1,1 Trichloroethane	2	0.425	0.421	0.004	0.94%	0.041	(-0.512 - 0.520)	0.099	N
1,2 Dichloroethane	2	0.009	0.017	-0.009	104.98%	0.004	(-0.060 - 0.043)	2.209	N
Benzene	2	0.349	0.766	-0.417	119.33%	0.255	(-3.660 - 2.827)	1.632	N
Carbon Tetrachloride	2	0.115	0.093	0.023	19.70%	0.013	(-0.143 - 0.188)	1.747	N
Trichloroethene	2	0.039	0.063	-0.025	63.73%	0.011	(-0.169 - 0.120)	2.173	N
Toluene	2	2.252	3.017	-0.764	33.94%	0.289	(-4.436 - 2.907)	2.646	N
1,1,2 Trichloroethane	0								
Tetrachloroethene	2	0.421	0.617	-0.196	46.64%	0.139	(-1.958 - 1.565)	1.417	N
Chlorobenzene	2	0.016	0.023	-0.007	46.01%	0.005	(-0.072 - 0.058)	1.431	N
Ethyl Benzene	2	0.519	0.686	-0.167	32.29%	0.071	(-1.069 - 0.734)	2.361	N
m/p Xylene	2	1.309	1.810	-0.501	38.27%	0.189	(-2.902 - 1.900)	2.652	N
Bromoform	0								
Styrene	2	0.044	0.090	-0.047	106.77%	0.023	(-0.338 - 0.245)	2.029	N
o Xylene	2	0.637	0.443	0.194	30.50%	0.290	(-3.494 - 3.882)	0.670	N
p Dichlorobenzene	2	0.069	0.240	-0.171	247.66%	0.021	(-0.438 - 0.097)	8.113	Y
m Dichlorobenzene	0								
o Dichlorobenzene	1	0.046	0.051	-0.005	11.93%				

Table 2.8

Organization: College of Staten Island **Sorbent: Tenax**

Quarter of July to September , 1989

DUPLICATE LOW FLOW

compound name	# Pairs run	Average Low Flow 1	Average Low Flow 2	Average Diff. (ppb)	% Diff. (ppb)	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	T Test	T > 0.0 (Y/N)
Methylene Chloride	7	0.059	0.051	0.008	14.02%	0.009	(-0.015 - 0.031)	0.881	N
1,1 Dichloroethane	5	0.173	0.091	0.082	47.53%	0.040	(-0.028 - 0.193)	2.069	N
Hexane	7	1.409	1.282	0.127	9.02%	0.090	(-0.094 - 0.348)	1.409	N
Chloroform	7	0.033	0.034	.000	1.15%	0.005	(-0.012 - 0.011)	0.083	N
1,1,1 Trichloroethane	7	0.436	0.426	0.011	2.43%	0.023	(-0.046 - 0.068)	0.455	N
1,2 Dichloroethane	7	0.027	0.028	.000	1.62%	0.001	(-0.004 - 0.003)	0.312	N
Benzene	7	1.134	1.091	0.044	3.86%	0.050	(-0.079 - 0.167)	0.872	N
Carbon Tetrachloride	7	0.042	0.044	-0.002	5.15%	0.008	(-0.023 - 0.018)	0.256	N
Trichloroethene	7	0.060	0.043	0.018	29.24%	0.013	(-0.015 - 0.050)	1.329	N
Toluene	7	3.915	3.575	0.340	8.69%	0.179	(-0.098 - 0.779)	1.899	N
1,1,2 Trichloroethane	1	0.010	0.010	.000	3.05%				
Tetrachloroethene	7	0.473	0.405	0.068	14.36%	0.062	(-0.083 - 0.219)	1.100	N
Chlorobenzene	5	0.011	0.010	0.001	6.53%	0.001	(-0.003 - 0.005)	0.511	N
Ethyl Benzene	7	0.433	0.375	0.058	13.49%	0.018	(0.013 - 0.103)	3.178	Y
m/p Xylene	7	1.556	1.303	0.253	16.28%	0.082	(0.054 - 0.453)	3.104	Y
Bromoform	0								
Styrene	7	0.074	0.059	0.014	19.42%	0.006	(.000 - 0.029)	2.394	Y
o Xylene	7	0.311	0.214	0.096	31.01%	0.029	(0.025 - 0.168)	3.307	Y
† Dichlorobenzene	7	0.091	0.052	0.039	43.16%	0.028	(-0.029 - 0.108)	1.401	N
₦ Dichlorobenzene	0								
o Dichlorobenzene	1	0.009	0.007	0.002	18.19%				

Table 2.9

Organization: College of Staten Island Sorbent: Tenax

Quarter of July to September , 1988

DUPLICATE HIGH FLOW

compound name	# Pairs run	Average High Flow 1	Average High Flow 2	Average Diff. (ppb)	% Diff. (ppb)	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	T Test	T > 0.0 (Y/N)
Methylene Chloride	4	0.179	0.213	-0.033	18.64%	0.040	(-0.161 - 0.095)	0.831	N
1,1 Dichloroethane	0								
Hexane	5	0.769	0.708	0.061	7.93%	0.063	(-0.113 - 0.235)	0.972	N
Chloroform	3	0.066	0.071	-0.005	7.84%	0.015	(-0.069 - 0.059)	0.350	N
1,1,1 Trichloroethane	5	0.369	0.275	0.094	25.43%	0.137	(-0.286 - 0.474)	0.685	N
1,2 Dichloroethane	4	0.177	0.163	0.014	7.75%	0.012	(-0.024 - 0.051)	1.171	N
Benzene	5	0.902	0.863	0.038	4.24%	0.080	(-0.183 - 0.259)	0.481	N
Carbon Tetrachloride	5	0.058	0.057	0.001	1.71%	0.004	(-0.011 - 0.013)	0.227	N
Trichloroethene	5	0.112	0.104	0.008	7.01%	0.007	(-0.012 - 0.028)	1.072	N
Toluene	5	4.293	4.162	0.131	3.05%	0.174	(-0.353 - 0.615)	0.751	N
1,1,2 Trichloroethane	0								
Tetrachloroethene	5	0.509	0.478	0.031	6.15%	0.018	(-0.019 - 0.082)	1.712	N
Chlorobenzene	3	0.010	0.010	.000	3.04%	0.001	(-0.004 - 0.004)	0.342	N
Ethyl Benzene	5	0.782	0.831	-0.050	6.37%	0.078	(-0.267 - 0.167)	0.637	N
m/p Xylene	5	2.781	2.881	-0.100	3.60%	0.245	(-0.779 - 0.579)	0.409	N
Bromoform	0								
Styrene	5	0.080	0.083	-0.003	4.04%	0.003	(-0.012 - 0.006)	0.963	N
o Xylene	5	0.397	0.413	-0.015	3.88%	0.024	(-0.082 - 0.051)	0.645	N
p Dichlorobenzene	1	0.293	0.456	-0.163	55.48%				
m Dichlorobenzene	0								
o Dichlorobenzene	0								

Table 2.10

Organization: College of Staten Island Sorbent: Tenax

Quarter of October to December , 1988

DUPLICATE HIGH FLOW

compound name	# Pairs run	Average High Flow 1	Average High Flow 2	Average Diff. (ppb)	% Diff.	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	T Test	T > 0 (Y/N)
Methylene Chloride	8	0.147	0.132	0.015	10.48%	0.018	(-0.027 - 0.058)	0.851	N
1,1 Dichloroethane	0								
Hexane	8	0.839	0.899	-0.061	7.22%	0.034	(-0.142 - 0.021)	1.759	N
Chloroform	8	0.021	0.024	-0.003	12.39%	0.003	(-0.009 - 0.004)	0.943	N
1,1,1 Trichloroethane	8	0.324	0.305	0.019	5.78%	0.013	(-0.011 - 0.049)	1.468	N
1,2 Dichloroethane	8	0.029	0.028	0.001	2.15%	0.001	(-0.002 - 0.003)	0.599	N
Benzene	8	1.577	1.486	0.091	5.77%	0.071	(-0.077 - 0.260)	1.279	N
Carbon Tetrachloride	8	0.098	0.093	0.005	5.19%	0.003	(-0.003 - 0.013)	1.461	N
Trichloroethene	8	0.055	0.056	-0.001	1.61%	0.002	(-0.005 - 0.003)	0.491	N
Toluene	8	2.955	2.968	-0.012	0.42%	0.055	(-0.143 - 0.118)	0.227	N
1,1,2 Trichloroethane	2	0.047	0.047	.000	0.63%	0.001	(-0.013 - 0.012)	0.303	N
Tetrachloroethene	8	0.697	0.663	0.034	4.88%	0.038	(-0.056 - 0.124)	0.898	N
Chlorobenzene	1	0.014	0.012	0.002	13.96%				
Ethyl Benzene	8	0.493	0.506	-0.013	2.67%	0.010	(-0.036 - 0.009)	1.381	N
m/p Xylene	8	1.840	1.882	-0.042	2.30%	0.029	(-0.112 - 0.027)	1.444	N
Bromoform	0								
Styrene	7	0.087	0.094	-0.006	7.42%	0.002	(-0.012 - -0.001)	2.903	Y
o Xylene	8	0.366	0.378	-0.012	3.26%	0.006	(-0.026 - 0.002)	1.955	N
p Dichlorobenzene	8	0.109	0.105	0.005	4.22%	0.012	(-0.024 - 0.033)	0.381	N
m Dichlorobenzene	0								
o Dichlorobenzene	3	0.002	0.002	.000	14.96%	0.001	(-0.003 - 0.002)	0.418	N

Table 2.11

Organization: College of Staten Island **Surbent: Tenax**

Quarter of January to March , 1989

DUPLICATE HIGH FLOW

compound name	# Pairs run	Average High Flow 1	Average High Flow 2	Average Diff. (ppb)	% Diff. (ppb)	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	T Test	T > 0.0 (Y/N)
Methylene Chloride	5	0.162	0.145	0.017	10.65%	0.003	(0.009 - 0.026)	5.574	Y
1,1 Dichloroethane	0								
Hexane	5	0.778	0.755	0.023	2.95%	0.017	(-0.025 - 0.071)	1.331	N
Chloroform	5	0.038	0.033	0.005	12.97%	0.002	(.000 - 0.010)	2.612	Y
1,1,1 Trichloroethane	5	0.494	0.488	0.006	1.17%	0.010	(-0.021 - 0.032)	0.601	N
1,2 Dichloroethane	5	0.036	0.036	.000	0.93%	0.001	(-0.003 - 0.002)	0.418	N
Benzene	5	2.022	1.867	0.155	7.65%	0.128	(-0.199 - 0.509)	1.212	N
Carbon Tetrachloride	5	0.167	0.175	-0.008	4.78%	0.009	(-0.033 - 0.017)	0.901	N
Trichloroethene	5	0.058	0.059	.000	0.74%	0.002	(-0.005 - 0.004)	0.258	N
Toluene	5	3.535	3.661	-0.127	3.58%	0.154	(-0.556 - 0.302)	0.820	N
1,1,2 Trichloroethane	0								
Tetrachloroethene	4	0.411	0.391	0.020	4.84%	0.011	(-0.014 - 0.054)	1.841	N
Chlorobenzene	2	0.005	0.006	-0.001	10.97%	0.001	(-0.019 - 0.018)	0.403	N
Ethyl Benzene	4	0.700	0.686	0.014	1.99%	0.014	(-0.030 - 0.058)	1.005	N
m/p Xylene	4	2.501	2.399	0.102	4.08%	0.052	(-0.063 - 0.267)	1.961	N
Bromoform	0								
Styrene	4	0.124	0.115	0.009	6.90%	0.005	(-0.007 - 0.024)	1.793	N
o Xylene	4	0.629	0.593	0.035	5.63%	0.019	(-0.024 - 0.094)	1.906	N
p Dichlorobenzene	4	0.139	0.147	-0.008	5.91%	0.012	(-0.047 - 0.030)	0.679	N
M Dichlorobenzene	0								
o Dichlorobenzene	2	0.007	0.008	-0.001	7.88%	.000	(-0.003 - 0.001)	3.686	N

Table 2.12

Organization: College of Staten Island Sorbent: Tenax

Quarter of April to June , 1989

DUPLICATE HIGH FLOW

compound name	# Pairs run	Average High Flow 1	Average High Flow 2	Average Diff. (ppb)	% Diff. (ppb)	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	T Test	T > 0.0 (Y/N)
Methylene Chloride	4	0.040	0.045	-0.005	12.46%	0.012	(-0.042 - 0.032)	0.433	N
1,1 Dichloroethane	2	0.011	0.015	-0.004	39.76%	0.001	(-0.023 - 0.014)	2.896	N
Hexane	4	0.312	0.369	-0.056	17.93%	0.082	(-0.318 - 0.206)	0.681	N
Chloroform	4	0.070	0.053	0.016	23.55%	0.008	(-0.008 - 0.041)	2.113	N
1,1,1 Trichloroethane	4	0.346	0.321	0.025	7.29%	0.049	(-0.130 - 0.181)	0.515	N
1,2 Dichloroethane	4	0.014	0.014	.000	0.08%	0.003	(-0.010 - 0.010)	0.003	N
Benzene	4	0.345	0.351	-0.006	1.63%	0.058	(-0.189 - 0.178)	0.098	N
Carbon Tetrachloride	4	0.101	0.094	0.007	7.25%	0.017	(-0.046 - 0.061)	0.436	N
Trichloroethene	4	0.045	0.040	0.004	9.91%	0.012	(-0.033 - 0.042)	0.377	N
Toluene	4	1.901	1.907	-0.005	0.29%	0.289	(-0.926 - 0.915)	0.019	N
1,1,2 Trichloroethane	0								
Tetrachloroethene	4	0.246	0.272	-0.026	10.76%	0.032	(-0.129 - 0.076)	0.824	N
Chlorobenzene	4	0.012	0.011	0.001	7.51%	0.002	(-0.005 - 0.007)	0.503	N
Ethyl Benzene	4	0.402	0.381	0.020	5.08%	0.068	(-0.196 - 0.237)	0.300	N
m/p Xylene	4	1.076	1.040	0.036	3.39%	0.184	(-0.550 - 0.623)	0.198	N
Bromoform	0								
Styrene	4	0.037	0.036	.000	0.73%	0.009	(-0.028 - 0.028)	0.030	N
o Xylene	4	0.261	0.252	0.009	3.59%	0.051	(-0.153 - 0.172)	0.183	N
p Dichlorobenzene	4	0.144	0.214	-0.070	48.41%	0.088	(-0.351 - 0.211)	0.791	N
m Dichlorobenzene	0								
o Dichlorobenzene	3	0.023	0.027	-0.004	18.62%	0.002	(-0.014 - 0.006)	1.880	N

Table 2.13

Organization: College of Staten Island Sorbent: Tenax

Quarter of July to September , 1989

DUPLICATE HIGH FLOW

compound name	# Pairs run	Average High Flow 1	Average High Flow 2	Average Diff. (ppb)	% Diff. (ppb)	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	T Test	T > 0.0 (Y/N)
Methylene Chloride	3	0.026	0.027	-0.001	5.32%	0.010	(-0.043 - 0.040)	0.145	N
1,1 Dichloroethane	2	0.014	0.076	-0.062	444.75%	0.029	(-0.436 - 0.312)	2.118	N
Hexane	3	0.293	0.296	-0.003	1.17%	0.028	(-0.122 - 0.115)	0.125	N
Chloroform	3	0.013	0.013	.000	2.21%	0.004	(-0.017 - 0.016)	0.072	N
1,1,1 Trichloroethane	3	0.190	0.174	0.016	8.29%	0.020	(-0.069 - 0.100)	0.800	N
1,2 Dichloroethane	3	0.013	0.012	0.001	7.39%	0.001	(-0.004 - 0.006)	0.758	N
Benzene	3	0.377	0.366	0.011	2.86%	0.027	(-0.105 - 0.127)	0.401	N
Carbon Tetrachloride	3	0.016	0.024	-0.008	50.17%	0.006	(-0.034 - 0.018)	1.354	N
Trichloroethene	3	0.045	0.040	0.005	10.55%	0.030	(-0.125 - 0.135)	0.157	N
Toluene	3	2.357	2.569	-0.213	9.03%	0.357	(-1.747 - 1.322)	0.596	N
1,1,2 Trichloroethane	0								
Tetrachloroethene	3	0.673	0.673	.000	0.01%	0.087	(-0.374 - 0.374)	0.001	N
Chlorobenzene	3	0.005	0.006	-0.001	18.09%	0.001	(-0.007 - 0.005)	0.660	N
Ethyl Benzene	3	0.281	0.285	-0.004	1.59%	0.034	(-0.150 - 0.141)	0.132	N
m/p Xylene	3	0.959	0.988	-0.029	3.00%	0.129	(-0.583 - 0.526)	0.223	N
Bromoform	0								
Styrene	3	0.057	0.059	-0.003	4.65%	0.013	(-0.057 - 0.051)	0.210	N
o Xylene	3	0.239	0.255	-0.017	6.93%	0.036	(-0.171 - 0.138)	0.461	N
p Dichlorobenzene	3	0.043	0.020	0.023	53.73%	0.028	(-0.098 - 0.144)	0.823	N
m Dichlorobenzene	0								
o Dichlorobenzene	1	0.003	0.004	-0.001	17.80%				

Table 3.1. Comparison of low and high flow values on basis of paired sample t test

Measurable most of time			Measurable < 33% of time		
Compound	Quarters #	% diff*	Compound	Quarters #	% diff*
Hexane	9	78	Methylene chloride	8	75
Chloroform	9	67	1,1-Dichloroethane	7	71
111-Trichloroethane	9	100	112-Trichloroethane	4	75
12-Dichloroethane	9	78	Chlorobenzene	9	100
Benzene	9	67	Bromoform	4	25
Carbon tetrachloride	9	100	m Dichlorobenzene	3	33
Trichloroethene	8	62	o Dichlorobenzene	4	100
Toluene	8	50	p Dichlorobenzene	4	100
Tetrachloroethene	8	25			
Ethylbenzene	7	57			
m/p Xylene	7	29			
Styrene	7	43			
o Xylene	7	43			
Mean % quarters different		61.5			72.4
S		23.8			29.5

* difference based paired t test

Table 3.2. Distributed Volume Comparison: Quarterly mean low flow divided by quarterly mean high flow for each compound

Compound	4Q87	1Q88	2Q88	3Q88	4Q88	1Q89	2Q89	3Q89	Mean	S
A. Measurable most of the time										
Hexane	0.96	0.99	1.14	1.43	1.02	0.94	1.04	1.31	1.10	0.17
Chloroform	1.02	0.96	1.34	1.83	1.19	1.01	1.47	1.75	1.32	0.32
111-Trichloroethane	1.20	1.23	1.32	1.60	1.31	1.19	1.26	1.48	1.32	0.14
1,2-Dichloroethane	0.95	0.75	0.94	1.30	0.86	0.75	1.16	1.56	1.03	0.27
Benzene	0.98	1.03	1.02	1.17	0.94	0.86	1.00	1.21	1.03	0.11
Carbon Tetrachloride	1.19	1.16	1.29	1.43	1.27	1.12	1.40	1.61	1.31	0.15
Trichloroethene	0.89	0.90	1.03	1.28	0.96	0.93	1.00	0.95	0.99	0.12
Toluene	0.99	1.03	1.02	1.09	1.10	1.02	1.01	0.99	1.03	0.04
Tetrachloroethene	0.96	0.98	0.90	0.91	0.97	0.97	0.91	1.02	0.95	0.04
Ethylbenzene		1.09	1.00	1.05	1.07	0.99	1.01	1.10	1.04	0.04
m/p Xylenes		1.02	1.02	1.09	1.09	1.01	0.94	1.07	1.03	0.05
Styrene		0.98	0.92	1.00	1.01	0.97	0.91	1.14	0.99	0.07
o Xylene	0.96	1.06	0.92	1.05	1.07	0.98	0.94	0.95	0.99	0.06
Mean for all 13	1.01	1.01	1.07	1.25	1.07	0.98	1.08	1.24	1.09	0.10
S	0.10	0.11	0.15	0.25	0.12	0.10	0.18	0.26	0.13	
Mean for first 7	1.03	1.00	1.15	1.43	1.08	0.97	1.19	1.41	1.16	0.17
S	0.11	0.15	0.15	0.21	0.16	0.14	0.18	0.25	0.14	
Mean for last 6	0.97	1.03	0.96	1.03	1.05	0.99	0.95	1.05	1.01	0.04
S	0.01	0.04	0.05	0.06	0.05	0.02	0.04	0.06	0.03	
B. Measurable less than 31% of the time*										
Methylene chloride	2.15	1.78	2.11	1.87	2.02	1.93	2.00	2.00	1.98	0.11
1,1-Dichloroethane		1.78	1.99	2.00	1.73	1.74	1.60	1.28	1.73	0.23
112-Trichloroethane					1.99	1.99	1.94	2.00	1.98	0.02
Chlorobenzene	1.38	1.30	1.19	1.41	1.57	1.39	1.59	1.61	1.43	0.14
Bromoform				2.00	1.85	2.00	2.00	1.98	1.97	0.06
m Dichlorobenzene					1.92	1.63	2.00	2.00	1.89	0.15
p Dichlorobenzene					1.93	2.02	1.97	1.97	1.97	0.03
o Dichlorobenzene					1.68	1.54	2.07	1.96	1.81	0.21
Mean	1.77	1.62	1.76	1.82	1.84	1.78	1.90	1.85	1.79	0.08
S	0.39	0.23	0.41	0.24	0.15	0.23	0.18	0.25		
Mean of columm means									1.85	
S									0.18	

* most or all values at or below MDL

Table 3.3. Means of lo-hi values > MDL for 7 compounds seen on < 31% of days

Compound (N=)		Flow Low	High	Diff	% Diff	t-value
Methylene chloride	Mean	1.40	0.77	0.63	81.8	6.43
58*	S					
1,1-Dichloroethane	Mean	0.032	0.032	0.000	0.0	0.00
10	S	1.874	1.874	0.005		
Chlorobenzene	Mean	0.036	0.032	0.004	13.1	
112	S	0.033	0.026			
Bromoform	Mean	0.022	0.024	0.0025	10.3	
6	S	0.029	0.025			
m-Dichlorobenzene	Mean	0.100	0.140	0.040	28.6	
1	S					
o-Dichlorobenzene	Mean	0.037	0.031	0.006	19.4	
20	S	0.032	0.033			
p-Dichlorobenzene	Mean	0.592	0.292	0.300	102.7	
14	S	0.183	0.086			

* values through December 1988

Table 3.4

Organization: College of Staten Island Sorbent: Tenax

Quarter of July to September , 1987

DISTRIBUTED VOLUMES

compound name	# Pairs run	Average Low Flow	Average MDL Study	Average Diff. (ppb)	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	T-Test	T > 0.05 (Y/N)
1,1 Dichloroethane								
1,1,1 Trichloroethane	89	0.467	0.041	0.170	0.025	(0.121 - 0.219)	6.839	Y
1,2 Dichloroethane	91	0.065	0.020	0.012	0.007	(-0.001 - 0.026)	1.813	N
1,1,2 Trichloroethane								
Benzene	86	1.381	0.130	0.414	0.046	(0.323 - 0.505)	9.020	Y
Bromoform								
Chloroform	78	0.031	0.021	0.012	0.002	(0.008 - 0.016)	6.003	Y
Chlorobenzene	79	0.032	0.020	0.010	0.002	(0.007 - 0.014)	5.255	Y
Carbon Tetrachloride	91	0.117	0.040	0.045	0.006	(0.033 - 0.057)	7.397	Y
Ethyl Benzene								
Hexane	91	0.907	0.100	0.310	0.037	(0.237 - 0.383)	8.415	Y
Methylene Chloride								
m Dichlorobenzene								
m/p Xylene								
o Dichlorobenzene								
o Xylene								
p Dichlorobenzene								
Styrene								
Toluene								
Tetrachloroethene								
Trichloroethene								

* = </= MDL

MDL(HIGH FLOW) = 1/2 MDL(LOW FLOW)

Table 3.5

Organization: College of Staten Island Sorbent: Tenax

Quarter of October to December , 1987

DISTRIBUTED VOLUMES

compound name	# Pairs run	Average Low Flow	Average MDL Study	Average Diff. (ppb)	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	T-Test	T > 0.05 (Y/N)
1,1 Dichloroethane								
1,1,1 Trichloroethane	243	0.456	0.041	0.078	0.008	(0.063 - 0.093)	10.241	Y
1,2 Dichloroethane	244	0.055	0.020	-0.003	0.002	(-0.008 - 0.002)	1.160	N
1,1,2 Trichloroethane								
Benzene	244	1.848	0.130	-0.023	0.022	(-0.066 - 0.020)	1.035	N
Bromoform								
Chloroform	244	0.055	0.021	0.001	0.001	(-0.001 - 0.004)	0.949	N
Chlorobenzene	249	0.013 *	0.020	0.004	.000	(0.003 - 0.004)	12.946	Y
Carbon Tetrachloride	244	0.131	0.040	0.022	0.002	(0.018 - 0.025)	12.150	Y
Ethyl Benzene								
Hexane	244	1.122	0.100	-0.037	0.017	(-0.071 - -0.003)	2.120	Y
Methylene Chloride	244	0.759 *	1.300	0.406	0.021	(0.364 - 0.448)	18.936	Y
m Dichlorobenzene								
m/p Xylene								
o Dichlorobenzene								
o Xylene	243	0.567	0.139	-0.016	0.005	(-0.026 - -0.006)	3.094	Y
p Dichlorobenzene								
Styrene								
Toluene	245	5.130	0.400	-0.005	0.058	(-0.118 - 0.108)	0.094	N
Tetrachloroethene	236	0.428	0.050	-0.015	0.009	(-0.033 - 0.002)	1.688	N
Trichloroethene	245	0.072	0.025	-0.008	0.002	(-0.011 - -0.005)	5.198	Y

* = </= MDL

MDL(HIGH FLOW) = 1/2 MDL(LOW FLOW)

Table 3.6

Organization: College of Staten Island Sorbent: Tenax

Quarter of January to March , 1988

DISTRIBUTED VOLUMES

compound name	# Pairs run	Average Low Flow	Average MDL Study	Average Diff. (ppb)	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	T-Test	T > 0.05 (Y/N)
1,1 Dichloroethane	112	0.010 *	0.020	0.004	.000	(0.004 - 0.005)	18.741	Y
1,1,1 Trichloroethane	184	0.493	0.041	0.094	0.008	(0.078 - 0.109)	11.786	Y
1,2 Dichloroethane	184	0.026	0.020	-0.009	0.001	(-0.010 - -0.007)	9.134	Y
1,1,2 Trichloroethane								
Benzene	185	1.843	0.130	0.064	0.028	(0.009 - 0.119)	2.292	Y
Bromoform								
Chloroform	183	0.045	0.021	-0.002	0.001	(-0.004 - .000)	1.697	N
Chlorobenzene	185	0.014 *	0.020	0.003	.000	(0.003 - 0.004)	10.056	Y
Carbon Tetrachloride	184	0.112	0.040	0.016	0.002	(0.012 - 0.020)	8.691	Y
Ethyl Benzene	183	1.230	0.223	0.104	0.017	(0.071 - 0.137)	6.144	Y
Hexane	180	1.084	0.100	-0.005	0.022	(-0.047 - 0.038)	0.212	N
Methylene Chloride	181	0.700 *	1.100	0.308	0.024	(0.260 - 0.355)	12.701	Y
m Dichlorobenzene								
m/p Xylene	101	1.995	0.417	0.045	0.040	(-0.034 - 0.124)	1.137	N
o Dichlorobenzene								
o Xylene	184	0.420	0.139	0.027	0.007	(0.014 - 0.040)	4.095	Y
p Dichlorobenzene								
Styrene	183	0.109	0.031	-0.001	0.002	(-0.005 - 0.002)	0.771	N
Toluene	184	4.917	0.400	0.164	0.059	(0.049 - 0.278)	2.793	Y
Tetrachloroethene	165	0.419	0.050	-0.004	0.007	(-0.018 - 0.009)	0.636	N
Trichloroethene	184	0.076	0.025	-0.008	0.001	(-0.010 - -0.006)	7.009	Y

* = </= MDL

MDL(HIGH FLOW) = 1/2 MDL(LOW FLOW)

Table 3.7

Organization: College of Staten Island Sorbent: Tenax

Quarter of April to June , 1988

DISTRIBUTED VOLUMES

compound name	# Pairs run	Average Low Flow	Average MDL Study	Average Diff. (ppb)	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	T-Test	T > 0.05 (Y/N)
1,1 Dichloroethane	251	0.010 *	0.020	0.005	.000	(0.005 - 0.005)	347.008	Y
1,1,1 Trichloroethane	250	0.337	0.041	0.083	0.008	(0.068 - 0.098)	10.826	Y
1,2 Dichloroethane	251	0.018 *	0.020	-0.001	0.001	(-0.003 - 0.001)	0.907	N
1,1,2 Trichloroethane								
Benzene	243	0.042	0.130	0.020	0.022	(-0.023 - 0.063)	0.905	N
Bromoform								
Chloroform	251	0.022	0.021	0.006	0.001	(0.004 - 0.007)	6.821	Y
Chlorobenzene	221	0.013 *	0.020	0.002	.000	(0.001 - 0.003)	6.301	Y
Carbon Tetrachloride	248	0.097	0.040	0.022	0.002	(0.018 - 0.026)	10.703	Y
Ethyl Benzene	216	0.487	0.223	0.001	0.008	(-0.015 - 0.017)	0.152	N
Hexane	248	0.558	0.100	0.070	0.013	(0.044 - 0.096)	5.255	Y
Methylene Chloride	251	0.310 *	0.611	0.163	0.016	(0.131 - 0.196)	9.961	Y
m Dichlorobenzene								
m/p Xylene	216	1.498	0.417	0.044	0.026	(-0.007 - 0.094)	1.682	N
o Dichlorobenzene								
o Xylene	218	0.247	0.139	-0.020	0.015	(-0.049 - 0.009)	1.366	N
p Dichlorobenzene								
Styrene	218	0.057	0.031	-0.005	0.001	(-0.007 - -0.002)	4.097	Y
Toluene	224	2.785	0.400	0.061	0.031	(-0.001 - 0.123)	1.942	N
Tetrachloroethene	226	0.317	0.050	-0.032	0.008	(-0.048 - -0.016)	3.870	Y
Trichloroethene	245	0.069	0.025	0.003	0.002	(-0.001 - 0.007)	1.293	N

* = </= MDL

MDL(HIGH FLOW) = 1/2 MDL(LOW FLOW)

Table 3.8

Organization: College of Staten Island Sorbent: Tenax

Quarter of July to September , 1988

DISTRIBUTED VOLUMES

compound name	# Pairs run	Average Low Flow	Average MDL Study	Average Diff. (ppb)	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	T-Test	T > 0.05 (Y/N)
1,1 Dichloroethane	53	0.010 *	0.020	0.005	0.000	(0.005 - 0.005)	0.000	N
1,1,1 Trichloroethane	211	0.390	0.041	0.147	0.017	(0.115 - 0.179)	8.879	Y
1,2 Dichloroethane	211	0.024	0.020	0.006	0.001	(0.003 - 0.008)	3.883	Y
1,1,2 Trichloroethane								
Benzene	115	1.181	0.130	0.173	0.034	(0.105 - 0.241)	5.060	Y
Bromoform	74	0.010 *	0.020	0.005	0.000	(0.005 - 0.005)	0.000	N
Chloroform	212	0.037	0.021	0.017	0.002	(0.014 - 0.020)	10.727	Y
Chlorobenzene	196	0.012 *	0.020	0.004	.000	(0.003 - 0.004)	11.980	Y
Carbon Tetrachloride	158	0.074	0.040	0.022	0.002	(0.018 - 0.027)	9.853	Y
Ethyl Benzene	189	0.646	0.223	0.034	0.013	(0.009 - 0.059)	2.681	Y
Hexane	210	0.701	0.100	0.211	0.015	(0.182 - 0.240)	14.303	Y
Methylene Chloride	212	0.229 *	0.611	0.107	0.009	(0.090 - 0.124)	12.502	Y
m Dichlorobenzene								
m/p Xylene	189	2.237	0.417	0.186	0.039	(0.110 - 0.262)	4.806	Y
o Dichlorobenzene								
o Xylene	192	0.361	0.139	0.018	0.007	(0.004 - 0.031)	2.611	Y
p Dichlorobenzene								
Styrene	192	0.078	0.031	.000	0.002	(-0.003 - 0.004)	0.142	N
Toluene	205	4.165	0.400	0.345	0.045	(0.257 - 0.432)	7.708	Y
Tetrachloroethene	205	0.435	0.050	-0.039	0.008	(-0.056 - -0.022)	4.631	Y
Trichloroethene	211	0.077	0.025	0.017	0.002	(0.014 - 0.020)	10.349	Y

* = <= MDL

MDL(HIGH FLOW) = 1/2 MDL(LOW FLOW)

Table 3.9

Organization: College of Staten Island Sorbent: Tenax

Quarter of October to December , 1988

DISTRIBUTED VOLUMES

compound name	# Pairs run	Average Low Flow	Average MDL Study	Average Diff. (ppb)	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	T-Test	T > 0.05 (Y/N)
1,1 Dichloroethane	227	0.011 *	0.020	0.005	.000	(0.004 - 0.005)	34.515	Y
1,1,1 Trichloroethane	220	0.491	0.041	0.117	0.011	(0.096 - 0.138)	10.989	Y
1,2 Dichloroethane	228	0.022	0.020	-0.004	0.001	(-0.005 - -0.002)	5.808	Y
1,1,2 Trichloroethane	231	0.020 *	0.040	0.010	.000	(0.010 - 0.010)	2265.860	Y
Benzene	228	1.709	0.130	-0.094	0.021	(-0.135 - -0.052)	4.444	Y
Bromoform	210	0.010 *	0.020	0.004	.000	(0.004 - 0.005)	34.380	Y
Chloroform	228	0.039	0.021	0.006	0.001	(0.004 - 0.009)	5.403	Y
Chlorobenzene	210	0.011 *	0.020	0.004	.000	(0.004 - 0.005)	17.060	Y
Carbon Tetrachloride	228	0.127	0.040	0.027	0.002	(0.024 - 0.030)	16.062	Y
Ethyl Benzene	210	0.653	0.223	0.044	0.009	(0.027 - 0.061)	5.060	Y
Hexane	228	0.969	0.100	0.025	0.011	(0.004 - 0.047)	2.306	Y
Methylene Chloride	228	0.327 *	0.611	0.166	0.016	(0.135 - 0.196)	10.603	Y
m Dichlorobenzene	209	0.011 *	0.020	0.005	.000	(0.005 - 0.006)	14.118	Y
m/p Xylene	210	2.357	0.417	0.198	0.024	(0.151 - 0.244)	8.340	Y
o Dichlorobenzene	210	0.012 *	0.020	0.005	.000	(0.004 - 0.005)	16.272	Y
o Xylene	210	0.479	0.133	0.034	0.005	(0.023 - 0.044)	6.448	Y
p Dichlorobenzene	209	0.236 *	0.400	0.114	0.008	(0.099 - 0.129)	14.688	Y
Styrene	209	0.103	0.031	0.001	0.002	(-0.003 - 0.006)	0.523	N
Toluene	228	3.855	0.400	0.358	0.030	(0.300 - 0.416)	12.028	Y
Tetrachloroethene	228	0.352	0.050	-0.010	0.006	(-0.021 - 0.001)	1.723	N
Trichloroethene	227	0.074	0.025	-0.003	0.001	(-0.005 - -0.001)	2.840	Y

* = <= MDL

MDL(HIGH FLOW) = 1/2 MDL(LOW FLOW)

Table 3.10

Organization: College of Staten Island Sorbent: Tenax

Quarter of January to March , 1989

DISTRIBUTED VOLUMES

compound name	# Pairs run	Average Low Flow	Average MDL Study	Average Diff. (ppb)	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	T-Test	T > 0.05 (Y/N)
1,1 Dichloroethane	228	0.010 *	0.020	0.004	.000	(0.004 - 0.005)	23.132	Y
1,1,1 Trichloroethane	228	0.490	0.041	0.080	0.007	(0.067 - 0.094)	11.489	Y
1,2 Dichloroethane	228	0.027	0.020	-0.009	0.001	(-0.011 - -0.007)	9.339	Y
1,1,2 Trichloroethane	228	0.020 *	0.040	0.010	.000	(0.010 - 0.010)	227.000	Y
Benzene	225	1.598	0.130	-0.259	0.021	(-0.301 - -0.217)	12.140	Y
Bromoform	225	0.010 *	0.020	0.005		(All at MDL)		
Chloroform	227	0.039	0.021	.000	0.001	(-0.002 - 0.002)	0.384	N
Chlorobenzene	222	0.012 *	0.020	0.003	.000	(0.003 - 0.004)	9.407	Y
Carbon Tetrachloride	225	0.178	0.040	0.020	0.002	(0.015 - 0.025)	8.322	Y
Ethyl Benzene	219	0.587	0.223	-0.004	0.005	(-0.015 - 0.006)	0.800	N
Hexane	228	0.780	0.100	-0.043	0.010	(-0.063 - -0.023)	4.269	Y
Methylene Chloride	228	0.326 *	0.611	0.158	0.009	(0.141 - 0.175)	17.991	Y
m Dichlorobenzene	219	0.010 *	0.020	0.005		(All at MDL)		
m/p Xylene	219	2.028	0.417	0.021	0.018	(-0.013 - 0.056)	1.205	N
o Dichlorobenzene	217	0.011 *	0.020	0.004	.000	(0.003 - 0.005)	10.526	Y
o Xylene	217	0.451	0.139	-0.008	0.005	(-0.018 - 0.002)	1.595	N
p Dichlorobenzene	217	0.213 *	0.400	0.101	0.004	(0.094 - 0.108)	28.232	Y
Styrene	217	0.091	0.031	-0.003	0.001	(-0.005 - .000)	2.322	Y
Toluene	225	3.804	0.400	0.071	0.033	(0.006 - 0.136)	2.140	Y
Tetrachloroethene	223	0.416	0.050	-0.009	0.006	(-0.021 - 0.004)	1.397	N
Trichloroethene	226	0.077	0.025	-0.005	0.001	(-0.007 - -0.003)	5.242	Y

* = <= MDL

MDL(HIGH FLOW) = 1/2 MDL(LOW FLOW)

Table 3.11

Organization: College of Staten Island Sorbent: Tenax

Quarter of April to June , 1989

DISTRIBUTED VOLUMES

compound name	# Pairs run	Average Low Flow	Average MDL Study	Average Diff. (ppb)	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	T-Test	T > 0.05 (Y/N)
1,1 Dichloroethane	14	0.015 *	0.020	0.006	0.001	(0.003 - 0.009)	4.277	Y
1,1,1 Trichloroethane	14	0.445	0.041	0.093	0.028	(0.033 - 0.153)	3.350	Y
1,2 Dichloroethane	14	0.012 *	0.020	0.002	0.001	(.000 - 0.003)	2.350	Y
1,1,2 Trichloroethane	14	0.020 *	0.040	0.010	.000	(0.009 - 0.010)	31.534	Y
Benzene	13	0.725	0.130	0.006	0.072	(-0.150 - 0.162)	0.082	N
Bromoform	14	0.010 *	0.020	0.005	0.000	(0.005 - 0.005)	0.000	N
Chloroform	14	0.043	0.021	0.014	0.004	(0.006 - 0.022)	3.787	Y
Chlorobenzene	14	0.015 *	0.020	0.006	0.001	(0.003 - 0.009)	3.958	Y
Carbon Tetrachloride	14	0.123	0.040	0.035	0.004	(0.026 - 0.045)	8.097	Y
Ethyl Benzene	14	0.295	0.223	0.004	0.023	(-0.045 - 0.053)	0.158	N
Hexane	14	0.519	0.100	0.022	0.053	(-0.092 - 0.136)	0.422	N
Methylene Chloride	14	0.200 *	0.611	0.100	0.000	(0.100 - 0.100)	0.000	N
m Dichlorobenzene	14	0.010 *	0.020	0.005	0.000	(0.005 - 0.005)	0.000	N
m/p Xylene	14	1.010	0.417	-0.057	0.074	(-0.216 - 0.102)	0.768	N
o Dichlorobenzene	14	0.011 *	0.020	0.006	.000	(0.005 - 0.007)	12.071	Y
o Xylene	14	0.230	0.139	-0.014	0.016	(-0.048 - 0.020)	0.885	N
p Dichlorobenzene	14	0.219 *	0.400	0.108	0.007	(0.093 - 0.123)	15.474	Y
Styrene	14	0.037	0.031	-0.003	0.004	(-0.012 - 0.005)	0.898	N
Toluene	14	2.330	0.400	0.039	0.109	(-0.197 - 0.275)	0.357	N
Tetrachloroethene	14	0.267	0.050	-0.024	0.018	(-0.062 - 0.015)	1.308	N
Trichloroethene	14	0.037	0.025	.000	0.002	(-0.004 - 0.004)	0.055	N

* = </= MDL

MDL(HIGH FLOW) = 1/2 MDL(LOW FLOW)

Table 3.12

Organization: College of Staten Island Sorbent: Tenax

Quarter of July to September , 1989

DISTRIBUTED VOLUMES

compound name	# Pairs run	Average Low Flow		Average MDL Study	Average Diff. (ppb)	Std. Err. Avg. Dif.	95% CL interval (ppb)			T-Test	T > 0.05 (Y/N)
1,1 Dichloroethane	39	0.011	*	0.020	0.002	0.002	(-0.001	-	0.006)	1.307	N
1,1,1 Trichloroethane	39	0.254		0.041	0.083	0.011	(0.060	-	0.105)	7.550	Y
1,2 Dichloroethane	39	0.018	*	0.020	0.007	0.001	(0.005	-	0.008)	10.405	Y
1,1,2 Trichloroethane	39	0.020	*	0.040	0.010	0.000	(0.010	-	0.010)	0.000	N
Benzene	32	0.844		0.130	0.148	0.029	(0.089	-	0.206)	5.080	Y
Bromoform	39	0.010	*	0.020	0.005	.000	(0.005	-	0.005)	122.024	Y
Chloroform	41	0.013	*	0.021	0.006	0.001	(0.004	-	0.007)	8.392	Y
Chlorobenzene	39	0.011	*	0.020	0.004	.000	(0.003	-	0.005)	14.210	Y
Carbon Tetrachloride	39	0.030	*	0.040	0.012	0.001	(0.009	-	0.014)	8.429	Y
Ethyl Benzene	39	0.337		0.223	0.091	0.013	(0.064	-	0.058)	2.350	Y
Hexane	35	0.782		0.100	0.188	0.035	(0.118	-	0.258)	5.443	Y
Methylene Chloride	39	0.200	*	0.611	0.100	.000	(0.100	-	0.100)	.000	N
m Dichlorobenzene	39	0.010	*	0.020	0.005	.000	(0.005	-	0.005)	.000	N
m/p Xylene	39	1.175		0.417	0.085	0.043	(-0.002	-	0.172)	1.978	N
o Dichlorobenzene	39	0.010	*	0.020	0.005	.000	(0.005	-	0.005)	101.025	Y
o Xylene	39	0.227		0.139	-0.012	0.014	(-0.040	-	0.016)	0.854	N
p Dichlorobenzene	39	0.200	*	0.400	0.099	0.001	(0.097	-	0.101)	88.625	Y
Styrene	39	0.056		0.031	0.007	0.003	(0.002	-	0.012)	2.612	Y
Toluene	39	2.835		0.400	-0.026	0.086	(-0.200	-	0.147)	0.307	N
Tetrachloroethene	40	0.378		0.050	0.010	0.028	(-0.046	-	0.067)	0.373	N
Trichloroethene	39	0.023	*	0.025	-0.001	0.003	(-0.007	-	0.005)	0.357	N

* = </= MDL

MDL(HIGH FLOW) = 1/2 MDL(LOW FLOW)

Table 4.1

Organization: College of Staten Island Sorbent: Tenax

Regression summary for January 1988 to September 1989

TENAX vs. CANISTER

Compound name	df	Regression coefficient	Slope	t-Test on slope	Significance	df	Regression coefficient	Slope	t-Test on slope	Significance
Methylene chloride	49	0.011	0.005	0.714	p > 0.2					
Chloroform	0									
111-Trichloroethane	41	0.308	0.341	4.272	p < 0.001	40	0.570	0.570	7.280	p < 0.001
Carbon Tetrachloride	3	0.073	0.677	0.486	p > 0.5					
Benzene	63	0.529	1.080	8.425	p < 0.001					
Trichloroethene	2	0.266	2.130	0.851	p > 0.5	1	0.994	3.008	12.873	p < 0.001
Tetrachloroethene	24	0.909	1.134	15.501	p < 0.001					
Ethylbenzene	39	0.242	1.090	3.531	p < 0.002	37	0.436	1.236	5.347	p < 0.001
m/p Xylenes	59	0.281	0.660	4.800	p < 0.001	58	0.681	1.387	11.138	p < 0.001
o Xylene	46	0.613	0.680	8.527	p < 0.001					
Hexane	43	0.109	0.008	2.292	p < 0.050	34	0.516	1.118	6.015	p < 0.001
Toluene	64	0.363	0.578	6.048	p < 0.001	63	0.588	0.939	9.501	p < 0.001

Table 4.2

Organization: College of Staten Island Sorbent: Tenax

Summary of January 1988 to September 1989

TENAX VS. CANISTER

Compound name	# Pairs run	Average Difference (ppb)	Average Difference (%)	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	t Test	T > 0.0 (Y/N)
Methylene chloride	51	1.251	552.4	0.470	(0.301 - 2.201)	2.662	Y
Chloroform	5	0.381	3814.0	0.039	(0.274 - 0.488)	9.896	Y
111-trichloroethane	43	0.347	152.3	0.065	(0.216 - 0.479)	5.350	Y
Carbon Tetrachloride	5	-0.014	101.4	0.100	(-0.292 - 0.263)	0.144	N
Benzene	65	0.042	36.1	0.075	(-0.108 - 0.192)	0.555	N
Trichloroethene	4	0.067	770.8	0.091	(-0.223 - 0.358)	0.736	N
Tetrachloroethene	26	-0.052	10.1	0.048	(-0.151 - 0.046)	1.092	N
Ethylbenzene	41	-0.204	0.5	0.069	(-0.345 - -0.064)	2.950	Y
m/p Xylenes	61	-0.366	-1.7	0.121	(-0.607 - -0.125)	3.034	Y
o Xylene	48	0.230	115.6	0.025	(0.178 - 0.281)	9.029	Y
Hexane	45	8.747	1032.2	3.750	(1.169 - 16.326)	2.333	Y
Toluene	66	-0.335	0.3	0.221	(-0.776 - 0.107)	1.517	N

Table 4.3

New York State DOH ELAP Proficiency Test Results for CSI-CES
(reported value/mean for compound)

	Conc.	Jan 88	Jul 88	Jan 89	Jul 89	Jan 90
1,2-Dichlorobenzene	low					1.161
	high					1.057
1,3-Dichlorobenzene	low			1.136	1.153	
	high			0.996	0.981	
1,4-Dichlorobenzene	low			1.278 *	1.150	
	high			1.071	0.992	
1,1-Dichloroethane	low	1.325 *	0.987			
	high	1.169	1.004			
1,2-Dichloroethane	low	1.025	1.381			
	high	0.809	1.034			
Benzene	low					1.041
	high					1.038
Bromoform	low			1.258	1.145	
	high			1.028	1.024	
Carbon Tetrachloride	low					1.098
	high					1.182
Chlorobenzene	low	1.018	1.012			
	high	0.975	0.919			
Chloroform	low					1.023
	high					1.033
Ethyl benzene	low	1.243	1.062			1.102
	high	1.094	1.032			1.097
Methylene Chloride	low					0.885
	high					1.048
m/p-Xylene	low	1.155	1.124	1.285 *	1.170	
	high	1.018	1.079	1.164	0.992	
Tetrachloroethene	low	1.227	1.120			
	high	1.059	1.063			
Toluene	low			1.336 **	1.160	1.153
	high			1.162	0.972	1.067
Trichloroethene	low			1.337 *	1.374 *	
	high			1.145	1.269 *	

* between 2 and 3 S from mean ** beyond 3 S

Table 4.4

Organization: College of Staten Island Sorbent: Tenax

Quarter of January to March , 1988

TENAX VS. CANISTER

compound name	# Pairs run	Average Difference (ppb)	Average Difference (%)	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	t Test	T > 0.0 (Y/N)
Methylene chloride	6	4.275	1124.7	3.88	(-5.688 - 14.238)	1.103	N
Chloroform	0						
111-trichloroethane	4	0.055	25.5	0.10	(-0.254 - 0.364)	0.566	N
Carbon Tetrachloride	0						
Benzene	9	-0.260	-15.1	0.13	(-0.561 - 0.041)	1.995	N
Trichloroethene	0						
Tetrachloroethene	1	0.040	6.5				
Ethylbenzene	2	-0.535	215.7	1.64	(-21.309 - 20.239)	0.327	N
m/p Xylenes	8	0.325	53.1	0.67	(-1.262 - 1.911)	0.484	N
o Xylene	4	0.490	267.9	0.14	(0.044 - 0.936)	3.499	Y
Hexane	8	48.690	5563.3	14.82	(13.644 - 83.737)	3.286	Y
Toluene	9	-1.133	-28.1	0.33	(-1.898 - -0.367)	3.412	Y

Table 4.5

Organization: College of Staten Island Sorbent: Tenax

Quarter of April to June , 1988

TENAX VS. CANISTER

Compound name	# Pairs run	Average Difference (ppb)	Average Difference (%)	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	t Test	T > 0.05 (Y/N)
Methylene chloride	10	1.120	554.6	0.474	(0.048 - 2.192)	2.363	Y
Chloroform	0						
111-trichloroethane	6	0.388	217.1	0.159	(-0.021 - 0.797)	2.438	N
Carbon Tetrachloride	2	0.143	276.9	0.027	(-0.200 - 0.486)	5.296	Y
Benzene	12	0.356	94.8	0.268	(-0.234 - 0.945)	1.328	N
Trichloroethene	0						
Tetrachloroethene	4	-0.038	-0.8	0.058	(-0.222 - 0.145)	0.663	N
Ethylbenzene	3	-0.268	-39.1	0.025	(-0.376 - -0.160)	10.658	Y
m/p Xylenes	11	-0.159	3.4	0.148	(-0.489 - 0.170)	1.078	N
o Xylene	7	0.227	173.5	0.075	(0.043 - 0.411)	3.022	Y
Hexane	6	0.729	188.5	0.757	(-1.218 - 2.676)	0.962	N
Toluene	12	-0.214	15.0	0.643	(-1.629 - 1.202)	0.332	N

Table 4.6

Organization: College of Staten Island Sorbent: Tenax

Quarter of July to September ,1988

TENAX VS. CANISTER

Compound name	# Pairs run	Average Difference (ppb)	Average Difference (%)	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	t Test	T > 0.05 (Y/N)
Methylene chloride	10	0.794	516.2	0.140	(0.477 - 1.111)	5.671	Y
Chloroform	0						
111-trichloroethane	8	0.231	151.7	0.071	(0.062 - 0.400)	3.236	Y
Carbon Tetrachloride	1	0.122	42.1				
Benzene	13	0.219	60.0	0.148	(-0.104 - 0.542)	1.479	N
Trichloroethene	1	-0.041	-11.7				
Tetrachloroethene	8	-0.230	-31.0	0.037	(-0.318 - -0.142)	6.193	Y
Ethylbenzene	11	-0.392	-44.9	0.051	(-0.506 - -0.279)	7.714	Y
m/p Xylenes	12	-1.192	-43.5	0.116	(-1.447 - -0.937)	10.274	Y
o Xylene	12	0.195	46.8	0.037	(0.114 - 0.276)	5.290	Y
Hexane	6	-0.010	11.4	0.123	(-0.327 - 0.306)	0.083	N
Toluene	13	-1.242	-26.3	0.146	(-1.561 - -0.924)	8.498	Y

Table 4.7

Organization: College of Staten Island Sorbent: Tenax

Quarter of October to December ,1988

TENAX VS. CANISTER

Compound name	# Pairs run	Average Difference (ppb)	Average Difference (%)	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	t Test	T > 0.05 (Y/N)
Methylene chloride	6	0.415	254.6	0.088	(0.189 - 0.641)	4.716	Y
Chloroform	0						
111-trichloroethane	3	0.190	33.7	0.107	(-0.270 - 0.650)	1.777	N
Carbon Tetrachloride	0						
Benzene	10	-0.291	-14.0	0.100	(-0.517 - -0.065)	2.913	Y
Trichloroethene	0						
Tetrachloroethene	7	-0.059	2.5	0.094	(-0.288 - 0.171)	0.625	N
Ethylbenzene	6	-0.242	-25.0	0.087	(-0.465 - -0.019)	2.786	Y
m/p Xylenes	10	-0.473	-18.3	0.146	(-0.804 - -0.142)	3.235	Y
o Xylene	6	0.225	102.1	0.049	(0.099 - 0.351)	4.582	Y
Hexane	6	-0.260	-22.2	0.109	(-0.539 - 0.019)	2.395	N
Toluene	10	-0.244	-9.9	0.149	(-0.581 - 0.093)	1.697	N

Table 4.8

Organization: College of Staten Island Sorbent: Tenax

Quarter of January to March , 1989

Compound name	# Pairs run	Average Difference (ppb)	Average Difference (%)	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	t Test	T > 0.0 (Y/N)
Methylene chloride	9	0.591	191.4	0.254	(0.005 - 1.177)	2.327	Y
Chloroform	0						
1,1,1-trichloroethane	11	0.367	62.8	0.084	(0.179 - 0.555)	4.352	Y
Carbon Tetrachloride	2	-0.240	-44.4	0.120	(-1.765 - 1.285)	2.000	N
Benzene	11	-0.293	-9.0	0.128	(-0.578 - -0.007)	2.286	Y
Trichloroethene	2	0.005	47.5	0.115	(-1.456 - 1.466)	0.043	N
Tetrachloroethene	2	0.035	13.4	0.065	(-0.791 - 0.861)	0.538	N
Ethylbenzene	8	-0.172	-23.6	0.086	(-0.377 - 0.032)	2.000	N
m/p Xylenes	9	-0.621	-21.5	0.269	(-1.242 - .000)	2.307	Y
o Xylene	9	0.129	26.6	0.053	(0.008 - 0.250)	2.451	Y
Hexane	9	0.038	7.8	0.077	(-0.140 - 0.216)	0.493	N
Toluene	10	-0.407	-1.8	0.386	(-1.280 - 0.466)	1.054	N

Table 4.9

Organization: College of Staten Island Sorbent: Tenax

Quarter of April to June ,1989

TENAX VS. CANISTER

compound name	# Pairs run	Average Difference (ppb)	Average Difference (%)	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	t Test	T > 0.0 (Y/N)
Methylene chloride	4	0.775	516.7	0.405	(-0.514 - 2.064)	1.914	N
Chloroform	0						
111-trichloroethane	4	0.096	33.0	0.027	(0.010 - 0.182)	3.555	Y
Carbon Tetrachloride	0						
Benzene	4	0.270	45.1	0.037	(0.153 - 0.387)	7.348	Y
Trichloroethene	0						
Tetrachloroethene	0						
Ethylbenzene	4	-0.008	-3.2	0.020	(-0.071 - 0.054)	0.419	N
m/p Xylenes	4	-0.158	-13.5	0.012	(-0.196 - -0.121)	13.448	Y
o Xylene	3	0.110	44.0	0.029	(-0.015 - 0.235)	3.793	Y
Hexane	3	-0.167	9.5	0.239	(-1.196 - 0.862)	0.697	N
Toluene	4	-0.181	-8.5	0.159	(-0.686 - 0.324)	1.141	N

Table 4.10

Organization: College of Staten Island Sorbent: Tenax

Quarter of July to September ,1989

TENAX VS. CANISTER

Compound name	# Pairs run	Average Difference (ppb)	Average Difference (%)	Std. Err. Avg. Dif. (ppb)	95% CL interval (ppb)	t Test	T > 0.0 (Y/N)
Methylene chloride	6	1.350	900.0	0.771	(-0.632 - 3.332)	1.751	N
Chloroform	5	0.381	3814.0	0.039	(0.274 - 0.488)	9.896	Y
111-trichloroethane	7	0.793	429.4	0.285	(0.095 - 1.491)	2.780	Y
Carbon Tetrachloride	0						
Benzene	5	0.436	101.8	0.111	(0.128 - 0.744)	3.928	Y
Trichloroethene	1	0.300	3000.0				
Tetrachloroethene	4	0.233	115.8	0.182	(-0.345 - 0.811)	1.284	N
Ethylbenzene	7	0.129	81.9	0.039	(0.032 - 0.225)	3.268	Y
m/p Xylenes	7	0.299	62.6	0.081	(0.100 - 0.497)	3.685	Y
o Xylene	7	0.328	245.2	0.032	(0.249 - 0.408)	10.106	Y
Hexane	7	0.217	111.1	0.100	(-0.027 - 0.461)	2.172	N
Toluene	7	1.976	81.1	1.317	(-1.248 - 5.199)	1.500	N

Table 4.11

Tenax quarterly mean divided by canister quarterly mean

Compound	1988				1989			Mean
	1st Q	2nd Q	3rd Q	4th Q	1st Q	2nd Q	3rd Q	
Hexane	0.02	0.47	1.01	1.34	0.96	1.23	0.70	0.09
1,1,1-Trichloroethane	0.90	0.39	0.58	0.78	0.64	0.76	0.20	0.55
Benzene	1.22	0.71	0.84	1.22	1.19	0.69	0.60	0.97
Carbon tetrachloride		0.31	0.70		1.87			1.05
Trichloroethene			1.13		0.98		0.03	0.77
Toluene	1.20	1.08	1.34	1.08	1.11	1.09	0.54	1.11
Tetrachloroethene	0.94	1.12	1.39	1.07	0.95		0.78	1.07
Ethylbenzene	1.46	1.56	1.82	1.49	1.37	1.03	0.68	1.42
m/p-Xylene	0.81	1.15	1.76	1.29	1.35	1.16	0.76	1.26
o-Xylene	0.46	0.51	0.70	0.66	0.81	0.69	0.38	0.63
Mean	0.88	0.81	1.13	1.12	1.12	0.95	0.52	0.89

Table 4.12

Absolute percent difference between mean tenax and canister values

Compound	1988				1989			Mean
	1st Q	2nd Q	3rd Q	4th Q	1st Q	2nd Q	3rd Q	
Hexane		0.53	0.01	0.34	0.04	0.23	0.30	0.24
1,1,1-Trichloroethane	0.10	0.61	0.42	0.22	0.36	0.24	0.80	0.39
Benzene	0.22	0.29	0.16	0.22	0.19	0.31	0.40	0.26
Carbon tetrachloride		0.69	0.30		0.87			0.62
Trichloroethene			0.13		0.02		0.97	0.37
Toluene	0.20	0.08	0.34	0.08	0.11	0.09	0.46	0.20
Tetrachloroethene	0.06	0.12	0.39	0.07	0.05		0.22	0.15
Ethylbenzene	0.46	0.56	0.82	0.49	0.37	0.03	0.32	0.44
m/p-Xylene	0.19	0.15	0.76	0.29	0.35	0.16	0.24	0.31
o-Xylene	0.54	0.49	0.30	0.34	0.19	0.31	0.62	0.40
Mean	0.25	0.39	0.36	0.26	0.25	0.19	0.48	0.31

Table 5.1a

Agency: College of Staten Island

Table : Minimum Detection Limit (ppb)

	1DCE		1TCE		2DCE		2TCE		B		BF		C		CB		CT		EB		E		
M/Y	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	M/Y
JUL87			0.08	0.04	0.02	0.01			0.13	0.06			0.02	0.01	0.02	0.01	0.04	0.02			0.1	0.05	JUL87
AUG87			0.04	0.02	0.02	0.01			0.13	0.06			0.02	0.01	0.02	0.01	0.04	0.02			0.1	0.05	AUG87
SEP87			0.04	0.02	0.02	0.01			0.13	0.06			0.02	0.01	0.04	0.02	0.02	0.01			0.1	0.05	SEP87
OCT87			0.04	0.02	0.02	0.01			0.13	0.06			0.02	0.01	0.02	0.01	0.04	0.02			0.1	0.05	OCT87
NOV87			0.04	0.02	0.02	0.01			0.13	0.06			0.04	0.02	0.02	0.01	0.04	0.02			0.1	0.05	NOV87
DEC87			0.04	0.02	0.02	0.01			0.13	0.06			0.02	0.01	0.02	0.01	0.04	0.02	0.4	0.2	0.1	0.05	DEC87
JAN88	0.02	0.01	0.04	0.02	0.02	0.01			0.13	0.06			0.02	0.01	0.02	0.01	0.04	0.02	0.4	0.2	0.1	0.05	JAN88
FEB88			0.04	0.02	0.02	0.01			0.13	0.06			0.02	0.01	0.02	0.01	0.04	0.02	0.4	0.2	0.1	0.05	FEB88
MAR88	0.02	0.01	0.04	0.02	0.02	0.01			0.13	0.06			0.02	0.01	0.02	0.01	0.04	0.02	0.2	0.1	0.1	0.05	MAR88
APR88	0.02	0.01	0.04	0.02	0.02	0.01			0.13	0.06			0.02	0.01	0.02	0.01	0.04	0.02	0.2	0.1	0.1	0.05	APR88
MAY88	0.02	0.01	0.04	0.02	0.02	0.01			0.13	0.06			0.04	0.02	0.02	0.01	0.04	0.02	0.2	0.1	0.1	0.05	MAY88
JUN88	0.02	0.01	0.04	0.02	0.02	0.01			0.13	0.06			0.02	0.01	0.02	0.01	0.04	0.02	0.2	0.1	0.1	0.05	JUN88
JUL88			0.04	0.02	0.02	0.01			0.13	0.06	0.02	0.01	0.02	0.01	0.02	0.01	0.04	0.02	0.2	0.1	0.1	0.05	JUL88
AUG88	0.02	0.01	0.04	0.02	0.02	0.01			0.13	0.06	0.02	0.01	0.02	0.01	0.02	0.01	0.04	0.02	0.2	0.1	0.1	0.05	AUG88
SEP88			0.04	0.02	0.02	0.01			0.13	0.06	0.02	0.01	0.02	0.01	0.02	0.01	0.04	0.02	0.2	0.1	0.1	0.05	SEP88
OCT88	0.02	0.01	0.04	0.02	0.02	0.01	0.04	0.02	0.13	0.06	0.02	0.01	0.02	0.01	0.02	0.01	0.04	0.02	0.2	0.1	0.1	0.05	OCT88
NOV88	0.02	0.01	0.04	0.02	0.02	0.01	0.04	0.02	0.13	0.06	0.02	0.01	0.02	0.01	0.02	0.01	0.04	0.02	0.2	0.1	0.1	0.05	NOV88
DEC88	0.02	0.01	0.04	0.02	0.02	0.01	0.04	0.02	0.13	0.06	0.02	0.01	0.02	0.01	0.02	0.01	0.04	0.02	0.2	0.1	0.1	0.05	DEC88
JAN89	0.02	0.01	0.04	0.02	0.02	0.01	0.04	0.02	0.13	0.06	0.02	0.01	0.02	0.01	0.02	0.01	0.04	0.02	0.2	0.1	0.1	0.05	JAN89
FEB89	0.02	0.01	0.04	0.02	0.02	0.01	0.04	0.02	0.13	0.06	0.02	0.01	0.02	0.01	0.02	0.01	0.04	0.02	0.2	0.1	0.1	0.05	FEB89
MAR89	0.02	0.01	0.04	0.02	0.02	0.01	0.04	0.02	0.13	0.06	0.02	0.01	0.02	0.01	0.02	0.01	0.04	0.02	0.2	0.1	0.1	0.05	MAR89
APR89	0.02	0.01	0.04	0.02	0.02	0.01	0.04	0.02	0.13	0.06	0.02	0.01	0.02	0.01	0.02	0.01	0.04	0.02	0.2	0.1	0.1	0.05	APR89
MAY89	0.02	0.01	0.04	0.02	0.02	0.01	0.04	0.02	0.13	0.06	0.02	0.01	0.02	0.01	0.02	0.01	0.04	0.02	0.2	0.1	0.1	0.05	MAY89
JUN89																							JUN89
JUL89																							JUL89
AUG89	0.02	0.01	0.04	0.02	0.02	0.01	0.04	0.02	0.13	0.06	0.02	0.01	0.02	0.01	0.02	0.01	0.04	0.02	0.2	0.1	0.1	0.05	AUG89
SEP89	0.02	0.01	0.04	0.02	0.02	0.01	0.04	0.02	0.13	0.06	0.02	0.01	0.02	0.01	0.02	0.01	0.04	0.02	0.2	0.1	0.1	0.05	SEP89
OCT89																							OCT89
NOV89	0.02	0.01	0.04	0.02	0.02	0.01	0.04	0.02	0.13	0.06	0.02	0.01	0.02	0.01	0.02	0.01	0.04	0.02	0.2	0.1	0.1	0.05	NOV89
DEC89	0.02	0.01	0.04	0.02	0.02	0.01	0.04	0.02	0.13	0.06	0.02	0.01	0.02	0.01	0.02	0.01	0.04	0.02	0.2	0.1	0.1	0.05	DEC89
JAN90	0.02	0.01	0.04	0.02	0.02	0.01	0.04	0.02	0.13	0.06	0.02	0.01	0.02	0.01	0.02	0.01	0.04	0.02	0.2	0.1	0.1	0.05	JAN90
FEB90	0.02	0.01	0.04	0.02	0.02	0.01	0.04	0.02	0.13	0.06	0.02	0.01	0.02	0.01	0.02	0.01	0.04	0.02	0.2	0.1	0.1	0.05	FEB90
MAR90	0.02	0.01	0.04	0.02	0.02	0.01	0.04	0.02	0.13	0.06	0.02	0.01	0.02	0.01	0.02	0.01	0.04	0.02	0.2	0.1	0.1	0.05	MAR90
M/Y	1DCE		1TCE		2DCE		2TCE		B		BF		C		CB		CT		EB		E		M/Y
	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	

Table 5.1b

Agency: College of Staten Island

Table : Minimum Detection Limit (ppb)

	MC		MDCB		MPI		ODCB		OI		PDCB		S		T		T4CB		TCB		
M/T	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	M/T
JUL87									0.2	0.1					0.4	0.2	0.05	0.03	0.02	0.01	JUL87
AUG87									0.2	0.1					0.4	0.2	0.05	0.03	0.04	0.02	AUG87
SEP87									0.2	0.1					0.4	0.2	0.05	0.03	0.04	0.02	SEP87
OCT87	1.3	0.6							0.2	0.1					0.4	0.2	0.05	0.03	0.04	0.02	OCT87
NOV87	1.3	0.6							0.2	0.1					0.4	0.2	0.05	0.03	0.04	0.02	NOV87
DEC87	1.3	0.6							0.2	0.1					0.4	0.2	0.05	0.03	0.04	0.02	DEC87
JAN88	1.3	0.6							0.2	0.1			0.03	0.015	0.4	0.2	0.05	0.03	0.02	0.01	JAN88
FEB88	1.0	0.5			0.4	0.2			0.2	0.1			0.03	0.015	0.4	0.2	0.05	0.03	0.04	0.02	FEB88
MAR88	1.0	0.5			0.4	0.2			0.1	0.05			0.03	0.015	0.4	0.2	0.05	0.03	0.04	0.02	MAR88
APR88	0.6	0.3			0.4	0.2			0.1	0.05			0.03	0.015	0.4	0.2	0.05	0.03	0.02	0.01	APR88
MAY88	1.3	0.6			0.4	0.2			0.1	0.05			0.03	0.015	0.4	0.2	0.05	0.03	0.02	0.01	MAY88
JUN88	0.4	0.2			0.4	0.2			0.1	0.05			0.03	0.015	0.4	0.2	0.05	0.03	0.02	0.01	JUN88
JUL88	0.4	0.2	0.02	0.01	0.4	0.2	0.02	0.01	0.2	0.1			0.06	0.03	0.4	0.2	0.05	0.03	0.02	0.01	JUL88
AUG88	0.4	0.2			0.4	0.2			0.2	0.1			0.03	0.015	0.4	0.2	0.05	0.03	0.01	0.01	AUG88
SEP88	0.4	0.2			0.4	0.2			0.2	0.1			0.03	0.015	0.4	0.2	0.05	0.03	0.02	0.01	SEP88
OCT88	0.4	0.2	0.02	0.01	0.4	0.2	0.02	0.01	0.2	0.1	0.4	0.2	0.03	0.015	0.4	0.2	0.05	0.03	0.02	0.01	OCT88
NOV88	0.4	0.2	0.02	0.01	0.4	0.2	0.02	0.01	0.1	0.05	0.4	0.2	0.03	0.015	0.4	0.2	0.05	0.03	0.02	0.01	NOV88
DEC88	0.4	0.2	0.02	0.01	0.8	0.4	0.02	0.01	0.1	0.05	0.4	0.2	0.03	0.015	0.4	0.2	0.05	0.03	0.02	0.01	DEC88
JAN89	0.4	0.2	0.02	0.01	0.4	0.2	0.02	0.01	0.1	0.05	0.4	0.2	0.03	0.015	0.4	0.2	0.05	0.03	0.02	0.01	JAN89
FEB89	0.4	0.2	0.02	0.01	0.4	0.2	0.02	0.01	0.1	0.05	0.4	0.2	0.03	0.015	0.4	0.2	0.05	0.03	0.02	0.01	FEB89
MAR89	0.4	0.2	0.02	0.01	0.4	0.2	0.02	0.01	0.1	0.05	0.4	0.2	0.03	0.015	0.4	0.2	0.05	0.03	0.02	0.01	MAR89
APR89	0.4	0.2	0.02	0.01	0.4	0.2	0.02	0.01	0.1	0.05	0.4	0.2	0.03	0.015	0.4	0.2	0.05	0.03	0.02	0.01	APR89
MAY89	0.4	0.2	0.02	0.01	0.4	0.2	0.02	0.01	0.1	0.05	0.4	0.2	0.03	0.015	0.4	0.2	0.05	0.03	0.02	0.01	MAY89
JUN89																					JUN89
JUL89																					JUL89
AUG89	0.4	0.2	0.02	0.01	0.4	0.2	0.02	0.01	0.1	0.05	0.4	0.2	0.03	0.015	0.4	0.2	0.05	0.03	0.02	0.01	AUG89
SEP89	0.4	0.2	0.02	0.01	0.4	0.2	0.02	0.01	0.1	0.05	0.4	0.2	0.03	0.015	0.4	0.2	0.05	0.03	0.02	0.01	SEP89
OCT89																					OCT89
NOV89	0.4	0.2	0.02	0.01	0.4	0.2	0.02	0.01	0.1	0.05	0.4	0.2	0.03	0.015	0.4	0.2	0.05	0.03	0.02	0.01	NOV89
DEC89	0.4	0.2	0.02	0.01	0.4	0.2	0.02	0.01	0.1	0.05	0.4	0.2	0.03	0.015	0.4	0.2	0.05	0.03	0.02	0.01	DEC89
JAN90	0.4	0.2	0.02	0.01	0.4	0.2	0.02	0.01	0.1	0.05	0.4	0.2	0.03	0.015	0.4	0.2	0.05	0.03	0.02	0.01	JAN90
FEB90	0.4	0.2	0.02	0.01	0.4	0.2	0.02	0.01	0.1	0.05	0.4	0.2	0.03	0.015	0.4	0.2	0.05	0.03	0.02	0.01	FEB90
MAR90	0.4	0.2	0.02	0.01	0.4	0.2	0.02	0.01	0.1	0.05	0.4	0.2	0.03	0.015	0.4	0.2	0.05	0.03	0.02	0.01	MAR90

	MC		MDCB		MPI		ODCB		OI		PDCB		S		T		T4CB		TCB		
M/T	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	M/T

Table 5.2a

Agency: College of Staten Island

Table : Minimum Detection Limit (ng)
(Converted from ppb)

M/Y	1DCE	1TCE	2DCE	2TCE	B	BF	C	CB	CT	EB	E	M/Y
JUL87		5.25	0.97		4.99		1.15	1.11	3.02		4.24	JUL87
AUG87		2.63	0.97		4.99		1.15	1.11	3.02		4.24	AUG87
SEP87		2.63	0.97		4.99		1.15	2.21	1.51		4.24	SEP87
OCT87		2.63	0.97		4.99		1.15	1.11	3.02		4.24	OCT87
NOV87		2.63	0.97		4.99		2.29	1.11	3.02		4.24	NOV87
DEC87		2.63	0.97		4.99		1.15	1.11	3.02	20.88	4.24	DEC87
JAN88	0.97	2.63	0.97		4.99		1.15	1.11	3.02	20.88	4.24	JAN88
FEB88		2.63	0.97		4.99		1.15	1.11	3.02	20.88	4.24	FEB88
MAR88	0.97	2.63	0.97		4.99		1.15	1.11	3.02	10.44	4.24	MAR88
APR88	0.97	2.63	0.97		4.99		1.15	1.11	3.02	10.44	4.24	APR88
MAY88	0.97	2.63	0.97		4.99		2.29	1.11	3.02	10.44	4.24	MAY88
JUN88	0.97	2.63	0.97		4.99		1.15	1.11	3.02	10.44	4.24	JUN88
JUL88		2.63	0.97		4.99	2.49	1.15	1.11	3.02	10.44	4.24	JUL88
AUG88	0.97	2.63	0.97		4.99	2.49	1.15	1.11	3.02	10.44	4.24	AUG88
SEP88		2.63	0.97		4.99	2.49	1.15	1.11	3.02	10.44	4.24	SEP88
OCT88	0.97	2.63	0.97	2.63	4.99	2.49	1.15	1.11	3.02	10.44	4.24	OCT88
NOV88	0.97	2.63	0.97	2.63	4.99	2.49	1.15	1.11	3.02	10.44	4.24	NOV88
DEC88	0.97	2.63	0.97	2.63	4.99	2.49	1.15	1.11	3.02	10.44	4.24	DEC88
JAN89	0.97	2.63	0.97	2.63	4.99	2.49	1.15	1.11	3.02	10.44	4.24	JAN89
FEB89	0.97	2.63	0.97	2.63	4.99	2.49	1.15	1.11	3.02	10.44	4.24	FEB89
MAR89	0.97	2.63	0.97	2.63	4.99	2.49	1.15	1.11	3.02	10.44	4.24	MAR89
APR89	0.97	2.63	0.97	2.63	4.99	2.49	1.15	1.11	3.02	10.44	4.24	APR89
MAY89	0.97	2.63	0.97	2.63	4.99	2.49	1.15	1.11	3.02	10.44	4.24	MAY89
JUN89												JUN89
JUL89												JUL89
AUG89	0.97	2.63	0.97	2.63	4.99	2.49	1.15	1.11	3.02	10.44	4.24	AUG89
SEP89	0.97	2.63	0.97	2.63	4.99	2.49	1.15	1.11	3.02	10.44	4.24	SEP89
OCT89												OCT89
NOV89	0.97	2.63	0.97	2.63	4.99	2.49	1.15	1.11	3.02	10.44	4.24	NOV89
DEC89	0.97	2.63	0.97	2.63	4.99	2.49	1.15	1.11	3.02	10.44	4.24	DEC89
JAN90	0.97	2.63	0.97	2.63	4.99	2.49	1.15	1.11	3.02	10.44	4.24	JAN90
FEB90	0.97	2.63	0.97	2.63	4.99	2.49	1.15	1.11	3.02	10.44	4.24	FEB90
MAR90	0.97	2.63	0.97	2.63	4.99	2.49	1.15	1.11	3.02	10.44	4.24	MAR90

M/Y	1DCE		1TCE		2DCE		2TCE		B		BF		C		CB		CT		EB		E		M/Y
	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	LF	HF	

0

Table 5.2b

Agency: College of Staten Island

Table : Minimum Detection Limit (ng)
(Converted from ppb)

M/T	MC	MDCB	MPX	ODCB	OX	PDCB	S	T	T4CE	TCE	
JUL87					10.44			18.14	4.08	1.29	JUL87
AUG87					10.44			18.14	4.08	2.58	AUG87
SEP87					10.44			18.14	4.08	2.58	SEP87
OCT87	54.29				10.44			18.14	4.08	2.58	OCT87
NOV87	54.29				10.44			18.14	4.08	2.58	NOV87
DEC87	54.29				10.44			18.14	4.08	2.58	DEC87
JAN88	54.29				10.44		1.54	18.14	4.08	1.29	JAN88
FEB88	41.76		20.88		10.44		1.54	18.14	4.08	2.58	FEB88
MAR88	41.76		20.88		5.22		1.54	18.14	4.08	2.58	MAR88
APR88	25.06		20.88		5.22		1.54	18.14	4.08	1.29	APR88
MAY88	54.29		20.88		5.22		1.54	18.14	4.08	1.29	MAY88
JUN88	16.70		20.88		5.22		1.54	18.14	4.08	1.29	JUN88
JUL88	16.70	1.45	20.88	1.45	10.44		3.07	18.14	4.08	1.29	JUL88
AUG88	16.70		20.88		10.44		1.54	18.14	4.08	1.29	AUG88
SEP88	16.70		20.88		10.44		1.54	18.14	4.08	1.29	SEP88
OCT88	16.70	1.45	20.88	1.45	10.44	28.94	1.54	18.14	4.08	1.29	OCT88
NOV88	16.70	1.45	20.88	1.45	5.22	28.94	1.54	18.14	4.08	1.29	NOV88
DEC88	16.70	1.45	41.76	1.45	5.22	28.94	1.54	18.14	4.08	1.29	DEC88
JAN89	16.70	1.45	20.88	1.45	5.22	28.94	1.54	18.14	4.08	1.29	JAN89
FEB89	16.70	1.45	20.88	1.45	5.22	28.94	1.54	18.14	4.08	1.29	FEB89
MAR89	16.70	1.45	20.88	1.45	5.22	28.94	1.54	18.14	4.08	1.29	MAR89
APR89	16.70	1.45	20.88	1.45	5.22	28.94	1.54	18.14	4.08	1.29	APR89
MAY89	16.70	1.45	20.88	1.45	5.22	28.94	1.54	18.14	4.08	1.29	MAY89
JUN89											JUN89
JUL89											JUL89
AUG89	16.70	1.45	20.88	1.45	5.22	28.94	1.54	18.14	4.08	1.29	AUG89
SEP89	16.70	1.45	20.88	1.45	5.22	28.94	1.54	18.14	4.08	1.29	SEP89
OCT89											OCT89
NOV89	16.70	1.45	20.88	1.45	5.22	28.94	1.54	18.14	4.08	1.29	NOV89
DEC89	16.70	1.45	20.88	1.45	5.22	28.94	1.54	18.14	4.08	1.29	DEC89
JAN90	16.70	1.45	20.88	1.45	5.22	28.94	1.54	18.14	4.08	1.29	JAN90
FEB90	16.70	1.45	20.88	1.45	5.22	28.94	1.54	18.14	4.08	1.29	FEB90
MAR90	16.70	1.45	20.88	1.45	5.22	28.94	1.54	18.14	4.08	1.29	MAR90

	MC		MDCB		MPX		ODCB		OX		PDCB		S		T		T4CE		TCE		
M/T	LP	HP	LP	HP	LP	HP	LP	HP	LP	HP	LP	HP	LP	HP	LP	HP	LP	HP	LP	HP	M/T

Table 6.1

Proficiency Test Results for CES/CSI (reported value/spiked value for compound)

Testbnd → Test cmpd	Conc.	ENSL Aug 87	ENSL Nov 87	ELAP Jan 88	ELAP Jul 88	ENSL Jul 88	ELAP Jan 89	ENSL Mar 89	ELAP Jul 89	ELAP Jan 90	Total Mean	Total S	Mean Low	Mean High	Mean ELAP	Mean ENSL
Benzene	low	1.04	0.92			0.85		1.02		1.04	0.97	0.06	0.97		1.04	0.96
	high	0.89	0.92			0.54		1.07		1.04	0.89	0.19		0.89	1.04	0.85
Bromoform	low		0.89				1.26		1.16		1.10	0.16	1.10		1.20	0.89
	high		0.81				1.03		1.02		0.96	0.10		0.96	1.03	0.81
Carbon Tetra-	low	0.82	1.04			0.87		0.86		1.10	0.98	0.15	0.98		1.10	0.86
chloride	high	0.66	1.02			0.47		0.78		1.10	0.87	0.24		0.87	1.10	0.79
Chloro-	low	0.87	0.90	1.02	1.01	0.89		0.91			0.93	0.06	0.93		1.01	0.89
benzene	high	0.73	0.78	0.97	0.92	0.84		0.76			0.83	0.09		0.83	0.96	0.78
Chloroform	low	1.08	1.35			1.30		0.99		1.02	1.29	0.20	1.29		1.02	1.30
	high	1.30	1.31			1.15		0.96		1.03	1.27	0.24		1.27	1.03	1.23
1,2-Dichloro-	low									1.10	1.10	0.00	1.10		1.10	
benzene (o)	high									1.00	1.00	0.00		1.00	1.00	
1,3-Dichloro-	low						1.14		1.10		1.14	0.01	1.14		1.14	
benzene (m)	high						1.00		0.90		0.99	0.01		0.99	0.99	
1,4-Dichloro-	low						1.28 s		1.15		1.21	0.06	1.21		1.21	
benzene (p)	high						1.07		0.99		1.03	0.04		1.03	1.03	
1,1-Dichloro-	low			1.32 s	0.99						1.16	0.17	1.16		1.16	
ethane.	high			1.17	1.00						1.09	0.06		1.09	1.09	
1,2-Dichloro-	low	1.24	0.92	1.03	1.31	1.29		0.99			1.13	0.16	1.13		1.20	1.09
ethane	high	1.13	1.11	0.81	1.03	1.15		0.94			1.03	0.12		1.03	0.92	1.04
Ethyl	low		(3.05)	1.24	1.08	1.30		1.43		1.10	1.23	0.13	1.23		1.14	1.37
benzene	high		(2.76)	1.09	1.03	1.29		1.26		1.10	1.15	0.09		1.15	1.07	1.26
Methylene	low									0.89	0.89	0.00	0.89		0.89	
chloride	high									1.00	1.05	0.00		1.05	1.05	
Tetrachloro-	low	1.19	1.15	1.23	1.12	1.43		1.00			1.19	0.13	1.19		1.17	1.19
ethene	high	1.07	1.08	1.06	1.06	1.33		0.87			1.08	0.13		1.08	1.06	1.09
Toluene	low		1.20			1.18	1.34 ss	1.11	1.16	1.15	1.19	0.07	1.19		1.22	1.16
	high		1.28			1.14	1.16	1.03	0.97	1.07	1.11	0.10		1.11	1.07	1.15
111Trichloro	low		1.17			0.99		0.81			0.99	0.15	0.99			0.99
ethane	high		1.19			0.97		0.78			0.98	0.17		0.98		0.98
Trichloro-	low	1.03	0.99			0.75	1.24 s	0.88	1.37 s		1.06	0.23	1.06		1.30	0.91
ethene	high	0.92	1.16			0.98	1.15	0.88	1.27 s		1.05	0.16		1.05	1.21	0.97
m/p-Xylene	low			1.15	1.12		1.29 s		1.17		1.18	0.06	1.18		1.16	
	high			1.02	1.08		1.18		0.99		1.06	0.07		1.06	1.06	
o-Xylene	low	0.83	0.89			0.89		0.75			0.79	0.08	0.79			0.79
	high	0.88	0.78			0.71		0.70			0.76	0.06		0.76		0.78
Mean		1.04	1.07	1.09	1.07	0.99	1.18	0.94	1.12	1.07	1.05		1.06	1.01	1.09	1.01
S		0.25	0.24	0.13	0.11	0.27	0.11	0.17	0.12	0.07	0.13		0.14	0.12	0.10	0.10

@ ENSL = Environmental Monitoring Systems Laboratory, U.S. Environmental Protection Agency
ELAP = New York State Health Department, Environmental Laboratory Approval Program
s between 2 and 3 S from mean ss beyond 3 S

Table 6.2a

Headspace Results for CES/CSI Relative to Values Reported by Others (CSI value/value for compound reported by other group)

Test compound		Flow	Oct 87 20	Oct 87 21	Oct 87 22	Jul 88 25	Jul 88 26	Jul 88 27	Jul 88 28	Sep 89 88 1	Sep 89 88 15	Sep 89 88 19	Sep 89 88 25	Mean	S	Overall mean & S
Benzene	DEC	low		0.858	0.564	5.200	0.857	0.625	0.848	0.569	0.298	0.189	0.757	1.010	1.414	
		high		0.878	0.564									0.721	0.157	0.987
	NJIT	low	1.596	2.667	1.400	0.852		0.398	0.578	0.838	0.500	0.647	2.178	1.165	0.732	
		high	1.766	2.000	1.240									1.669	0.518	
	CNN					0.658	1.103	0.406	0.557	0.285	0.179	0.200	0.669	0.532	0.309	
	RTP	low	0.949	1.000		0.531	0.889							0.842	0.184	0.885
		high	1.107	1.579										1.340	0.233	
Carbon Tetra-chloride	DEC	low				2.714	0.667	0.800	1.000					1.295	0.828	
		high														0.740
	NJIT	low	1.625	1.111	0.667	1.000		0.308	0.125					0.806	0.505	
		high	0.556	0.279	0.591									0.475	0.140	
	CNN					0.633	0.667	0.167	0.139					0.401	0.249	
	RTP	low	0.813	0.769										0.791	0.022	0.538
		high	0.667	0.767		0.543	0.421							0.599	0.130	
Chloroform	DEC	low				0.333	0.038	0.333	0.286					0.248	0.122	
		high	0.287	1.124										0.665	0.459	0.314
	NJIT	low	0.881		0.178	0.667		0.500	0.500					0.385	0.220	
		high	0.843		0.225									0.134	0.091	
	CNN					0.105	0.100	0.286	0.333					0.206	0.105	
	RTP	low														0.264
		high														
Dichloro-methane	DEC	low		0.811			0.518		0.977	0.444	0.417	0.118	0.278	0.498	0.251	
		high		0.195										0.195	.000	1.182
(methylene chloride)	NJIT	low			0.891				0.265	5.000	5.000	5.000	5.000	3.389	2.288	
		high														
	CNN						0.477		0.315	0.500	0.200	0.071	0.143	0.284	0.162	
	CNN						0.729		0.377					0.553	0.176	
	RTP	low	0.188	0.266										0.227	0.039	1.769
		high	0.221	0.585			4.300							1.702	1.849	
Ethyl benzene	DEC	low				2.571	3.429	4.100	3.577					3.419	0.549	
		high														2.146
	CNN					0.783	2.057	1.541	2.793					1.793	0.734	
	CNN					0.783	1.895	1.783	2.325					1.696	0.565	
	RTP	low														1.010
		high				1.000	1.412							1.206	0.206	

Table 6.2b

Shootout Results for CES/CSI Relative to Values Reported by Others CSI value/value for compound reported by other group)

Test compound		Flow	Oct 87 20	Oct 87 21	Oct 87 22	Jul 88 I 25	Jul 88 I 26	Jul 88 I 27	Jul 88 I 28	Sep 88 II 1	Sep 88 II 15	Sep 88 II 19	Sep 88 II 25	Mean	S	Overall mean & S
Tetrachloro-ethene	DEC	low		1.167	1.329	2.000	1.636	1.300	2.136	0.100	1.000	1.150	1.283	1.330	0.851	
		high		1.090	1.250									1.170	0.080	1.964
	MJET	low	5.800	4.667	1.091	1.297		0.944	1.469	0.167	0.477		19.250	3.874	5.719	
		high	1.167	1.000	1.000									2.036	1.499	
	CMM					1.188	1.429	0.904	2.350	0.250	0.306	0.821	3.050	1.397	1.111	
	GMM					1.110	1.390	0.850	1.660					1.253	0.309	
	RTP	low				1.043	0.747							0.895	0.148	3.073
Toluene	DEC	low		1.905	1.626	2.670	1.264	1.647	1.651	0.571	0.542	0.561	0.711	1.315	0.670	
		high		1.791	1.858									1.825	0.834	3.312
	MJET	low	0.811	9.524	2.128	1.397	0.920	1.009		1.091	0.790		63.667	9.893	19.281	
		high	4.375	4.615	2.917									3.361	0.751	
	CMM					1.405	1.059	1.066	1.177	0.600	0.500	0.779	0.926	0.940	0.281	
	GMM					1.240	1.186	1.062	1.241					1.182	0.873	
	RTP	low	1.487	1.619										1.553	0.066	9.681
		high	1.736	1.812		1.320	1.052							1.502	0.351	
111Trichloro-ethane	DEC	low		0.764	0.769	0.895	1.039	1.040	0.854	0.221	0.143	0.143	0.398	0.627	0.346	
		high		0.620	0.667									0.643	0.823	0.783
	MJET	low	1.824	4.857	3.286	0.810		0.759	0.719	0.441	0.256	0.431	1.000	1.438	1.423	
		high	0.750	1.107	0.625									0.827	0.284	
	CMM					0.659	0.996	0.576	0.820	0.107	0.071	0.104	0.194	0.441	0.349	
	GMM					0.425	0.803	0.524	0.774					0.631	0.161	
	RTP	low	0.984	0.680										0.617	0.869	0.817
		high	0.500	0.721		0.447	0.318							0.495	0.148	
Trichloro-ethene	DEC	low				2.000	2.667	2.000	2.333					2.250	0.276	
		high														1.050
	MJET	low				0.667		0.500	1.167					0.778	0.288	
		high														
	GMM					0.083	0.889	0.100	0.538					0.403	0.335	
	RTP	low														0.863
		high				0.200	0.500							0.350	0.150	

Table 6.2c

Shootout Results for CDC/CSI Relative to Values Reported by Others (CSI value/value for compound reported by other group)

Test compound		Flow	Oct 87 28	Oct 87 31	Oct 87 32	Jul 88 § 25	Jul 88 § 26	Jul 88 § 27	Jul 88 § 28	Sep 84 15 1	Sep 84 15 15	Sep 84 15 29	Mean	S	Overall mean & S
m/p-Xylene	DEC	low				1.698	1.794	2.063	2.253	0.191	0.475	0.538	0.672	1.264	0.718	4.745
		high														
	NJIT	low				1.837		1.970	2.276	1.100	0.683		97.500	17.544	35.762	
		high														
	CRH					1.834	1.599	1.625	1.943	0.192	0.304	0.595	0.788	1.020	0.588	
	GHH					1.000	1.618	1.711	2.179					1.625	0.420	
	RTP	low														17.861
		high				1.475	1.277							1.376	0.099	
o-Xylene	DEC	low		0.938	0.850	1.111	1.020	1.000	1.163	0.206	0.375	0.407	0.945	0.739	0.320	
		high		0.849	0.647									0.745	0.098	1.078
	NJIT	low	2.108	5.714	0.857	1.429		0.833	1.163	0.157	0.400		9.000	2.411	2.799	
		high	2.500	1.826	0.536									1.621	0.815	
	CRH					0.446	0.592	0.541	0.682	0.045	0.079	0.229	0.375	0.374	0.221	
	GHH					0.435	0.642	0.645	0.877					0.650	0.150	
	RTP	low	0.488	0.816										0.652	0.164	1.530
		high	0.714	0.857		0.714	0.433							0.680	0.154	
		Mean	1.667	1.768	1.656	1.151	1.168	1.226	1.225	0.478	0.425	0.558	9.561			1.724
		S	1.097	1.864	0.789	0.662	0.690	0.759	0.695	1.012	1.004	1.119	23.004			5.541

& CSI divided by grand mean of all reported values for July 88 shootout
§ values for benzene and tetrachloroethylene interpolated from bar graphs, values for canisters include PEI & RTP
all values interpolated from bar graphs, values for canisters only for PEI, data for only 7 compounds provided by EPA

Table 6.3a

Shootout Mean Values for CES/CSI Relative to Values Reported by Others

Test compound		Flow	Oct 87 Lo & Hi Mean	Oct 87 Lo & Hi S	Oct 87 Combined Mean	Oct 87 Combined S	Jul 88 Combined Mean	Jul 88 Combined S	Sep 89* Combined Mean	Sep 89* Combined S	Overall Combined Mean	Overall Combined S	Means DEC	Means NJIT	Means CAN	Means GM	Means RTP
Benzene	DEC	low	0.47	0.09	0.60		1.68		0.41		0.98		0.98				
		high	0.72	0.16		0.18		1.32		0.21		1.30					
	NJIT	low	1.89	0.56	1.78		0.61		0.86		1.21			1.21			
		high	1.67	0.32		0.47		0.19		0.14		0.87					
	CAN						0.73		0.33		0.53				0.53		
								0.27		0.20		0.31					
	RTP	low	0.97	0.03	1.16		0.71				1.01						1.01
		high	1.34	0.23		0.25		0.18				0.31					
Carbon Tetra-chloride	DEC	low					1.30				1.30		1.30				
		high						0.83				0.83					
	NJIT	low	1.13	0.39	0.80		0.46				0.70			0.70			
		high	0.46	0.14		0.44		0.38				0.45					
	GM*						0.40				0.40					0.40	
								0.25				0.25					
	RTP	low	0.79	0.02	0.75						0.86						0.86
		high	0.72	0.05		0.05	0.48					0.14					
								0.08									
Chloroform	DEC	low			0.87		0.25				0.39		0.39				
		high	0.87	0.46		0.46		0.12				0.34					
	NJIT	low	0.13	0.05	0.13		0.56				0.31			0.31			
		high	0.13	0.09		0.07		0.08				0.22					
	GM*						0.21				0.21					0.21	
								0.10				0.10					
	RTP	low															
		high															
Dichloro-methane	DEC	low	0.31	0.00	0.25		0.76		0.31		0.41		0.41				
		high	0.19	0.00		0.06		0.23		0.13		0.25					
(methylene chloride)	NJIT	low	0.03	0.00	0.03		0.27		5.00		3.38			3.38			
		high				0.00		0.00		0.00		2.29					
	CAN						0.40		0.23		0.28				0.28		
								0.08		0.16		0.16					
	GM*						0.55				0.55					0.55	
								0.18				0.18					
	RTP	low	0.23	0.04	0.31		4.30				1.11						1.11
		high	0.40	0.18		0.16						1.60					

Table 6.3b

Shootout Mean Values for CES/CSI Relative to Values Reported by Others

Test compound		Flow	Oct 87 Lo & Hi Mean	S	Combined Mean	S	Jul 88 Combined Mean	S	Sep 88 Combined Mean	S	Overall Combined Mean	S	Means DEC	NJIT	CAN	GM	RTP
Ethyl benzene	DEC	low					3.42				3.42		3.42				
		high						0.55				0.55					
	CAN						1.79				1.79			1.79			
								0.73				0.73					
	GM*						1.70				1.70					1.70	
								0.57				0.57					
	RTP	low					1.21				1.21						1.21
		high						0.21				0.21					
Tetrachloro-ethene	DEC	low	1.24	0.08	1.21		1.82		0.88		1.30		1.30				
		high	1.17	0.08		0.09		0.31		0.48		0.51					
	NJIT	low	3.75	1.91	2.90		1.24		0.32		1.98			1.98			
		high	2.06	1.49		1.91		0.22		0.15		1.77					
	CAN						1.47		1.33		1.40				1.40		
								0.54		1.47		1.11					
	GM*						1.25				1.25					1.25	
								0.30				0.30					
	RTP	low					0.90				0.90						0.90
								0.15				0.15					
Toluene	DEC	low	1.77	0.14	1.80		1.81		0.80		1.40		1.40				
		high	1.82	0.03		0.11		0.52		0.07		0.85					
	NJIT	low	6.72	3.27	5.34		1.11		0.94		3.39			3.39			
		high	3.97	0.75		2.74		0.21		0.15		2.95					
	CAN						1.18		0.70		0.94				0.94		
								0.14		0.16		0.28					
	GM*						1.18				1.18					1.18	
								0.07				0.07					
	RTP	low	1.55	0.07	1.68						1.52						1.52
		high	1.81	0.08		0.15	1.19					0.27					
								0.14									
111Trichloro ethane	DEC	low	0.77	0.00	0.70		0.96		0.23		0.63		0.63				
		high	0.64	0.02		0.06		0.09		0.10		0.32					
	NJIT	low	3.32	1.24	2.07		0.76		0.37		1.32			1.32			
		high	0.83	0.20		1.53		0.04		0.09		1.33					
	CAN						0.76		0.12		0.44				0.44		
								0.16		0.05		0.34					
	GM*						0.63				0.63					0.63	
								0.16				0.16					
	RTP	low	0.62	0.06	0.61		0.38				0.54						0.54
		high	0.61	0.11		0.09		0.07				0.14					

Table 6.3c

Shootout Mean Values for CES/CSI Relative to Values Reported by Others

Test compound		Flow	Oct 87 Lo & Hi Mean	S	Oct 87 Combined Mean	S	Jul 86 Combined Mean	S	Sep 898 Combined Mean	S	Overall Combined Mean	S	Means DEC	NJIT	CAN	GH	RTP
Trichloro-ethene	DEC	low					2.25				2.25		2.25				
		high						0.28				0.28					
	NJIT	low					0.78				0.78			0.78			
		high						0.28				0.28					
	GH*						0.40				0.40					0.40	
								0.33				0.33					
	RTP	low					0.35										0.35
		high						0.15				0.15					
a/p-Xylene	DEC	low					1.98		0.57		1.27		1.27				
		high						0.23		0.07		0.72					
	NJIT	low					2.03		0.84		1.55			1.55			
		high						0.16		0.16		0.61					
	CAN						1.55		0.51		1.03				1.03		
								0.33		0.19		0.59					
	GH*						1.62				1.62					1.62	
								0.42				0.42					
	RTP	low									1.38						1.38
		high					1.38					0.10					
								0.10									
o-Xylene	DEC	low	0.74	0.19	0.74		1.07		0.40		0.74		0.74				
		high	0.74	0.10		0.15		0.07		0.09		0.29					
	NJIT	low	2.89	2.06	2.28		1.14		0.33		1.60			1.60			
		high	1.62	0.81		1.69		0.24		0.07		1.47					
	CAN						0.57		0.18		0.37				0.37		
								0.09		0.13		0.22					
	GH*						0.85				0.86					0.86	
								0.15				0.15					
	RTP	low	0.85	0.16	0.72		0.57				0.67						0.67
		high	0.79	0.07		0.14		0.14				0.16					
		Mean			1.51		1.14		0.75		1.15		1.28	1.64	0.71	0.86	0.93
		S				1.67		0.83		1.12		1.22	0.85	0.95	0.38	0.51	0.36

* CSI divided by grand mean of all reported values for July 86 shootout

@ NJIT values for 25 September 1989 omitted as obvious outlier due to desorption failure

Table 6.4. Comparison of mean winter (4th and 1st quarters) values over the two years of the study *

	Aromatics			Chlorinated HCs			Mean
	NJIT	DEC	CSI	NJIT	DEC	CSI	
Benzene	0.55	1.57	1.09				1.07
m/p-xylene	0.46	1.29	1.19				0.98
o-Xylene	0.59	1.26	1.21				1.02
Toluene	0.46	0.90	1.38				0.91
Carbon tetrachloride				1.20	0.50	0.81	0.83
Trichloroethylene				2.00	0.90	1.01	1.30
1,1,1-trichloroethane				0.18	1.42	1.03	0.88
Tetrachloroethylene				1.53	1.75	1.26	1.52
Chloroform				1.00	0.75	1.35	1.03
Mean	0.52	1.25	1.22	1.18	1.06	1.09	1.06
S	0.06	0.24	0.10	0.60	0.46	0.19	0.21

* (4Q87 + 1Q88)/(4Q88 + 1Q89)

Table 7.1a

Chronology of maintenance for ATD50, GC and MS 3rd Quarter 1987

Date	Helium	ATD50	Cold trap	ATD prog/ method	Tune	Mass spec	Fila- ments	Leaks	Comments
1Jul				5/1			broke		
3Jul			replace		auto		switch		
9Jul	new tank								
14Jul	*			6/1					*increase He pressure to increase flow rate
16Jul							change		open column to tighten liquid nitrogen line
17Jul						*			*high air background, tighten fittings
20Jul								*	*still high background, replace seal between GC & MS, pump down over night
21Jul				6/1	auto				
23Jul			prob.	6/2	auto				
24Jul to 28th	system down		*						*replaced cold trap, resolder wires in heat sink, apply new silicone heat sink compound to plates
29Jul				6/2	auto				
4Aug				6/2	auto				
19Aug			*	6/2		*			*failed to reach max temp. *shut off 22 min into sample
20Aug	*		replace						*replace moisture, O2, and HC traps on He line
21Aug	*								*water still high, replace charcoal in trap on helium line
24Aug					auto				
21Sep						*			*abnormal termination in 2nd, 4th & 5th traps
22Sep				3/2					
23Sep	new tank			6/2					

Table 7.1b

Chronology of maintenance for ATD50, GC and MS 4th Quarter 1987

Date	Helium	ATD50	Cold trap	ATD prog/ method	Tune	Mass spec	Fila- ments	Leaks	Comments
10ct				6/2					
20ct				7/2	twice				
90ct									EM at 2200V
130ct									EM at 2000V
140ct			*	7A/2					*resolder wires in CT
160ct				7/2	manual				
190ct				7/2					power failure & lost sample, fuse blown
210ct				7/2					
2Nov				7/2					
25Nov	*				auto				*switch the He tank and start at 1500 psi
									EM at 1800 eV
27Nov					manual				EM at 1800 eV, ion focus high >40
30Nov				3/1					
20ec				8/2		*			*retune, adjust ion focus, slit entrance and adjust amu effect
40ec		#				*			*adjust parameters, # ATD50 fuse blown
80ec									change MS pump oil
170ec									split GC/MS, and replace cryogenic valve
220ec									split GC/MS, check liq. nitrogen connection
230ec						*			*readjust tune
300ec									MS turned off, excessive pressure

Table 7.1c

Chronology of maintenance for ATD50, GC and MS 1st and 2nd Quarter 1988

Date	Helium	ATD50 Cold trap	ATD prog/ method	Tune	Mass spec	Filaments	Leaks	Comments
1st Q 1988								
6Jan	new tank							
8Jan								out of liq. Nit. and use dry ice
22Jan						#1 burn		
25Jan								MS turned off due to excess pressure. He flow rate too high
26Jan					x	#2 burn replace		xopen MS
27Jan					x			xair leak, open system, and adjust O-ring around sourc
29Jan			8/2					
8Feb		replace						split system to repair liq. Nit. valve
10Feb			8/2					
1Mar			8/1					
3Mar			8/1			#1 burn		
7Mar			8/2			use #2		
22Mar			8/1					
23Mar			8/2					
2nd Q 1988								
2Apr					x			xrough pump making noise
5Apr	new tank				x			xrough pump replaced
6Apr								replace Si seals on hot box and carriage
25May								resolder wires in cold traps
21Jun								tighten clips on ATD50 traps

Table 7.1d

Chronology of maintenance for ATD50, GC and MS 3rd Quarter 1988

Date	Helium	ATD50 Cold trap	ATD prog/ method	Tune	Mass spec	Fila- ments	Leaks	Comments
3rd Q 1988								
8Jul	new tank							change Oxygen trap
13Jul	*			x				xreadjust aau gain and offset, ant lens, ion focus and EM at 2200. * He flow rate adjusted
19Jul								change Si seal on hot box
9Aug		replace						put 2nd moisture trap on He line
16Aug	new tank							remove 2nd moisture trap (Supelco), reset purge flow to 80 cc/m, 28 pks & 900 ct on Btune, drop pressure to 34.5-35 psi and tank at 2720 psi.
17Aug								tank at 2620 psi, lower purge to 50 cc/min
7Sep	x							xreplace oxygen trap on line
9Sep	new tank							
14Sep	new tank				x			MS high water peek, put on new He tank. MS shut off - excessive pressure separated, capped column, no voltage in MS, replaced filament, cleaned source
20Sep					x			replace floppy disk drive

Table 7.1e

Chronology of maintenance for ATD50, GC and MS 4th Quarter 1988

Date	Helium	ATD50	Cold trap	ATD prog/ method	Tune	Mass spec	Fila- ments	Leaks	Comments
6Oct									high air background, leak in moisture trap-replaced
7Oct									replace Si O-ring on hot box
10Oct							burn		
14Oct			icing up						
18Oct		*							*replace fuses in ATD50
26Oct		*	replace						*ATD50 hot box not functioning
28Oct		*							*replace fuse
4Nov				8/2		*			*air background started high , and then dropped dur day
10Nov	new tank								start tank at 2000 psi
11Nov		*							*hot box fuse blown and replaced
2Dec		*							*change Si O-ring on hot box
14Dec									cryogenic valve making noise
21Dec			replace				replace		
22Dec			tighten						replace cryogenic valve and change pump
23Dec			replace						
25Dec									column broke at MS end, reinsert, rough pump does not start
27Dec							use #1		got rough pump started by moving rotor, replace moisture trap
28Dec							use #2		

Table 7.1f

Chronology of maintenance for ATD50, GC and MS 1st Quarter 1989

Date	Helium	ATD50	Cold trap	ATD prog/ method	Tune	Mass spec	Fila- ments	Leaks	Comments
23Jan			x						xgas leak at inlet of trap - replace Si O-ring, cold trap not reaching -30 C, replace heat sink compound top plate
31Jan			x						xCT not reaching high temp, remove resistance (ice)
1Feb				8/2					
28Feb		x							HF9
2Mar		x							HF9
3Mar									replace Si O-ring on ATD50 oven
6Mar		x							HF9, caused by metal flange on heated valve, sensor should rotate in about 1 second
10Mar			replace						change transfer line between ATD50 and GC
11Mar						x			replace rough pump, install ion gauge
15Mar									connect transfer line and column with new union from J & W
17Mar	new tank								
22Mar									tune shows other compounds present, raised GC temperature to 220 degrees C for several minutes

Table 7.1g

Chronology of maintenance for ATD50, GC and MS 2nd, 3rd and 4th Quarters 1989

Date	Helium	ATD50	Cold trap	ATD prog/ method	Tune	Mass spec	File- ments	Leaks	Comments
2nd Q 1989									
4Apr						x			background air count very high from column bleed, unable to run, heated column for several hours at 225 C, and over night at 150 C
5Apr		0/2			manual				small column bleed in 240/260
15Apr									Dr. Weisel leaves for UMDNJ
16Apr									Dr. Oppenheimer takes over
18May			x						xcold trap blows fuses
19May									both MDL 8 amp and fuses blown, cold trap cannot reach high temp., replace fuses with new BUSS MDL8
27May									prior to run raised temp to 250 C for 1 hr
29May					x				xunable to do Btune, sensitivity too low
28Jun									Dr. Zha takes over
30Jun									replace GC column, ion source cleaned retention times shifted, and thus computer program for indentifying compounds has to be adjusted, ATD50 blowing fuses when CT fires
3st Q 1989									
6Jul									raise GC temp. to 275 C to remove H20
11Jul to 30th									system down due to loss of system volume on workstation 2, version 3.1 software installed by HP
20Jul									cold trap and spacer nuts replaced, sensitivity for higher masses lost, autotune fails, suspect dirty source
7Aug									EM voltage changed from 2200 to 1800, change ATDQUANB threshold from 50 to 700
8Aug							x		xfilament damage suspected
9Aug									EM change back to 2200
15Aug							change		
18Aug new tank									
20Aug to 5Sep			change						MS shut down, 3 circuit boards replaced in MS
18Sep to 50ct		x							xATD50 shut down with HF4 signal
4th Q 1989									
50ct		x							xfinally fixed by replacing #10 relay
60ct		x						x	xtransfer line cracked, replaced
70ct		x						x	xfix leak in hot box
80ct									start to analyze mid September samples
100ct									complete analysis of September samples

Table 7.2

Number and date or name of calibration curves by quarter*

Weisel -->						¦ Zha -->		
3Q87	4Q87	1Q88	2Q88	3Q88	4Q88	1Q89	2Q89	3Q89
70387	100287	11888	4 588	70888	101188	11789	62889	81189
71087	100587	12888	MAY	70988	111288	20289		91289
72187	101287	20288		90288	111588	20389		
73087	101387	2 388			NOV	30289		
80487	102187	2 588			NOVEX	31689		
80787	103087	2 688			DECEX	32089		
81087	110487	21088			122888	3ave89		
81487	112487							
82187	112587							
82187	120287							
82487	120487							
82887	121187							
90487	121887							
90887	122387							
91587								
91787								
91887								
92787								
18	14	7	2	3	7	7	1	2

*Reduction related to shift from autotune to manual tune

Table 7.3a

Tune parameters and mass spectrometer condition (ATune file)
3rd and 4th Quarters, 1987

Date	Multi-plier	Amu gain	Amu off-set	Ion fo-cus	Ent lens	Repel-ler	X-ray	Mass gain	Mass offset	Air-H2O count	Thres-hold
30Jun	1800	154	69	0	50	10.2	64	-2	16	1064	5
3Jul		151			55		90			377	
7Jul							80			764	
14Jul		152					76	-4		304	
16Jul	1800	155	67		45		68	117	15	1916	
17Jul	2000	152	68				68	-9	16	4652	
21Jul	2000	151		4	55		88	-4		675	
23Jul			67	0	50		76	-7		563	
29Jul		152	68		60		92	-4		359	8
4Aug		150	67		50		80	4		581	
7Aug		151	68	4	60		88	-5	17	305	7
10Aug		152	67				80	-6		596	
21Aug				8	70			-1	16	2015	5
24Aug	2000	149	67	4	75			2		15188	
27Aug		154	66	0	60			-1		1275	
31Aug										2307	
04Sep					55		72			783	
15Sep		151			45		84	2	17	813	
ATune (auto)			BTune (manual tune with BFB to USEPA conditions)								
10ctA	2000	151	66	0	45	10.2	84	2	17	787	5
20ctA		149		4	70		68	4	16	2857	
50ctA		156		0			76	-8	17	405	
90ctA	2200	155		16	60			-1	16	22424	
130ctA	2000			4	65		72	2	17	585	
25NovA	1800	158		0	50			31	16	259	
27NovB	1600	157		52	55	8.8	52	44	15	19480	
30NovA	1800	158	69	0	50			31	16	323	
2DecB	1400	150	72	20	50	10.2	48	46	16	13301	5
4DecB	2200		73	25						1002	
8DecB	2000	151	71	22		8.4	60	38	15	602	
23DecB			72	0	62	10.2				168	

Table 7.3b

Tune parameters and mass spectrometer condition (B & A Tunes)
1st, 2nd, 3rd, and 4th Quarters, 1988

Date	Multi-plier	Amu gain	Amu off-set	Ion fo-cus	Ent lens	Repel-ler	X-ray	Mass gain	Mass offset	Air-H2O count	Thres-hold
11JanB	2000	151	72	0	62	10.2	60	38	15	677	5
14JanB	2200	158	68	4	55	10	84	-4	16	543	
18JanB	2000	151	72	0	62	10.2	60	38	18	195	
3FebB									15	333	
9FebB	2200	162	68	4	50		80	-3	16	533	
22FebA	2000	159	66	8	55		72	0		18208	
1MarA	1800	158	68	12	50		60	7	16	151	
3MarB	2000	151	72	0	62			38	15	168	
7MarA		158	66		55			-3	16	17672	
17MarA					50		76	-1	16	288	
19MarA	1800		68		45		60	-4	17	12088	
22MarA	1600			48	60		56	-6		16251	
31MarA	2000			16	45		88	2	16	162	
6AprA	2000	155	68	16	45	10.2	76	-1	16	180	5
12AprA		158		0			60	0		275	
12AprB		151	72		62			38	15	275	
18AprA	1800	158	68	16	50			-7	18	257	
25AprA				12			58	-6	17	182	
29AprA	2000				45		60	-4	16	294	
5MayB		151	72	0	62			38	15	169	
11MayA	1800	157	68	8	45		80	-6	18	156	
1JulB	2000	151	72	?	?	10.2	?	?	?	132	
13JulA	2200	154	88	16	48	10.2	88	-12	18	167	
14JulB		155		24	47					914	
6SepB										488	20
19SepB			69				68		16	650	20
6OctB	2200	155	69	24	47	10.2	68	-12	16	1038	20
29DecB										378	

Table 7.3c

Tune parameters and mass spectrometer condition (B & A Tunes)
1st, 2nd, and 3rd Quarters, 1989

Date	Multi-plier	Amu gain	Amu off-set	Ion fo-cus	Ent lens	Repel-ler	X-ray	Mass gain	Mass offset	Air-H2O count	Thres-hold
13JanB	2200	155	69	24	47	10.2	68	-12	16	399	20
19JanB		152								291	
23JanB										291	5
29MarB	2200	152	69	24	47	10.2	68	-12	16	304	5
1AprB	2200	152	69	24	47	10.2	68	-12	16	304	5
27May	system shut down, source needs cleaning										
New HP Software installed (Increase sensitivity 100x)											
2AugB	2200	148	70	8	65	10.2	88	20	15	1278	20
9AugB		145		22	55		104	-3		637	500
19AugB		150		28	65		64	-11		452	750
23AugB	2400			0	50		112	-465	11	850	
24AugB		152	71					-696	12	339	
25AugB	2200									600	
26AugB		159	68	10	55		88	-175	11	239	
29AugB	2400	150	70	4	45		84	-327	12	500	
30AugB								-235		373	
1SepB								-580		352	
6SepB		210	68		48		112	47	6	481	
8SepB	2600							53	4	450	
30OctB	2400	204	71	0	65	10.2	88	16	-5	670	
14NovB		212	68		55			18	-19	411	
15NovB		210	70		47			40	-17	318	
16NovB		203	71		37		84	61	-23	236	
1DecB	2000	196	68		40		92	3	-12	374	

Table 7.3d

Tune parameters and mass spectrometer condition (B & A Tunes)
Summary of quarterly means from 3rd quarter 87 to 4th quarter 89

Date	Multi-plier	Amu gain	Amu off-set	Ion fo-cus	Ent lens	Repel-ler	X-ray	Mass gain	Mass offset	Air-H2O count	Thres-hold
3q87	1920	152	67	3	56	10	80	6	16	1919	6
4q87	1889	154	71	12	56	10	66	19	16	5183	5
1q88	1960	156	69	10	54	10	70	9	16	5175	5
2q88	1900	155	70	9	52	10	67	7	16	224	5
3q88	2100	153	76	20	48	10	78	-12	17	470	20
4q88	2200	155	69	24	47	10	68	-12	16	708	20
1q89	2200	153	69	24	47	10	68	-12	16	321	10
2q89	2200	152	69	24	47	10	68	-12	16	304	5
3q89	2360	159	69	12	55	5	93	-216	10	546	318
4q89	2200	205	70	0	49	10	88	28	-15	402	

Table 7.4

Mean value for internal standards for a subset of samples from each quarter

Quarter	N	Stat.	Internal standards 1st*	2nd*	2nd/1st	S/Mean 1st	2nd
3q87#	10	Mean	214.6	111.9	0.52	0.16	0.73
		S	34.3	81.4			
4q87	10	Mean	237.5	141.1	0.59	0.18	0.28
		S	42.5	39.1			
1q88	19	Mean	263.1	90.6	0.34	0.18	0.35
		S	48.7	32.1			
2q88	23	Mean	204.3	74.4	0.36	0.18	0.51
		S	37.7	37.9			
3q88	24	Mean	232.0	41.6	0.18	0.30	0.40
		S	70.6	16.7			
4q88	32	Mean	206.9	31.1	0.15	0.19	0.17
		S	39.2	5.3			
1q89	20	Mean	197.8	41.7	0.21	0.19	0.22
		S	37.2	9.2			
2q89	16	Mean	192.3	102.4	0.53	0.26	0.41
		S	50.5	42.0			
3q89	28	Mean	164.8	85.9	0.52	0.26	0.58
		S	42.8	49.9			

* 4q87 to 1q89 adjusted to fit ratio of 1st to 2nd of 1:0.46
# without splitless valve, which was installed at start of next quarter

Table 8.1

Organization: College of Staten Island Sorbent: Tenax

Regression summary for January to March 1989

DISTRIBUTED VOLUME (Low-High Flow)

Compound name	df		Regression coefficient	Slope	t-Test on slope	Significance
Methylene chloride	226	*	0.788	0.558	29.368	p < 0.001
1,1-Dichloroethane	226	*	0.241	0.989	8.526	p < 0.001
Hexane	226		0.927	0.906	56.625	p < 0.001
Chloroform	225		0.626	0.549	19.607	p < 0.001
111-Trichloroethane	226		0.909	0.727	48.467	p < 0.001
1,2-Dichloroethane	226		0.472	0.451	14.548	p < 0.001
Benzene	223		0.941	1.071	63.000	p < 0.001
Carbon Tetrachloride	223		0.951	0.853	71.083	p < 0.001
Trichloroethene	224		0.934	1.006	59.176	p < 0.001
Toluene	223		0.968	1.039	86.583	p < 0.001
112-Trichloroethane	226	*	1.00	all at MDL		
Tetrachloroethene	221		0.951	0.896	68.923	p < 0.001
Chlorobenzene	220	*	0.541	0.823	16.137	p < 0.001
Ethylbenzene	217		0.963	0.952	79.333	p < 0.001
m/p Xylenes	217		0.968	1.013	84.417	p < 0.001
Bromoform	223	*	1.00	all at MDL		
Styrene	215		0.946	1.019	63.688	p < 0.001
o Xylene	215		0.952	1.043	69.533	p < 0.001
m Dichlorobenzene	217	*	0.294	0.323	9.500	p < 0.001
p Dichlorobenzene	215	*	0.127	0.054	6.000	p < 0.001
o Dichlorobenzene	215	*	0.118	0.318	5.390	p < 0.001

* most or all values at or below MDL

Table 8.2

Table 8.2

Regression summary for study

COMPARISON ACROSS CATEGORIES

Compound name		Duplicate Low		Duplicate High		1st Q 1989 Low-High		Canister - Tenax	
		df	R^2	df	R^2	df	R^2	df	R^2
Methylene chloride	*	25	0.81	24	0.81	226	0.79		
1,1-Dichloroethane	*	6	0.83	4	0.67	226	0.24		
Hexane		27	0.97	25	0.96	226	0.93	34	0.52
Chloroform		20	0.93	23	0.81	225	0.63		
111-Trichloroethane		27	0.95	25	0.76	226	0.91	40	0.57
1,2-Dichloroethane		15	0.99	24	1.00	226	0.47		
Benzene		27	0.95	25	0.98	223	0.94	63	0.53
Carbon Tetrachloride		27	0.95	25	0.95	223	0.95	3	0.07
Trichloroethene		26	0.89	25	0.92	224	0.93	1	0.99
Toluene		27	0.95	25	0.97	223	0.97	63	0.59
112-Trichloroethane	*	2		4	1.00	226			
Tetrachloroethene		26	0.96	27	1.00	221	0.95	24	0.91
Chlorobenzene	*	7	0.60	13	0.67	220	0.54		
Ethylbenzene		26	0.92	24	0.93	217	0.96	37	0.44
m/p Xylenes		26	0.94	24	0.95	217	0.97	58	0.68
Bromoform	*					223			
Styrene		26	0.86	23	0.96	215	0.95		
o Xylene		26	0.83	24	0.96	215	0.95	46	0.61
m Dichlorobenzene	*					215	0.13		
p Dichlorobenzene	*	22	0.46	20	0.71	217	0.29		
o Dichlorobenzene	*	3	0.98	9	0.96	215	0.12		
All compounds									
Mean		20.6	0.88	20.7	0.89	221.4	0.72	37	0.59
S		8.8	0.14	7.1	0.11	4.3	0.31	21	0.24
For measurable compounds only									
Mean		23.4	0.93	23.1	0.94	221.9	0.89	37	0.59
S		6.8	0.04	5.4	0.07	4.0	0.15	21	0.24

* most or all values at or below MDL

Table 8.3

Comparison of results from Blind Samples and Shootouts (CSI/Other)

Compound (in order of retention time)	Blind samples		Shootouts			CSI-PEI	Mean
	ELAP	EMSL	DEC	NJIT	CAN	CAN	
Methylene chloride	0.97		0.41	3.38	0.28		1.26
Chloroform	1.03	1.34	0.39	0.31			0.77
111Trichloroethane		0.99	0.63	1.32	0.44	0.59	0.79
Benzene	1.04	0.91	0.96	1.21	0.53	0.97	0.94
Carbon Tetrachloride	1.14	0.82	1.30	0.70		1.04	1.00
Trichloroethene	1.28	0.94	2.25	0.78		0.79	1.21
Toluene	1.14	1.15	1.40	3.39	0.94	1.10	1.52
Tetrachloroethene	1.12	1.14	1.30	1.98	1.40	1.07	1.33
Ethylbenzene	1.10	1.31	3.42		1.79	1.41	1.81
m/p-Xylene	1.12		1.27	1.55	1.03	1.25	1.24
o-Xylene		0.77	0.74	1.60	0.37	0.63	0.82

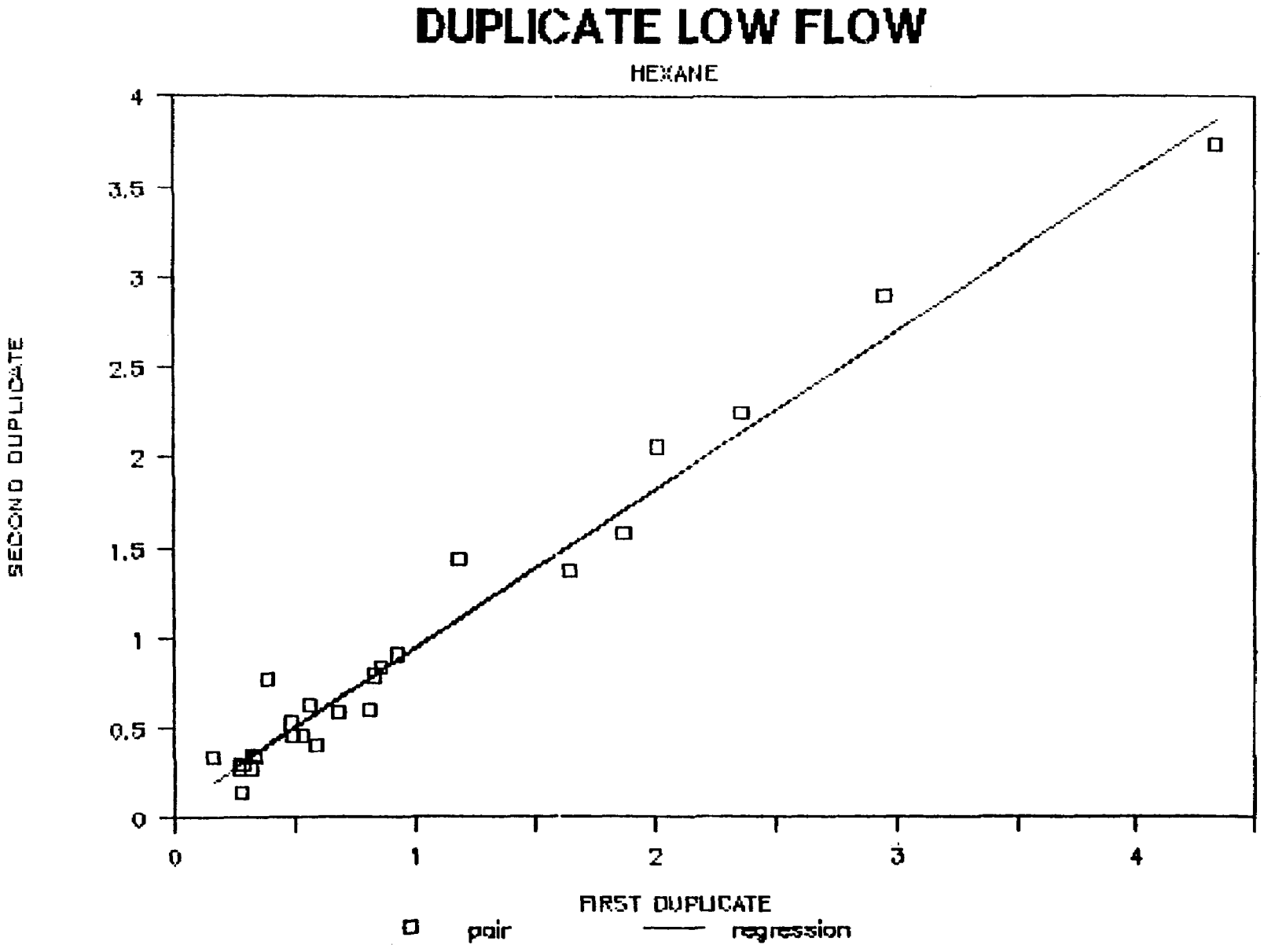

DUPLICATE LOW FLOW
HEXANE
SECOND DUPLICATE
FIRST DUPLICATE
0
0.5
1
1.5
2
2.5
3
3.5
4
pair
regression

Figure 2.1

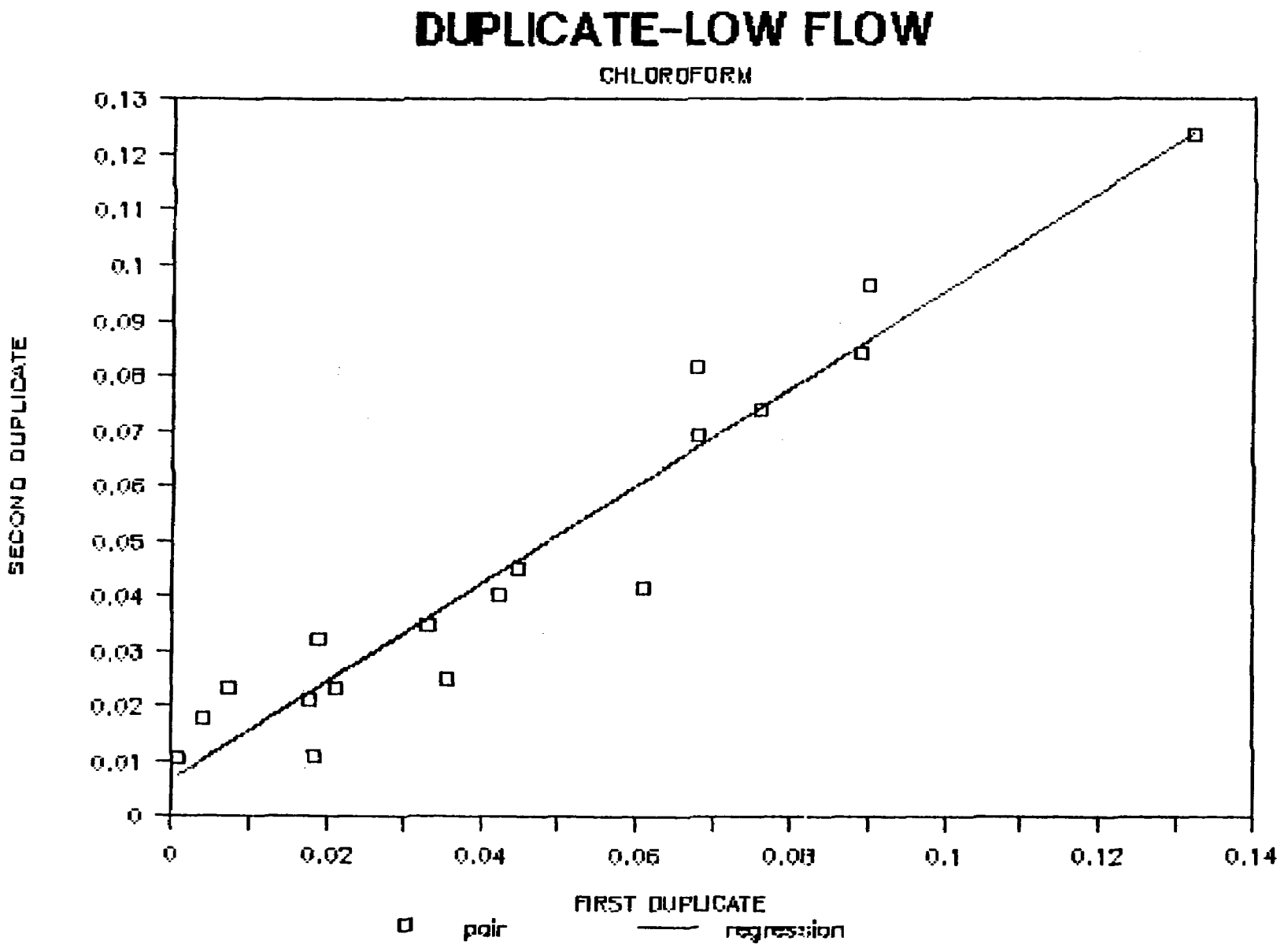

DUPLICATE-LOW FLOW
CHLOROFORM
SECOND DUPLICATE
0.13
0.12
0.11
0.1
0.09
0.08
0.07
0.06
0.05
0.04
0.03
0.02
0.01
0
0
0.02
0.04
0.06
0.08
0.1
0.12
0.14
FIRST DUPLICATE
pair
regression

Figure 2.2

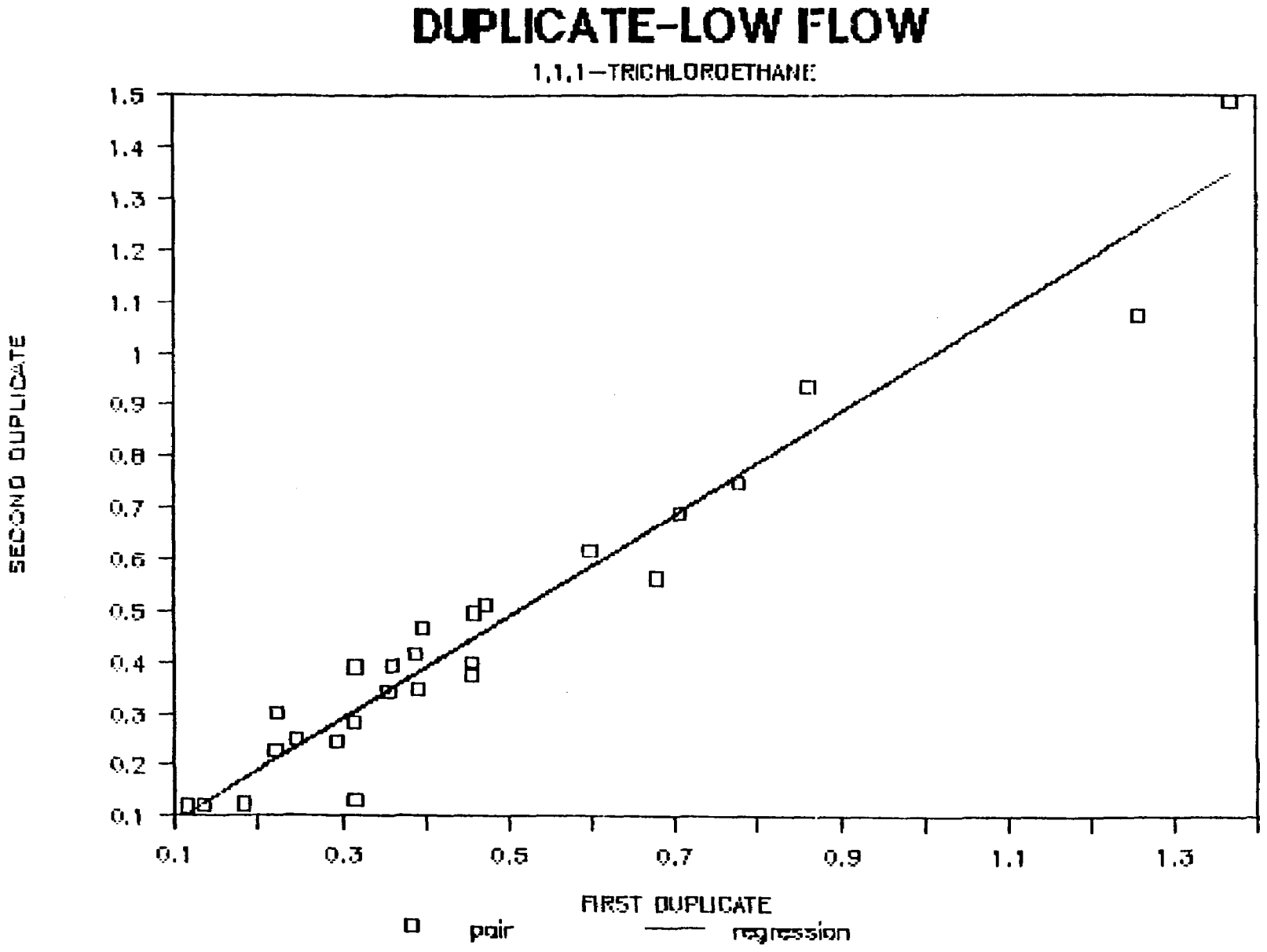

DUPLICATE-LOW FLOW
1,1,1-TRICHLOROETHANE
SECOND DUPLICATE
1.5
1.4
1.3
1.2
1.1
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0.1
0.3
0.5
0.7
0.9
1.1
1.3
FIRST DUPLICATE
pair
regression

Figure 2.3

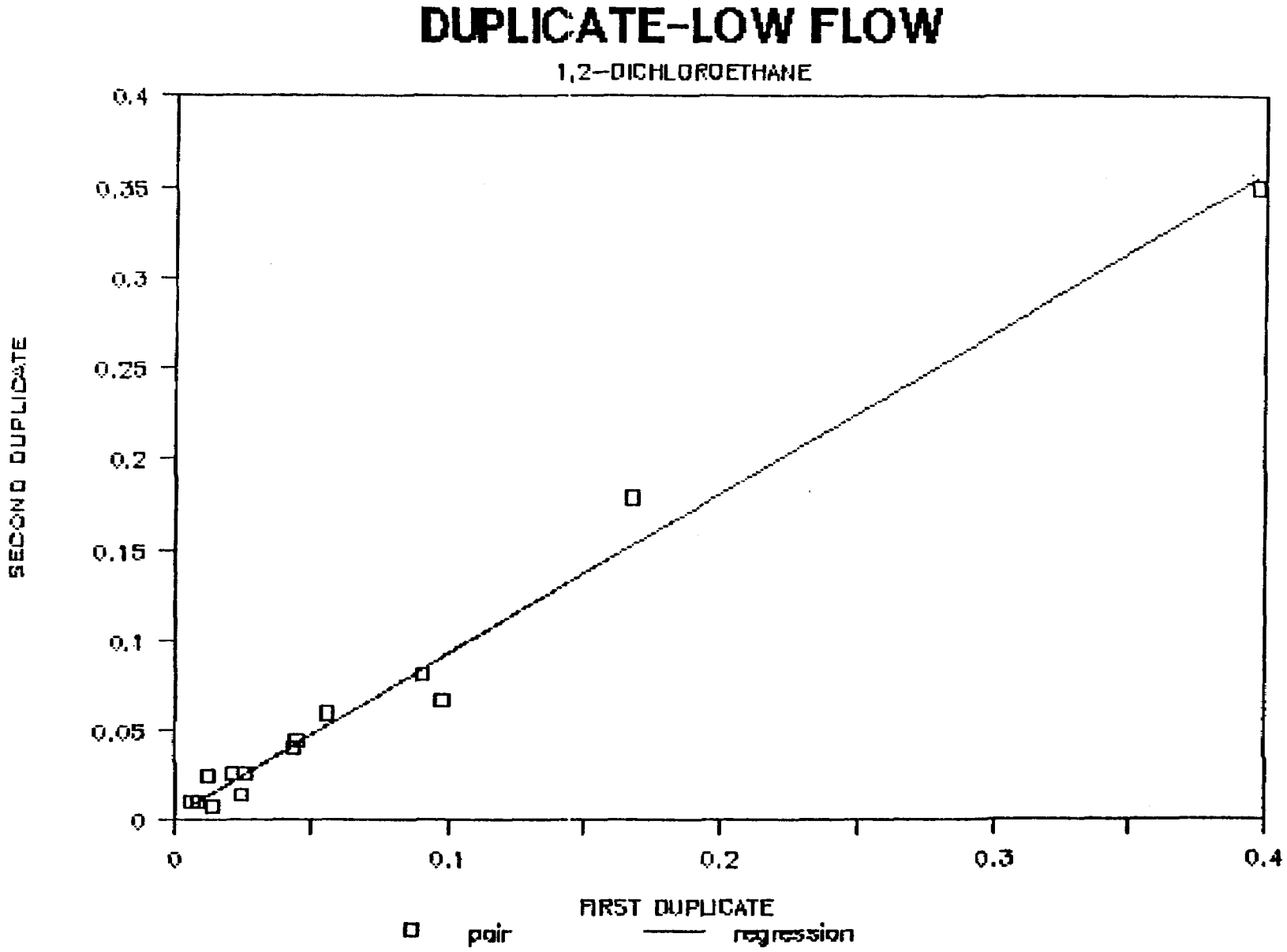

DUPLICATE-LOW FLOW
1,2-DICHLOROETHANE
SECOND DUPLICATE
0.4
0.35
0.3
0.25
0.2
0.15
0.1
0.05
0
0
0.1
0.2
0.3
0.4
FIRST DUPLICATE
pair
regression

Figure 2.4

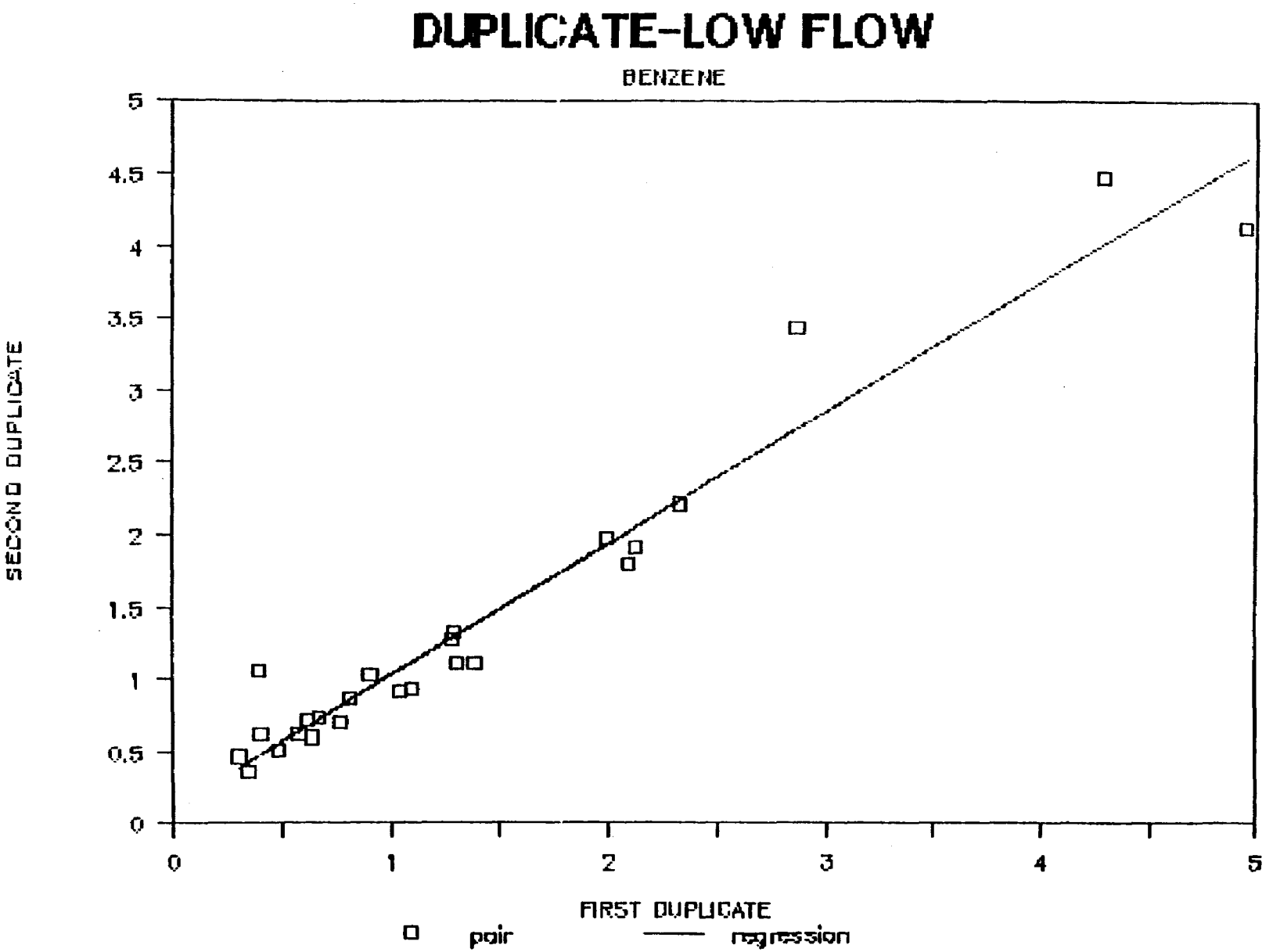

DUPLICATE-LOW FLOW
BENZENE
SECOND DUPLICATE
FIRST DUPLICATE
0
0.5
1
1.5
2
2.5
3
3.5
4
4.5
5
pair
regression

Figure 2.5

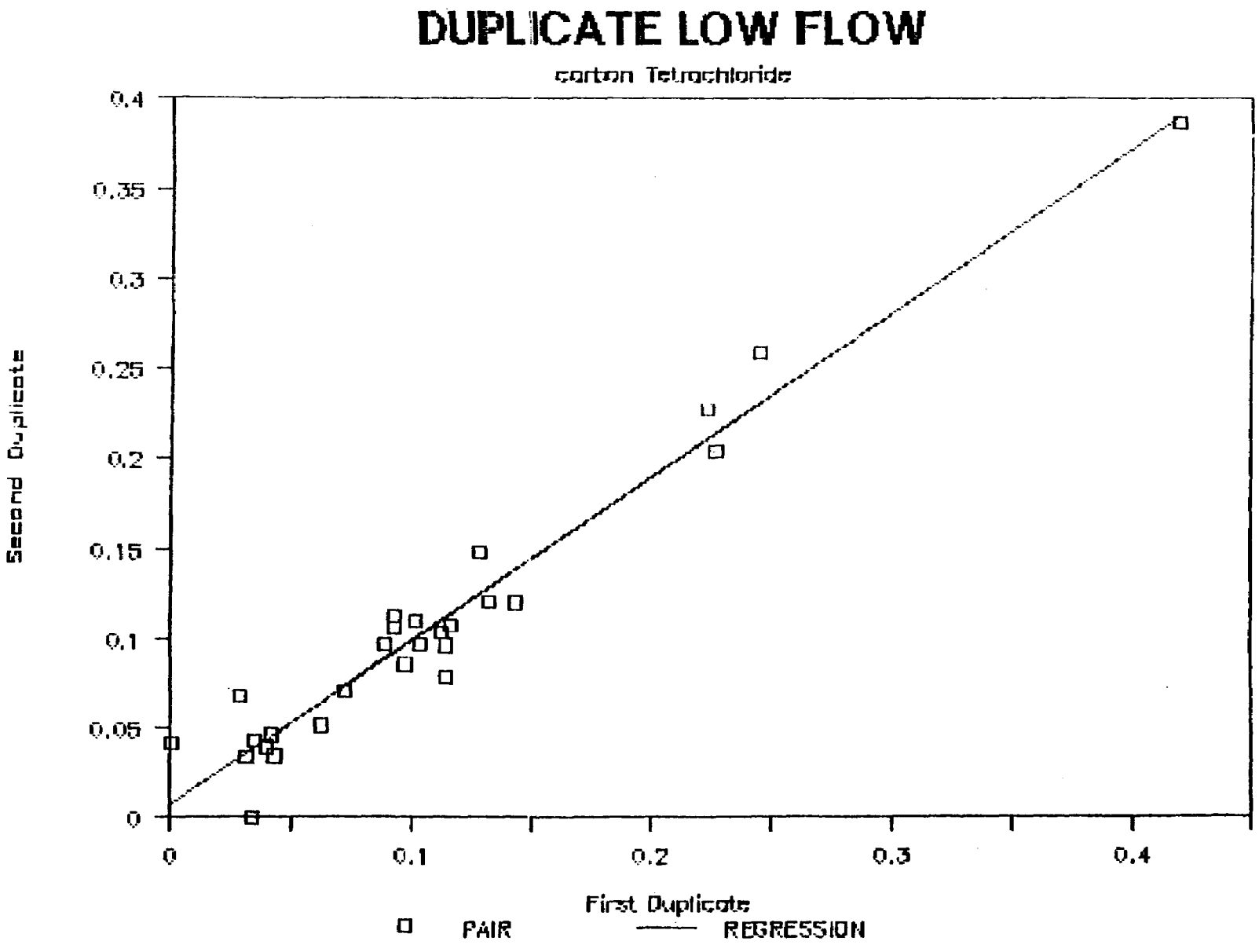

DUPLICATE LOW FLOW
carbon Tetrachloride
Second Duplicate
0.4
0.35
0.3
0.25
0.2
0.15
0.1
0.05
0
0
0.1
0.2
0.3
0.4
First Duplicate
PAIR
REGRESSION

Figure 2.6

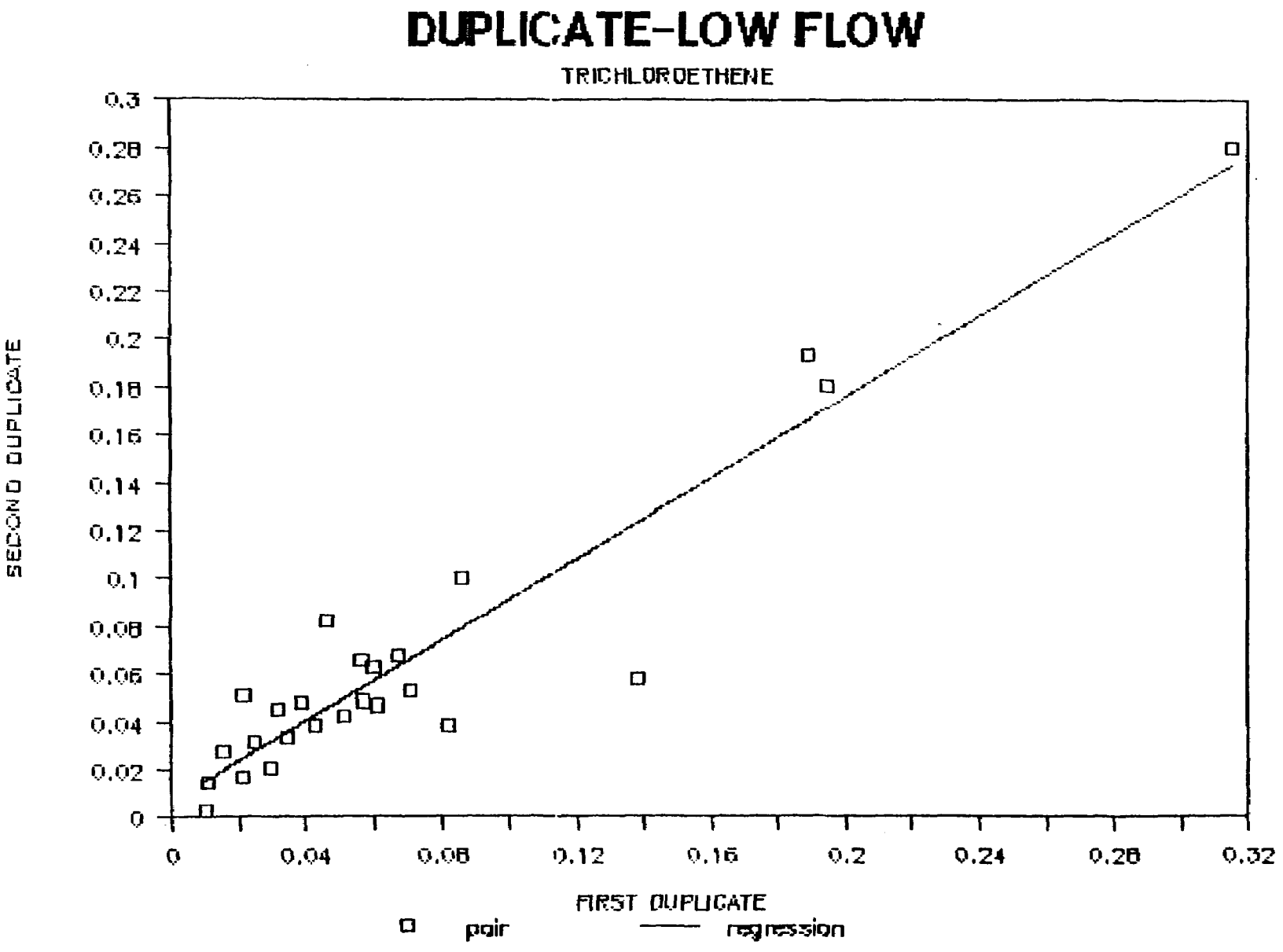

DUPLICATE-LOW FLOW
TRICHLOROETHENE
SECOND DUPLICATE
0.3
0.28
0.26
0.24
0.22
0.2
0.18
0.16
0.14
0.12
0.1
0.08
0.06
0.04
0.02
0
0
0.04
0.08
0.12
0.16
0.2
0.24
0.28
0.32
FIRST DUPLICATE
pair
regression

Figure 2.7

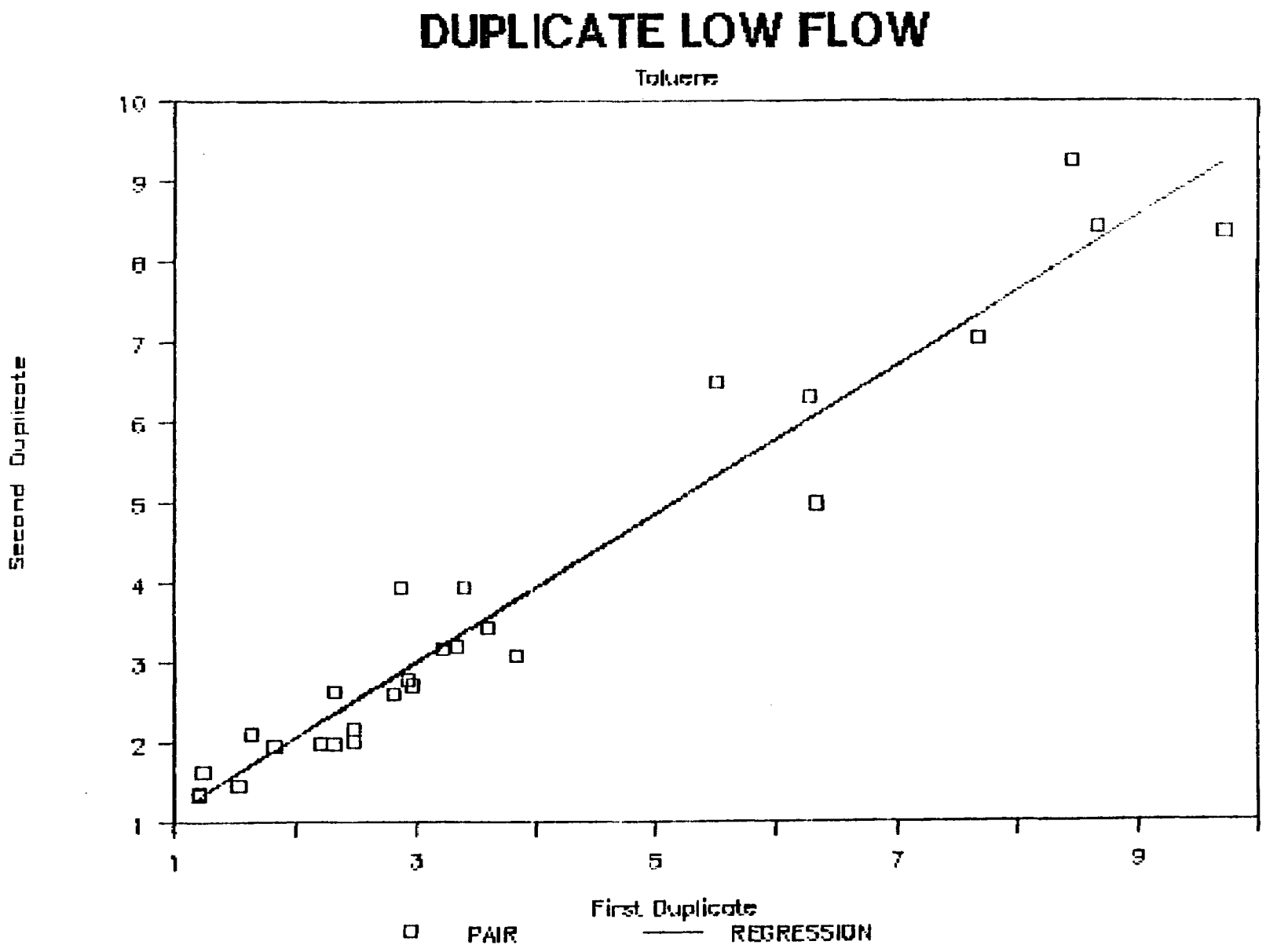

DUPLICATE LOW FLOW
Toluene
Second Duplicate
First Duplicate
PAIR
REGRESSION
1
2
3
4
5
6
7
8
9
10

Figure 2.8

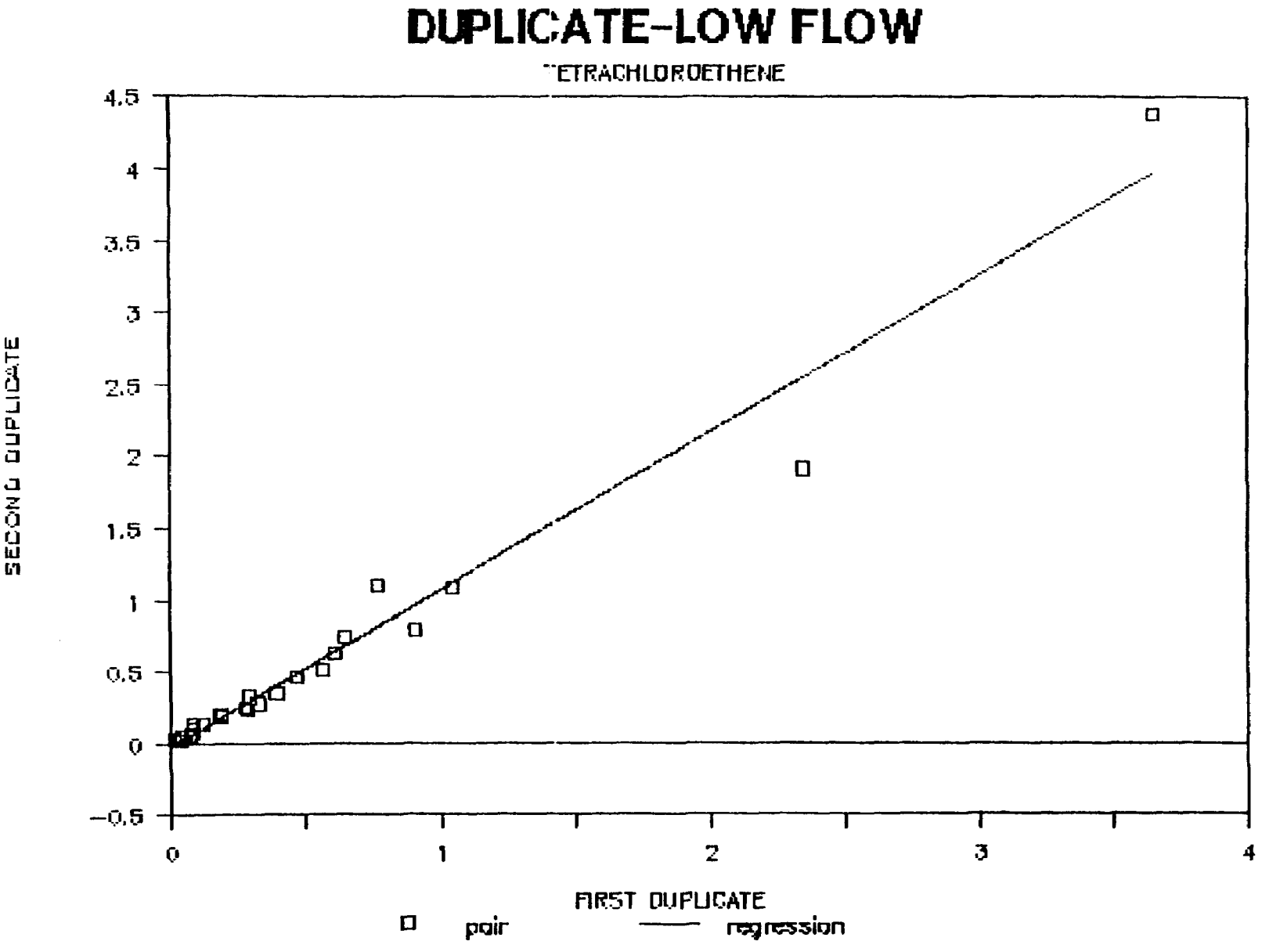

DUPLICATE-LOW FLOW
TETRACHLOROETHENE
SECOND DUPLICATE
FIRST DUPLICATE
4.5
4
3.5
3
2.5
2
1.5
1
0.5
0
-0.5
0
1
2
3
4
pair
regression

Figure 2.9

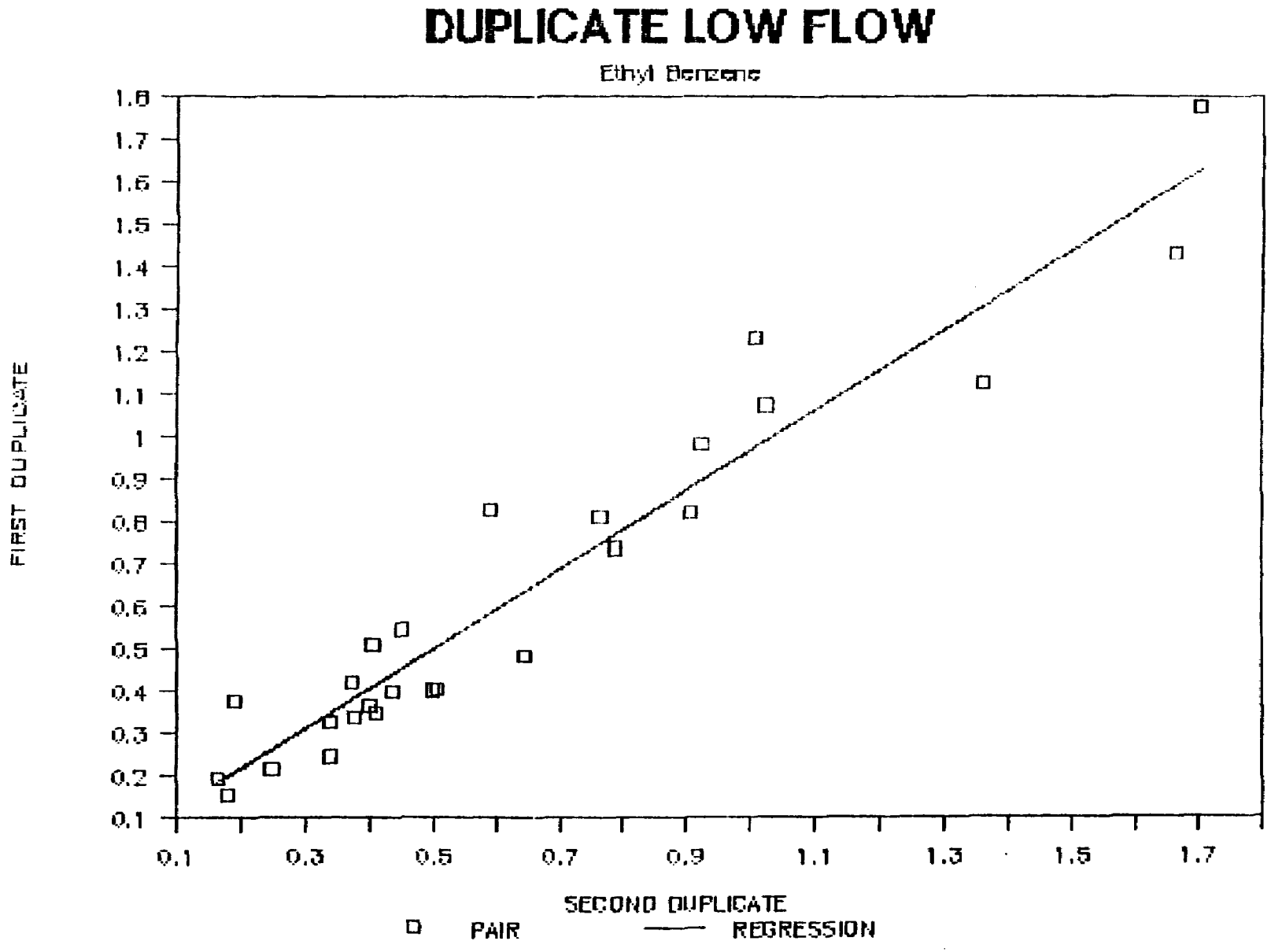

DUPLICATE LOW FLOW
Ethyl Benzene
FIRST DUPLICATE
1.8
1.7
1.6
1.5
1.4
1.3
1.2
1.1
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0.1
0.3
0.5
0.7
0.9
1.1
1.3
1.5
1.7
SECOND DUPLICATE
PAIR
REGRESSION

Figure 2.10

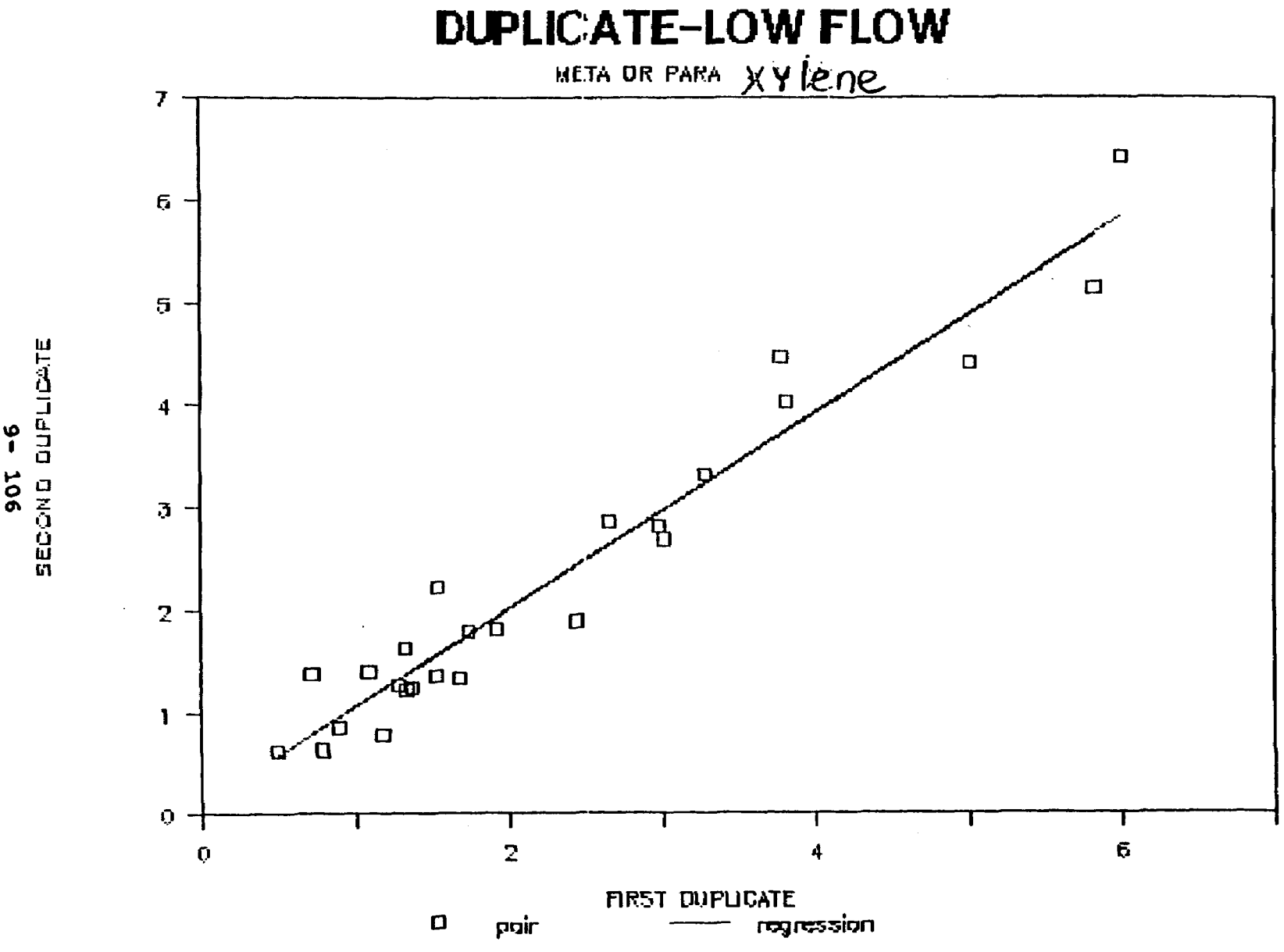

DUPLICATE-LOW FLOW
META OR PARA Xylene
SECOND DUPLICATE
9- 106
0
1
2
3
4
5
6
7
0
2
4
6
FIRST DUPLICATE
pair
regression

Figure 2.11

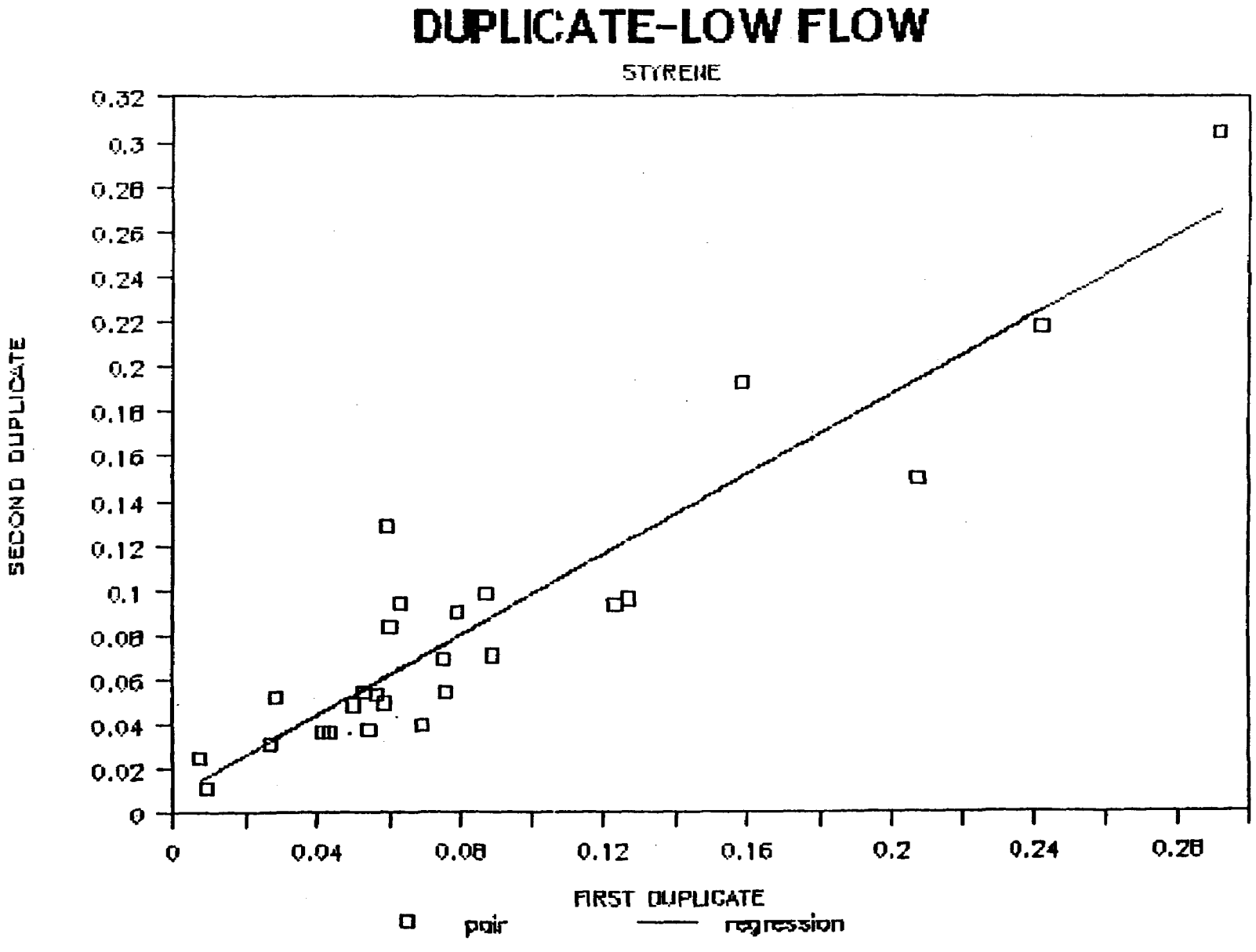

DUPLICATE-LOW FLOW
STYRENE
SECOND DUPLICATE
0.32
0.3
0.28
0.26
0.24
0.22
0.2
0.18
0.16
0.14
0.12
0.1
0.08
0.06
0.04
0.02
0
0
0.04
0.08
0.12
0.16
0.2
0.24
0.28
FIRST DUPLICATE
pair
regression

Figure 2:12

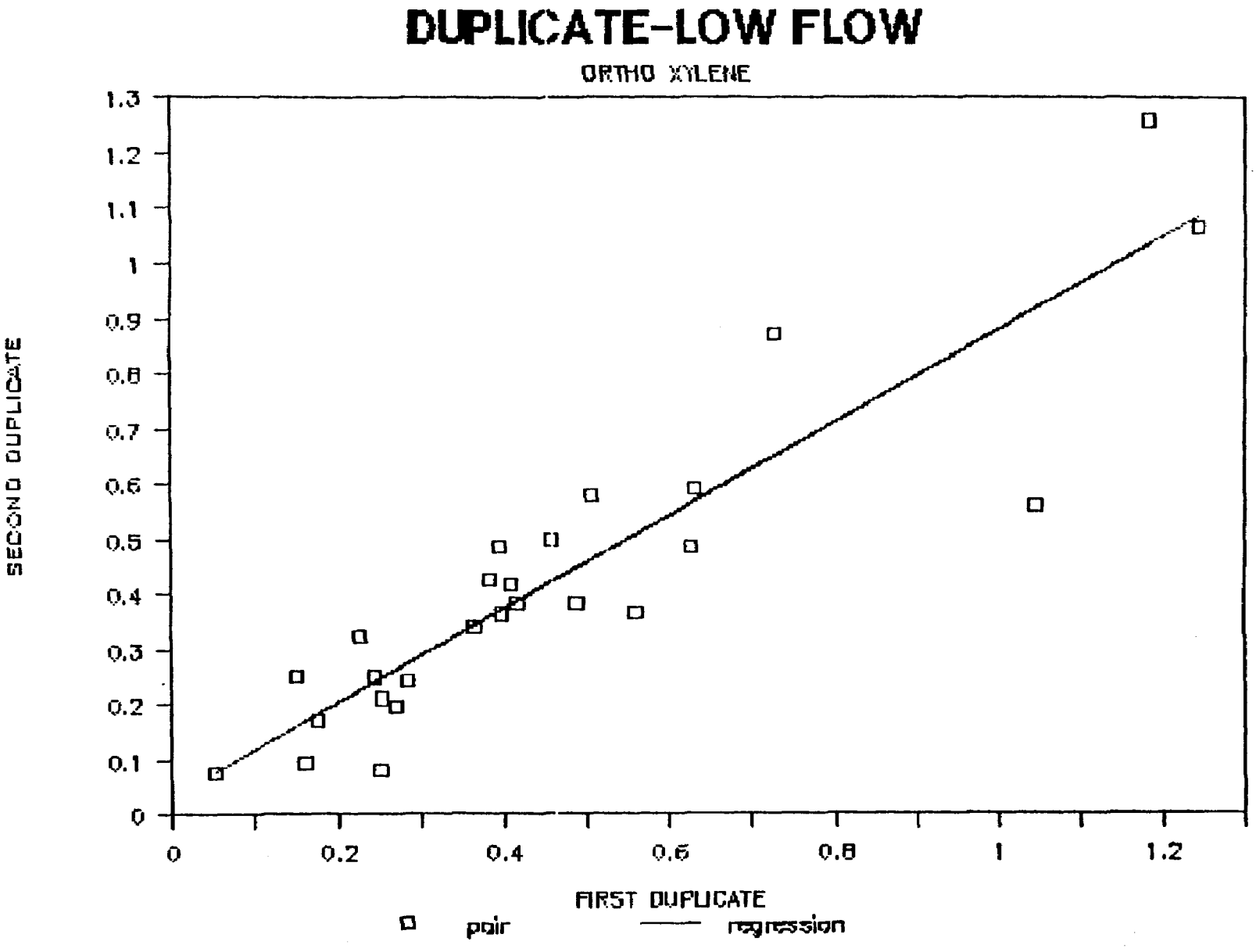

DUPLICATE-LOW FLOW
ORTHO XYLENE
SECOND DUPLICATE
1.3
1.2
1.1
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0
0.2
0.4
0.6
0.8
1
1.2
FIRST DUPLICATE
pair
regression

Figure 2.13

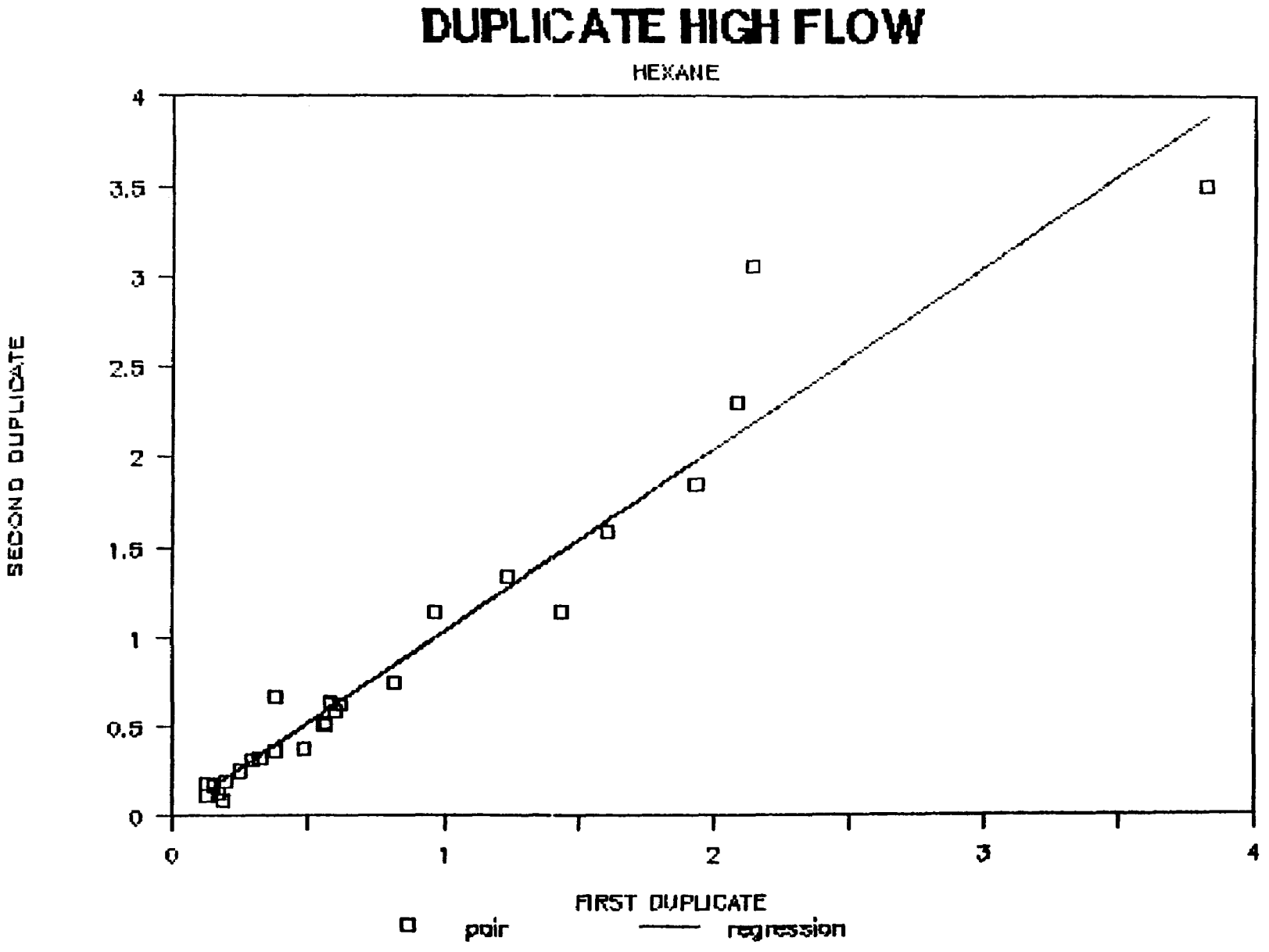

DUPLICATE HIGH FLOW
HEXANE
SECOND DUPLICATE
FIRST DUPLICATE
0
0.5
1
1.5
2
2.5
3
3.5
4
pair
regression

Figure 2.14

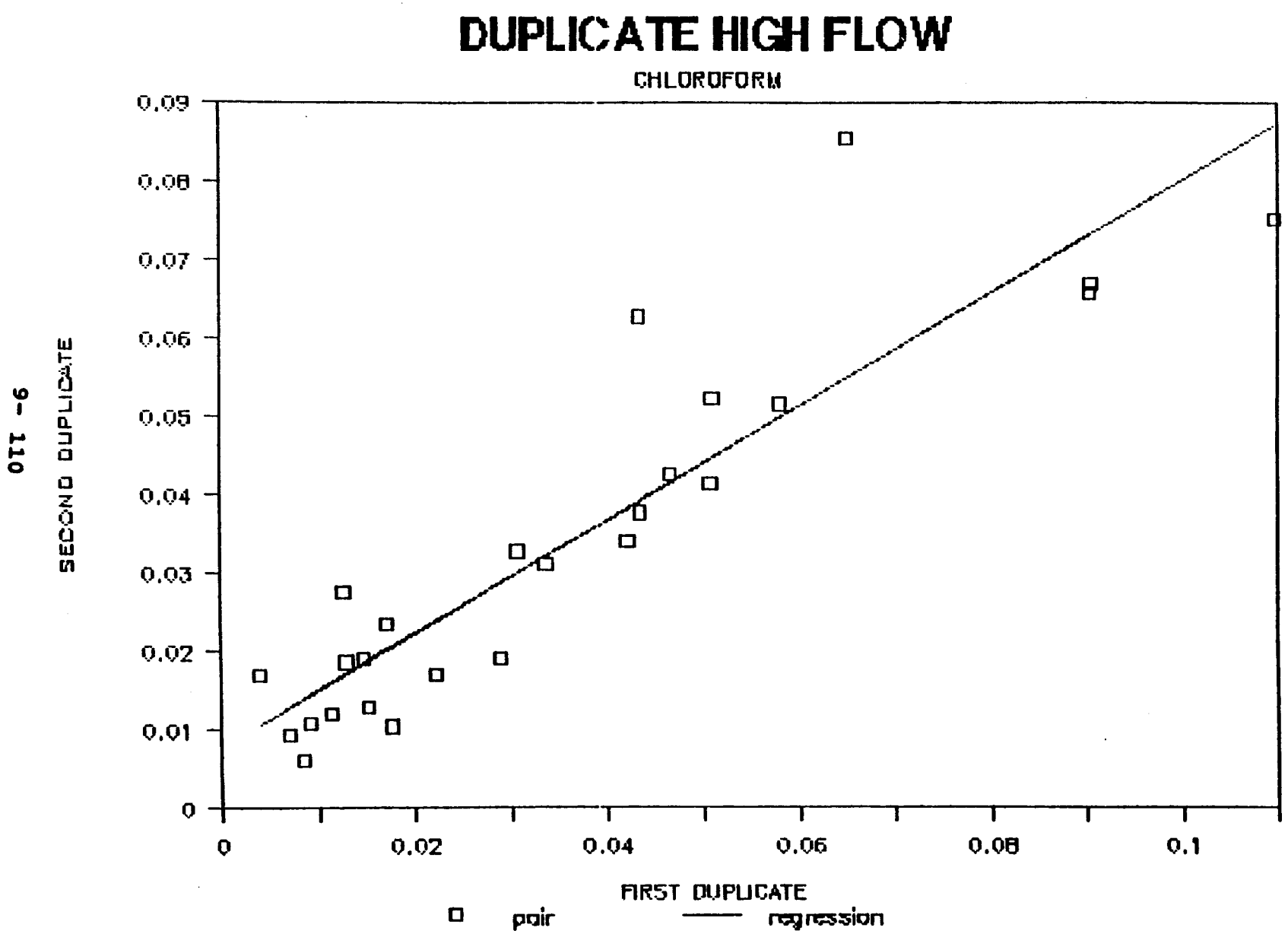

DUPLICATE HIGH FLOW
CHLOROFORM
SECOND DUPLICATE
0.09
0.08
0.07
0.06
0.05
0.04
0.03
0.02
0.01
0
0
0.02
0.04
0.06
0.08
0.1
FIRST DUPLICATE
pair
regression

Figure 2.15

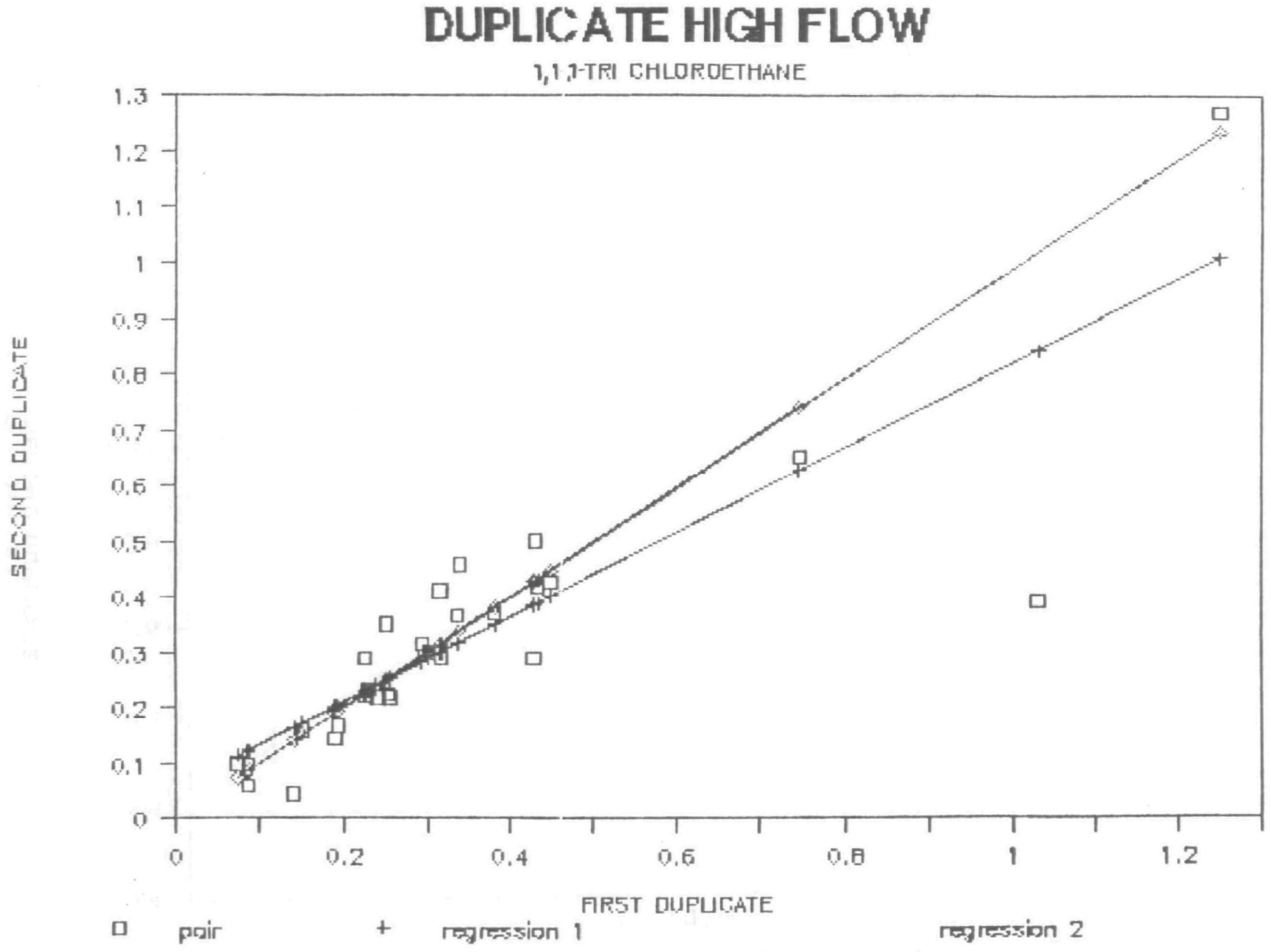

DUPLICATE HIGH FLOW
1,1,1-TRI CHLOROETHANE
SECOND DUPLICATE
1.3
1.2
1.1
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0
0.2
0.4
0.6
0.8
1
1.2
FIRST DUPLICATE
pair
regression 1
regression 2

Figure 2.16

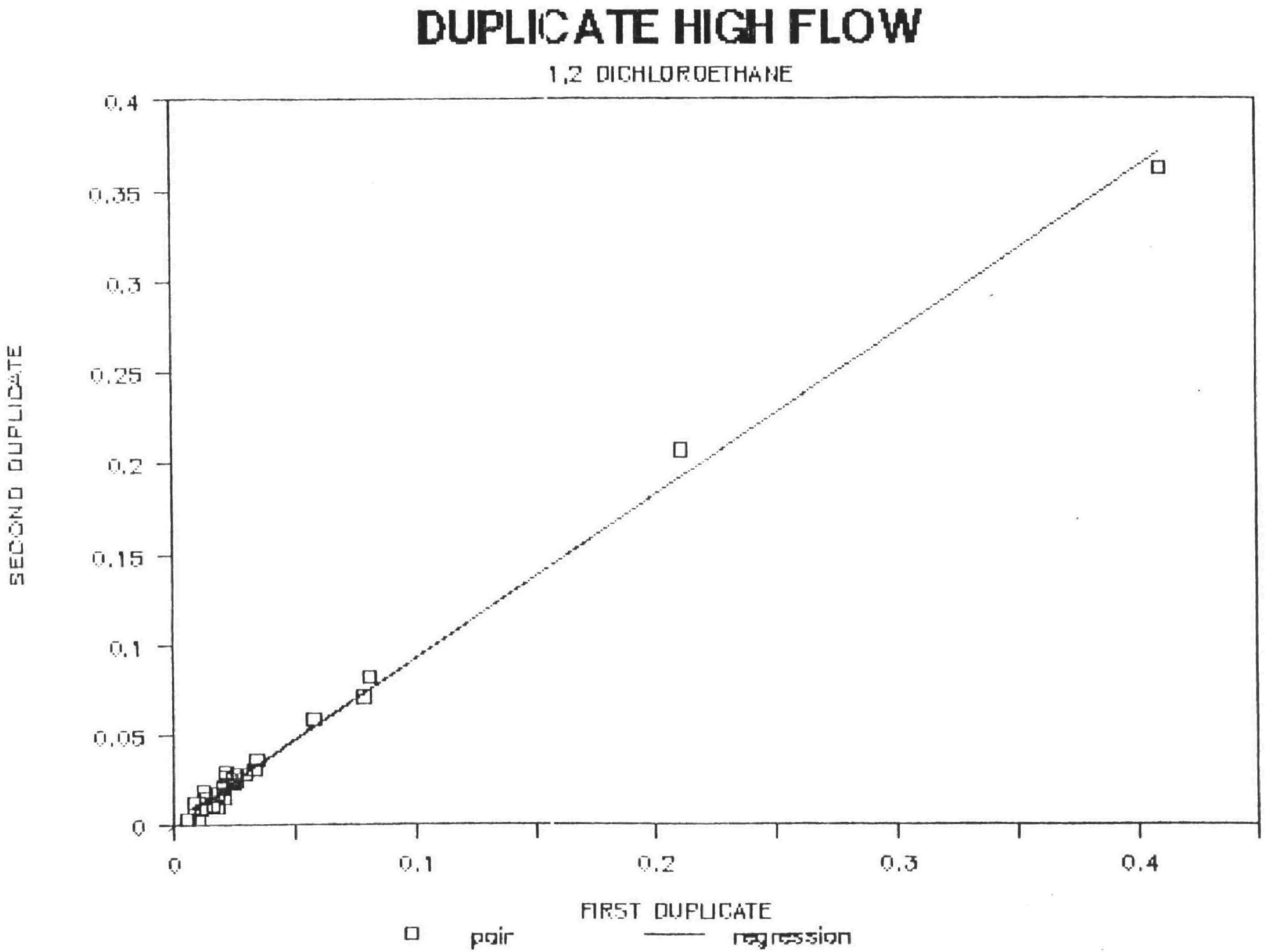

DUPLICATE HIGH FLOW
1,2 DICHLOROETHANE
SECOND DUPLICATE
0.4
0.35
0.3
0.25
0.2
0.15
0.1
0.05
0
0
0.1
0.2
0.3
0.4
FIRST DUPLICATE
pair
regression

Figure 2.17

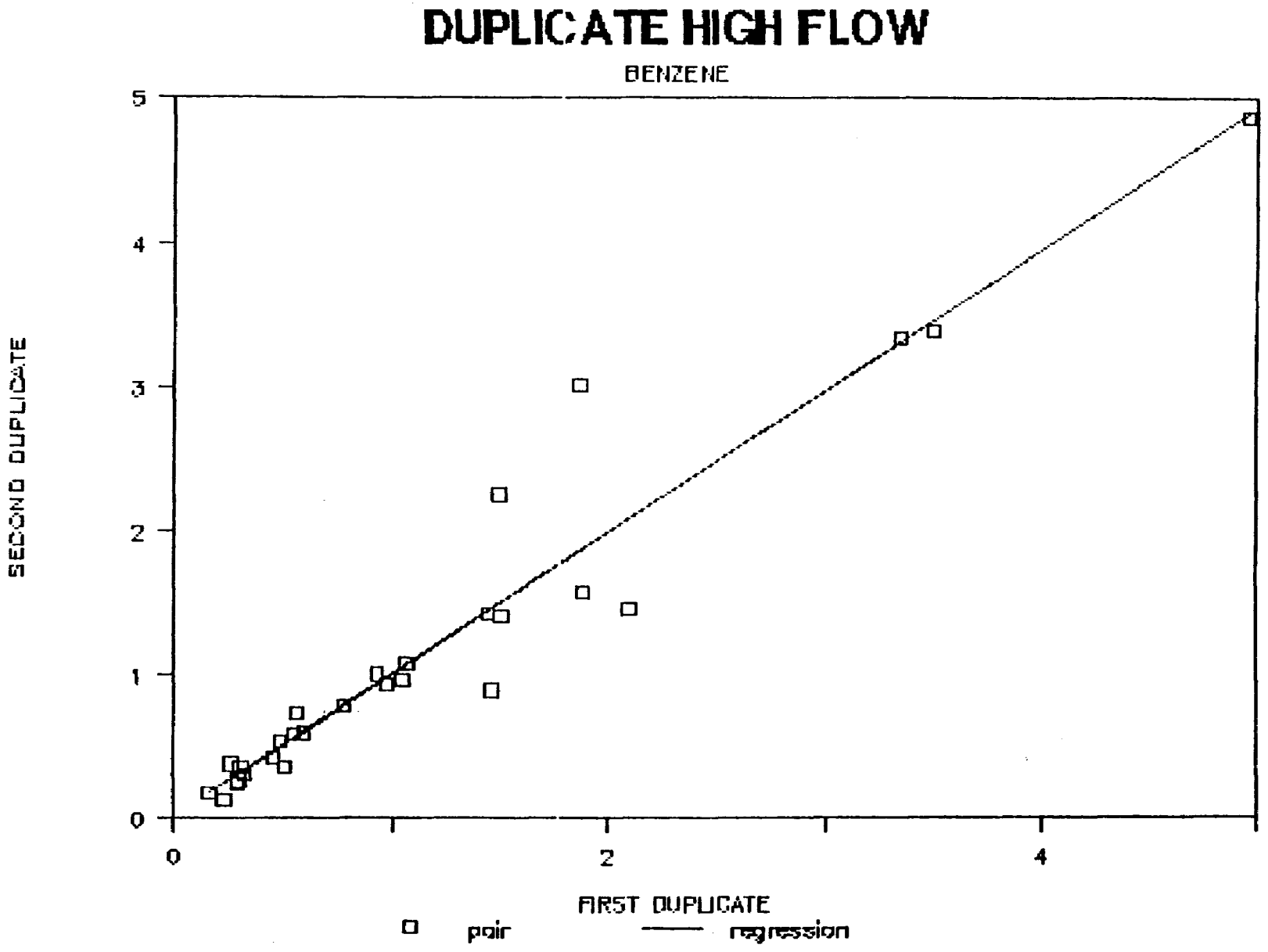

DUPLICATE HIGH FLOW
BENZENE
SECOND DUPLICATE
FIRST DUPLICATE
0
1
2
3
4
5
0
2
4
pair
regression

Figure 2.18

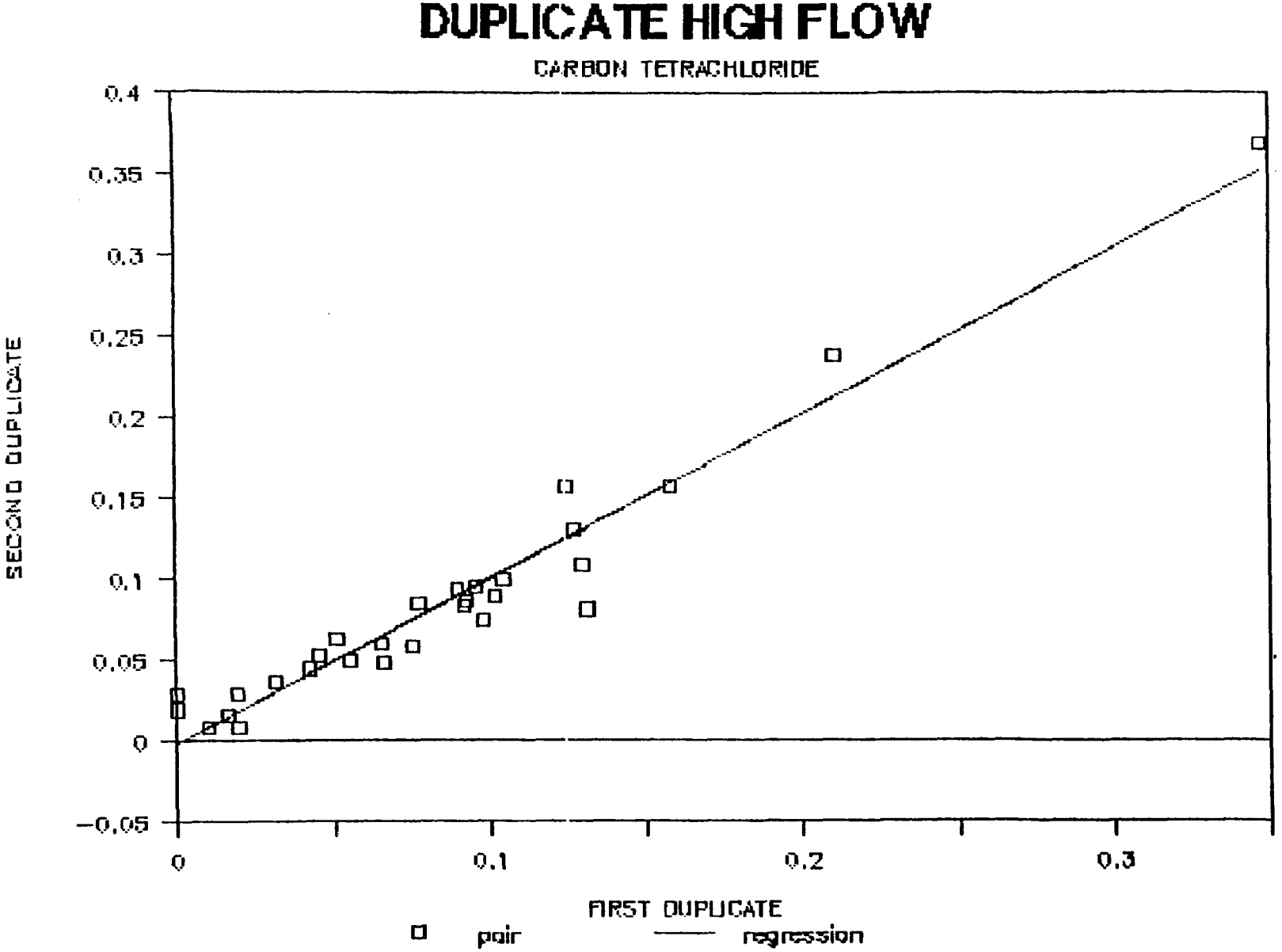

DUPLICATE HIGH FLOW
CARBON TETRACHLORIDE
SECOND DUPLICATE
0.4
0.35
0.3
0.25
0.2
0.15
0.1
0.05
0
-0.05
0
0.1
0.2
0.3
FIRST DUPLICATE
pair
regression

Figure 2.19

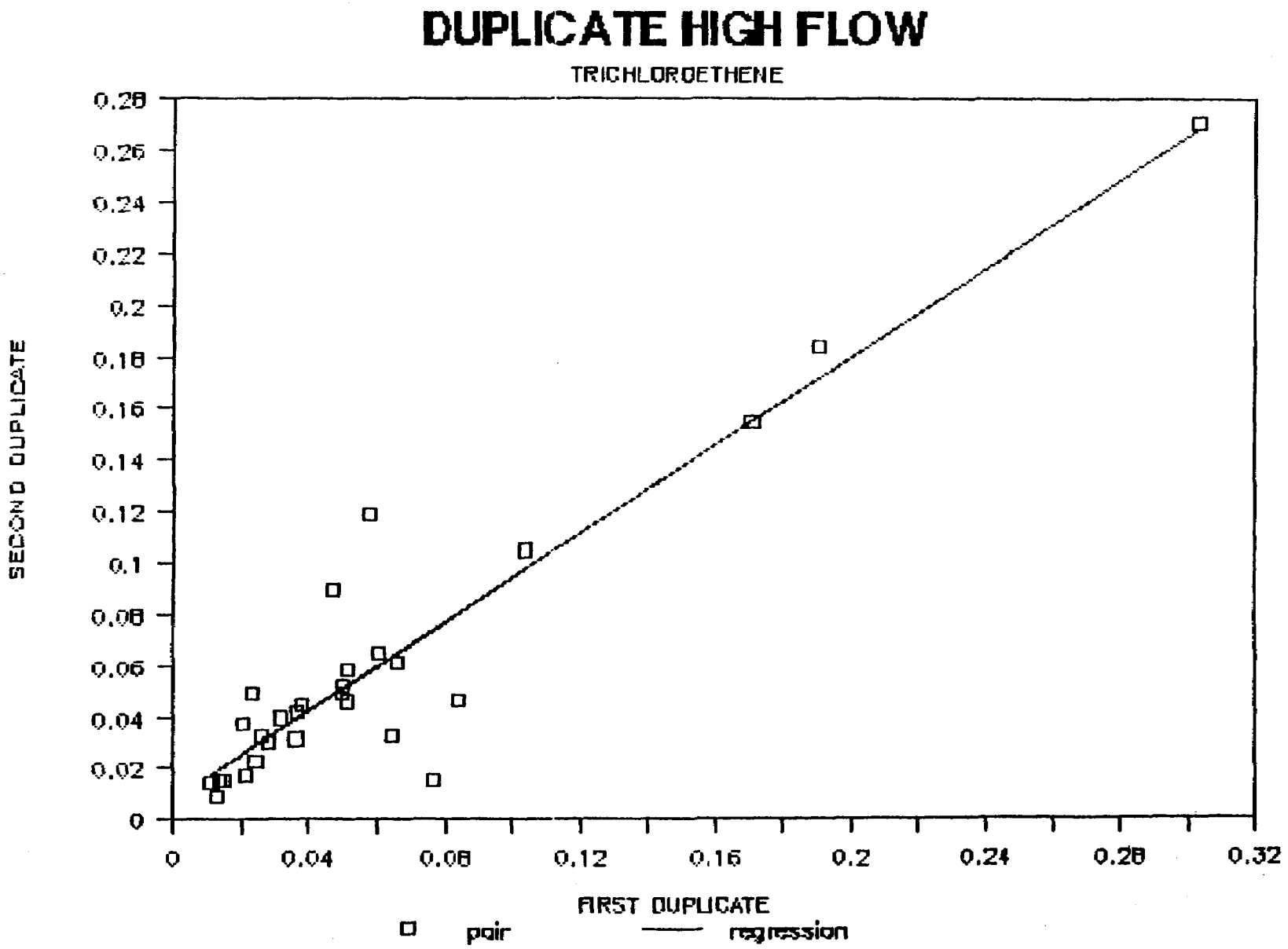

DUPLICATE HIGH FLOW
TRICHLOROETHENE
SECOND DUPLICATE
0.28
0.26
0.24
0.22
0.2
0.18
0.16
0.14
0.12
0.1
0.08
0.06
0.04
0.02
0
0
0.04
0.08
0.12
0.16
0.2
0.24
0.28
0.32
FIRST DUPLICATE
pair
regression

Figure 2.20

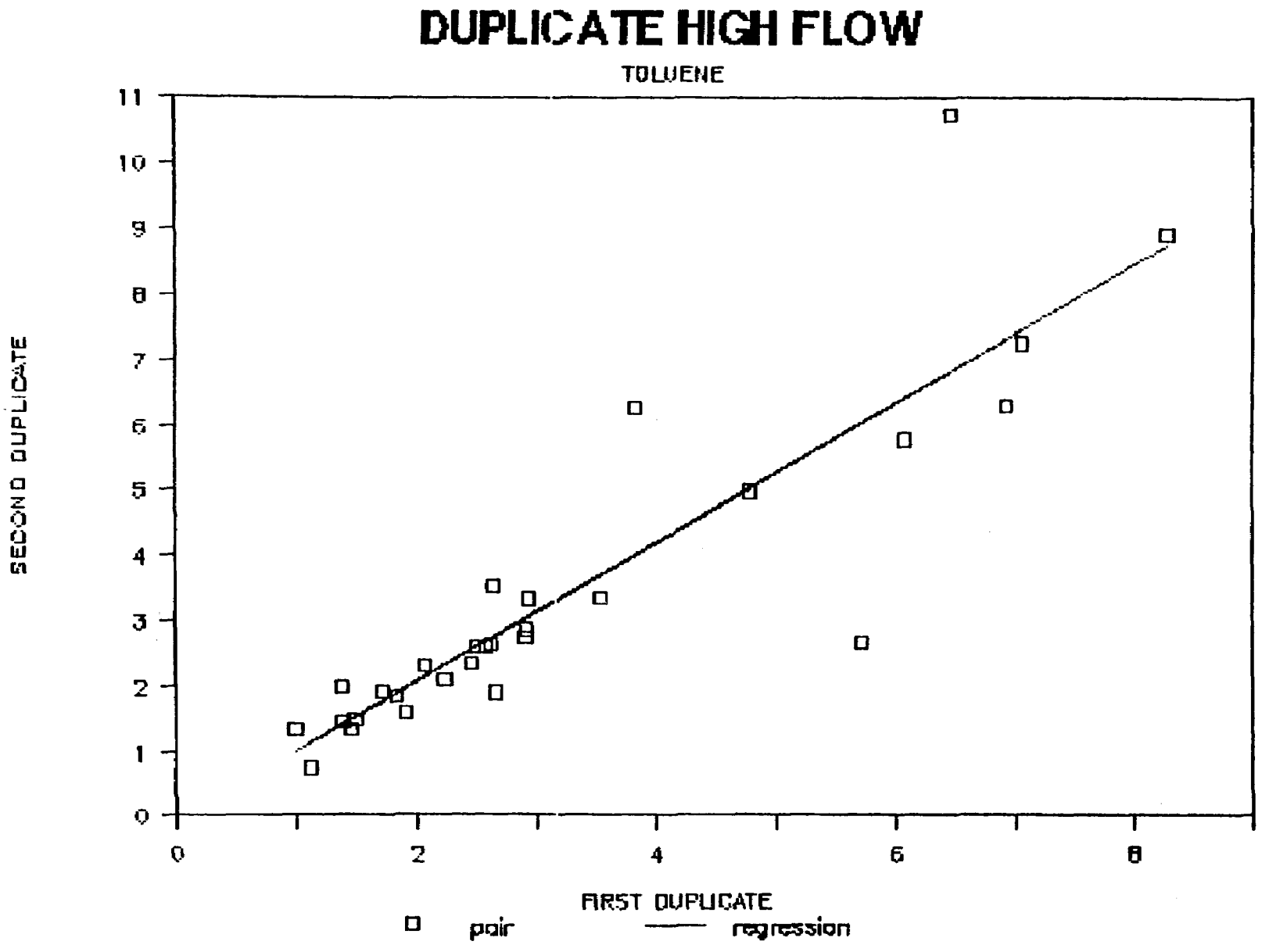

DUPLICATE HIGH FLOW
TOLUENE
SECOND DUPLICATE
FIRST DUPLICATE
pair
regression
0
1
2
3
4
5
6
7
8
9
10
11
0
2
4
6
8

Figure 2.21

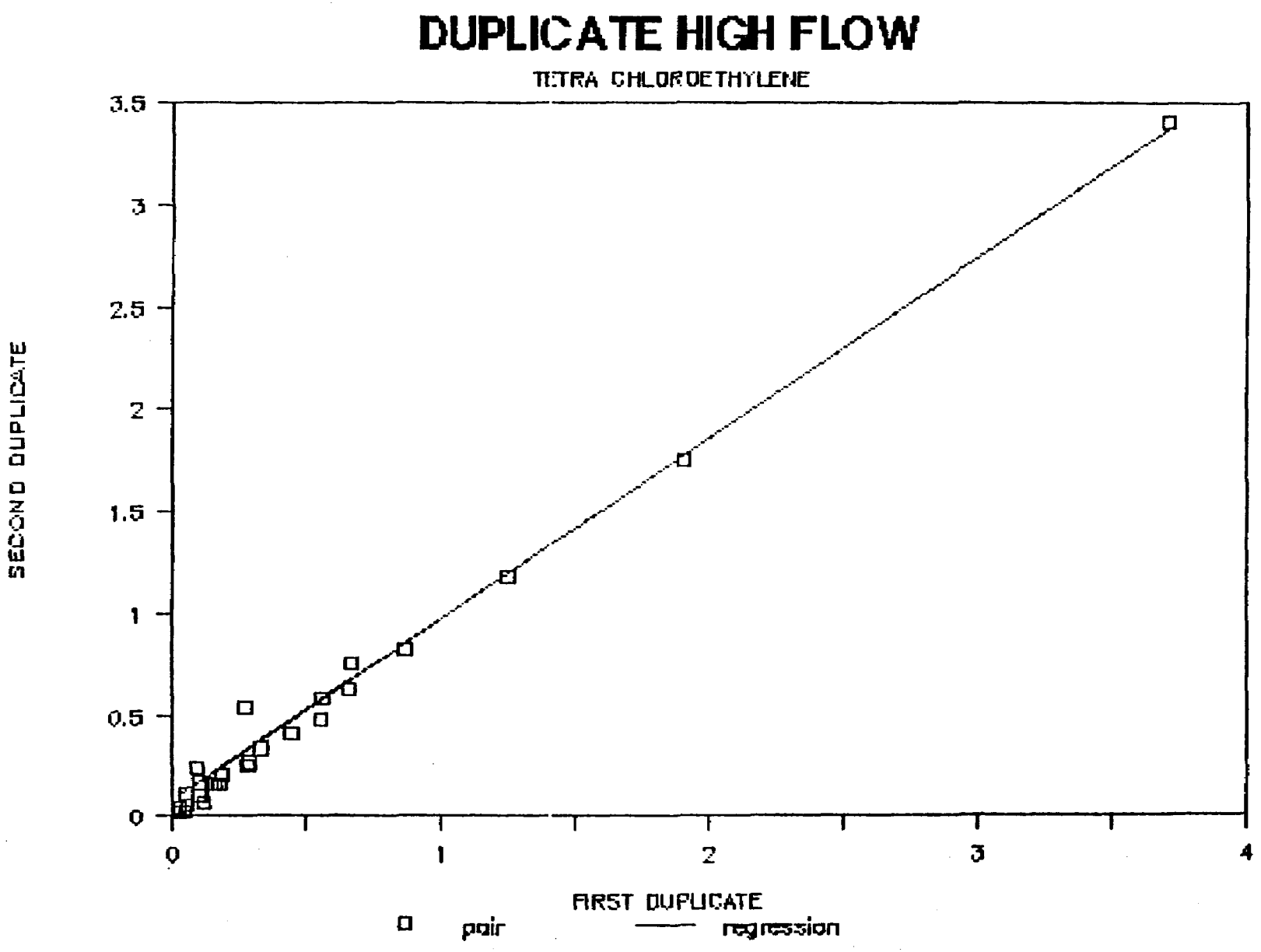

DUPLICATE HIGH FLOW
TETRA CHLOROETHYLENE
SECOND DUPLICATE
FIRST DUPLICATE
3.5
3
2.5
2
1.5
1
0.5
0
0
1
2
3
4
pair
regression

Figure 2.22

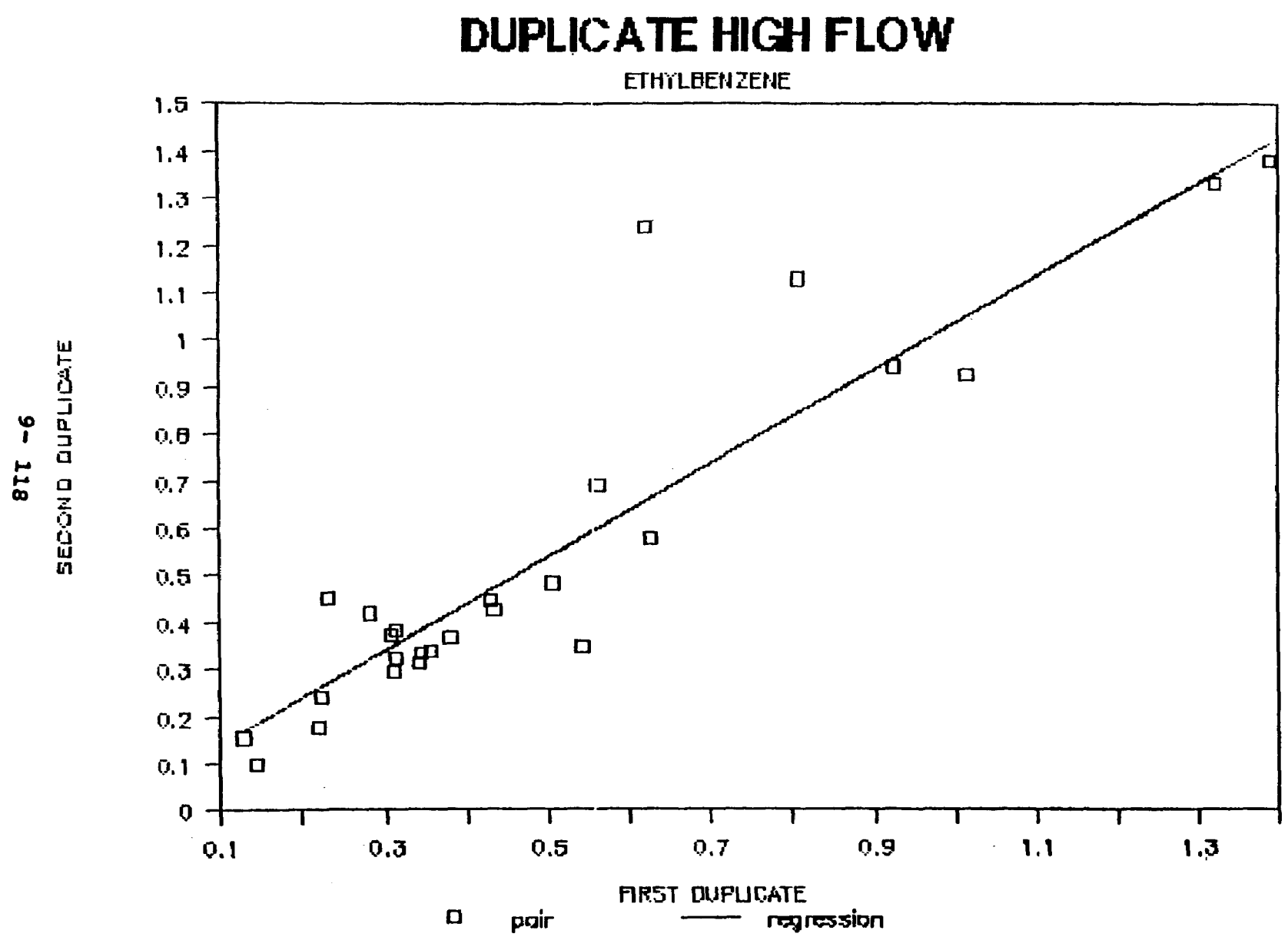

DUPLICATE HIGH FLOW
ETHYLBENZENE
SECOND DUPLICATE
8II -6
1.5
1.4
1.3
1.2
1.1
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0.1
0.3
0.5
0.7
0.9
1.1
1.3
FIRST DUPLICATE
pair
regression

Figure 2.23

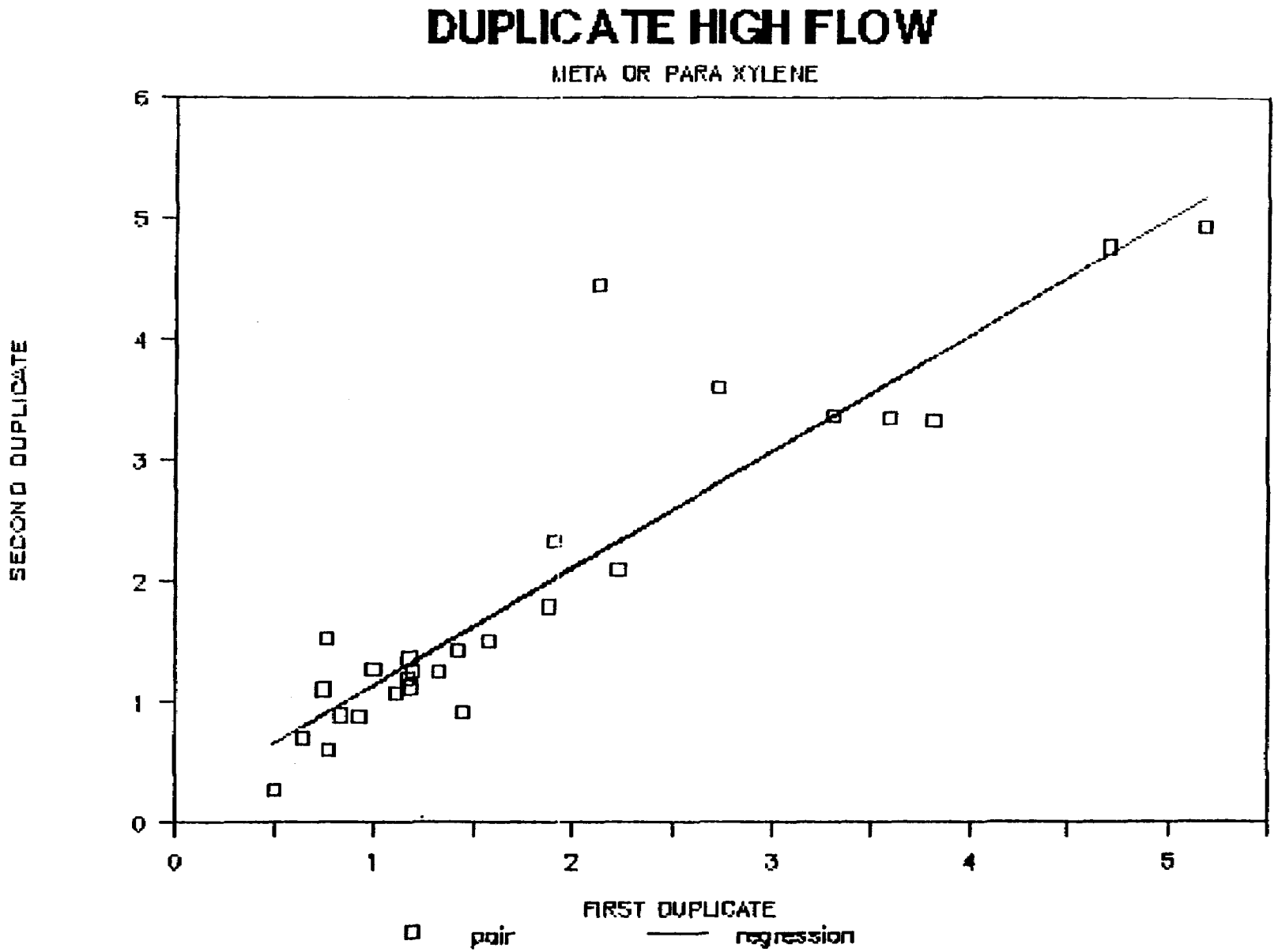

DUPLICATE HIGH FLOW
META OR PARA XYLENE
SECOND DUPLICATE
FIRST DUPLICATE
0
1
2
3
4
5
6
pair
regression

Figure 2.24

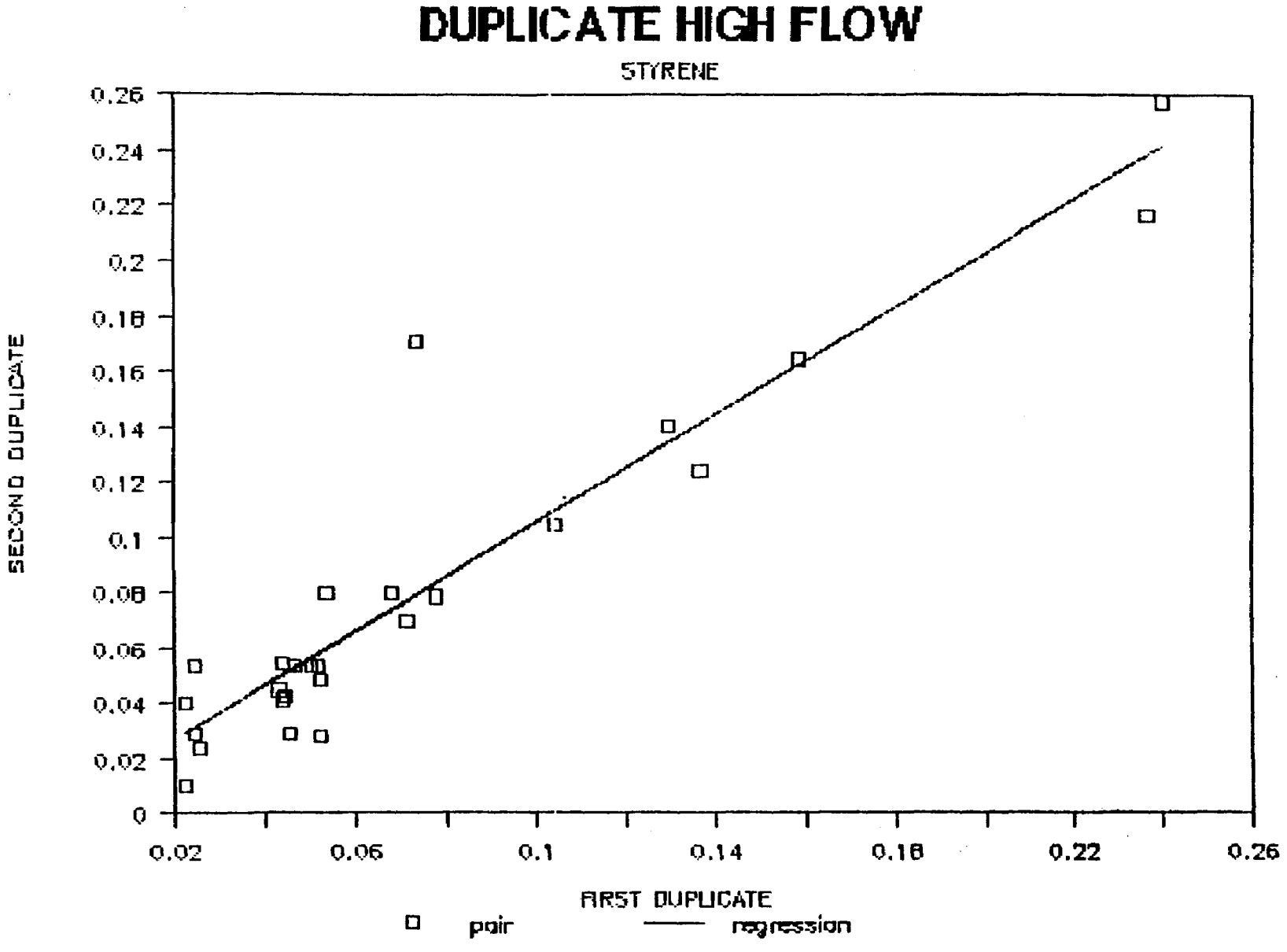

DUPLICATE HIGH FLOW
STYRENE
SECOND DUPLICATE
0.26
0.24
0.22
0.2
0.18
0.16
0.14
0.12
0.1
0.08
0.06
0.04
0.02
0
0.02
0.06
0.1
0.14
0.18
0.22
0.26
FIRST DUPLICATE
pair
regression

Figure 2.25

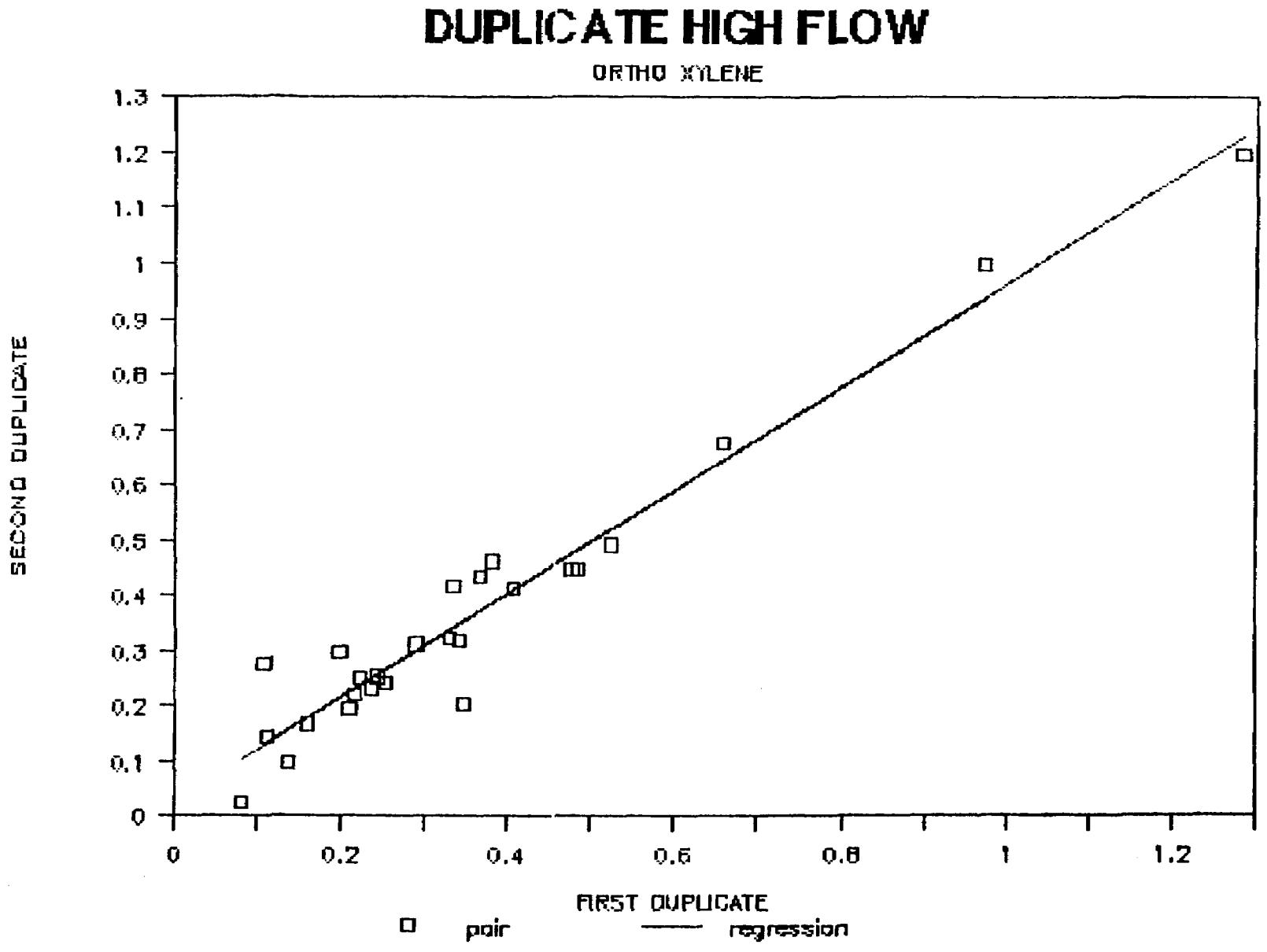

DUPLICATE HIGH FLOW
ORTHO XYLENE
SECOND DUPLICATE
1.3
1.2
1.1
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0
0.2
0.4
0.6
0.8
1
1.2
FIRST DUPLICATE
pair
regression

Figure 2.26

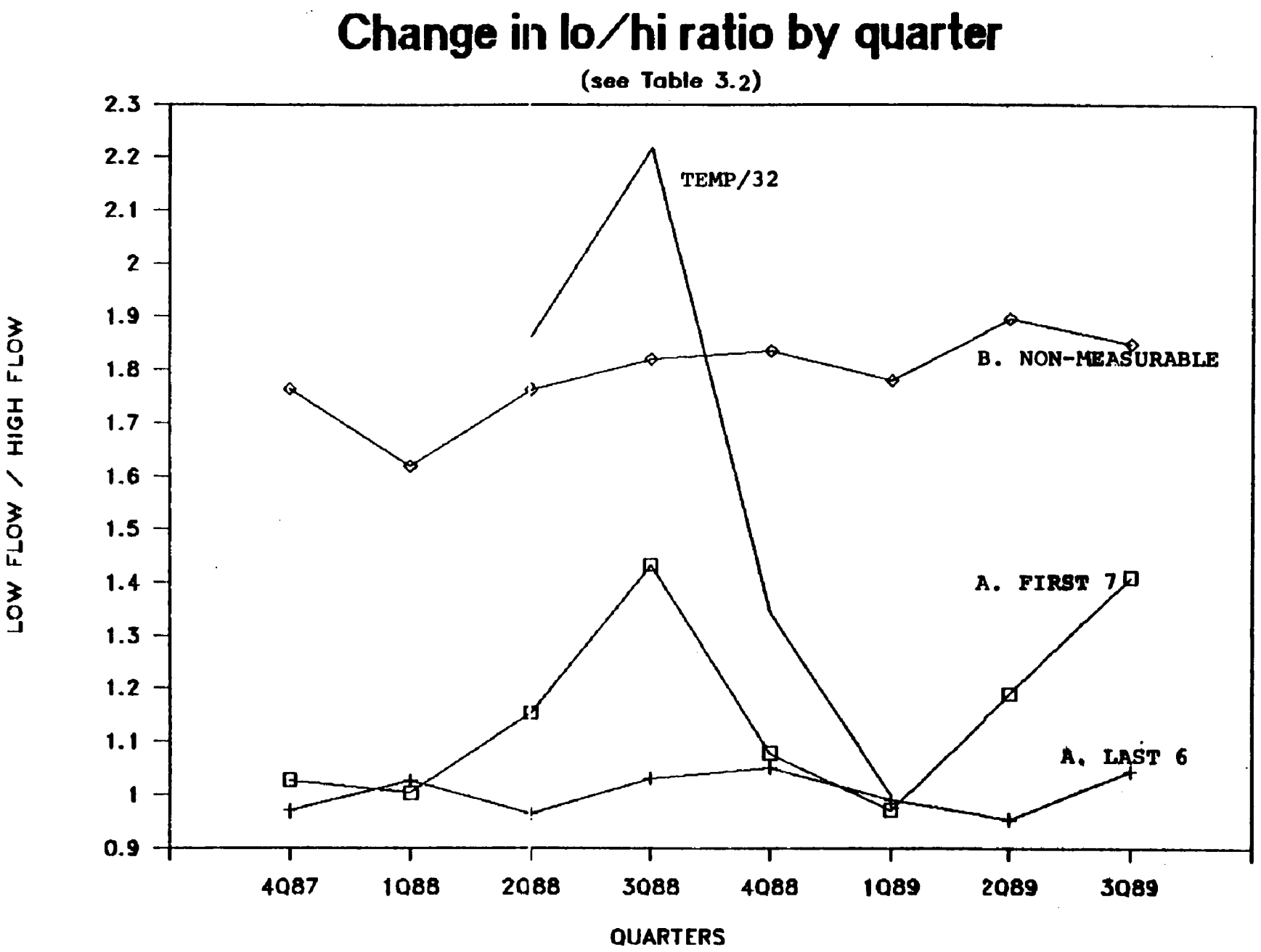

Change in lo/hi ratio by quarter
(see Table 3.2)
LOW FLOW / HIGH FLOW
2.3
2.2
2.1
2
1.9
1.8
1.7
1.6
1.5
1.4
1.3
1.2
1.1
1
0.9
TEMP/32
B. NON-MEASURABLE
A. FIRST 7
A. LAST 6
4Q87
1Q88
2Q88
3Q88
4Q88
1Q89
2Q89
3Q89
QUARTERS

Figure 3.1

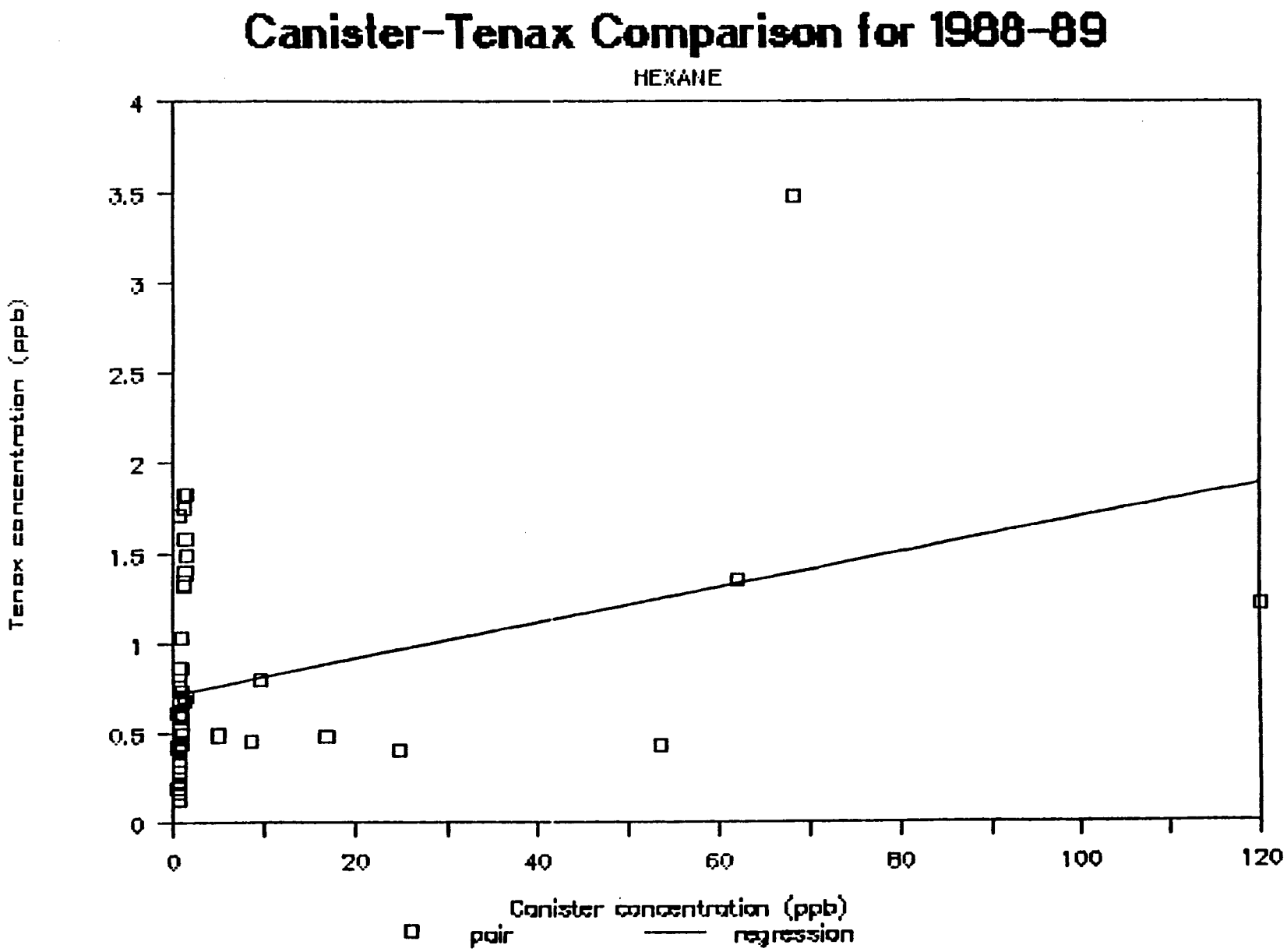

Canister-Tenax Comparison for 1988-89
HEXANE
Tenax concentration (ppb)
4
3.5
3
2.5
2
1.5
1
0.5
0
0
20
40
60
80
100
120
Canister concentration (ppb)
pair
regression

Figure 4.1a

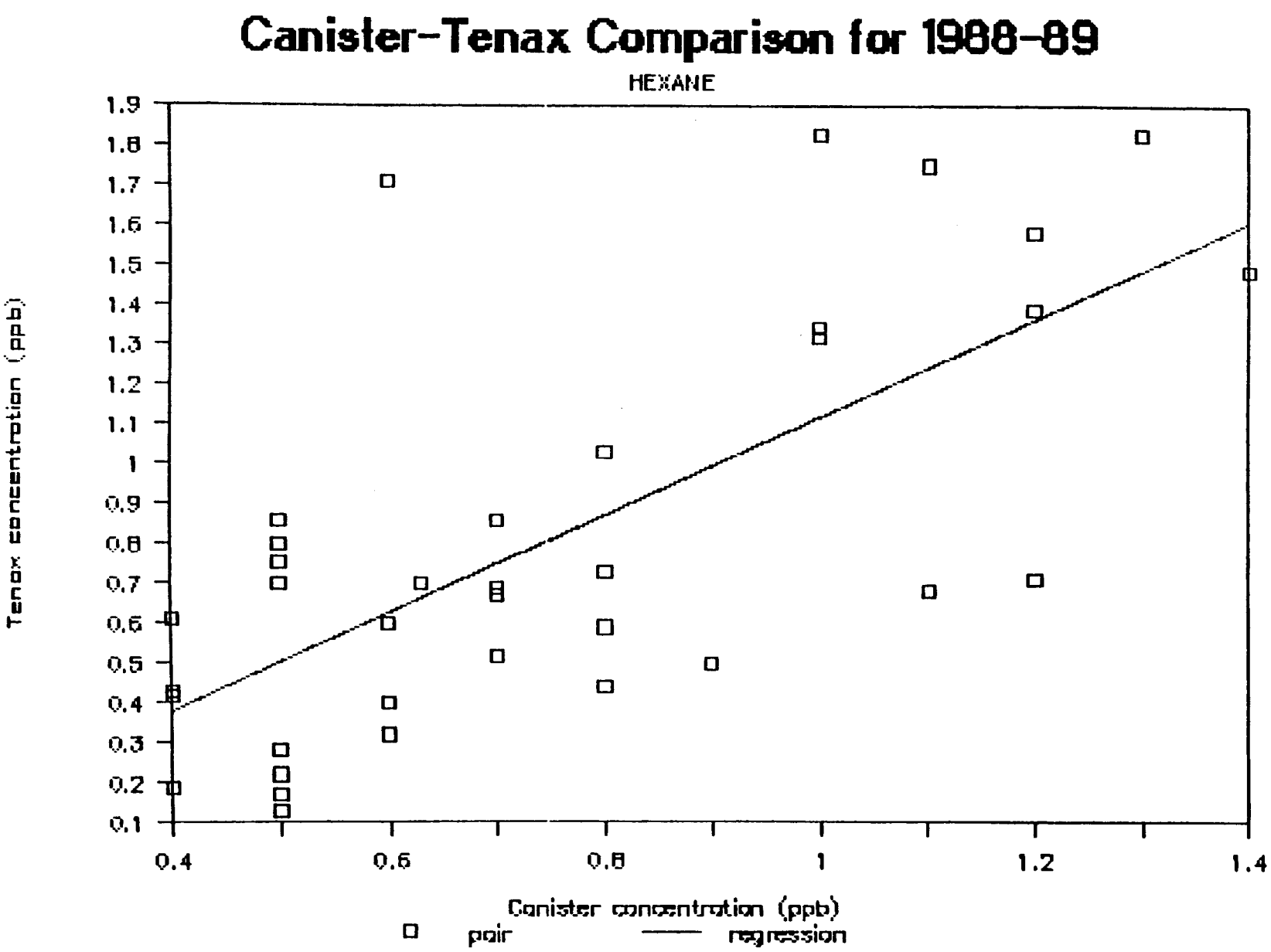

Canister-Tenax Comparison for 1988-89
HEXANE
Tenax concentration (ppb)
1.9
1.8
1.7
1.6
1.5
1.4
1.3
1.2
1.1
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0.4
0.6
0.8
1
1.2
1.4
Canister concentration (ppb)
pair
regression

Figure 4.1b

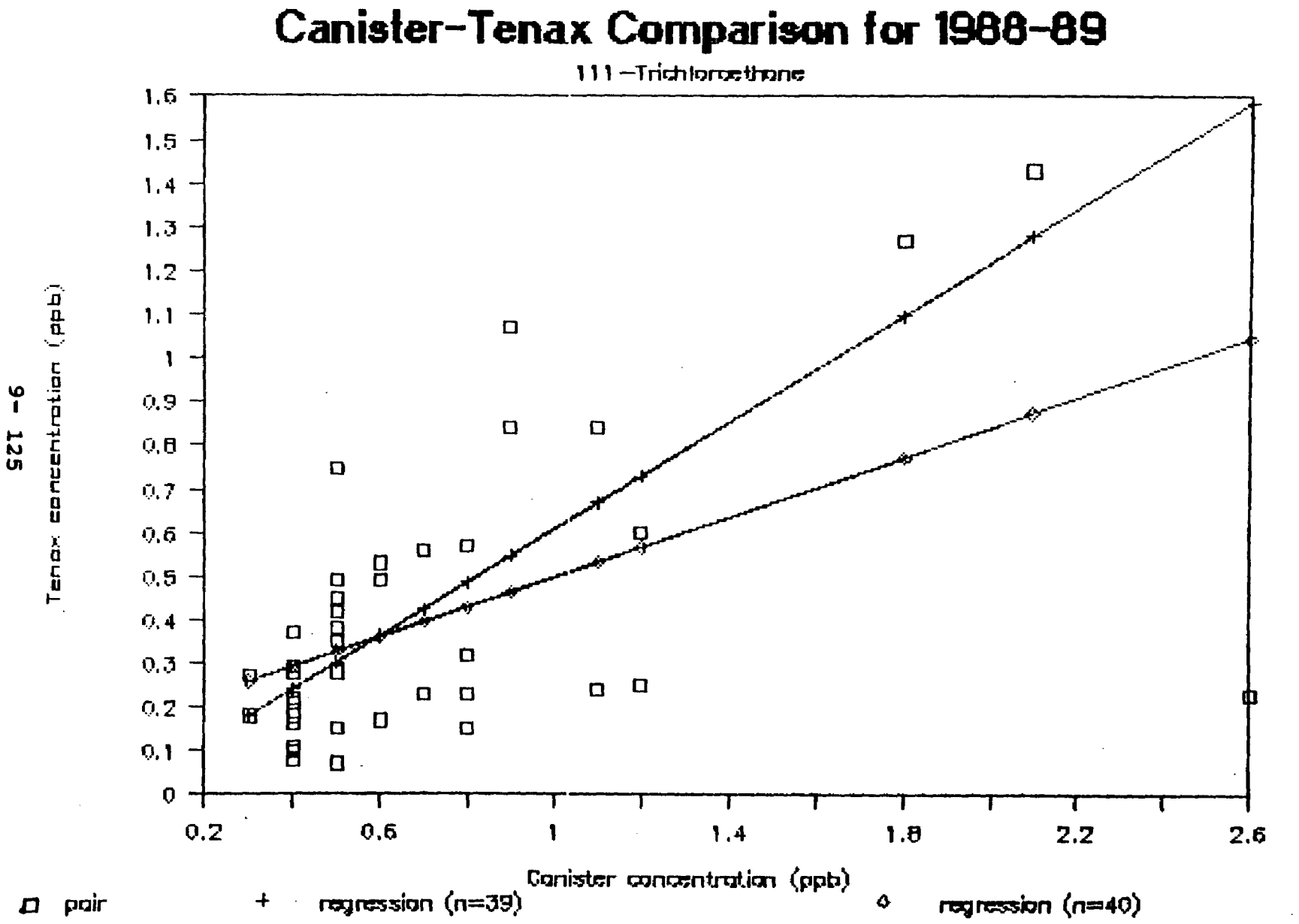

Canister-Tenax Comparison for 1988-89
111-Trichloroethane
Tenax concentration (ppb)
Canister concentration (ppb)
1.6
1.5
1.4
1.3
1.2
1.1
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0.2
0.6
1
1.4
1.8
2.2
2.6
pair
regression (n=39)
regression (n=40)

Figure 4.2

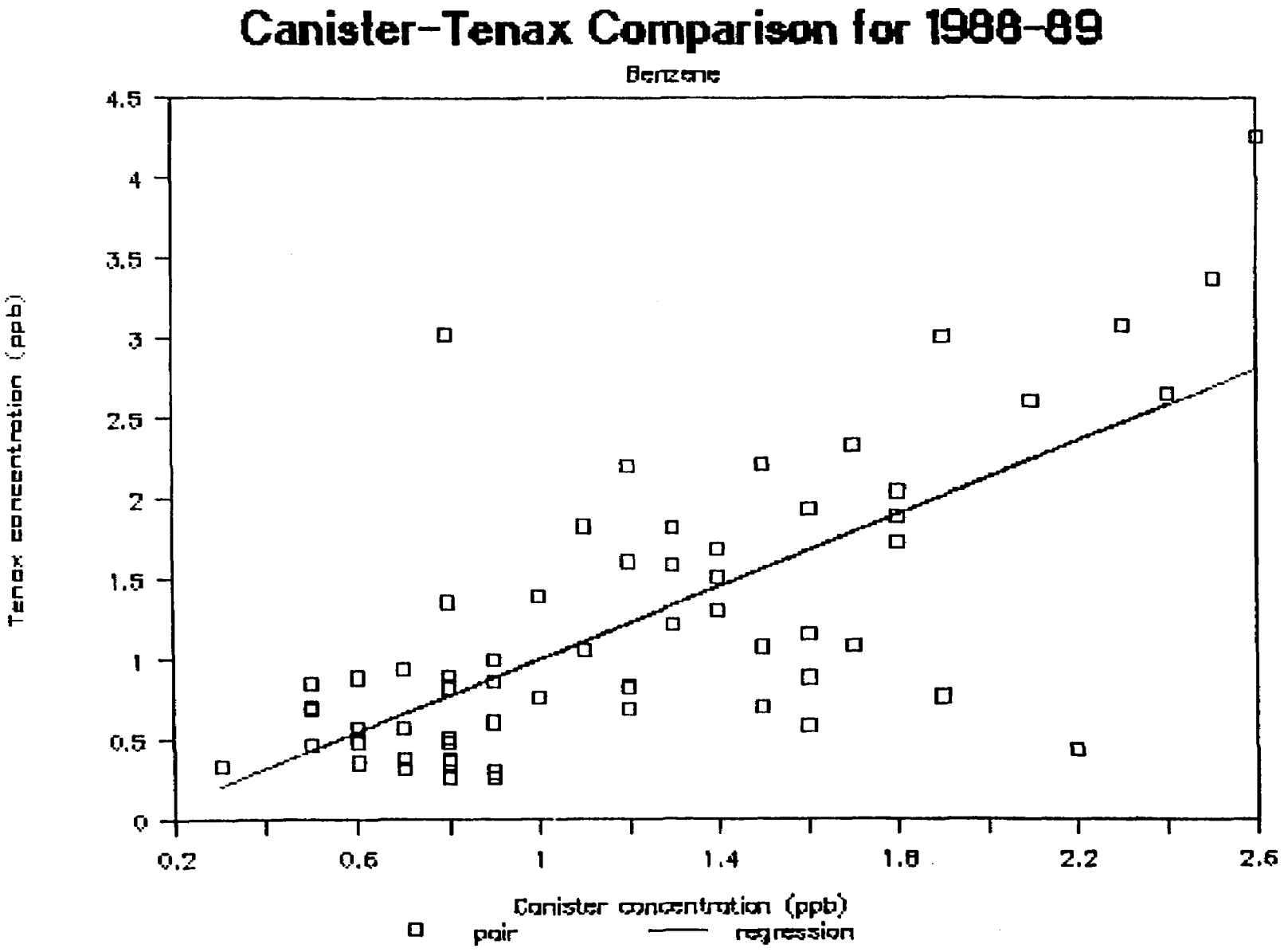

Canister-Tenax Comparison for 1988-89
Benzene
Tenax concentration (ppb)
4.5
4
3.5
3
2.5
2
1.5
1
0.5
0
0.2
0.6
1
1.4
1.8
2.2
2.6
Canister concentration (ppb)
pair
regression

Figure 4.3

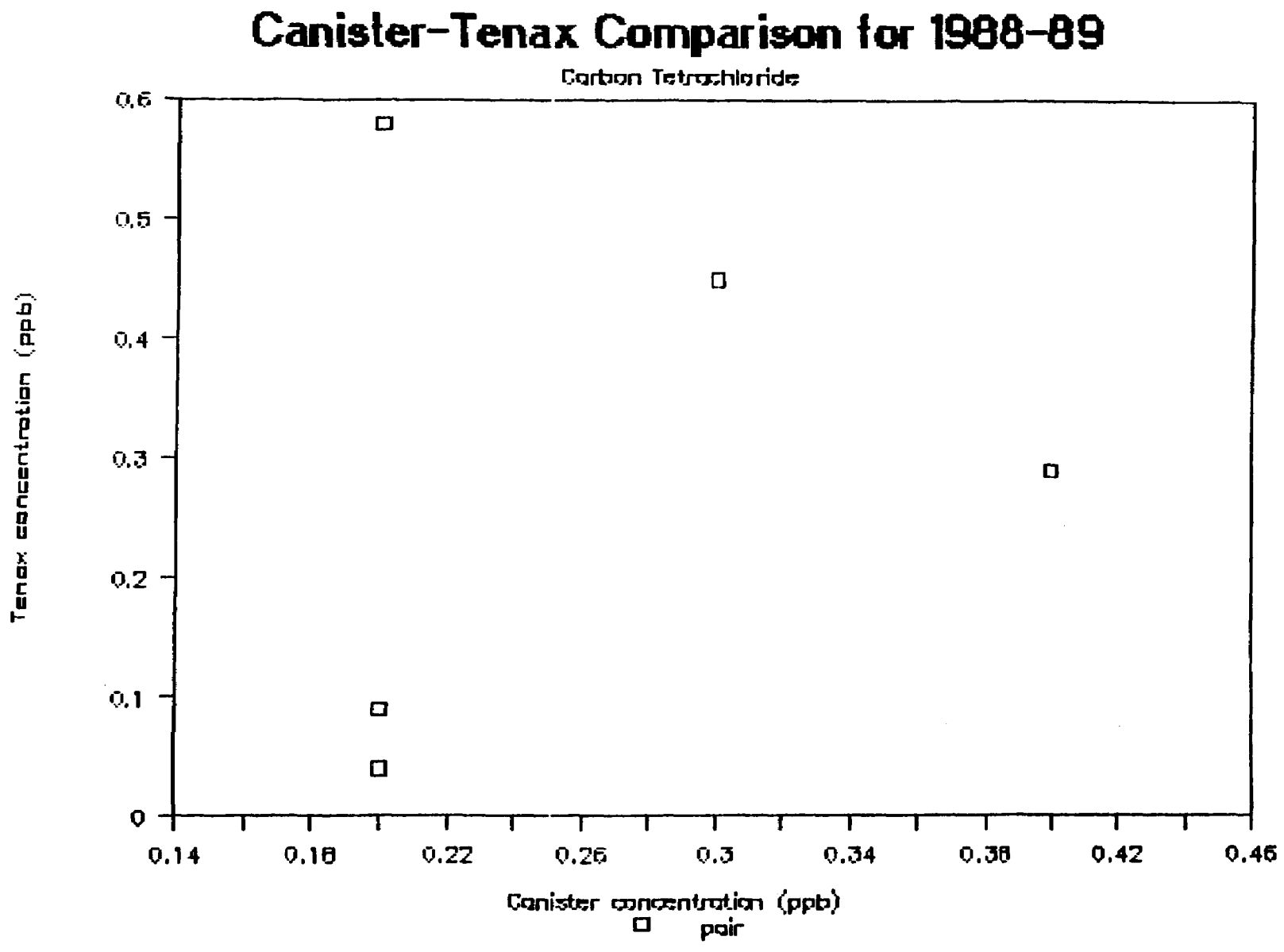

Canister-Tenax Comparison for 1988-89
Carbon Tetrachloride
Tenax concentration (ppb)
0.6
0.5
0.4
0.3
0.2
0.1
0
0.14
0.18
0.22
0.26
0.3
0.34
0.38
0.42
0.46
Canister concentration (ppb)
pair

Figure 4.4

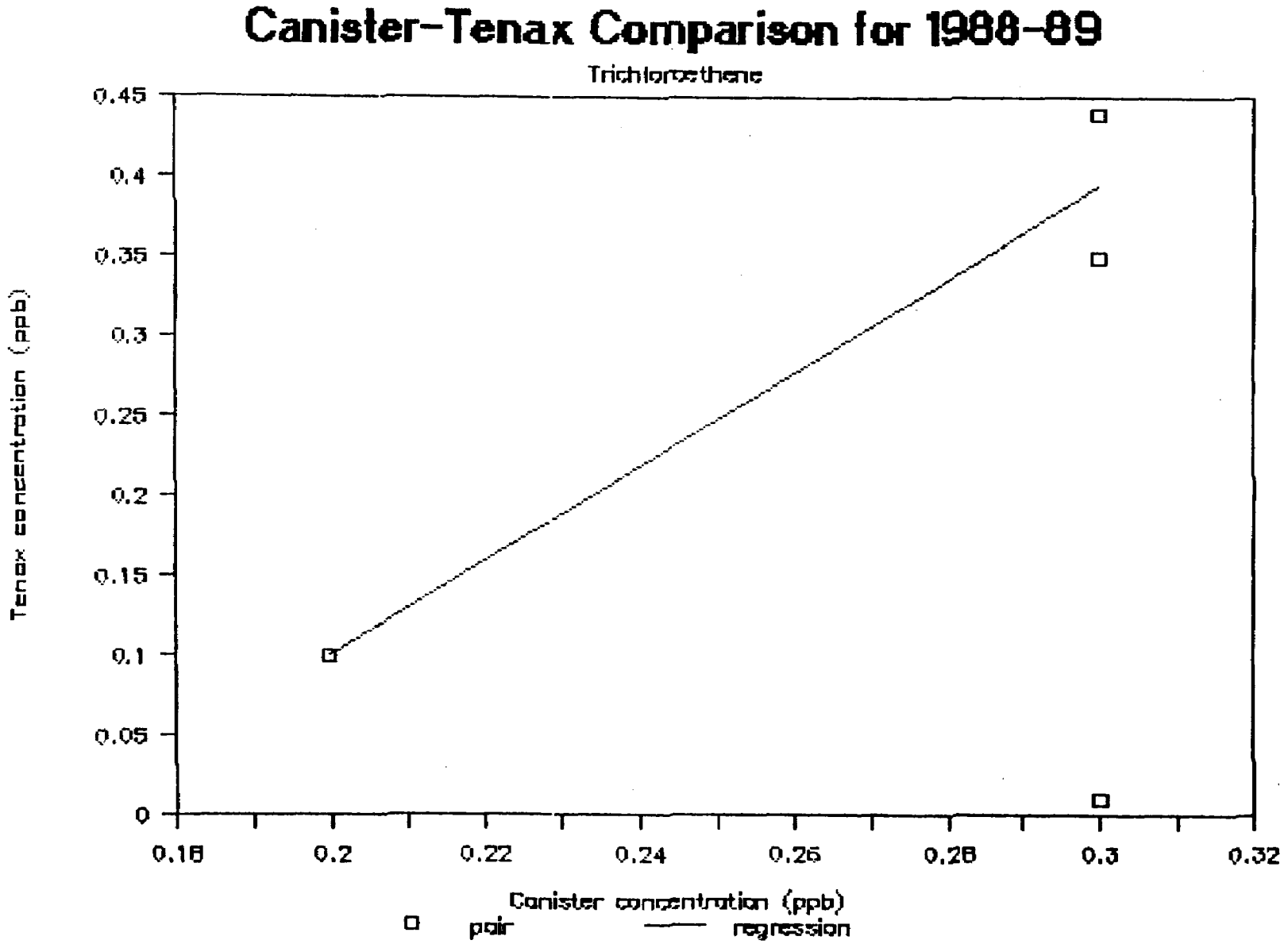

Canister-Tenax Comparison for 1988-89
Trichloroethene
Tenax concentration (ppb)
0.45
0.4
0.35
0.3
0.25
0.2
0.15
0.1
0.05
0
0.18
0.2
0.22
0.24
0.26
0.28
0.3
0.32
Canister concentration (ppb)
pair
regression

Figure 4.5

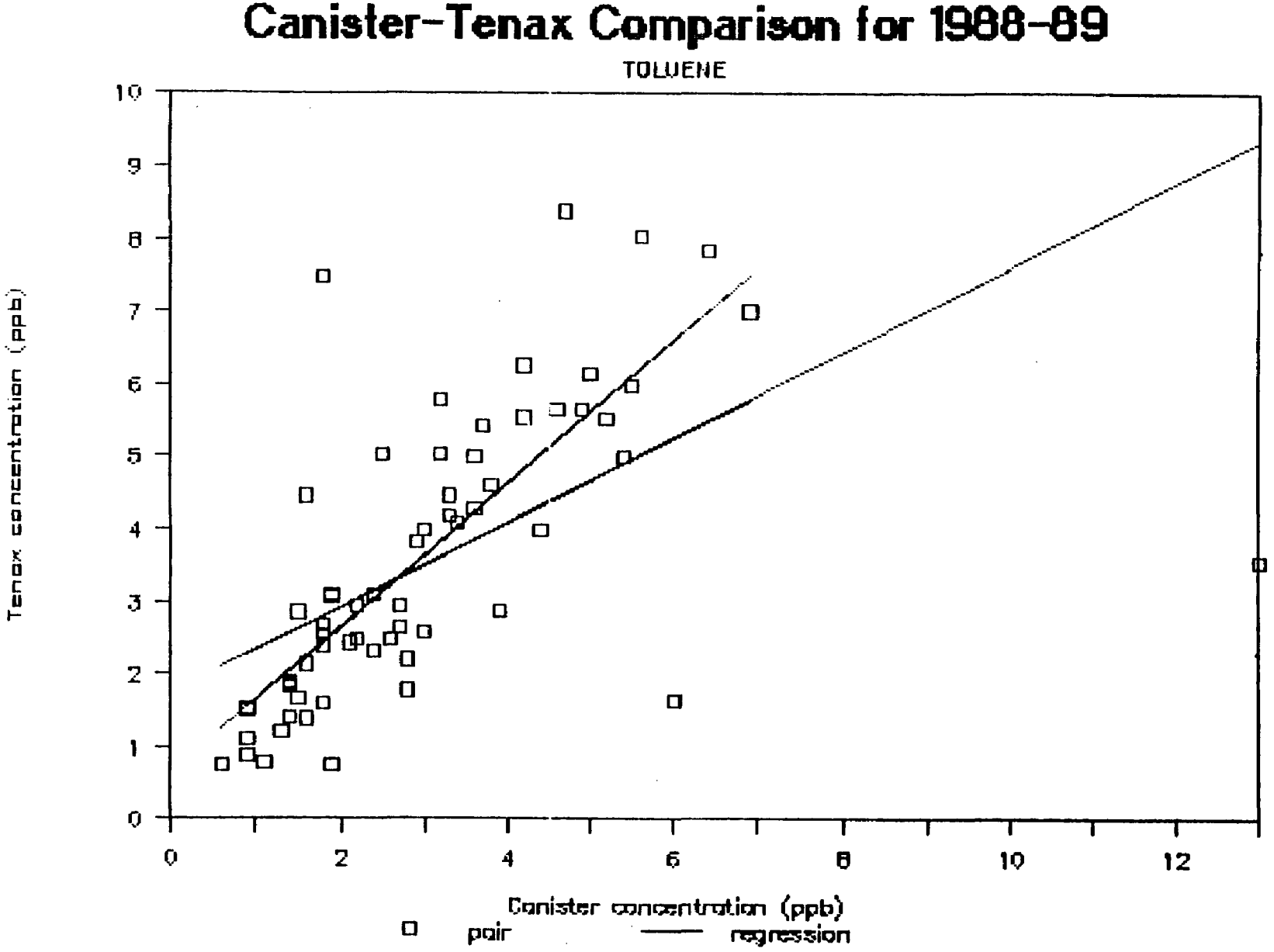

Canister-Tenax Comparison for 1988-89
TOLUENE
Tenax concentration (ppb)
Canister concentration (ppb)
pair
regression
0
1
2
3
4
5
6
7
8
9
10
0
2
4
6
8
10
12

Figure 4.6

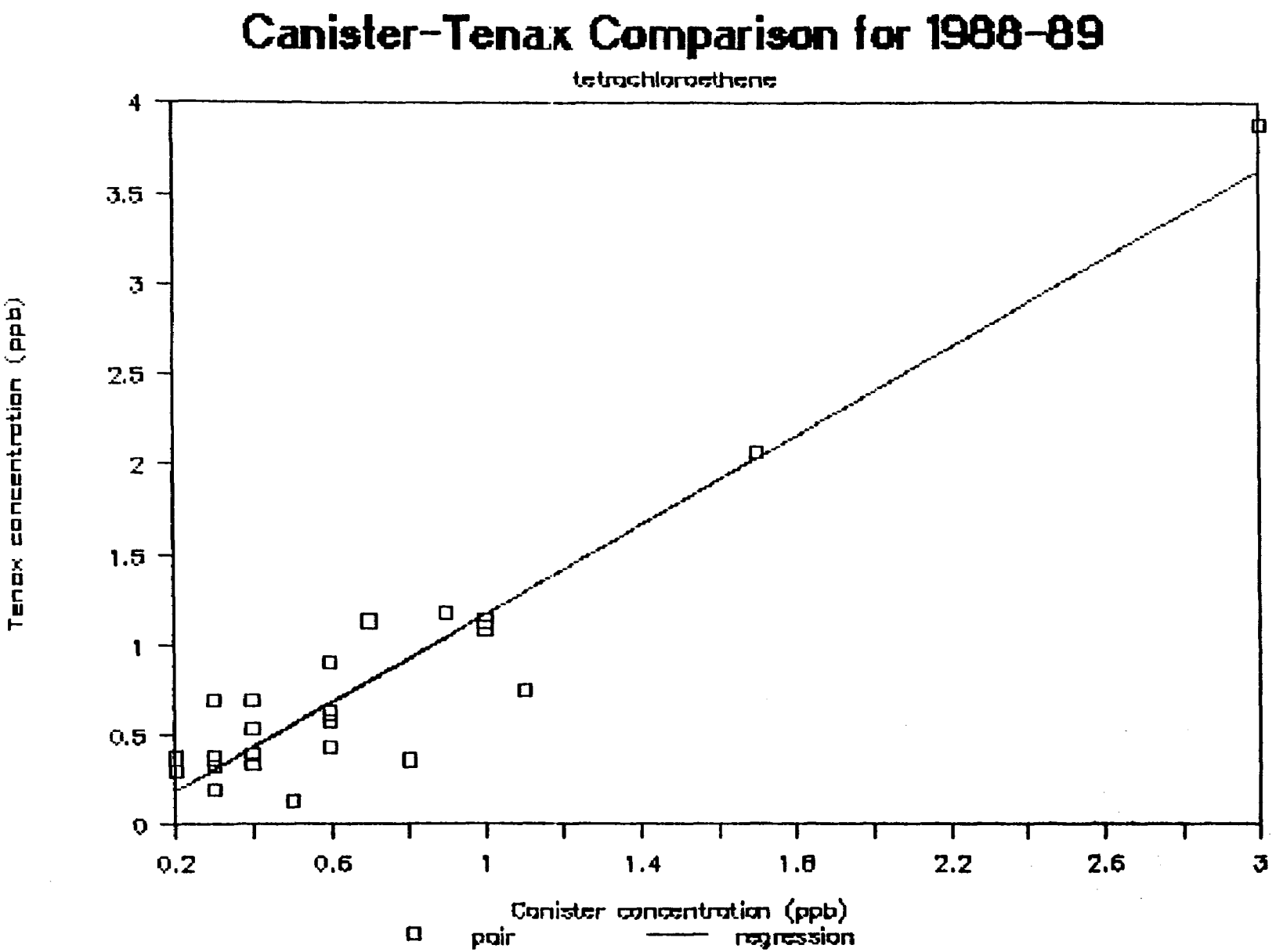

Canister-Tenax Comparison for 1988-89
tetrachloroethene
Tenax concentration (ppb)
Canister concentration (ppb)
0
0.5
1
1.5
2
2.5
3
3.5
4
0.2
0.6
1
1.4
1.8
2.2
2.6
3
pair
regression

Figure 4.7

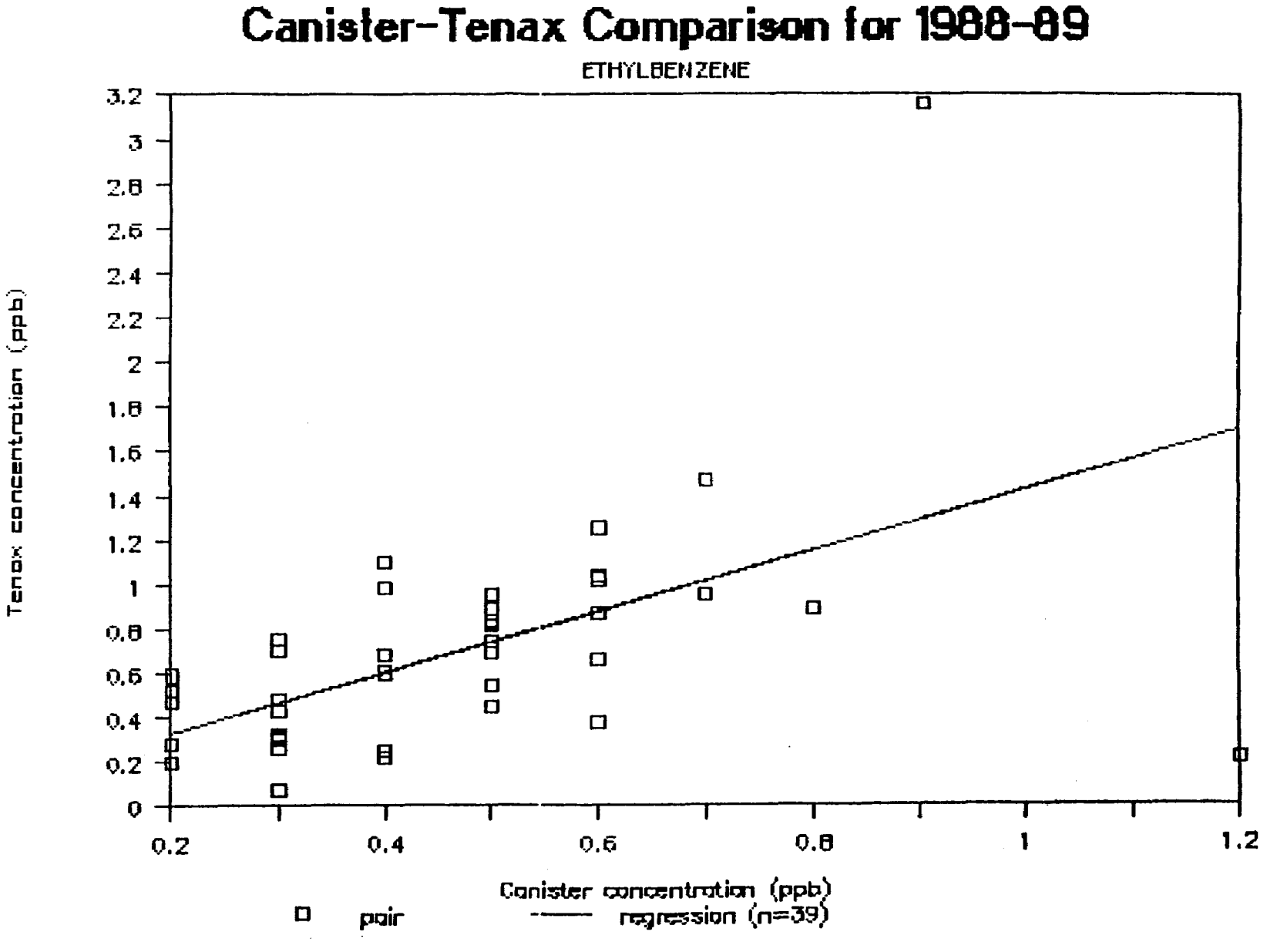

Canister-Tenax Comparison for 1988-89
ETHYLBENZENE
Tenax concentration (ppb)
3.2
3
2.8
2.6
2.4
2.2
2
1.8
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
0.2
0.4
0.6
0.8
1
1.2
Canister concentration (ppb)
pair
regression (n=39)

Figure 4.8

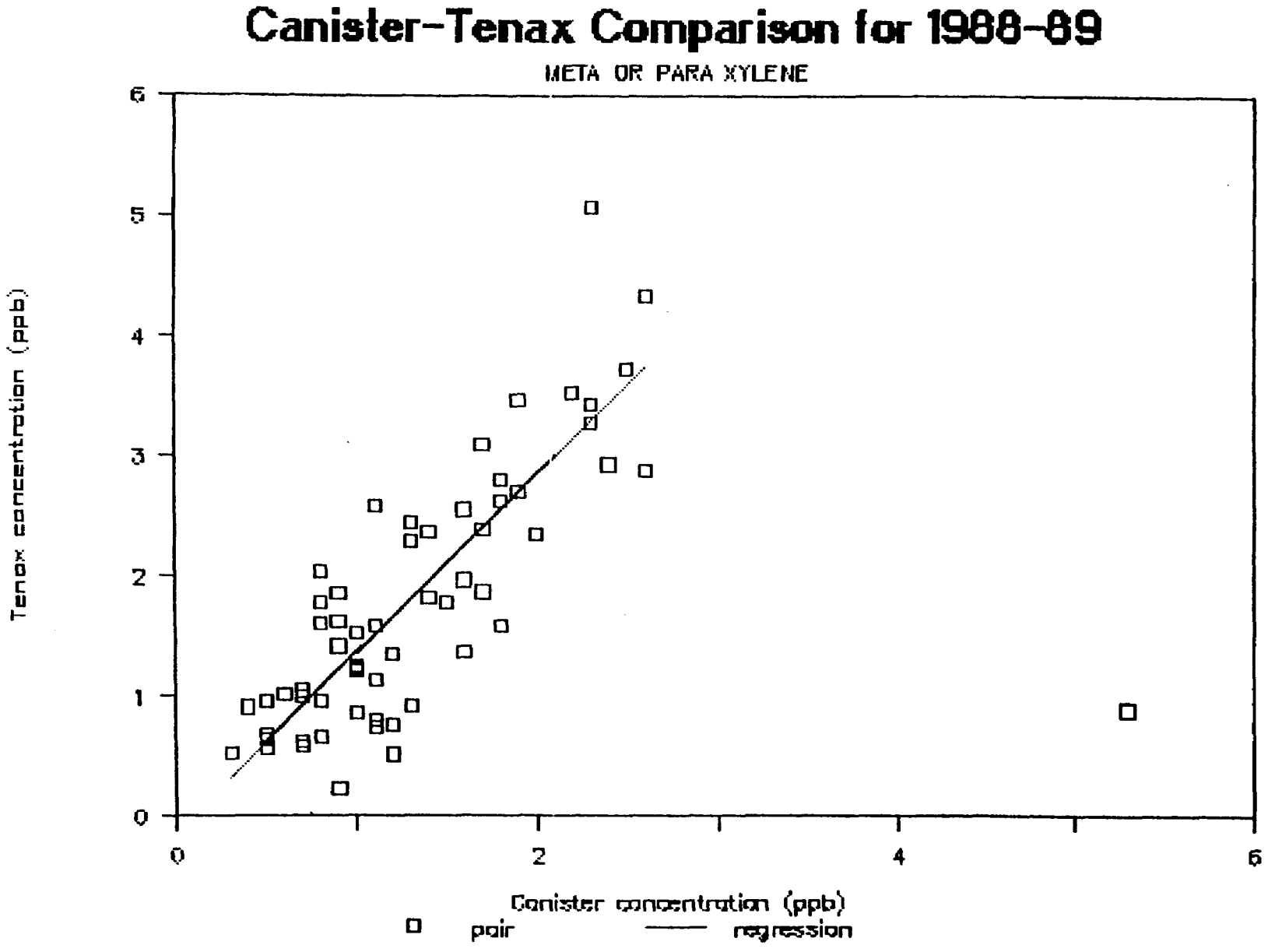

Canister-Tenax Comparison for 1988-89
META OR PARA XYLENE
Tenax concentration (ppb)
0
1
2
3
4
5
6
0
2
4
6
Canister concentration (ppb)
pair
regression

Figure 4.9

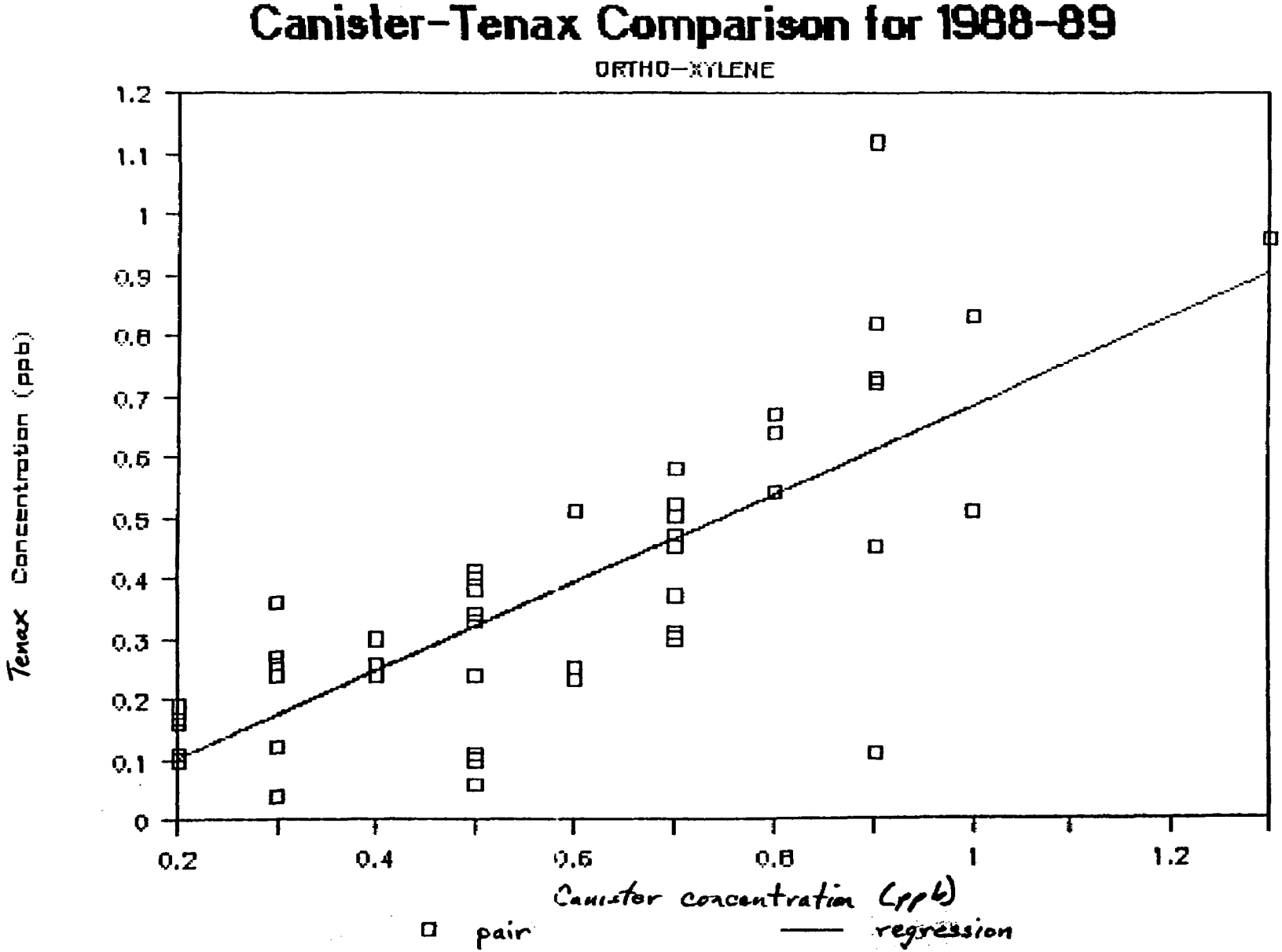

Canister-Tenax Comparison for 1988-89
ORTHO-XYLENE
Tenax Concentration (ppb)
1.2
1.1
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0.2
0.4
0.6
0.8
1
1.2
Canister concentration (ppb)
pair
regression

Figure 4.10

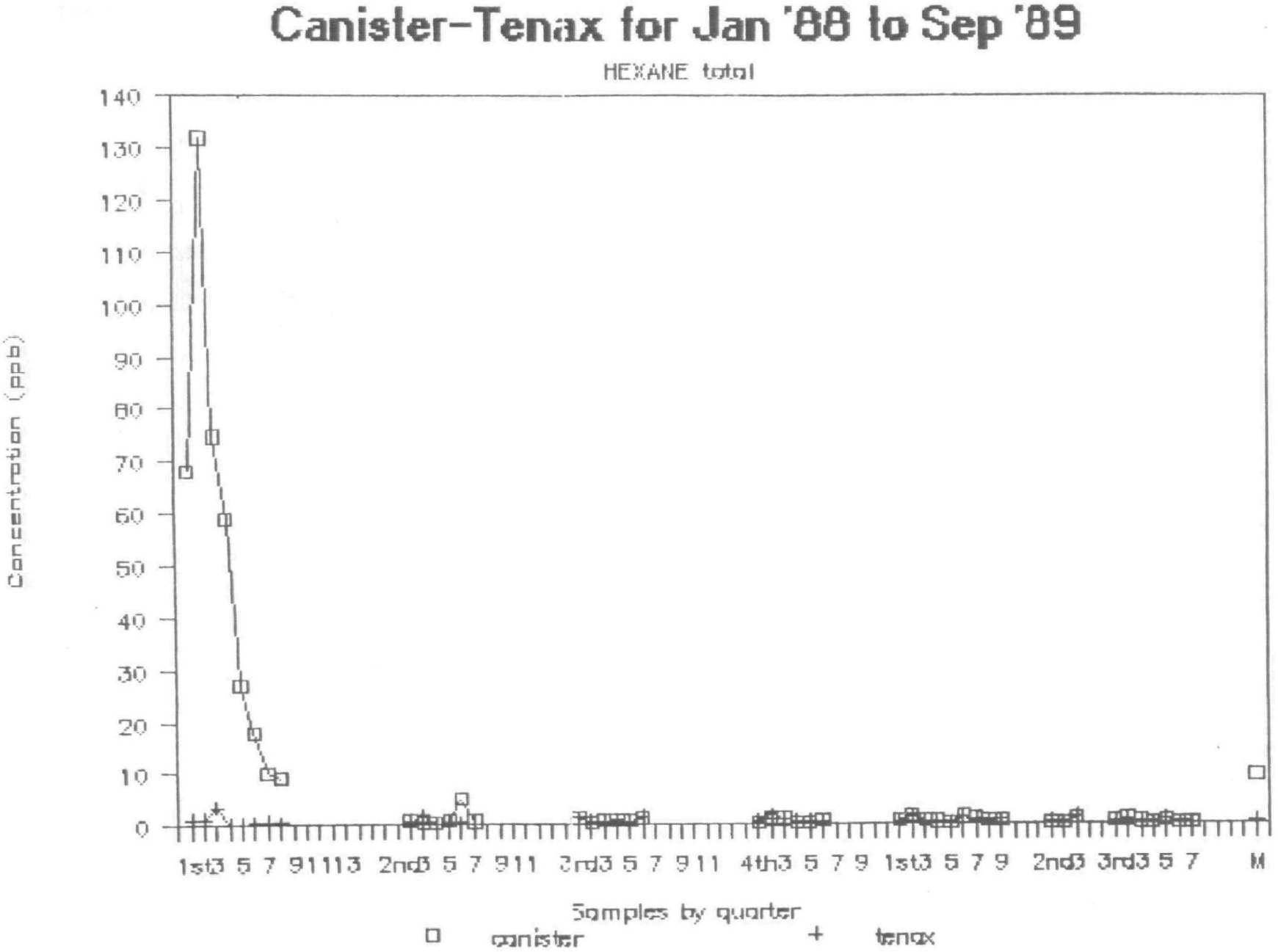

Canister-Tenax for Jan '88 to Sep '89
HEXANE total
Concentration (ppb)
140
130
120
110
100
90
80
70
60
50
40
30
20
10
0
1st3 5 7 9 11 13 2nd3 5 7 9 11 3rd3 5 7 9 11 4th3 5 7 9 1st3 5 7 9 2nd3 3rd3 5 7 M
Samples by quarter
canister
tenax

Figure 4.11a

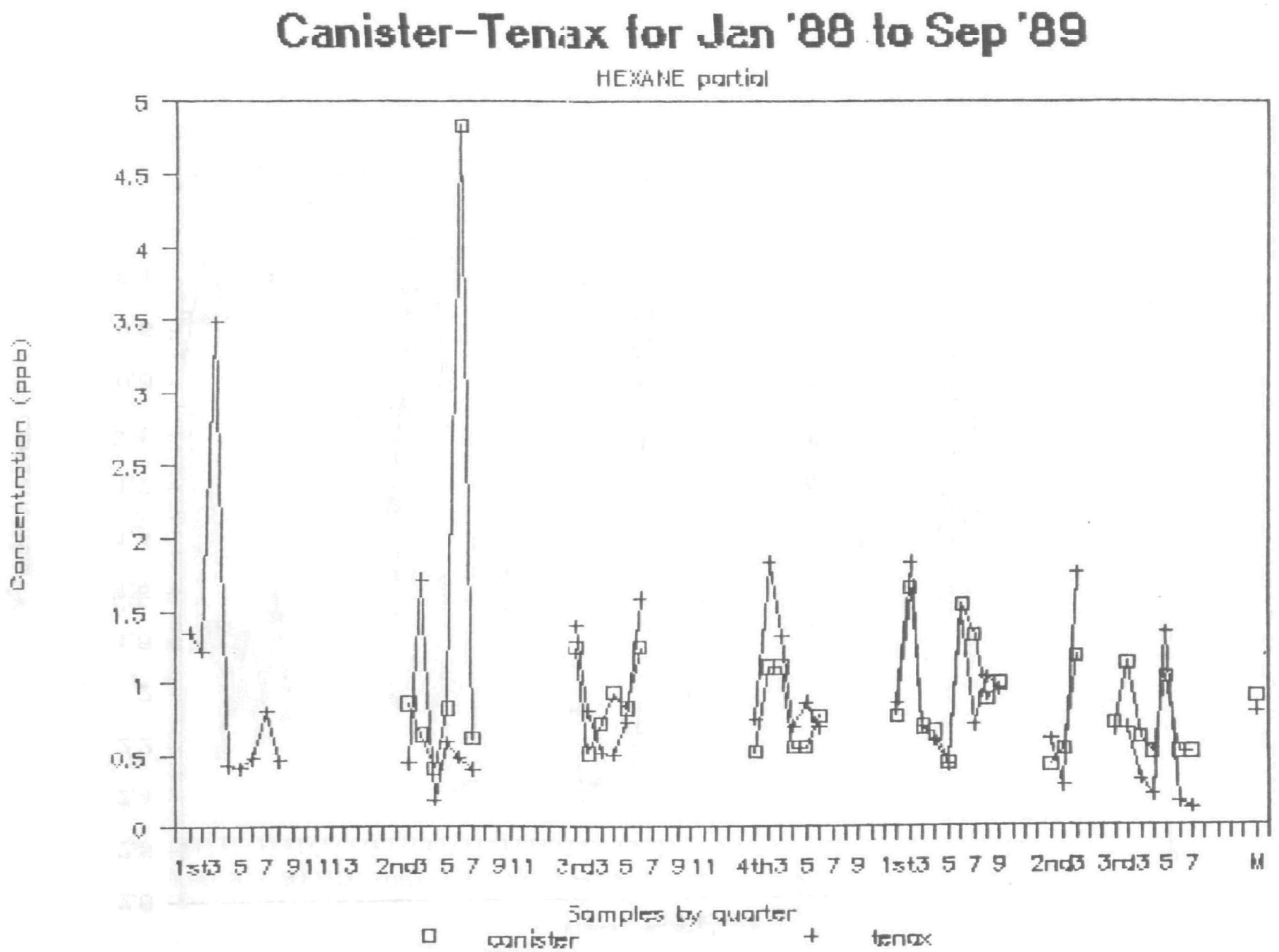

Canister-Tenax for Jan '88 to Sep '89
HEXANE partial
Concentration (ppb)
5
4.5
4
3.5
3
2.5
2
1.5
1
0.5
0
1st3 5 7 9 11 13 2nd3 5 7 9 11 3rd3 5 7 9 11 4th3 5 7 9 1st3 5 7 9 2nd3 3rd3 5 7 M
Samples by quarter
canister
tenax

Figure 4.11b

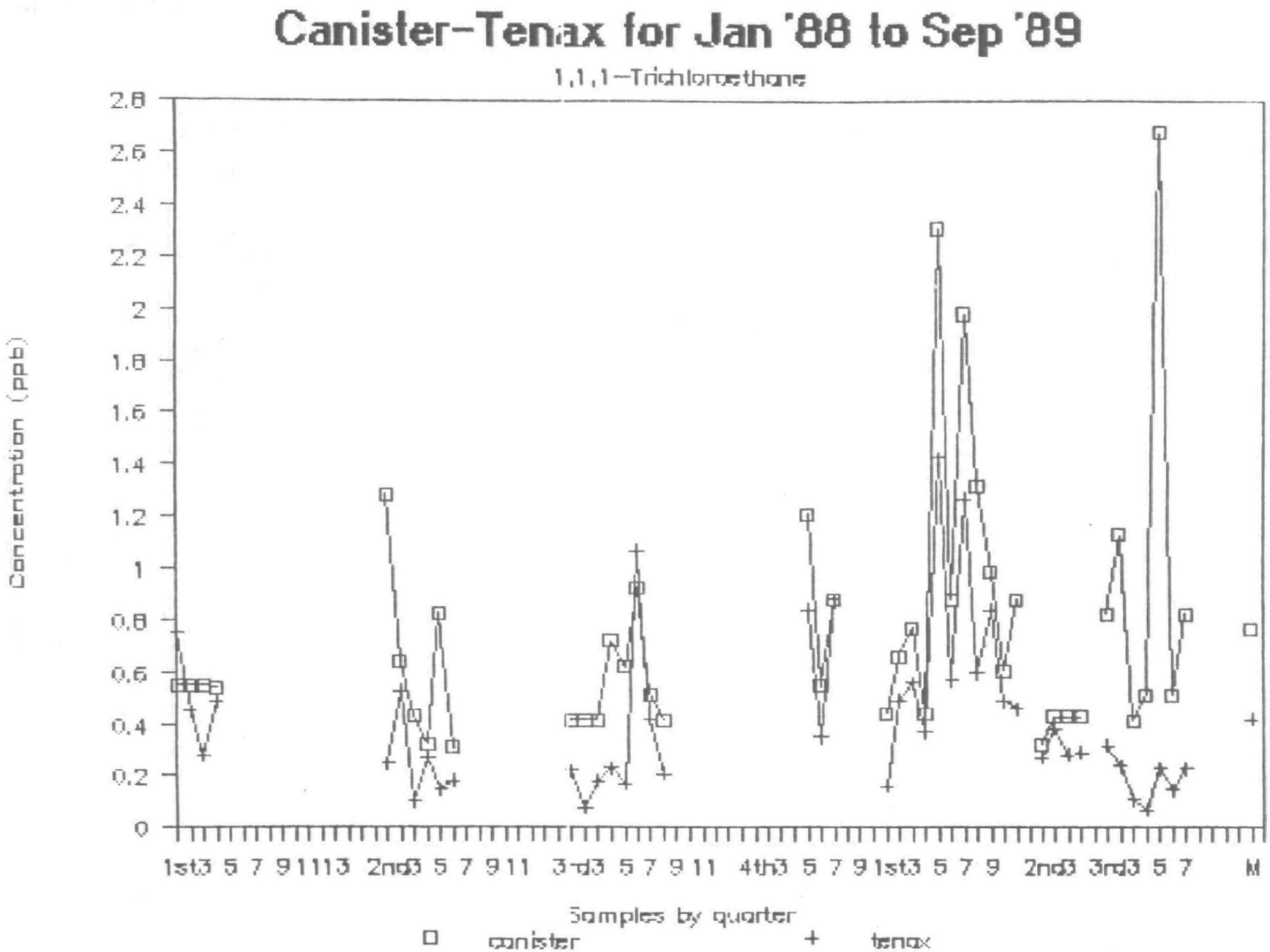

Canister-Tenax for Jan '88 to Sep '89
1,1,1-Trichloroethane
Concentration (ppb)
2.8
2.6
2.4
2.2
2
1.8
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
1st3 5 7 9 11 13 2nd3 5 7 9 11 3rd3 5 7 9 11 4th3 5 7 9 1st3 5 7 9 2nd3 3rd3 5 7 M
Samples by quarter
canister
tenax

Figure 4.12

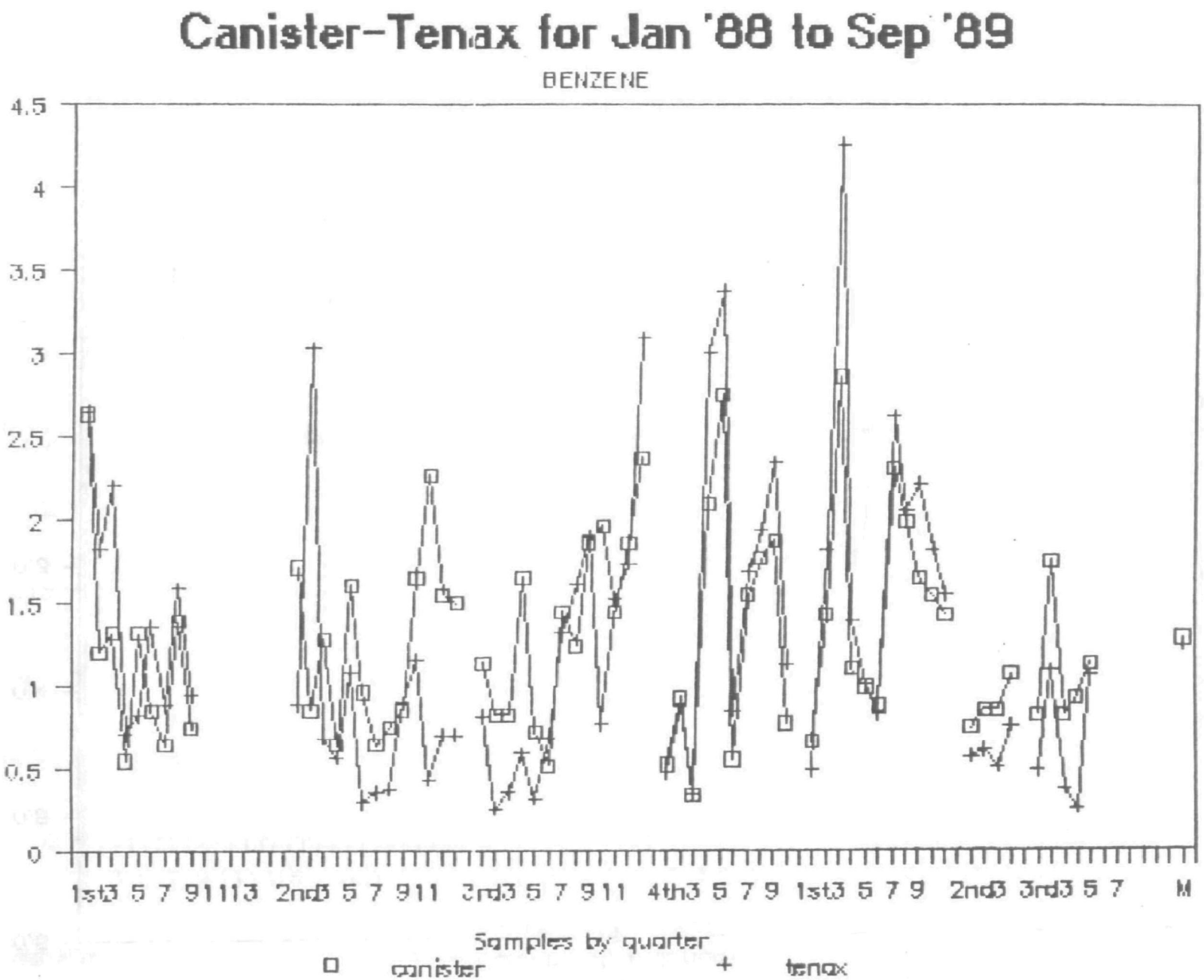

Canister-Tenax for Jan '88 to Sep '89
BENZENE
Concentration (ppb)
4.5
4
3.5
3
2.5
2
1.5
1
0.5
0
1st3 5 7 9 11 13 2nd3 5 7 9 11 3rd3 5 7 9 11 4th3 5 7 9 1st3 5 7 9 2nd3 3rd3 5 7 M
Samples by quarter
canister
tenax

Figure 4.13

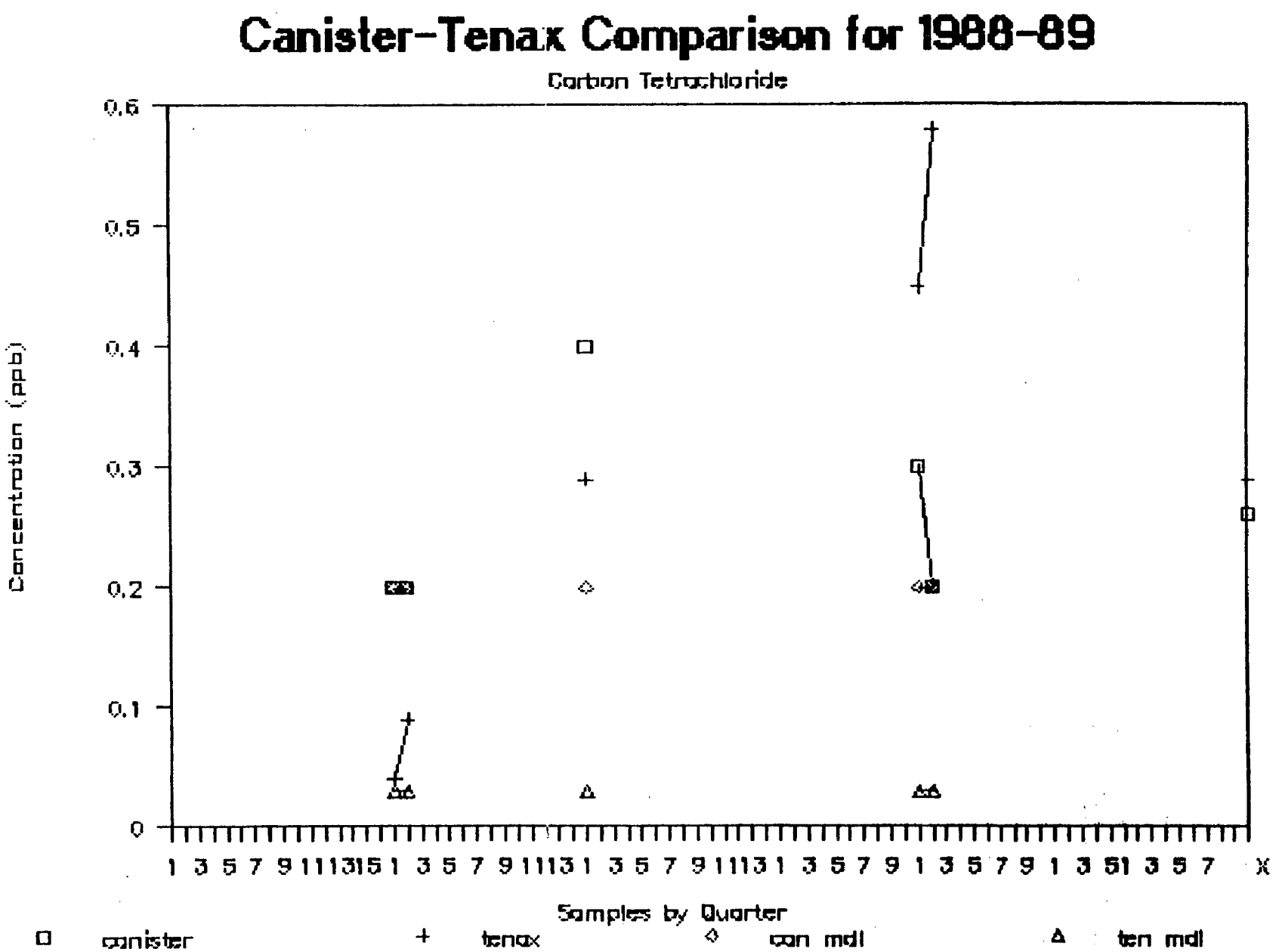

Canister-Tenax Comparison for 1988-89
Carbon Tetrachloride
Concentration (ppb)
0.6
0.5
0.4
0.3
0.2
0.1
0
1 3 5 7 9 11 13 15 1 3 5 7 9 11 13 1 3 5 7 9 11 13 1 3 5 7 9 1 3 5 7 9 1 3 5 7 X
Samples by Quarter
canister
tenax
can mdl
ten mdl

Figure 4.14

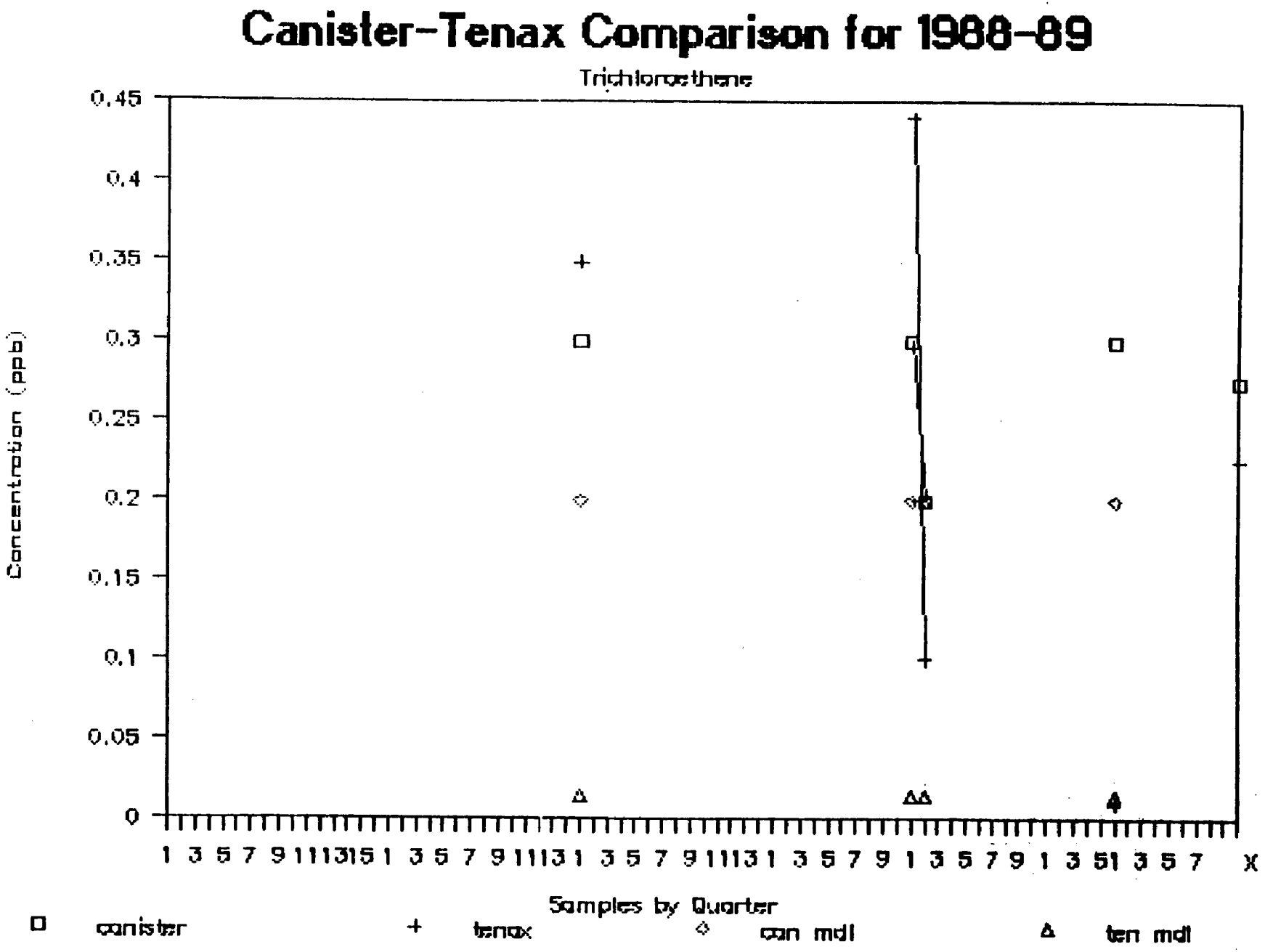

Canister-Tenax Comparison for 1988-89
Trichloroethene
Concentration (ppb)
0.45
0.4
0.35
0.3
0.25
0.2
0.15
0.1
0.05
0
1 3 5 7 9 11 13 15 1 3 5 7 9 11 13 1 3 5 7 9 11 13 1 3 5 7 9 1 3 5 7 9 1 3 5 7 X
Samples by Quarter
canister
tenax
can mdl
ten mdl

Figure 4.15

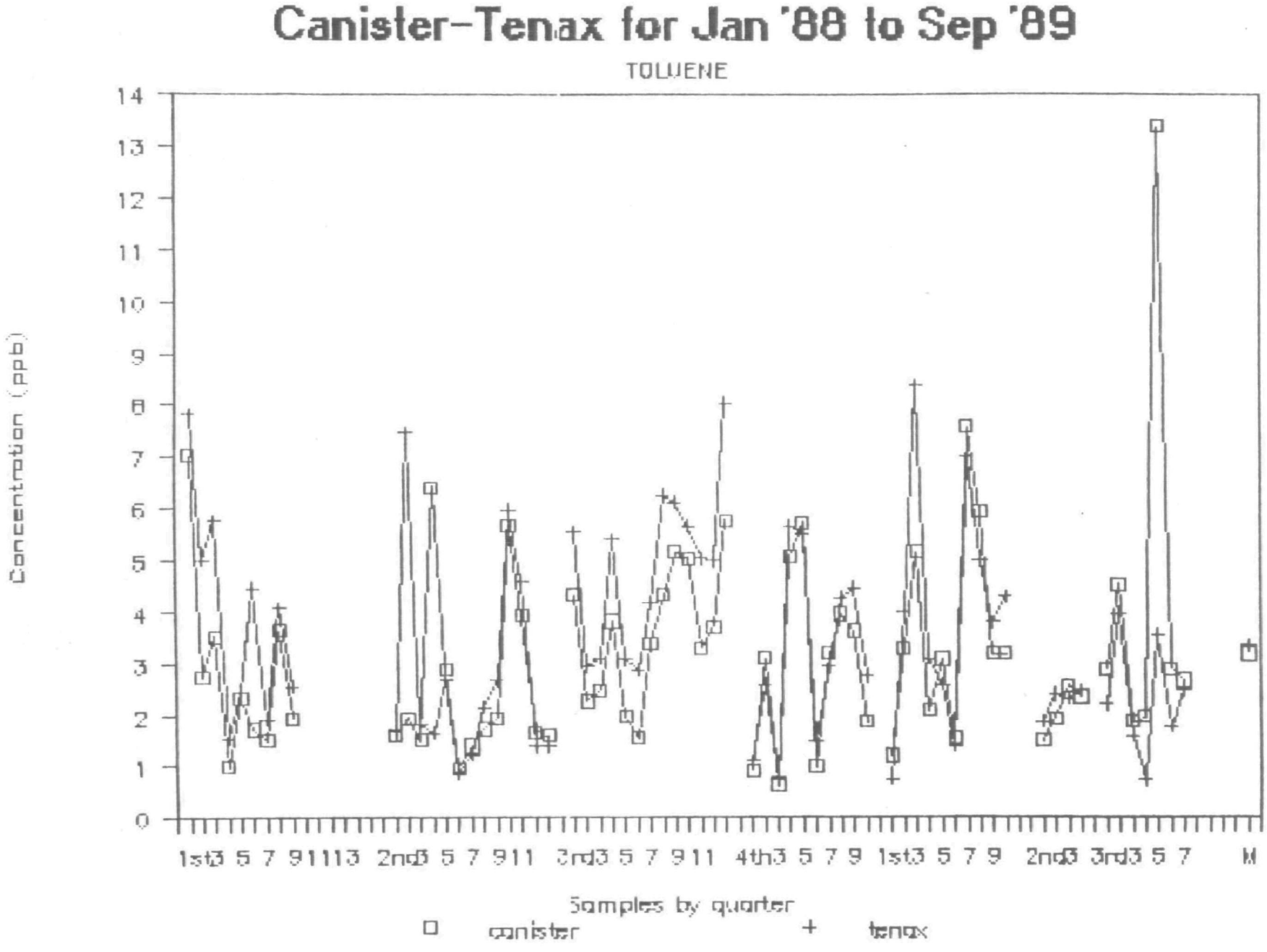

Canister-Tenax for Jan '88 to Sep '89
TOLUENE
Concentration (ppb)
0
1
2
3
4
5
6
7
8
9
10
11
12
13
14
1st3 5 7 9 11 13 2nd3 5 7 9 11 3rd3 5 7 9 11 4th3 5 7 9 1st3 5 7 9 2nd3 3rd3 5 7 M
Samples by quarter
canister
tenax

Figure 4.16

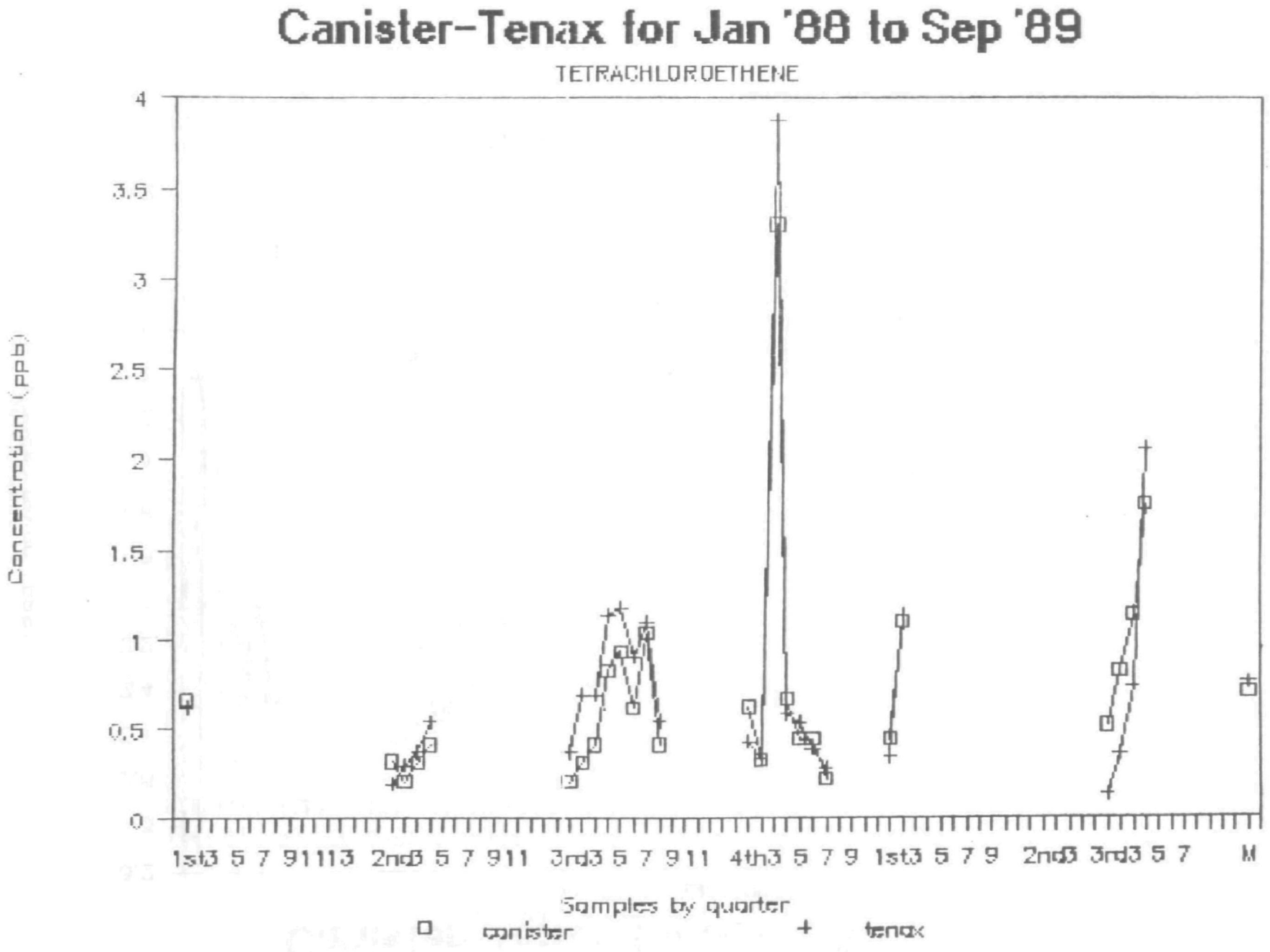

Canister-Tenax for Jan '88 to Sep '89
TETRACHLOROETHENE
Concentration (ppb)
4
3.5
3
2.5
2
1.5
1
0.5
0
1st3 5 7 9 11 13 2nd3 5 7 9 11 3rd3 5 7 9 11 4th3 5 7 9 1st3 5 7 9 2nd3 3rd3 5 7 M
Samples by quarter
canister
tenax

Figure 4.17

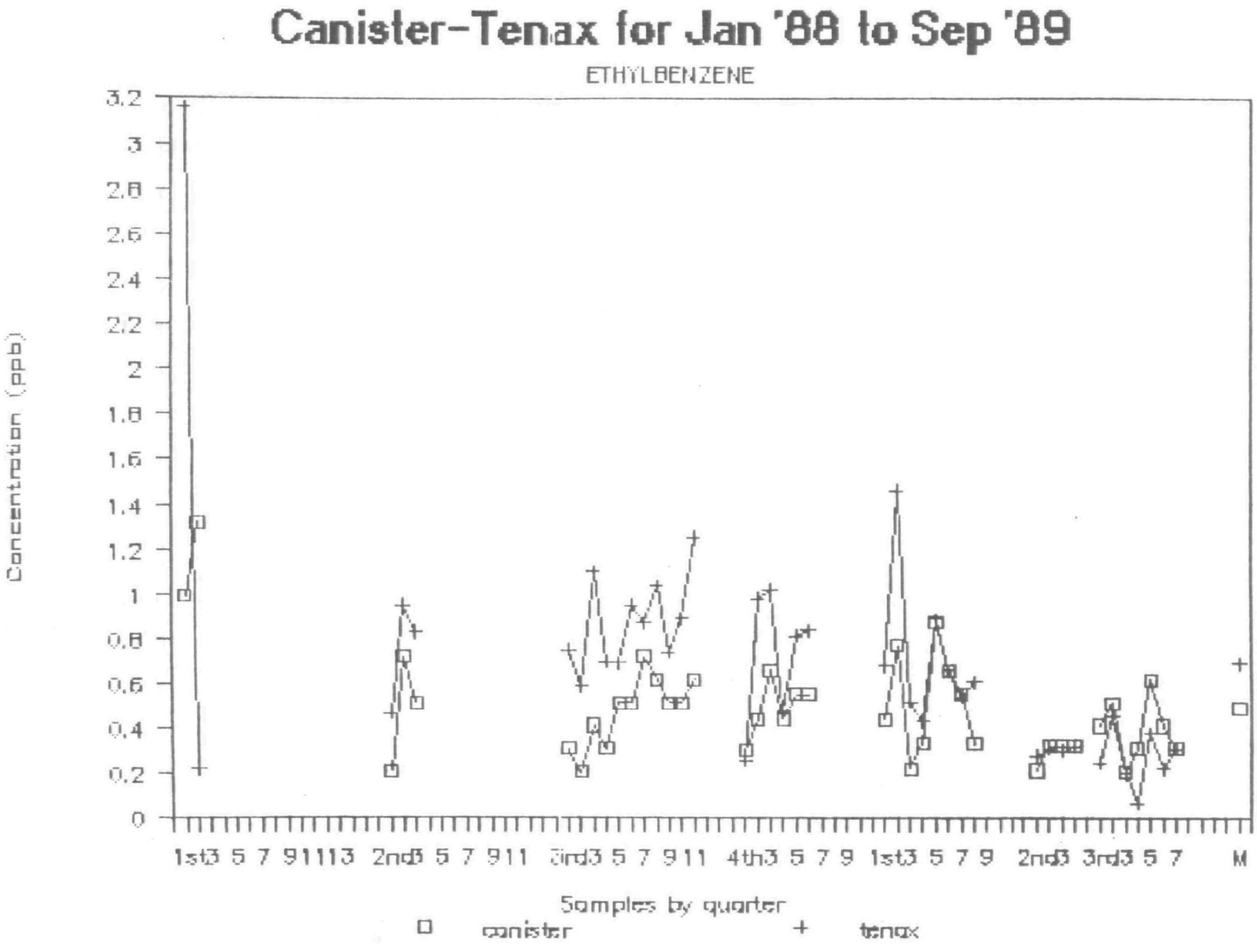

Canister-Tenax for Jan '88 to Sep '89
ETHYLBENZENE
Concentration (ppb)
3.2
3
2.8
2.6
2.4
2.2
2
1.8
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
1st3 5 7 9 11 13 2nd3 5 7 9 11 3rd3 5 7 9 11 4th3 5 7 9 1st3 5 7 9 2nd3 3rd3 5 7 M
Samples by quarter
canister
tenax

Figure 4.18

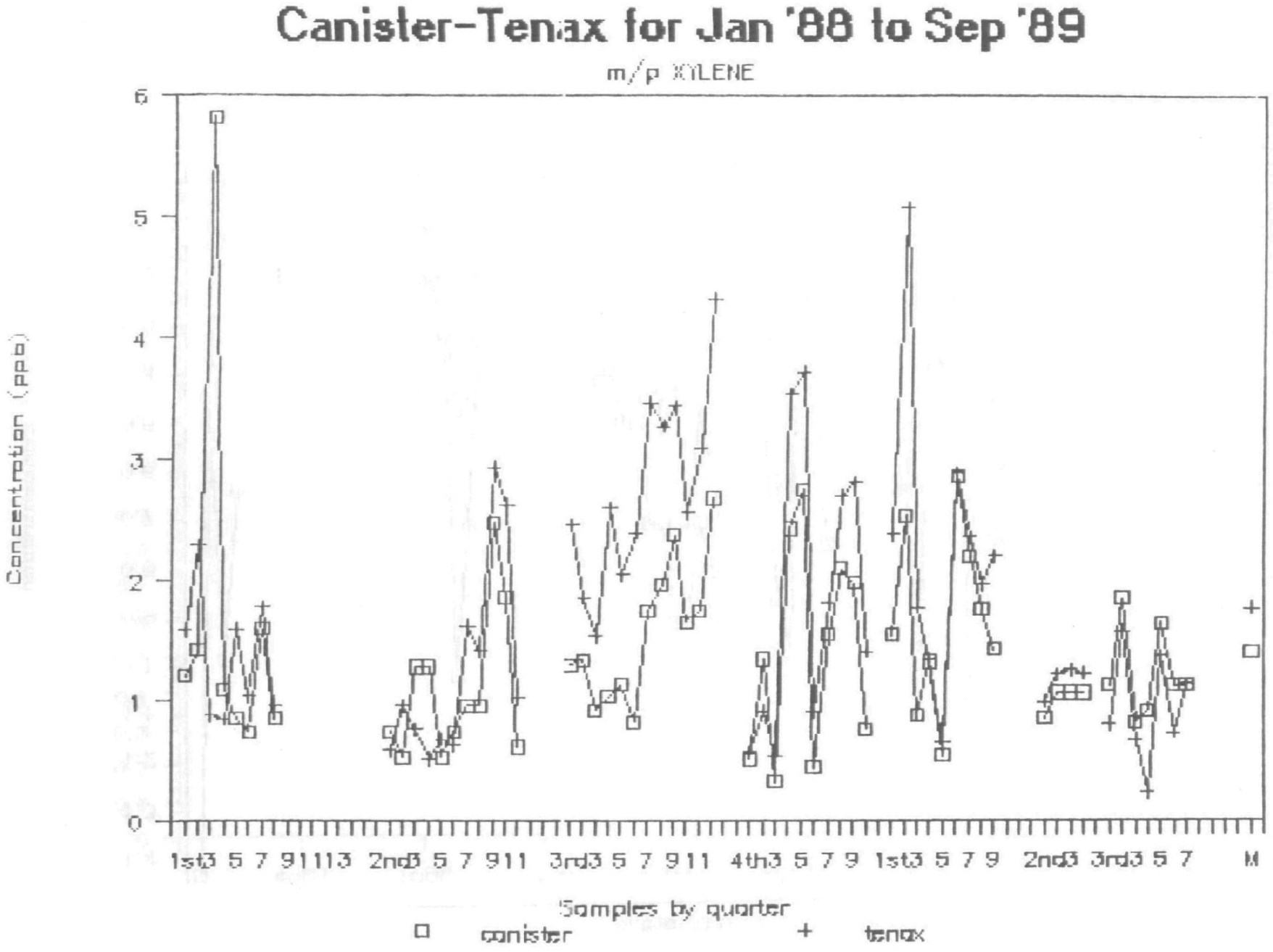

Canister-Tenax for Jan '88 to Sep '89
m/p XYLENE
Concentration (ppb)
0
1
2
3
4
5
6
1st3 5 7 9 11 1 3 2nd3 5 7 9 11 3rd3 5 7 9 11 4th3 5 7 9 1st3 5 7 9 2nd3 3rd3 5 7 M
Samples by quarter
canister
tenax

Figure 4.19

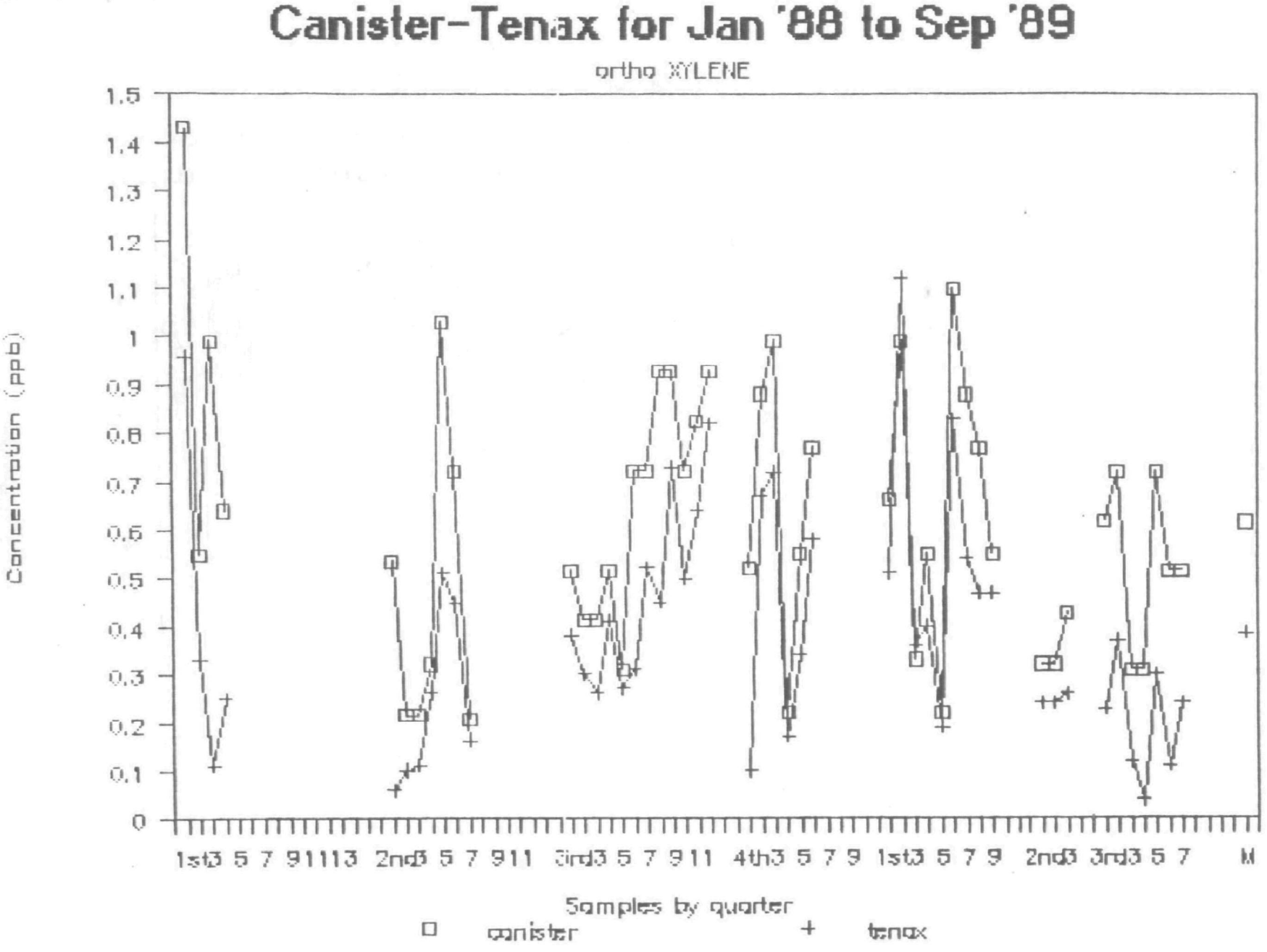

Canister-Tenax for Jan '88 to Sep '89
ortho XYLENE
Concentration (ppb)
1.5
1.4
1.3
1.2
1.1
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
1st3 5 7 9 11 13 2nd3 5 7 9 11 3rd3 5 7 9 11 4th3 5 7 9 1st3 5 7 9 2nd3 3rd3 5 7 M
Samples by quarter
canister
tenax

Figure 4.20

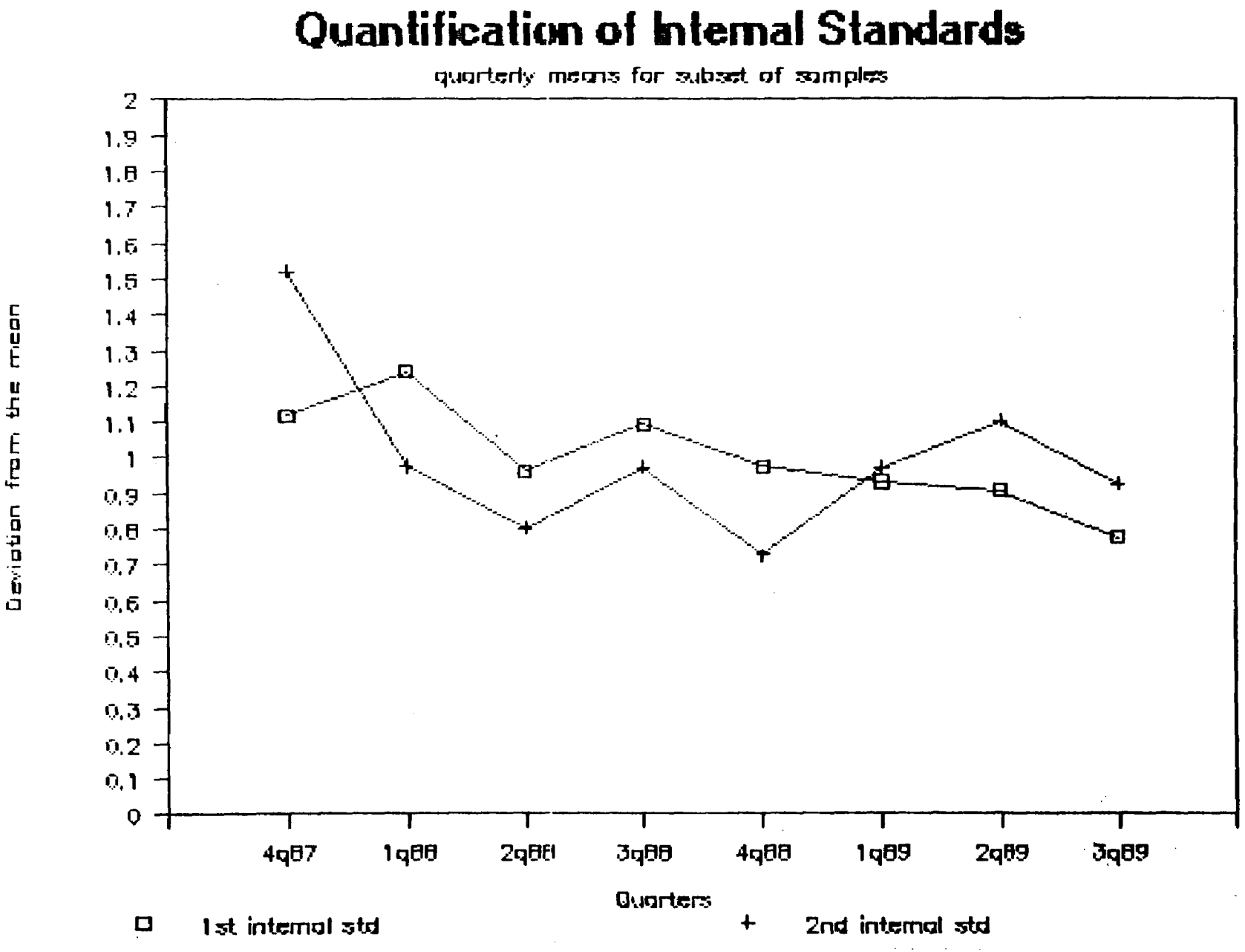

Quantification of Internal Standards
quarterly means for subset of samples
Deviation from the mean
2
1.9
1.8
1.7
1.6
1.5
1.4
1.3
1.2
1.1
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
4q87
1q88
2q88
3q88
4q88
1q89
2q89
3q89
Quarters
1st internal std
2nd internal std

Figure 7.1

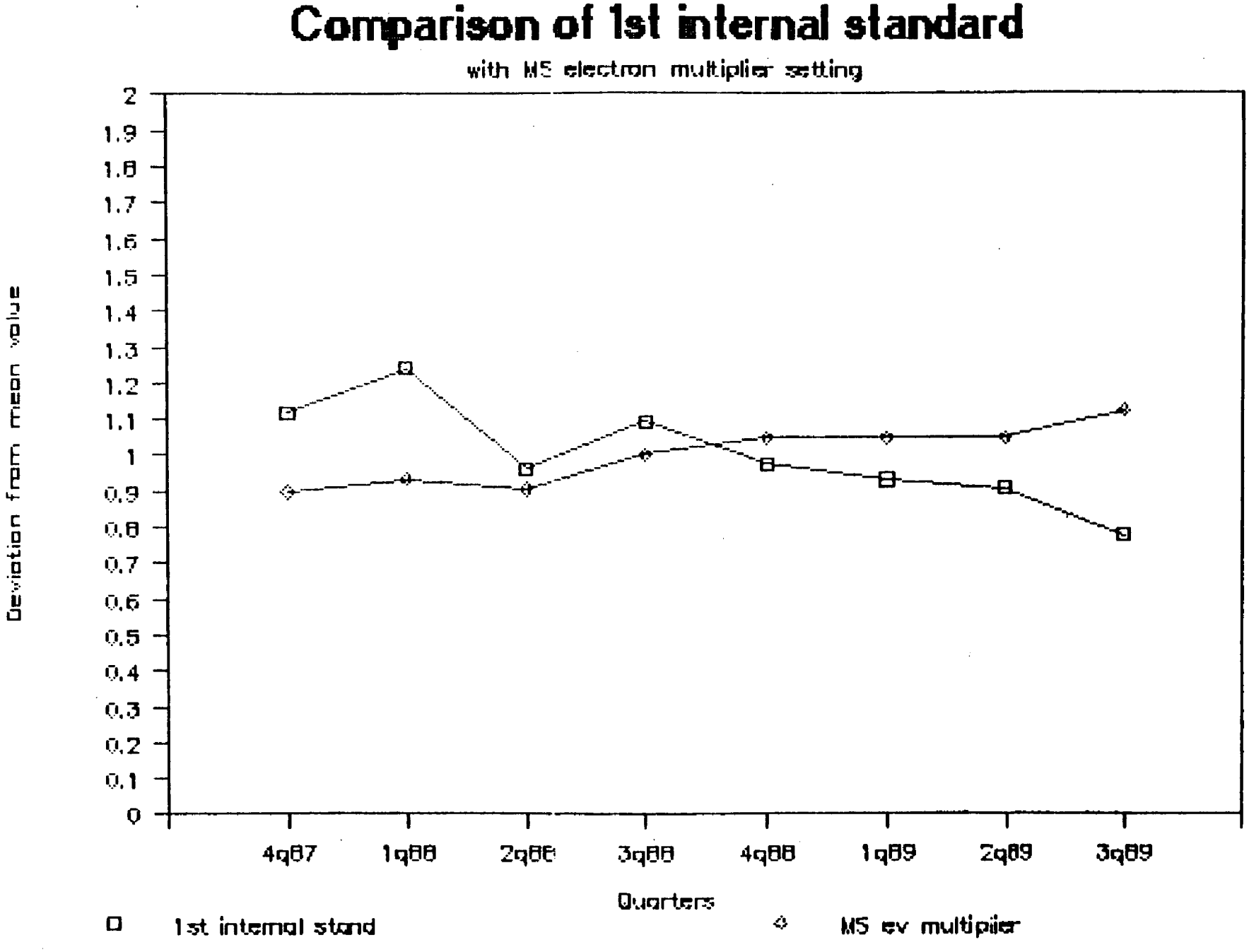

Comparison of 1st internal standard
with M5 electron multiplier setting
Deviation from mean value
2
1.9
1.8
1.7
1.6
1.5
1.4
1.3
1.2
1.1
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
4q87
1q88
2q88
3q88
4q88
1q89
2q89
3q89
Quarters
1st internal stand
M5 ev multiplier

Figure 7.2

10. NJIT VOCS QUALITY ASSURANCE REPORT

New Jersey Institute of Technology

Air Pollution Research Laboratory

Final Quality Assurance Report

Staten Island/Northern New Jersey Air Toxics Project

April 1990

Dr. B. Kebbekus
Professor of Chemistry
Department of Chemical Engineering, Chemistry
and
Environmental Sciences

Chapter 1

Blanks

Tenax:

Blanks are done on each batch of Tenax traps before they are used for sampling. These blank data are not kept or reported, since they are simply used to determine that the set of traps on the manifold has been properly cleaned. The blanks which accompany each day's set of samples are the blanks which are reported here, and which are used for the correction of data. The data show that the average quantity of individual target compounds varied considerably over the course of the project. However, the levels were usually below 1 ng/trap, with benzene, toluene and xylene occasionally rising as high as 5 ng. These amounts are usually much less than 10% of the amount found in samples, and have not posed a problem.

Canisters:

The canisters are each blanked before being sent to the field. No measurable conatminaton is expected in the canisters, and they are not used if there is any measurable amount of the target compounds. The only major problem which arose with blanks in the canister system was the problem with methylene chloride contamination in one of the bellows pumps. This contamination was impossible to remove, although extensive purging and mild warming of the pump was tried. Finally, a considerable amount of data for this compound at the Carteret site was lost because of the contamination of the pump. Since the daily blanks for the canisters could not show problems with the sampling system and pumps on site, the problem continued over a major period of time before it was detected and its source determined.

Blank Data:

The data on the Tenax blanks is shown in Tables 1-1 to 1-9. These tables show the average quantity in nanograms for each compound over each quarter of sampling. The standard deviation of each group of data is given and the limits of the range of blanks at the 95% confidence level is also calculated. Figures 1-1 to 1-4 show the blanks for several compound plotted against time, showing that elevated blanks tend to come in sequential groups. This is an indication that the techniques which lead to high quality analyses by this method are sometimes quite subtle, and the causes of deviations are not always obvious. Remediation of these problems takes time and careful checking.

In the smaller volume samples, those taken at the lower flow on Tenax, a 1 ppb sample contains approximately 20 ng of the compound, so it can be seen that the blanks were not excessive.

Table 1-1

Organization: NJIT Sorbent: Tenax

QUARTER OF Jul. TO Sep. , 1987

BLANKS

	# Blanks Run	Avg. (ng)	Std.Dev (ng)	95% CL Interval (ng)	
1.MeCL	12	0.30	0.62	-0.09	0.69
2.DCM	12	0.00	0.00	0.00	0.00
3.C6	12	0.69	1.13	-0.02	1.40
4.CFor	12	0.00	0.00	0.00	0.00
5.111	12	0.56	0.79	0.06	1.06
6.Bz	12	1.84	1.72	0.76	2.92
7.CCl4	12	0.00	0.00	0.00	0.00
8.Tric	12	0.00	0.00	0.00	0.00
9.Tol	12	1.18	0.82	0.66	1.70
10.Perc.	12	0.09	0.17	-0.02	0.20
11.pmX	12	0.70	0.90	0.13	1.27
12.oX	12	1.02	0.61	0.64	1.40

Table 1-2

Organization: NJIT Sorbent: Tenax

QUARTER OF Oct. TO Dec. , 1987

BLANKS

	# Blanks Run	Avg. (ng)	Std.Dev (ng)	95% CL Interval (ng)	
1.MeCL	12	0.08	0.23	-0.06	0.22
2.DCM	12	0.00	0.00	0.00	0.00
3.C6	12	0.24	0.73	-0.22	0.70
4.CFor	12	0.00	0.00	0.00	0.00
5.111	12	0.00	0.00	0.00	0.00
6.Bz	12	0.62	1.36	-0.24	1.48
7.CCl4	12	0.00	0.00	0.00	0.00
8.Tric	12	0.00	0.00	0.00	0.00
9.Tol	12	0.17	0.33	-0.04	0.38
10.Perc.	12	0.00	0.00	0.00	0.00
11.pmX	12	0.15	0.36	-0.08	0.38
12.oX	12	0.16	0.44	-0.12	0.44

Table 1-3

Organization: NJIT Sorbent: Tenax

QUARTER OF Jan. TO Mar. , 1988

BLANKS

	# Blanks Run	Avg. (ng)	Std.Dev (ng)	95% CL Interval (ng)	
1.MeCL	15	0.02	0.08	-0.02	0.06
2.DCM	15	0.00	0.00	0.00	0.00
3.C6	15	0.27	0.38	0.06	0.48
4.CFor	15	0.00	0.00	0.00	0.00
5.111	15	0.14	0.42	-0.09	0.37
6.Bz	15	0.86	1.22	0.19	1.53
7.CCl4	15	0.00	0.00	0.00	0.00
8.Tric	15	0.00	0.00	0.00	0.00
9.Tol	15	0.27	0.47	0.01	0.53
10.Perc.	15	0.00	0.00	0.00	0.00
11.pmX	15	0.09	0.26	-0.05	0.23
12.oX	15	0.30	0.63	-0.05	0.65

Table 1-4

Organization: NJIT Sorbent: Tenax

QUARTER OF Apr. TO Jun. , 1988

BLANKS

	# Blanks Run	Avg. (ng)	Std.Dev (ng)	95% CL Interval (ng)	
1.MeCL	14	0.14	0.38	-0.08	0.36
2.DCM	14	0.00	0.00	0.00	0.00
3.C6	14	0.10	0.19	-0.01	0.21
4.CFor	14	0.00	0.00	0.00	0.00
5.111	14	0.15	0.54	-0.16	0.46
6.Bz	14	1.44	1.64	0.50	2.38
7.CCl4	14	0.00	0.00	0.00	0.00
8.Tric	14	0.00	0.00	0.00	0.00
9.Tol	14	0.72	0.86	0.22	1.21
10.Perc.	14	0.00	0.00	0.00	0.00
11.pmX	14	0.70	1.06	0.09	1.31
12.oX	14	0.70	0.90	0.18	1.21

Table 1-5

Organization: NJIT Sorbent: Tenax

QUARTER OF Jul. TO Sep. , 1988

BLANKS

	# Blanks Run	Avg. (ng)	Std.Dev (ng)	95% CL Interval (ng)	
1.MeCL	15	1.81	1.08	1.22	2.40
2.DCM	15	0.00	0.00	0.00	0.00
3.C6	15	2.50	1.10	1.90	3.10
4.CFor	15	0.00	0.00	0.00	0.00
5.111	15	0.07	0.18	-0.03	0.17
6.Bz	15	3.63	1.48	2.82	4.44
7.CCl4	15	0.00	0.00	0.00	0.00
8.Tric	15	0.00	0.00	0.00	0.00
9.Tol	15	1.04	0.49	0.77	1.31
10.Perc.	15	0.00	0.00	0.00	0.00
11.pmX	15	0.55	0.46	0.30	0.80
12.oX	15	0.18	0.34	-0.01	0.37

Table 1-6

Organization: NJIT Sorbent: Tenax

QUARTER OF Oct. TO Dec. , 1988

BLANKS

	# Blanks Run	Avg. (ng)	Std.Dev (ng)	95% CL Interval (ng)	
1.MeCL	15	2.50	1.01	1.95	3.05
2.DCM	15	0.00	0.00	0.00	0.00
3.C6	15	0.00	0.00	0.00	0.00
4.CFor	15	0.00	0.00	0.00	0.00
5.111	15	0.00	0.00	0.00	0.00
6.Bz	15	4.18	1.05	3.61	4.75
7.CCl4	15	0.00	0.00	0.00	0.00
8.Tric	15	0.00	0.00	0.00	0.00
9.Tol	15	1.32	0.78	0.89	1.75
10.Perc.	15	0.00	0.00	0.00	0.00
11.pmX	15	0.66	0.56	0.35	0.97
12.oX	15	0.24	0.59	-0.08	0.56

Table 1-7

Organization: NJIT Sorbent: Tenax

QUARTER OF Jan. TO Mar. , 1989

BLANKS

	# Blanks Run	Avg. (ng)	Std.Dev (ng)	95% CL Interval (ng)	
1.MeCL	14	1.42	0.95	0.88	1.96
2.DCM	14	0.00	0.00	0.00	0.00
3.C6	14	0.45	0.83	-0.02	0.93
4.CFor	14	0.00	0.00	0.00	0.00
5.111	14	0.00	0.00	0.00	0.00
6.Bz	14	3.69	2.49	2.26	5.11
7.CCl4	14	0.00	0.00	0.00	0.00
8.Tric	14	0.00	0.00	0.00	0.00
9.Tol	14	0.98	0.78	0.53	1.42
10.Perc.	14	0.00	0.00	0.00	0.00
11.pmX	14	1.13	1.00	0.56	1.70
12.oX	14	0.00	0.00	0.00	0.00

Table 1-8

Organization: NJIT Sorbent: Tenax

QUARTER OF Apr. TO Jun. , 1989

BLANKS

	# Blanks Run	Avg. (ng)	Std.Dev (ng)	95% CL Interval (ng)	
1.MèCL	15	0.92	1.29	0.20	1.63
2.DCM	15	0.00	0.00	0.00	0.00
3.C6	15	1.80	2.15	0.62	2.98
4.CFor	15	0.00	0.00	0.00	0.00
5.111	15	0.42	1.46	-0.39	1.22
6.Bz	15	3.08	1.71	2.14	4.01
7.CCl4	15	0.00	0.00	0.00	0.00
8.Tric	15	0.00	0.00	0.00	0.00
9.Tol	15	1.29	1.81	0.30	2.29
10.Perc.	15	0.00	0.00	0.00	0.00
11.pmX	15	1.00	1.13	0.37	1.62
12.oX	15	0.70	0.76	0.28	1.12

Table 1-9

Organization: NJIT Sorbent: Tenax

QUARTER OF Jul. TO Sep. , 1989

BLANKS

	# Blanks Run	Avg. (ng)	Std.Dev (ng)	95% CL Interval (ng)	
1.MeCL	13	4.43	5.87	0.92	7.95
2.DCM	13	0.00	0.00	0.00	0.00
3.C6	13	2.61	3.05	0.78	4.44
4.CFor	13	0.00	0.00	0.00	0.00
5.111	13	1.73	2.39	0.30	3.16
6.Bz	13	4.82	4.46	2.15	7.49
7.CCl4	13	0.67	0.81	0.18	1.15
8.Tric	13	0.09	0.33	-0.10	0.29
9.Tol	13	4.72	5.30	1.55	7.90
10.Perc.	13	0.24	0.60	-0.12	0.60
11.pmX	13	0.89	1.11	0.23	1.56
12.oX	13	0.63	0.63	0.25	1.01

Fig. 1-1

Tenax Blank, 1989

Carbon Tetrachloride

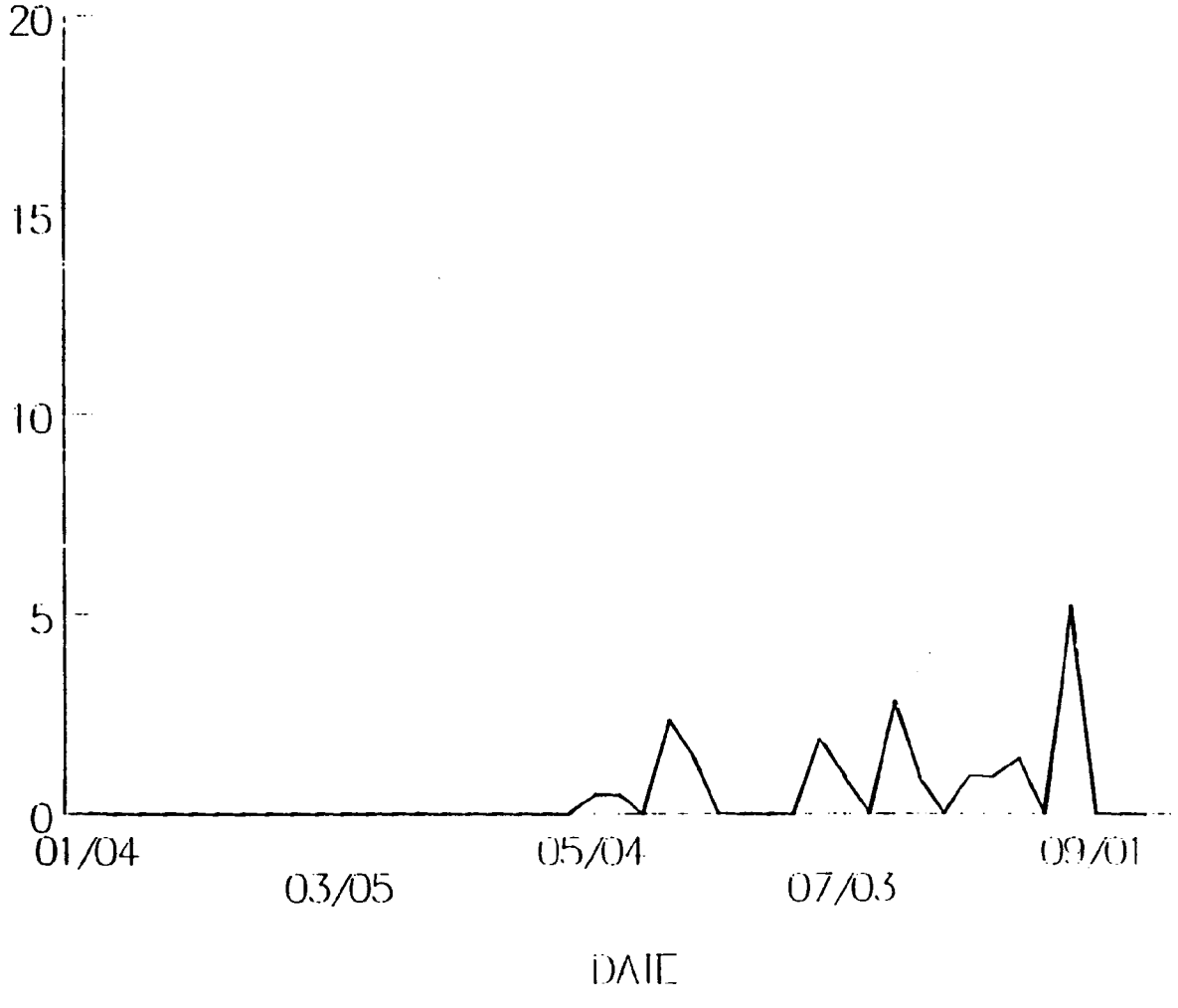
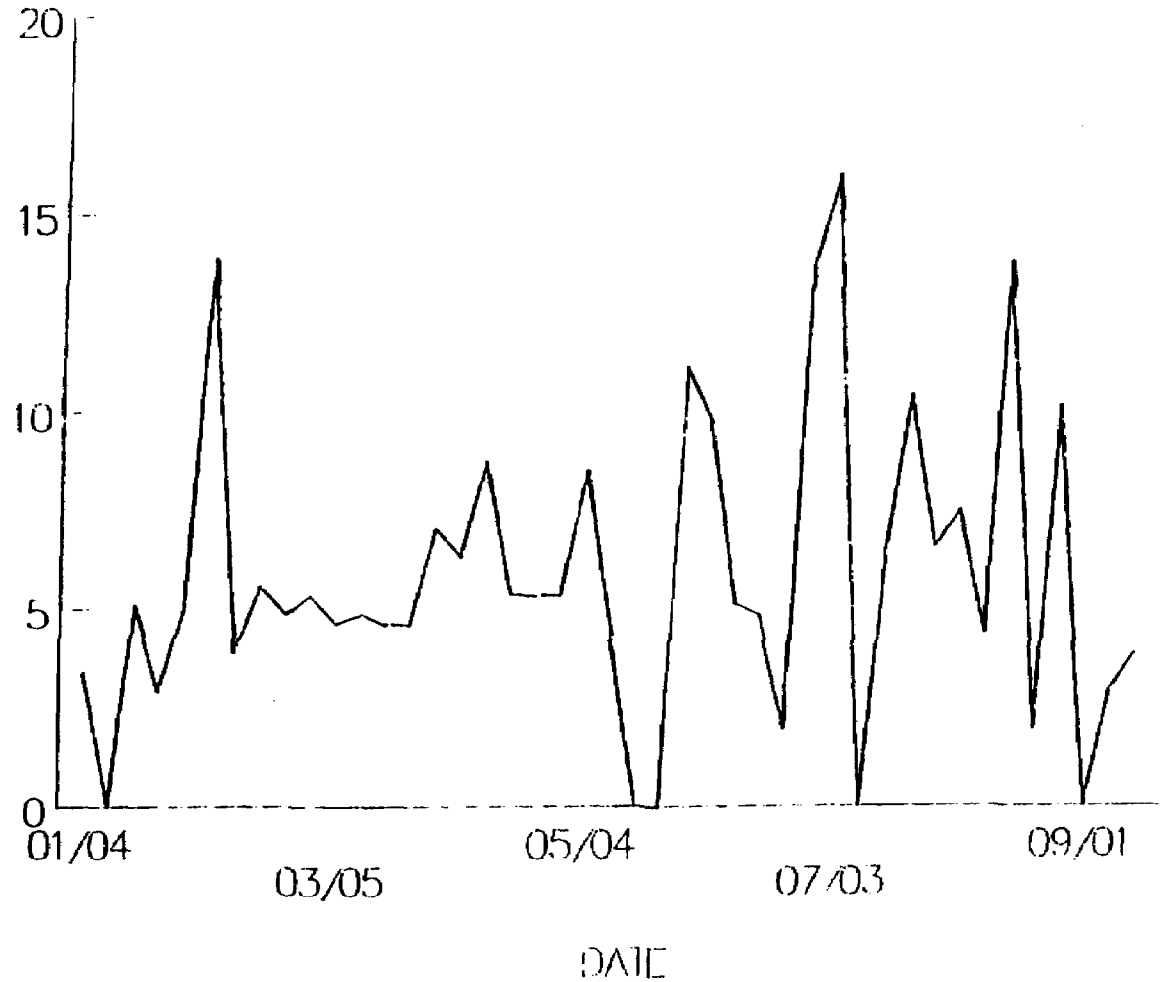


Fig. 1-2

Tenax Blank, 1989

Benzene

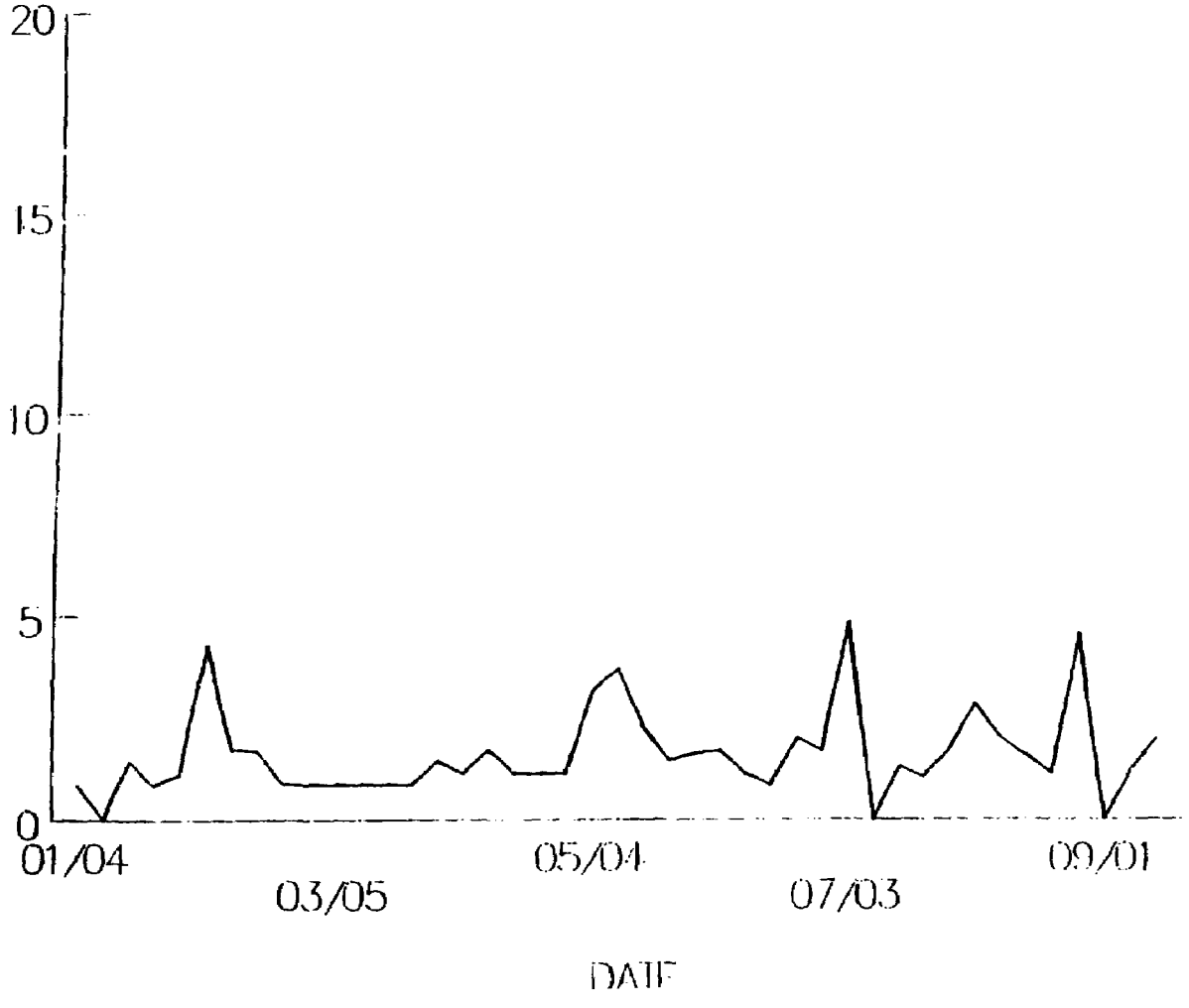


ng

Fig. 1-3

Tenax Blank, 1989

Toluene

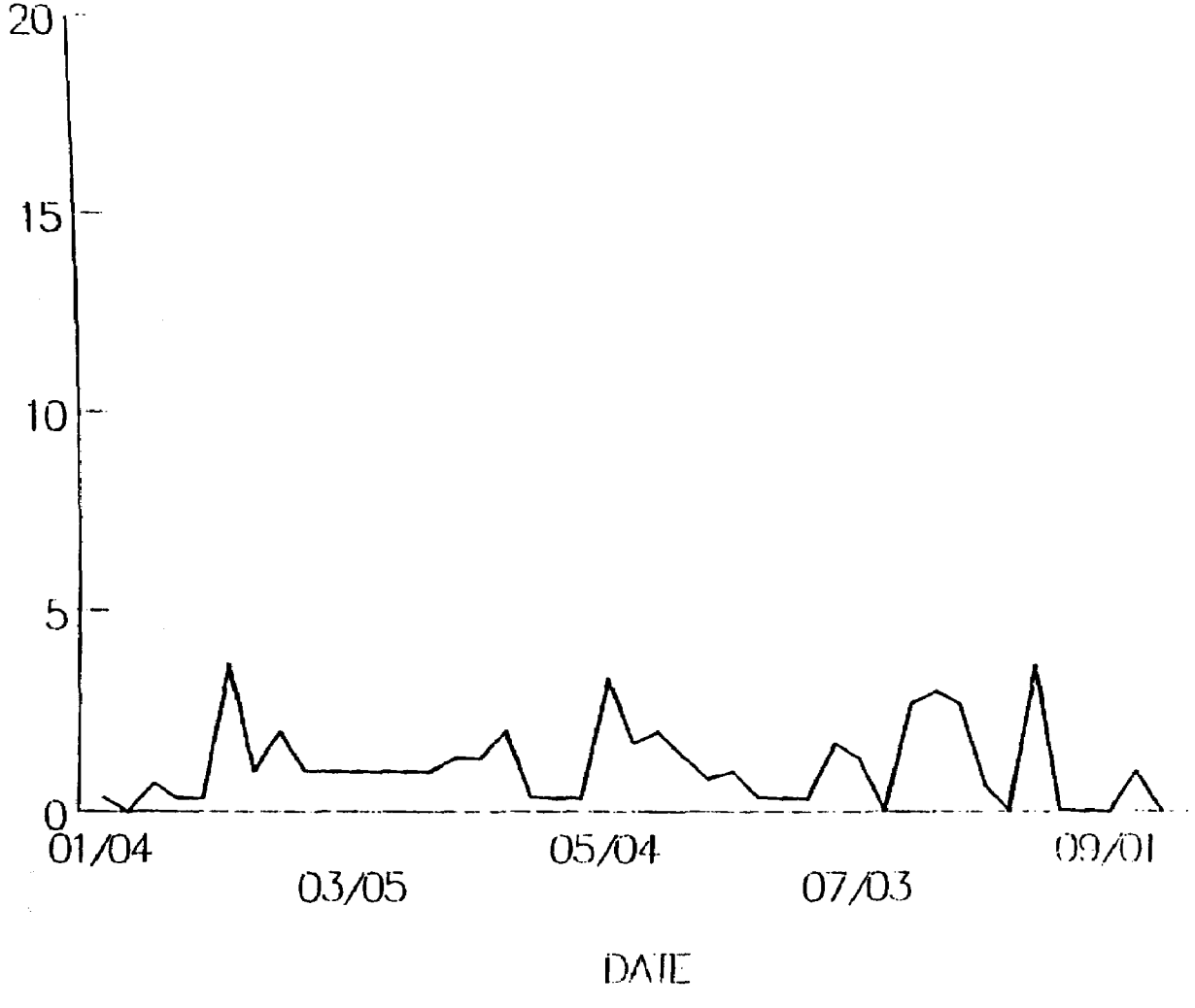


ng

Fig. 1-4

Tenax Blank, 1989

p & m-Xylene



ng

Chapter 2

Replicate Samples

Tenax: Because samples were being taken at two flows, and canister samples were being taken at the same sites every sampling day, duplicate Tenax samples were not done.

Canister Samples: As each canister contained sufficient sample for several injections into the gas chromatograph, routine analytical procedure was developed in which each canister was analyzed three times and the results averaged. These averages were reported in the data tables. The three replicates were not entered into the computer spreadsheets of record, and so are not readily available for study. However, two periods were studied to obtain the reproducibility data for the canister samples. These periods from April to December, 1988 and from January to September 1989 cover early and later samples. The data tables (Tables 2-1 and 2-2) show that the avarages of three replicate analyses showed average standard deviations on the order of 10%. It is also obvious there was some improvement in the analytical technique between the early and later samples. The number of compounds for which the relative standard deviation of the mean exceeded 10% dropped from 7 to only 3, and those were compounds which are detected at levels very close to the detection limit.

While these replicates are solely replications of the analytical procedure, and not of the sampling system, the possible error sources in the sampling system are many fewer than those found when sample flows and the constancy of these flows are critical, as in absorbent sampling. Flow rate into the canister has no bearing on the concentration found, as long as the total amount of sample is kept within the limits imposed by the pressure rating of the canister and the minimum volume of sample required for analysis. Therefore it appears that the analytical system contains the major sources of variability in this system, and the

replicates of the analysis are a good estimate of the overall error level of this analysis.

Table 2-1

Organization: NJIT Sampling: Canister

COVERING FROM Apr. TO Dec. , 1988

DUPLICATE SAMPLES

	# Run (each 3 dup.)	SD of MEAN (ppb)	SD of MEAN (%)
1.MeCL	19	0.00	-
2.DCM	19	0.00	-
3.C6	19	0.07	7.71
4.CFor	19	0.01	18.48
5.111	19	0.08	10.07
6.Bz	19	0.06	4.63
7.CCl4	19	0.03	13.42
8.Tric	19	0.02	15.89
9.Tol	19	0.14	5.01
10.Perc.	19	0.04	15.28
11.pmX	19	0.07	10.71
12.oX	19	0.03	12.78

Table 2-2

Organization: NJIT Sampling: Canister

COVERING FROM Jan. TO Sep. , 1989

DUPLICATE SAMPLES

	# Run (each 3 dup.)	SD of MEAN (ppb)	SD of MEAN (%)
1.MeCL	20	0.13	9.54
2.DCM	20	0.00	-
3.C6	20	0.05	5.67
4.CFor	20	0.01	15.30
5.111	20	0.08	6.75
6.Bz	20	0.08	5.36
7.CCl4	20	0.02	12.50
8.Tric	20	0.01	10.63
9.Tol	20	0.14	5.96
10.Perc.	20	0.01	6.39
11.pmX	20	0.10	8.81
12.oX	20	0.05	9.17

Chapter 3

Distributed Volumes

Tenax: To assure samples which were free of the influence of sample breakthrough, samples were taken at two flows, 5 and 10 ml/min. This resulted in total sample volumes over 24 hours of approximately 7 and 14 liters. If substantial and regular breakthrough is occurring for any particular compound, the concentration determined for the lower flow trap will exceed that determined for the higher flow sample. The statistics for the high and low flow traps are shown for each quarter in Tables 3-1 to 3-9. Evidence of breakthrough is most readily seen graphically. Figures 3-1 to 3-3 show the levels found for typical compounds, benzene, hexane, and tetrachloroethylene, with the concentrations determined on the two traps plotted against each other. The amount of difference between traps is indicated by the distance of each point from the straight line with a slope of 1. Points below the line are those where the high flow traps showed evidence of breakthrough.

It can be seen that for many of the compounds, the majority of the samples fall below the 1:1 line, but in most cases the deviation is within an acceptable limit, and in some cases does not appear to be concentration dependant. For example, in the case of benzene, the points lie mostly along a line below, but parallel to the 1:1 line, while in the case of tetrachloroethylene, the least squares line and the 1:1 line diverge as concentrations increase.

This would argue for separate causes of the differences. If sample is breaking through due to migration of the sample front through the tube, the amount lost should be proportional to the the concentration. The case of deviations which are constant, rather than proportional to the concentration may be related to problems with the blank.

The t-test is used to determine if there is a significant difference between the results for samples done at the different flows. It appears that breakthrough is more of a problem

later in the project than it was in the first few quarters. Actually, the small difference due to breakthrough is more apparent in the later samples when the precision of the measurement has improved. The larger amount of scatter in the earlier measurements masked the effect of breakthrough. The apparantly significant differences in some quarters for the higher boiling compounds are not due to breakthrough, but rather due to occasional cold spot problems in the analytical system. A constant loss from two samples which contain masses of material, differing by a factor of two will lead to apparent concentration differences.

Table 3-1

Organization: NJIT Sorbent: Tenax

QUARTER OF Jul. TO Sep. , 1987

DISTRIBUTED VOLUMES

	# Pairs Run	Avg. Dif. (ppb)	Std.Dev Avg.Dif (ppb)	95% CL Interval (ppb)		T Test	T> 0.05 (Y/N)
1.MeCL	9	0.14	0.11	-0.11	0.38	1.30	N
2.DCM	4	0.00	0.00	0.00	0.00	-	-
3.C6	9	0.11	0.22	-0.40	0.61	0.48	N
4.CFor	8	-0.01	0.01	-0.02	0.01	-0.83	N
5.111	9	-0.03	0.16	-0.39	0.34	-0.17	N
6.Bz	9	0.18	0.21	-0.30	0.66	0.83	N
7.CCl4	9	0.01	0.03	-0.06	0.07	0.19	N
8.Tric	9	0.01	0.03	-0.06	0.08	0.26	N
9.Tol	9	0.21	0.89	-1.80	2.21	0.23	N
10.Perc.	9	-0.03	0.03	-0.10	0.04	-0.90	N
11.pmX	9	0.17	0.43	-0.81	1.14	0.39	N
12.oX	9	0.04	0.16	-0.32	0.40	0.25	N

Table 3-2

QUARTER OF Oct. TO Dec. , 1987

DISTRIBUTED VOLUMES

	# Pairs Run	Avg. Dif. (ppb)	Std.Dev Avg.Dif (ppb)	95% CL Interval (ppb)		T Test	T> 0.05 (Y/N)
1.MeCL	9	0.00	0.06	-0.14	0.13	-0.03	N
2.DCM	4	0.00	0.00	0.00	0.00	-	-
3.C6	10	-0.42	0.42	-1.35	0.51	-1.01	N
4.CFor	10	0.00	0.00	0.00	0.00	-	N
5.111	11	-0.14	0.12	-0.40	0.12	-1.20	N
6.Bz	11	-0.24	0.48	-1.30	0.82	-0.50	N
7.CCl4	11	0.01	0.02	-0.03	0.04	0.31	N
8.Tric	11	0.01	0.03	-0.06	0.07	0.27	N
9.Tol	11	-0.50	1.42	-3.62	2.61	-0.36	N
10.Perc.	11	0.00	0.03	-0.06	0.07	0.14	N
11.pmX	11	-0.30	0.41	-1.20	0.60	-0.73	N
12.oX	11	-0.10	0.14	-0.41	0.21	-0.73	N

Table 3-3

Organization: NJIT Sorbent: Tenax

QUARTER OF Jan. TO Mar. , 1988

DISTRIBUTED VOLUMES

	# Pairs Run	Avg. Dif. (ppb)	Std.Dev Avg.Dif (ppb)	95% CL Interval (ppb)		T Test	T> 0.05 (Y/N)
1.MeCL	12	0.06	0.02	0.02	0.11	2.95	Y
2.DCM	12	0.00	0.00	0.00	0.00	-	-
3.C6	12	0.17	0.11	-0.08	0.41	1.50	N
4.CFor	12	-0.01	0.01	-0.02	0.01	-1.07	N
5.111	12	0.17	0.05	0.05	0.28	3.11	Y
6.Bz	11	0.36	0.22	-0.13	0.85	1.63	N
7.CCl4	12	0.05	0.02	0.00	0.09	2.40	Y
8.Tric	12	0.02	0.01	0.00	0.04	1.95	N
9.Tol	10	-0.04	0.27	-0.65	0.57	-0.14	N
10.Perc.	12	0.06	0.02	0.01	0.11	2.47	Y
11.pmX	12	0.13	0.09	-0.08	0.33	1.34	N
12.oX	12	0.14	0.07	-0.02	0.30	1.91	N

Table 3-4

QUARTER OF Apr. TO Jun. , 1988

DISTRIBUTED VOLUMES

	# Pairs Run	Avg. Dif. (ppb)	Std.Dev Avg.Dif (ppb)	95% CL Interval (ppb)		T Test	T> 0.05 (Y/N)
1.MeCL	13	0.05	0.05	-0.06	0.15	0.97	N
2.DCM	13	0.00	0.00	0.00	0.00	-	-
3.C6	13	0.10	0.27	-0.49	0.69	0.37	N
4.CFor	11	0.00	0.03	-0.06	0.05	-0.20	N
5.111	13	0.21	0.12	-0.05	0.48	1.72	N
6.Bz	13	0.20	0.25	-0.33	0.73	0.83	N
7.CCl4	13	0.05	0.04	-0.04	0.14	1.29	N
8.Tric	13	0.01	0.02	-0.04	0.05	0.40	N
9.Tol	13	0.41	1.00	-1.76	2.57	0.41	N
10.Perc.	13	0.07	0.05	-0.03	0.17	1.53	N
11.pmX	13	0.12	0.27	-0.47	0.71	0.44	N
12.oX	13	0.04	0.09	-0.15	0.23	0.46	N

Table 3-5

Organization: NJIT Sorbent: Tenax

QUARTER OF Jul. TO Sep. , 1988

DISTRIBUTED VOLUMES

	# Pairs Run	Avg. Dif. (ppb)	Std.Dev Avg.Dif (ppb)	95% CL Interval (ppb)		T Test	T> 0.05 (Y/N)
1.MeCL	27	0.14	0.03	0.09	0.19	5.43	Y
2.DCM	27	0.00	0.00	0.00	0.00	-	N
3.C6	27	0.34	0.28	-0.24	0.91	1.20	N
4.CFor	27	0.00	0.01	-0.02	0.02	0.35	N
5.111	28	0.19	0.14	-0.10	0.48	1.36	N
6.Bz	27	0.30	0.19	-0.10	0.70	1.54	N
7.CCl4	27	0.03	0.03	-0.03	0.09	0.93	N
8.Tric	24	0.01	0.02	-0.03	0.04	0.33	N
9.Tol	27	0.44	0.72	-1.04	1.92	0.61	N
10.Perc.	27	0.02	0.05	-0.08	0.12	0.36	N
11.pmX	27	0.09	0.26	-0.44	0.61	0.34	N
12.oX	26	0.03	0.08	-0.14	0.20	0.38	N

Table 3-6

QUARTER OF Oct. TO Dec. , 1988

DISTRIBUTED VOLUMES

	# Pairs Run	Avg. Dif. (ppb)	Std.Dev Avg.Dif (ppb)	95% CL Interval (ppb)		T Test	T> 0.05 (Y/N)
1.MeCL	29	0.20	0.03	0.14	0.25	7.78	Y
2.DCM	29	0.00	0.00	0.00	0.00	-	-
3.C6	29	0.08	0.03	0.03	0.14	3.23	Y
4.CFor	29	0.00	0.00	0.00	0.00	-	-
5.111	29	0.08	0.02	0.05	0.12	4.66	Y
6.Bz	29	0.17	0.06	0.05	0.29	2.84	Y
7.CCl4	29	0.01	0.00	0.00	0.02	3.48	Y
8.Tric	29	0.01	0.00	0.00	0.02	1.57	N
9.Tol	29	0.42	0.15	0.12	0.73	2.81	Y
10.Perc.	29	0.02	0.00	0.01	0.03	4.31	Y
11.pmX	29	0.16	0.08	-0.01	0.33	1.98	N
12.oX	29	0.05	0.03	0.00	0.10	1.99	N

Table 3-7

Organization: NJIT Sorbent: Tenax

QUARTER OF Jan. TO Mar. , 1989

DISTRIBUTED VOLUMES

	# Pairs Run	Avg. Dif. (ppb)	Std.Dev Avg.Dif (ppb)	95% CL Interval (ppb)		T Test	T> 0.05 (Y/N)
1.MeCL	-	-	-	-	-	-	-
2.DCM	31	0.00	0.00	0.00	0.00	-	-
3.C6	31	0.09	0.06	-0.03	0.21	1.57	N
4.CFor	28	0.00	0.00	0.00	0.00	-	-
5.111	29	0.05	0.03	-0.01	0.11	1.58	N
6.Bz	28	0.20	0.08	0.04	0.36	2.52	Y
7.CCl4	30	0.01	0.01	0.00	0.02	1.37	N
8.Tric	28	0.00	0.00	0.00	0.01	0.79	N
9.Tol	30	0.35	0.16	0.02	0.68	2.18	Y
10.Perc.	29	0.03	0.01	0.01	0.05	4.04	Y
11.pmX	30	0.22	0.10	0.01	0.43	2.11	Y
12.oX	31	0.12	0.05	0.01	0.23	2.30	Y

Table 3-8

QUARTER OF Apr. TO Jun. , 1989

DISTRIBUTED VOLUMES

	# Pairs Run	Avg. Dif. (ppb)	Std.Dev Avg.Dif (ppb)	95% CL Interval (ppb)		T Test	T> 0.05 (Y/N)
1.MeCL	-	-	-	-	-	-	-
2.DCM	50	0.00	0.00	0.00	0.00	-	-
3.C6	48	0.24	0.07	0.09	0.39	3.33	Y
4.CFor	51	0.00	0.00	0.00	0.01	1.07	N
5.111	50	0.08	0.02	0.04	0.12	4.04	Y
6.Bz	49	0.18	0.05	0.08	0.28	3.60	Y
7.CCl4	51	0.02	0.01	0.01	0.03	2.86	Y
8.Tric	51	0.00	0.00	0.00	0.01	0.95	N
9.Tol	48	0.11	0.12	-0.14	0.36	0.90	N
10.Perc.	51	0.01	0.01	-0.01	0.03	0.89	N
11.pmX	48	0.04	0.04	-0.04	0.12	1.07	N
12.oX	48	0.07	0.02	0.02	0.12	3.03	Y

Table 3-9

Organization: NJIT Sorbent: Tenax

QUARTER OF Jul. TO Sep. , 1989

DISTRIBUTED VOLUMES

	# Pairs Run	Avg. Dif. (ppb)	Std.Dev Avg.Dif (ppb)	95% CL Interval (ppb)		T Test	T> 0.05 (Y/N)
1.MeCL	47	0.93	0.42	0.09	1.77	2.21	Y
2.DCM	47	0.00	0.00	0.00	0.00	-	-
3.C6	47	0.17	0.09	-0.01	0.35	1.91	N
4.CFor	47	0.01	0.01	-0.01	0.04	1.19	N
5.111	47	0.22	0.07	0.07	0.36	3.02	Y
6.Bz	47	0.30	0.08	0.14	0.46	3.74	Y
7.CCl4	47	0.07	0.03	0.00	0.13	1.89	N
8.Tric	47	0.02	0.01	-0.01	0.04	1.48	N
9.Tol	47	0.51	0.31	-0.12	1.13	1.63	N
10.Perc.	47	0.07	0.05	-0.03	0.17	1.46	N
11.pmX	47	0.21	0.12	-0.04	0.45	1.70	N
12.oX	47	0.12	0.06	-0.01	0.25	1.81	N

Fig. 3-1

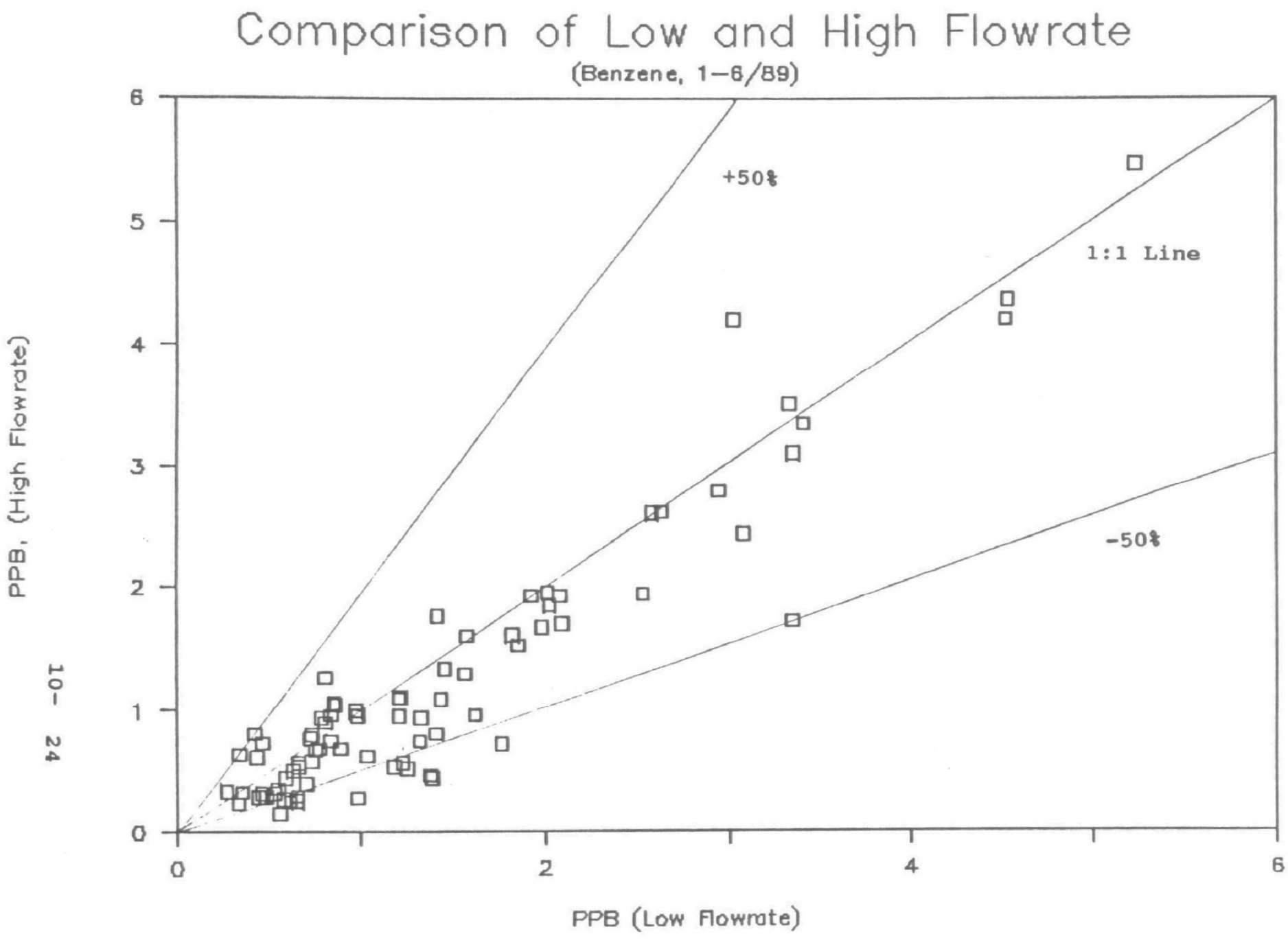

Comparison of Low and High Flowrate
(Benzene, 1-6/89)
+50%
1:1 Line
-50%
PPB, (High Flowrate)
PPB (Low Flowrate)
0
1
2
3
4
5
6
0
2
4
6

Fig. 3-2

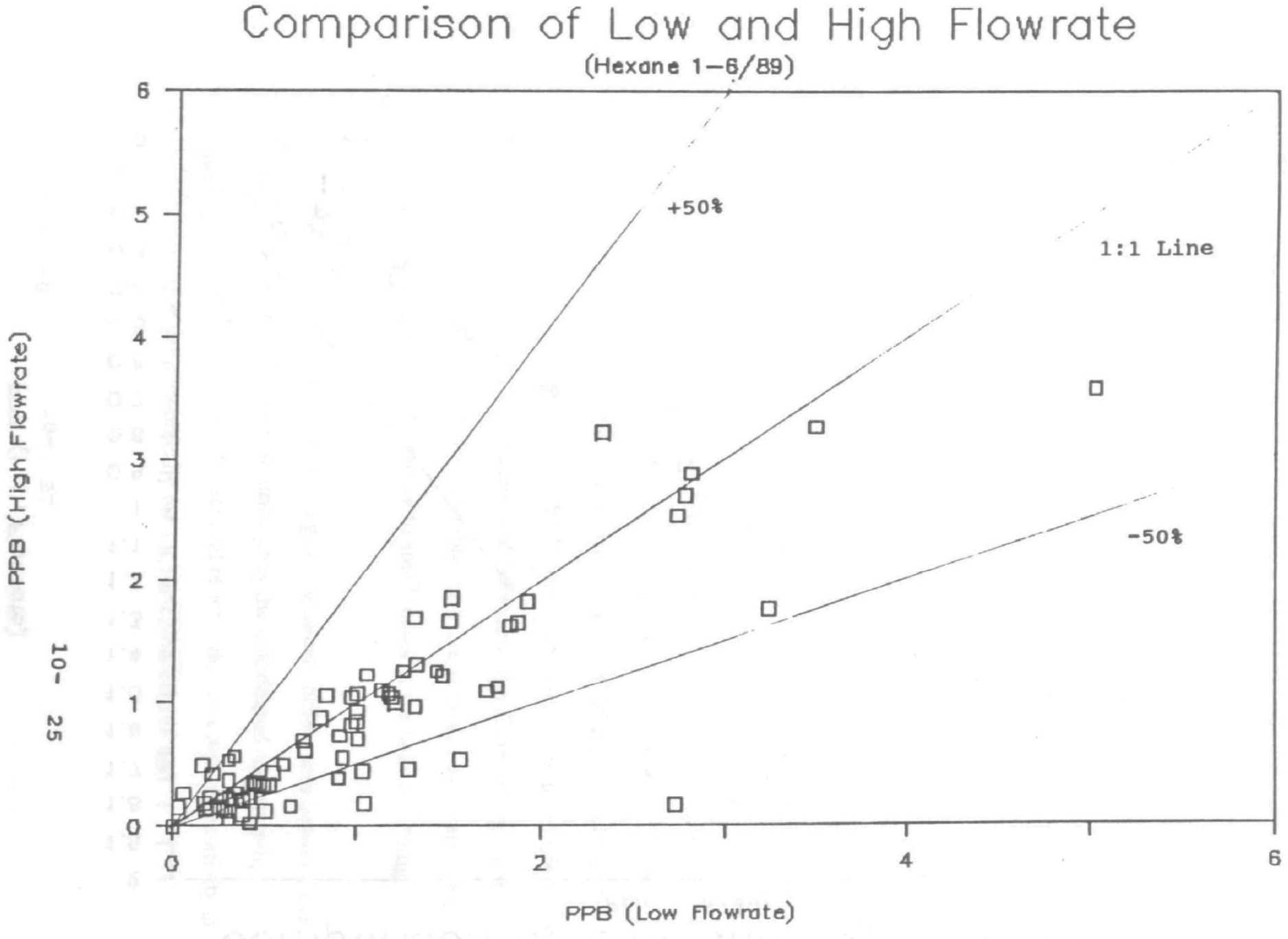
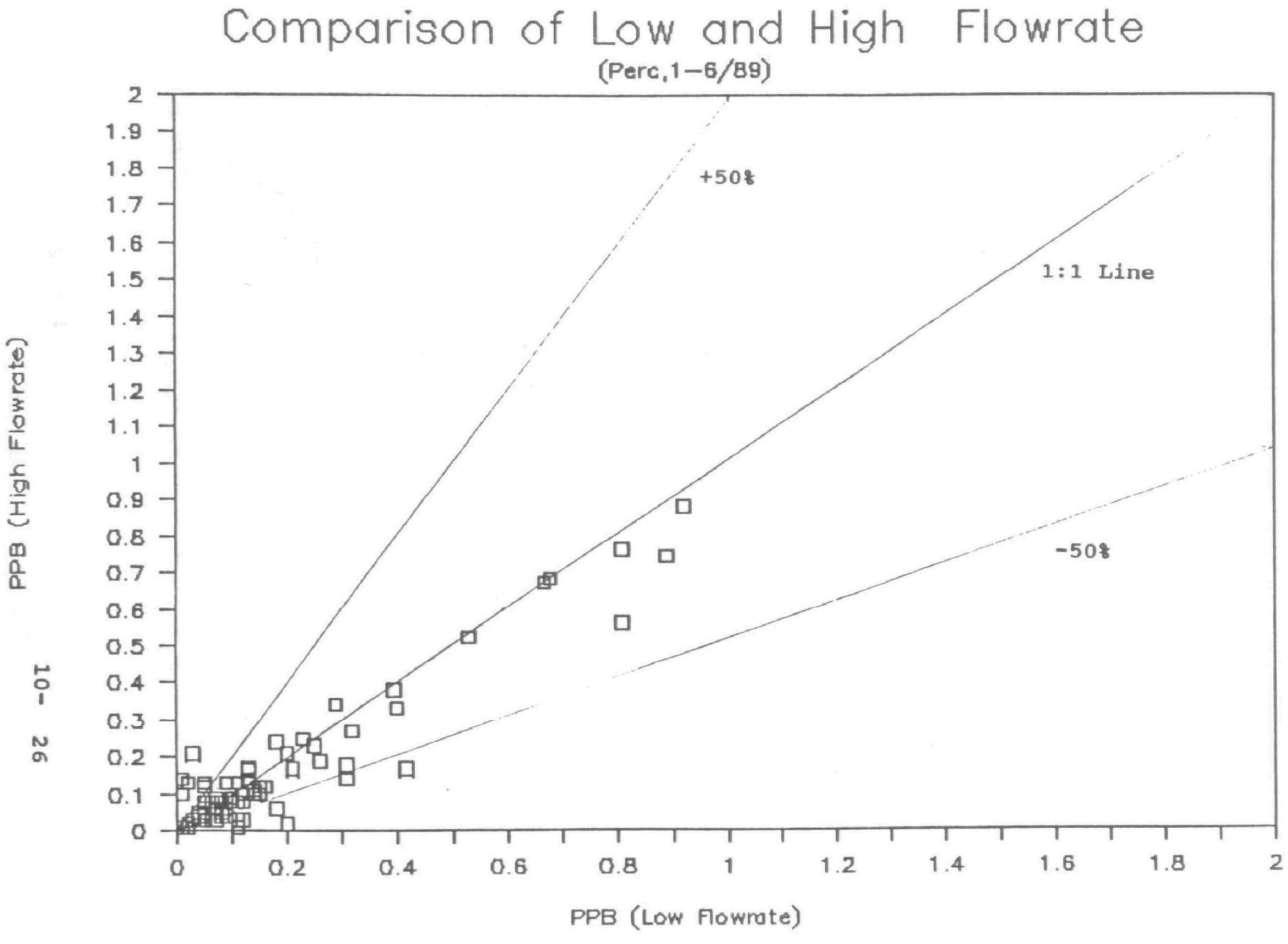

Comparison of Low and High Flowrate
(Hexane 1-6/89)
+50%
1:1 Line
-50%
PPB (High Flowrate)
PPB (Low Flowrate)
0
1
2
3
4
5
6
0
2
4
6

Fig. 3-3


Comparison of Low and High Flowrate
(Perc,1-6/89)
+50%
1:1 Line
-50%
PPB (High Flowrate)
PPB (Low Flowrate)
0
0.2
0.4
0.6
0.8
1
1.2
1.4
1.6
1.8
2

Chapter 4

Tenax vs. Canister Analyses

At the two sites, Elizabeth and Carteret, canister samples were taken each sampling day, along with the Tenax samples. Tables 4.1 to 4.9 show the differences in the data obtained by the two methods over the course of the project. While the differences between the two methods were greater than the differences between the two Tenax tubes, and also greater than the differences between the replicate canister analyses, the differences averaged below 1 ppb for most compounds and for most quarters. There are no easily discernible patterns of higher concentrations of certain compounds by one or the other method.

In the first quarter of the project only three compounds were reported by the canister method. These appear to show significantly higher levels on the canister analyses, but the numbers of analyses are small, and the canister method was not optimized, and was showing poor separation. In the second quarter only one compound, carbon tetrachloride showed a significant difference between the methods, which can be attributed to the fact that the canister method was being done with quantitation on the FID detector, while Tenax samples were being quantitated using the ECD, which was less prone to interference by the nearby benzene peak. The first two quarters of 1988 show significantly larger concentrations in the canister samples, which are probably the more accurate, as several cold spots had developed in the Tenax system, leading to small losses of the heavier boiling compounds.

Early in 1989, a group of compounds which gave significant differences between the two methods began to be evident. These were principally the chlorinated compounds, which gave low results on the Tenax system for several months due to a loss of sensitivity in the ECD detector. This can be seen graphically for 1,1,1-trichloroethane and

tetrachloroethylene, in Figures 4-1 and 4-2. Other statistically significant differences scattered over the length of the project occur mostly in the compounds which are present in the air at very low levels, just above the detection limits. The low levels of these samples makes the occurrence of subtle biases, of unknown origin, in the two systems more likely. This probably accounts for the fact that there appear to be significant differences in these compounds from time to time. The average differences, even those which are statistically significant, infrequently exceed 1 ppb, except during the period before April 1988, when the canister analysis system was being refined.

The variations between the two systems can be seen graphically for three typical compounds, benzene, toluene and tetrachloroethylene in Figures 4-3 to 4-5. Toluene was subject to some losses due to the recurring cold spot in the Tenax system in the early part of 1988.

The two analytical systems were calibrated with the same standard gas mixture, to prevent any bias due to calibration from occurring. Since all analytical biases which have been discovered have been negative, usually due to losses of analyte at some point in the system (cold spots, breakthrough, low detector sensitivity), when the two methods disagree, and there is sufficient difference between the results to require a choice to be made, the higher value is more likely to be closer to the actual level.

Table 4-1

Organization: NJIT Sorbent: Tenax

QUARTER OF Jul. TO Sep. , 1987

TENAX vs. CANISTER

	# Pairs Run	Avg. Dif. (ppb)	Std.Dev Avg.Dif (ppb)	95% CL Interval (ppb)		T Test	T> 0.05 (Y/N)
1.MeCL	-	-	-	-	-	-	-
2.DCM	-	-	-	-	-	-	-
3.C6	10	-0.51	0.22	-1.00	-0.02	-2.32	Y
4.CFor	-	-	-	-	-	-	-
5.111	-	-	-	-	-	-	-
6.Bz	-	-	-	-	-	-	-
7.CCl4	-	-	-	-	-	-	-
8.Tric	-	-	-	-	-	-	-
9.Tol	-	-	-	-	-	-	-
10.Perc.	-	-	-	-	-	-	-
11.pmX	7	-1.46	0.40	-2.41	-0.52	-3.67	Y
12.oX	8	-1.10	0.11	-1.35	-0.85	-10.35	Y

Table 4-2

QUARTER OF Oct. TO Dec. , 1987

TENAX vs. CANISTER

	# Pairs Run	Avg. Dif. (ppb)	Std.Dev Avg.Dif (ppb)	95% CL Interval (ppb)		T Test	T> 0.05 (Y/N)
1.MeCL	-	-	-	-	-	-	-
2.DCM	-	-	-	-	-	-	-
3.C6	13	-0.11	0.26	-0.68	0.46	-0.43	N
4.CFor	-	-	-	-	-	-	-
5.111	-	-	-	-	-	-	-
6.Bz	13	0.19	0.35	-0.57	0.95	0.53	N
7.CCl4	13	-0.07	0.02	-0.12	-0.02	-3.06	Y
8.Tric	-	-	-	-	-	-	-
9.Tol	13	-1.35	1.01	-3.53	0.83	-1.33	N
10.Perc.	13	-0.02	0.03	-0.09	0.05	-0.63	N
11.pmX	13	-0.12	0.31	-0.79	0.54	-0.40	N
12.oX	13	-0.03	0.12	-0.29	0.24	-0.24	N

Table 4-3

Organization: NJIT Sorbent: Tenax

QUARTER OF Jan. TO Mar. , 1988

TENAX vs. CANISTER

	# Pairs Run	Avg. Dif. (ppb)	Std.Dev Avg.Dif (ppb)	95% CL Interval (ppb)		T Test	T> 0.05 (Y/N)
1.MeCL	-	-	-	-	-	-	-
2.DCM	-	-	-	-	-	-	-
3.C6	15	-0.31	0.25	-0.85	0.24	-1.20	N
4.CFor	-	-	-	-	-	-	-
5.111	-	-	-	-	-	-	-
6.Bz	15	-0.05	0.29	-0.68	0.58	-0.16	N
7.CCl4	15	0.07	0.02	0.03	0.11	3.54	Y
8.Tric	-	-	-	-	-	-	-
9.Tol	14	-3.85	0.77	-5.50	-2.19	-4.99	Y
10.Perc.	15	0.09	0.03	0.03	0.14	3.06	Y
11.pmX	14	-2.11	0.46	-3.09	-1.12	-4.58	Y
12.oX	15	-0.50	0.16	-0.83	-0.16	-3.19	Y

Table 4-4

QUARTER OF Apr. TO Jun. , 1988

TENAX vs. CANISTER

	# Pairs Run	Avg. Dif. (ppb)	Std.Dev Avg.Dif (ppb)	95% CL Interval (ppb)		T Test	T> 0.05 (Y/N)
1.MeCL	14	0.02	0.04	-0.07	0.11	0.40	N
2.DCM	14	0.00	0.00	0.00	0.00	-	-
3.C6	14	-0.20	0.30	-0.84	0.45	-0.65	N
4.CFor	12	0.04	0.02	0.00	0.07	1.95	N
5.111	14	-0.24	0.14	-0.54	0.06	-1.73	N
6.Bz	14	-0.24	0.24	-0.75	0.27	-1.02	N
7.CCl4	14	0.00	0.03	-0.08	0.07	-0.06	N
8.Tric	14	0.03	0.02	0.00	0.06	1.98	N
9.Tol	14	-0.69	0.85	-2.52	1.14	-0.81	N
10.Perc.	14	0.10	0.03	0.03	0.17	3.08	Y
11.pmX	14	-1.60	0.32	-2.29	-0.90	-4.91	Y
12.oX	14	-0.55	0.12	-0.81	-0.29	-4.49	Y

Table 4-5

Organization: NJIT Sorbent: Tenax

QUARTER OF Jul. TO Sep. , 1988

TENAX vs. CANISTER

	# Pairs Run	Avg. Dif. (ppb)	Std.Dev Avg.Dif (ppb)	95% CL Interval (ppb)		T Test	T> 0.05 (Y/N)
1.MeCL	27	0.09	0.06	-0.04	0.22	1.42	N
2.DCM	27	0.00	0.00	0.00	0.00	-	N
3.C6	27	-0.73	0.57	-1.89	0.44	-1.28	N
4.CFor	27	0.00	0.01	-0.02	0.02	0.23	N
5.111	27	-0.30	0.15	-0.62	0.01	-2.01	N
6.Bz	27	-0.09	0.20	-0.50	0.31	-0.46	N
7.CCl4	27	0.00	0.03	-0.06	0.06	0.00	N
8.Tric	27	0.00	0.01	-0.03	0.03	-0.20	N
9.Tol	27	-0.15	0.74	-1.67	1.36	-0.21	N
10.Perc.	27	0.15	0.04	0.06	0.23	3.61	Y
11.pmX	27	0.37	0.22	-0.08	0.83	1.67	N
12.oX	27	0.10	0.08	-0.06	0.25	1.25	N

Table 4-6

QUARTER OF Oct. TO Dec. , 1988

TENAX vs. CANISTER

	# Pairs Run	Avg. Dif. (ppb)	Std.Dev Avg.Dif (ppb)	95% CL Interval (ppb)		T Test	T> 0.05 (Y/N)
1.MeCL	29	0.16	0.10	-0.04	0.36	1.61	N
2.DCM	27	0.00	0.00	0.00	0.00	-	N
3.C6	29	0.22	0.16	-0.11	0.55	1.35	N
4.CFor	29	-0.03	0.01	-0.04	-0.02	-4.74	Y
5.111	29	-0.24	0.17	-0.60	0.11	-1.40	N
6.Bz	29	0.50	0.18	0.13	0.88	2.76	Y
7.CCl4	29	-0.06	0.02	-0.10	-0.02	-3.19	Y
8.Tric	29	-0.03	0.02	-0.06	0.00	-1.86	N
9.Tol	29	1.90	0.55	0.78	3.01	3.47	Y
10.Perc.	29	0.01	0.03	-0.05	0.06	0.21	N
11.pmX	29	0.94	0.17	0.58	1.29	5.41	Y
12.oX	29	0.31	0.06	0.19	0.42	5.47	Y

Table 4-7

Organization: NJIT Sorbent: Tenax

QUARTER OF Jan. TO Mar. , 1989

TENAX vs. CANISTER

	# Pairs Run	Avg. Dif. (ppb)	Std.Dev Avg.Dif (ppb)	95% CL Interval (ppb)		T Test	T> 0.05 (Y/N)
1.MeCL	-	-	-	-	-	-	-
2.DCM	22	0.00	0.00	0.00	0.00	-	-
3.C6	22	0.17	0.18	-0.20	0.54	0.91	N
4.CFor	23	-0.02	0.00	-0.03	-0.02	-7.50	Y
5.111	24	-0.36	0.11	-0.58	-0.14	-3.39	Y
6.Bz	24	0.40	0.20	0.00	0.80	2.00	N
7.CCl4	24	-0.12	0.02	-0.16	-0.08	-5.90	Y
8.Tric	24	-0.05	0.01	-0.07	-0.03	-4.87	Y
9.Tol	24	1.28	0.63	0.02	2.55	2.05	Y
10.Perc.	24	-0.11	0.03	-0.17	-0.04	-3.43	Y
11.pmX	23	0.21	0.34	-0.49	0.90	0.62	N
12.oX	23	0.11	0.10	-0.09	0.31	1.13	N

Table 4-8

QUARTER OF Apr. TO Jun. , 1989

TENAX vs. CANISTER

	# Pairs Run	Avg. Dif. (ppb)	Std.Dev Avg.Dif (ppb)	95% CL Interval (ppb)		T Test	T> 0.05 (Y/N)
1.MeCL	-	-	-	-	-	-	-
2.DCM	27	0.00	0.00	0.00	0.00	-	-
3.C6	27	0.21	0.12	-0.02	0.45	1.81	N
4.CFor	27	-0.02	0.01	-0.04	-0.01	-3.88	N
5.111	27	-0.60	0.13	-0.86	-0.34	-4.80	Y
6.Bz	27	-0.02	0.13	-0.28	0.24	-0.13	N
7.CCl4	27	-0.05	0.02	-0.08	-0.02	-3.19	Y
8.Tric	26	-0.03	0.01	-0.04	-0.01	-3.10	Y
9.Tol	27	0.06	0.33	-0.60	0.71	0.18	N
10.Perc.	27	0.07	0.03	0.01	0.12	2.31	Y
11.pmX	28	-0.69	0.18	-1.05	-0.33	-3.92	Y
12.oX	27	-0.26	0.09	-0.43	-0.08	-2.92	Y

Table 4-9

Organization: NJIT Sorbent: Tenax

QUARTER OF Jul. TO Sep. , 1989

TENAX vs. CANISTER

	# Pairs Run	Avg. Dif. (ppb)	Std.Dev Avg.Dif (ppb)	95% CL Interval (ppb)		T Test	T> 0.05 (Y/N)
1.MeCL	28	-0.02	0.44	-0.92	0.89	-0.03	N
2.DCM	28	-0.01	0.00	-0.01	0.00	-5.29	Y
3.C6	28	-0.05	0.17	-0.40	0.30	-0.30	N
4.CFor	28	-0.03	0.01	-0.05	-0.01	-2.85	Y
5.111	28	-0.65	0.25	-1.15	-0.15	-2.65	Y
6.Bz	28	-0.78	0.27	-1.32	-0.23	-2.90	Y
7.CCl4	28	-0.05	0.03	-0.11	0.01	-1.66	N
8.Tric	28	-0.01	0.02	-0.06	0.04	-0.45	N
9.Tol	28	-0.39	0.85	-2.13	1.35	-0.46	N
10.Perc.	28	0.17	0.05	0.06	0.28	3.30	Y
11.pmX	28	-0.19	0.18	-0.55	0.18	-1.05	N
12.oX	28	-0.05	0.06	-0.18	0.08	-0.77	N

Fig. 4-1

1,1,1-Trichloroethane

Carteret, 1989

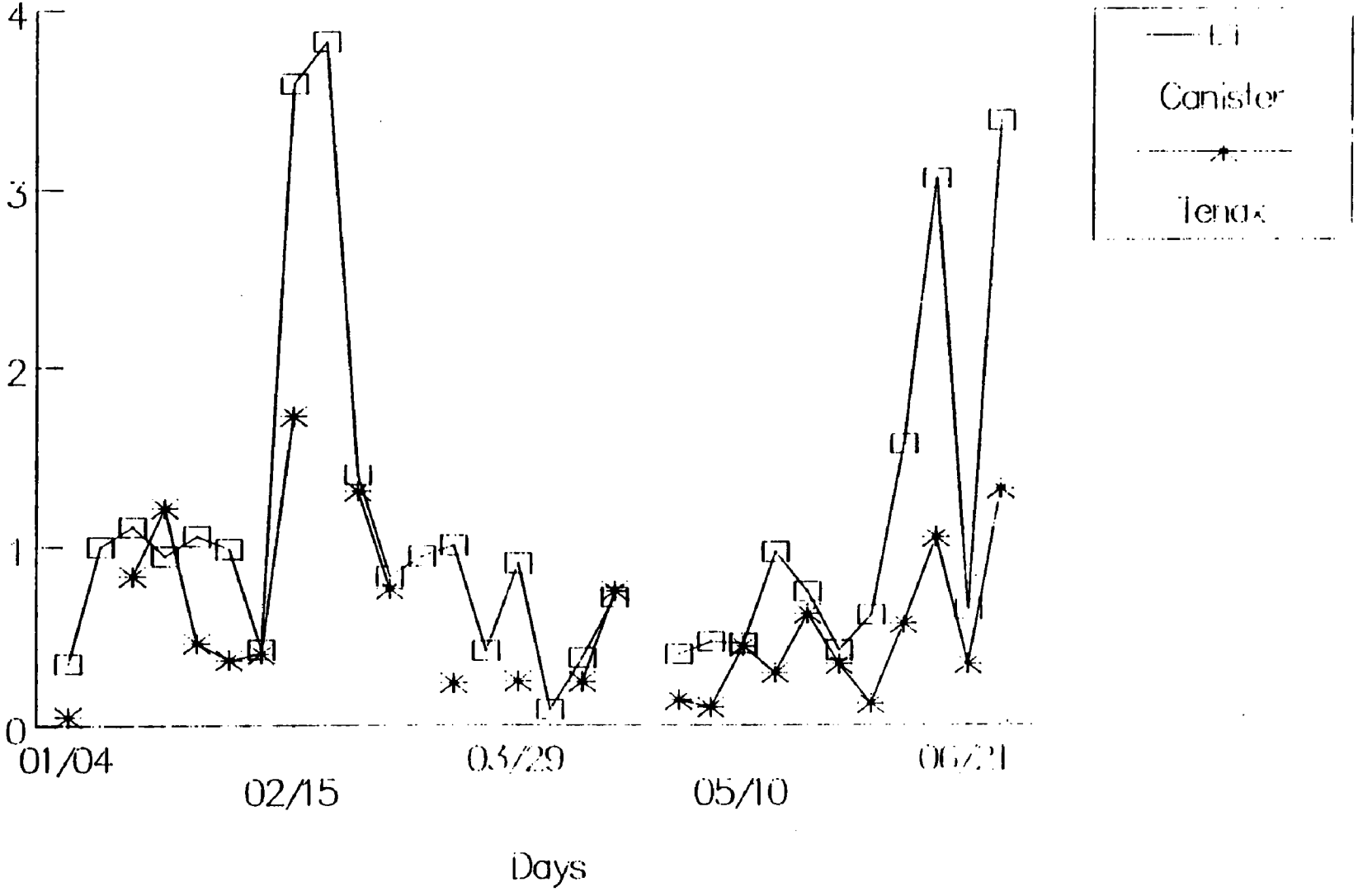
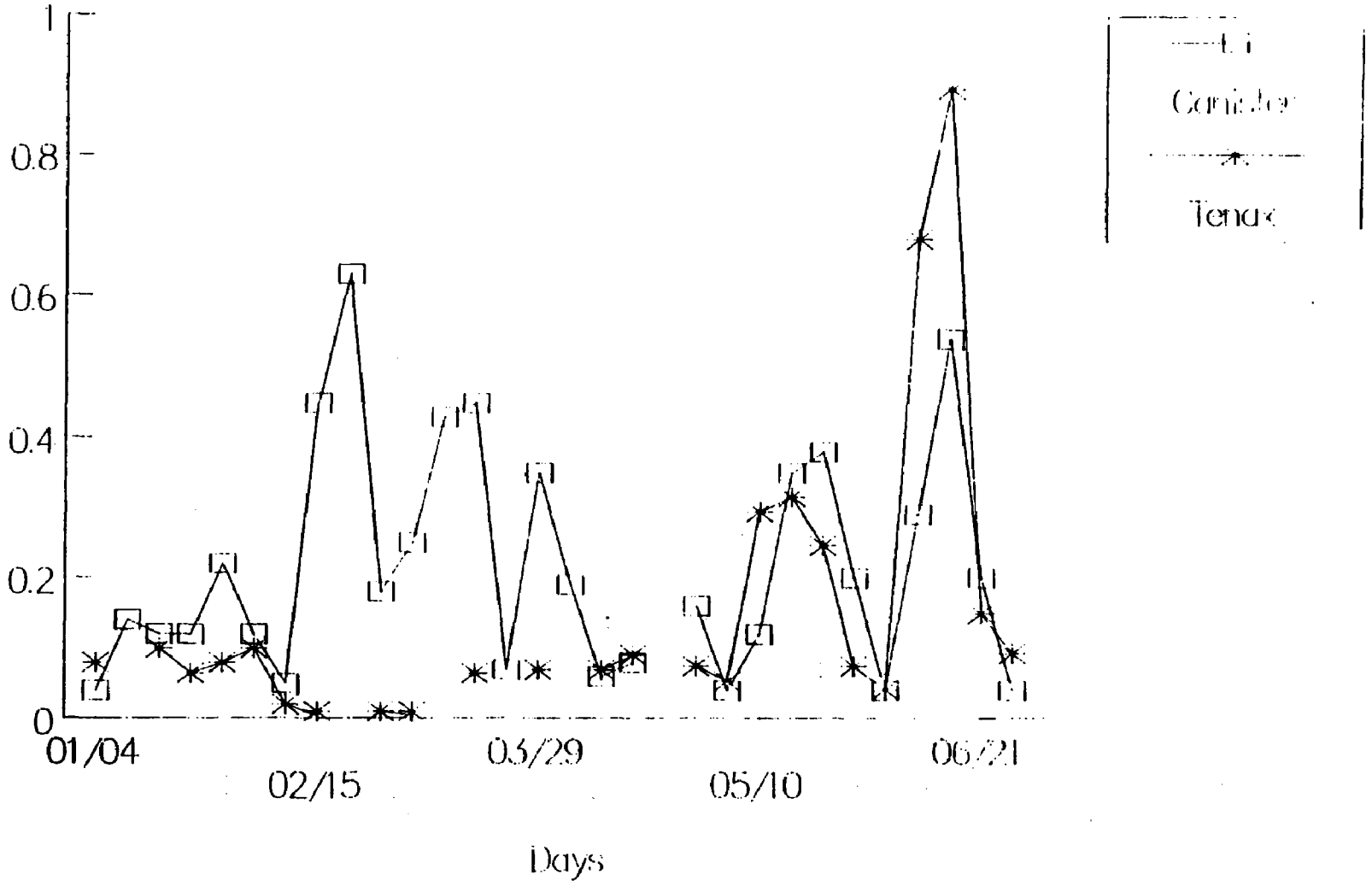


Fig. 4-2

Tetrachloroethylene

Carteret, 1989



ppb

10- 35

Fig. 4-3

Benzene

Elizabeth, 1988

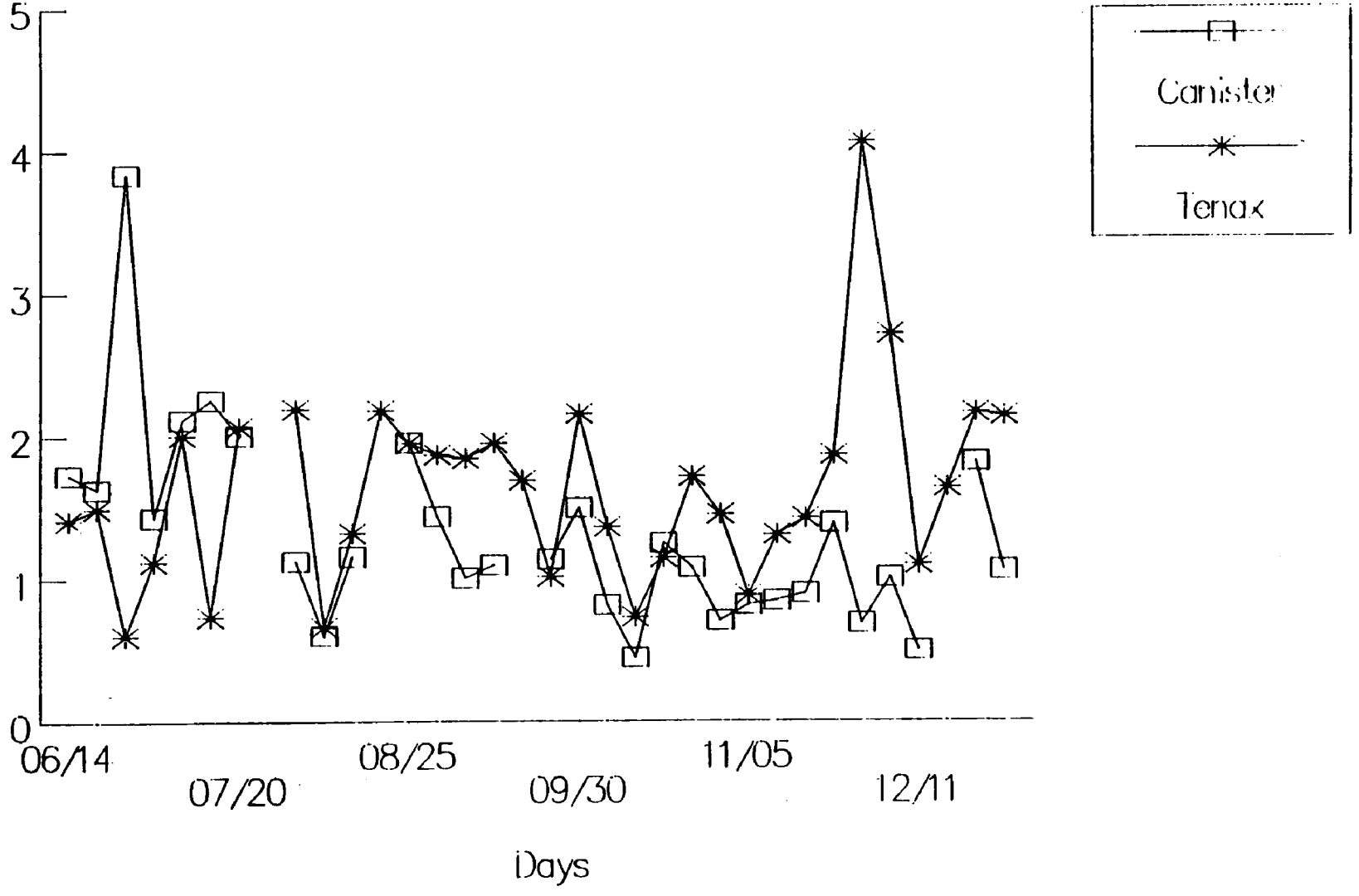
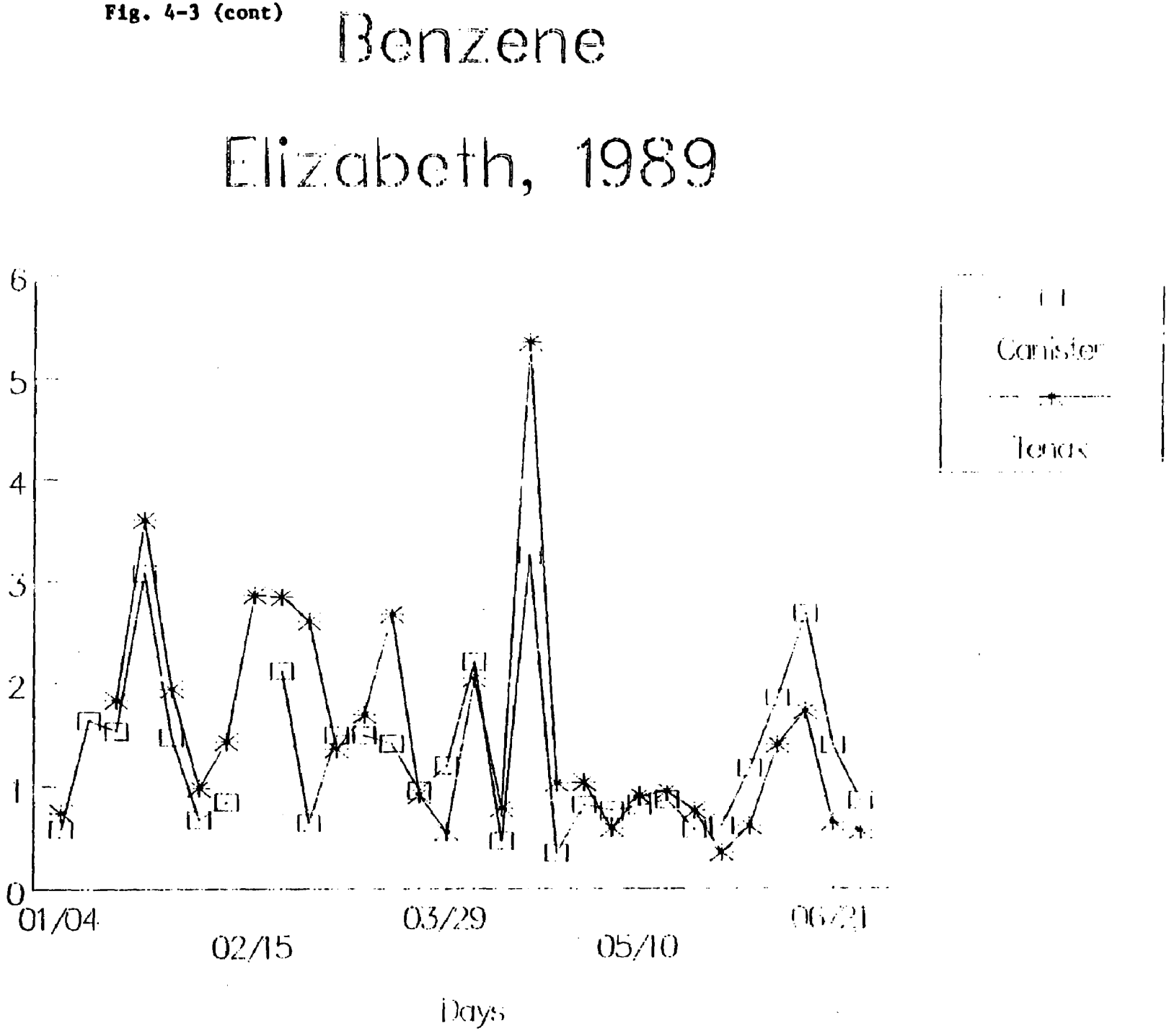


Fig. 4-3 (cont)



gas

Fig. 4-4

Toluene

Carteret, 1988

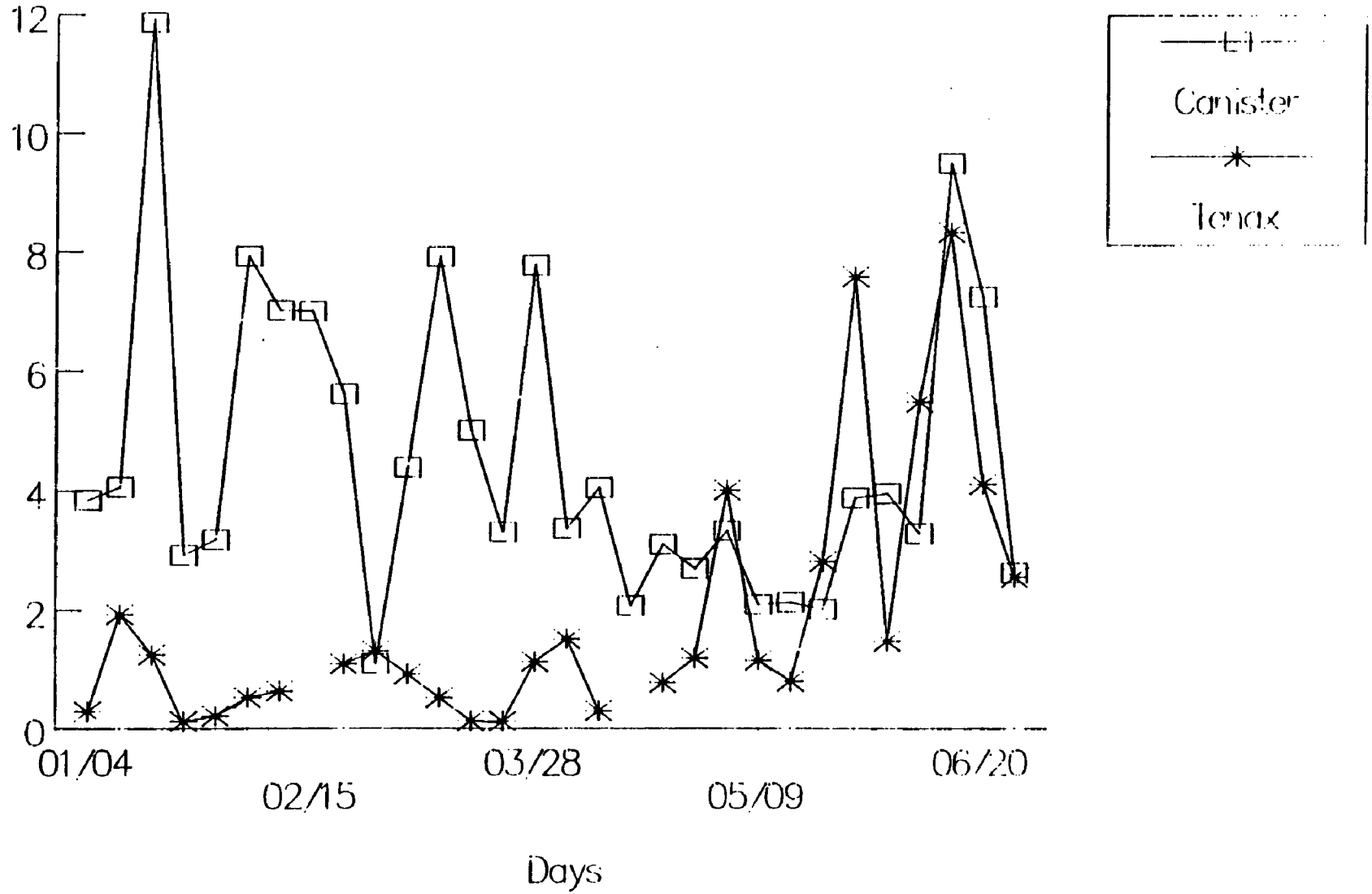


Fig. 4-4 (cont)

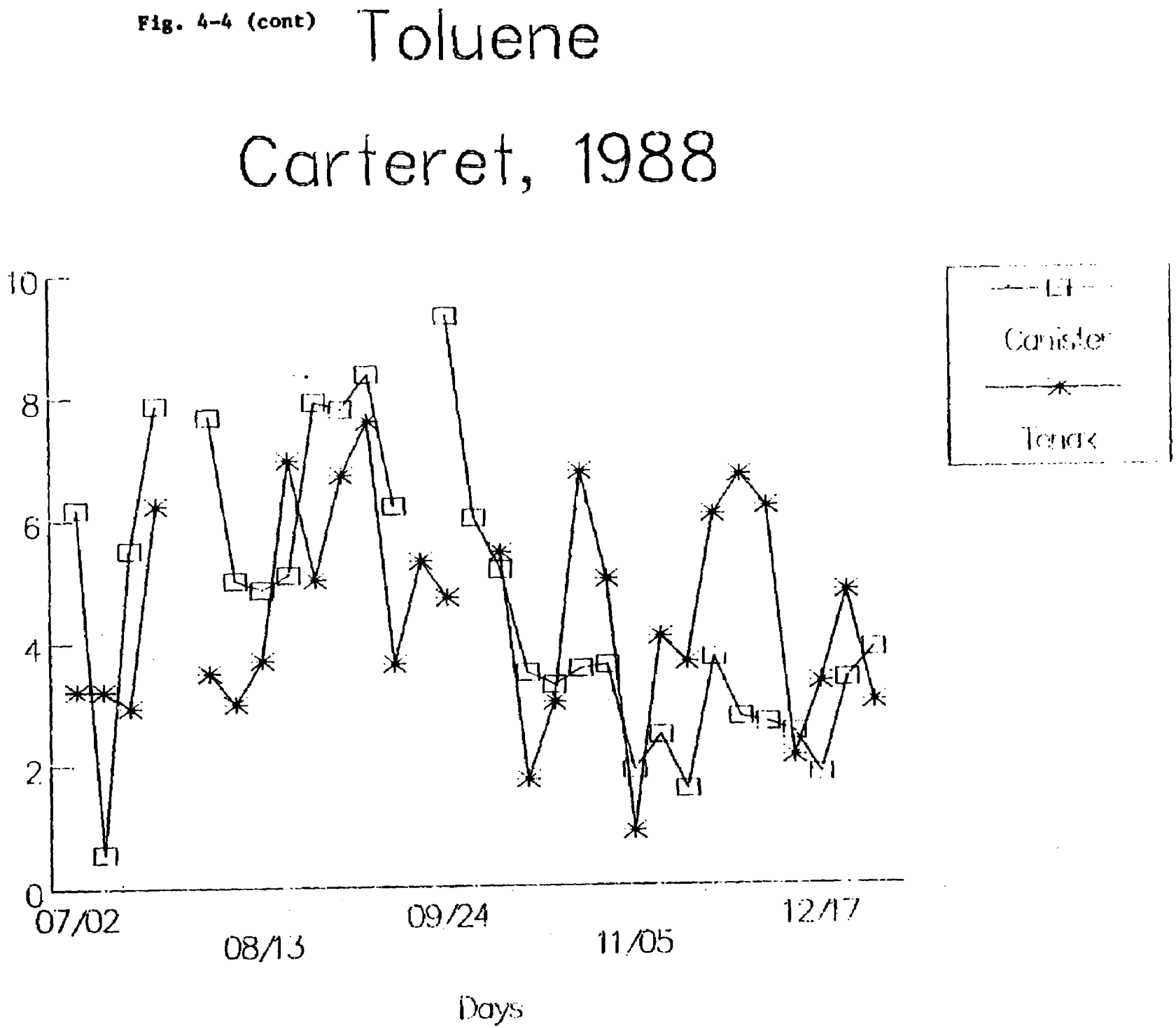

Toluene
Carteret, 1988
Canister
Tenax
10
8
6
4
2
0
07/02
08/13
09/24
11/05
12/17
Days

Fig. 4-5

Hexane

Elizabeth, 1988

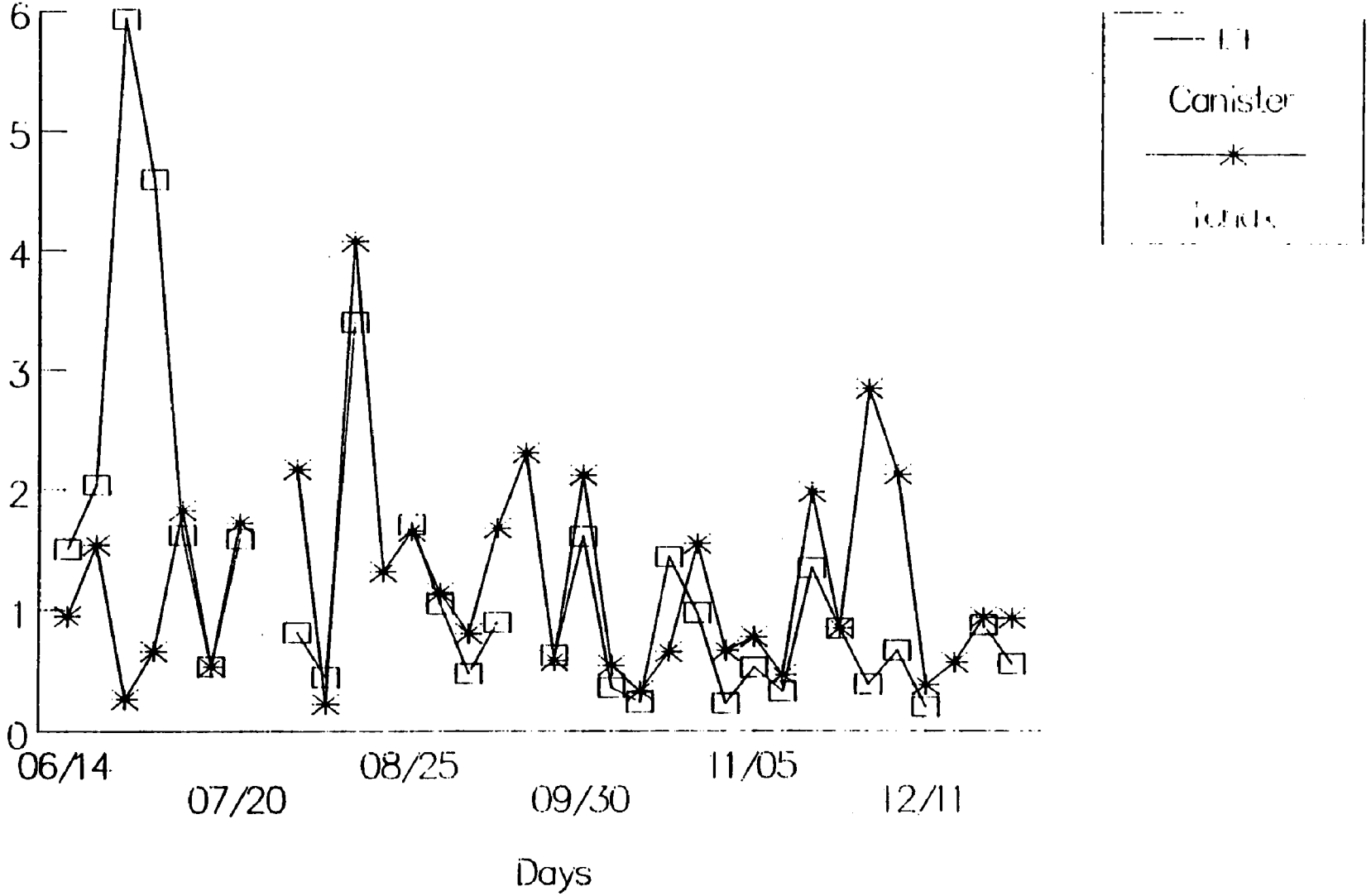
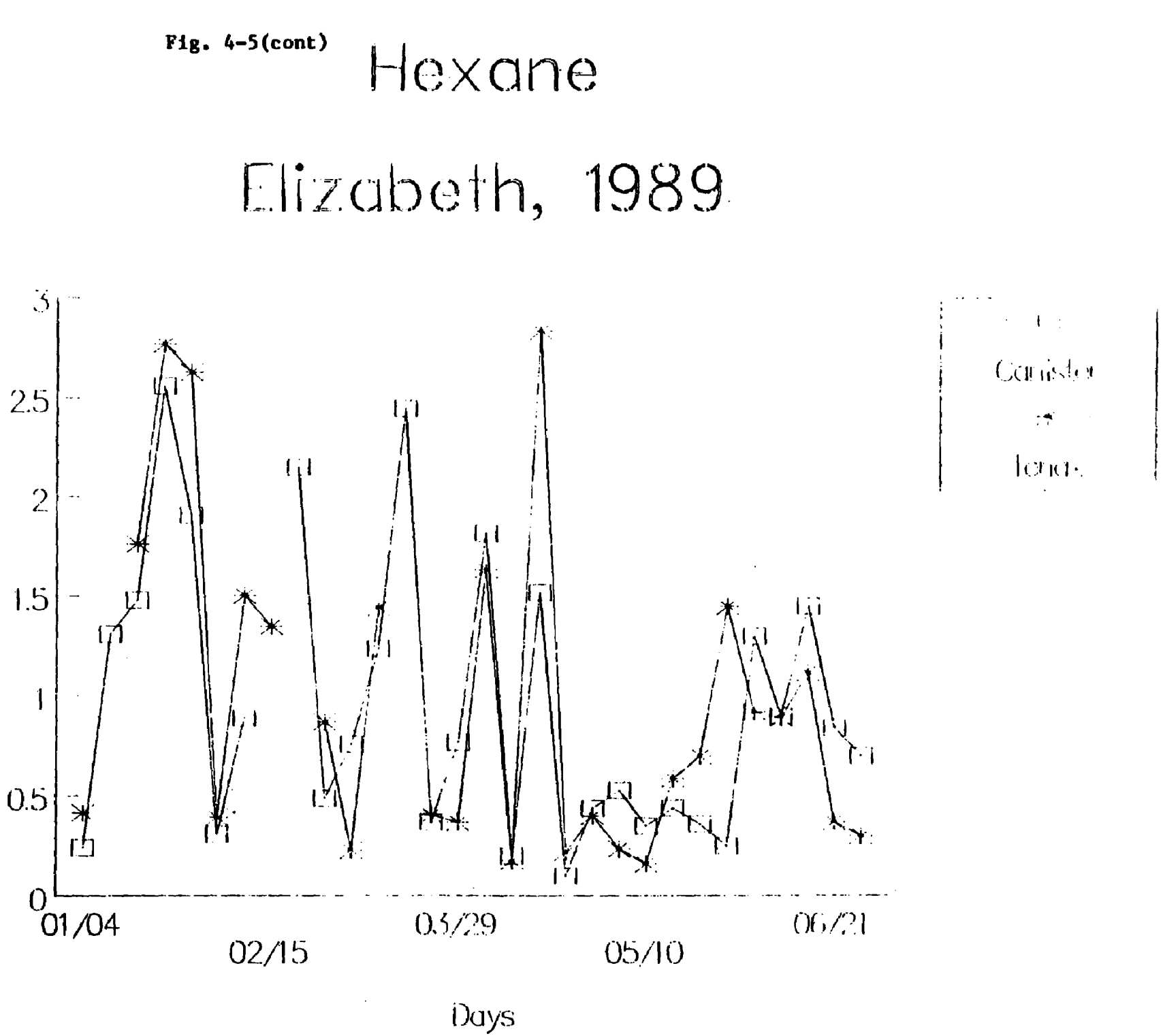


Fig. 4-5(cont)


Hexane
Elizabeth, 1989
3
2.5
2
1.5
1
0.5
0
01/04
02/15
03/29
05/10
06/21
Days
ppb
Canister
Tenax

Chapter 5

Detection Limits

Detection limits for all the target compounds are listed in Table 5-1. These were constant over the span of the project. The higher detection limit for carbon tetrachloride is due to its relatively difficult separation, as it elutes just after the much bigger benzene peak. Compounds not detected in each analytical run were listed in the reported data as being one half of the detection limit. The absolute quantitation for compounds whose usual ambient concentration falls at or near the detection limit cannot be considered accurate. Sub part-per-billion concentrations may show 100 to 200 percent errors, when the absolute error is of the order of 0.1 ppb.

Therefore, when a compound's level hovers around the detection limit, the results can only be discussed in terms of orders of magnitude. However, the assurance that the compound is not present at a substantially higher level than the detection limit is reliable. This is sufficient for risk assessment purposes.

Table 5-1

Minimum Detectable Levels
(ppb)

Chloromethane	0.01
Dichloromethane	0.01
Chloroform	0.01
1,1,1-Trichloroethane	0.01
Carbon Tetrachloride	0.05
Trichloroethylene	0.01
Tetrachloroethylene	0.01
Benzene	0.01
Toluene	0.01
Hexane	0.01
o-Xylene	0.01
m- and p-Xylene	0.01

Chapter 6

Performance Evaluation Samples

The EMSL Tenax and canister spikes from August 1987, November 1987 and June 1988 are shown in Table 6-1 to 6-3.The first data set showed wide variations in the data. The Tekmar desorber was exhibiting symptoms of a cold spot in the transfer system when these traps were being analyzed, so that most of the compounds were hanging up badly, and being carried over to subsequent samples. This was especially obvious in the trichloroethylene, perchloroethylene and toluene analyses. The results alternate from high to low, as sample material was lost from one sample and added to the next. Also, the losses due to a hot spot at the end of the oven had not yet been detected.

It should also be noted that the EPA was not informed, due to our oversight, of the proper end to load the tube. This may have led to some tubes being desorbed from the opposite end from that in which they were loaded. Finally, the blank trap was found to be contaminated, when it was analyzed, although all traps were clean when sent. The fact that the glass tube in which it was shipped was broken in transit may have contributed to the contamination. More seriously, the blank was subjected to the same sample carryover as the other samples.

The samples from the EPA filled canisters contained more moisture than we had encountered in the samples we had taken up to that time. Therefore, the on-column cryogenic trap plugged before the entire sample had been transferred to the column. This allowed only a portion of the sample to be focussed, while most of it passed onto the column without being focussed, after the separation had begun. The problem was eventually resolved by changing the carrier gas from pressure control to flow control, so that plugging was eliminated. With the flow controller, a plugged column is immediately obvious,

since the pressure would rise dramatically. Actually, this pressure rise seems sufficient to keep the trap open. The problem of slow release of components from the focussing trap was relieved by heating the trap momentarily before allowing it to return to the column temperature. This evaporates the water, and frees the organic compounds. Heating this trap sharpened the peaks, and improved the recoveries. Further, the liquid argon trap for concentration the sample was still in use at this time.

The second set of samples done in November, 1987, showed much more consistency, but were still low. This was finally attributed to the premature desorption taking place at the bottom of the oven. The canister samples were not much improved, since the major change, substitution of the -110 degree trap for the liquid argon trap had not yet been made.

The canisters sent to PEI for confirmatory analyses likewise showed improved agreement over the course of the project. The early samples analyzed at PEI were done with such high detection limits that few compounds were detected at all. PEI split canister data is shown in Table 6-4.

Table 6-1 Results of EPA Spiked Samples (August, 1987)

Tenax spiked tubes, ng/tube

Compound	Spike	Reported	%Diff.	Spike	Reported	%Diff
Chloroform	108	37,48	-60	54	trace	
111trichloroethane	292	201,180	-35	146	467,286,333	+147
Carbon Tet.	233	9,trace		116	125,118,tr	-30
Benzene	384	357,282	-16	192	238,26,273	-7
Trichloroethylene	320	288,213	-21	160	358,286,235	+83
Toluene	441	268,80	-60	221	450,294,386	+70
Perchloroethylene	354	122,26	-79	177	195,115,203	+3
o-Xylene	384	28,10	-95	192	13,3,100	-80
Overall average % difference			52%			60%

The Tekmar desorber was exhibiting symptoms of a cold spot in the transfer system when these traps were being analyzed, so that most of the compounds were hanging up badly, and being carried over to subsequent samples. This is especially obvious in the trichloroethylene, perchloroethylene and toluene analyses. The results alternate from high to low, as sample material is lost from one sample and added to the next.

It should also be noted that the EPA was not informed, due to our oversight, of the proper end to load the tube. This may have led to some tubes being desorbed from the opposite end from that in which they were loaded. Finally, the blank trap was found to be contaminated, when it was analyzed, although all traps were clean when sent. The fact that the glass tube in which it was shipped was broken in transit may have contributed to the contamination. More seriously, the blank was subjected to the same sample carryover as the other samples.

Tenax Blank:	ng/trap reported
Chloroform	-
111trichloroethane	205
Carbon Tet.	115
Benzene	5
trichloroethylene	190
toluene	100
perchloroethylene	10
o-xylene	6

Table 6--1 (cont)

Canister spikes (August, 1987)

Compound	Spike	Reported		% Difference	
		A	B	A	B
Chloroform	3.9	4.6	-	18	-
111trichloroethane	2.8	0.8	0.6	-71	-79
Carbon Tet.	3.2	2.7	-	-16	
Benzene	4.2	0.7	0.7	-83	-83
trichloroethylene	3.8	2.2	0.6	-42	-84
toluene	2.8	1.7	0.5	-39	-82
perchloroethylene	3.2	0.5	0.5	-84	-84
o-xylene	2.9	0.3	0.5	-90	-83

The samples from the EPA filled canisters contained more moisture than we had encountered in the samples we had taken all winter. Therefore, the on-column cryogenic trap plugged before the entire sample had been transferred to the column. This allowed only a portion of the sample to be focussed, while most of it passed onto the column without being focussed, after the separation had begun. The problem has been resolved by changing the carrier gas from pressure control to flow control, so that plugging has been eliminated. With the flow controller, a plugged column is immediately obvious, since the pressure would rise dramatically. Actually, this pressure rise seems sufficient to keep the trap open. The problem of slow release of components from the focussing trap has been relieved by heating the trap momentarily before allowing it to return to the column temperature. This evaporates the water, and frees the organic compounds. Heating this trap has sharpened the peaks, and improved the recoveries.

Table 6-2 Results of EPA Spiked Samples

Tenax QA Samples for Staten Island
VOC Methods Comparison (NJIT) (November, 1987)

	Spiked, ng		Reported, ng		Difference, %	
Compound	120	122	120	122	120	122
chloroform	54	108	15	45	-72	-58
1,1,1-trichloroethane	121	243	51	112	-58	-54
carbon tetrachloride	145	291	52	129	-64	-56
benzene	192	384	69	157	-64	-59
1,2-dichloroethane	137	275	NR	NR	--	--
trichloroethylene	213	426	49	159	-77	-63
1,2-dichloropropane	84	168	NR	NR	--	--
toluene	220	441	50	137	-77	-69
tetrachloroethylene	177	354	37	119	-79	-66
chlorobenzene	241	483	NR	NR	--	--
ethylbenzene	157	315	NR	NR	--	--
o-xylene	192	384	68	122	-65	-68
bromoform	210	420	NR	NR	--	--

NR - not reported

Tenax blank - o-xylene 5 ng (results uncorrected)

Table 6-2 (cont)

Results of EPA Spiked Samples
Canister QA Samples for Staten Island
VOC Methods Comparison (NJIT)
(November, 1987)

Compound	Spiked, ppb	Reported, ppb		Difference, %	
	A and B	A	B	A	B
vinyl chloride	2.5	NR	NR	--	--
bromomethane	5.9	NR	NR	--	--
trichlorofluoromethane	4.2	NR	NR	--	--
chloroform	5.2	0.1	5.0	-98	-3.8
carbon tetrachloride	4.2	3.9	5.8	-7.1	38
1,2-dichloroethane	3.4	NR	NR	--	--
1,1,1-trichloroethane	3.7	2.6	2.9	-30	-22
trichloroethylene	5.1	3.5	3.6	-31	-29
1,2-dichloropropane	3.1	NR	NR	--	--
benzene - d_6	5.5	3.0	3.7	-45	-33
1,2-dibromoethane	1.7	NR	NR	--	--
tetrachloroethylene	4.2	3.8	4.0	-9.5	-4.8
toluene - d_8	3.7	2.5	3.3	-32	-11
chlorobenzene	3.9	NR	NR	--	--
ethylbenzene	3.0	NR	NR	--	--
styrene	3.4	NR	NR	--	--
o-xylene	3.9	3.7	5.5	-5.1	41

NR - not reported

Table 6-3 Audit Sample Results for Staten Island Study
(Tenax Tubes and Canisters)

June 1988

Compound	Spiked, ng T-103	Spiked, ng T-109	Spiked, ng T-110	ppb Canister	Reported, ng T-103	Reported, ng T-109	Reported, ng T-110	ppb Canister
chloroform	72	72	108	4.8	79	85	134	4.1
1,1,1-trichloroethane	97	97	146	4.7	97	103	146	6.6
carbon tetrachloride	116	116	174	4.6	163	167	246	4.4
benzene	128	128	192	4.3	155	155	226	4.0
trichloroethylene	142	142	213	5.6	175	176	256	5.4
toluene	147	147	220	4.8	233	234	341	5.3
tetrachloroethylene	118	118	177	4.9	155	162	230	4.9
o-xylene	128	128	192	4.9	118	118	177	2.4
methylene chloride	---	---	---	4.6	---	---	---	3.4
m,p-xylene	---	---	---	---	---	---	---	0.1

Tenax-118 (Tenax blank): benzene 2 ng, toluene 2 ng, o-xylene 3 ng

Percent Bias of Audit Results

Compound	Bias, % T-103	T-109	T-110	Canister
chloroform	9.7	18	24	-15
1,1,1-trichloroethane	0	6.2	0	40
carbon tetrachloride	40	44	41	-4.3
benzene	21	21	18	-7.0
trichloroethylene	23	24	20	-3.6
toluene	58	59	55	10
tetrachloroethylene	31	37	30	0
o-xylene	-7.8	-7.8	-7.8	-51
methylene chloride	--	--	--	-26

Table 6-4

Comparison of PEI and NJIT for Canister

Date	MECL PEI	MECL NJIT	MECL BIAS	DCM PEI	DCM NJIT	DCM BIAS
01/16/88	ND	ND	-	ND	ND	-
03/22/88	ND	ND	-	1.40	ND	-
06/20/88	ND	ND	-	1.20	ND	-
09/06/88	ND	ND	-	2.50	ND	-
12/23/88	ND	ND	-	0.85	ND	-
01/22/89	ND	ND	-	0.40	ND	-
03/11/89	ND	ND	-	ND	ND	-
04/16/89	ND	ND	-	260.00	ND	-
06/27/89	ND	ND	-	1400.00	ND	-
07/15/89	ND	ND	-	570.00	ND	-
08/26/89	ND	ND	-	1.30	ND	-
AVG.	0.00	0.00	0.00	248.63	0.00	0.00
SD	0.00	0.00	0.00	415.31	0.00	0.00

Date	C6 PEI	C6 NJIT	C6 BIAS	CFOR PEI	CFOR NJIT	CFOR BIAS
01/16/88	1.80	2.00	-11.11	ND	ND	-
03/22/88	ND	0.15	-	ND	ND	-
06/20/88	ND	1.47	-	ND	0.08	-
09/06/88	ND	1.59	-	ND	0.02	-
12/23/88	1.20	1.51	-25.83	ND	0.04	-
01/22/89	1.30	1.83	-40.77	ND	0.04	-
03/11/89	ND	1.28	-	ND	0.04	-
04/16/89	1.50	1.52	-1.33	ND	0.04	-
06/27/89	ND	0.70	-	ND	0.07	-
07/15/89	1.00	1.11	-11.00	ND	0.04	-
08/26/89	2.20	1.33	39.55	ND	0.02	-
AVG.	1.50	1.32	-8.42	0.00	0.04	0.00
SD	0.80	0.49	18.85	0.00	0.02	0.00

BIAS= (PEI-NJIT)/PEI*100

Table 6-4 (cont)

Comparison of PEI and NJIT for Canister

	1,1,1			BZ		
Date	PEI	NJIT	BIAS	PEI	NJIT	BIAS
01/16/88	ND	ND	-	ND	2.49	-
03/22/88	0.75	ND	-	0.85	0.19	77.65
06/20/88	ND	0.89	-	1.00	1.70	-70.00
09/06/88	0.60	0.55	8.33	1.80	2.40	-33.33
12/23/88	0.90	0.95	-5.56	2.05	2.47	-20.49
01/22/89	0.80	0.95	-18.75	1.70	2.19	-28.82
03/11/89	ND	0.95	-	ND	2.01	-
04/16/89	0.70	1.26	-80.00	3.10	3.26	-5.16
06/27/89	0.60	0.74	-23.33	0.90	0.86	4.44
07/15/89	0.50	0.65	-30.00	1.00	1.07	-7.00
08/26/89	1.20	1.67	-39.17	2.10	2.61	-24.29
AVG.	0.76	0.96	-26.92	1.61	1.93	-11.89
SD	0.38	0.47	24.50	0.89	0.86	34.29

	CCl4			TRIC		
Date	PEI	NJIT	BIAS	PEI	NJIT	BIAS
01/16/88	ND	0.20	-	0.50	ND	-
03/22/88	ND	0.03	-	ND	ND	-
06/20/88	ND	0.30	-	ND	ND	-
09/06/88	ND	0.10	-	ND	0.08	-
12/23/88	ND	0.29	-	ND	0.07	-
01/22/89	0.20	0.33	-65.00	ND	0.09	-
03/11/89	ND	0.26	-	ND	0.08	-
04/16/89	ND	0.24	-	ND	0.09	-
06/27/89	ND	0.12	-	ND	0.05	-
07/15/89	0.30	0.52	-73.33	ND	0.06	-
08/26/89	ND	0.20	-	ND	0.09	-
AVG.	0.25	0.24	-69.17	0.50	0.08	0.00
SD	0.10	0.13	26.74	0.14	0.04	0.00

BIAS= (PEI-NJIT)/PEI*100

Table 6-4(cont)

Comparison of PEI and NJIT for Canister

Date	TOL PEI	TOL NJIT	TOL BIAS	PERC PEI	PERC NJIT	PERC BIAS
01/16/88	3.10	11.90	-283.87 *	ND	0.10	-
03/22/88	3.15	3.30	-4.76	ND	ND	-
06/20/88	5.10	7.20	-41.18	ND	0.09	-
09/06/88	5.30	8.30	-56.60	ND	0.09	-
12/23/88	3.40	3.30	2.94	0.20	0.18	10.00
01/22/89	3.10	3.80	-22.58	ND	0.12	-
03/11/89	ND	7.90	-	ND	0.43	-
04/16/89	6.80	5.70	16.18	0.20	0.15	25.00
06/27/89	1.90	1.50	21.05	0.20	0.06	70.00
07/15/89	2.30	1.90	17.39	ND	0.05	-
08/26/89	5.60	4.60	17.86	0.20	0.10	50.00
AVG.	3.98	5.40	-5.52	0.20	0.14	38.75
SD	1.85	3.01	25.53	0.10	0.11	23.24

Date	PMX PEI	PMX NJIT	PMX BIAS	OX PEI	OX NJIT	OX BIAS
01/16/88	0.60	3.80	-533.33 *	ND	0.87	-
03/22/88	1.75	6.43	-267.43 *	0.60	1.50	-150.00*
06/20/88	2.30	3.07	-33.48	0.90	1.01	-12.22
09/06/88	1.40	1.37	2.14	0.50	0.37	26.00
12/23/88	0.80	0.64	20.00	0.30	0.17	43.33
01/22/89	0.70	0.65	7.14	0.30	0.18	40.00
03/11/89	ND	2.38	-	ND	0.56	-
04/16/89	3.20	2.23	30.31	1.20	0.83	30.83
06/27/89	0.70	0.77	-10.00	0.30	0.28	6.67
07/15/89	0.90	0.57	36.67	0.40	0.21	47.50
08/26/89	2.10	1.43	31.90	0.90	0.59	34.44
AVG.	1.45	2.12	10.59	0.60	0.60	27.07
SD	0.89	1.71	21.50	0.36	0.40	19.81

BIAS= (PEI-NJIT)/PEI*100
*:Deleted in Avg. and SD.

Chapter 7

Changes in Analytical Methodology

The results of the QA samples done during the year showed that there were problems with the Tenax desorption system. It was determined that there was serious carryover between one sample and the next. This was attributed to a cold spot in the desorber, which was corrected by applying some insulation, to keep the tubing from being chilled when the cryo trap was cooled. The second set of traps loaded for us by EMSL indicated that our analysis was much more precise, but still had a significant bias on the low side. This was finally traced to the desorption oven, which was still hot at its lower end, when the temperature readout indicated that it was cool. This caused the sample at the end of the tube to begin to desorb much before the first cryotrap was cooled to collect the sample. This was addressed by removing the Tenax from the lower inch of the trap, so that the warmed end would contain no sample. The third set of traps from EMSL, analyzed after these improvements were made showed good sample recovery and quantitation.

It was determined late in the first year that there was some interference with some of the halogenated compounds, especially carbon tetrachloride, which was giving much higher values than were being obtained by other groups. The electron capture detector is not subject to this interference, so the system was modified to obtain areas for the ECD peaks as well as the FID. This change was made on the Tenax analyses in midsummer, and on the canister system in early November.

Finally, when the canister analysis system was first set up, the peaks were poorly resolved at the beginning of the chromatogram. This was thought to be due to water condensing in the second cryotrap and interfering with the rapid injection of the volatiles onto the column. A Permapure drier containing a semipermeable membrane to remove water

from the sample before the first trap was tried. This proved not to be successful. Further study showed that the problem with water was not the main cause of the poor resolution. If water did not enter the trap at a very rapid rate, it tended to freeze on the walls, allowing the gas flow to pass through the tube center. If the trap was not plugged, there was no significant effect on the resolution. However, the presence of carbon dioxide in the sample was the major cause of the peak broadening. The gaseous carbon dioxide, which occupies a volume of nearly half a milliter, in the average sample, expands rapidly from the solid state in the focussing trap, and spreads the previously concentrated sample band. The problem was addressed by raising the temperature of the first trap to -110°C, thus preventing the trapping of carbon dioxide. This change was made in April, 1988. The data from canister samples done prior to that time were removed from the database, except for a few late-eluting compounds which could be reasonably well measured.

A summary of the improvements made in the VOC analysis, with the dates the changes were implemented is shown in Table 7-1.

Table 7.1

Summary of Changes in Analytical Methodology

Tenax Samples

July 1987	Testing, no samples reported.
August/Sept	Late eluting compounds were not reported because a cold spot in the Tekmar desorber caused carryover between samples for these less volatile compounds.
Oct/March '88	All compounds reported, cold spot eliminated.
April '88	The length of Tenax in the traps was reduced to keep the adsorbent bed out of the hot spot at the bottom of the desorber oven. Data from previous analyses was not deleted, since the impact of this hot spot was felt to affect the loaded performance evaluation samples much more seriously than the actual samples.
Jan-June '89	Slightly lower values were reported for the chlorinated compounds due to a somewhat diminished detector sensitivity. No data was eliminated, as the results were not seriously different from the canister results.

Canister Samples

Sept 1987	Early compounds were not well separated. Only 3 compounds were reported, and only FID detection was used.
Oct/March '88	Separation still poor, but ECD was installed for identification of chlorinateds, so additional compounds are reported.
April/June	Major improvement in the canister analysis is accomplished by raising the temperature of the concentrating trap, to avoid the trapping of carbon dioxide. Some chlorinated compounds were still subject to interference, and the ECD was not calibrated for quantitation at this time.
July '88	All compounds were being determined, and the method was working well.

Chapter 8

Assessment of the Data for Each Target Compound

Chloromethane

The variability of chloromethane was constant over the project time. While the results appear to agree reasonably well from both analytical systems, experiments in our laboratory show that both methods show substantially low results for this compound, due to the fact that it breaks through on Tenax, and is not completely trapped if the temperature of the concentrating trap in the canister system is not rigidly controlled at -110°C. Since this temperature was controlled by a manually placed cold bath for much of the project, losses invariably occurred. The data for this compound should only be used as "minimum levels present".

Method	Time period	Number Samples Run	Mean Conc (ppb)	Mean Diff. Between Pairs	SD of Mean Diff. of Pairs (ppb)
Tenax	7/87-10/89	146	0.47	0.22	0.21
				Pooled SD for 3 Replicates	
Canister	7/87-10/89	98	0.61	0.07	

Dichloromethane

A substantial number of data points for this compound were lost because of a contamination in one of the bellows sampling pumps used in the canister system. The variability did not change substantially over the course of the project. Most of the data points were very near to the detection limits and therefore can only be considered as order of magnitude measurements.

Method	Time period	Number Samples Run	Mean Conc (ppb)	Mean Diff. Between Pairs	SD of Mean Diff. of Pairs (ppb)
Tenax	7/87-10/89	217	0.01	0.0	0.0
				Pooled SD for 3 Replicates	
Canister	7/87-10/89	145	0.01	-	

Hexane

Hexane showed good agreement between and within methods, and laboratory experiments as well as performance evaluation samples showed acceptable recovery for hexane.

Method	Time period	Number Samples Run	Mean Conc (ppb)	Mean Diff. Between Pairs	SD of Mean Diff. of Pairs (ppb)
Tenax	7/87-10/89	225	0.87	0.10	0.08
				Pooled SD for 3 Replicates	
Canister	7/87-10/89	185	1.17	0.06	

Chloroform

Chloroform levels throughout the project were very near to the detection limits and therefore the data must be be considered as order of magnitude measurements.

Method	Time period	Number Samples Run	Mean Conc (ppb)	Mean Diff. Between Pairs	SD of Mean Diff. of Pairs (ppb)
Tenax	7/87-10/89	223	0.02	0.0	0.01
				Pooled SD for 3 Replicates	
Canister	7/87-10/89	146	0.04	0.01	

1,1,1-Trichloroethylene

The reliability of the analysis for this compound was improved several times during the project, by substitution of the ECD detector for the FID in each system, and by a change in the concentrating trap temperature to improve resolution.

However, the Tenax method gave consistantly lower results for this compound than did the canister method. In the various performance evaluation and shootout samples, the Tenax method showed better agreement than did the canister. While we have no evidence that there was an interference in the canister analysis which was not a problem with the Tenax analysis, the PE samples and shootout results lead us to have more confidence in the Tenax results, where the two results differ.

Method	Time period	Number Samples Run	Mean Conc (ppb)	Mean Diff. Between Pairs	SD of Mean Diff. of Pairs (ppb)
Tenax	7/87-3/88	32	0.51	0.0	0.04
Tenax	4/88-10/89	196	0.56	0.10	0.04
				Pooled SD for 3 Replicates	
Canister	7/87-3/88	0			
Canister	4/88-10/89	149	1.06	0.08	

Benzene

Benzene showed good agreement between and within methods, and laboratory experiments as well as performance evaluation samples showed acceptable recovery for benzene.

Method	Time period	Number Samples Run	Mean Conc (ppb)	Mean Diff. Between Pairs	SD of Mean Diff. of Pairs (ppb)
Tenax	7/87-10/89	224	1.23	0.18	0.08
				Pooled SD for 3 Replicates	
Canister	7/87-10/89	177	1.41	0.07	

Carbon Tetrachloride

The reliability of the analysis for this compound was improved several times during the project, by substitution of the ECD detector for the FID in each system, and by a change in the concentrating trap temperature to improve resolution.

Method	Time period	Number Samples Run	Mean Conc (ppb)	Mean Diff. Between Pairs	SD of Mean Diff. of Pairs (ppb)
Tenax	7/87-3/88	32	0.14	0.02	0.0
Tenax	4/88-10/89	197	0.13	0.03	0.01
				Pooled SD for 3 Replicates	
Canister	7/87-3/88	0		0.03	
Canister	4/88-10/89	149	0.18	0.02	

Trichloroethylene

The reliability of the analysis for this compound was improved several times during the project, by substitution of the ECD detector for the FID in each system, and by a change in the concentrating trap temperature to improve resolution.

Method	Time period	Number Samples Run	Mean Conc (ppb)	Mean Diff. Between Pairs	SD of Mean Diff. of Pairs (ppb)
Tenax	7/87-3/88	32	0.12	0.01	0.01
Tenax	4/88-10/89	192	0.05	0.01	0.0
				Pooled SD for 3 Replicates	
Canister	7/87-3/88	0			
Canister	4/88-10/89	148	0.06	0.01	

Toluene

Changes in the concentrating trap temperatures for the canister system and the detection and elimination of the cold spot problems in the Tenax desorption system allowed improvements in the quantitation of this compound.

Method	Time period	Number Samples Run	Mean Conc (ppb)	Mean Diff. Between Pairs	SD of Mean Diff. of Pairs (ppb)
Tenax	7/87-6/88	43	2.47	0.02	0.43
Tenax	7/88-10/89	181	3.92	0.37	0.17
				Pooled SD for 3 Replicates	
Canister	7/87-3/88	27	4.83	0.14	
Canister	4/88-10/89	149	3.6	0.14	

Tetrachloroethylene

Remediation of the cold spot problems in the desorption system and substitution of the ECD in the analytical systems improved the analysis of this compound.

Method	Time period	Number Samples Run	Mean Conc (ppb)	Mean Diff. Between Pairs	SD of Mean Diff. of Pairs (ppb)
Tenax	7/87-6/88	45	0.18	0.03	0.01
Tenax	7/88-10/89	183	0.20	0.03	0.02
				Pooled SD for 3 Replicates	
Canister	7/87-3/88	28	0.11	0.04	
Canister	4/88-10/89	149	0.14	0.01	

m & p-Xylene

The cold spot problems in the Tenax analysis system caused low values for this compound in the early stages of the project. The samples done before 7/88 should be considered low.

Method	Time period	Number Samples Run	Mean Conc (ppb)	Mean Diff. Between Pairs	SD of Mean Diff. of Pairs (ppb)
Tenax	7/87-6/88	45	0.77	0.03	0.12
Tenax	7/88-10/89	181	1.12	0.14	0.06
				Pooled SD for 3 Replicates	
Canister	7/87-10/89	183	1.63	0.09	

o-Xylene

The cold spot problems in the Tenax analysis system caused low values for this compound in the early stages of the project. The samples done before 7/88 should be considered low.

Method	Time period	Number Samples Run	Mean Conc (ppb)	Mean Diff. Between Pairs	SD of Mean Diff. of Pairs (ppb)
Tenax	7/87-6/88	45	0.34	0.03	0.03
Tenax	7/88-10/89	181	0.37	0.08	0.02
				Pooled SD for 3 Replicates	
Canister	7/87-10/89	184	0.72	0.04	

11. PAPER ON EMISSIONS INVENTORY DEVELOPMENT FOR PUBLICLY-OWNED TREATMENT WORKS

Report on Emissions Inventory Development for Volatile Organic Compounds from Publicly Owned Treatment Works in the New York and New Jersey Ozone Nonattainment Areas

Roch C. Raimonde
Betty Martinovich

Air and Waste Management Division
Environmental Protection Agency
Region II
26 Federal Plaza
New York, New York 10278

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REPORT ON EMISSIONS INVENTORY DEVELOPMENT FOR VOLATILE ORGANIC COMPOUNDS FROM PUBLICLY OWNED TREATMENT WORKS IN THE NEW YORK AND NEW JERSEY OZONE NONATTAINMENT AREAS

Abstract

Publicly Owned Treatment Works (POTWs), more commonly known as sewage treatment plants, are increasingly gaining national attention as being large emitters of volatile organic compounds (VOCs) and airborne toxics. The Environmental Protection Agency (EPA) and the states do not now regulate VOC emissions from POTWs. We have, in anticipation of post-1987 SIP planning, conducted a preliminary study of the POTWs in the ozone nonattainment areas of New York (the five boroughs of New York City, Westchester, Rockland, Nassau and Suffolk Counties) and New Jersey (the entire State is nonattainment). Using data obtained from pretreatment reports on volatile organics in the influent streams to the POTWs along with the percent removals of these organics at each plant and estimated volatilization percentages of each pollutant (obtained from the Report to Congress on the Discharge of Hazardous Wastes to Publicly Owned Treatment Works), we have calculated estimates of emissions of VOCs from the POTWs which require pretreatment in our area.

The results are significant. POTWs in our area appear to emit more than 10,000 tons of VOCs per year. This is roughly equal to the emission reductions projected to be achievable through the implementation of individual "extraordinary measures" such as the control of VOCs from auto refinishing operations or architectural surface coatings. This report outlines the steps used in developing the estimated emissions inventory of VOCs from POTWs and further compares the results to the extraordinary measures under study in Region II. The results strongly suggest that a small number of POTWs in the Region may be major sources of VOCs, each emitting greater than 100 tons per year. While no air monitoring studies were done to support these estimates, some air data is available and these seem to support our conclusions. The results also suggest that EPA and the states must consider POTWs in a comprehensive post-1987 planning effort to attain the national ambient air quality standard for ozone.

Our estimates of the percent of the organics that volatilize from the amount removed were also based upon the assumption that each system was not acclimated. We used Table 4.8 in The Report to Congress, which also lists the average fractions released (we chose the "Unacclimated Median Release" values). When this assumption is made, volatilization becomes the dominant mechanism for the removal of volatile organics (in an acclimated system, biodegradation plays a much larger role). (4) The values listed in this table are based upon each individual chemical's Henry's Constant and solubility, among other factors.

There are better methods to estimate the percent subject to volatilization at a specific POTW, but these imply knowledge of detailed information at the POTW, including windspeeds and volumes of aeration tanks. However, our intention was to attempt to simplify these calculations because we didn't have such information available at the time this paper was written. For alternate methods, see Namkung and Rittman, "Estimating Volatile Organic Compound (VOC) Emissions from Publicly Owned Treatment Works (POTWs), JWPCF, 59, 670, 1987; and Corsi, Chang, Schroeder and Qiu, "Emissions of Volatile and Potentially Toxic Organic Compounds from Municipal Wastewater Treatment Plants" APCA meeting, New York, NY, 87-95.7, June 1987.

With this information, we simply multiplied the influent concentration by the percent removal to give the amount of the influent removed. Multiplying this product by the percent that volatilizes gave us the air emissions (the percent that volatilizes of the percent removed). This value represents the estimated air emissions (in ug/l) of priority pollutants. To get our low estimate of total emissions at the POTW, we assumed a 50 percent non-priority contribution. Our high estimate, based upon conversations with staff of the New Jersey Department of Environmental Protection (5) and staff from an environmental consulting firm, Science Applications International Corporation - the authors of The Report to Congress (6), reflects a non-priority contribution of 90 percent.

This value is then scaled to tons/year via the following conversion:
Air Emissions (ug/l) x Flow at POTW (Millions of Gal/day) x (10liters/2.64gal) x 365 days/year x 10exp-9 Kg/ug x 2.204lb/kg x 1 ton/2000lb.

Flow at each POTW is available in each pretreatment report. Our data was provided by the Water Management Division of EPA, Region II. (7)

Results

A sample calculation is shown in Table 3 for the Middlesex County POTW. Table 4 presents a summary of our results. It is important to note that our low and high estimates do not include photochemically unreactive VOCs (methylene chloride, 1,1,1-trichloroethane, and chlorinated fluorocarbons). However, their emissions tend to be quite sizable. The "Low Total" column includes these emissions. While not important for their contribution to ozone formation, these emissions may be subject to air toxics, NESHAPS or NSPS regulations. As Table 4 shows, the low estimate of photochemically reactive VOCs in New York is relatively low (460.14 tons/year). But the low estimate including non-reactive VOCs is over twice as high (1135.68 tons/year).

Once we established emissions estimates for POTWs for which there was sufficient data, further estimations had to be made to include those facilities for which no data was available. This was done by calculating the emissions per unit flow at each POTW (with data) in a state and then taking the average of these quotients. We then multiplied this by the total flow (for all POTWs requiring pretreatment) in the state. Note that there is a high and a low estimate that reflects the estimates made for individual facilities. In New York, the range of emissions per unit flow was from 0.21 to 1.09 tons VOC/yr/MGD, which gives a range of total emissions of 460.14 to 2300.71 tons VOC/year. In New Jersey, because of the uniqueness of the Middlesex County POTW (see Figure 1), we based the total for the rest of the State on the range of emissions per unit flow that we calculated without including this facility. The emissions from the Middlesex County facility were then added to this total to get the total estimates for the entire State. In New Jersey, the range of emissions per unit flow was from 4.62 to 23.12 tons VOC/yr/MGD. This translated to a range of emissions (including Middlesex County) of 5386.52 to 26932.62 tons VOC/year. Our estimated emissions for the region is 5846.67 to 29233.34 tons VOC/year. It is not surprising that the majority of VOC emissions are estimated to come from New Jersey POTWs. The mean industrial discharge to POTWs in New Jersey is almost five times as great as that in New York, with a much larger number of organics manufacturers located in New Jersey. (8)

We did not have the resources to verify our estimates through an air monitoring program. However, we obtained data from a monitoring program conducted by the New Jersey Department of Environmental Protection at the Middlesex

are of the same magnitude as those projected through the "extraordinary measures." Considering the numbers of sources needed for full compliance of the "extraordinary measures" (gasoline stations, dry cleaners, etc.), POTWs may be one of the most feasible and more easily achievable categories for reductions remaining.

In this study, we have not explored the actual methods for reducing VOC emissions from POTWs. Some plants now have odor controls, of which we may be able to take advantage in designing controls for VOCs. We hope to explore this issue in the future. If our estimates are correct, it appears that these controls will be essential in a comprehensive post-1987 effort to attain the national ambient air quality standard for ozone.

Referances:

1. Baamonde, Kapichak and Truchan, "An Evaluation of the Programs to Attain the Ozone and Carbon Monoxide Standards in New Jersey and New York", USEPA Air Programs Branch Region II, New York, NY, presented at APCA convention, June 1987.

2. USEPA, Office of Water Regulations and Standards, "Report to Congress on the Discharge of Hazardous Wastes to Publicly Owned Treatment Works", Washington, D.C., February 1986.

3. CFM Incorporated Environmental Engineering Services, "Report Upon Investigation of Organic Priority Pollutants in the Influent to the Passaic Valley Sewerage Commissioners Treatment Plant, Whippany, New Jersey, May 1986.

4. Report to Congress, Table 4.8.

5. J. Kelley, New Jersey Department of Environmental Protection, Trenton, New Jersey, private communication, 1986.

6. A. Jones, Science Applications International Corporation, McLean Virginia, private communication, 1986.

7. Gallup and Prorok, USEPA, Office of Water Enforcement and Permits, "Pretreatment Program Approval Status List" Washington, D.C., March 1986.

8. Ibid.

9. Harkov, Jenks, and Ruggeri, "Volatile Organic Compounds in the Ambient Air Near a Large, Regional Sewage Plant in New Jersey", New Jersey Department of Environmental Protection, Trenton, New Jersey, presented at APCA convention June, 1987.

10. Greenberg and Pause, ERT Inc, "Ambient Air Monitoring and Wastewater and Sludge Monitoring for VOCs and Trace Metals at the Fields Point POTW, Providence Rhode Island", Department of Environmental Management, State of Rhode Island, Providence, Rhode Island, March 1987.

11. Regulatory Integration Division, Office of Policy Planning and Evaluation, USEPA, "Draft Final Report of the Philadelphia Integrated Environmental Management Project", Washington, D.C., June 1986.

12. CFM Incorporated, "Report Upon Investigation of Organic Priority Pollutants in the Influent to the Passaic Valley Sewerage Commissioner's Treatment Plant", Whippany, New Jersey, May 1986.

Pretreatment Reports Used:

1. Stony Brook Regional Sewerage Authority, "Pretreatment Program Submission", Princeton, New Jersey, April 1983.

2. Malcolm Pirnie, Inc., "Industrial Pretreatment Program Phase One - The Pequannock, Lincoln Park and Fairfield Sewerage Authority" Paramus, New Jersey, September 1983.

3. BCM, Inc., "Final Draft Report, Hamilton Township, Municipal-Industrial Pretreatment Program" Plymouth Meeting, PA March 1983.

4. Elson T. Killam Associates, Inc. "Rahway Valley Sewerage Authority Industrial Pretreatment Program Part I" Milburn, New Jersey, August 1982.

5. Metcalf & Eddy, Inc., "Somerset Raritan Valley Sewerage Authority Industrial Pretreatment Program Phase 1, Part 2 Technical Report, New York, April 1984.

6. Buck, Siefert & Jost, Inc. "Ewing-Lawrence Sewerage Authority Industrial Pretreatment Program", Englewood Cliffs, New Jersey, April 1983.

Middlesex County Data Obtained From #7:

7. USEPA, "Fate of Priority Pollutants In Publicly Owned Treatment Works", Volumes I and II, Washington, D.C., September 1982.

TABLE 2

Priority Pollutants

PURGEABLE ORGANICS (31 COMPOUNDS)

Acrolein	1, 2-Dichloropropane
Acrylonitrile	1, 3-Dichloropropene
Benzene	Methylene chloride
Toluene	Methyl chloride
Ethylbenzene	Methyl bromide
Carbon tetrachloride	Bromoform
Chlorobenzene	Dichlorobromomethane
1, 2-Dichloroethane	Trichlorofluoromethane
1, 1, 1-Trichloroethane	Dichlorodifluoromethane
1, 1-Dichloroethane	Chlorodibromomethane
1, 1-Dichloroethylene	Tetrachloroethylene
1, 1, 2 Trichloroethane	Trichloroethylene
1, 1, 2, 2 Tetrachloroethane	Vinyl chloride
Chloroethane	1, 2-trans-Dichloroethylene
2-Chloroethyl vinyl ether	bis (Chloromethyl) ether
Chloroform	

BASE/NEUTRAL EXTRACTABLE ORGANICS (46 COMPOUNDS)

1, 2-Dichlorobenzene	Fluorene
1, 3-Dichlorobenzene	Fluoranthene
1, 4-Dichlorobenzene	Chrysene
Hexachloroethane	Pyrene
Hexachlorobutadiene	Phenanthrene
Hexachlorobenzene	Anthracene
1, 2, 4-Trichlorobenzene	Benzo (a) anthracene
bis (2-Chloroethoxy) methane	Benzo (b) fluoranthene
Naphthalene	Benzo (k) fluoranthene
2-Chloronaphthalene	Benzo (a) pyrene
Isophorone	Indeno (1, 2, 3-c, d) pyrene
Nitrobenzene	Dibenzo (a, h) anthracene
2, 4-Dinitrotoluene	Benzo (g, h, i) perylene
2, 6-Dinitrotoluene	4-Chlorophenyl phenyl ether
4-Bromophenyl phenyl ether	3, 3'-Dichlorobenzidine
bis (2 Ethylhexyl) phthalate	Benzidine
Di-n-octyl phthalate	bis (2-Chloroethyl) ether
Dimethyl phthalate	1, 2-Diphenylhydrazine
Diethyl phthalate	Hexachlorocyclopentadiene
Di-n-butyl phthalate	N-Nitrosodiphenylamine
Acenaphthylene	N-Nitrosodimethylamine
Acenaphthene	N-Nitrosodi-n-propylamine
Butyl benzyl phthalate	bis (2-Chloroisopropyl) ether

ACID EXTRACTABLE ORGANICS (11 COMPOUNDS)

Phenol	p-Chloro-m-cresol
2-Nitrophenol	2-Chlorophenol
4-Nitrophenol	2, 4-Dichlorophenol
2, 4-Dinitrophenol	2, 4, 6-Trichlorophenol
4, 6-Dinitro-o-cresol	2, 4-Dimethylphenol
Pentachlorophenol	

PESTICIDES/PCB'S (26 COMPOUNDS)

α-Endosulfan	Heptachlor
β-Endosulfan	Heptachlor epoxide
Endosulfan sulfate	Chlordane
α-BHC	Toxaphene
β-BHC	Aroclor 1016
δ-BHC	Aroclor 1221
γ-BHC	Aroclor 1232
Aldrin	Aroclor 1242
Dieldrin	Aroclor 1248
4, 4'-DDE	Aroclor 1254
4, 4'-DDD	Aroclor 1260
4, 4'-DDT	2, 3, 7, 8-Tetrachlorodibenzo-p-dioxin (TCDD)
Endrin	
Endrin aldehyde	

METALS (13 ELEMENTS)

Antimony	Mercury
Arsenic	Nickel
Beryllium	Selenium
Cadmium	Silver
Chromium	Thallium
Copper	Zinc
Lead	

MISCELLANEOUS (3 ANALYTES)

Ast...sos (P...os)
Tot... Ion
Tot... ...ons

TABLE 3

Sample Calculation

MIDDLESEX COUNTY

Pollutant	Influent (ug/l)	% Removal	% Volatilization	To Air (ug/l)
Benzene	220.00	100.00	80.00	179.20
Carbon Tetrachloride	111.00	88.00	90.00	103.75
1,2-Dichloroethane	6582.00	33.00	90.00	1954.85
Chlorobenzene	5.00	80.00	50.00	2.00
1,1-Dichloroethane	2.00	50.00	90.00	0.90
1,1,2-Trichloroethane	5.00	20.00	90.00	0.90
Tetrachloroethene	1.00	100.00	60.00	0.00
1,2-trans-Dichloroethylen	11.00	91.00	90.00	9.01
1,2-Dichloropropane	430.00	100.00	90.00	390.20
Ethylbenzene	157.00	97.00	80.00	121.83
Methyl Chloride	321.00	67.00	80.00	172.06
Methyl Bromide	90.00	70.00	80.00	56.16
Bromoform	1.00	100.00	50.00	0.50
Dichlorobromomethane	10.00	50.00	50.00	2.50
Chlorodibromomethane	1.00	100.00	50.00	0.50
Tetrachloroethylene	2062.00	05.00	80.00	1946.16
Toluene	6045.00	100.00	80.00	3876.00
Trichloroethylene	659.00	07.00	80.00	650.66
Vinyl chloride	15.00	100.00	95.00	14.25
1,2-Dichlorobenzene	66.00	2.00	90.00	0.01
1,4-Dichlorobenzene	4.00	5.00	90.00	0.18
Chloroform	110.00	50.00	90.00	53.90
1,1-Dichloroethylene	31.00	96.00	90.00	26.23
Subtotal				9376.37
Methylene Chloride	795.00	100.00	60.00	477.00
1,1,1-Trichloroethane	8575.00	87.00	90.00	5140.23
Trichlorofluoromethane	37.00	97.00	90.00	32.30
Subtotal (non-reactive)				5657.53
Calculations:	Reactive	Non-reactive	Total	
= 508 non-priority	18740.74	5657.53		
= flow	2249849.00	679903.12		
= conversion (=tons/yr)	3419.77	1031.93	4451.70	

FIGURE 1

Emissions/Flow at New Jersey POTWs

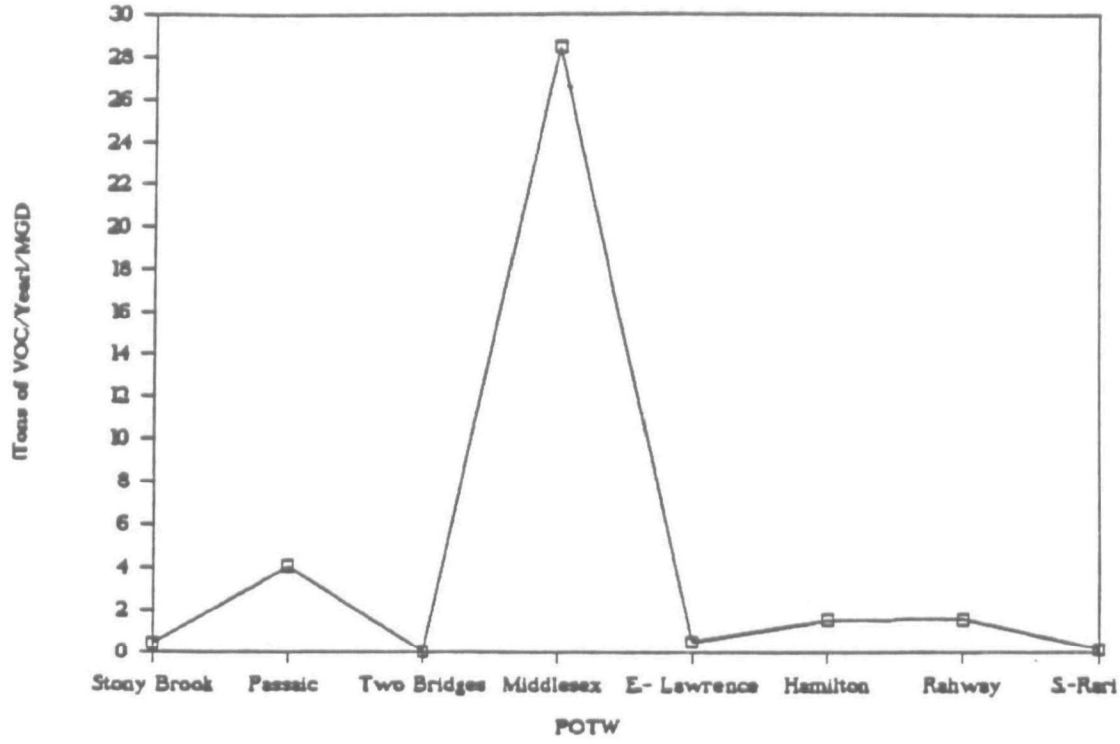

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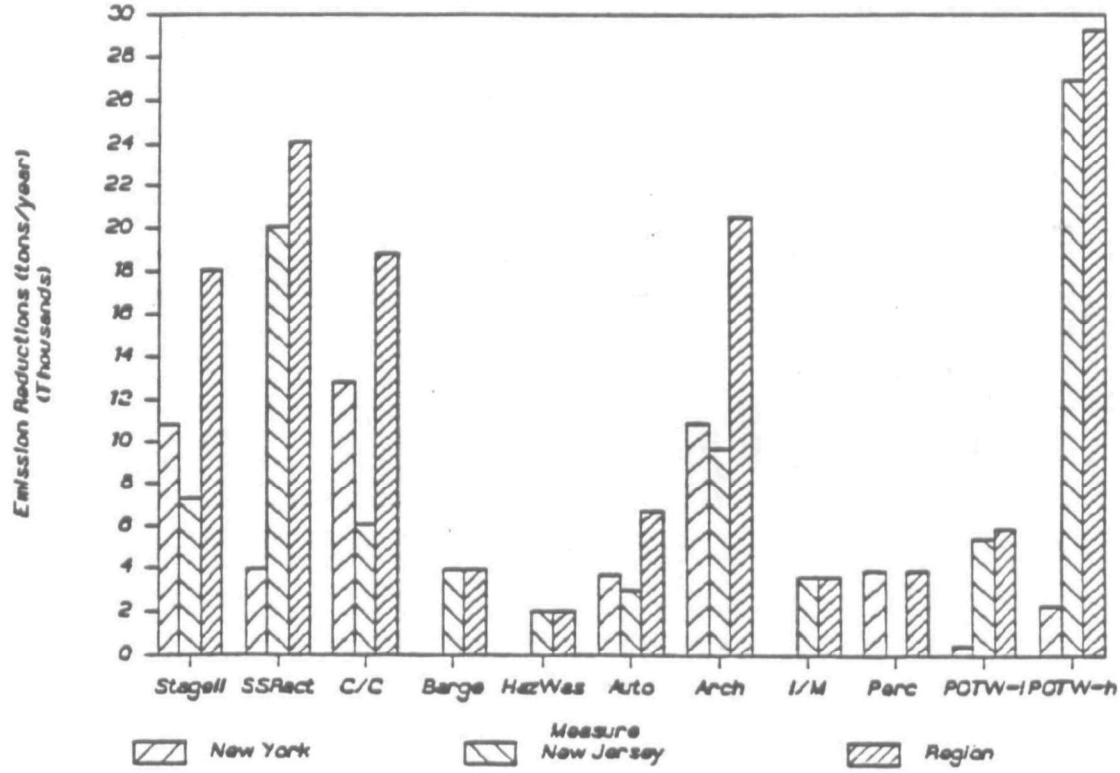
FIGURE 2

POTW Emissions v Extraordinary Measures



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12. SUPPORT DOCUMENTS FOR REFERENCE CONCENTRATIONS AND INHALATION UNIT RISK FACTORS FROM THE INTEGRATED RISK INFORMATION SYSTEM (IRIS)

Description of Carcinogenicity Section

The Carcinogenicity Assessment Section provides information on three aspects of the carcinogenic risk assessment for the agent in question; the U.S. EPA classification, and quantitative estimates of risk from oral exposure and from inhalation exposure. The classification reflects a weight-of-evidence judgment of the likelihood that the agent is a human carcinogen. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per mg/kg/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The Carcinogen Assessment Background Document provides details on the rationale and methods used to derive the carcinogenicity values found in IRIS. Users are referred to the Oral RfD and Inhalation RfC Sections for information on long-term toxic effects other than carcinogenicity.

Note: Under Review indicates that the chemical is currently being review by the Carcinogen Risk Assessment Verification Endeavor.

CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name: Xylenes
CASRN: 1330-20-7

EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification: D; not classifiable as to human carcinogenicity

BASIS

Orally administered technical xylene mixtures did not result in significant increases in incidences in tumor responses in rats or mice of both sexes.

HUMAN CARCINOGENICITY DATA

None.

ANIMAL CARCINOGENICITY DATA

Inadequate. In an NTP (1986) study, 50 male and 50 female F344/N rats were treated by gavage with mixed xylenes in corn oil (60% m-xylene, 14% p-xylene, 9% o-xylene and 17% ethylbenzene) at dosages of 0, 250 or 500 mg/kg/day, 5 days/week for 103 weeks. Similarly, 50 male and 50 female B6C3F1 mice were treated with the same xylene mixture at dosages of 0, 500 or 1000 mg/kg/day. Animals were killed and examined histologically when moribund or after 104-105 weeks. An apparent dose-related increased mortality was observed in male rats, but this difference was statistically significant for the high dose group, only. No other differences in survival between dosage groups of either sex were observed. Interstitial cell tumors of the testes could not be attributed to administration of the test compound observed in male rats (43/50 control, 38/50 low-dose and 41/49 high-dose). NTP (1986) reported that there were no significant changes in the incidence of neoplastic or nonneoplastic lesions in either the rats or mice that could be considered related to the mixed xylene treatment, and concluded that under the conditions of these 2-year gavage studies, there was "no evidence of carcinogenicity" of xylene (mixed) for rats or mice of either sex at any dosage tested.

Maltoni et al. (1985), in a limited study, reported higher incidences (compared with controls) of malignant tumors in male and female Sprague-Dawley rats treated by gavage with xylene in olive oil at 500 mg/kg/day, 4 or 5 days/week for 104 weeks. This study did not report survival rates or specific tumor types; therefore, the results cannot be interpreted.

Berenblum (1941) reported that "undiluted" xylene applied at weekly intervals produced one tumor-bearing animal out of 40 after 25 weeks in skin-painting experiments in mice. No control groups were described. Pound (1970) reported negative results in initiation-promotion experiments with xylene as the

initiator and croton oil as the promotor.

SUPPORTING DATA FOR CARCINOGENICITY

The frequency of sister chromatid exchanges and chromosomal aberrations were nearly identical between a group of 17 paint industry workers exposed to xylene and their respective referents (Haglund et al., 1980). In vitro, xylene caused no increase in the number of sister chromatid exchanges in human lymphocytes (Gerner-Smidt and Friedrich, 1978). Studies indicate that xylene isomers, technical grade xylene or mixed xylene are not mutagenic in tests with Salmonella typhimurium (Florin et al., 1980; NTP, 1986; Bos et al., 1981) nor in mutant reversion assays with Escherichia coli (McCarroll et al., 1981). Technical grade xylene, but not o- and m-xylene, was weakly mutagenic in Drosophila recessive lethal tests. Chromosomal aberrations were not increased in bone marrow cells of rats exposed to xylenes by inhalation (Donner et al., 1980).

——— QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE ———

No Data Available

— QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE —

No Data Available

———————— EPA DOCUMENTATION AND REVIEW ————————

Source Document: U.S. EPA. 1987. Drinking Water Criteria Document for Xylene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. Final.

The Drinking Water Criteria Document for Xylene has received Agency and external review.

Agency Work Group Review: 12/02/87

Verification Date: 12/02/87

———————— EPA CONTACTS (CARCINOGENICITY ASSESSMENT) ————————

Bruce Mintz / OST -- (202)260-9569

W. Bruce Peirano / OHEA -- (513)569-7540

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Pound, A.W. 1970. Induced cell proliferation and the initiation of skin tumor formation in mice by ultraviolet light. Pathology. 2: 269-275.

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CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name: Ethylbenzene
CASRN: 100-41-4

EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification: D; not classifiable as to human carcinogenicity

BASIS

nonclassifiable due to lack of animal bioassays and human studies.

HUMAN CARCINOGENICITY DATA

None.

ANIMAL CARCINOGENICITY DATA

None. NTP has plans to initiate bioassay. Metabolism and excretion studies at 3.5, 35 and 350 mg/kg are to be conducted as well.

SUPPORTING DATA FOR CARCINOGENICITY

The metabolic pathways for humans and rodents are different (Engstrom et al., 1984). Major metabolites in humans, mandelic acid and phenylglyoxylic acid, are minor metabolites in rats and rabbits (Kiese and Lenk, 1974). The major animal metabolites were not detected in the urine of exposed workers (Engstrom et al., 1984).

Ethylbenzene at 0.4 mg/plate was not mutagenic for Salmonella strains TA98, TA1535, TA1537 and TA1538 with or without Aroclor 1254 induced rat liver homogenates (S9) (Nestmann et al., 1980). Ethylbenzene was shown to increase the mean number of sister chromatid exchanges in human whole blood lymphocyte culture at the highest dose examined without any metabolic activation system (Norppa and Vainio, 1983).

Dean et al. (1985) used a battery of short-term tests including bacterial mutation assays, mitotic gene conversion in Saccharomyces cerevisiae JD1 in the presence and absence of S9 and chromosomal damage in a cultured rat liver cell line. Ethylbenzene was not mutagenic in the range of concentrations tested (0.2, 2, 20, 50 and 200 ug/plate) for S. typhimurium TA98, TA100, TA1535, TA1537 and TA1538 or for Escherichia coli WP2 and WP2uvrA. Ethylbenzene also showed no response in the S. cerevisiae JD1 gene conversion assay. In contrast, ethylbenzene hydroperoxide showed positive responses with E. coli WP2 at 200 ug/plate in the presence of S9 and an equally significant response with the gene conversion system of yeast.

QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

No Data Available

QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

No Data Available

EPA DOCUMENTATION AND REVIEW

Source Document: U.S. EPA. 1980. Ambient Water Quality Criteria Document for Ethylbenzene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Water Regulations and Standards, Washington, DC. EPA 440/5-80-048. NTIS PB 81-117590.

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U.S. EPA. 1987. Drinking Water Criteria Document for Ethylbenzene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC.

The Ambient Water Quality Criteria Document and the Health Assessment Document have received Agency and external review. The Drinking Water Criteria Document has been extensively reviewed.

Agency Work Group Review: 10/07/87

Verification Date: 10/07/87

EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Arthur Chiu / OHEA -- (202)260-6764

Linda Papa / OHEA -- (513)569-7587

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CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name: Dichloromethane
CASRN: 75-09-2
Primary Synonym: Methylene Chloride

EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

WEIGHT-OF-EVIDENCE CLASSIFICATION
Classification: B2; probable human carcinogen

BASIS

Based on inadequate human data and sufficient evidence of carcinogenicity in animals; increased incidence of hepatocellular neoplasms and alveolar/bronchiolar neoplasms in male and female mice, and increased incidence of benign mammary tumors in both sexes of rats, salivary gland sarcomas in male rats and leukemia in female rats. This classification is supported by some positive genotoxicity data, although results in mammalian systems are generally negative.

HUMAN CARCINOGENICITY DATA

Inadequate. Neither of two studies of chemical factory workers exposed to dichloromethane showed an excess of cancers (Ott et al., 1983; Friedlander et al., 1978; Hearne and Friedlander, 1981). The Ott et al. (1983) study was designed to examine cardiovascular effects, and consequently the study period was too short to allow for latency of site-specific cancers. In the Friedlander et al. (1978) study, exposures were low, but the data provided some suggestion of an increased incidence of pancreatic tumors. This study was recently updated to include a larger cohort, followed through 1984, and an investigation of possible confounding factors (Hearne et al., 1986, 1987). A nonsignificant excess in pancreatic cancer deaths was observed, which was interpreted by EPA (1987a) as neither clear evidence of carcinogenicity in humans, nor evidence of noncarcinogenicity. An update of the Ott et al. (1983) study, based on longer follow-up, indicated possible elevation of liver and biliary tract cancers (TSCA section 8(e) submission no. 8eHQ-0198-0772 FLWP et seq., 1989).

ANIMAL CARCINOGENICITY DATA

Sufficient. Dichloromethane administered in the drinking water induced a significant increase in combined hepatocellular carcinoma and neoplastic nodules in female F344 rats and a nonsignificant increase in combined hepatocellular carcinoma and neoplastic nodules in male B6C3F1 mice (NCA, 1982, 1983). Two inhalation studies with dichloromethane have shown an increased incidence of benign mammary tumors in both sexes of Sprague-Dawley (Burek et al., 1984) and F344 (NTP, 1986) rats. Male Sprague-Dawley rats had increased salivary gland sarcoma (Burek et al., 1984) and female F344 rats had increased

leukemia incidence (NTP, 1986). Both sexes of B6C3F1 mice developed liver and lung tumors after dichloromethane treatment (NTP, 1986).

In a 2-year study by the National Coffee Association (1982, 1983), groups of 85 F344 rats/sex/dose received 5, 50, 125, or 250 (mg/kg)/day of dichloromethane in the drinking water. Control groups consisted of 135 rats/sex. In female rats the incidence of combined hepatocellular carcinoma and neoplastic nodules was statistically significantly increased in the 50 and 250 mg/kg dose groups when compared with matched controls (0/134, 1/85, 4/83, 1/85, and 6/85 in the five dose groups 0, 5, 50, 125, and 250 (mg/kg)/day, respectively). The incidence of hepatocellular carcinoma alone was not significantly increased (0/134, 0/85, 2/83, 0/85, 2/85). The combined incidence of hepatocellular carinoma and neoplastic nodules in controls and the 4 dose groups (472 rats: 4 with carcinoma and 8 with neoplastic nodules) was similar to that for historical controls (419 rats; 5 with carcinoma, 19 with neoplastic nodules). Male rats showed no increase in liver tumors.

In the same National Coffee Association study (1982, 1983), B6C3F1 mice received 0, 60, 125, 185, or 250 (mg/kg)/day of dichloromethane in drinking water. Treatment groups consisted of 50 female mice and 200, 100, 100, and 125 male mice (low to high dose). One hundred females and 125 males served as controls. Male mice had an increased incidence of combined neoplastic nodules and hepatocellular carcinoma (24/125, 51/200, 30/100, 31/99, 35/125). The increase was not dose-related, but the pairwise comparisons for the two mid-dose groups were reported to be statistically significant (U.S. EPA, 1985a). The hepatocellular carcinoma incidence alone for male mice (which was about 55 to 65% of the total) was not significantly elevated. Female mice did not have increased liver tumor incidence. The EPA (1985b) regarded this study as suggestive but not conclusive evidence for carcinogenicity of dichloromethane.

A gavage bioassay of dichloromethane conducted by NTP (1982) has not been published because of high mortality, much of which was attributed to gavage accidents.

Inhalation exposure of 107 to 109 Syrian hamsters/sex/dose to 0, 500, 1500, or 3500 ppm of dichloromethane for 6 hours/day, 5 days/week for 2 years did not induce neoplasia (Burek et al., 1984). Sprague-Dawley rats (129/sex/ dose) were exposed under the same conditions. Female rats administered the highest dose experienced significantly reduced survival from 18-24 months. Female rats showed a dose-related increase in the average number of benign mammary tumors per rat (1.7, 2.3, 2.6, 3.0), although the numbers of rats with tumors were not significantly increased. A similar response was observed in male rats, but to a lesser degree. In the male rats there was a statistically significant positive trend in the incidence of sarcomas of the salivary gland (1/93, 0/94, 5/91, 11/88); the incidence was significantly elevated at the high dose. There is a question as to whether these doses reached the MTD, particularly in the hamsters and the male rats. In another study (Dow Chemical Co., 1982), 90 Sprague-Dawley rats/sex were exposed by inhalation to 0, 50, 200, or 500 ppm dichloromethane for 20 months (male) or 24 months (female). No salivary tumors were observed, but there was an exposure-related increase in the total number

of benign mammary tumors in female rats, although the increase was not statistically significant in any individual exposure group.

Groups of 50 each male and female F344/N rats and B6C3F1 mice were exposed to dichloromethane by inhalation, 6 hours/day, 5 days/week for 2 years (NTP, 1986). Exposure concentrations were 0, 1000, 2000, or 4000 ppm for rats and 0, 2000, or 4000 ppm for mice. Survival of male rats was low; however, this apparently was not treatment-related. Survival was decreased in a treatment-related fashion for male and female mice and female rats. Mammary adenomas and fibroadenomas were significantly increased in male and female rats after survival adjustment, as were mononuclear cell leukemias in female rats. Among treated mice of both sexes there were significantly increased incidences of hepatocellular adenomas and carcinomas, and of alveolarbronchiolar adenomas and carcinomas, by life table tests. Adenomas and carcinomas were significantly increased alone as well as in combination. In addition, there were significant dose-related increases in the number of lung tumors per animal multiplicity in both sexes of mice.

Two inhalation assays using dogs, rabbits, guinea pigs, and rats showed no tumors, but were not conducted for the lifetime of the animals (Heppel et al., 1944; MacEwen et al., 1972). Theiss et al., (1977) injected Strain A male mice intraperitoneally with 0, 160, 400, or 800 mg/kg of dichloromethane 16 to 17 times, over 5 to 6 weeks. Survival of the animals was poor. The animals remaining 24 weeks after the first treatment were killed and examined for lung tumors; pulmonary adenomas were found.

SUPPORTING DATA FOR CARCINOGENICITY

Dichloromethane was mutagenic for Salmonella typhimurium with or without the addition of hepatic enzymes (Green, 1983) and produced mitotic recombination in yeast (Callen et al., 1980). Results in cultured mammalian cells have generally been negative, but dichloromethane has been shown to transform rat embryo cells and to enhance viral transformation of Syrian hamster embryo cells (Price et al., 1978; Hatch et al., 1983). Although chlorinated solvents have often been suspected of acting through a nongenotoxic mechanism of cell proliferation, Lefevre and Ashby (1989) found methylene chloride to be unable to induce hepatocellular division in mice.

— QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE —

Unit Risk: 4.7E-7 per (ug/cu.m)
Extrapolation Method: Linearized multistage procedure, extra risk

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	2E+2 per (ug/cu.m)
E-5 (1 in 100,000)	2E+1 per (ug/cu.m)
E-6 (1 in 1,000,000)	2E+0 per (ug/cu.m)

DOSE-RESPONSE DATA (CARCINOGENICITY, INHALATION EXPOSURE)

Tumor Type: combined adenomas and carcinomas
Test Animals: mouse/B6C3F1, female
Route: inhalation

	Dose			
Tumor Type	Administered (ppm)	Transformed Animal (mg/kg)/day	Human Equivalent (mg/kg)/day	Tumor Incidence
Liver	0	0	0	3/45
	2000	1582	356	16/46
	4000	3162	712	40/46
Lung	0	0	0	3/45
	2000	1582	356	30/46
	4000	3162	712	41/46

ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION EXPOSURE)

The unit risk of 4.7E-7 per (ug/cu.m), which incorporates information on pharmacokinetics and metabolism of dichloromethane, is approximately nine-fold lower than the previous applied dose estimate (U.S. EPA, 1987a,b). Internal dose estimates were based on the metabolism of dichloromethane by the glutathione-s-transferase pathway, as estimated by the model developed by Andersen et al. (1987). The internal dose was corrected for interspecies differences in sensitivity by using the surface area correction factor.

Calculation of a slope factor from the unit risk is inappropriate when pharmacokinetic models are used. (When dose-response relationships are figured on the basis of internal or metabolized dose, a slope factor in terms of per (mg/kg)/day represents a back calculation using different absorption assumptions than the pharmacokinetic models. This introduces possible contradictions.)

The unit risk should not be used if the air concentration exceeds 2E+4 ug/cu.m, since above this concentration the unit risk may differ from that stated. Since the unit risk is based on a pharmacokinetic model, the risk may change with alterations in exposure patterns. Thus, the unit risk presented here may not be applicable to acute, high exposures.

DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION EXPOSURE)

Adequate numbers of animals were observed and tumor incidences were significantly increased in a dose-dependent fashion. Analysis excluding animals that died before observation of the first tumors produced similar risk estimates, as did time-to-tumor analysis. The use of animal and human metabolism and pharmacokinetic data reduces some of the uncertainty typically associated with dose-risk extrapolation. A great deal of uncertainty still

exists, however, in the estimates of internal dose generated by the model of Andersen et al. (1987). Important uncertainties remain regarding the pharmacokinetics, pharmacodynamics, and mechanisms of carcinogenicity for dichloromethane.

EPA DOCUMENTATION AND REVIEW

Source Document: U.S. EPA. 1985a. Health Assessment Document for Dichloromethane (Methylene Chloride). Final Report. Office of Health and Environmental Assessment, Washington, D.C. EPA/600/8-82/004F.

U.S. EPA. 1985b. Addendum to the Health Assessment Document for Dichloromethane (methylene chloride). Updated carcinogenicity assessment. Prepared by the Carcinogen Assessment Group, OHEA, Washington, DC. EPA/600/8-82/004FF.

U.S. EPA. 1987a. Update to the Health Assessment Document and Addendum for Dichloromethane (Methylene Chloride): Pharmacokinetics, Mechanism of Action and Epidemiology. Review Draft. Office of Health and Environmental Assessment, Washington, DC. EPA/600/8-87/030A.

U.S. EPA. 1987b. Technical Analysis of New Methods and Data Regarding Dichloromethane Hazard Assessments. Review Draft. Office of Health and Environmental Assessment, Washington, DC. EPA/600/8-87/029A.

The Addendum to the Health Assessment Document, the Update to the Health Assessment Document and Addendum, and the Technical Analysis of New Methods and Data for dichloromethane have received Agency and external review, including a review by the Science Advisory Board (SAB). Although the last two documents are not yet finalized and the SAB comments are not yet incorporated, these do not alter this document's analyses or conclusions.

Agency Work Group Review: 11/12/86, 12/04/86, 04/06/89

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EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Lorenz Rhomberg / OHEA -- (202)260-5723

Dharm Singh / OHEA -- (202)260-5958

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U.S. EPA. 1985b. Addendum to the Health Assessment Document for Dichloromethane (methylene chloride). Updated carcinogenicity assessment. Prepared by the Carcinogen Assessment Group, OHEA, Washington, DC. EPA/600/8-82/004FF.

U.S. EPA. 1987a. Update to the Health Assessment Document and Addendum for Dichloromethane (Methylene Chloride): Pharmacokinetics, Mechanism of Action and Epidemiology. Review Draft. Office of Health and Environmental Assessment, Washington, DC. EPA/600/8-87/030A.

U.S. EPA. 1987b. Technical Analysis of New Methods and Data Regarding Dichloromethane Hazard Assessments. Review Draft. Office of Health and Environmental Assessment, Washington, DC. EPA/600/8- 87/029A.

CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name: Benzene
CASRN: 71-43-2

EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification: A; human carcinogen

BASIS

Several studies of increased incidence of nonlymphocytic leukemia from occupational exposure, increased incidence of neoplasia in rats and mice exposed by inhalation and gavage, and some supporting data form the basis for this classification.

HUMAN CARCINOGENICITY DATA

Aksoy et al. (1974) reported effects of benzene exposure among 28,500 Turkish workers employed in the shoe industry. Mean duration of employment was 9.7 years (1-15 year range) and mean age was 34.2 years. Peak exposure was reported to be 210-650 ppm. Twenty-six cases of leukemia and a total of 34 leukemias or preleukemias were observed, corresponding to an incidence of 13/100,000 (by comparison to 6/100,000 for the general population). A follow-up paper (Aksoy, 1980) reported eight additional cases of leukemia as well as evidence suggestive of increases in other malignancies.

In a retrospective cohort mortality study Infante et al. (1977a,b) examined leukemogenic effects of benzene exposure in 748 white males exposed while employed in the manufacturing of rubber products. Exposure occurred from 1940-1949, and vital statistics were obtained through 1975. A statistically significant increase (p less than or equal to 0.002) of leukemias was found by comparison to the general U.S. population. There was no evidence of solvent exposure other than benzene. Air concentrations were generally found to be below the recommended limits in effect during the study period.

In a subsequent retrospective cohort mortality study Rinsky et al. (1981) observed seven deaths from leukemia among 748 workers exposed to benzene and followed for at least 24 years (17,020 person-years). This increased incidence was statistically significant; standard mortality ratio (SMR) was 560. For the five leukemia deaths that occurred among workers with more than 5 years exposure, the SMR was 2100. Exposures (which ranged from 10-100 ppm 8-hour TWA) were described as less than the recommended standards for the time period of 1941-1969.

In an updated version of the Rinsky et al. (1981) study, the authors followed the same cohort to 12/31/81 (Rinsky et al., 1987). An in his earlier study, cumulative exposure was derived from historic air-sampling data or interpolated

estimates based on exisitng data. Standardized mortality rates ranged from 109 at cumulative benzene exposures under 40 ppm-years and increased montonically to 6637 (6 cases) at 400 ppm-years or more. The authors found significantly elevated risks of leukemia at cumulative exposures less than the equivalent current standard for occupational exposure which is 10 ppm over a 40-year working lifetime.

Ott et al. (1978) observed three deaths from leukemia among 594 workers followed for at least 23 years in a retrospective cohort mortality study, but the increase was not statistically significant. Exposures ranged from <2 to >25 ppm 8-hour TWA.

Wong et al. (1983) reported on the mortality of male chemical workers who had been exposed to benzene for at least 6 months during the years 1946-1975. The study population of 4062 persons was drawn from seven chemical plants, and jobs were categorized as to peak exposure. Those with at least 3 days/week exposure (3036 subjects) were further categorizeed on the basis of an 8-hour TWA. The control subjects held jobs at the same plants for at least 6 months but were never subject to benzene exposure. Dose-dependent increases were seen in leukemia and lymphatic and hematopoietic cancer. The incidence of leukemia was responsible for the majority of the increase. It was noted that the significance of the increase is due largely to a less than expected incidence of neoplasia in the unexposed subjects.

Numerous other epidemiologic and case studies have reported an increased incidence or a causal relationship between leukemia and exposure to benzene (IARC, 1982).

ANIMAL CARCINOGENICITY DATA

Both gavage and inhalation exposure of rodents to benzene have resulted in development of neoplasia. Maltoni and Scarnato (1979) and Maltoni et al. (1983) administered benzene by gavage at dose levels of 0, 50, 250, and 500 mg/kg bw to 30-40 Sprague-Dawley rats/sex for life. Dose-related increased incidences of mammary tumors were seen in females and of Zymbal gland carcinomas, oral cavity carcinomas and leukemias/lymphomas in both sexes.

In an NTP (1986) study, benzene was administered by gavage doses of 0, 50, 100, or 200 mg/kg bw to 50 F344/N rats/sex or 0, 25, 50, or 100 mg/kg bw to 50 B6C3F1 mice/sex. Treatment was 5 times/week for 103 weeks. Significantly increased incidences ($p<0.05$) of various neoplasic growths were seen in both sexes of both species. Both male and female rats and mice had increased incidence of carcinomas of the Zymbal gland. Male and female rats had oral cavity tumors, and males showed increased incidences of skin tumors. Mice of both sexes had increased incidence of lymphomas and lung tumors. Males were observed to have harderian and preputial gland tumors and females had tumors of mammary gland and ovary. In general, the increased incidence was dose-related.

Slightly increased incidences of hematopoietic neoplasms were reported for male C57B1 mice exposed by inhalation to 300 ppm benzene 6 hours/day, 5 days/ week for 488 days. There was no increase in tumor incidence in male AKR or CD-1 mice similarly exposed to 100 ppm or 100 or 300 ppm benzene, respectively.

Likewise male Sprague-Dawley rats exposed by inhalation to 300 ppm benzene were not observed to have increased incidence of neoplasia (Snyder et al., 1981). Maltoni et al. (1983) treated male and female Sprague-Dawley rats in the following manner. Starting at 13 weeks of age rats were exposed to 200 ppm benzene 4 hours/day, 5 days/week for 7 weeks; 200 ppm 7 hours/day, 5 days/week for 12 weeks; 300 ppm 7 hours/day, 5 days/week for 85 weeks. An 8-hour/day TWA for 5 days/week was calculated to be 241 ppm. A statistically significant increase was noted in hepatomas and carcinomas of the Zymbal gland.

SUPPORTING DATA FOR CARCINOGENICITY

Numerous investigators have found significant increases in chromosomal aberrations of bone marrow cells and peripheral lymphocytes from workers with exposure to benzene (IARC, 1982). Benzene also induced chromosomal aberrations in bone marrow cells from rabbits (Kissling and Speck, 1973), mice (Meyne and Legator, 1980) and rats (Anderson and Richardson, 1979). Several investigators have reported positive results for benzene in mouse micronucleus assays (Meyne and Legator, 1980). Benzene was not mutagenic in several bacterial and yeast systems, in the sex-linked recessive lethal mutation assay with Drosophila melanogaster or in mouse lymphoma cell forward mutation assay.

— QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE —

Unit Risk: 8.3E-6 per (ug/cu.m)
Extrapolation Method: One-hit (pooled data)

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	1E+1 per (ug/cu.m)
E-5 (1 in 100,000)	1E+0 per (ug/cu.m)
E-6 (1 in 1,000,000)	1E-1 per (ug/cu.m)

——— DOSE-RESPONSE DATA (CARCINOGENICITY, INHALATION EXPOSURE) ———

Tumor Type: leukemia
Test Animals: human
Route: inhalation, occupational exposure

No additional data.

——— ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION EXPOSURE) ———

The unit risk estimate is the geometric mean of four ML point estimates using pooled data from the Rinsky et al. (1981) and Ott et al. (1978) studies, which was then adjusted for the results of the Wong et al. (1983) study. The Rinsky data used were from an updated tape which reports one more case of leukemia than was published in 1981. Equal weight was given to cumulative dose and weighted cumulative dose exposure categories as well as to relative and absolute risk model forms. The results of the Wong et al. (1983) study were incorporated by assuming that the ratio of the Rinsky-Ott-Wong studies to the Rinsky-Ott studies for the relative risk cumulative dose model was the same as

for other model-exposure category combinations and multiplying this ratio by the Rinsky-Ott geometric mean. The age-specific U.S. death rates for 1978 (the most current year available) were used for background leukemia and total death rates. It should be noted that a recently published paper (Rinsky et al., 1987) reported yet another case of leukemia from the study population.

The unit risk should not be used if the air concentration exceeds 100 ug/cu.m, since above this concentration the unit risk may not be appropriate.

—— DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION EXPOSURE) ——

The pooled cohorts were sufficiently large and were followed for an ade quate time period. The increases in leukemias were statistically significant and dose-related in one of the studies. Wong et al. (1983) disagrees that exposures reported in Rinsky et al. (1981) were within the recommended standards. For the five leukemia deaths in persons with 5 or more years exposure, the author notes that mean exposure levels (range 15-70 ppm) exceeded the recommended standard (25 ppm) in 75% of the work locations sampled. The risk estimate above based on reconsideration of the Rinsky et al. (1981) and Ott et al. (1978) studies is very similar to that of 2.4E-2/ppm (cited in U.S. EPA, 1980) based on Infante et al. (1977a,b), Ott et al. (1978) and Aksoy et al. (1974). It was felt by the authors of U.S. EPA (1985) that the exposure assessment provided by Aksoy was too imprecise to warrant inclusion in the current risk estimate. A total of 21 unit risk estimates were prepared using 6 models and various combinations of the epidemiologic data. These range over slightly more than one order of magnitude. A geometric mean of these estimates is 2.7E-2/ppm. Regression models give an estimate similar to the geometric mean.

—————————— EPA DOCUMENTATION AND REVIEW ——————————

Source Document: U.S. EPA. 1980. Ambient Water Quality Criteria Document for Benzene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office (Cincinnati, OH) and Carcinogen Assessment Group (Washington, DC), and the Environmental Research Labs (Corvalis, OR; Duluth, MN; Gulf Breeze, FL) for the Office of Water Regulations and Standards, Washington, DC. EPA 440/5-80-018.

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The 1985 Interim Evaluation was reviewed by the Carcinogen Assessment Group.

The 1987 memorandum is an internal document.

Agency Work Group Review: 03/05/87, 10/09/87

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EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

David Bayliss / OHEA -- (202)260-5726

Robert McGaughy / OHEA -- (202)260-5898

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CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name: Toluene
CASRN: 108-88-3

EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification: D; not classifiable as to human carcinogenicity

BASIS

No human data and inadequate animal data. Toluene did not produce positive results in the majority of genotoxic assays.

HUMAN CARCINOGENICITY DATA

None.

ANIMAL CARCINOGENICITY DATA

A chronic (106-week) bioassay of toluene in F344 rats of both sexes reported no carcinogenic responses (CIIT, 1980). A total of 960 rats were exposed by inhalation for 6 hours/day, 5 days/week to toluene at 0, 30, 100, or 300 ppm. Groups of 20/sex/dose were sacrificed at 18 months. Gross and microscopic examination of tissues and organs identified no increase in neoplastic tissue or tumor masses among treated rats when compared with controls. The study is considered inadequate because the highest dose administered was well below the MTD for toluene and because of the high incidence of lesions and pathological changes in the control animals.

Several studies have examined the carcinogenicity of toluene following repeated dermal applications. Toluene (dose not reported) applied to shaved interscapular skin of 54 male mice (strains A/He, C3HeB, SWR) throughout their lifetime (3 times weekly) produced no carcinogenic response (Poel, 1963). One drop of toluene (about 6 mL) applied to the dorsal skin of 20 random-bred albino mice twice weekly for 50 weeks caused no skin papillomas or carcinomas after a 1-year latency period was allowed (Coombs et al., 1973). No increase in the incidence of skin or systemic tumors was demonstrated in male or female mice of three strains (CF, C3H, or CBaH) when toluene was applied to the back of 25 mice of each sex of each strain at 0.05-0.1 mL/mouse, twice weekly for 56 weeks (Doak et al., 1976). One skin papilloma and a single skin carcinoma were reported among a group of 30 mice treated dermally with one drop of 0.2% (w/v) solution toluene twice weekly, administered from droppers delivering 16-20 uL per drop for 72 weeks (Lijinsky and Garcia, 1972). It is not reported whether evaporation of toluene from the skin was prevented during these studies.

SUPPORTING DATA FOR CARCINOGENICITY

Toluene was found to be nonmutagenic in reverse mutation assays with S. typhimurium (Mortelmans and Riccio, 1980; Nestmann et al., 1980; Bos et al., 1981; Litton Bionetics, Inc., 1981; Snow et al., 1981) and E. coli (Mortelmans and Riccio, 1980), with and without metabolic activation. Toluene did not induce mitotic gene conversion (Litton Bionetics, Inc., 1981; Mortelmans and Riccio, 1980) or mitotic crossing over (Mortelmans and Riccio, 1980) in S. cerevisiae. Although Litton Bionetics, Inc. (1981) reported that toluene did not cause increased chromosomal aberrations in bone marrow cells, several Russian studies (Dobrokhotov, 1972; Lyapkalo, 1973) report toluene as effective in causing chromosal damage in bone marrow cells of rats. There was no evidence of chromosomal aberrations in blood lymphocytes of workers exposed to toluene only (Maki-Paakkanen et al., 1980; Forni et al., 1971), although a slight increase was noted in workers exposed to toluene and benzene (Forni et al., 1971; Funes-Craviota et al., 1977). This finding is supported by studies of cultured human lymphocytes exposed to toluene in vitro; no elevation of chromosomal aberrations or sister chromatid exchanges was observed (Gerner-Smidt and Friedrich, 1978).

— QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE —

No Data Available

EPA DOCUMENTATION AND REVIEW

Source Document: U.S. EPA. 1987. Drinking Water Criteria Document for Toluene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC.

The values in the 1987 Drinking Water Criteria Document for Toluene have received peer and administrative review.

Agency Work Group Review: 09/15/87

Verification Date: 09/15/87

EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Dharm Singh / OHEA -- (202)260-5958

Robert McGaughy / OHEA -- (202)260-5898

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CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name: Arsenic, inorganic
CASRN: 7440-38-2

Note: Important*** Page down to see oral quantitative estimate message.

EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification: A; human carcinogen

BASIS

based on observation of increased lung cancer mortality in populations exposed primarily through inhalation and on increased skin cancer incidence in several populations consuming drinking water with high arsenic concentrations.

HUMAN CARCINOGENICITY DATA

Studies of smelter worker populations (Tacoma, WA; Magma, UT; Anaconda, MT; Ronnskar, Sweden; Saganoseki-Machii, Japan) have all found an association between occupational arsenic exposure and lung cancer mortality (Enterline and Marsh, 1982; Lee-Feldstein, 1983; Axelson et al., 1978; Tokudome and Kuratsune, 1976; Rencher et al., 1977). Both proportionate mortality and cohort studies of pesticide manufacturing workers have shown an excess of lung cancer deaths among exposed persons (Ott et al., 1974; Mabuchi et al., 1979). One study of a population residing near a pesticide manufacturing plant revealed that these residents were also at an excess risk of lung cancer (Matanoski et al., 1981). Case reports of arsenical pesticide applicators have also demonstrated an association between arsenic exposure and lung cancer (Roth, 1958).

A cross-sectional study of 40,000 Taiwanese exposed to arsenic in drinking water found significant excess skin cancer prevalence by comparison to 7500 residents of Taiwan and Matsu who consumed relatively arsenic-free water (Tseng et al., 1968). This study design limited its usefulness in risk estimation. Arsenic-induced skin cancer has also been attributed to water supplies in Chile, Argentina and Mexico (Borgono and Greiber, 1972; Bergoglio, 1964; Cebrian et al., 1983). No excess skin cancer incidence has been observed in U.S. residents consuming relatively high levels of arsenic in drinking water (Morton et al., 1976; Southwick et al., 1981). The results of these U.S. studies, however, are not necessarily inconsistent with the existing findings from the foreign populations. The statistical powers of the U.S. studies are considered to be inadequate because of the small sample size.

A follow-up study (Tseng, 1977) of the population living in the same area of Taiwan, where arsenic contamination of the water supply was endemic, found significantly elevated standard mortality ratios for cancer of the bladder, lung, liver, kidney, skin and colon. This study of bladder, liver and lung

cancer cases in the endemic area found a significant association with arsenic exposure that was dose-related. The association of arsenic ingestion and cancer of various internal organs has also been cited in a number of case reports (Chen et al., 1985, 1986). Persons treated with arsenic-containing medicinals have also been shown to be at a risk of skin cancer (Sommers and McManus, 1953).

ANIMAL CARCINOGENICITY DATA

None. There has not been consistent demonstration of arsenic carcinogenicity in test animals for various chemical forms administered by different routes to several species (IARC, 1980). There are some data to indicate that arsenic may produce animal tumors if retention time in the lung can be increased (Pershagen et al., 1982, 1984).

SUPPORTING DATA FOR CARCINOGENICITY

Sodium arsenate has been shown to transform Syrian hamster embryo cells (Dipaolo and Casto, 1979) and to produce sister-chromatid-exchange in DON cells, CHO cells and human peripheral lymphocytes exposed in vitro (Wan et al., 1982; Ohno et al., 1982; Larramendy et al., 1981; Andersen, 1983; Crossen, 1983). While arsenic compounds have not been shown to mutate bacterial strains, it produces preferential killing of repair deficient strains (Rossman, 1981).

— QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE —

Unit Risk: 4.3E-3 per (ug/cu.m)
Extrapolation Method: absolute-risk linear model

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	2E-2 per (ug/cu.m)
E-5 (1 in 100,000)	2E-3 per (ug/cu.m)
E-6 (1 in 1,000,000)	2E-4 per (ug/cu.m)

——— DOSE-RESPONSE DATA (CARCINOGENICITY, INHALATION EXPOSURE) ———

Tumor Type: lung cancer
Test Animals: human, male
Route: inhalation, occupational exposure

Ambient Unit Risk Estimates

Exposure Source	Study	Unit Risk	Geometric Mean Unit Risk	Final Estimates Unit Risk
Anaconda smelter	Brown and Chu, 1983a,b,c	1.25 E-3		
	Lee-Feldstein, 1983	2.80 E-3	2.56 E-3	
	Higgins, 1982; Higgins et al., 1982; Welch et al., 1982	4.90 E-3		4.29 E-3
ASARCO smelter	Enterline and Marsh, 1982	6.81 E-3 7.60 E-3	7.19 E-3	

——— ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION EXPOSURE) ———

A geometric mean was obtained for data sets obtained within distinct exposed populations (U.S. EPA, 1984). The final estimate is the geometric mean of those two values. It was assumed that the increase in age-specific mortality rate of lung cancer was a function only of cumulative exposures.

The unit risk should not be used if the air concentration exceeds 2 ug/cu.m, since above this concentration the unit risk may not be appropriate.

—— DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION EXPOSURE) ——

Overall a large study population was observed. Exposure assessments included air measurements for the Anaconda smelter and both air measurements and urinary arsenic for the ASARCO smelter. Observed lung cancer incidence was significantly increased over expected values. The range of the estimates derived from data from two different exposure areas was within a factor of 6.

——————— EPA DOCUMENTATION AND REVIEW ———————

Source Document: U.S. EPA. 1984. Health Assessment Document for Inorganic Arsenic. Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA 600/8-83-021F.

The 1984 Health Assessment Document for Inorganic Arsenic received Agency and external review including a review by SAB.

Agency Work Group Review: 01/13/88

Verification Date: 01/13/88

EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Herman Gibb / OHEA -- (202)260-5898

Chao Chen / OHEA -- (202)260-5719

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CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name: Cadmium
CASRN: 7440-43-9

EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification: B1; probable human carcinogen

BASIS

Limited evidence from occupational epidemiologic studies of cadmium is consistent across investigators and study populations. There is sufficient evidence of carcinogenicity in rats and mice by inhalation and intramuscular and subcutaneous injection. Seven studies in rats and mice wherein cadmium salts (acetate, sulfate, chloride) were administered orally have shown no evidence of carcinogenic response.

HUMAN CARCINOGENICITY DATA

Limited. A 2-fold excess risk of lung cancer was observed in cadmium smelter workers. The cohort consisted of 602 white males who had been employed in production work a minimum of 6 months during the years 1940-1969. The population was followed to the end of 1978. Urine cadmium data available for 261 workers employed after 1960 suggested a highly exposed population. The authors were able to ascertain that the increased lung cancer risk was probably not due to the presence of arsenic or to smoking (Thun et al., 1985). An evaluation by the Carcinogen Assessment Group of these possible confounding factors has indicated that the assumptions and methods used in accounting for them appear to be valid. As the SMRs observed were low and there is a lack of clear cut evidence of a causal relationship of the cadmium exposure only, this study is considered to supply limited evidence of human carcinogenicity.

An excess lung cancer risk was also observed in three other studies which were, however, compromised by the presence of other carcinogens (arsenic, smoking) in the exposure or by a small population (Varner, 1983; Sorahan and Waterhouse, 1983; Armstrong and Kazantzis, 1983).

Four studies of workers exposed to cadmium dust or fumes provided evidence of a statistically significant positive association with prostate cancer (Kipling and Waterhouse, 1967; Lemen et al., 1976; Holden, 1980; Sorahan and Waterhouse, 1983), but the total number of cases was small in each study. The Thun et al. (1985) study is an update of an earlier study (Lemen et al., 1976) and does not show excess prostate cancer risk in these workers. Studies of human ingestion of cadmium are inadequate to assess carcinogenicity.

ANIMAL CARCINOGENICITY DATA

Exposure of Wistar rats by inhalation to cadmium as cadmium chloride at concentrations of 12.5, 25 and 50 ug/cu.m for 18 months, with an additional 13-month observation period, resulted in significant increases in lung tumors (Takenaka et al., 1983). Intratracheal instillation of cadmium oxide did not produce lung tumors in Fischer 344 rats but rather mammary tumors in males and tumors at multiple sites in males (Sanders and Mahaffey, 1984). Injection site tumors and distant site tumors (for example, testicular) have been reported by a number of authors as a consequence of intramuscular or subcutaneous administration of cadmium metal and chloride, sulfate, oxide and sulfide compounds of cadmium to rats and mice (U.S. EPA, 1985). Seven studies in rats and mice where cadmium salts (acetate, sulfate, chloride) were administered orally have shown no evidence of a carcinogenic response.

SUPPORTING DATA FOR CARCINOGENICITY

Results of mutagenicity tests in bacteria and yeast have been inconclusive. Positive responses have been obtained in mutation assays in Chinese hamster cells (Dom and V79 lines) and in mouse lymphoma cells (Casto, 1976; Ochi and Ohsawa, 1983; Oberly et al., 1982).

Conflicting results have been obtained in assays of chromosomal aberrations in human lymphocytes treated in vitro or obtained from exposed workers. Cadmium treatment in vivo or in vitro appears to interfere with spindle formation and to result in aneuploidy in germ cells of mice and hamsters (Shimada et al., 1976; Watanabe et al., 1979; Gilliavod and Leonard, 1975).

——— DOSE-RESPONSE DATA (CARCINOGENICITY, INHALATION EXPOSURE) ———

Tumor Type: lung, trachea, bronchus cancer deaths
Test Animals: human/white male
Route: inhalation, occupational exposure

Cumulative Exposure (mg/day/cu.m)	Median Observation	24 hour/ ug/cu.m Equivalent	No. of Expected Lung, Trachea and Bronchus Cancers Assuming No Cadmium Effect	Observed No. of Deaths (lung, trachea, bronchus cancers)
less than or equal to 584	280	168	3.77	2
585-2920	1210	727	4.61	7
greater than or equal to 2921	4200	2522	2.50	7

The 24-hour equivalent = median observation x 1E+3 x 8/24 x 1/365 x 240/365.

ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION EXPOSURE)

The unit risk should not be used if the air concentration exceeds 6 ug/cu.m, since above this concentration the unit risk may not be appropriate.

DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION EXPOSURE)

The data were derived from a relatively large cohort. Effects of arsenic and smoking were accounted for in the quantitative analysis for cadmium effects.

An inhalation unit risk for cadmium based on the Takenaka et al. (1983) analysis is 9.2E-2 per (ug/cu.m). While this estimate is higher than that derived from human data [1.8E-3 per (ug/cu.m)] and thus more conservative, it was felt that the use of available human data was more reliable because of species variations in response and the type of exposure (cadmium salt vs. cadmium fume and cadmium oxide).

EPA DOCUMENTATION AND REVIEW

Source Document: U.S. EPA. 1985. Updated Mutagenicity and Carcinogenicity Assessment of Cadmium: Addendum to the Health Assessment Document for Cadmium (May 1981, EPA 600/B-B1-023). EPA 600/B-83-025F.

The Addendum to the Cadmium Health Assessment has received both Agency and external review.

Agency Work Group Review: 11/12/86
Verification Date: 11/12/86

EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

William Pepelko / OHEA -- (202)260-5904

David Bayliss / OHEA -- (202)260-5726

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CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name: Chromium(VI)
CASRN: 18540-29-9

EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification: A; human carcinogen

BASIS

Results of occupational epidemiologic studies of chromium-exposed workers are consistent across investigators and study populations. Dose-response relationships have been established for chromium exposure and lung cancer. Chromium-exposed workers are exposed to both chromium III and chromium VI compounds. Because only chromium VI has been found to be carcinogenic in animal studies, however, it was concluded that only chromium VI should be classified as a human carcinogen.

HUMAN CARCINOGENICITY DATA

Sufficient. Epidemiologic studies of chromate production facilities in the United States (Machle and Gregorius, 1948; Brinton et al., 1952; Mancuso and Hueper, 1951, Mancuso, 1975; Baetjer, 1950; Taylor, 1966; Enterline, 1974; Hayes et al., 1979; Hill and Ferguson, 1979), Great Britain (Bidstrup, 1951; Bidstrup and Case, 1956; Alderson et al., 1981), Japan (Watanabe and Fukuchi, 1975; Ohsaki et al., 1978; Sano and Mitohara, 1978; Satoh et al., 1981) and West Germany (Korallus et al., 1982; Bittersohl, 1971) have established an association between chromium (Cr) exposure and lung cancer. Most of these studies did not attempt to determine whether Cr III or Cr VI compounds were the etiologic agents.

Three studies of the chrome pigment industry, one in Norway (Langard and Norseth, 1975), one in England (Davies, 1978, 1979), and the third in the Netherlands and Germany (Frentzel-Beyme, 1983) also found an association between occupational chromium exposure (predominantly to Cr VI) and lung cancer.

Results of two studies of the chromium plating industry (Royle, 1975; Silverstein et al., 1981) were inconclusive, while the findings of a Japanese study of chrome platers were negative (Okubo and Tsuchiya, 1979). The results of studies of ferrochromium workers (Pokrovskaya and Shabynina, 1973; Langard et al., 1980; Axelsson et al., 1980) were inconclusive as to lung cancer risk.

ANIMAL CARCINOGENICITY DATA

Sufficient. Hexavalent chromium compounds were carcinogenic in animal assays producing the following tumor types: intramuscular injection site tumors in

Fischer 344 and Bethesda Black rats and in C57BL mice (Furst et al., 1976; Maltoni, 1974, 1976; Payne, 1960; Heuper and Payne, 1959); intraplural implant site tumors for various chromium VI compounds in Sprague-Dawley and Bethesda Black rats (Payne, 1960; Heuper 1961; Heuper and Payne, 1962); intrabronchial implantation site tumors for various Cr VI compounds in Wistar rats (Levy and Martin, 1983; Laskin et al., 1970; Levy as quoted in NIOSH, 1975); and subcutaneous injection site sarcomas in Sprague-Dawley rats (Maltoni, 1974, 1976).

SUPPORTING DATA FOR CARCINOGENICITY

A large number of chromium compounds have been assayed in in vitro genetic toxicology assays. In general, hexavalent chromium is mutagenic in bacterial assays whereas trivalent chromium is not (Lofroth, 1978; Petrellie and Flora, 1977, 1978). Likewise Cr VI but not Cr III was mutagenic in yeasts (Bonatti et al., 1976) and in V79 cells (Newbold et al., 1979). Chromium III and VI compounds decrease the fidelity of DNA synthesis in vitro (Loeb et al., 1977), while Cr VI compounds inhibit replicative DNA synthesis in mammalian cells (Levis et al., 1978) and produce unscheduled DNA synthesis, presumably repair synthesis, as a consequence of DNA damage (Raffetto, 1977). Chromate has been shown to transform both primary cells and cell lines (Fradkin et al., 1975; Tsuda and Kato, 1977; Casto et al., 1979). Chromosomal effects produced by treatment with chromium compounds have been reported by a number of authors; for example, both Cr VI and Cr III salts were clastogenic for cultured human leukocytes (Nakamuro et al., 1978).

There are no long-term studies of ingested Cr VI. There appears to be significant in vivo conversion of Cr VI to Cr III and III to VI; Cr III is an essential trace element.

— QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE —

Unit Risk: 1.2E-2 per (ug/cu.m)
Extrapolation Method: Multistage, extra risk

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	8E-3 per (ug/cu.m)
E-5 (1 in 100,000)	8E-4 per (ug/cu.m)
E-6 (1 in 1,000,000)	8E-5 per (ug/cu.m)

——— DOSE-RESPONSE DATA (CARCINOGENICITY, INHALATION EXPOSURE) ———

Tumor Type:	lung cancer
Test Animals:	human
Route:	inhalation, occupational exposure

Exposure Level Subject Age (years)	Midrange (ug/cu.m)	Deaths from Lung Cancer	Person Years
50	5.66	3	1345
	25.27	6	931
	46.83	6	299
60	4.68	4	1063
	20.79	5	712
	39.08	5	211
70	4.41	2	401
	21.29	4	345

——— ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION EXPOSURE) ———

The cancer mortality in Mancuso (1975) was assumed to be due to Cr VI, which was further assumed to be no less than one-seventh of total chromium. It was also assumed that the smoking habits of chromate workers were similar to those of the U.S. white male population. The unit risks of Langard et al. (1980), Axelsson et al. (1980), and Pokrovskaya and Shabynina (1973) are 1.3E-1, 3.5E-2 and 9.2E-2 per (ug/cu.m), respectively.

Hexavalent chromium compounds have not produced lung tumors in animals by inhalation. Trivalent chromium compounds have not been reported as carcinogenic by any route of administration.

The unit risk should not be used if the air concentration exceeds 8E-1 ug/cu.m, since above this concentration the unit risk may not be appropriate.

—— DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION EXPOSURE) ——

Results of studies of chromium exposure are consistent across investigators and countries. A dose-relationship for lung tumors has been established. The assumption that the ratio of Cr III to Cr VI is 6:1 may lead to a 7-fold underestimation of risk. The use of 1949 hygiene data, which may underestimate worker exposure, may result in an overestimation of risk. Further overestimation of risk may be due to the implicit assumption that the smoking habits of chromate workers were similar to those of the general white male population, since it is generally accepted that the proportion of smokers is higher for industrial workers than for the general population.

———————— EPA DOCUMENTATION AND REVIEW ————————

Source Document: U.S. EPA. 1984. Health Assessment Document for Chromium. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. EPA 600/8-83-014F.

The quantification of cancer risk in the 1984 Health Assessment Document has received peer review in public sessions of the Environmental Health Committee of the U.S. EPA's Science Advisory Board.

Agency Work Group Review: 06/26/86

Verification Date: 06/26/86

EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Herman Gibb / OHEA -- (202)260-5898

Chao Chen / OHEA -- (202)260-5719

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CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name: Copper
CASRN: 7440-50-8

EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification: D; not classifiable as to human carcinogenicity

BASIS

There are no human data, inadequate animal data from assays of copper compounds, and equivocal mutagenicity data.

HUMAN CARCINOGENICITY DATA

None.

ANIMAL CARCINOGENICITY DATA

Inadequate. Bionetics Research Labs (1968) studied the carcinogenicity of a copper-containing compound, copper hydroxyquinoline, in two strains of mice (B6C3F1 and B6AKF1). Groups of 18 male and 18 female 7-day-old mice were administered 1000 mg copper hydroxyquinoline/kg bw (180.6 mg Cu/kg) suspended in 0.5% gelatin daily until they were 28 days old, after which they were administered 2800 ppm (505.6 ppm Cu) in the feed for 50 additional weeks. No statistically significant increases in tumor incidence were observed in the treated 78-week-old animals.

In the same study, Bionetics Research Labs (1968) administered a single subcutaneous injection of gelatin (control) or 1000 mg of copper hydroxyquinoline/kg bw (180.6 mg Cu/kg) suspended in 0.5% gelatin to groups of 28-day-old mice of both strains. After 50 days of observation, the male B6C3F1 had an increased incidence of reticulum cell sarcomas compared with controls. No tumors were observed in the treated male B6AKF1 mice, and a low incidence of reticulum cell sarcomas was observed in the treated female mice of both strains.

Gilman (1962) administered intramuscular injections containing 20 mg of cupric oxide (16 mg Cu), cupric sulfide (13.3 mg Cu), and cuprous sulfide (16 mg Cu) into the left and right thighs of 2- to 3-month-old Wistar rats. After 20 months of observations, no injection-site tumors were observed in any animals, but other tumors were observed at very low incidence in the animals receiving cupric sulfide (2/30) and cuprous sulfide (1/30). As the relevance of the organic copper compound to the observation of sarcoma induction is uncertain and the incidence of tumors in rats treated i.m. with inorganic copper was very low, data are considered inadequate for classification.

SUPPORTING DATA FOR CARCINOGENICITY

Moriya et al. (1983) reported no increase in mutations in E. coli and S. typhimurium strains TA98, TA1535, TA1537 and TA1538 incubated with up to 5 mg copper quinolinolate/plate and in S. typhimurium TA98 and TA100 incubated with up to 5 mg copper sulfate/plate. Demerec et al. (1951) reported dose-related mutagenic effects in E. coli with 2 to 10 ppm copper sulfate in a reverse mutation assay. Negative results were obtained with copper sulfate or copper chloride in assays using S. cerevisiae (Singh, 1983) and Bacillus subtilis (Nishioka, 1975, Matsui, 1980, Kanematsu et al., 1980). Errors in DNA synthesis from poly(c)templates have been induced in viruses incubated with copper chloride or copper acetate (Sirover and Loeb, 1976). Chromosomal aberrations were induced in isolated rat hepatocytes when incubated with copper sulfate (Sina et al., 1983). Casto et al. (1979) showed enhanced cell transformation in Syrian hamster embryo cells infected with simian adenovirus with the addition of cuprous sulfide and copper sulfate. High concentrations of copper compounds have been reported to induce mitosis in rat ascites cells and recessive lethals in Drosophila melanogaster. Law (1938) reported increases in the percent lethals observed in Drosophila larvae and eggs when exposed to copper by microinjection (0.1% copper sulfate) or immersion (concentrated aqueous copper sulfate), respectively.

— QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE —

No Data Available

EPA DOCUMENTATION AND REVIEW

Source Document: U.S. EPA. 1987. Drinking Water Criteria Document for Copper. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC.

The values in the 1987 Drinking Water Criteria Document for Copper have received peer and administrative review.

Agency Work Group Review: 09/15/87

Verification Date: 09/15/87

EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

David J. Reisman / OHEA -- (513)569-7588

W. Bruce Peirano / OHEA -- (513)569-7540

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U.S. EPA. 1987. Drinking Water Criteria Document for Copper. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC.

CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name: Lead and compounds (inorganic)
CASRN: 7439-92-1

EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification: B2; probable human carcinogen

BASIS

Sufficient animal evidence. Ten rat bioassays and one mouse assay have shown statistically significant increases in renal tumors with dietary and subcutaneous exposure to several soluble lead salts. Animal assays provide reproducible results in several laboratories, in multiple rat strains with some evidence of multiple tumor sites. Short term studies show that lead affects gene expression. Human evidence is inadequate.

HUMAN CARCINOGENICITY DATA

Inadequate. There are four epidemiologic studies in occupational cohorts exposed to lead and lead compounds. Two studies (Dingwall-Fordyce and Lane 1963, Nelson et al., 1982) did not find any association between exposure to lead and cancer mortality. Selevan et al. (1985) in their retrospective cohort mortality study of primary lead smelter workers found a slight decrease in the total cancer mortality (SMR=95). Apparent excesses were observed for respiratory cancer (SMR=111, obs=41, $p>0.05$) and kidney cancer (SMR=204, obs=6, $p>0.05$). Cooper and Gaffey (1975) and Cooper (1985 update) in their cohort mortality study of battery plant workers and lead smelter workers found statistically significant excesses for total cancer mortality (SMR=113, obs=344), stomach cancer (SMR=168, obs=34) and lung cancer (SMR=124, obs=109) in battery plant workers. Although similar excesses were observed in smelter workers, they were not statistically significant. Cooper and Gaffey (1975) felt it was possible that individual subjects were monitored primarily on the basis of obvious signs of lead exposure, while others who show no symptoms of lead poisoning would not be monitored.

All of the available studies lacked quantitative exposure information, as well as information on the possible contribution of smoking. All studies also had exposures to other metals such as arsenic, cadmium and zinc for which no adjustment was done. The cancer excesses observed in the lung and stomach were relatively small (<200). There was no consistency of site among the various studies, and no study showed any dose-response relationship. Thus, the available human evidence is considered to be inadequate to refute or demonstrate potential carcinogenicity for humans from lead exposure.

ANIMAL CARCINOGENICITY DATA

Sufficient. The carcinogenic potential of lead salts, primarily phosphates and acetates, administered by the oral route, diet or by injection has been demonstrated in rats and mice by more than 10 investigators. The most characteristic cancer response is bilateral renal carcinoma. Rats given lead acetate or subacetate orally have developed gliomas, and lead subacetate also produced lung adenomas in mice after i.p. adminstration. Most of these investigations found a carcinogenic response only at the highest dose. The lead compounds tested in animals are almost all soluble salts. Metallic lead, lead oxide and lead tetralkyls have not been tested adequately. Studies with inhalation exposure have not been located in the literature.

Azar et al. (1973) administerd 10, 50, 100, and 500 ppm lead as lead acetate in dietary concentrations to fifty rats/sex/treatment group for 2 years. One hundred control rats of each sex received the basal laboratory diet. In a second 2-year feeding study, 20 rats/group were given diets containing 0, 1000, and 2000 ppm lead as lead acetate. No renal tumors were reported in the control groups or in treated animals of either sex receiving 10 to 100 ppm. Male rats fed 500, 1000, and 2000 ppm lead acetate had an increased renal tumor incidence of 5/50, 10/20, and 16/20, while 7/20 females in the 2000 ppm developed renal tumors.

The Azar et al. (1973) study is limited by the lack of experimental detail. The possibility of environmental contamination from lead in the air or drinking water was not mentioned. The strains of rats used were not specified in the study, but the Health Effects Assessment for Lead (U.S. EPA, 1984) indicated the rats were Wistar strain. The weight gain at 1000 and 2000 ppm was reported to be depressed, but details were not given.

Kasprzak et al. (1985), in investigating the interaction of dietary calcium on lead carcinogenicity, fed a diet with 1% lead subacetate (8500 ppm Pb) to male Sprague-Dawley rats for 79 weeks. Of the rats surviving (29/30) in this treatment group beyond 58 weeks, 44.8% had renal tumors. Four rats had adenocarcinomas; the remainaing nine had adenomas. Bilateral tumors were noted. No renal tumors were noted among the controls.

As part of a study to determine interactions between sodium nitrite, ethyl urea and lead, male Sprague-Dawley rats were given lead acetate in their drinking water for 76 weeks (Koller et al., 1986). The concentration of lead was 2600 ppm. No kidney tumors were detected among the 10 control rats. Thirteen of 16 (81%) lead-treated rats had renal tubular carcinoma, with three tumors detected at 72 weeks and the remainder detected at the termination of the study.

Van Esch and Kroes (1969) fed basic lead acetate at 0, 0.1%, and 1.0% in the diet to 25 Swiss mice/sex/treatment group for 2 years. No renal tumors developed in the control group, but 6/25 male mice of 0.1% basic lead acetate group had renal tumors (adenomas and carcinomas combined). In the 1.0% group, one female had a renal tumor. The authors felt that the low incidence in the 1.0% group was due to early mortality.

Hamsters given lead subacetate at 0.5% and 1% in the diet had no significant renal tumor response (Van Esch and Kroes, 1969).

SUPPORTING DATA FOR CARCINOGENICITY

Lead acetate induces cell transformation in Syrian hamster embryo cells (DiPaolo et al., 1978) as well as enhances the incidence of simian adenovirus induction. Lead oxide showed similar enhanced adenovirus induction (Casto et al., 1979).

Under certain conditions lead compounds are capable of inducing chromosomal aberrations in vivo and in tissue cultures. Grandjean et al. (1983) showed a relationship between SCE and lead exposure in exposed workers. Lead has been shown, in a number of DNA structure and function assays, to affect the molecular processes associated with the regulation of gene expression (U.S. EPA, 1986).

— QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE —

No Data Available

EPA DOCUMENTATION AND REVIEW

Source Document: U.S. EPA. 1984. Health Effects Assessment for Lead. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Emergency and Remedial Response, Washington, DC. EPA/540/1-86/055. NTIS PB85-163996/AS.

U.S. EPA. 1986. Air Quality Criteria Document for Lead. Volumes III, IV. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC, for the Office of Air Quality Planning and Standards. EPA-600/8-83/028dF.

U.S. EPA. 1987. Preliminary review of the carcinogenic potential of lead associated with oral exposure. Prepared by the Office of Health and Environmental Assessment, Carcinogenic Assessment Group, Washington DC, for the Office of Drinking Water, Office of Solid Waste and the Office of Emergency and Remedial Response (Superfund). OHEA-C-267. Internal Review Draft.

The review of the carcinogenic potential of lead associated with oral exposure has received Agency review.

The 1986 Air Quality Criteria Document for Lead has received Agency and External Review.

Agency Work Group Review: 05/04/88

Verification Date: 05/04/88

EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

William Pepelko / OHEA -- (202)260-5904
Jim Cogliano / OHEA -- (202)260-7338

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13. SUPPORT DOCUMENTS FOR REFERENCE CONCENTRATIONS FROM THE NEW YORK STATE DEPARTMENT OF HEALTH

STATE OF NEW YORK - DEPARTMENT OF HEALTH

INTEROFFICE MEMORANDUM

TO: John K. Hawley, Ph.D., Research Director
Division of Environmental Health Assessment

FROM: Mary Jo Miller, Research Scientist II
Bureau of Toxic Substance Assessment

SUBJECT: Staten Island Project - RfCs

DATE: March 26, 1992

1. Most of the RfC's listed in your 3/23/92 memorandum were derived from oral RfDs assuming a 70 kg adult inhales 23 m^3/day. The oral RfD, calculated RfC and source of the oral RfD are listed below. Short paragraphs on these chemicals are attached.

Chemical	Oral RfD (mg/kg/d)	RfC (ug/m^3)	Oral RfD Source
chloroform	1E-2	30	IRIS (1991); HEAST (1991)
carbon tetrachloride		7E-4	2.1 IRIS (1991): HEAST (1991)
tetrachloroethene	1E-2	30	IRIS (1991); HEAST (1991)
benzene	7E-4	2.1	US EPA (1986)
trichloroethene	7.4E-3	23	US EPA (1987)

2. The RfC for toluene (2,000 ug/m^3) is from HEAST (1991). (Verified and IRIS input pending.)

3. The RfC for chloromethane (826 ug/m^3) is equivalent to the minimal risk level for inhalation exposure derived by ATSDR (1990). A short toxicological paragraph and ATSDR's risk derivation is attached.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1990. Toxicological Profile for chloromethane (TP-90-07). ATSDR, Atlanta, GA.

Health Effects Assessment Summary Tables (HEAST). 1991. Annual FY-1991. US EPA, Office of Research and Development, Washington, D.C.

Integrated Risk Information System (IRIS). 1991. Online. US EPA, Environmental Criteria and Assessment Office, Cincinnati, OH.

U.S. Environmental Protection Agency (US EPA). 1986. Volatile organic chemicals. US EPA, Office of Drinking Water, Washington, DC. (As cited in Draft ATSDR Toxicological Profile on Benzene, 1987.)

U.S. Environmental Protection Agency (US EPA). 1987. Drinking Water Health Advisory for trichloroethene. US EPA, Office of Drinking Water, Washington, DC.

Chloromethane

Chronic inhalation exposure to chloromethane causes liver and kidney damage, nervous system effects, decreased body weight gain, and male reproductive system effects. Male mice exposed by inhalation to high concentrations of chloromethane over their lifetimes developed kidney tumors, but no tumors were observed in female mice and male and female rats. It is not known whether chloromethane could cause reproductive effects or cancer in humans.

Based on a NOAEL of 225 ppm (464 mg/m^3) for reduced body weight gain in rats and mice (CIIT, 1981), the Agency for Toxic Substances and Disease Registry derived a minimum risk level of 0.4 ppm (826 ug/m^3) (ATSDR, 1990). This risk level was derived by adjusting the NOAEL for intermittent exposure (6 hr/day, 5 day/week) and dividing by a 100 fold uncertainty factor.

References

ATSDR. 1990. Toxicological Profile for Chloromethane (TP-90-07). ATSDR, Atlanta, GA.

CIIT. 1981. Final report on a chronic inhalation toxicology study in rats and mice exposed to methyl chloride. Unpublished study: OTS Submission Document ID 40-8120717. (As cited in ATSDR, 1990.)

Benzene

Chronic exposure to benzene causes damage to the blood-forming system (leukopenia and anemia), immune system and nervous system. Benzene has also been associated with leukemia in occupational exposed workers and in experimental animals. The US EPA proposed an oral RfD of 0.0007 mg/kg/day using the wolf et al. (1956) inhalation study in rats and leukopenia as the adverse health effect of concern (US EPA, 1986). A RfC of 2.1 ug/m^3 was derived from the oral RfD assuming a 70 kg adult inhales 23 m^3/day.

References

Wolf, M.A., V.K. Rowe, D.D. McCollister, R.R. Hollingsworth and F. Oyin. 1956. Toxicological studies of certain alkylated benzenes and benzene. AMA Arch. Ind. Health. 14: 387-398.

U.S. Environmental Protection Agency (US EPA). 1986. Volatile organic chemicals. US EPA, Office of Drinking Water, Washington, DC. (As cited in Draft ATSDR Toxicological Profile on Benzene, 1987.)

Chloroform

Chronic exposure to chloroform causes liver and kidney damage and central nervous system effects. Chloroform has been associated with cancer of the liver and kidney in experimental animals. Based on a LOAEL of 12.9 mg/kg/d for liver damage in beagle dogs (Heywood et al., 1979), and using a 1,000 fold uncertainty factor, the US EPA derived an oral RfD of 0.01 mg/kg/day for chloroform (IRIS, 1991). A RfC of 30 ug/m^3 was derived from the oral RfD assuming a 70 kg adult inhales 23 m^3/day.

References

Heywood, R., R.J. Sortwell, P.R.B. Noel, et al. 1979. Safety evaluation of toothpaste containing chloroform. III. Long-term study in beagle dogs. J. Environ. Pathol. Toxicol. 2: 835-851.

Integrated Risk Information System (IRIS). 1991. Online. US EPA, Environmental Criteria and Assessment Office, Cincinnati, OH.

Tetrachloroethene

Chronic exposure to tetrachloroethene causes liver and kidney damage and central nervous system effects. Tetrachloroethene has been associated with cancer of the liver and kidney in experimental animals. Based on a NOAEL of 14 mg/kg/day for liver damage in rats (Buben and O'Flaherty, 1985) and using a 1,000 fold uncertainty factor, the US EPA derived an oral RfD of 0.01 mg/kg/day for tetrachloroethene (IRIS, 1991). A RfC of 30 ug/m^3 was derived from the oral RfD assuming a 70 kg adult inhales 23 m^3/day.

References

Buben, J.A. and E.J. O'Flaherty. 1985. Delineation of the role of metabolism in the hepatotoxicity of trichloroethylene and perchloroethylene: a dose-effect study. Tox. Appl. Pharm. 78: 105-122.

Integrated Risk Information System (IRIS). 1991. Online. US EPA, Environmental Criteria and Assessment Office, Cincinnati, OH.

Trichloroethene

Chronic exposure to trichloroethene causes liver and kidney damage and effects on the nervous system, immune system and blood. Trichloroethene has been associated with liver cancer in experimental animals. The US EPA derived an oral RfD of 0.0074 mg/kg/day based on an inhalation study in rats by Kimmerle and Eben (1973) and increased liver weight as the adverse health effect of concerns (US EPA, 1987). A RfC of 23 ug/m^3 was derived from the oral RfD assuming a 70 kg adult inhales 23 m^3/day. The oral RfD for trichloroethene is currently under review by the US EPA.

References

Kimmerle, G. and A. Eben. 1973. Metabolism, excretion and toxicology of trichloroethylene after inhalation. 1. Experimental exposure on rats. Arch. Toxicol. 30: 115-126.

U.S. Environmental Protection Agency (US EPA). 1987. Drinking Water Health Advisory for Trichloroethene. US EPA, Office of Drinking Water, Washington, DC.

Carbon Tetrachloride

Chronic exposure to carbon tetrachloride causes liver and kidney damage and central nervous system effects. Carbon tetrachloride has been associated with liver cancer in experimental animals. Based on a NOAEL of 0.71 mg/kg/day for liver damage in rats (Bruckner et al., 1986) and sing a 1,000 fold uncertainty factor, the US EPA derived an oral RfD of 0.0007 mg/kg/day for carbon tetrachloride (IRIS, 1991). A RfC of 2.1 ug/m^3 was derived from the oral RfD assuming a 70 kg adult inhales 23 m^3/day.

References

Bruckner, J.W., W.F. Mackenzie, S. Muralidhara, R. Luthra, G.M. Kyle and D. Acosta. 1986. Oral toxicity of carbon tetrachloride: acute, subacute and subchronic studies in rats. Fund. Appl. Toxicol. 6: 16-34.

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STATE OF NEW YORK
DEPARTMENT OF HEALTH
ALBANY

DAVID AXELROD, M.D.
COMMISSIONER

January 4, 1990

Dear Commissioner Jorling:

Enclosed is the document on formaldehyde prepared by staff of the Health Department's Bureau of Toxic Substance Assessment.

Exposure to formaldehyde causes irritation of the eyes, mucous membranes of the upper respiratory tract and skin. Studies of people exposed for five hours or less under controlled conditions provide the best data upon which to base an ambient air criterion for irritant effects. The most sensitive objective response to irritation is a reduction in nasal mucous flow. An air concentration of 30 ug/m³ provides an exposure margin of 10 over the no-observed-effect level (NOEL) for this effect. Although a NOEL for eye, nose, or throat irritation has not been determined, the incidence of these complaints during short-term exposure to 30 ug/m³ is not expected to be above background. The 30 ug/m³ value should not be used as an incremental concentration above non-industrial background because of the potential for irritation effects.

Epidemiologic studies on people exposed to formaldehyde have found significant excesses of cancer of the nose, mouth, oropharynx, and lungs. Other studies have found no excess or non-statistically significant excesses of these cancers. As a whole, the epidemiologic studies provide limited evidence for the carcinogenicity of formaldehyde. Formaldehyde is carcinogenic in both rats and mice. The maximum likelihood estimate (MLE) and lower confidence limit (LCL) estimate of the formaldehyde concentration associated with one in one million excess cancer risk calculated from the rat data are 10 ug/m³ and 0.06 ug/m³, respectively. Because the linearized LCL dose-response curve does not fit the formaldehyde data points well, the latter value contains a greater degree of uncertainty than is commonly found in the LCL calculations for carcinogenic risk.

Because of the uncertainties in the carcinogenic risk estimates and the normal levels of formaldehyde in ambient air (14 to 25 ug/m³ in Singh's 1983 survey), DEC may want to consider alternatives to using the LCL

cancer risk estimate. One approach would be to use the carcinogenic data to support formaldehyde's placement in the high-toxicity category which would require Best Available Control Technology control. In addition, the non-carcinogenic guidance value (30 ug/m^3) would be used to evaluate short-term emissions; it should be used as a short-term ambient concentration (e.g. one to four hours).

Sincerely,

David Axelrod, M.D.
Commissioner of Health

Enc.

Hon. Thomas Jorling
Commissioner
NYS Department of Environmental Conservation
50 Wolf Road
Albany, New York 12233

STATE OF NEW YORK
DEPARTMENT OF HEALTH
ALBANY

DAVID AXELROD, M.D.
COMMISSIONER

April 3, 1990

Dear Commissioner Jorling:

Enclosed is the document on cadmium (Cd) exposure prepared by staff of the Health Department's Bureau of Toxic Substance Assessment.

Most human exposure to cadmium results from ingestion for which the critical toxic endpoint is kidney damage. Inhalation of cadmium also represents a risk for respiratory tract cancer at concentrations that also contribute to kidney toxicity.

Although kidney damage generally occurs when kidney levels exceed 200 ppm Cd, some individuals are at risk at lower concentrations. A kidney concentration of 40 ppm Cd, which corresponds to a daily uptake of 2.9 ug Cd, is a conservative estimate of a 0.1 percent probability for renal dysfunction. This value has been used for estimating the recommended ambient air limit of 20 ng Cd/m^3.

There is limited evidence in humans and sufficient evidence in animals to conclude that cadmium compounds are carcinogenic by inhalation. An increased risk of respiratory cancer was observed among cadmium production workers exposed primarily to cadmium oxide dusts. These data have been used in this document to estimate an air concentration expected to result in an excess human lung cancer risk of 1×10^{-6} after lifetime exposure. This quantitative risk estimate which resulted in an air concentration of 0.5 ng/Cd m^3, was based on human data from a well-conducted epidemiology study, with good exposure estimates, and a sufficient follow-up period to detect an effect. Because of the quality of these data, human-based risk estimates rather than animal-based estimates were used. These risk estimates are applicable to all cadmium compounds.

Exposures from airborne cadmium emissions which accumulate in the soil and foodchain were considered in the derivation of the air limit of 20 ng Cd/m^3, a maximum concentration designed to protect against renal effects. Cadmium has not been demonstrated to be carcinogenic by ingestion exposure alone. If air concentrations are less than 20 ng/m^3, the ingestion pathway is not a critical pathway and would not be separately considered for carcinogenic risk.

Sincerely,

David Axelrod, M.D.
Commissioner of Health

Attachment

Hon. Thomas C. Jorling
Commissioner
NYS Department of Environmental Conservation
50 Wolf Road
Albany, New York 12233

STATE OF NEW YORK
DEPARTMENT OF HEALTH
ALBANY

DAVID AXELROD, M D
COMMISSIONER

May 2, 1990

Dear Commissioner Jorling:

Enclosed is the health risk assessment of chromium (Cr) exposure prepared by staff of the Health Department's Bureau of Toxic Substance Assessment.

Occupational exposure to chromium compounds causes allergic dermatitis, respiratory tract irritation, ulcerations of the nasal septum and cancer. In experimental animals, inhalation exposure to chromium compounds produces pathological changes in the lungs, respiratory impairment, immunosuppression, nasal irritation, kidney damage, liver damage and cancer. In general, hexavalent chromium (CrVI) compounds are more toxic than trivalent chromium (CrIII) compounds due to their relatively higher solubility, greater absorption by body tissues, and greater reactivity.

The most sensitive endpoints on which to base a non-carcinogenic guideline are chromium's effects on the pulmonary system. The severity of these effects depends on the amount inhaled, the duration of the exposure, and the solubility and valence state of the chromium compound. These factors, along with the likelihood that chromium in ambient air will not be present in just one form or valence state, were taken into consideration in calculating an ambient air limit of 0.1 ug total Cr/m^3 for non-cancer endpoints.

Workers in the chromate industry have an increased risk of respiratory cancer. Most studies of the carcinogenic effects of chromium compounds in humans involved exposures to a combination of metallic, trivalent and hexavalent chromium. Although the relative contributions of each of these three forms of chromium to cancer risk is unknown, hexavalent chromium itself has been implicated as an etiologic agent.

A cohort study of workers in a chromate production facility was chosen for the quantitative cancer risk assessment as it has a relatively long follow-up period and adequate exposure data. The estimate of the ambient air concentration of Cr(VI) that corresponds to an excess human lung cancer risk of 1×10^{-6} after lifetime exposure is 0.02 ng Cr(VI)/m^3. Since Cr(VI) is implicated as the principal, if not the only carcinogenic agent, the cancer risk estimate should only be used for evaluating emissions where Cr(VI) is present.

People can be exposed to chromium emissions by inhalation or ingestion of foods (vegetables or animal products). Increased exposures from chromium emissions which accumulate in the soil are expected to be small. If the suggested guidelines are not exceeded, the ingestion pathway is not critical and normally would not have to be evaluated.

Sincerely,

David Axelrod, M.D.
Commissioner of Health

Enclosure

Hon. Thomas C. Jorling
Commissioner
New York State Department of
Environmental Conservation
50 Wolf Road
Albany, New York 12233-1010

STATE OF NEW YORK
DEPARTMENT OF HEALTH
ALBANY

DAVID AXELROD, M.D.
COMMISSIONER

January 4, 1989

Dear Commissioner Jorling:

Enclosed is the document on nickel (Ni) prepared by staff of the Health Department's Bureau of Toxic Substance Assessment.

Occupational exposure to nickel compounds causes allergic dermatitis, nasal and respiratory tract irritation and asthmatic lung disease. In experimental animals, subchronic and chronic inhalation exposure to nickel compounds (0.025 to 2 mg/m^3) causes pathological changes in the lungs, respiratory impairment, immunosuppression, hyperglycemia and fetotoxicity. An ambient air limit of 20 ng Ni/m^3 is recommended to provide the general population with a sufficient margin-of-safety over the inhaled dose associated with adverse respiratory effects.

Nickel subsulfide and refinery dust created during the nickel refining process are associated with an excess cancer risk in humans or animals. The data on the carcinogenic potential for other forms of nickel are insufficient to make definitive conclusions about their carcinogenicity by inhalation. Some evidence, although limited, suggests that nickel powder, sulfide, oxide, hydroxide, carbonate, acetate and nickelocene forms may have carcinogenic activity.

From the animal data, we estimated that the concentration of nickel subsulfide in air associated with one in one million excess cancer risk is 0.2 ng Ni/m^3. For those compounds having limited evidence of carcinogenicity, the process being regulated should determine whether the air concentration associated with a one in one million excess cancer risk is based on either the animal nickel subsulfide data (0.2 ng Ni/m^3) or the human nickel refinery dust data (4 ng Ni/m^3); the limited strength of the cancer data should also be noted. Other nickel compounds should be regulated on the basis of non-carcinogenic endpoints.

Presently, the risk assessments for nickel exposure from air emissions evaluate both the inhalation and ingestion pathways. Increased ingestion exposures and the risks from nickel emissions which accumulate in the soil are expected to be small. The ingestion pathway is not critical and normally would not have to be evaluated.

Sincerely,

David Axelrod, M.D.
Commissioner of Health

Attachment

Hon. Thomas Jorling
Commissioner
NYS Department of Environmental Conservation
50 Wolf Road
Albany, New York 12233

STATE OF NEW YORK
DEPARTMENT OF HEALTH
ALBANY

DAVID AXELROD M D
Commissioner

January 28, 1991

Dear Commissioner Jorling:

Enclosed is the report on vanadium prepared by staff of the Health Department's Bureau of Toxic Substance Assessment.

A wide array of adverse effects on the respiratory tract, including bronchitis, emphysema, tracheitis, pulmonary edema and bronchial pneumonia, have been associated with exposure to vanadium compounds in air. Pulmonary sensitization has also been reported. Although several studies have been conducted on factory workers exposed to vanadium pentoxide dust, these studies have deficiencies which limit their usefulness for developing an ambient air criterion. Animal studies, though limited with respect to duration of exposure and/or presentation of study details, provide dose-response data on lung effects which were used to derive a criterion of 0.2 ug vanadium/m^3 of air.

Exposures from airborne cadmium emissions which accumulate in the soil and foodchain were considered in the derivation of the criterion of 0.2 ug/m^3. The health risk from ingested vanadium is less than that of inhaled vanadium. If air concentrations are less than 0.2 ug/m^3, the ingestion pathway is not critical and would not need to be considered.

Sincerely,

David Axelrod, M.D.
Commissioner of Health

Enclosure

Hon. Thomas C. Jorling
Commissioner
NYS Department of Environmental Conservation
50 Wolf Road
Albany, New York 12233

STATE OF NEW YORK
DEPARTMENT OF HEALTH
ALBANY

DAVID AXELROD, M.D.
COMMISSIONER

May 23, 1989

Dear Commissioner Jorling:

In September of 1984 the Departments of Health and Environmental Conservation entered into a memorandum of understanding which formalized the working relationship between the two agencies for health risk assessments for chemicals associated with air emissions from municipal waste incinerators. The Department of Health agreed to develop ambient air guidance for six heavy metals, hydrogen chloride, polychlorinated dibenzodioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), polychlorinated hydrocarbons (PAHs), benzo[alpha]pyrene (BaP), crysene, formaldehyde, and PCBs. Now PCDDs and PCDFs are in one document, as are PAHs, BaP, and crysene; three additional metals are added to the list of chemicals.

Scientific documentation to support air guidelines are being finalized for fourteen compounds or groups of compounds. These drafts have undergone scientific peer review by independent scientists outside of the Department of Health. The drafts have also been reviewed by Department of Environmental Conservation staff. Responsiveness summaries have been prepared to address comments and final revisions to the documents are expected to be completed soon. Each document is being transmitted to the Department of Environmental Conservation with recommendations which are protective of public health.

The zinc document is enclosed, along with the peer reviewers' comments, and a response to the reviewers' comments. The fourteen other documents will be completed during the next few months, and transmitted at the rate of two per month.

Zinc is an essential element necessary for normal growth and many body functions. The respiratory tracts of humans and animals have been adversely affected by inhalation of particulate zinc compounds. However, the respiratory effects elicited by particulate matter containing zinc and/or zinc oxide are the same as those elicited by particulate matter which does not contain zinc. Therefore, control of particulates in ambient air should adequately protect the public health from exposure to zinc and zinc oxide.

Specific zinc compounds (i.e., zinc chloride, zinc sulfate, and zinc chromate) have a greater toxic potential than zinc and zinc oxide; these effects are primarily attributable to the anionic component of the compound and not zinc itself. Control of these specific zinc compounds, when warranted, should be achieved by compliance with specific, more stringent, criteria associated with the anions (e.g., zinc chloride will be covered by the hydrochloric acid criterion and zinc chromate by the chromium criteria).

I recommend that all the documents, the Health Department's recommendations, and control information from DEC be taken to public hearings, along with proposed ambient air standards. Following the public hearings, ambient air standards should be adopted. Our staff is available to work with yours on this project. As further discussions are warranted, please feel free to contact me.

Sincerely,

David Axelrod, M.D.
Commissioner of Health

Enclosures

Hon. Thomas C. Jorling
Commissioner
NYS Dept. of Environmental Conservation
50 Wolf Road
Albany, New York 12233

14. MEMORANDUM ON THE REFERENCE CONCENTRATION FOR CHROMIUM

Attachment II

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF RESEARCH AND DEVELOPMENT
ENVIRONMENTAL CRITERIA AND ASSESSMENT OFFICE
CINCINNATI, OHIO 45268

August 26, 1992

SUBJECT: RfC for Total Chromium

FROM: Joan S. Dollarhide
Chemical Mixtures Assessment Branch
Environmental Criteria and Assessment Office

TO: Marian Olsen
Policy and Program Integration Branch
U.S. EPA, Region II

This memo is in response to your request for ECAO's assistance in evaluating the appropriateness of various RfCs for total chromium. As I mentioned during our phone conversation, I feel that the uncertainties surrounding the chromium RfC are so great that your best approach would be to postpone a final decision regarding an appropriate RfC until the results of a public meeting on the subject, to be held in December, are available. However, since your time-frame makes this impossible, I will summarize the information we currently have.

Review of the NYSDOH RfC for Chromium

The RfC for chromium derived by the New York State Department of Health (NYSDOH) is not consistent with the U.S. EPA approach described in Interim Methods for Development of Inhalation Reference Doses (U.S. EPA, 1989; EPA/600/8-88/066F). The specific aspects of the NYSDOH number which are inconsistent are described below:

1. Use of animal data: The Interim Methods state that human data are preferred over animal data for deriving an RfC. The NYSDOH uses animal data although adequate human studies are available.

2. Selection of critical study and LOAEL: U.S. EPA methodology states that the study which defines the highest NOAEL, or lowest LOAEL if a NOAEL is not defined, and demonstrates a dose-response relationship should be selected as the critical study. The NOAEL or LOAEL from the critical study is then used to estimate the RfC by applying the appropriate uncertainty factors. The NYSDOH report did not select a single critical study, but rather selected a LOAEL, defined as "reasonable", from a range of LOAELs defined by several animal studies.

3. Estimation of animal inhaled dose and human ADI: The Interim Methods state that dosimetric adjustments are to be used to convert

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an animal LOAEL to a Human Equivalent Concentration (HEC) LOAEL [$LOAEL_{(HEC)}$]. For aerosols and particulates, the dosimetric adjustment consists of multiplying the animal LOAEL by the Regional Deposited Dose Ratio (RDDR) for the region of the respiratory tract in which an adverse effect was observed to obtain the $LOAEL_{(HEC)}$. The RDDR takes into account the aerodynamic properties of the particles and the differences between animal and human in the respiratory tract surface area and architecture. The $LOAEL_{(HEC)}$ is then divided by the appropriate uncertainty factor to obtain the RfC in mg/cu.m. The NYSDOH method of calculating animal inhaled dose (air concentration multiplied by animal inhalation rate and divided by animal body weight) does not consider the aerodynamic properties of the particles. By using the animal inhaled dose to derive the human ADI, NYSDOH does not account for interspecies differences in respiratory system surface area and structure.

U.S. EPA RfC for Chromium

As you know, in 1990 the RfD/RfC Work Group reviewed and subsequently verified an RfC for total chromium of 2E-6 mg/cu.m. This RfC was based on a human occupational study (Lindberg and Hedenstierna, 1983) which identified nasal mucosa atrophy as the critical effect at a $LOAEL_{(HEC)}$ of 0.000714 mg/cu.m. An uncertainty factor of 300 was applied. The sources of the differences between this RfC and the NYSDOH RfC include different critical study (human vs. animal), different critical effect (nasal mucosa atrophy vs. effects on alveolar macrophages), different choice of $LOAEL_{(HEC)}$ (.000714 mg/cu.m vs. .03 mg/kg/day), and different choice of uncertainty factor (300 vs. 1000).

Since this RfC was reviewed, EPA has received numerous public comments which have resulted in the RfC being placed "under review and also withdrawn from the HEAST. Below is a summary of the public comments received. These issues will be considered as part of the ongoing development process for a chromium RfC.

1. The proposed RfC is below the analytical limits of detection recommended by EPA and below naturally occurring levels of Cr(III) and Cr(VI) in much of the United States.

2. In the critical study, workers were exposed to chromic acid mists, which contain almost no Cr(III). Therefore, it is inappropriate to use this study to derive an RfC for Cr(III). Furthermore, separate RfCs for Cr(III) and Cr(VI) have been suggested as more appropriate than a single RfC.

3. Since the physico-chemical and toxicological properties of chromic acid appear to be different from those associated with environmental chromium, separate RfCs for mists and particulates have been suggested.

4. The critical effect selected may be due to the irritant nature

of the chromic acid mist and not to any direct effect of chromium. In other words, other types of chromates which have a higher pH might not have the same effects.

5. The critical study appears to define a NOAEL of .001 mg/cu.m that would be more appropriate than the LOAEL of .002 mg/cu.m used by the RfD/RfC Work Group.

6. The choice of uncertainty factors was questioned. In particular, comments suggested that the UF for absence of developmental studies and the UF for extrapolation from a subchronic study were not necessary.

As you can see, there are significant problems with both the NYSDOH and EPA RfCs for chromium that make each less than ideal. Whichever RfC you select, be sure to include a complete discussion of all the uncertainties associated with that RfC. Also note that, given the uncertainties associated with a chromium RfC, using a Hazard Index of 1 as the threshold for action may be unnecessarily restrictive. I have enclosed a summary of the previously reviewed EPA chromium RfC. It should give you more information on how the RfC was derived, and it gives the $NOAEL_{(HEC)}$ and $LOAEL_{(HEC)}$ using dosimetric adjustments for several of the animal studies cited in the NYSDOH report. If you have additional questions, please call.

15. MEMORANDUM ON THE REFERENCE CONCENTRATION FOR XYLENE

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF RESEARCH AND DEVELOPMENT
ENVIRONMENTAL CRITERIA AND ASSESSMENT OFFICE
CINCINNATI, OHIO 45268

September 3, 1992

SUBJECT: Reference Concentration (RfC) listings for xylene (mixed and o-, m- and p-isomers) in HEAST

FROM: Kenneth A. Poirier, Ph.D.
Environmental Health Scientist
Chemical Mixtures Assessment Branch

TO: Marian Olsen
U.S. EPA - Region II
OPM, Policy and Program Integration Branch

This memo is a response to your telephone query on September 1 regarding the Reference Concentration (RfC) listings for xylene (mixed and o-, m- and p-isomers) in HEAST. As you pointed out, the most recent update of HEAST (March 1992), no longer has these values listed. The reason for this is as follows:

The HEAST lists toxicity values for chemicals that come from a number of sources that have various levels of Agency review. Many times a listing for a RfC or Reference Dose (RfD) is taken from an Agency document that has had some programmatic review, though not necessarily an Agency consensus review as occurs within the RfD/RfC Work Group. It is for this reason that the user of HEAST carefully check the source of the toxicity value when choosing to use a value that is listed on HEAST.

For the RfC and RfD toxicity values, the ultimate Agency review occurs when a risk assessment for a chemical is reviewed and verified by the Agency's RfD/RfC Work Group. When a risk assessment reaches verified status, it is loaded onto the Agency's Integrated Risk Information System (IRIS). When this occurs a notation of "IRIS" is made in HEAST. Because the documentation of a verified risk assessment is extensive, the HEAST user is strongly urged to consult IRIS. By not listing the IRIS value, the user is forced to use IRIS and hopefully read the rationale used and limits placed on each risk assessment.

In the case of the xylenes, the RfC values have been left blank pending loading onto IRIS. The RfCs for mixed, o-, m- and p-xylene have reviewed and verified by the Agency's RfD/RfC Work Group. These four inhalation risk assessments have been found to have inadequate data on which to establish a chronic risk assessment and have been designated as NOT VERIFIABLE. A statement

to this effect will soon appear on IRIS. At that time an IRIS notation will be placed on HEAST. Until appropriate data is made available to reassess the chronic inhalation effects, a RfC can not be derived.

cc: J. Dollarhide
L. Knauf
C. Sonich-Mullin

16. MEMORANDUM ON THE WEIGHT-OF-EVIDENCE CLASSIFICATION OF TETRACHLOROETHYLENE AND TRICHLOROETHYLENE

Memorandum to M. Olsen concerning carcinogen characterizations for perchloroethylene and trichloroethylene. Cincinnati, OH: Office of Research and Development, Environmental Criteria and Assessment Office, U.S. Environmental Protection Agency. 1992.

Tetrachloroethylene (perchloroethylene, PERC) (CASRN 127-18-4)

The carcinogenicity characterization has a long history. A July 1985 Health Assessment Document for Tetrachloroethylene (Perchloroethylene), EPA # 600/8-82/005F, classified the agent in Weight-of-Evidence Group "C - Possible Human Carcinogen" mentioning that this would be reevaluated because of new information. The 1985 document also provided upper bound inhalation and oral risk estimates. An April 1987 Addendum to the Health Assessment Document, EPA# 600/8-82/005FA, proposed that the Weight-of-Evidence be upgraded to "B2 - Probable Human Carcinogen" and provided a revised inhalation risk estimate. A February 1991 document titled Response to Issues and Data Submissions on the Carcinogenicity of Tetrachloroethylene, EPA# 600/6-91/002A discussed newer data relative to weight-of-evidence classification. The Agency's Science Advisory Board has reviewed these documents finding them to be technically adequate while offering an opinion that the weight-of-evidence is on C-B2 continuum (C=Possible Human Carcinogen, B2=Probable Human Carcinogen). At present time, the Agency has not adopted a final position on the weight-of-evidence classification.

The upper bound risk estimates from the 1985 Health Assessment Document as amended by updated inhalation values from the 1987 Addendum have not as yet been verified by the IRIS-CRAVE Workgroup. The estimates are viewed as useful information in the context of the information available in the 1985-1987 period.

ORAL: 1985 HAD; Unit risk = 1.5E-6 per ug/L

Slope Factor = 5.2E-2 per mg/kg/day

INHALATION: 1987 Addendum; Unit risk = range form 2.9E-7 to 9.5E-7 with a geometric mean of 5.8E-7 per ug/cu.m

Slope factor = 2.0E-3 per mg/kg/day

Those needing to make a choice about carcinogenicity have found the 1985, 1987 and 1991 EPA documents and the 1988 and 1991 Science Advisory Board letters of advice useful background information. When the Agency makes a decision about weight-of-evidence, the CRAVE-IRIS verification will be completed and the information put on IRIS.

Trichloroethylene (TCE) (CASRN 79-01-6)

The current phase of the carcinogenicity characterization for trichloroethylene started with a July 1985 Health Assessment Document for Trichloroethylene, EPA# 600/8-82/006F which classified trichloroethylene in Weight-of-Evidence Group "B2 - Probable Human Carcinogen". Inhalation and oral upper bound risk estimates were provided. This information was verified on IRIS from 3/87 through 7/89. A June 1987 Addendum to the Health Assessment Document for Trichloroethylene, EPA# 600/8-82/006FA proposed that the Weight-of-Evidence finding of "B2" was further supported by newly available animal bioassay data and offered a minor revision to the inhalation upper bound risk estimate. In 1988 the Agency's Science Advisory Board offered an opinion that the weight-of-evidence was on C-B2 continuum (C=Possible Human Carcinogen, B2=Probable Human Carcinogen). The Agency withdrew the IRIS carcinogenicity file in 7/89 and has not adopted a current position on the weight-of-evidence classification.

The quantitative risk estimates provided in the 1985 Health Assessment Document and 1987 Addendum have been reviewed by the IRIS-Crave Workgroup but are not verified as such pending resolution of the weight-of-evidence classification. The upper bound risk values in these documents are as follows:

ORAL: 1985 HAD; Unit Risk = 3.2E-7 per ug/L
Slope Factor = 1.1E-2 per mg/kg/day

INHALATION: 1987 Addendum; Unit Risk = 1.7E-6 per ug/cu.m.
Slope Factor = 6.0E-3 per mg/kg/day

When the Agency adopts a current position on weight-of-evidence classification, the trichloroethylene file will be reentered on IRIS.

17. TOXICOLOGICAL SUMMARIES FOR CHEMICALS FOR WHICH TOXICOLICAL INFORMATION WAS INSUFFICIENT FOR RISK ASSESSMENT IN THE SI/NJ UATAP

Barium and compounds
Chlorobenzene
Cobalt
Copper
Dichloroethane, 1,1-
Selenium
Styrene
Trichloroethane, 1,1,1-
Xylenes
Zinc

TOXICOLOGICAL PROFILE FOR BARIUM AND COMPOUNDS

Prepared by:

Clement International Corporation
Under Contract No. 205-88-0608

Prepared for:

Agency for Toxic Substances and Disease Registry
U.S. Public Health Service

July 1992

Toxicological Profile
TP-91/03

Barium

2.4 RELEVANCE TO PUBLIC HEALTH

No acute-, intermediate-, or chronic-duration inhalation MRLs were derived for barium because studies evaluating the effects of barium in humans and animals following acute, intermediate, and chronic inhalation exposure were inadequate for establishing the exposure concentrations associated with adverse health effects. The human studies (Doig 1976; Essing et al. 1976; Seaton et al. 1986; Shankle and Keane 1988) were limited by the small number of subjects and the lack of quantitative exposure information. The animal studies (Hicks et al. 1986; Tarasenko et al. 1977) were limited by inadequate descriptions of the experimental design.

No acute-, intermediate-, or chronic-duration oral MRLs were derived for barium because of limitations of the studies evaluating oral exposure to barium for such durations. Case studies of acute exposures in humans did not provide adequate characterization of the doses associated with adverse health effects and acute-duration animal studies did not provide sufficient data to identify a target organ.

Intermediate-duration oral studies in humans either did not provide adequate characterization of doses associated with adverse health effects (Brenniman and Levy 1985; Brenniman et al. 1979a, 1981) or the number of subjects examined was too small (Wones et al. 1990). The observation of increased blood pressure in an intermediate-duration oral study in rats (Perry et al. 1983, 1985, 1989) was not used to set an MRL because the resulting MRL would be approximately 1.5-4-fold lower than the estimated daily intake of barium from air, water, and dietary sources combined.

No chronic-duration oral MRL was established for barium, despite the observation of a NOAEL and a LOAEL for blood pressure effects in a chronic rat study by Perry et al. (1983, 1985, 1989), because the resulting MRL would have been approximately 19-50-fold lower than the estimated daily intake of barium from air, water, and dietary sources combined.

No acute-, intermediate-, or chronic-duration dermal MRLs were derived for barium because of the lack of an appropriate methodology for the development of dermal MRLs.

Barium is naturally present to some extent in water and food. Consequently, the general population is exposed normally to barium through the ingestion of drinking water or food. The general population also is exposed by inhalation to low levels of barium in ambient air. Exposure to barium

through public drinking water supplies, food, or ambient air generally should not pose a significant health risk to humans because of the very low levels of barium that would typically be associated with these types of exposures.

Since barium is a frequent contaminant at hazardous waste sites, humans living or working near these sites may potentially become exposed to barium. Concentrations of barium in soil or groundwater may be significantly elevated over background levels at hazardous waste sites, thereby posing a potential health risk to humans. Soil contaminated with barium is of concern because airborne dusts generated from contaminated surface soil through the action of wind may potentially expose individuals by inhalation. Airborne barium dusts generated from contaminated surface soil could potentially form residues on foods that are ingested. There is also the potential that children may ingest barium through hand to mouth contact following playing in contaminated soil. Groundwater contaminated with barium is of concern because of the potential for humans to ingest such water. Contaminated soil and groundwater also are of concern because individuals may directly become exposed dermally through airborne dusts, through direct contact with contaminated soil from construction, excavation, or recreational activities, and/or through direct contact by showering with contaminated water.

There is little quantitative information regarding the extent of barium absorption following inhalation, oral, or dermal exposure. Available evidence indicates that barium is absorbed to some extent following inhalation, oral, and dermal exposure; however, absorption in some cases is expected to be limited. For example, there is some evidence that gastrointestinal absorption of barium in humans is less than 5-30% of the administered dose. These latter data suggest that although individuals may become exposed orally to high levels of barium, adverse health effects may not necessarily develop because of the limited gastrointestinal absorption. Another important factor affecting the development of adverse health effects in humans is the solubility of the barium compound to which the individual is exposed. Soluble barium compounds would generally be expected to be of greater health concern than insoluble barium compounds because of the greater potential of soluble barium compounds to be absorbed by the body.

The different barium compounds have different solubilities in water and body fluids and therefore they serve as variable sources of the Ba^{2+} ion. The Ba^{2+} ion and the soluble compounds of barium (notably chloride, nitrate, hydroxide) are toxic to humans. The insoluble compounds of barium (notably sulfate and carbonate) are inefficient sources of Ba^{2+} ion because of limited solubility and are therefore generally nontoxic to humans (ILO 1983). The insoluble, nontoxic nature of barium sulfate has made it practical to use this particular barium compound in medical applications such as enema procedures and in x-ray photography of the gastrointestinal tract. Barium provides an opaque contrasting medium when ingested or given by enema prior to x-ray examination. Under these routine medical situations, barium sulfate is generally safe. However, barium sulfate or other insoluble barium compounds

may potentially be toxic when it is introduced into the gastrointestinal tract under conditions where there is colon cancer (Princenthal et al. 1983) or perforations of the gastrointestinal tract and barium is allowed to enter the blood stream.

Barium has been associated with a number of adverse health effects in both humans and experimental animals. Both human and animal evidence suggests that the cardiovascular system may be one of the primary targets of barium toxicity. In addition to cardiovascular effects, exposure of humans and/or animals to barium has been associated with respiratory, gastrointestinal, hematological, musculoskeletal, hepatic, renal, neurological, developmental, and reproductive effects. No data or insufficient data are available to draw conclusions regarding the immunological, genotoxic, or carcinogenic effects of barium. Death has been observed in some individuals following acute oral exposure to high concentrations of barium. The following section evaluates the significance of existing toxicity data on barium with regard to human health.

Death. No studies were available regarding death in humans or animals after inhalation or dermal exposure to barium. However, mortality has been reported to occur in a number of cases where humans have been exposed acutely to barium through accidental or intentional ingestion (Das and Singh 1970; Diengott et al. 1964; McNally 1925; Ogen et al. 1967; Talwar and Sharma 1979). The observations from human case reports are supported by findings from acute studies with rodents that indicate barium is toxic by the oral route (Borzelleca et al. 1988; Tardiff et al. 1980). Reduced lifespan also has been observed in chronic oral studies with mice (Schroeder and Mitchener 1975b). The results from human case studies and animal studies suggest that humans who are exposed orally to high levels of barium may be at increased risk for mortality.

One death in an adult female due to acute intravasation of barium sulfate during a barium enema was found in the literature. Direct entry of barium sulfate into the circulatory system apparently resulted in cardiorespiratory failure (Cove and Snyder 1974). Acute parenteral administration of barium compounds to animals has resulted in death. Rate of administration, total dose, species, and individual differences are all factors affecting the ability of barium and its compounds to cause death. Major symptoms leading to death are hypokalemia (Jalinski et al. 1967; Roza and Berman 1971; Schott and McArdle 1974), muscle paralysis (Roza and Berman 1971; Schott and McArdle 1974), cardiorespiratory failure (Cove and Snyder 1974; Roza and Berman 1971), and convulsions (Segreti et al. 1979; Welch et al. 1983). Parenteral administration is not a normal route of barium exposure in humans and only on the rare occurrence of intravasation during barium enema would it be expected to be a problem. However, many of the symptoms experienced are the same as those experienced by humans and animals exposed to acute doses by inhalation and ingestion.

Systemic Effects

Respiratory Effects. Studies evaluating the respiratory effects of barium following inhalation, oral, and dermal exposure are limited. Benign pneumonoconiosis has been observed in workers exposed occupationally by inhalation to barium (Doig 1976). However, no respiratory effects were observed in another study of workers exposed to barium carbonate dust by inhalation (Essing et al. 1976). There are case reports of individuals who developed respiratory weakness and paralysis following acute ingestion of barium (Das and Singh 1970; Gould et al. 1973; Lewi and Bar-Khayim 1964; Morton 1945; Ogen et al. 1967; Phelan et al. 1984; Wetherill et al. 1981). Respiratory effects have not been evaluated in humans following dermal exposure. Accumulation of fluid in the trachea has been noted in acute oral studies with rats (Borzelleca et al. 1988). The results from human case and occupational studies and from acute oral studies with rats suggest that humans who are exposed orally or by inhalation to barium may be at increased risk for minor respiratory effects.

Acute intravasation of barium sulfate into the circulatory system of an adult female patient following a barium enema procedure caused the compound to be deposited in blood vessels throughout the body, including the lungs, and resulted in respiratory failure (Cove and Snyder 1974). Acute parenteral administration of barium compounds to animals has been shown to result in paralysis of the respiratory muscles (Roza and Berman 1971). Similar respiratory paralysis is frequently encountered in cases of acute exposure in humans and animals by ingestion or inhalation. Intratracheal administration of barium sulfate into rat lungs produced a mild inflammatory reaction (Huston et al. 1952). Barium sulfate could not be removed by either polymorphonuclear leukocytes or monocytes. A tissue reaction followed; however, no fibrosis was observed. Since this mode of entry is similar to inhalation, these results may be significant for cases of inhalation exposure.

Cardiovascular Effects. No reliable information is available regarding cardiovascular effects in humans or animals for inhalation or dermal exposure. However, case reports of humans exposed orally by acute ingestion and results of acute, intermediate, and chronic oral studies with experimental animals indicate that barium induces a number of cardiovascular effects. These effects include increased blood pressure, changes in heart rhythm, myocardial damage, and changes in heart physiology and metabolism (Das and Singh 1970; Diengott et al. 1964; Gould et al. 1973; Kopp et al. 1985; Lewi and Bar-Khayim 1964; McNally 1925; Perry et al. 1983, 1985, 1989; Talwar and Sharma 1979; Wetherill et al. 1981). The results from this study suggest that humans who are exposed orally to barium may be at increased risk for cardiovascular effects.

In addition to cardiovascular effects following oral exposure, cardiovascular effects have been observed in humans following intravasation of barium and in animals following parenteral barium exposure. During a barium

sulfate enema procedure on an adult female, the patient developed cardiorespiratory failure (Cove and Snyder 1974). On necropsy, barium sulfate was found throughout the circulatory system, including the heart. The authors attributed the death of the woman to barium intravasation. In animals, parenteral administration of barium compounds has been shown to cause hypertension and dysrhythmias (Foster et al. 1977; Mattila et al. 1986; Roza and Berman 1971). Although parenteral exposure is not a common exposure route for humans, similar symptoms are observed in cases of acute oral and inhalation exposure in humans and animals.

In vitro research in mammalian systems indicated barium induces both contraction and automaticity in isolated hearts and heart muscles (Delfino et al. 1988; Ehara and Inazawa 1980; Hiraoka et al. 1980; Katzung and Morgenstern 1976; Mascher 1973; Munch et al. 1980; Saeki et al. 1981; Slavicek 1972; Toda 1970). Electrical and mechanical effects caused by barium appear to be primarily calcium dependent, although barium could still induce contractions and pacemaker activity in calcium deficient media (Ebeigbe and Aloamaka 1987; Ehara and Inazawa 1980; Hiraoka et al. 1980; Slavicek 1972; Toda 1970). Barium has also been shown to cause significant alterations of most myocyte components and degeneration of mitochondria and the contractile apparatus (Delfino et al. 1988). Repeated exposures to barium in isolated heart systems resulted in tachycardia (Ebeigbe and Aloamaka 1987). These in vitro findings offer some possible explanations for the heart abnormalities seen in barium toxicity in humans and animals.

Gastrointestinal Effects. Reliable human and animal studies evaluating the gastrointestinal effects of barium following inhalation and dermal exposure were not available. Data from case reports of humans suggest that gastrointestinal hemorrhage and gastrointestinal disturbances, including gastric pain, vomiting, and diarrhea, have been associated with acute oral exposure to barium (Das and Singh 1970; Diengott et al. 1964; Gould et al. 1973; Lewi and Bar-Khayim 1964; McNally 1925; Morton 1945; Ogen et al. 1967; Phelan et al. 1984; Talwar and Sharma 1979; Wetherill et al. 1981). Inflammation of the intestines has been noted in acute oral studies with rats (Borzelleca et al. 1988). No data were available from intermediate or chronic exposure studies. Results from human case studies and acute studies with rats suggest that humans exposed orally to barium for acute periods may develop gastrointestinal effects.

Two case studies of acute intrusion of barium sulfate into the peritoneal space during barium enema examination of four men showed barium sulfate caused an acute inflammatory tissue response (Kay 1954; Yamamura et al. 1985), and in one case resulted in formation of a fibrous granuloma (Kay 1954). This is an extremely rare mode of entry and not of significant concern for individuals exposed at a hazardous waste site. Increased fluid accumulation in the intestinal lumen of rats was observed after intraperitoneal injection of barium chloride (Hardcastle et al. 1983b, 1985); however, this observation is not significant for individuals exposed at

hazardous waste sites because of the route of exposure and because there has been no documentation of this effect occurring in humans following normal exposure routes.

Limited studies have been done in vitro on mammalian gastrointestinal systems. Generally, they indicated that barium induced intestinal secretion by releasing intracellular calcium, which combined with calmodulin to stimulate the secretory process (Hardcastle et al. 1983a, 1983b, 1985). Barium also increased gastrointestinal tissue sugar accumulation and decreased mucosal to serosal galactose fluxes. The two proposed mechanisms for this are (1) activation of the calcium-calmodulin complex or (2) direct action of barium on smooth muscle tone (Alcalde and Ilundain 1988). The relevance of these effects on the gastrointestinal tract is unknown.

Hematological Effects. No reliable studies were available regarding hematological effects in humans or animals following inhalation or dermal exposure to barium. There is suggestive evidence from case reports that acute inhalation, oral, and dermal exposure of humans is associated with lowered blood potassium levels (Diengott et al. 1964; Gould et al. 1973; Lewi and Bar-Khayim 1964; Phelan et al. 1984; Shankle and Keane 1988; Stewart and Hummel 1984; Talwar and Sharma 1979; Wetherill et al. 1981). These findings suggest that humans exposed to barium by various routes may be at increased risk for minor hematological effects.

Several studies of animals exposed to barium by parenteral routes indicate that barium decreases in serum potassium (Foster et al. 1977; Jaklinski et al. 1967; Roza and Berman 1971; Schott and McArdle 1974). In one study, dogs intravenously administered barium chloride demonstrated a decrease in serum potassium accompanied by an increase in red blood cell potassium concentration (Roza and Berman 1971). The authors concluded that the observed hypokalemia was due to a shift of potassium from extracellular to intracellular compartments and not to excretion. Additional intravenous studies have linked the observed hypokalemia to muscle paralysis in rats (Schott and McArdle 1974) and cardiac arrhythmias in dogs (Foster et al. 1977). These experiments in animals strongly support the suggestive human case study evidence indicating hypokalemia is an important effect of acute barium toxicity.

Musculoskeletal Effects. No studies were available in humans or animals regarding musculoskeletal effects of barium following dermal exposure. Case reports of humans indicate that acute inhalation and acute oral exposure to barium has been associated with muscle weakness and paralysis (Das and Singh 1970; Diengott et al. 1964; Gould et al. 1973; Lewi and Bar-Khayim 1964; McNally 1925; Morton 1945; Ogen et al. 1967; Phelan et al. 1984; Shankle and Keane 1988; Wetherill et al. 1981). Occupational exposure has not, however, been found to result in radiologically apparent barium deposits in skeletal muscle or bone (Essing et al. 1976). Very limited animal data are available regarding musculoskeletal effects. No adverse effects on the musculoskeletal

system were reported in an intermediate oral study with rats (Tardiff et al. 1980). The findings from human case reports suggest that humans having acute oral or inhalation exposure to barium may develop musculoskeletal effects.

No data on musculoskeletal involvement in cases of barium exposure by other than oral or inhalation modes have been reported for humans. In animals receiving acute doses of barium compounds parenterally, both muscle twitching and paralysis have been reported. Muscle twitching usually occurred within minutes of injection with flaccid paralysis following (Roza and Berman 1971; Schott and McArdle 1974). Parenteral administration is a very rare route of barium exposure, but once barium has entered the bloodstream and has been systemically distributed, it will have the same effects on the same organ. Similar symptoms are expected to occur in humans acutely exposed to barium via inhalation and oral routes.

Barium induced smooth muscle contractions in a variety of in vitro mammalian systems (Antonio et al. 1973; Breuing et al. 1987; Clement 1981; Ebeigbe and Aloamaka 1987; Ehara and Inazawa 1980; Karaki et al. 1967; Mishra et al. 1988; Munch et al. 1980; Saeki et al. 1981; Saito et al. 1972; Slavicek 1972). Contraction appears to be calcium dependent (Antonio et al. 1973; Breuing et al. 1987; Clement 1981; Karaki et al. 1967; Saito et al. 1972), although the exact mechanism is unknown (Breuing et al. 1987; Clement 1981; Mishra et al. 1988).

Hepatic Effects. No reliable human or animal data were available regarding hepatic effects following inhalation or dermal exposure. Degeneration of the liver following acute oral exposure to barium has been noted in one human case report (McNally 1925). Increased liver/brain weight ratio and darkened liver were observed in rats following acute oral exposure to barium (Borzelleca et al. 1988). Decrease blood urea nitrogen, a potential sign of altered hepatic activity, was also noted in this study (Borzelleca et al. 1988). The available data are too limited to conclusively determine whether or not oral exposure to barium is associated with increased risk of hepatic effects in humans.

Renal Effects. No dermal studies evaluating renal effects in humans or animals were available. Renal failure was reported in one case study of a human exposed by acute inhalation to barium (Shankle and Keane 1988). Case studies of humans developing renal failure, renal insufficiency, and renal degeneration following acute oral barium poisoning have been reported (Gould et al. 1973; Lewi and Bar-Khayim 1964; McNally 1925; Phelan et al. 1984; Wetherill et al. 1981). Increased kidney/body weight ratio has been observed in rats following acute oral exposure to barium (Borzelleca et al. 1988). Renal effects have not been observed in intermediate or chronic oral studies with rats (Schroeder and Mitchener 1975a; Tardiff et al. 1980). Together, the findings from human case reports and animal studies suggest that individuals exposed to barium by acute inhalation or ingestion may be at increased risk of developing minor renal effects.

One in vitro study on rat renal tissue homogenate showed barium weakly inhibited the sodium-potassium-adenosine triphosphatase enzyme system (Kramer et al. 1986). A second study on mouse kidney tubules showed barium chloride could depolarize the membrane and inhibit potassium transport (Völkl et al. 1987). A similar defect in cell membrane transport in humans could be responsible for the renal involvement observed in some cases of acute barium poisoning.

Dermal/Ocular Effects. Few inhalation or dermal studies evaluating dermal/ocular effects in humans or animals are available. Results of one limited study suggested that barium carbonate was a dermal and ocular irritant when applied to the skin and eye of animals; however, it was not clear whether or not control animals were used (Tarasenko et al. 1977). In studies with Sprague-Dawley rats, both ocular discharge following acute oral exposure (Borzelleca et al. 1988) and nonsignificant increases in retinal dystrophy following intermediate and chronic oral exposure (McCauley et al. 1985) have been observed. Although the retinal dystrophy was not statistically significant, a dose-related trend was noted in several groups of rats if different duration exposure groups were combined. Both ocular discharge and retinal dystrophy are commonly observed in Sprague-Dawley rats; consequently, these ocular lesions cannot necessarily be attributed to oral barium exposure. Together, these results from animal studies provide unreliable information to draw firm conclusions about dermal/ocular effects in humans following barium exposure.

Other Systemic Effects. Other systemic effects have been observed. Barium sulfate was observed to act as an appendocolith in two cases following barium enema procedures (Palder and Dalessandri 1988). This is a rare occurrence and probably not significant in cases of human barium toxicity. Intravenous injection of barium sulfate into pigs increased calcitonin secretion from the thyroid (Pento 1979). This is probably not a significant effect for humans since intravenous exposure is not a common route and the dose required was so high (1.7 mg/kg/minute for 20 minutes) it caused cardiotoxicity.

Limited data are available on in vitro effects of barium on the endocrine system. Studies done with isolated pancreatic islet cells from mice show barium is transported across the cell membrane and incorporated into organelles, especially the mitochondria and secretory granules (Berggren et al. 1983). Barium was found to increase cytoplasmic calcium; consequently, the insulin-releasing action of barium may be mediated by calcium. Barium has also been found capable of stimulating the calcitonin secretion system of the thyroid in pigs (Pento 1979).

Immunological Effects. No information was available regarding immunotoxicity in humans following exposure to barium. Acute oral exposure of rats to barium failed to induce changes in thymus weight or gross or

microscopic lesions of the thymus (Borzelleca et al. 1988). Information from this study is too limited to draw any conclusions regarding relevance to human health.

An *in vitro* immunological study indicated that barium sulfate in low doses for relatively short periods posed no serious toxic hazard to phagocytic cells (Rae 1977).

Neurological Effects. No data were available regarding neurological effects in humans and/or animals following dermal exposure. One case study of a human accidentally exposed by acute inhalation to barium noted the absence of deep tendon reflexes (Shankle and Keane 1988). Case studies of humans having acute oral exposure to barium have reported such effects as numbness and tingling of the mouth and neck, partial and complete paralysis, and brain congestion and edema (Das and Singh 1970; Diengott et al. 1964; Gould et al. 1973; Lewi and Bar-Khayim 1964; McNally 1925; Morton 1945; Ogen et al. 1967; Phelan et al. 1984; Wetherill et al. 1981). Acute and intermediate oral exposure of rats to barium has not been associated with changes in brain weight or with gross or microscopic changes of the brain (Borzelleca et al. 1988; Tardiff et al. 1980). Based on the limited, but suggestive evidence from human case studies, there is the potential that individuals exposed by acute inhalation or acute oral exposure to barium may be at increased risk of developing neurological effects.

There are no cases of neurological effects in humans following parenteral exposure to barium compounds. In a few animal studies where barium chloride was injected intracerebroventricularly, insensitivity to pain occurred within minutes (Welch et al. 1983) followed by fatal convulsions if the dose was sufficient (Segreti et al. 1979; Welch et al. 1983). The significance of these data is difficult to assess since this unusual mode of entry would not occur in humans, and could be partially responsible for the rapid and extreme effects. Intraperitoneal injection of barium sulfate into mice produced an immediate increase in electroshock sensitivity followed by a decrease in sensitivity 24 hours later (Peyton and Borowitz 1978). These results are also difficult to assess in terms of effects observed in cases of human exposure, but suggest that barium in sufficient amounts may potentially influence brain function.

In most *in vitro* studies of nerve fibers, barium prolonged the action potential and caused rhythmic discharges (de No and Feng 1946; Greengard and Straub 1959). Barium released catecholamines in the absence of calcium both after nerve stimulation and in the absence of stimulation (Boullin 1965, 1967; Douglas and Rubin 1964a; Nakazato and Onoda 1980; Shanbaky et al. 1978). Barium also inhibited potassium flux in glial cells (Walz et al. 1984). These *in vitro* effects provide clues to the possible mechanism by which barium induces toxic effects on the cardiovascular and musculoskeletal systems. Barium had only a weak effect in blocking activation of spinal cord neurons by

excitatory amino acids (Ault et al. 1980). Barium was also taken up by mitochondria in bovine adrenal medulla (Shanbaky et al. 1982). These organelles therefore maybe more susceptible to the toxic effects of barium.

Developmental Effects. Little information is available regarding developmental effects in humans and/or animals following inhalation, oral, or dermal exposure to barium. One study reported reduced survival, underdevelopment, lowered weight, decreased lability of the peripheral nervous system, and various blood disorders in offspring of female rats exposed by intermediate inhalation to barium (Tarasenko et al. 1977). The same study also reportedly observed increased mortality, increased leukocyte count, disturbances in liver function, and increased urinary excretion of hippuric acid in offspring of female rats treated orally with barium during conception and pregnancy (Tarasenko et al. 1977). These studies are inadequate for evaluating the developmental effects of barium because of a number of significant study limitations (see Sections 2.2.1.2 and 2.2.2.5). In view of the major study limitations, and until verified by further tests, results from these studies should be regarded as providing only preliminary and/or suggestive evidence that inhalation and oral exposure to barium is potentially associated with adverse developmental effects.

Reproductive Effects. No studies were available regarding reproductive effects in humans following inhalation, oral, or dermal exposure. Disturbances in spermatogenesis, shortened estrous cycle, and alterations in the morphological structure of the ovaries and testes were reportedly observed in intermediate exposure experiments in which rats were treated by inhalation with barium carbonate dust (Tarasenko et al. 1977). However, these experiments suffered from a number of major limitations (see Section 2.2.1.2). Acute oral exposure of rats to barium has been associated with decreased ovary/brain weight ratio and decreased ovary weight (Borzelleca et al. 1988). These latter animal findings suggest that humans exposed orally to barium may be at increased risk of reproductive effects.

Genotoxic Effects. No data on in vivo studies of barium genotoxicity were available. In vitro studies were limited and primarily involve prokaryotic test systems. Tests of the fidelity of DNA synthesis using an avian myeloblastosis virus (AMV) DNA polymerase system showed that neither barium acetate nor barium chloride affect the accuracy of DNA replication (Sirover and Loeb 1976a; Sirover and Loeb 1976b). Barium chloride produced negative test results for its ability to inhibit growth in wild and recombination deficient strains of Bacillus subtilis. These results indicate that barium chloride is not mutagenic (Nishioka 1975). However, studies with a DNA polymerase I system from Micrococcus luteus, demonstrated that concentrations of barium ion less than or equal to 0.1 mM stimulated DNA polymerase activity while concentrations greater than this inhibited polymerase activity (Korman et al. 1978). The significance of the inhibitory and stimulatory effects has not been determined. Results from an experiment

designed to test the effect of barium chloride on sporulation frequency, recombination frequency, and meiotic failures in Saccharomyces cerevisiae demonstrated a definite inhibition of sporulation. Effects on recombination frequency and meiotic failures were ambiguous. Barium chloride may have caused a marginal increase in recombination frequency and information of diploid clones (Sora et al. 1986), but the data are inconclusive. The data available to date are insufficient to support a conclusive statement regarding the genotoxicity of barium and barium compounds.

Cancer. No adequate human studies were available that evaluated the carcinogenic potential of barium. Two chronic oral studies were available that examined the incidence of tumors in rats and mice exposed to barium acetate in drinking water for lifetime (Schroeder and Mitchener 1975a, 1975b). Although results of these oral studies were negative for carcinogenicity, they were inadequate for evaluating carcinogenic effects because insufficient numbers of animals were used, it was not determined whether or not a maximum tolerated dose was achieved, a complete histological examination was not performed, and only one exposure dose was evaluated. Precancerous lesions (dysplasia) were reported in one study in which a woman was treated on the cervix with a barium chloride solution; however, the relevance of this limited observation cannot be determined because only one subject was treated and because the vehicle solution was not specified (Ayre 1966). Results of one skin-painting study with mice suggest that barium hydroxide extract derived from tobacco leaf acted as a tumor-promoting agent; however, it cannot be determined whether or not this apparent positive tumorigenic response was due to barium hydroxide or some other component of the tobacco leaf extract (Van Duuren et al. 1968). Barium has not been evaluated by EPA for human carcinogenic potential (IRIS 1991).

TOXICOLOGICAL PROFILE FOR CHLOROBENZENE

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Toxicological
Profile
TP-90-06

Chlorobenzene

2.4 RELEVANCE TO PUBLIC HEALTH

Inhalation studies in humans and animals and oral studies in animals demonstrate that chlorobenzene can affect the central nervous system, liver, and kidneys. Chlorobenzene did not affect the developing fetus, was not genotoxic, and did not affect reproduction. Data has not provided clear evidence that chlorobenzene causes cancer in animals. Existing data are considered inadequate to derive human minimal risk levels for acute and chronic exposures.

Death. No case studies of human fatalities have been reported following exposure to chlorobenzene by inhalation, ingestion, or dermal contact. Death has been reported in animals at high doses for brief periods of exposure. Rabbits died within 2 weeks after removal from exposure at approximately 537 ppm (Rozenbaum et al. 1947). The cause of death has been attributed to central nervous system depression resulting in respiratory failure. Animal data suggest that lethality may not be a concern for humans unless the exposure level is very high.

2. HEALTH EFFECTS

Systemic Effects. No studies were located regarding effects on the respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, or dermal/ocular systems in humans or animals by any route of exposure to chlorobenzene.

Hepatic Effects. No studies were located demonstrating that chlorobenzene causes hepatic toxicity in humans by any route of exposure. Acute and intermediate exposures in animals demonstrated that chlorobenzene causes changes in liver weights and enzyme levels, degeneration, necrosis, and alterations in microsomal enzymes. These effects were first evident during acute exposure (5 days) at 1,140 mg/kg/day by gavage (Rimington and Ziegler 1963) and intermediate exposure (5 days/wk for 24 weeks) at 75 ppm via inhalation (Dilley 1977). Similar effects were also observed following ingestion of ≥ 250 mg chlorobenzene/kg/day for 91 days. The precise mechanism for liver damage is not known; however, direct binding of chlorobenzene metabolites to cellular protein may be involved (Reid et al. 1973; Reid and Krishna 1973). There were differential sensitivities in animal species tested which may be due to differences in metabolism. Based on animal studies, liver toxicity may be an area of concern in humans.

Renal Effects. No studies were located demonstrating that chlorobenzene causes renal effects in humans by any route of exposure. Intermediate studies in animals showed effects on the kidney at doses comparable to those causing liver effects. Typical signs included tubular degeneration and necrosis as well as changes in organ weight. Changes in organ weights with accompanying histopathology occurred at ≥ 250 mg/kg/day (90 days) (Kluwe et al. 1985). The precise mechanism of kidney damage is not clear. However, necrosis was associated with covalent binding of substantial amounts of radiolabeled chlorobenzene to kidney protein in intraperitoneal studies (Reid 1973). This study also reported that autoradiograms revealed that most of the covalently bound material was localized within necrotic tubular cells (Reid 1973). Based on animal studies, renal toxicity may be an area of concern in humans.

Immunological Effects. Histopathologic evaluations in animals suggest that chlorobenzene may be immunotoxic; however, direct tests of immune function have not been performed. In the absence of functional assessment, the potential for chlorobenzene to affect the immune system in humans can not be determined.

Neurological effects. Case reports of humans demonstrated that chlorobenzene caused disturbances of the central nervous system, but there were no reports of changes in the structure of the brain and other parts of the nervous system. Effects were observed in humans who inhaled vapors of chlorobenzene in the workplace for up to 2 years (Rozenbaum et al. 1947). Effects included headaches, dizziness, and

sleepiness. Unconsciousness, lack of response to skin stimuli, and muscle spasms were noted following accidental ingestion. While there is qualitative evidence for central nervous system effects in humans, a quantitative assessment can not be made since exposure levels were not reported. Because work practices have changed significantly since these studies, it is reasonable to assume that exposure levels in this study were higher than current permissible federal exposure levels. Acute studies in animals confirm that chlorobenzene is potentially neurotoxic. These effects appear to be the result of narcotic effects of chlorobenzene on the central nervous system. Acute inhalation exposure produced narcosis preceded by muscle spasms in rabbits at 1,090 ppm (Rozenbaum et al. 1947).

Developmental Effects. No studies were found regarding the developmental toxicity of chlorobenzene in humans. In inhalation and oral exposure studies, the animals did not demonstrate significant developmental toxicity when compared with untreated controls. Negative responses in two animal species suggest that developmental toxicity may not be an area of concern for chlorobenzene.

Reproductive Effects. No studies were found regarding the reproductive toxicity of chlorobenzene in humans. In a two-generation inhalation study, chlorobenzene did not adversely affect various reproductive parameters in rats (Nair et al. 1987). While results of this study suggest reproductive toxicity may not be an area of concern to humans, other considerations are warranted before firm conclusions can be made regarding risk to humans. The slight increase in the occurrence of degeneration of the germinal epithelium of the testes provides some evidence for further consideration. Also, the study did not provide histopathological data on other organs related to reproductive functions (i.e., epididymis, vas deferens, accessory sex glands, and pituitary). While the authors reported no treatment-related impairment of fertility, it should be noted that fertility assessments in test animals are limited by their insensitivity as measures of reproductive injury in humans.

Genotoxic Effects. No studies were located regarding the genotoxic effects of chlorobenzene in humans. No *in vivo* animal assays were found, except the micronuclear test in mice which was moderately positive (Mohtashamipur et al. 1987) (Table 2-3). Furthermore, *in vitro* tests employing bacterial and yeast assay systems with and without metabolic activation were negative (Haworth et al. 1983; NTP 1985; Prasad 1970). Chlorobenzene induced transformation in adult rat liver epithelial cells but was not genotoxic to hepatocytes (Shimada et al. 1983). Since transformations may occur through nongenotoxic mechanisms, results do not necessarily indicate that chlorobenzene is potentially genotoxic. Results of *in vitro* assays for chlorobenzene are presented

in Table 2-4. Existing data suggest that genotoxicity may not be an area of concern for chlorobenzene exposure in humans.

Cancer. No studies were found regarding the carcinogenicity of chlorobenzene in humans. In a chronic bioassay in animals, chlorobenzene (up to 120 mg/kg/day) did not produce increased tumor incidences in mice of both sexes or in female rats (NTP 1985). It was noted, however, that male rats showed a statistically significant increase in neoplastic nodules at the highest dose level tested. While there is strong evidence for neoplastic nodules, existing data are inadequate to characterize the potential for chlorobenzene to cause cancer in humans and animals.

TOXICOLOGICAL PROFILE FOR COBALT

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2.4 RELEVANCE TO PUBLIC HEALTH

Cobalt has been found to produce adverse effects by the inhalation, oral, and dermal routes. Effects in humans following inhalation exposure to cobalt included lung effects (respiratory irritation, fibrosis, asthma, pneumonia, wheezing), cardiovascular effects (cardiomyopathy), liver and kidney congestion, ocular effects (congestion of the conjunctiva), and weight loss. Effects in humans observed following ingestion of cobalt, as cobalt sulfate in beer or as cobalt chloride as a treatment for anemia, included cardiomyopathy, gastrointestinal effects, visual disturbances, and thyroid effects. Cobalt dermatitis and sensitization as a result of dermal exposure to cobalt are well documented.

Cobalt is essential for the growth and development of ruminants (Becker and Smith 1951; Keener et al. 1949). Overexposure of other animals to cobalt resulted in effects similar to those in humans following inhalation and dermal exposure. After ingestion of cobalt, effects in animals were similar to effects in humans although some additional effects, including hypothermia, neurological effects (effects on reactivity), developmental effects (stunted fetuses), and reproductive effects (testicular degeneration and atrophy), were found in animals, but not humans. In the animal studies, the doses tested were higher than the levels to which humans would be expected to be exposed.

An MRL for inhalation exposure to cobalt was derived for intermediate duration only. The intermediate inhalation MRL of $3x10^{-5}$ mg cobalt/m^3 was based on a LOAEL of 0.11 mg cobalt/m^3 for metaplasia of the larynx in rats exposed for 13 weeks (6 hours/day, 5 days/week) (NTP 1991). Similar effects were reported in mice exposed to the same concentration, but the rats appeared to be more sensitive. NOAEL values were not defined for the rats or the mice. A decrease in lung compliance was reported in pigs exposed to a comparable level of cobalt for 3 months (0.1 mg cobalt/m^3 as cobalt dust, 6 hours/day, 5 days/week) (Kerfoot 1973).

The intermediate inhalation MRL was derived by adjusting the LOAEL of 0.11 mg cobalt/m^3 for intermittent exposure (6 hours/24 hours x 5 days/7 days), converting to an equivalent human dose, and dividing by an

uncertainty factor of 1,000 (10 for the use of a LOAEL, 10 for human variability, and 10 for interspecies extrapolation). An acute inhalation MRL was not derived because the threshold was not defined for human effects and animal studies reported effects that were serious and occurred at levels above those reported in the few human studies. A chronic inhalation MRL was not derived because the lowest LOAEL is human sensitization and ATSDR does not derive MRLs based on health effects such as sensitization.

Oral MRL values were not derived for acute, intermediate or chronic exposure to cobalt. Acute and intermediate MRLs were not derived because the reported effects in animals were serious and occurred at levels above those reported in the few human oral studies. The intermediate-duration human oral studies were also insufficient for derivation of MRL values because the reported effects were serious. No chronic oral studies were available for humans or animals, therefore, a chronic oral MRL was not derived for cobalt.

Acute-duration, intermediate-duration, and chronic-duration dermal MRLs were not derived for cobalt due to the lack of appropriate methodology for the development of dermal MRLs.

Death. Lethal cardiomyopathy in humans was reported following repeated inhalation of airborne cobalt or ingestion of beer that contained cobalt (Alexander 1969, 1972; Barborik and Dusek 1972; Morin et al. 1971). Inhalation exposure levels associated with cardiomyopathy have not been determined. In the 1960s, breweries in the United States, Canada, and Europe added cobalt salts to beer to improve foaming properties at the tap. Several deaths occurred among heavy beer drinkers who consumed beer containing 0.04-0.14 mg cobalt/kg/day (8-30 pints of beer daily). The addition of cobalt to beer has since been discontinued. Although the ingestion of cobalt was identified as a key causative factor in the beer drinkers cardiomyopathy, other etiologic factors were significant, including heavy alcohol consumption and related nutritional deficits. Repeated oral ingestion of 1 mg cobalt/kg/day to raise the hematocrit of anemic, but otherwise healthy, patients did not cause cardiac injury (Davis and Fields 1958).

In animals, deaths from inhalation exposure were related to respiratory effects and secondary infections (NTP 1991; Palmes et al. 1959). Deaths in animals following oral exposure resulted from cardiomyopathy (Mohiuddin et al. 1970) or from multiple lesions (kidney, liver, and heart lesions) (Speijers et al. 1982). Acute lethality in animals varies with the chemical form administered, with soluble compounds generally being more toxic than insoluble compounds. In rats, cobalt fluoride is more toxic than cobalt chloride by a factor of two (Speijers et al. 1982). With the exception of tricobalt tetroxide, the LD_{50} values of the cobalt compounds for which acute oral lethality data are available (Table 2-2), all lie within the same order of magnitude when expressed in terms of the cobalt ion. Tricobalt tetroxide (LD_{50} >3,672 mg cobalt/kg) is insoluble in water and, therefore, is relatively nontoxic (FDRL 1984c).

Systemic Effects. The primary target organ systems for the effects of cobalt in humans are the respiratory system following inhalation exposure and the cardiac and hematopoietic systems following oral exposure.

Respiratory. Effects on the respiratory system include irritation, fibrosis, asthma, pneumonia, and wheezing following inhalation exposure (Hartung et al. 1982; Kusaka et al. 1986b; Shirakawa et al. 1988, 1989; Sprince et al. 1988). Individuals can develop a sensitivity to cobalt, and inhalation exposure to airborne cobalt can precipitate asthmatic attacks in sensitized individuals (Shirakawa et al. 1988, 1989). Studies in animals report similar effects following inhalation exposure. Intermediate-duration inhalation studies in rats and mice reported that the larynx was the part of the respiratory tract most sensitive to the effects of cobalt, with the lungs, nose, and trachea being affected at higher exposure levels (NTP 1991).

Cardiovascular. In humans, lethal cardiomyopathy resulted from oral and inhalation exposure to cobalt. Along with the severe cardiac effects, beer-cobalt cardiomyopathy was characterized by initial effects on the gastrointestinal system (vomiting, nausea, diarrhea), pulmonary rales and edema (resulting from the cardiac failure), liver injury (resulting from hepatic ischemia), and polycythemia (Alexander 1969, 1972; Morin et al. 1971). Beer-cobalt cardiomyopathy was similar to both alcoholic cardiomyopathy and beriberi, except that beer-cobalt cardiomyopathy had an abrupt onset, characterized by left ventricular failure, cardiogenic shock, polycythemia, and acidosis. Evidence that ingestion of ethanol was not required for development of cobalt cardiomyopathy came from studies in animals. A cardiomyopathy similar to that observed in humans occurred in guinea pigs after repeated exposure to cobalt (20 mg/kg/day) in foods, with or without ethanol consumption (Mohiuddin et al. 1970).

Gastrointestinal. Gastrointestinal effects, including nausea, vomiting, and diarrhea, were reported in humans after ingestion of cobalt-contaminated beer (Morin et al. 1971) and treatment with cobalt for anemia (Duckham and Lee 1976; Holly 1955). No effects on the gastrointestinal system, however, were reported in animals after inhalation or oral exposure (Domingo et al. 1984; Holly 1955; NTP 1991). The data in humans suggest that therapeutic exposure to cobalt is likely to result in gastrointestinal effects.

Hematopoietic. Because cobalt induces polycythemia in humans following oral exposure, it has been used in the treatment of anemia (Davis and Fields 1958; Duckham and Lee 1976; Taylor et al. 1977). Polycythemia was not observed in humans following inhalation exposure. Animal data show increased hematocrit and hemoglobin levels following both oral and inhalation exposure

(Brewer 1940; Davis and Fields 1958; Domingo and Llobet 1984; Domingo et al. 1984; Holly 1955; Krasovskii and Fridlyand 1971; Murdock 1959; NTP 1991; Palmes et al. 1959; Stanley et al. 1947). The increase in hematocrit in both the humans and animals does not necessarily constitute an adverse effect.

A possible mechanism of cobalt toxicity is the irreversible formation of a cobalt chelate with the sulfhydryl groups of dihydrolipoic acid, resulting in inhibition of two conversions in the tricarboxylic acid cycle (pyruvate to acetyl CoA and alpha-ketoglutarate to succinyl-CoA) (Taylor and Marks 1978). The resulting inhibition of cellular respiration and oxidative phosphorylation may lead to decreased oxygen uptake by the tissues, resulting in cardiac effects. Tissue hypoxia may in turn stimulate the release of erythropoietin, resulting in increased hematocrit and hemoglobin levels (Taylor and Marks 1978).

Hepatic. No conclusive evidence that cobalt is a direct liver toxicant in humans has been reported following exposure to low levels; however, liver injury has been associated with cobalt-related cardiomyopathy following either inhalation or oral exposure (Alexander 1972; Barborik and Dusek 1972; Morin et al. 1971). Although the mechanism for the liver effects is not known, it is likely that hepatic ischemia related to cardiovascular impairment is a significant causative factor. Liver injury was observed in animals orally exposed to near lethal levels of cobalt (Speijers et al. 1982). Whether this represents a direct effect of cobalt on the liver or an indirect effect of cardiac impairment is not known. A direct effect of cobalt on the liver is plausible since the liver is the major site of accumulation of orally absorbed cobalt.

Renal. No conclusive evidence that cobalt is a kidney toxicant in humans has been reported. Congestion of the kidneys, however, has been associated with cobalt cardiomyopathy resulting from occupational exposure to cobalt (Barborik and Dusek 1972). Effects on the proximal tubules of the kidneys were observed in animals orally exposed to cobalt (Holly 1955; Murdock 1959; Speijers et al. 1982). Adverse effects on the kidneys of both humans and animals are a possibility because a substantial amount of cobalt absorbed into the blood is excreted in the urine.

Dermal/Ocular. Effects on the human eye have been observed following occupational exposure (congestion of the conjunctiva) and oral exposure (optic atrophy, impaired choroidal perfusion) to cobalt (Barborik and Dusek 1972; Licht et al. 1972). Occupational exposure to cobalt also causes dermatitis (Alomar et al. 1985; Dooms-Goossens et al. 1980; Kanerva et al. 1988).

Other Systemic Effects. A decrease in iodine uptake by the thyroid resulted from acute oral exposure of humans to 1 mg cobalt/kg/day or longer-term exposure to 0.54 mg/kg/day (Paley et al. 1958; Roche and Layrisse 1956).

Weight loss was found in workers occupationally exposed to cobalt. Similar weight loss was seen in animals; in addition, time- and dose-related hypothermia was observed in rats given cobalt orally.

In various species of animals, parenteral administration of cobalt resulted in cytotoxic effects on the alpha cells of the pancreas (Beskid 1963; Goldner et al. 1952; Lacy and Cardeza 1958; Lazarus et al. 1953; Van Campenhout 1955). Because this effect has never been reported in humans or animals following inhalation, oral, or dermal exposure to cobalt, the relevance of the effect to humans is not known.

Immunological Effects. Exposure to cobalt can lead to sensitization. In its most serious form, cobalt-sensitization can result in or exacerbate asthma (Shirakawa et al. 1988, 1989). Dermal sensitization and related cobalt-dermatitis have also been described. The mechanism for cobalt sensitization is not completely understood. Antibodies to cobalt have been detected in individuals sensitized to cobalt, suggesting that a humoral immune response may be a component of the sensitization phenomenon (Bencko et al. 1983; Shirakawa et al. 1988, 1989).

Neurological Effects. No studies were located regarding neurological effects in humans following inhalation, oral, or dermal exposure to cobalt. Enhanced behavioral reactivity to stress, a slower rate of lever pushing, and effects on conditioned reflexes were observed in rats orally exposed to cobalt (Bourg et al. 1985; Krasovskii and Fridlyand 1971; Nation et al. 1983). The relevance of these findings to humans is not known. In rats, cobalt applied directly to the brain has been found to induce epilepsy and has been used extensively as a model toward a better understanding of epilepsy in humans (Bernstein and Keilhoff 1986; Bregman et al. 1985; Esclapez and Trottier 1989; Hartman et al. 1974; Hocherman and Reichenthal 1983; Lee and Malpeli 1986; Pavan and Conard 1974; Pitkanen et al. 1987; Sugaya et al. 1988; Zhao et al. 1985).

Developmental Effects. No obvious developmental effects were observed in human fetuses from mothers who were given cobalt orally to counteract decreases in hematocrit and hemoglobin levels that often occur during pregnancy (Holly 1955). No studies were located regarding developmental effects in humans following inhalation or dermal exposure. Animal studies, however, reported that oral exposure to cobalt results in developmental effects including stunted fetuses, a decrease in the number of litters and average litter weights, and an increase in the number of dead pups per litter (Domingo et al. 1985). Toxic maternal effects were also observed in this study. The relevance of the effects found in animals to possible human effects is not known.

Reproductive Effects. No studies were located regarding reproductive effects in humans following inhalation, oral, or dermal exposure to cobalt.

Following both inhalation and oral exposure of animals to cobalt, adverse effects on the testes were observed (degeneration, atrophy, decreased weight) (Corrier et al. 1985; Domingo et al. 1985; Mollenhauer et al. 1985; NTP 1991; Pedigo et al. 1988). An increase in the length of the estrous cycle was also reported in female mice following inhalation exposure (NTP 1991). Because no effects on the reproductive system were found in patients who died as a result of beer-cobalt cardiomyopathy, the significance of the animal results to humans is not clear.

Genotoxic Effects. No studies were located regarding genotoxic effects in humans or animals following inhalation, oral, or dermal exposure to cobalt.

Results of genetic testing of cobalt are presented in Table 2-8. Several different forms of cobalt, including cobalt chloride and cobalt sulfide, were tested. No profound differences were found among the various forms. Cobalt was found to be generally nonmutagenic in bacteria (Salmonella typhimurium, Escherichia coli) and yeast when compounds with a valence state of II were tested (Arlauskas et al. 1985; Fukunaga et al. 1982; Kanematsu et al. 1980; Kharab and Singh 1985; Ogawa et al. 1986; Singh 1983; Tso and Fung 1981). A very weak mutagenic response was found with Bacillus subtilis (Kanematsu et al. 1980). A mutagenic response to cobalt was found, however, when compounds with a valence state of III were tested in S. typhimurium and E. coli (Schultz et al. 1982). The authors suggested that this may be due to the formation of cobalt III complexes that are inert to ligand substitution, allowing optimal interaction of cobalt with genetic material (Schultz et al. 1982). Other studies have shown cobalt to be a comutagen in combination with 4-substituted pyridines in S. typhimurium (Ogawa et al. 1988). It has been reported that cobalt acts as an antimutagen in bacterial (S. typhimurium, B. subtilis, E. coli) and yeast test systems (S. cerevisiae) (Inoue et al. 1981; Kada 1982; Kada et al. 1986; Kuroda and Inoue 1988; Mochizuki and Sora et al. 1986). A possible explanation was that cobalt acts by correcting the error-proneness of deoxyribonucleic acid (DNA) replicating enzymes by improving their performance in DNA synthesis (Inoue et al. 1981; Kada et al. 1986; Kuroda and Inoue 1988; Mochizuki and Kada 1982). Stable cobalt was genotoxic in other assay systems: genetic conversions in S. cerevisiae (Funkunaga et al. 1982; Kharab and Singh 1985; Singh 1983); clastogenic effects in mammalian cells (Hamilton-Koch et al. 1986; Painter and Howard 1982); transformation in hamster cells (Costa et al. 1982); and sister chromatid exchanges in human lymphocytes (Andersen 1983).

Cancer. Cobalt has not been shown to cause cancer in humans by any exposure route. An occupational study reported an increased incidence of death from lung cancer (SMR=4.66) in workers exposed to cobalt (Mur et al. 1987), but the difference was not statistically significant; the characteristic lung diseases commonly found in cobalt workers were not observed, and the workers were exposed to arsenic and nickel as well as cobalt. The induction of tumors (fibrosarcomas) following intramuscular

injection of cobalt oxide into rats has been shown (Gilman 1962; Gilman and Ruckerbauer 1962; Heath 1956, 1960). No tumors were induced in mice after intramuscular injection of cobalt (Gilman 1962; Gilman and Ruckerbauer 1962). Tumors were also induced following subcutaneous (Shabaan et al. 1977) and intrathoracic injections in rats (Heath and Daniel 1962). The significance of these results to humans is not clear because these are not relevant routes of exposure and no tumors were found in humans with metal-alloy prostheses. IARC (1991), however, has classified cobalt and cobalt compounds as group 2B, possible human carcinogens.

TOXICOLOGICAL PROFILE FOR COPPER

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Toxicological Profile

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Copper

2.4 RELEVANCE TO PUBLIC HEALTH

Copper is a metallic element that occurs naturally as the free metal. Most copper compounds occur in +1 (Cu(I)) and +2 (Cu(II)) valence states. Although copper is present as numerous chemical species, the biological availability and toxicity of copper is probably related to free Cu(II) ion activity. In this section, the term copper refers to Cu(II).

Copper is an essential nutrient that is incorporated into numerous enzymes. These enzymes are involved in hemoglobin formation, carbohydrate metabolism, catecholamine biosynthesis, and cross-linking of collagen, elastin, and hair keratin. There are numerous copper dependent enzymes, some of which are cytochrome c oxidase, superoxide dismutase, dopamine β-hydroxylase, and ascorbic acid oxidase.

Copper homeostasis plays an important role in the prevention of copper toxicity. Copper is readily absorbed from the stomach and small intestine; after copper requirements are met, there are several mechanisms that prevent copper overload. Excess copper absorbed into gastrointestinal mucosal cells is bound to metallothionein. This bound copper is excreted when the cell is sloughed off. Copper that eludes the intestinal barrier can be stored in the liver or incorporated into bile and excreted in the feces. Because of the body's efficient means of blocking the absorption of excess copper, the most likely pathway for the entry of toxic amounts of copper would be long-term inhalation or possibly through the skin. Both of these pathways would allow copper to pass unimpeded into blood.

There is little information on copper toxicity in man. Most reports of copper toxicity in humans involve the consumption of water contaminated with high levels of copper or suicide attempts using copper sulfate. Effects observed in humans include gastrointestinal, hepatic, immunological (following dermal exposure), and respiratory effects (following inhalation of high concentrations of fine copper metal particles). Death as a result of copper toxicity has also been observed. Because the taste threshold of copper, 2.6 ppm (Cohen et al. 1960), is much lower than the levels associated with toxicity and the emetic properties of copper, accidental poisoning can be prevented.

The only significant example of copper toxicity in humans is Wilson's disease (hepatolenticular degeneration), an autosomal recessive disorder that affects normal copper homeostasis. The disease is characterized by excessive retention of hepatic copper, decreased concentration of plasma

ceruloplasmin, impaired biliary copper excretion, and hypercupruria. The systemic manifestations of Wilson's disease are hepatic and renal lesions and hemolytic anemia (Schroeder et al. 1966).

The effects observed in humans following exposure to high levels of copper and in individuals with Wilson's disease are also observed in animals. Developmental and reproductive effects are also observed in animals; these effects have not been reported in humans.

Death. There are several reports of humans dying as a result of copper poisoning. Most of these involve the intentional ingestion of large amounts of copper. Chuttani et al. (1965) attributed these deaths to extensive hepatic centrilobular necrosis. Deaths in animals given >250 mg Cu/kg/day in the diet have also been attributed to extensive hepatic centrilobular necrosis.

Systemic Effects. The primary toxicological effect of consuming high levels of copper in humans is gastrointestinal irritation, manifested as vomiting, nausea, diarrhea, and anorexia. Centrilobular necrosis of the liver and necrosis and sloughing of tubular cells in the kidney have been observed in individuals dying from copper poisoning (Chuttani et al. 1965).

Gastrointestinal effects (hyperplasia in forestomach) have been observed in animals following exposure to large levels of copper in the drinking water or food. Centrilobular necrosis and extensive degeneration of the proximal convoluted tubule epithelium have also been observed in rats. These effects are followed by regeneration of the tissue and development of tolerance to continued dosing. Tolerance is defined as a state of decreased responsiveness to a chemical's toxic effect, resulting from prior exposure to the chemical. There appears to be an upper limit to the amount of copper that can be tolerated. In rats, the limit is ≈250 mg Cu/kg/d (Haywood 1985). Tolerance has also been observed in pigs (Suttle and Mills 1966a,b).

The mechanisms of tolerance are not known but may involve changes in the distribution and molecular association of copper. Tolerance apparently represents an adjustment of homeostasis: rather than copper being stored in the liver and excreted in bile, it is released from the liver and excreted into the urine (Haywood et al. 1985b). It is not known if humans develop a tolerance to copper.

Hematological effects have been observed in healthy humans exposed to high levels of copper and in individuals with Wilson's disease (Forman and Kumar 1980). A decrease in hemoglobin and hematocrit values has also been reported in rats and pigs orally exposed to copper (Kline et al. 1971; Kumar and Sharma 1987; NTP 1990a,b; Rana and Kumar 1980; Suttle and Mills 1966a,b). An increase in hemoglobin levels without a change in hematocrit levels was also observed (Liu and Medeiros 1986). The Liu and Medeiros (1986) study was a longer term study than the studies that observed

decreased hemoglobin and hematocrit levels (Kline et al. 1971; Kumar and Sharma 1987; NTP 1990a,b; Rana and Kumar 1980; Suttle and Mills 1966a,b). The discrepancy between the hematological effects observed in these shorter term studies and the Liu and Medeiros (1986) study may represent a tolerance to high copper intake.

A statistically significant increase in systolic blood pressure has been observed in rats fed a diet containing a moderately high level of copper. Because this is the only study that examined the cardiovascular effects associated with increased copper intake, the risk of increased blood pressure in humans following exposure to high levels of copper is not known.

Immunological Effects. Dermal exposure to copper results in contact allergic dermatitis in some individuals (Barranco 1972; Saltzer and Wilson 1968). A report of a similar dermatitis in a woman after the insertion of a copper IUD (Barranco 1972) suggests that the dermatitis is mediated by the immune system rather than dermal irritation.

Inhalation studies in mice confirm the finding of impaired immune function after exposure to copper (Drummond et al. 1986). However, species apparently differ in effects of inhaled copper on the immune system.

Neurological Effects. Neurological effects have not been observed in healthy humans exposed to high levels of copper in drinking water. However, a clinical manifestation of Wilson's disease is central nervous system degenerative changes. Symptoms include poor coordination, psychological impairment, tremor, disturbed gait, and rigidity (Strickland and Leu 1975). Increased copper levels in the brain have also been observed in individuals with Wilson's disease (Stokinger 1981). In a child who died of Wilson's disease, increased norepinephrine and decreased dopamine concentrations in the basal ganglia were observed (Nyberg et al. 1982).

Although neurotoxicity has not been observed in animals exposed to high levels of copper, increased concentration of copper in the brain has been observed in rats given high levels of copper orally or intraperitoneally (DeVries et al. 1986; Lal et al. 1974; Murthy et al. 1981). The effects of the ingestion of high levels of copper on levels of neurotransmitters is equivocal (DeVries et al. 1986; Lal et al. 1974; Murthy et al. 1981). Because of the development of tolerance to copper toxicity, brain copper levels may not reach a level that would interfere with the synthesis or degradation of neurotransmitters or elicit neurological impairment.

If humans, like rats and pigs, develop a tolerance to high levels of copper, then central nervous system degenerative changes would not be an outcome of copper toxicity.

Developmental Effects. Developmental effects have not been observed in healthy humans or in the offspring of mothers with Wilson's disease, whereas

increased fetal mortality and developmental abnormalities have been observed in mice, mink, and hamsters injected with copper or fed a diet high in copper (Aulerich et al. 1982; DiCarlo 1979, 1980; Ferm and Hanlon 1974; Lecyk 1980; O'Shea and Kaufman 1979, 1980). Developmental effects have been observed in minks administered doses 50 times lower than those given to mice and hamsters. Increased kit mortality was observed between birth and 4 weeks due to impaired lactation in females consuming high levels of copper. Maternal toxicity was not observed at this level.

Developmental effects have not been reported in humans. Because of the animal developmental toxicity, the possibility that developmental effects might occur in humans cannot be ruled out.

Reproductive Effects. Reproductive effects following exposure to high levels of copper have not been observed in humans. However, intrauterine devices (IUDs) have been used in women as a method of birth control. Although reproductive performance was not adversely affected in minks fed a diet high in copper (Aulerich et al. 1982), the insertion of copper wires into the vas deferens or uterus prior to conception or at gestational day 3 resulted in decreased fertility or decreased number of implantation sites in monkeys, rats, hamsters, and rabbits (Chang and Tatum 1970; Chang et al. 1970; Kapur et al. 1984; Zipper et al. 1969). No adverse effects were observed in control animals in which wires of cadmium, cobalt, nickel, platinum, silver, or zinc were implanted *in utero* (Chang et al. 1970; Zipper et al. 1969), suggesting that the copper, rather than the insertion of an exogenous object, is the contraceptive agent.

Genotoxicity Effects. Several *in vitro* studies (Table 2-4) have examined genotoxic effects of copper in nonhuman systems. The results of the tests using prokaryotic organisms are equivocal. However, positive results have been observed *in vitro* (Table 2-4) and *in vivo* (Table 2-5) in mammalian systems. At low levels of copper (0.01-0.1 mM Cu as copper sulfate), DNA strand breaks were not observed in rat hepatocytes; however, strand breaks did occur at high concentrations (0.04 mM Cu as copper sulfate) (Sina et al. 1983). *In vivo* studies with Inbred Swiss mice showed that copper exposure resulted in chromosomal aberrations and micronuclei and sperm abnormalities (Bhunya and Pati 1987). Copper binds with the phosphate on nucleotides and nucleic acids of DNA. Its mutagenic potential may be the result of this binding (Sharma and Talukder 1987).

Although there is no data on the mutagenicity of copper in humans, *in vivo* studies and mammalian system *in vitro* studies suggest that copper is a potential human mutagen.

Cancer. An elevated incidence of cancer has not been observed in humans or animals exposed to copper via inhalation, oral, or dermal routes of exposure. Copper exposure via intramuscular injection has not been shown to induce cancer in rats (Furst 1971; Gilman 1962). A slightly increased incidence of reticulum cell sarcoma was observed in mice 18 months after a

single subcutaneous injection of copper 8-hydroxyquinoline (BRL 1968). The significance of this finding is difficult to determine because of the contrary findings of Yamane et al. (1984) and because subcutaneous injection is not a normal route of copper exposure.

TOXICOLOGICAL PROFILE FOR 1,1-DICHLOROETHANE

Prepared by:

Clement International Corporation
Under Contract No. 205-88-0608

Prepared for:

Agency for Toxic Substances and Disease Registry
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Toxicological Profile TP-90-12

1, 1-dichloroethane

2.4 RELEVANCE TO PUBLIC HEALTH

Relatively little information is available on the health effects of 1,1-dichloroethane in humans or animals. However, the limited data available in animals indicate that it is less toxic than its isomer, 1,2-dichloroethane, and most other chlorinated aliphatics (Bruckner 1989). Chlorinated aliphatics as a class are known to cause central nervous system depression, and respiratory tract and dermal irritation when humans are exposed by inhalation to sufficiently high levels (Parker et al. 1979).

The available data in animals suggest that inhaled 1,1-dichloroethane may be nephrotoxic. However, this finding is limited to one species (cat) and was not observed in three other species tested under the same conditions. Another effect observed in animals but not humans following inhalation exposure to 1,1-dichloroethane exposure is fetotoxicity. Suggestive, but inconclusive, evidence of carcinogenicity was obtained in an oral chronic bioassay of 1,1-dichloroethane in rats and mice.

Death. No reports of death in humans following exposure to 1,1-dichloroethane were found. Death has been observed in laboratory animals following inhalation and oral exposure to 1,1-dichloroethane. No reliable LC_{50} or LD_{50} data were found, but lethal doses of 1,1-dichloroethane are perhaps 5 to 10 times higher than those required to produce death following exposure to 1,2-dichloroethane or tetrachlorocarbons (EPA 1985; Hofmann et

al. 1971; Smyth 1956). Thus, it is likely that 1,1-dichloroethane can be fatal to humans, if exposure to high enough levels occurs.

The cause of death in animals following exposure to 1,1-dichloroethane has not been well-defined, but Plaa and Larson (1965) reported that deaths observed following intraperitoneal injection of this compound appeared to be due to fatal central nervous system depression.

Systemic Effects. The use of 1,1-dichloroethane as an anesthetic was discontinued when it was discovered that this compound induced cardiac arrhythmias in humans at anesthetic doses (approximately 105,000 mg/m^3, or 26,000 ppm). The mechanism of action for the induction of cardiac arrhythmias by 1,1-dichloroethane is not known. However, when the cardiac muscle is markedly depressed, it is more susceptible to the effects of catecholamines. Secretion of catecholamines is increased in this situation by compensatory and other mechanisms, resulting in excessive spontaneous contractions of the heart. This is an effect common to exposure to other chlorinated aliphatics at high concentrations (Reinhardt et al. 1971). Cardiovascular toxicity has not been reported in animals following exposure to 1,1-dichloroethane.

No reports of adverse renal effects in humans following exposure to 1,1-dichloroethane were found. Nephrotoxicity has been observed in cats following subchronic inhalation exposure to 1,1-dichloroethane. However, rats, rabbits, and guinea pigs exposed under the same conditions failed to exhibit any toxic effects on the kidney (Hofmann et al. 1971). Plaa and Larson (1965) tested renal function in mice following intraperitoneal injection of 1,1-dichloroethane, and found that adverse effects on the kidney were only observed at lethal doses. These effects included increased glucose and protein in the urine and tubular swelling. Though data obtained following intraperitoneal injection provides information on potential health effects, data from oral, inhalation and dermal experiments are more relevant to possible exposures in humans. No histopathological changes in the kidney were noted after chronic ingestion of 1,1-dichloroethane by rats and mice (Klaunig et al. 1986; NCI 1977). The toxicological significance of the nephrotoxicity observed in cats and the mice with regard to human health is not known given the small number of animals tested (cats), the lack of a nephrotoxic effect in other species and in other studies where 1,1-dichloroethane was administered orally, and the fact that nephrotoxicity is not an effect commonly attributed to the halogenated hydrocarbons.

Immunological Effects. No studies were located regarding immunologic effects in humans or animals following exposure to 1,1-dichloroethane, and it is not known if 1,1-dichloroethane is immunotoxic in humans.

Neurological Effects. Chlorinated aliphatics as a class are known to cause central nervous system depression following high-level exposure in humans and animals. No reliable dose-response data were found on the central

nervous system depression induced by 1,1-dichloroethane, though 1,1-dichloroethane was once used as an anesthetic agent in humans. However, Plaa and Larson (1965) attributed deaths observed in mice following intraperitoneal injection to fatal central nervous system depression. Neurologic effects associated with long-term exposure to 1,1-dichloroethane in humans or animals have not been reported.

Developmental Effects. Adverse developmental effects in humans associated with exposure to 1,1-dichloroethane have not been reported. One study in rats indicated that inhalation exposure to 1,1-dichloroethane resulted in retarded fetal development (delayed ossification of vertebrae) in the absence of significant maternal toxicity (Schwetz et al. 1974). The absence of maternal toxicity implies a direct effect on the fetus, rather than effects due to illness in the dam. The implications of the findings from one study with regard to potential developmental effects in humans are not known.

Reproductive Effects. No studies were located regarding reproductive effects in humans or animals following exposure to 1,1-dichloroethane, and it is not known if 1,1-dichloroethane has the potential to cause adverse reproductive effects in humans.

Genotoxic Effects. No studies were located regarding *in vivo* genotoxic effects in humans. The genotoxic potential of 1,1-dichloroethane has been investigated *in vitro* in *Salmonella typhimurium* (Riccio et al. 1983; Simmon et al. 1977), *Saccharomyces cerevisiae* (Bronzetti et al. 1987; Simmon et al. 1977), and Syrian hamster embryo cells (Hatch et al. 1983). In addition *in vitro* and *in vivo* assays have been conducted using rat and mouse organs (Colacci et al. 1985). Results of these studies are summarized in Table 2-5. Results from three studies conducted in *S. typhimurium* tester strains were conflicting. 1,1-Dichloroethane was nonmutagenic in yeast cells even in the presence of metabolic activation system. However, because of insufficient reporting of data by Bronzetti et al. (1987) and Simmon et al. (1977), no assessment of the genotoxic potential of 1,1-dichloroethane in *S. cerevisiae* can be made. The available data from the remaining studies indicate that, although 1,1-dichloroethane did not induce cell transformation in BALB/c-3T3 cells (Tu et al. 1985), it increased the frequency of transformations induced by Simian adenovirus (SA7) in hamster embryo cells (Hatch et al. 1983).

In the Ames assay, 1,1-dichloroethane was nonmutagenic in *Salmonella* strains TA97, TA98, TA100, and TA102 (Nohmi et al. 1985). The compound was tested with and without metabolic activation. The highest dose was toxic to all strains of bacteria. In contrast, 1,1-dichloroethane was mutagenic to strains TA1537, TA98, TA100, and TA1535 exposed to its vapor in a desiccator in the presence and absence of S9 mix (Riccio et al. 1983). Although the tests were conducted using three dose levels, the authors did not report the actual doses tested, and therefore the presence of a dose-dependent response could not be assessed. Simmon et al. (1977) on the other hand obtained

negative results using the same strains of Salmonella and a similar protocol. The concentrations of 1,1-dichloroethane tested were not reported. Because the reporting of data was insufficient in studies by Riccio et al. (1983) and Simmon et al. (1977), the discrepancies in their reported results cannot be explained at this time.

1,1-Dichloroethane was nonmutagenic in yeast strains D3 and D7, even in the presence of S9 mix (Bronzetti et al. 1987; Simmon et al. 1977). Bronzetti et al. (1987) conducted an assay using strain D7 of Saccharomyces cerevisiae from the stationary and logarithmic growth phase. The cells harvested from the log phase cultures contained cytochrome P-450 and were capable of metabolizing promutagens to genetically active products. Both studies lacked details regarding doses of 1,1-dichloroethane tested, though conflicting results may also be due to impurities in the chemicals used.

Tu et al. (1985) exposed BALB/c-3T3 cells to 1,1-dichloroethane in a sealed chamber for 24 hours. No cell transformation was detected. This lack of effect may be due to the short period of exposure. However, 1,1-dichloroethane increased the frequency of transformation induced by SA-7 virus in Syrian hamster embryo cells (Hatch et al. 1983). Embryo cell cultures were exposed in a sealed treatment chamber to volatilized 1,1-dichloroethane for 20 hours and then treated with SA7 virus for 3 hours. 1,1-Dichloroethane treatment significantly increased the viral transformation frequency in cells in a dose-dependent manner. The highest concentration (1,000 µg/mL) was cytotoxic. These results reflect the capacity of 1,1-dichloroethane to interact with cellular DNA in hamster embryo cells.

In an in vivo study by Colacci et al. (1985) 1,1-dichloroethane (98% purity) was found covalently bound to nucleic acids and proteins from liver, lung, kidney, and stomach of male rats and mice 22 hours following a single intraperitoneal injection of approximately 1.2 mg/kg. In vitro binding of 1,1-dichloroethane to nucleic acids and proteins was mediated by liver P-450 dependent microsomal mixed function oxidase system. Glutathione-s-transferase shifted the equilibrium of the enzymatic reaction and thereby decreased binding, presumably by reducing the amount of toxic metabolite available for binding to macromolecules. On the other hand, phenobarbital increased binding by increasing cytochrome P-450 activity, thus generating more toxic metabolites available for binding to macromolecules. Presumably the metabolites generated from P-450 enzymatic action on 1,1-dichloroethane bind to cellular macromolecules. Lung microsomes were weakly effective whereas kidney and stomach microsomal fractions were ineffective. Therefore, the binding to macromolecules of various organs detected in vivo may have been due to a stable hepatic metabolite that was circulated to reach extrahepatic organs. Pretreatment with phenobarbital enhanced the binding to DNA, microsomal RNA and proteins while addition of glutathione-s-transferase (GSH) to the microsomal systems caused suppression of binding. Because only radioactivity was measured it is difficult to determine whether the µmole bound represents 1,1-dichloroethane or its metabolite(s). However, the fact

that binding is enhanced with induction of P-450 suggests that it represents the metabolite(s). Thus, GSH appears to play a detoxification role in the metabolism of 1,1-dichloroethane. The fact that 1,1-dichloroethane binds to nucleic acid suggests that it may have a potential to produce mutation in a mammalian system.

Cancer. There is inconclusive evidence that 1,1-dichloroethane may be carcinogenic in humans. A significant positive dose-related trend was observed for the incidence of hemangiosarcomas and mammary adenocarcinomas in female rats, hepatocellular carcinoma in male mice, and endometrial stromal polyps in female mice. However, only the incidence of endometrial stromal polyps in female mice was significantly increased over the corresponding control animals. Limitations in this study (e.g., poor survival in both treated and control animals) preclude the consideration of these results as conclusive evidence of carcinogenicity (NCI 1977).

Results of a recently reported drinking water bioassay in mice indicated that 1,1-dichloroethane is not carcinogenic (Klaunig et al. 1986). Possible differences in the pharmacokinetics of 1,1-dichloroethane between the NCI (1977) and Klaunig et al. (1986) studies because of the different methods of administration and different vehicle and/or differences in dose levels employed may account for the disparate results. An <u>in vitro</u> assay of carcinogenicity initiation also yielded negative results for 1,1-dichloroethane (Herren-Freund and Pereira 1986).

The induction of γ-glutamyltranspeptidase (GTP) foci, which are putative preneoplastic lesions, in isolated rat liver hepatocytes correlates well with carcinogenicity. 1,1-Dichloroethane failed to induce GTP foci in liver hepatocytes obtained from rats and mice treated with 1,1-dichloroethane for 7 days followed by promotion with phenobarbital (Herren-Freund and Pereira 1986). This suggests that 1,1-dichloroethane is not carcinogenic, though these results are not conclusive.

There is limited evidence that neither confirms or dispels the carcinogenic potential of 1,1-dichloroethane. Thus, these results are inconclusive as to whether it poses a cancer threat for humans. The EPA has classified 1,1-dichloroethane as a Class C chemical which is defined as a possible human carcinogen (IRIS 1990).

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TOXICOLOGICAL PROFILE FOR SELENIUM

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In collaboration with:

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Selenium

2.3 RELEVANCE TO PUBLIC HEALTH

Death. Information regarding death in humans following inhalation or dermal exposure was not found. Deaths due to respiratory failure in humans following ingestion of selenium compounds have been reported, but the amount of selenium ingested was not quantified. Concentrations and doses causing death in animals have been reported for acute inhalation exposures, and for acute, intermediate, and chronic oral exposures. The causes of acute lethality of selenium compounds in animals following either inhalation or oral exposures appears to be respiratory failure. The toxic effects of selenium following oral and inhalation exposure are cumulative. In guinea pigs, the LC_{50} for inhalation exposure to hydrogen selenide decreases with increasing

duration of exposure: the LC_{50} values are 12.7 mg selenium/m^3, 9 mg selenium/m^3, and 1-4 mg selenium/m^3 for durations of 1, 4, and 8 hours respectively (Dudley and Miller 1941). Similarly, sodium selenite administered to rats at a dose of 0.69 mg selenium/kg/day killed 5 out of 8 male rats between days 28 and 42 (Palmer and Olson 1974), whereas the same compound administered at less than half that dose (i.e., 0.28 mg selenium/kg/day) killed half of a test group of male rats after 58 days of administration (Schroeder and Mitchener 1971a). Water soluble selenium compounds are more lethal to animals than elemental selenium by any route.

Systemic Effects. Systemic effects in humans and animals following inhalation and oral exposure to selenium compounds are similar. One proposed mechanism of toxicity for selenium compound is that under conditions of excess body levels of selenium, selenium atoms begin to replace sulfur atoms in structural and enzymatic proteins (Shamberger 1970), destroying the proteins' structural and functional integrity. This mechanism of action is unlikely to be organ specific and toxic levels of selenium are expected to affect multiple organ systems. Differential sensitivities of the various organ systems to selenium exposure would be expected on the basis of differential accumulation or retention of selenium compounds.

The primary target organ in humans and in animals upon acute exposure by inhalation or oral routes is the lung, with cardiovascular, hepatic, and renal systems also affected. Lesser effects are observed in all other organ systems but the muscular/skeletal system. No studies were located regarding effects in humans or in animals following intermediate or chronic inhalation exposure to selenium or to selenium compounds. Following intermediate or chronic oral exposure to selenium compounds, the primary effects in humans are dermal and neurological. Following intermediate and chronic oral exposure to selenium compounds, the primary effects in livestock exposed to naturally occurring selenium in range plants are also dermal and neurological. The primary effects in laboratory animals exposed to inorganic selenium salts or to selenium-containing amino acids are cardiovascular, gastrointestinal, hematological, hepatic, dermal, immunological, neurological, and reproductive.

Respiratory Effects. Following acute inhalation or acute oral exposure to selenium compounds, the primary sign of selenium toxicity in both humans and animals is respiratory distress. Clinical signs in humans have been reported to include bronchial spasms, severe bronchitis, and bronchial pneumonia (Buchan 1947; Wilson 1962). Signs in animals have included labored breathing and bronchial pneumonia (Hall et al. 1951). Death is usually from respiratory failure associated with pulmonary edema. Lethal doses of parenteral sodium selenite (1-4 mg/kg) have also produced moderate pulmonary edema in pigs (Van Vleet et al. 1974). Following intermediate and chronic oral exposure to selenium compounds, however, the lungs do not appear to be a primary target organ in either humans or animals.

Cardiovascular Effects. Following acute inhalation or acute oral exposure to selenium compounds, some cardiovascular signs such as elevated pulse rate and tachycardia have been reported in humans. In livestock, lesions of the heart have been observed following acute oral exposure to seleniferous plants. Harr et al. (1967) reported myocardial hyperemia, hemorrhage, and degeneration in old rats following a lifetime administration of sodium selenate or sodium selenite in the diet. In animals, intraperitoneal administration of sodium selenite at 1.0 mg selenium/kg bw has produced ultrastructural abnormalities of mitochondria in myocardial tissue but not in liver or kidney tissues (Dini et al. 1981). The dose was lethal for the guinea pigs, with animals dying 24 hours or later following the intraperitoneal administration. Pretreatment with pyruvate or methionine protected against the mitochondrial changes. Only one dose was used, however, and it is not known whether mitochondrial changes occur at sublethal doses.

Hepatic Effects. In animals, the liver is affected following both inhalation and oral exposure to several different selenium compounds. Liver effects were observed in guinea pigs following inhalation exposure to elemental selenium dust and hydrogen selenide. The liver appears to be the primary target organ for the oral toxicity of sodium selenate, sodium selenite, and organic forms of selenium following intermediate and chronic exposure, whereas liver cirrhosis or dysfunction has not been a notable component of the clinical manifestations of chronic selenosis in humans. Selenium sulfide administration to rats has also produced hepatic effects.

Renal Effects. In animals, the kidney appears not to be seriously affected by acute inhalation exposure to elemental selenium or hydrogen selenide or acute to chronic oral exposure to sodium selenate, sodium selenite, or dietary selenium compounds. Selenium sulfide administration to mice has produced interstitial nephritis.

Hematological Effects. A variety of changes occur in the blood chemistry of animals after acute oral exposure to sodium selenite (Anderson and Moxon 1942). Anemia, red cell hemolysis, and reduced blood hemoglobin concentration have been observed in rats following intermediate and chronic oral exposure to sodium selenite (Halverson et al. 1966; Halverson et al. 1970). Young et al. (1981) have reported a lytic effect for sodium selenite on normal sheep erythrocytes in vitro.

Dermal/Ocular Effects. Following chronic oral exposure to organic selenium compounds found in food, two principal clinical conditions observed in humans are dermal and neurological effects, as described in the epidemiological study of endemic selenosis in the People's Republic of China (Yang et al. 1983). The dermal manifestations include loss of hair, deformation and loss of nails, and discoloration and excessive decay of teeth. Similar clinical manifestations occur in livestock following intermediate and chronic exposure to seleniferous plants, including loss of hair and

malformation of hooves characteristic of "alkali disease" (Rosenfeld and Beath 1964).

Other Systemic Effects. One of the most common effects in animals following intermediate or chronic oral administration of inorganic and organic compounds of selenium is reduced growth rate of young animals and loss of weight in older animals (Halverson et al. 1966; Harr et al. 1967; Nelson et al. 1943; Palmer and Olson 1974; Schroeder 1967). Selenium sulfide administration has also caused a reduction in body weight in female mice (NTP 1980c).

Neurological Effects. Following chronic oral exposure to selenium compounds in the diet, neurological manifestations in humans have been reported to include numbness, paralysis, and occasionally hemiplegia. Analogous clinical manifestations (i.e., loss of hair, malformation of hooves, and "blind staggers" in cattle and poliomyelomalacia in swine) occur in livestock following intermediate and chronic grazing exposure to plants containing high levels of selenium (see Sections 2.2.2.2 and 2.2.2.4). Histologically, swine with neurological symptoms also exhibit bilateral macroscopic lesions of the ventral horn of the spinal cord.

The neurological symptoms and histopathology observed in livestock following oral exposure to excess selenium compounds have not been recorded in laboratory animals. This suggests that small laboratory mammals might not be appropriate models for selenium toxicity in humans (e.g., laboratory animals metabolize and/or excrete selenium compounds more quickly), that some as yet unidentified organic form of selenium contributes to the neurological manifestations of chronic selenosis in humans and in livestock, or that unrecognized confounding factors have contributed to the neurological syndrome associated with chronic selenosis in field studies of humans and livestock.

Reproductive/Developmental Effects. There is no evidence from experimental animals administered selenium compounds via the oral route or via injection that selenium compounds are teratogenic in mammals. Lee et al. (1979) reported reduced fetal weights following subcutaneous injection of pregnant mice with sodium selenite (1.6 mg selenium/kg/day) on days 9-12 of gestation. The incidence of cleft-palate was not statistically different from the control group. Yonemoto et al. (1984) observed a dose-dependent elevation of maternal death rates and decreases in body weight of offspring of pregnant mice receiving a single intravenous injection of sodium selenite at doses ranging from 1.30 to 2.52 mg selenium/kg/day on day 12 of gestation. The failure to observe teratogenic effects in these studies is consistent with the other mammalian studies involving oral administration of potassium selenate and unspecified selenate salt (Rosenfeld and Beath 1954; Schroeder and Mitchener 1971b). In hamsters, Holmberg and Fern (1969) also failed to produce teratogenic effects with intravenous injection of sodium selenite at 0.91 mg selenium/kg selenium on day 8 of pregnancy. The administered dose was

just sublethal to the dam, yet caused only six percent resorptions, a percentage that was comparable with controls.

Selenium might, however, be a particular hazard to the developing eye. Ostadalova and Babicky (1980) administered single subcutaneous doses of sodium selenate, DL-selenomethionine, DL-selenocystine, dimethyl selenide, or trimethyl selenonium to groups of 10-day-old male rats and observed the effects on the eye between the ages of 14-16 days (when the eyes opened) to 60 days. The range of doses administered was adjusted for each compound such that the lowest dose did not produce cataracts and the highest dose was lethal. A dose-related increase in eye cataracts was induced by the administration of sodium selenate, DL-selenomethionine, and DL-selenocystine; dimethyl selenide and trimethylselenonium chloride failed to produce cataracts.

Daily intraperitoneal injections of male rats with selenium dioxide at daily doses up to 0.035 mg selenium/kg/day for 90 days produced dose-dependent histological changes in the testes with significant testicular degeneration and atrophy at the highest dose tested (Chowdhury and Venkatakrishna-Bhatt 1983).

The relevance of these reproductive/developmental effects of selenium exposure in animals to potential reproductive or developmental effects in humans is not known.

Carcinogenicity. The majority of epidemiological studies in humans do not suggest that excess exposure to selenium is associated with an increased risk of cancer. To the contrary, investigators report an association between low selenium intake or body levels and an increased risk of developing many types of cancers (Hocman 1988; Shamberger 1970; Vernie 1984).

Despite early reports that orally administered selenium produced hepatic tumors in rats, subsequent chronic experimental exposure of rats and mice to selenium salts and organic forms of selenium in the diet have provided no further evidence of hepatic carcinogenicity (Harr et al. 1967; Schroeder 1967; Schroeder and Mitchener 1972). Instead, in the majority of studies conducted in the last 15 years, selenium supplementation has significantly inhibited spontaneous and chemical-, viral-, and UV-induced neoplasia in animals, although there are a few exceptions (Hocman 1988; Medina 1986; Vernie 1984). In a review of 39 experiments testing the relationship between selenium supplementation and tumorigenesis in animals, Medina (1986) noted that 34 out of 39 studies demonstrated an inhibitory effect of selenium on tumorigenesis; in three studies, selenium administration had no effect on tumorigenicity; and in two studies, selenium enhanced tumorigenicity. Thus, selenium appears to inhibit tumorigenesis in most, but not all, of the test situations investigated.

A variety of mechanisms of action have been proposed to explain the inhibitory effect of selenium on carcinogenesis (Hocman 1988). Because oxygen radicals such as $O_2^{-\cdot}$ or $OH^{\cdot}$ may initiate the process of carcinogenesis, one hypothesis is that adequate or increased activity levels of the selenium-dependent enzyme glutathione peroxidase (GSH-Px) helps to prevent the initiation of cancers (Hocman 1988). Another hypothesis is that selenium inhibits cell proliferation (Vernie 1984). It is unlikely, however, that a single mechanism of action is responsible for selenium's putative anticarcinogenic properties.

Selenium sulfide, on the other hand, has been demonstrated to be carcinogenic following oral daily administration by gavage in rats and female mice (NTP 1980c).

Genotoxic Effects. Inorganic selenium compounds have been observed to have both genotoxic and antigenotoxic effects. The antigenotoxic effects generally occur at lower selenium exposure levels than the genotoxic effects. This discussion will focus on genotoxic effects only.

In general, sodium selenite and sodium selenate have produced mixed results in bacterial mutagenicity test systems (Table 2-4). Sodium selenite has tested positive for base-pair substitution mutations using the Ames test in Salmonella typhimurium and was also positive in the transformation assay using Bacillus subtilis (Nakamuro et al. 1976; Noda et al. 1979). However, negative results have also been reported for sodium selenite both in the Ames test in S. typhimurium and the rec assay using B. subtilis (Lofroth and Ames 1978; Noda et al. 1979). Sodium selenate on the other hand has tested positive in the Ames test using S. typhimurium (base-pair substitution) and in the rec assay using B. subtilis (Lofroth and Ames 1978; Noda et al. 1979), but has tested negative using the transformation assay in B. subtilis (Nakamuro et al. 1976).

Results with mammalian cell systems are also mixed, although sodium selenite is more consistently genotoxic in these systems. Sodium selenite has been observed to induce unscheduled DNA synthesis (UDS), chromosomal aberrations, and sister-chromatid exchange in cultured human fibroblasts (Lo et al. 1978; Ray et al 1978; Whiting et al. 1980), UDS in Chinese hamster V79 cells (Sirianni and Huang 1983), and chromosomal aberrations in Chinese hamster ovary cells (Whiting et al. 1980). However, sodium selenate induced chromosomal aberrations in Chinese hamster ovary cells (Whiting et al. 1980), and UDS in Chinese hamster V79 cells (Sirianni and Huang 1983), but did not induce chromosomal aberrations in human leukocytes or cultured human fibroblasts (Lo et al. 1978; Nakamuro et al. 1976).

Addition of glutathione to test mixtures enhances the genotoxicity of sodium selenite, sodium selenate, and sodium selenide in bacterial test systems indicating that production of a mutagenic species occurs via a reductive mechanism following exposure to these compounds (Whiting et

al. 1980). This mechanism is supported by results in mammalian test systems. For example, in cultured human leukocytes, sodium selenite induces chromosome aberrations and sister chromatid exchanges (Nakamuro et al. 1976; Ray et al. 1978; Ray and Altenburg 1978). Sister chromatid exchange was not observed at similar sodium selenite concentrations in a human lymphoblastoid cell line; however, exchanges were observed when these same cells were incubated with sodium selenite and red blood cell lysate (Ray and Altenburg 1978). The observation that internal constituents of red blood cells may contribute to the genotoxicity of sodium selenite supports the suggestion that metabolism is involved in the production of an active species following exposure to sodium selenite in these test systems. The active species responsible for the genotoxic effects is not known.

At high concentrations, sodium selenite induces unscheduled DNA synthesis and chromosome aberrations in cultured human fibroblasts (Lo et al. 1978). Addition of a metabolic activator (S9 fraction) or glutathione have increased both the number of aberrations and the toxicity of sodium selenite (Whiting et al. 1980) and sodium selenate (Lo et al. 1978; Whiting et al. 1980).

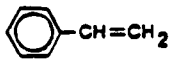
Sodium selenite has also been studied in vivo in the rat and Chinese hamster. An increased number of bone marrow cells with chromosome aberrations were observed following intraperitoneal injections of sodium selenite. The doses at which aberrations were higher than controls ranged from 3 to 6 mg selenium/kg of body weight, a level comparable to reported LD_{50} values for intraperitoneal injection of sodium selenite in rats (Norppa et al. 1980; Newton and Lilly 1986; Olson 1986).

STYRENE

Synonyms: phenyl ethylene, vinyl benzene, cinnamene

CAS Registry Number: 100-42-5

Molecular Weight: 104.14

Molecular Formula: 

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Chelsea, Michigan.

Toxicity Profile

Carcinogenicity: There is some evidence for an association between styrene exposure in the styrene-butadiene rubber and styrene-polystyrene manufacturing industries and increased risk of lymphatic and hematopoietic tumors. However, the evidence is insufficient to designate styrene as a human carcinogen on account of limitations in the available studies such as small cohort sizes and concurrent exposure to multiple chemicals.

Jersey et al.[7] (cited in NIOSH[2]) exposed male and female Sprague-Dawley rats to 0, 600, or 1200 ppm styrene via inhalation for 6 hours per day, 5 days per week for 18.3 (males) or 20.7 (females) months with final sacrifice at 24 months; after 2 months the highest exposure level was lowered to 1000 ppm because of excessive mortality among the male rats. There was an increased combined incidence of leukemia and lymphosarcoma tumor types in female rats in both exposure groups which was statistically significant when data from both exposure groups were combined and for each group compared individually to historical, but not concurrent, controls. In addition, there was a higher incidence of alveolar histiocytosis and increased liver weights in high-dose female rats. There was no increase in incidence of tumors attributable to treatment in the male rats; concurrent high mortality due to chronic murine pneumonia limits the conclusions that may be drawn from the male rat data.

In an NCI bioassay,[8] male and female B6C3F$_1$ mice and F344 rats received 150 or 300 mg/kg/day and 500, 1000, or 2000 mg/kg/day, respectively, by gavage in corn oil 5 days a week for 103 weeks (low-dose rats) or 78 weeks (all other groups). There was an increased incidence of lung adenomas in male mice compared with vehicle, but not historical, controls. NCI concluded that there was "suggestive evidence of carcinogenicity in male B6C3F$_1$ mice, but the evidence was not "convincing" for either species.

The possible carcinogenicity of styrene has been investigated in several additional studies that are reviewed in the drinking water criteria document[4] and the NIOSH criteria document.[2] Regarding carcinogenicity, NIOSH states that "from the experimental animal investigations and from the epidemiological studies, there seems to be little basis to conclude that styrene is carcinogenic."[2] It seems more reasonable to tentatively place styrene in group C, possible human carcinogen under the EPA weight-of-evidence classification.

Mutagenicity: Positive results are listed in the Gene-Tox database as summarized in RTECS for the following assays: (1) micronucleus test—in vitro human lymphocytes, (2) Ames assay, (3) *Drosophila* SLRL test, (4) *S. cerevisiae* gene conversion, and (5) in vivo cytogenetics—human lymphocytes. Negative results are listed for the following assays: (1) cell transformation—Syrian hamster embryo cells by adenovirus SA7, (2) gene mutation—V79 cell culture, and (3) in vitro unscheduled DNA synthesis-human fibroblasts.[9] The evidence for genetic activity of styrene in short-term tests was also recently reviewed by IARC.[10] Based on the

IARC classification scheme, it is our judgment that the evidence for genetic activity of styrene in short-term tests is sufficient.

Developmental Toxicity: Although there have been some reports of birth defects[11] (cited in NIOSH[2]) and increased incidence of spontaneous abortions[12] (cited in NIOSH[2]) in women occupationally exposed to styrene, evidence of developmental toxicity has not been found in several apparently well-conducted animal studies. These include a multigeneration study in which rats were exposed to styrene in drinking water[13] and a study in which rabbits were exposed to styrene via inhalation and rats were exposed via inhalation and gavage.[14]

In another study, however, Kankaanpaa et al.[15] exposed BMR/T6T6 mice to 0 or 250 ppm styrene for 6 hours per day on gestation days 6 through 16 of gestation and Chinese hamsters to 0, 300, 500, 750, or 1000 ppm styrene on days 6 through 18 of gestation. Embryotoxicity, in the form of significantly higher incidences of dead and resorbed fetuses, were observed in both species only in the highest exposure group relative to controls. Some degree of maternal toxicity was apparently observed, but no details were given and the authors did not attribute the embryotoxicity to maternal toxicity.

Reproductive Toxicity: No data were found implicating styrene as a reproductive toxin.

Systemic Toxicity: The best-documented form of systemic toxicity resulting from styrene exposure involves the central nervous system. Subjectively, this is manifest as complaints of headache, fatigue, dizziness, confusion, drowsiness, malaise, difficulty in concentrating, and a feeling of intoxication. Clinical signs include altered equilibrium, delayed reaction times, and abnormal EEGs. Alterations such as these have been reported in experimental studies at concentrations as low as 100 ppm[16,17] (cited in NIOSH[2]). Numerous studies, both clinical and experimental, documenting the C.N.S. toxicity of styrene are reviewed in the NIOSH criteria document.[2]

In addition to C.N.S. toxicity, there is also more limited evidence for other adverse effects attributable to styrene. These include peripheral neuropathy, abnormal pulmonary function, and alterations in liver function. The evidence for these effects is also reviewed in the criteria document.[2]

In a recent review of the toxicity of styrene, Bond et al.[16] concluded that many of the effects of styrene in humans and laboratory animals are similar. Further, it was noted that the major metabolic pathway in both humans and animal models involves oxidation of the vinyl group to styrene oxide with further metabolism to several products, at least two of which—mandelic and phenylglyoxylic acids—have been detected in both human and rodent urine subsequent to styrene exposure.

Irritation: The irritancy of styrene to the eyes, nose, skin, and respiratory tract is well documented in both clinical and experimental studies. A large proportion of subjects report upper respiratory tract irritation at exposures as low as 100 ppm, and

some report eye irritation at concentrations of 20 or 50 ppm[16,17,19] (cited in NIOSH[2]). Styrene is reported to have an aromatic odor[1] and an odor threshold of 0.32 ppm (geometric mean of reported literature values).[20]

Basis for the AALG: Styrene is considered "very slightly" soluble in water and has a vapor pressure of 6.1 mm Hg at 25°C.[20] It is used in the production of polystyrene plastics, protective coatings, styrenated polyesters, copolymer resins, and as a chemical intermediate.[1] Styrene has been detected in both ambient air and finished drinking water in the United States.[21] Based on the lack of data on the environmental fate and distribution of styrene and the expected contribution of various media to human exposure, 50% of the contribution to total exposure is allowed from air.

Given the equivocal nature of the evidence for styrene carcinogenicity, two AALGs were calculated for styrene: one based on carcinogenicity and another based on systemic toxicity. Also, use of quantitative risk assessment was not deemed appropriate on account of the equivocal evidence of carcinogenicity. The AALG is instead based on the rat LOAEL of 600 ppm from the study of Jersey et al.[7] (cited in NIOSH[2]) using an uncertainty factor approach. The LOAEL was adjusted for continuous exposure (600 ppm × 5/7 × 6/24 × 20.7/24), and the total uncertainty factor used was 10,000 (10 for a LOAEL × 10 for interindividual variation × 10 for interspecies variation × 10 for the database factor). Use of the database factor was justified based on the severity of the effect and, in addition, data from human experimental studies[22,23] (cited in Amoore and Hautala[20]). Retention of 60% of the exposure dose was assumed. Given the weight-of-evidence ranking for styrene and the pending review of its carcinogenicity by EPA,[24] the AALG should be considered provisional.

An AALG was also calculated for systemic toxicity, specifically neurotoxicity using the NIOSH REL-TWA as a basis. This limit was treated as a human LOAEL (REL/210; 10 for interindividual variation × 5 for a LOAEL × 4.2) based on the review in the criteria document,[2] in which it was stated that there were "effects such as slower reactions, subjective complaints related to CNS depression and abnormal EEGs at styrene concentrations around 50 ppm." This AALG should also be considered provisional.

AALG:
- carcinogenicity—6.2 ppb (26.3 $\mu g/m^3$) annual TWA
- systemic toxicity—119 ppb (507 $\mu g/m^3$) 8-hour TWA

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DRAFT

TOXICOLOGICAL PROFILE FOR 1,1,1-TRICHLOROETHANE

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Toxicological Profile
TP-90-27

1,1,1-Trichloroethane

2.4 RELEVANCE TO PUBLIC HEALTH

Clinical symptoms associated with exposure to 1,1,1-trichloroethane that have been reported in humans include hypotension, diarrhea and vomiting, central nervous system depression and dermal and ocular irritation. Mild hepatic effects may also occur in humans. Deaths have been attributed to cardiac arrythmia and respiratory failure secondary to central nervous system depression. Effects reported in humans that also occur in animals include hypotension, cardiac arrythmia, mild hepatic effects, central nervous system depression, and dermal irritation. Effects that have been observed in animals but not investigated in humans include mild developmental effects.

Death. The volatility of 1,1,1-trichloroethane coupled with the rapid and extensive absorption and elimination of inhaled 1,1,1-trichloroethane makes acute inhalation (as compared to intermediate and chronic duration) the most likely lethal exposure scenario in humans. The acute lethal air concentration for humans is unknown; however, simulations of several lethal exposure scenarios suggest that it may be as low as 6000 ppm. The results of animal studies indicate that the acutely lethal exposure concentration increases substantially with decreasing exposure duration. Thus, death may occur at a 3- to 4-fold lower concentration after a 6-7 hour exposure than after a 15-minute exposure.

Human deaths from inhalation of 1,1,1-trichloroethane have been attributed to respiratory failure secondary to central nervous system depression and to cardiac arrythmia. Based on the results of animal

studies, lethal arrhythmias probably result from sensitization of the heart to epinephrine. Therefore, acutely lethal exposure levels may be lower in individuals exposed during physical exertion. Physical exertion may also decrease the acutely lethal exposure level by increasing lung perfusion and lung retention of inhaled 1,1,1-trichloroethane.

Very little is known about the lethality of orally ingested 1,1,1-trichloroethane in humans. In one case of accidental acute oral exposure, ingestion of 600 mg/kg of 1,1,1-trichloroethane did not prove lethal. Based on the results of animal studies, it is possible that much higher acute oral doses can be tolerated.

Human lethality involving dermal exposure has not been reported. However, such an occurrence is extremely unlikely in view of the high volatility of 1,1,1-trichloroethane, which would limit the absorption of 1,1,1-trichloroethane in contact with the skin. Lethality in animals has been reported only when extremely high doses (15,800 mg/kg) are applied to the skin for prolonged periods (e.g., 24 hours) under occlusion.

Systemic Effects. Systemic effects of 1,1,1-trichloroethane reported in humans include hypotension, mild hepatic effects, diarrhea and vomiting, and dermal and ocular irritation. Cardiac arrhythmias have been implicated in cases of death following inhalation exposure to high concentrations of 1,1,1-trichloroethane.

1,1,1-Trichloroethane can reduce blood pressure (mild-to-severe reduction) in humans. However, such effects are likely only after exposure to high concentrations of 1,1,1-trichloroethane vapor. Humans exposed to low levels daily for up to 6 years did not have abnormalities in blood pressure, heart rate, or electrocardiogram. Reduced blood pressure accompanies exposure to anesthetic concentrations of 1,1,1-trichloroethane vapor (10,000-26,000 ppm). The effects are not permanent and subside shortly after exposure ceases. The mechanism has been studied in animals and appears to involve cardiac depression and peripheral vasodilation.

Human deaths following inhalation of 1,1,1-trichloroethane are often attributed to cardiac arrhythmia. Such conclusions are based on animal studies in which arrhythmias have been produced during or immediately following acute inhalation exposure to 1,1,1-trichloroethane. The mechanism for the arrhythmias involves sensitization of the heart to endogenous epinephrine. The level at which cardiac sensitization occurs in humans is not known, but in animals concentrations as low as 5000 ppm are effective after only 10 minutes of exposure. Physical exertion, stress, or any other stimulus for the release of epinephrine from the adrenal medulla may render an individual more vulnerable to 1,1,1-trichloroethane.

2. HEALTH EFFECTS

Nausea, vomiting, and diarrhea have been reported to occur in humans after acute oral or inhalation exposure to high levels of 1,1,1-trichloroethane. Vomiting and diarrhea have not been reported in animals, and the mechanisms for these effects are not known.

1,1,1-Trichloroethane may be a hepatotoxicant in humans, although the evidence is not conclusive. Increased levels of urinary urobilinogen and serum bilirubin, suggestive of liver injury, have been reported in humans exposed to 1,1,1-trichloroethane by inhalation or ingestion. Mild hepatic changes have also been found at autopsy in people who died following acute inhalation of high concentrations of 1,1,1-trichloroethane. Studies in animals have shown that exposure to relatively high concentrations of 1,1,1-trichloroethane in air (1000 ppm or more) or high oral doses (1334 mg/kg or more) are required to produce liver injury, although some effects have been observed at 200-500 ppm in air. Effects observed in animals include fatty degeneration, slight increases in liver weight, and changes in liver and serum enzyme levels. The effects are reversible and subside after exposure is terminated.

1,1,1-Trichloroethane is mildly irritating when applied to the skin. Effects include slight, transient, reversible erythema and edema. Exposure to 1,1,1-trichloroethane vapor is associated with mild eye irritation in humans and animals. Based on these results and the results of direct-application animal studies, it is likely that 1,1,1-trichloroethane applied directly to the eye will produce irritation in humans as well.

Immunological Effects. Immunological effects of 1,1,1-trichloroethane have not been reported in humans, and have not been studied extensively in animals. Acute inhalation exposure had no effect on survival from a bacterial pathogen challenge in mice. Histological evaluation of immune system tissues from rats and mice (including lymph nodes, thymus, and spleen) have not revealed any lesions attributable to 1,1,1-trichloroethane exposure. However, more extensive studies of immune function would be required to adequately evaluate the immunotoxic potential of 1,1,1-trichloroethane in humans.

Neurological Effects. Neurological effects are the preeminent symptoms of acute inhalation exposure to 1,1,1-trichloroethane in humans. The intoxicating effects of inhaled 1,1,1-trichloroethane create a potential for abuse of this chemical. Neurological effects have not been reported following oral or dermal exposure, and are likely to occur, if at all, only after exposure to very high levels or doses. Effects reported following acute inhalation in humans increase in severity with increasing exposure level. Impaired performance on tests of psychophysiological function have been reported in subjects exposed to low concentrations (175 ppm or more). Dizziness, lightheadedness, and loss of coordination are found after exposure to moderate concentrations (more than 500 ppm). General anesthesia occurs at high levels (10,000 ppm or more). These effects subside rapidly

after exposure is terminated. Irreversible neurological impairment has not been reported in humans.

Animals appear to provide useful models for examining the neurological effects of 1,1,1-trichloroethane. As in humans, central nervous system depression is the predominant effect of inhaled 1,1,1-trichloroethane in animals; symptoms include ataxia, anesthesia, and death at increasing concentrations. There was no evidence of gross or histologic damage found in the brains of most exposed animals, but lasting physical changes to the brain have been indicated by reports of increased levels of glial fibrillary acid protein and decreased DNA content in the brain of gerbils following intermediate exposure to low levels. Alterations of brain metabolism have also been found in exposed animals. Behavioral changes, including impaired performance on behavioral tests and increased motor activity, have been widely reported; however, the sites of action and biochemical mechanisms of neurotoxicity have not been identified.

Little information was located regarding neurological effects in man or animals following oral or dermal exposure to 1,1,1-trichloroethane. Existing data indicate that a single oral exposure to moderate levels may not produce outward signs of neurotoxicity. However, it is assumed that high doses of 1,1,1-trichloroethane administered orally or dermally will result in neurotoxicity.

Developmental Effects. Developmental effects of 1,1,1-trichloroethane in humans have not been reported. Minor embryotoxic effects were reported in rats were and rabbits exposed to high concentrations of 1,1,1-trichloroethane by inhalation. Effects included decreased fetal weights, increased minor soft tissue and skeletal anomalies, and delayed ossification. For one of these studies, developmental defects may have been associated with the significant maternal toxicity observed. Neither an inhalation study using a lower concentration nor a drinking water study found any developmental effects. Although there are some positive reports of minor developmental effects in experimental animals, 1,1,1-trichloroethane does not appear to be a potent developmental toxicant in animals. However, only one study examined potential effects on the developing nervous system. In view of the known neurological effects of 1,1,1-trichloroethane in humans and animals, additional developmental studies that examine neurological endpoints would be an important component of a complete investigation of the potential for developmental toxicity of 1,1,1-trichloroethane in humans.

Reproductive Effects. Reproductive effects of 1,1,1-trichloroethane in humans have not been reported. Histological evaluation of reproductive organs and tissues from male and female rats and mice revealed no lesions attributable to 1,1,1-trichloroethane exposure. More sensitive tests are required before a full evaluation of the potential for reproductive effects in humans can be made.

2. HEALTH EFFECTS

Genotoxic Effects. The genotoxic effects of 1,1,1-trichloroethane have been studied extensively. The results are summarized in Tables 2-4 through 2-7. Although most tests of mutagenicity in the Ames Salmonella assay produced negative results, those conducted in a desiccator, to minimize evaporation and maximize exposure, were positive. These results indicate that 1,1,1-trichloroethane may be mutagenic in Salmonella. The results were negative in other tests of genotoxicity in bacteria and yeast. 1,1,1-Trichloroethane is a relatively volatile compound; therefore, a high evaporation rate could result in lower doses to the microorganisms and thus affect the outcome of genotoxicity tests. This explanation could account for the negative results observed in tests with bacteria and yeast.

Most assays of genotoxicity in mammalian cells were negative, but 1,1,1-trichloroethane did produce chromosomal aberrations in Chinese hamster ovary cells in vitro. In vivo micronucleus tests for chromosomal aberrations were all negative, however. Positive or weakly positive results were reported in assays for degranulation of endoplasmic reticulum, which measures the ability of a compound to displace polysomes from endoplasmic reticulum in rat hepatocytes in vitro, and formation of DNA adducts (binding of the compound to DNA) in mouse liver in vivo. Tests of cell transformation in rat embryo cells, hamster embryo cells, baby hamster kidney cells, and mouse BALB/c-3T3 cells were all positive. It is believed that cell transformation systems may be similar to the process of neoplastic transformations.

Although 1,1,1-trichloroethane was mutagenic in a few assays with Salmonella, induced chromosomal aberrations in a Chinese hamster ovary cell assay, and was positive in mammalian cell transformation assays, the existing genotoxicity data are largely negative. In addition, there is a possibility that positive results were produced by stabilizers and not 1,1,1-trichloroethane itself. Therefore, a firm conclusion regarding the genotoxic potential of 1,1,1-trichloroethane in humans is not possible.

Cancer. Evidence for or against an association between exposure to 1,1,1-trichloroethane and cancer in humans has not been reported. Among animals, no effects were found in a well-designed inhalation study at exposure levels up to 1500 ppm. The results of an oral study indicated that 1,1,1-trichloroethane may have produced an increase in the occurrence of immunoblastic lymphosarcoma in rats. However, the biological and statistical significance of the results of this study are questionable because of the limitations of the study design.

There is also limited information on the role of 1,1,1-trichloroethane metabolites in the toxicity of the compound. Reactive metabolites are important in the carcinogenicity of other chloroethanes. Binding to DNA, which is correlated with carcinogenicity in chlorinated ethanes (Lattanzi et al. 1988), was weak in both in vivo and in vitro tests. Even weak binding, however, indicates an ability to interact with DNA. Cell biotransformation tests were positive for this chemical. The results of

these assays may have been confounded by the presence of stabilizing agents, however. Two of the possible stabilizing additives in commercial formulations of 1,1,1-trichloroethane are 1,2-epoxybutane (butylene oxide), and 1,4-dioxane (diethylene dioxide). Both stabilizers have been identified as potential carcinogens based on animal studies (NTP 1989a).

At this time, it does not appear that exposure to 1,1,1-trichloroethane presents a clear cancer risk in animals; however, as discussed above, limitations surrounding the studies performed to date prevent a definitive assessment of the carcinogenic potential of this compound in humans.

TOXICOLOGICAL PROFILE FOR TOTAL XYLENES

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Toxicological Profile TP-90-30

Xylenes

2.4 RELEVANCE TO PUBLIC HEALTH

The concentrations of mixed xylene and xylene isomers used in animal studies are much higher than the ambient levels encountered in urban and industrial areas. However, information about the effects observed at high concentrations of xylenes is important because potentially high levels may be present at hazardous waste sites. In addition, subgroups of the population may be extremely sensitive and effects seen at high levels in animals may be a predictor of effects seen in these subgroups when they are exposed at much lower levels.

Both human and animal data suggest that mixed xylene, m-xylene, o-xylene, and p-xylene all produce similar effects, although the individual isomers are not necessarily equal in potency with regard to a given effect. Human data indicate that both short and long-term xylene exposure result in a variety of nervous system effects that include headache, mental confusion, narcosis, alterations in body balance, impaired short-term memory, dizziness, and tremors. In animals, xylene also produces nervous system effects. The respiratory system may also be affected. Higher doses of xylene have produced unconsciousness and death in humans and animals. The liver and kidney may also be targets of xylene toxicity in humans, although more thorough data are needed to better assess the relationship.

Death. Xylene can be fatal to both humans and animals following inhalation and oral exposure. Death has been observed in animals following dermal exposure to xylene, but no cases have been reported in humans. Death in humans and animals appears to be caused by either respiratory failure or ventricular fibrillation. The amount of xylene necessary to cause death is relatively large in both animals and humans, and reports of death in humans following inhalation of xylene occurred in areas of poor ventilation. Therefore, it is highly unlikely that inhalation or ingestion of the small amounts of xylene likely to be present in contaminated water or air would pose a risk of death.

Systemic Effects. In humans, acute inhalation of xylene produced nose and throat irritation (Goldie 1960; Hake et al. 1981; Klaucke et al. 1982; Nersesian et al. 1985). Severe lung congestion with pulmonary hemorrhages and edema were noted in a worker who died following acute inhalation of paint fumes containing xylene (Morley et al. 1970). In addition, chronic occupational exposure to xylene vapors has been associated with labored breathing and impaired pulmonary function (Hipolito 1980; Roberts et al. 1988).

Animal data provide supporting evidence for the respiratory effects observed in humans following exposure to xylene. Adverse respiratory effects noted in rats, mice, and guinea pigs following acute and intermediate inhalation exposure to xylene included decreased respiratory rate, labored breathing, irritation of the respiratory tract, pulmonary edema, and pulmonary inflammation (Carpenter et al. 1975; De Ceaurriz et al. 1981; Furnas and Hine 1958; Smyth and Smyth 1928).

Chronic occupational exposure of workers to xylene by inhalation has been associated with increased heart palpitation and abnormal ECGs (Hipolito 1980; Sukhanova et al. 1969). However, these particular reports provide no conclusive evidence that xylene causes cardiovascular effects in humans because exposure conditions were not well characterized and workers may have been exposed to other chemical agents in addition to xylene.

Data from animal studies provide limited evidence that humans could be at increased risk of developing cardiovascular effects following exposure to xylene. Cardiovascular effects observed in rats following acute and intermediate inhalation exposure to xylene have included ventricular repolarization disturbances, atrial fibrillation, arrhythmias, occasional cardiac arrest, changes in ECG, morphological changes in coronary microvessels, decreased myocardial blood flow, and increased heart weight (Morvai et al. 1976, 1987). However, histopathologic lesions of the heart have not been observed in other studies (Carpenter et al. 1975; Hazleton Labs 1988a, 1988b; Jenkins et al. 1970; NTP 1986).

2. HEALTH EFFECTS

Symptoms of nausea, vomiting, and gastric discomfort have been noted in workers following inhalation of xylene. Gastrointestinal effects have not been reported in animals. However, there are sufficient human data to conclude that exposure to xylene could produce such effects (e.g., nausea and vomiting).

Human and animal data provide no indications of adverse hematological effects following inhalation of xylene. In the past, chronic occupational exposure to xylene by inhalation was thought to be associated with a variety of hematological effects. However, exposure in all cases was to solvent mixtures known or suspected to contain benzene. Because benzene is an agent strongly suspected of causing leukemia and other blood dyscrasias in humans, these effects cannot be attributed solely to xylene.

Hematological effects have not been observed in rats, dogs, or guinea pigs exposed by inhalation to mixed xylene or o-xylene for an intermediate period (Carpenter et al. 1975; Jenkins et al. 1970). These negative results from animal studies suggest that humans might not develop hematological effects from intermediate inhalation of xylene; however, the hematological effects from chronic inhalation, oral, and dermal exposure are not known.

No data were available regarding the musculoskeletal effects of xylene in humans following inhalation exposure to mixed xylene, m-, o-, or p-xylene. Animal da regarding musculoskeletal effects following xylene exposure are limited. icroscopic examination of skeletal muscle of rats exposed for an intermediate period of time to mixed xylenes, m-xylene, or p-xylene revealed no treatment-related lesions (Carpenter et al. 1975; Hazleton Labs 1988a, 1988b; NTP 1986). Skeletal anomalies, delayed ossification, and extra ribs have been observed in the fetuses and offspring of pregnant mice and rats exposed by inhalation to mixed xylene and o-xylene (Mirkova et al. 1983; Ungvary et al. 1980b). These latter results suggest that the human fetus might be at increased risk of such skeletal effects following maternal exposure to high levels of xylene. The above studies are not definitive, however, in terms of possible skeletal effects.

Human data regarding the hepatic effects following inhalation of xylene are limited to several case and occupational studies (Dolara et al. 1982; Kurppa and Husman 1982; Morley et al. 1970). However, these studies provide limited evidence for evaluating the hepatic effects of xylene in humans because these subjects were concurrently exposed to other chemical agents in addition to xylene.

Available animal studies indicate that mixed xylene and individual isomers produce a variety of mild hepatic effects, and they provide evidence that humans might be at increased risk of developing such effects following xylene exposure. Effects seen in animals have included increased hepatic

cytochrome P-450 and b5 content, increased hepatic weight, increased liver to body weight ratios, decreased hepatic glycogen, ultrastructural changes in hepatic endoplasmic reticulum, changes in the distribution of hepatocellular nuclei, congestion of liver cells, and/or degeneration of the liver (Bowers et al. 1982; Condie et al. 1988; Elovaara 1982; Elovaara et al. 1980; Muralidhara and Krishnakumari 1980; Patel 1979; Pyykko 1980; Smyth and Smyth 1928; Tatrai and Ungvary 1980; Tatrai et al. 1981; Toftgard and Nilsen 1981, 1982; Toftgard et al. 1981; Ungvary et al. 1980a). Many of the observed hepatic effects in animals following inhalation and oral exposure to xylene are likely due to increased metabolism of the solvent and are not necessarily adverse effects (EPA 1985a; Tatrai et al. 1981).

The available human studies that investigate the renal effects following inhalation of xylene are of limited value because exposure conditions were not well characterized and subjects were exposed to other solvents in addition to xylene. However, they provide suggestive evidence that subjects exposed by inhalation to solvent mixtures containing xylene may be at an increased risk of developing renal dysfunction and/or renal damage (Askergren 1982; Franchini et al. 1983; Morley et al. 1970). Indications of renal effects in humans exposed to solvent mixtures containing xylene have included increased blood urea concentrations, decreased urinary clearance of endogenous creatinine, increased lysozymuria, increased urinary levels of β-glucuronidase, and increased urinary excretion of albumin, erythrocytes, and leukocytes (Askergren 1982; Franchini et al. 1983; Morley et al. 1970). No human data were available regarding the renal toxicity of xylene following oral or dermal exposure.

Data from ar nal studies provide additional evidence that humans could be at risk of developing renal effects following inhalation exposure to xylene. Effects noted in studies with rats, guinea pigs, dogs, and monkeys have included increased renal enzyme activity, increased renal cytochrome P-450 content, increased renal microsomal protein, and increased kidney-to-body weight ratios (Condie et al. 1988; Elovaara 1982; Toftgard and Nilsen 1982). In the study by Condie et al. (1988), tubular dilation and atrophy consistent with early chronic nephropathy were observed, however in studies by Carpenter et al. (1975) and Jenkins et al. (1970), the biochemical changes were not associated with any histopathologic lesions of the kidney.

It has been suggested that xylene induces renal effects by causing increased capillary (at the glomerulus) and/or tubular permeability (EPA 1985a). Increased renal permeability caused by irritant or fluidization effects could result in physiological and possibly histological effects (EPA 1985a). In humans exposed to solvent mixtures containing xylene, the increased urinary levels of β-glucuronidase may be due to a faster cellular turnover in the renal tubular epithelium because of a mild toxic effect (Franchini et al. 1983). The lysozymuria and increase in urinary excretion of

albumin may be indicative of potential damage to the renal tubules and renal glomeruli, respectively (Askergren 1982; Franchini et al. 1983). Increased urinary excretion of erythrocytes and leukocytes are also indicators of potential toxic injury to the kidney (Askergren 1982).

Dermal exposure of humans to xylene causes skin irritation, dryness and scaling of the skin, and vasodilation of the skin (Engstrom et al. 1977; Riihimaki 1979). Exposure of humans to xylene vapors causes ocular irritation (Carpenter et al. 1975; Hake et al. 1981; Klaucke et al. 1982; Nelson et al. 1943).

Animal data provide additional evidence that dermal exposure to xylene produces dermal and ocular effects. These included skin erythema and edema, eschar formation in some animals, and epidermal thickening (Hine and Zuidema 1970). No studies were available regarding potential dermal/ocular effects in animals following exposure to xylene vapor.

Immunological Effects. Very limited human and no animal data are available to evaluate the immunological effects of xylene. Therefore, the relevance to public health is not known.

Neurological Effects. Neurological effects in humans following oral or dermal exposure to xylene have not been studied, although one case was reported of a man who developed a coma following ingestion of xylene (Recchia et al. 1985). Results of experimental studies with humans indicate that acute inhalation exposure to mixed xylene or m-xylene causes impaired short-term memory, impaired reaction time, performance decrements in numerical ability, and alterations in equilibrium and body balance (Gamberale et al. 1978; Riihimaki and Savolainen 1980; Savolainen et al. 1985; Savolainen et al. 1979b; Savolainen and Riihimaki 1981b; Savolainen and Linnavuo 1979; Savolainen et al. 1984). Available case and occupational studies together provide suggestive evidence that acute and chronic inhalation exposure to xylene or solvent mixtures containing xylene may be associated with many neurological effects and symptoms (Arthur and Curnock 1982; Goldie 1960; Hipolito 1980; Klaucke et al. 1982; Morley et al. 1970; Nersesian et al. 1985; Roberts et al. 1988). In several case reports, isolated instances of unconsciousness, amnesia, brain hemorrhage, and epileptic seizure have been associated in a limited number of individuals with acute inhalation exposure to solvent mixtures containing xylene (Arthur and Curnock 1982; Goldie 1960; Morley et al. 1970).

Results of experimental studies with animals provide further evidence that mixed xylene and individual isomers are neurotoxicants following inhalation exposure. Signs of neurotoxicity observed in rats, mice, and gerbils following acute and intermediate inhalation exposure to the various

xylene isomers have included narcosis, prostration, incoordination, tremors, muscular spasms, labored breathing, behavioral changes, hyperactivity, elevated auditory thresholds, hearing loss, changes in brain enzyme activity and changes in levels of brain proteins (Andersson et al. 1981; Carpenter et al. 1975; De Ceaurriz et al. 1983; Furnas and Hine 1958; Ghosh et al. 1987; Kyrklund et al. 1987; Molnar et al. 1986; NTP 1986; Pryor et al. 1987; Rank 1985; Rosengren et al. 1986; Savolainen and Seppalainen 1979; Savolainen et al. 1978; Savolainen et al. 1979a; Wimolwattanapun et al. 1987). No animal studies evaluating the neurological effects of xylene following chronic inhalation exposure were available.

Although a number of mechanisms of action have been proposed, the toxic mechanism of xylene on the nervous system is not fully understood. Because xylene is lipid soluble, it can distribute to the central nervous system. A number of investigators have noted the affinity of xylene for nervous system tissue, such as myelin and axonal membrane, in humans and animals (Desi et al. 1967; EPA 1985a; Gerarde 1959; Savolainen and Pfaffli 1980).

Neurological effects, including narcosis and anesthesia, are noted after acute exposure to high concentration of xylene when high blood and brain levels of the solvent occur (EPA 1985a). It has been suggested that xylene and other alkylbenzenes act simply by being in the nervous system at sufficiently high concentrations to inhibit normal function (Desi et al. 1967; EPA 1985a; Gerarde 1959). A number of experimental studies with humans on CNS function indicate that the first observable effects of m-xylene are on the central vestibular system, which controls equilibrium and body balance (Riihimaki and Savolainen 1980; Savolainen and Linnavuo 1979; Savolainen et al. 1979b; Savolainen and Riihimaki 1981b; Savolainen et al. 1984; Savolainen et al. 1985).

Also, xylene may directly affect nerve conductivity by altering the lipid components of the axonal membrane (EPA 1985a; Savolainen and Seppalainen 1979). Altered lipid components in turn could alter sodium permeability and decrease action potentials, resulting in signs of intoxication (EPA 1985a).

Results of experimental studies with rats suggest that mixed xylene and m-, o-, or p-xylene can cause alterations in dopamine and/or noradrenaline levels in the brain (Andersson et al. 1981). These changes can produce disturbances in catecholamine neurotransmission, which in turn can potentially alter brain function, particularly mental, motor, and neuroendocrine control (Andersson et al. 1981). Two possible modes of action have been suggested. Xylene or a metabolite of xylene could act directly on adrenergic receptors in the brain, causing increased catecholamines and postsynaptic stimulation. The second possibility involves alteration of axonal membrane fluidity, which causes permeability changes and alters neurotransmitter release (Andersson et al. 1981; EPA 1985a).

Some authors have also suggested that metabolic intermediates, such as arene oxides or methylbenzaldehyde, may be responsible for the toxic effects of xylene (Savolainen and Pfaffli 1980). Oxidation of xylene to these intermediates by microsomal enzyme systems may occur within brain cells (Savolainen and Pfaffli 1980).

Developmental Toxicity. Limited human studies were available regarding the developmental or teratogenic effects of xylene. However, because of concurrent exposure with chemical agents in addition to xylene, they cannot be used to assess the relationship between xylene exposure and developmental effects in humans. Findings in animal studies suggest that adverse effects might occur in the unborn and offspring of women exposed to xylene or its isomers. Results of studies with rats and mice indicate that inhalation exposure to mixed xylene or xylene isomers may induce increased fetal death, decreased fetal weight, delayed skeletal development, skeletal anomalies, enzymatic changes in fetal organs, and maternal toxicity (Hudak and Ungvary 1978; Marks et al. 1982; Mirkova et al. 1983; Ungvary et al. 1980b, 1981). Oral exposure to mixed xylene has been associated with cleft plate and decreased fetal weight (Marks et al. 1982). Dermal exposure of rats to xylene has been associated with biochemical changes in fetal and maternal brain tissue (Mirkova et al. 1979). However, p-xylene produced no developmental effects, with maternal toxicity, in rats (Rosen et al. 1986). These studies were generally limited but, taken together, suggest fetotoxic effects, although most of these may have been secondary to maternal toxicity.

The exact mechanism by which mixed xylene or its individual isomers produce toxic effects in fetuses has not been fully investigated. Based on results of studies with rats, p-xylene-induced delayed fetal development may have been caused by decreased levels of progesterone and estradiol (Ungvary et al. 1981). The titers of these hormones were apparently lowered due to xylene's inductive effect on metabolism, which caused increased hormone catabolism.

Reproductive Toxicity. The relevance to public health regarding xylene exposure and adverse reproductive effects is not known because of the limitations of the human and animal data. Occupational exposure of men to xylenes, in addition to other solvents, was found to increase the potential for their wives to experience spontaneous abortions, however, this study was limited by exposure of the men to other solvents and the limited size of the population studied (Taskinen et al. 1989). No reproductive effects were found in rats following inhalation of xylene before mating and during gestation and lactation (Bio/dynamics 1983). Histopathological examination following intermediate and chronic oral bioassays revealed no adverse effects on the reproductive organs of rats and mice (Hazleton Labs 1988a, 1988b; NTP 1986).

No other studies were located regarding reproductive effects in animals following inhalation or dermal exposure to xylene or its isomers.

Genotoxicity. Mixed xylene, as well as each of the individual xylene isomers, has been tested for genotoxicity in a variety of *in vitro* and *in vivo* assays. Results of the various assays indicate that mixed xylene and xylene isomers are nongenotoxic (Tables 2-10 and 2-11). As summarized in Table 2-10, the results of the various assays indicate that mixed xylene and xylene isomers are nongenotoxic following *in vitro* exposure (Bos et al. 1981; Connor et al. 1985; Florin et al. 1980; Haworth et al. 1983; Litton Bionetics 1978b; McCarroll et al. 1981a, 1981b; NTP 1986; Shimizu et al. 1985).

The induction of genotoxic effects following *in vivo* exposure to xylene has been evaluated in the bone marrow chromosomal aberration test with rats (Litton Bionetics 1978b), the bone marrow micronucleus test with mice (Mohtashamipur et al. 1985), and the sperm morphology test with rats (Washington et al. 1983). The incidence of sister-chromatid exchanges and chromosomal aberrations in the peripheral lymphocytes of workers exposed occupationally to xylene also has been evaluated (Haglund et al. 1980; Pap and Varga 1987). Both human studies involved occupational exposure to other chemicals in addition to xylene. As summarized in Table 2-11, the results of these studies indicate that mixed xylene, *m*-, *o*-, and *p*-xylene are nongenotoxic following *in vivo* exposure.

No mutagenic activity was demonstrated for any of the various metabolites of xylene in bacterial test systems. *S. typhimurium* strains TA98, TA100, TA1535, TA1537, and TA1538, with and without S9 metabolic activation, have been used to test the mutagenic activity of *p*-xylenol (Epler et al. 1979; Florin et al. 1980; Hejtmankova et al. 1979; Pool and Lin 1982), *m*-xylenol (Epler et al. 1979; Florin et al. 1980), and *o*-methylbenzyl alcohol (Bos et al. 1981). 2,4-Dimethylphenol has been evaluated in a gene reversion assay with *E. coli* strain Sd-4-73 (Szybalski 1958).

Ethylbenzene, a common component of many technical grades of mixed xylene, also demonstrated no mutagenic effects in the gene reversion assay with *S. cerevisiae* (Nestmann and Lee 1983), the *Salmonella*/microsome assay with strains TA98, TA100, TA1535, TA1537, and TA1538 (Florin et al. 1980; Nestmann et al. 1980), or in cytogenic assays with cultured Chinese hamster ovary cells (NTP 1986). However, in studies with cultured human lymphocytes, ethylbenzene induced a slight but statistically significant ($p<0.01$) increase in the number of the sister-chromatid exchanges (Norppa and Vainio 1983). The authors of this latter study suggested that ethylbenzene may be a "weak, ineffective mutagen." Ethylbenzene is the subject of a separate toxicological profile, and the reader should refer to that document for a more detailed review of its genotoxicity potential.

In summary, genotoxicity studies on mixed xylene and the individual isomers of xylene have provided consistently negative results in a variety of *in vitro* and *in vivo* assays and test systems (bacteria, yeast, insects, cultured mammalian cells, mice, rats, and humans). Based on the genotoxicity studies conducted to date, there is sufficient evidence to conclude that mixed xylene, *m*-xylene, *o*-xylene, and *p*-xylene are nonmutagenic. There is also limited evidence from bacterial test systems that suggest that xylene metabolites, specifically *m*-xylenol, *p*-xylenol, 2,4-dimethylphenol, and *o*-methylbenzyl alcohol, are nonmutagenic as well.

Cancer. No data were available regarding the development of cancer in humans following inhalation, oral, or dermal exposure to mixed xylene or individual isomers. Animal carcinogenicity data for the xylenes are limited to oral studies with mixed xylene (Maltoni et al. 1983, 1985; NTP 1986) and dermal studies in which the isomeric composition of the xylene was not specified, exposures were less than lifetime, and involved multiple chemicals (Berenblum 1941; Pound 1970; Pound and Withers 1963). No animal carcinogenicity data for the xylenes were available for inhalation exposure. Because of the limited data, no conclusions can be drawn regarding the relationship between xylene exposure and cancer in humans.

EPA has classified mixed xylene as a Group D agent (not classifiable as to human carcinogenicity) (IRIS 1989). This classification applies to those chemical agents for which there is inadequate evidence of carcinogenicity in animals. No cancer potency factor (q1*) or other quantitative estimate of carcinogenicity has been developed by EPA for mixed xylene, *m*-xylene, *o*-xylene, or *p*-xylene.

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TOXICOLOGICAL PROFILE FOR ZINC

Prepared by:

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In collaboration with:

U.S. Environmental Protection Agency

December 1989

ZINC

2.3 RELEVANCE TO PUBLIC HEALTH

Death. Death due to respiratory failure in humans following acute inhalation of zinc chloride has been reported. However, the amount of zinc exposure was not determined. Furthermore, exposure to zinc was concomitant with exposure to other chemicals. Hence, death could not be exclusively attributed to zinc exposure.

No information regarding death in animals following inhalation exposure was found. However, death was reported in ferrets (Straube et al. 1980) and mice (Maita et al. 1981) following acute and intermediate oral exposures,

respectively. Adverse systemic effects were observed in these animals, but the specific cause of death could not be determined.

Systemic Effects. Effects in humans and animals following acute inhalation exposure to zinc compounds are primarily limited to the respiratory tract. Oral exposure to zinc and its compounds in humans and animals primarily affects the gastrointestinal system. Zinc also affects the hematological and renal systems in both humans and animals following acute, intermediate or chronic exposures. Hepatic effects were observed in animals after acute oral exposure. No adverse hepatic effects were observed in humans after intermediate exposure.

Cardiovascular Effects. Intermediate duration oral administration of zinc to humans has resulted in decreased serum HDL-cholesterol levels. Although this is not a direct effect on the cardiovascular system, the decline in HDL levels may be associated with increased risk of coronary artery disease.

Gastrointestinal Effects. The ingestion of small amounts of zinc is essential to maintain one's health. However, evidence shows that high level ingestion of zinc presents a potential for gastrointestinal disorders. Following acute, intermediate or chronic ingestion of zinc, the primary effects in humans or animals are pancreatic abnormalities and gastrointestinal irritation. No adverse gastrointestinal effects were observed after inhalation exposure. Biochemical changes, including increased serum amylase levels and hypocalcemia, found in an individual after drinking a zinc chloride solution (unknown amount) are indicative of acute pancreatitis (Chobanian 1981). More severe effects are seen in the pancreas as exposure levels increase in various test animals. Although morphological changes, such as pancreatic fibrosis and degeneration and necrosis of acinar cells of the pancreas, are seen in most animal studies, these changes occur over a wide range of exposure levels (59 mg/kg/day in cats to 3,900 mg/kg/day in mice) (Aughey et al. 1977; Drinker et al. 1927d; Maita et al. 1981). This variability indicates the existence of species differences with regard to gastrointestinal disorders, specifically pancreatic toxicity. Zinc compounds, when ingested at high levels, also cause intestinal bleeding in humans (Moore 1978) and in animals (Maita et al. 1981; Straube et al. 1980).

Respiratory Effects. Respiratory disorders have been observed in humans and animals following the acute inhalation exposure to zinc compounds. No adverse respiratory effects have been observed following ingestion of zinc compounds.

Acute exposure to high concentrations of airborne zinc oxide in humans causes metal fume fever. Zinc oxide penetrates the alveoli, damages the lung tissue, and transiently impairs pulmonary function (Brown 1988; Drinker et al. 1927b; Vogelmeier et al. 1987). Lung volumes are decreased as is the carbon monoxide diffusion capacity (Drinker et al. 1972b; Mueller and Seger 1985;

respectively. Adverse systemic effects were observed in these animals, but the specific cause of death could not be determined.

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Sturgis et al. 1927). Metal fume fever is believed to be the result of an immune reaction to inhaled metal oxide particles (Mueller and Seger 1985).

Respiratory tract irritation occurs in both humans and animals (Drinker and Drinker 1928; Sturgis et al. 1927) after exposure to zinc oxide. Most laboratory animals, except guinea pigs, begin to present respiratory abnormalities (e.g., pulmonary congestion, peribronchial leukocytic infiltration) at similar exposure levels as humans (Drinker and Drinker 1928). Cats exhibited more severe effects than other animals, including bronchopneumonia (Drinker and Drinker 1928).

In humans, inhalation of zinc chloride causes greater damage to respiratory tissue than zinc oxide. Lesions reported were acute pneumonitis, ulceration of mucous membranes, subpleural hemorrhage, and pulmonary fibrosis. Exposed individuals may die with respiratory distress syndrome (Evans 1945; Hjortso et al. 1988; Johnson and Stonehill 1961; Matarese and Matthews 1966; Milliken et al. 1963; Schenker et al. 1981).

The studies in humans and animals reveal that inhalation of zinc as particulate or fume can result in respiratory ailments (Drinker and Drinker 1928; Sturgis et al. 1927). However, zinc fume or particles are primarily generated through grinding or welding of zinc-containing metal. This form of exposure presents a particular problem for industrial workers.

Hematological Effects. Anemia occurs in both humans and animals after high level acute, intermediate or chronic oral exposure to zinc and its compounds (Allen et al. 1983; Drinker et al. 1927c; Maita et al. 1981; Moore 1978; Straube et al. 1980). This effect occurs over a wide range of doses (6.47 mg/kg/day in humans to 3,900 mg/kg/day in mice), indicating species differences regarding the hematological effects of zinc. The anemia could be exacerbated by gastrointestinal bleeding that has been associated with the ingestion of high levels of zinc. Inhibition of intestinal absorption of copper and iron may also be a factor (Prasad et al. 1978). Based on the reviewed data, humans appear to be susceptible to these effects of zinc.

Anemia has been observed in patients after they were treated with renal dialysis for 2 months using water containing high levels of zinc. This suggests that anemia can result from treatments of intermediate duration. The zinc was presumed to have eluted from galvanized hinges in storage containers. Anemia was reversed after carbon filtration or deionization of the dialysis water (Gallery et al. 1972; Petrie and Row 1977).

Hepatic Effects. Following acute and intermediate oral exposure to zinc compounds, hepatic necrosis was reported in sheep (Allen et al. 1983). Intermediate oral exposure of rats to zinc compounds at a dose similar to that administered to sheep resulted in enzymatic changes (Kadiiska et al. 1985). Intermediate oral exposure of humans to zinc resulted in no adverse hepatic effects. No adverse hepatic effects were reported after inhalation exposure.

The weight of evidence suggests that the liver is not a primary target organ in humans or animals for zinc toxicity.

Renal Effects. No adverse renal effects have been observed in humans following inhalation or oral exposure to zinc compounds. Following intermediate oral exposure to zinc compounds, renal lesions have been reported in laboratory animals. Sheep were the most susceptible to renal toxicity (Allen et al. 1983).

Central Nervous System Effects. Of additional interest with respect to neurological symptoms and lethargy observed in human subjects following oral administration of zinc (Murphy 1970; Potter 1981) are in vitro investigations of the effects of zinc on neurological tissue. These studies indicate that zinc inhibits (competes for) the entry of calcium ions into the nerve terminals, thereby influencing the release of neurotransmitters (Nishimura 1987). In addition, zinc (at concentrations that may occur in vivo during certain pathophysiologic states) has been observed to be toxic to neurons and glia cells of the central nervous system (Choi et al. 1988; Yokoyama et al. 1986).

Reproductive and Developmental Effects. Oral exposure to high doses of zinc (200-500 mg/kg/day) has resulted in reduced fetal growth and altered fetal and maternal concentrations of zinc and copper in rats (Cox et al. 1969; Ketcheson et al. 1969; Schlicker and Cox 1967) and, in extreme cases, cessation of reproduction (Sutton and Nelson 1937).

Congenital malformations such as exencephaly and rib fusions have been observed in the offspring of pregnant golden hamsters injected intravenously with a single dose of 2 mg zinc sulfate/kg on the 8th day of gestation (Ferm and Carpenter 1968). Similarly, zinc chloride injected intraperitoneally in single doses of 12.5, 20.5, and 25 mg/kg on day 8, 9, 10, or 11 of gestation in mice produced skeletal anomalies, including delayed ossification and ripple ribs without accompanying soft tissue defects. Ripple ribs, an unusual anomaly, first appeared when zinc salt was given on day 9 of gestation at a dose of 20.5 mg/kg, becoming more prevalent when 25 mg/kg of the salt was administered on day 11 of gestation (Chang et al. 1977). There is no evidence of similar effects in humans.

Genotoxic Effects. Genotoxicity studies conducted in a variety of test systems have failed to provide evidence for mutagenicity of zinc. However, there are indications of weak clastogenic effects following zinc exposure. Results of in vitro studies are shown in Table 2-3. Exposure to zinc as zinc sulfate or zinc chloride does not increase mutation frequencies in bacterial or mammalian cell culture test systems (Amacher and Paillet 1980; Marzin and Vo Phi 1985; Nishioka 1975; Venitt and Levy 1974). Treatment of human lymphocytes in culture with zinc has, however, been observed to result in a slight increase in chromosomal aberrations (Deknudt and Deminatti 1978).

Results of in vivo studies are shown in Table 2-4. A dominant lethal study in mice was negative, indicating a lack of a mutagenic effect. However, chromosomal aberrations have been observed in bone marrow cells following exposure to zinc in vivo both in mice fed 650 mg/kg/day zinc chloride in diet (Deknudt 1982) and in mice exposed to zinc oxide by inhalation (Voroshilin et al. 1978). Chromosomal aberrations due to zinc were observed in bone marrow cells of mice maintained on a low calcium diet (Deknudt and Gerber 1979). Calcium may be displaced by zinc in calcium-depleted conditions, thus leading to chromosome breaks and/or interfering in the repair process (Deknudt and Gerber 1979).

Cancer. The carcinogenicity of zinc following oral exposure has been evaluated in a single study with mice (Walters and Roe 1965). In this study, mice were administered zinc sulfate at elemental zinc doses of either 0, 170, or 850 mg/kg/day in drinking water for 1 year. Relative to controls, no increase in tumor incidence was observed in treated mice. The investigation was not adequate for evaluating the carcinogenicity of zinc because of several study limitations. Teratomas of the testes were observed in fowl given testicular injections of 2 ml of a 10% zinc sulfate solution (Falin and Gromzewa 1939). The relevance of this study to public health is not known.

No studies were located regarding carcinogenicity in experimental animals following inhalation or dermal exposure to zinc and its compounds.