Urban Air Toxics Concentrations In Denver

May 2002 through April 2003

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Prepared by the

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Urban Air Toxics Concentrations In Denver

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Executive Summary

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Executive Summary

This report discusses results for ambient air toxics monitoring conducted at three locations, CAMP, Welby, and Swansea, in Denver during the period May 2002 through April 2003. The CAMP and Welby sites were sampled for a year, while Swansea sampled for six months. Twenty-four hour long samples were collected on a once every six day basis for a year as part of the U.S. Environmental Protection Agency's (EPA) Urban Air Toxics Monitoring Project (UATMP). The CAMP site was at 2105 Broadway in the central business district. The Welby site was at 78th Avenue and Steele Street, along the Platte River. This site is just north of the main industrial section of Denver, and also is in the path of downriver drainage winds from the downtown area. The Swansea site was located at an elementary school at 4650 Columbine Street. This location is in a mixed residential /industrial area between CAMP and Welby.

Aldehyde, volatile organic compound, and metal samples were taken with equipment provided by Eastern Research Group (ERG), a consulting firm contracted by EPA to provide support to the national network. The ERG samplers collected two different types of samples. A dinitrophenylhydrazine (DNPH) cartridge collected carbonyl samples by EPA Method TO-11A. DNPH cartridges were analyzed for twelve different carbonyls. Air was also drawn into a stainless steel canister. The canisters were analyzed for 58 volatile organic compounds (VOCs) by EPA method TO-15. In addition, high volume samplers collected total suspended particulate matter samples that were analyzed for eleven different metals. Thus, the total number of chemical compounds assessed was 81. Of the 81 chemicals assessed, 34 were detected less than 10 percent of the time.

Three carbonyls were present in all samples at all sites. These were formaldehyde, acetaldehyde, and acetone. However, levels of acetone and formaldehyde were well below the cancer screening concentration, so they were not retained in the risk analysis. Crotonaldehyde was present over 98% of the time, and is significant in the risk analyses. However, this crotonaldehyde significance is based on the use of a toxicity number based on an oral dose, not an inhalation dose. The inhalation health effects may be quite different. Automobiles are believed to be the largest emission source for formaldehyde, crotonaldehyde, and acetaldehyde, either as direct emissions, or as compounds forming from photochemical reactions. The impacts from aldehydes are difficult to control, because they can form as hydrocarbons emitted from automobiles and industrial processes react in the presence of sunlight. Analysis of results from the EPA national Urban Air Toxics Network indicates that acetaldehyde and acetone are problems on a nationwide scale. Levels of crotonaldehyde observed at the three sites are well within the normal range, as listed in the 2003 Urban Air Toxics Monitoring Program: Final Report. Thus, the situation in Denver is typical of most American cities.

Highest VOCs at Each Site

CAMP	**Welby**	**Swansea**
Acetylene	acetylene	acetylene
Propylene		propylene
Toluene	toluene	toluene
m,p - xylenes	m,p - xylenes	m,p - xylenes
o-xylenes		o-xylene
methyl ethyl ketone	methyl ethyl ketone	methyl ethyl ketone
dichlorodifluoromethane	dichlorodifluoromethane	dichlorodifluoromethane
acetonitrile		
trichlorofluoromethane		trichlorofluoromethane
benzene	benzene	benzene
1,2,4-trimethylbenzene		
		ethylbenzene

The highest volatile organic compounds at each site are shown in the table above. Except for methyl ethyl ketone, these compounds were detected 90 percent or more of the time, at all three monitoring locations. Methyl ethyl ketone detections varied from site-to-site, suggesting local influences. Results from EPA's national network indicate that 1,3-butadiene, benzene, tetrachloroethylene and carbon tetrachloride are also a problem on a nationwide scale. 1-3 butadiene and benzene are believed to result from automobile emissions, while carbon

tetrachloride and tetrachloroethylene are industrially-emitted compounds. Some other VOCs were present on a more localized basis, appearing at one site, but less often at the other. These are likely emitted from local industrial operations.

Tetrachloroethylene, or perchloroethylene, occurred at CAMP and Welby, but less than 7 percent of the time. It was never detected at Swansea. Concentrations suggest that this compound, used in dry cleaning, presents a greater than one-in-a-million risk of cancer. These results are consistent with EPA's national analyses, which indicate that levels of tetrachloroethylene are of concern in urban areas throughout the United States. p-Dichloro-benzene occurred less than 5 percent of the time at CAMP and Welby, and was never detected at Swansea. Although calculated annual averages indicate this compound may be a concern, the use of ½ the detection limit for all the non-detect days make these results highly uncertain. Unlike many of the others discussed, this compound appears to be a local problem.

Almost every metals sample had very low, but measurable, levels. Except for beryllium, all metals were detected in 100 percent of the samples. Lead and manganese were the metals detected at the highest concentrations. However, lead levels were well below the standards of 1.5 $\mu g/m^3$, as a monthly (Colorado standard) or a quarterly (federal standard) average. The levels of arsenic detected were low, were typical of other cities in Colorado, and were similar to other national air toxics monitoring sites. Manganese levels are believed to be related to smelting operations or may be naturally-occurring background levels.

In conclusion, a number of compounds related to vehicular emissions are present in Denver air. These are formaldehyde, acetaldehyde, benzene, and 1,3-butadiene. Carbon tetrachloride and tetrachloroethylene, which are from industrial sources, also may be a concern. Except for formaldehyde, these compounds appear to be at problem levels throughout the urban areas of the United States. Arsenic and manganese may also be of concern. Arsenic and chromium are present at low levels, while manganese may be from natural or industrial sources.

A screening-level risk assessment of the potential human health impacts from inhalation of air toxics was conducted using data collected from air monitoring stations in Denver, Colorado. The purpose of the evaluation was to determine if residents at any of these locations are being exposed to airborne concentrations of toxic air pollutants via inhalation that may pose unacceptable risks to human health. In general, this risk assessment can be considered a conservative estimate of the exposure assessment. For example, the chronic risk estimates are based on an individual that is exposed to the monitored concentrations over 70 years, for 24 hours per day. The potential human health implications of these exposures were characterized for both cancer and non-cancer health effects.

Total cancer risks were found to range from 1E-04 (100 excess cancers per 1 million individuals) to 2E-04 (200 excess cancers per 1 million individuals) across the various monitoring locations. These total risk estimates are based on all carcinogenic chemicals in this study including crotonaldehyde, which was deemed to have a highly uncertain toxicity value. Crotonaldehyde is one of the major contributors to the total risk at each monitoring station, with risk estimates for this chemical ranging from 4E-05 to 8E-05 (40 to 80 in a million). A range of "acceptable" health risk values for carcinogens has been historically proposed by U.S. EPA. Acceptability ranges from one in one million (1×10^{-06}) to one hundred per million (1×10^{-04}). The cancer risks are fairly comparable across all three sites. Total cancer risks for the CAMP monitoring location slightly exceeds the upper end of EPA's acceptable risk range with estimated total cancer risks of 2E-04, with or without crotonaldehyde. Most large urban areas in the United States exhibit aggregate or total carcinogenic risks in the 10^{-04} to 10^{-05} range.

Non-cancer risks were assessed at all monitoring stations by comparing the location specific exposure point concentration to a concentration that is considered to be without an appreciable risk of deleterious effects during a lifetime, for even the most sensitive individual. None of the individual chemicals that were assessed at any monitoring location were found to have a hazard quotient exceeding a value of one. Hazard indices for each monitoring station were calculated by summing hazard quotients of individual chemicals that contribute to specific categories of known critical effects. For all critical effects other than respiratory and neurologic, hazard indices did not exceed a level of one at any monitoring location. For non-carcinogenic estimation of hazard indices, an individual calculated index below one is generally regarded as an acceptable (or "safe") level of exposure.

Hazard indices of two were seen for respiratory effects at the CAMP and Swansea monitoring stations. The largest chemical contributors to the hazard indices at each of these locations were formaldehyde, acetaldehyde

and 1,2,4-trimethylbenzene. Hazard indices of two were also seen for neurologic effects at the CAMP, Welby, and Swansea monitoring stations. The largest chemical contributor to the hazard indices at each of the monitoring locations was manganese, which contributed to approximately 50% of the neurologic risk at each location. It is important to recognize that concentrations of manganese may be naturally occurring and investigations have not been conducted to determine if manganese represents background concentrations. These elevated hazard indices indicate that there may be a potential for respiratory and/or neurologic effects to occur in an individual exposed for 7 years or longer to air concentrations measured at several of the monitoring locations.

It should be noted that the results of this study and screening analysis are subject to some significant uncertainties. For example, EPA believes that acrolein contributes significantly to overall cancer risk. However, no monitoring method currently exists for acrolein in air. EPA is working to develop one for the future. Another uncertainty lies in the fact that polycyclic aromatic hydrocarbons (PAHs) were not monitored. Since diesel vehicles emit these compounds, an important vehicular air pollution source is absent from this analysis. Additionally, the study is based on one year of data collected at fixed monitoring stations. These fixed points may not adequately characterize exposure of a mobile population in a major metropolitan area. Most importantly, science is currently unable to assess exposures to multiple air toxics, simultaneously.

Calculations in this report use Colorado Department of Public Health and Environment and EPA's most recent, best estimates of a health risk values for each chemical compound. However, these health risk concentrations, as well as actual concentrations of chemicals in the air, change over time. Therefore, this study is best viewed as a "snapshot" in time.

A major goal of the study was to determine whether there are toxic compounds in air that are unique to Denver. If there are compounds that are significant locally, but not at the national level, then the EPA National Air Toxics Strategy may not be adequate to reduce air toxic cancer and non-cancer health risk in Colorado. The study results indicate that the compounds measured in Denver are the ones that EPA is focusing on nationally. For example, acetaldehyde and formaldehyde, two of the most important aldehydes monitored, are on the EPA list of Mobile Source Air Toxics (MSATs). The MSAT list of compounds contains 21 air toxics upon which the Environmental Protection Agency is focusing its control strategies. Benzene, 1,3-butadiene, toluene, and xylenes are also on this list. The two highest concentration metals observed in Denver, lead and manganese, are on the list. Acrolein and diesel exhaust, two air toxics this study did not address, are also MSAT targets.

It should also be noted that many of the compounds observed in Denver are also on EPA's list of 33 Urban Hazardous Air Pollutants (HAPs). These compounds are believed to be the most important contributors to inhalation risk from outdoor air. Acetaldehyde, formaldehyde, benzene, 1,3-butadiene, lead and manganese are on this list, just as they are on the MSAT list above. Tetrachloroethylene, while not an MSAT, is on this list of Urban HAPs. Crotonaldehyde is not on the EPA MSAT or HAPs lists, but Denver levels are within the range cited in national results for the Urban Air Toxics Monitoring Network in 2003. EPA's failure to list this compound is probably due to the fact that it lacks inhalation reference doses or cancer risk factors. However, strategies directed against acetaldehyde and formaldehyde will likely be effective in reducing crotonaldehyde. The annual report of the 2003 nationwide UATMP results gives a range of 0.03 – 2.69 ppbv observed throughout the network. The range for the Denver sites was 0.21 – 2.20 ppbv. The study did not identify any compounds that were of local-only significance.

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Section 1 - Introduction

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Introduction

This report discusses results for ambient air toxics monitoring conducted at three locations in Denver during the year May 2002 through April 2003. As part of the U.S. Environmental Protection Agency's (EPA) Urban Air Toxics Monitoring Project (UATMP), twenty-four hour samples were collected on a once every six day schedule for a year at two sites, and for a six-month period at a third site. Aldehyde, volatile organic compound and metals samples were taken with equipment provided by Eastern Research Group (ERG), a consulting firm contracted by EPA to provide support to the national network. The ERG samplers collected two different types of samples. A dinitrophenylhydrazine (DNPH) cartridge collected carbonyl samples by EPA Method TO-11A. DNPH cartridges were analyzed for twelve different carbonyls. Air was also drawn into a stainless steel canister. The canister samples were analyzed for 58 volatile organic compounds (VOCs) by EPA method TO-15. In addition, high volume samplers collected total suspended particulate matter (TSP) samples that were analyzed for eleven different metals. Thus, the total number of chemical compounds assessed was 81.

These compounds are believed, based on EPA analyses, to cover many of the most important air pollutants. However, it should be noted that these 81 compounds are a very limited subset of the pollutants potentially present in ambient air. For example, this report does not discuss six pollutants that have National Ambient Air Quality Standards (NAAQS). Four of these are compounds: carbon monoxide, ozone, nitrogen dioxide, and sulfur dioxide. These NAAQS compounds are regulated differently than the air toxics discussed in this report. The other two NAAQS pollutants not covered here concern particulate matter concentrations in air, measured as PM_{10} or $PM_{2.5}$.These are not discussed in this report, as they are mixtures of many different chemical compounds. Further information about NAAQS pollutants can be found on the web at http://www.epa.gov/ttn/naaqs/.

The air monitoring project discussed in this report did not include monitoring for semi-volatile organic compounds. Thus, potential health effects for inhalation of pesticides or diesel emissions cannot be assessed. These compounds have not been monitored in recent years, except at some specific hazardous waste site clean-ups.

The results section of this report is separated into chapters by the monitoring method employed. Thus, one chapter discusses the carbonyls, one presents volatile organic compound (VOC) information, and the next one summarizes the metals analyzed by the ICP method. Sections 2 to 4 each follow the same format. They begin with a summary of statistics for the compounds analyzed, then discuss the percentage of samples in which each chemical was detected. Some summary graphs of certain compounds are presented. The section then presents a brief discussion of quality assurance statistics, such as blank and precision results, that are available upon request to the Colorado Department of Public Health and Environment (CDPHE). Section 5 presents a risk assessment, estimating whether measured concentrations are likely to cause adverse health effects. Attachment 1 discusses sources and health effects for compounds that have a hazard quotient of 0.1 or greater, or contribute more than 1 % to total cancer risk calculated for the compounds sampled at the site. This Attachment gives a brief summary of each chemical's use, air emission sources, and air concentration.

Site Information

The Urban Air Toxics Pilot Project at Denver, Colorado sampled at three separate locations. The first was the downtown Denver CAMP station at 2105 Broadway. This site was chosen because it has existing monitoring for CO, SO_2, NO_2, TSP, lead, $PM_{2.5}$ and PM_{10}, thus providing a wealth of contemporaneous air pollution data. The site represents public exposure in a downtown core business area, with lots of traffic. The second site was the Welby site at 78^{th} Avenue and Steele Street. It is another long-term air pollution monitoring site, with CO, SO_2, NO_2, ozone and PM_{10} monitoring. It is located on the northern edge of the most densely–populated area of Denver, along the South Platte River. An industrial area is located to the south. The Welby site provides an excellent location for measuring air pollution as it drains out of the city, down the river. The third station was the Swansea Elementary School, located at 4650 Columbine Street. The school is located next to the elevated viaduct for highway I-70. It is in an area of mixed industrial and residential use. The area has a number of small businesses that emit toxic air pollutants, such as automotive repair facilities, printing operations, and metalworking facilities. A large processing plant for animal feeds is nearby. Several blocks to the east, there is a large truck refueling center. Due to the large number of businesses in the area, the neighborhood has a lot of diesel truck traffic. Residents of the area have been requesting air pollution sampling for a number of years. This site was chosen for six months of monitoring, to

assess how concentrations in this environmental justice area compare with those in other portions of the city. Photographs of these three locations follow in Figures 1.1 through 1.3.

Meteorological data were available from the CAMP and Welby stations, which have permanent towers. A city map showing all three stations follows (Figure 1.4).

Figure 1.1 - Denver – CAMP Site Photos
2105 Broadway

CAMP - Looking North

CAMP - Looking East

CAMP - Looking South

CAMP - Looking West

Figure 1.2 - Denver – Welby Site Photos
78th Avenue and Steele Street

Welby - Looking North

Welby - Looking East

Welby - Looking South

Welby - Looking West

Figure 1.3 - Denver – Swansea Site Photos
4650 Columbine Street

Swansea - Looking North

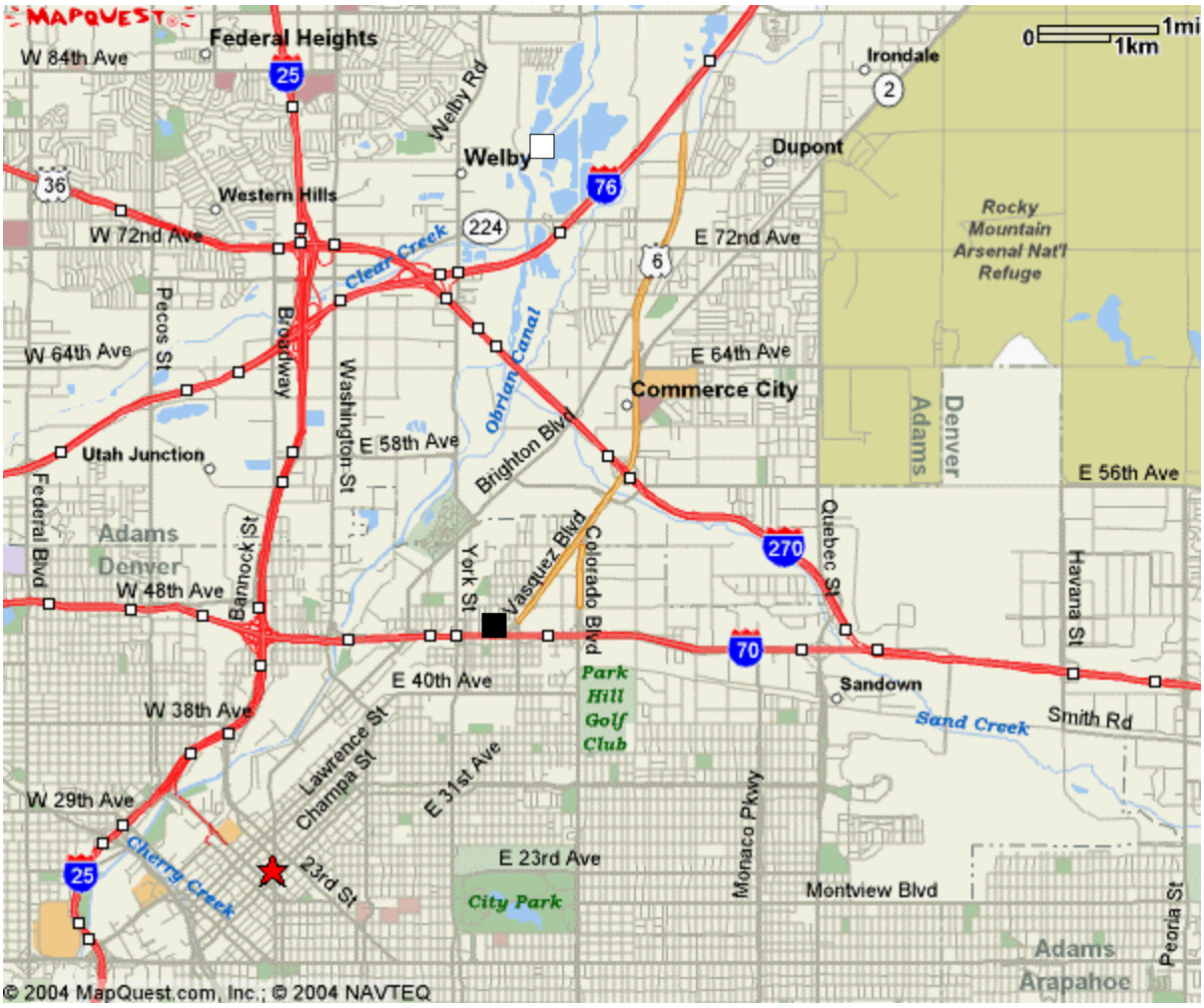
Swansea - Looking East

Swansea - Looking South

Swansea - Looking West

Swansea – VOC/Carbonyl Sample Probe

Figure 1.4 - Denver Sites Map



Red Star = CAMP Site, 2105 Broadway
White Square = Welby, 78th Avenue and Steele Street
Black Square = Swansea, 46th Avenue and Columbine Street

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Section 2 - Carbonyls at Denver Stations

May 2002 to April 2003

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Summary Statistics - Carbonyls

Maximum and Mean – All Samples

Carbonyl data collected at the Denver stations from May 2002 through April 2003 are presented in this section. Carbonyls were sampled on a one-in-six day basis for a year, for a total of 60 samples attempted at CAMP and Welby. During the half-year of sampling at Swansea, 30 samples were attempted. A few samples were lost due to equipment problems, such as inadequate sample run times. However, all sites met the EPA goal for over 85 percent sample recovery. (See Table 2.1).

Tables 2.2 through 2.4 summarize the annual maximum and mean concentrations for each carbonyl compound measured during the study. Results show that the most prevalent carbonyls in Denver air are formaldehyde, acetone, and acetaldehyde, in that order. The other nine carbonyl compounds measured occur at concentration levels significantly below those of these top three compounds.

Table 2.1 - Percentage Data Recovery For Carbonyl Samples

Station	Sample Days Scheduled	Samples Recovered	Percentage Recovered
Denver - CAMP	**60**	**58**	**96.7%**
Denver - Welby	**60**	**53**	**88.3%**
Denver - Swansea	**30**	**29**	**96.7%**

It should be noted that the annual means reported in Tables 2.2 through 2.4 were calculated by substituting one-half of the detection level for the “non-detect” days. This calculation method should not significantly affect the annual means for substances that were at measurable levels most of the time. All of the carbonyls, except for isovaleraldehyde and 2,5-dimethylbenzaldehyde, were present at least 90 percent of the time. However, the true annual means of isovaleraldehyde and 2,5-dimethylbenzaldehyde may be well below the numbers reported here.

Percentage of Samples For Which Compound Was Detected

Tables 2.2 through 2.4 show that most of these compounds were present in air over 90 percent of the time the air was sampled. However, isovaleraldehyde and 2,5-dimethylbenzaldehyde were seen less frequently, with detections in less than one-quarter of the air samples taken. This frequency of occurrence is similar to that noted in the 2000 – 2001 study of similar compounds in downtown Denver.

Table 2.2 - Carbonyl Compounds Data Summary – 24 Hour Samples at CAMP Site

CAMP Site	Summary Statistics (ug/m3)		Count of Non-Detects		Percentage of Samples In Which Compound Was Detected
	Maximum	Mean	Number	Percentage	
Formaldehyde	18.20	8.15	0	0	100
Acetaldehyde	9.29	4.14	0	0	100
Acetone	13.17	6.62	0	0	100
Propionaldehyde	1.34	0.45	0	0	100
Crotonaldehyde	0.37	0.12	0	0	100
Butyr/Isobutyraldehyde	2.26	0.86	0	0	100
Benzaldehyde	1.06	0.44	0	0	100
Isovaleraldehyde	0.36	0.01	54	93.1	6.9
Valeraldehyde	1.22	0.37	0	0	100
Tolualdehydes	1.18	0.43	1	1.7	98.3
Hexaldehyde	2.60	0.67	0	0	100
2,5-Dimethylbenzaldehyde	0.19	0.01	47	81.0	19.0

Table 2.3 - Carbonyl Compounds Data Summary – 24 Hour Samples at Welby Site

Welby Site	Summary Statistics (ug/m3)		Count of Non-Detects		Percentage of Samples In Which Compound Was Detected
	Maximum	Mean	Number	Percentage	
Formaldehyde	7.46	3.49	0	0	100
Acetaldehyde	6.40	2.87	0	0	100
Acetone	8.41	4.30	0	0	100
Propionaldehyde	0.89	0.31	1	1.9	98.1
Crotonaldehyde	0.28	0.08	1	1.9	98.1
Butyr/Isobutyraldehyde	1.00	0.50	0	0	100
Benzaldehyde	0.51	0.19	0	0	100
Isovaleraldehyde	0.04	0.01	52	98.1	1.9
Valeraldehyde	0.35	0.12	0	0	100
Tolualdehydes	0.80	0.21	0	0	100
Hexaldehyde	0.44	0.17	0	0	100
2,5-Dimethylbenzaldehyde	0.26	0.01	50	94.3	5.7

Table 2.4 - Carbonyl Compounds Data Summary – 24 Hour Samples at Swansea Site

Swansea Site	Summary Statistics (ug/m3)		Count of Non-Detects		Percentage of Samples In Which Compound Was Detected
	Maximum	Mean	Number	Percentage	
Formaldehyde	9.42	5.01	0	0	100
Acetaldehyde	5.55	3.26	0	0	100
Acetone	8.37	4.77	0	0	100
Propionaldehyde	0.73	0.36	0	0	100
Crotonaldehyde	0.14	0.06	0	0	100
Butyr/Isobutyraldehyde	0.83	0.54	0	0	100
Benzaldehyde	1.01	0.28	0	0	100
Isovaleraldehyde	0.05	0.01	28	96.6	3.4
Valeraldehyde	0.29	0.16	0	0	100
Tolualdehydes	0.54	0.30	0	0	100
Hexaldehyde	0.33	0.20	0	0	100
2,5-Dimethylbenzaldehyde	0.27	0.02	24	82.8	17.2

Graphs - Carbonyls

The carbonyl compounds measured during the study are graphed in Figures 2.1 through 2.4. Figure 2.1 shows that CAMP generally had the highest mean levels, followed by Swansea, and then Welby. According to Figure 2.2, CAMP had the highest daily maximums for all compounds, with Swansea and Welby maxima being about equal. Figures 2.3 and 2.4 show daily results for formaldehyde and acetaldehyde. For formaldehyde, the spring/summer period (May through September) showed higher concentrations than the rest of the year. For acetaldehyde, the values did not show much seasonal variation. Generally, concentrations of these two compounds rise and fall together, suggesting a common emissions source. Figure 2.5 shows that crotonaldehyde was at very low concentrations, but increased a bit during spring.

Figure 2.1 - Average Carbonyls at Sites

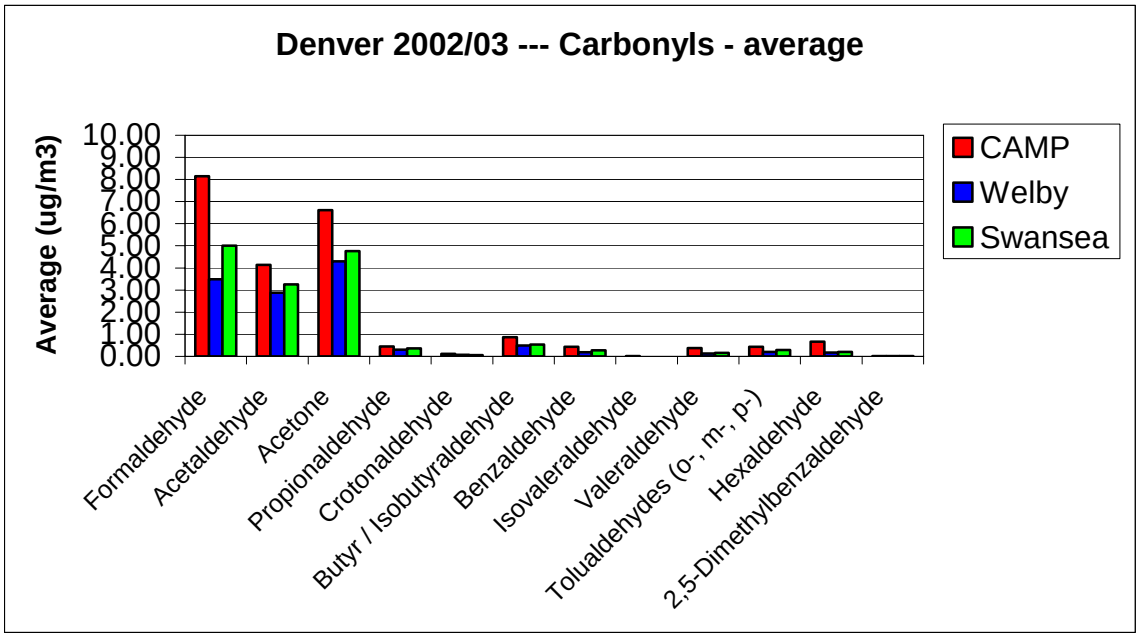


Figure 2.2 - Maximum Carbonyls at Sites

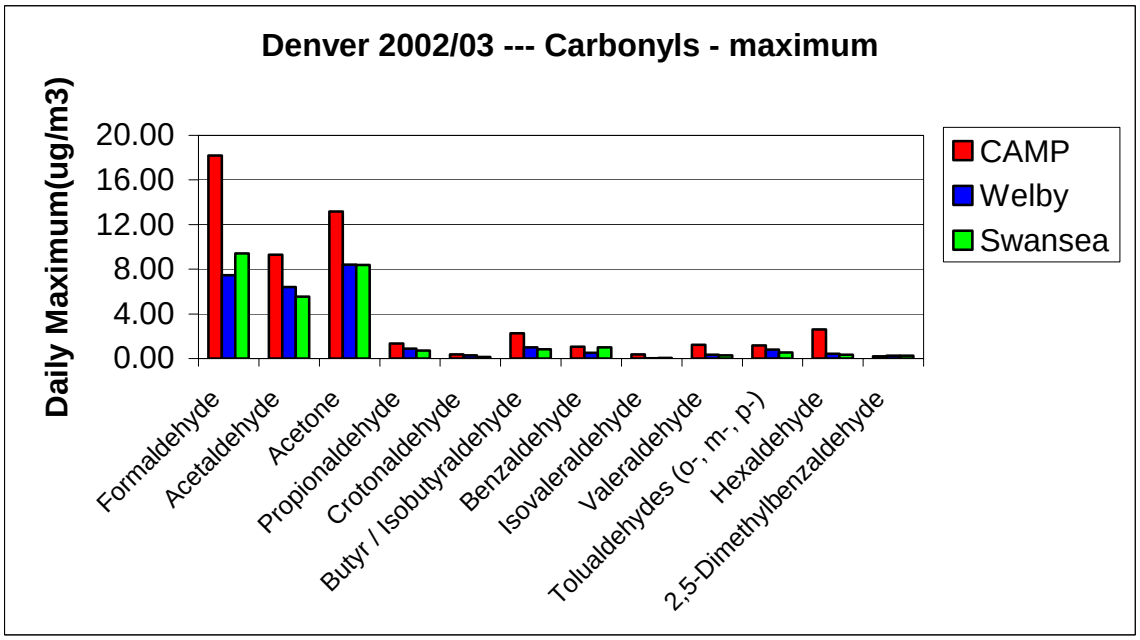


Figure 2.3 - Formaldehyde At Sites

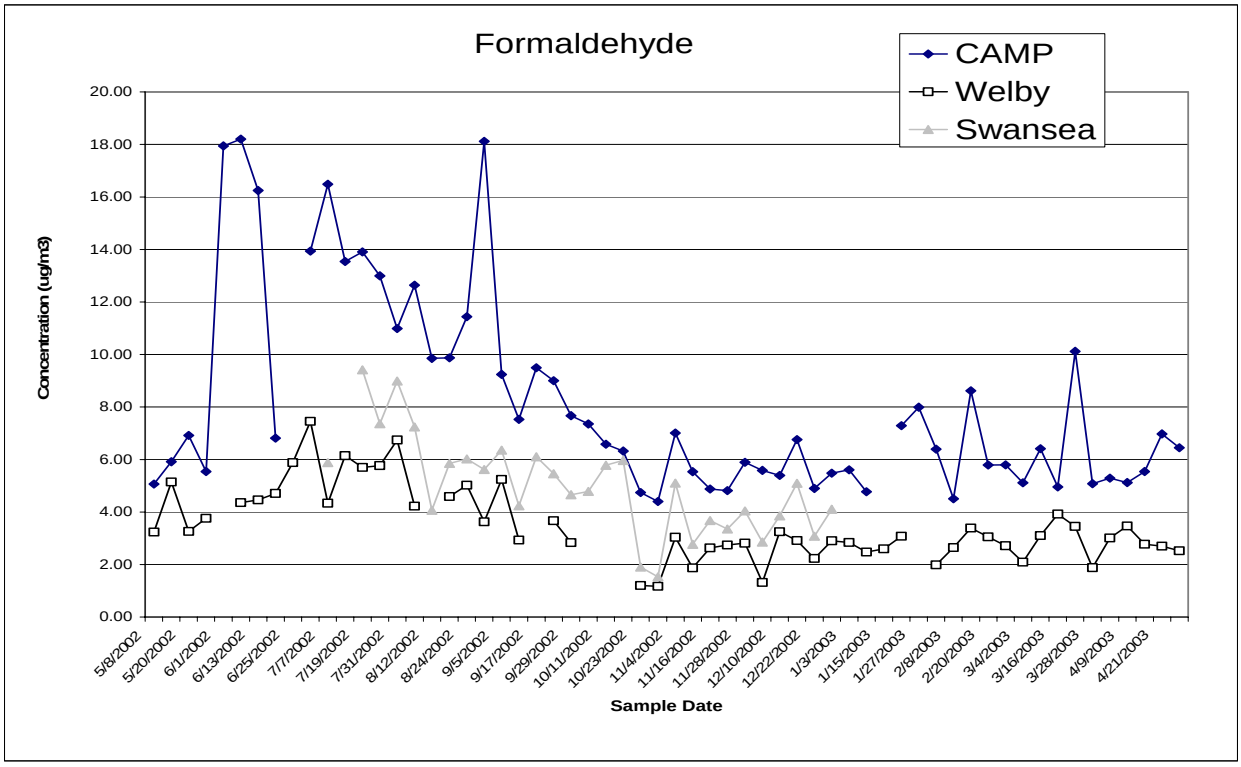


Figure 2.4 - Acetaldehyde At Sites

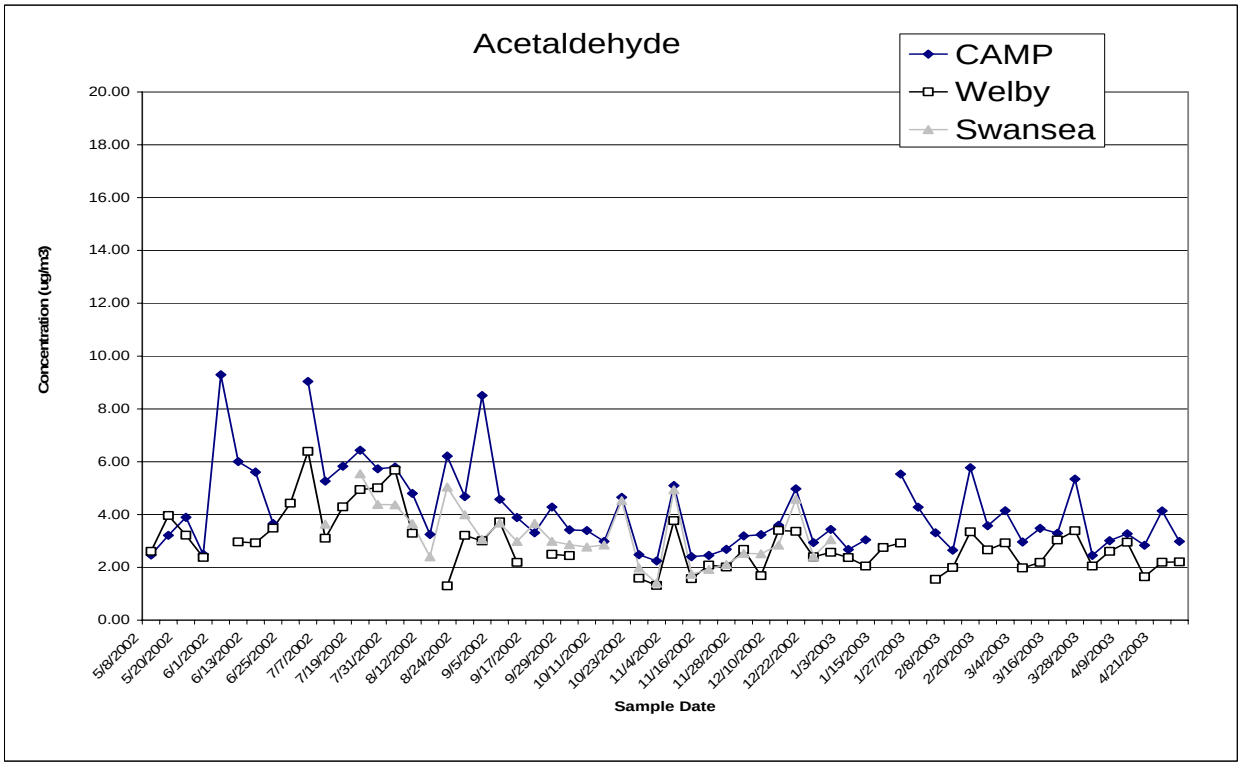
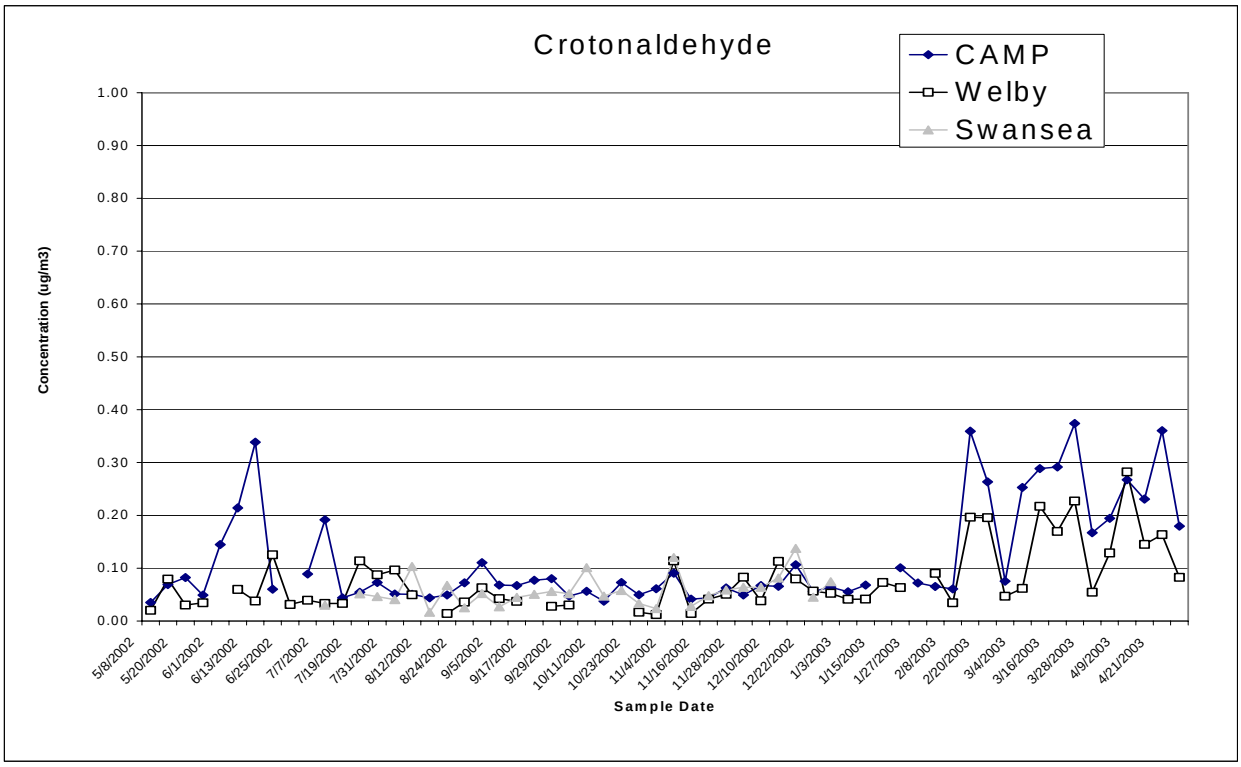


Figure 2.5 - Crotonaldehyde At Sites



Precision of Sample Results - Carbonyls

Periodically throughout the year, a second carbonyl cartridge was sampled simultaneously with the main sample. These additional samples, known as duplicates, were collected in order to assess the precision (repeatability) of the carbonyl sampling method. On the duplicate sampling dates, the laboratory also conducted a test of the precision of the analytical process by injecting two samples of each cartridge's liquid extract into the liquid chromatograph/ mass spectrometer. These samples are known as the laboratory replicates. Thus, this project collected two types of precision data – duplicate data, which assesses both sampling and analysis procedures, and replicate data, which assesses laboratory analytical method repeatability. Detailed information regarding precision and laboratory replicate results is available upon request.

Field Blanks - Carbonyls

Field blanks were periodically taken by attaching a blank DNPH cartridge to the sampler briefly, and then removing it. The purpose of these blanks was to assess contamination that might exist in the cartridge media, or contamination that might occur in sample installation or shipping. Most cartridges had small amounts of formaldehyde, acetaldehyde, and acetone. The other nine compounds occasionally had detectable amounts on the blanks. Detailed information regarding field blank results is available upon request.

Section 3 - Volatile Organic Compounds at Denver Stations

May 2002 to April 2003

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Summary Statistics - Volatile Organic Compounds

Maximum and Mean - All Samples

Volatile organic compound (VOC) data collected at the Denver stations from May 2002 through April 2003 are presented in this section. Volatile organic compounds were sampled at CAMP and Welby for one year on a one-in-six day basis, for a total of 60 samples attempted. Of these, the laboratory successfully processed 57 and 52, for percentage data recovery rates exceeding 87. The Swansea site was a special study location that ran for six months, with 97 percent data recovery (see Table 3.1).

Tables 3.2 through 3.4 summarize the annual maximum and mean concentrations for each of the 58 volatile organic compounds measured during the study. It should be noted that the annual means were calculated by replacing all "non-detect" values with one-half of the sample detection limit. This is an accepted conservative technique for calculating annual values when some of the samples were less than the laboratory's ability to measure.

Table 3.1 - Percentage Data Recovery For Volatile Organic Compound Samples

Station	Sample Days Scheduled	Samples Recovered	Percentage Recovered
CAMP	**60**	**57**	**95**
Welby	**60**	**52**	**87**
Swansea	**31**	**30**	**97**

Table 3.2 - Volatile Organic Compound Data Summary – CAMP

CAMP Site	Summary Statistics (ug/m3)		Count of Non-Detects		Percentage of Samples In Which Compound Was Detected
	Maximum	Mean	Number	Percentage	
Acetylene	10.96	2.92	0	0	100
Propylene	5.27	2.35	0	0	100
Dichlorodifluoromethane	5.24	3.21	0	0	100
Chloromethane	2.27	1.31	0	0	100
Dichlorotetrafluoroethane	0.38	0.27	57	100	0
Vinyl Chloride	0.17	0.13	57	100	0
1,3-Butadiene	0.95	0.32	10	18	82
Bromomethane	0.25	0.23	57	100	0
Chloroethane	0.17	0.17	57	100	0
Acetonitrile	87.14	2.44	47	82	18
Trichlorofluoromethane	4.66	2.15	0	0	100
Acrylonitrile	5.56	0.61	56	98	2
1,1-Dichloroethene	0.22	0.20	56	98	2
Methylene Chloride	31.89	1.40	4	7	93
Trichlorotrifluoroethane	1.99	0.83	1	2	98
trans - 1,2 - Dichloroethylene	0.24	0.16	57	100	0
1,1 - Dichloroethane	0.16	0.15	57	100	0
Methyl tert-Butyl Ether	0.41	0.37	57	100	0
Methyl Ethyl Ketone	34.89	2.57	34	60	40
Chloroprene	0.13	0.10	57	100	0
cis-1,2-Dichloroethylene	0.28	0.24	57	100	0
Bromochloromethane	0.56	0.39	57	100	0
Chloroform	0.83	0.19	48	84	16
Ethyl tert-Butyl Ether	0.38	0.35	57	100	0
1,2 - Dichloroethane	0.22	0.21	56	98	2
1,1,1 - Trichloroethane	4.15	0.30	45	79	21
Benzene	7.38	3.17	0	0	100
Carbon Tetrachloride	1.13	0.47	7	12	88
tert-Amyl Methyl Ether	0.38	0.36	57	100	0
1,2 - Dichloropropane	0.16	0.16	57	100	0
Ethyl Acrylate	0.68	0.64	57	100	0
Bromodichloromethane	0.30	0.26	57	100	0
Trichloroethylene	3.17	0.41	53	93	7
Methyl Methacrylate	0.82	0.63	56	98	2
cis -1,3 - Dichloropropene	0.25	0.24	57	100	0
Methyl Isobutyl Ketone	2.70	0.53	55	96	4

CAMP Site	Summary Statistics (ug/m3)		Count of Non-Detects		Percentage of Samples In Which Compound Was Detected
	Maximum	Mean	Number	Percentage	
trans - 1,3 - Dichloropropene	0.30	0.26	57	100	0
1,1,2 - Trichloroethane	0.52	0.43	57	100	0
Toluene	50.35	9.81	0	0	100
Dibromochloromethane	0.43	0.38	57	100	0
1,2-Dibromoethane	0.42	0.37	57	100	0
n-Octane	5.42	0.71	18	32	68
Tetrachloroethylene	10.58	0.64	34	60	40
Chlorobenzene	0.23	0.21	57	100	0
Ethylbenzene	3.13	1.54	0	0	100
m,p - Xylene	10.29	4.48	0	0	100
Bromoform	0.67	0.57	57	100	0
Styrene	1.45	0.33	31	54	46
1,1,2,2 - Tetrachloroethane	0.65	0.54	57	100	0
o - Xylene	5.04	2.08	0	0	100
1,3,5-Trimethylbenzene	1.77	1.09	0	0	100
1,2,4-Trimethylbenzene	5.06	3.07	0	0	100
m - Dichlorobenzene	0.54	0.48	57	100	0
Chloromethylbenzene	0.36	0.32	57	100	0
p - Dichlorobenzene	0.45	0.43	54	95	5
o - Dichlorobenzene	0.51	0.46	57	100	0
1,2,4-Trichlorobenzene	0.59	0.47	57	100	0
Hexachloro-1,3-Butadiene	1.07	0.89	57	100	0

Table 3.2, completed.

Table 3.3 - Volatile Organic Compound Data Summary - Welby

Welby Site	**Summary Statistics (ug/m3)**		**Count of Non-Detects**		**Percentage of Samples In Which Compound Was Detected**
	Maximum	**Mean**	**Number**	**Percentage**	
Acetylene	7.42	2.30	0	0	100
Propylene	5.82	1.70	0	0	100
Dichlorodifluoromethane	4.30	2.97	0	0	100
Chloromethane	1.98	1.22	0	0	100
Dichlorotetrafluoroethane	0.38	0.27	52	100	0
Vinyl Chloride	0.17	0.13	52	100	0
1,3-Butadiene	0.93	0.22	21	40	60
Bromomethane	5.71	0.39	50	96	4
Chloroethane	0.17	0.17	52	100	0
Acetonitrile	VOID	VOID			
Trichlorofluoromethane	7.47	1.82	0	0	100
Acrylonitrile	0.56	0.51	52	100	0
1,1-Dichloroethene	0.22	0.21	52	100	0
Methylene Chloride	5.77	0.67	11	21	79
Trichlorotrifluoroethane	1.53	0.76	3	6	94
trans - 1,2 - Dichloroethylene	0.24	0.16	52	100	0
1,1 - Dichloroethane	0.16	0.15	52	100	0
Methyl tert-Butyl Ether	7.46	1.79	28	54	46
Methyl Ethyl Ketone	33.21	3.13	30	58	42
Chloroprene	0.13	0.10	52	100	0
cis-1,2-Dichloroethylene	0.28	0.24	52	100	0
Bromochloromethane	0.56	0.40	52	100	0
Chloroform	0.44	0.19	46	88	12
Ethyl tert-Butyl Ether	0.38	0.35	52	100	0
1,2 - Dichloroethane	0.22	0.21	52	100	0
1,1,1 - Trichloroethane	0.49	0.21	43	83	17
Benzene	7.19	2.47	0	0	100
Carbon Tetrachloride	1.01	0.43	9	17	83
tert-Amyl Methyl Ether	0.38	0.36	52	100	0
1,2 - Dichloropropane	0.16	0.16	52	100	0
Ethyl Acrylate	0.68	0.63	52	100	0
Bromodichloromethane	0.30	0.26	52	100	0
Trichloroethylene	0.48	0.34	50	96	4
Methyl Methacrylate	0.74	0.61	52	100	0
cis -1,3 - Dichloropropene	0.25	0.24	52	100	0
Methyl Isobutyl Ketone	41.54	1.49	46	88	12
trans - 1,3 - Dichloropropene	0.30	0.27	52	100	0
1,1,2 - Trichloroethane	0.52	0.42	52	100	0
Toluene	68.82	8.72	0	0	100

Welby Site	**Summary Statistics (ug/m3)**		**Count of Non-Detects**		**Percentage of Samples In Which Compound Was Detected**
	Maximum	**Mean**	**Number**	**Percentage**	
Dibromochloromethane	0.43	0.38	52	100	0
1,2-Dibromoethane	0.42	0.37	52	100	0
n-Octane	1.40	0.50	25	48	52
Tetrachloroethylene	1.90	0.36	38	73	27
Chlorobenzene	0.23	0.22	52	100	0
Ethylbenzene	3.52	1.44	3	6	94
m,p - Xylene	12.07	4.46	0	0	100
Bromoform	0.67	0.56	52	100	0
Styrene	2.60	0.27	43	83	17
1,1,2,2 - Tetrachloroethane	0.65	0.53	52	100	0
o - Xylene	4.04	1.81	1	2	98
1,3,5-Trimethylbenzene	1.08	0.38	26	50	50
1,2,4-Trimethylbenzene	3.39	1.15	3	6	94
m - Dichlorobenzene	0.54	0.48	52	100	0
Chloromethylbenzene	0.36	0.32	52	100	0
p - Dichlorobenzene	0.45	0.45	50	96	4
o - Dichlorobenzene	0.51	0.46	52	100	0
1,2,4-Trichlorobenzene	0.59	0.48	52	100	0
Hexachloro-1,3-Butadiene	1.07	0.90	52	100	0

(Note: Acetonitrile VOID at Welby and Swansea due to contamination in sampler.)

Table 3.3, completed.

Table 3.4 - Volatile Organic Compound Data Summary - Swansea

Swansea Site	**Summary Statistics (ug/m3)**		**Count of Non-Detects**		**Percentage of Samples In Which Compound Was Detected**
	Maximum	**Mean**	**Number**	**Percentage**	
Acetylene	10.92	2.90	0	0	100
Propylene	6.28	2.06	0	0	100
Dichlorodifluoromethane	4.75	3.12	1	3	97
Chloromethane	1.65	1.16	1	3	97
Dichlorotetrafluoroethane	0.38	0.23	29	97	3
Vinyl Chloride	0.17	0.12	30	100	0
1,3-Butadiene	1.04	0.28	7	23	77
Bromomethane	0.25	0.22	30	100	0
Chloroethane	0.17	0.17	30	100	0
Acetonitrile	VOID	VOID			
Trichlorofluoromethane	14.21	2.26	1	3	97
Acrylonitrile	2.08	0.60	29	97	3
1,1-Dichloroethene	0.22	0.20	30	100	0
Methylene Chloride	3.09	0.78	7	23	77
Trichlorotrifluoroethane	1.38	0.70	1	3	97
trans - 1,2 - Dichloroethylene	0.24	0.13	30	100	0
1,1 - Dichloroethane	0.16	0.16	30	100	0
Methyl tert-Butyl Ether	0.41	0.40	30	100	0
Methyl Ethyl Ketone	24.72	2.94	19	63	37
Chloroprene	0.13	0.09	30	100	0
cis-1,2-Dichloroethylene	0.28	0.22	30	100	0
Bromochloromethane	0.56	0.34	30	100	0
Chloroform	0.78	0.20	26	87	13
Ethyl tert-Butyl Ether	0.38	0.37	30	100	0
1,2 - Dichloroethane	0.22	0.20	30	100	0
1,1,1 - Trichloroethane	0.27	0.16	26	87	13
Benzene	7.03	2.80	0	0	100
Carbon Tetrachloride	1.01	0.47	4	13	87
tert-Amyl Methyl Ether	0.38	0.37	30	100	0
1,2 - Dichloropropane	0.16	0.16	30	100	0
Ethyl Acrylate	0.68	0.66	30	100	0
Bromodichloromethane	0.30	0.24	30	100	0
Trichloroethylene	0.46	0.29	30	100	0
Methyl Methacrylate	0.74	0.70	30	100	0
cis -1,3 - Dichloropropene	0.25	0.25	30	100	0
Methyl Isobutyl Ketone	1.88	0.53	27	90	10
trans - 1,3 - Dichloropropene	0.30	0.25	30	100	0
1,1,2 - Trichloroethane	0.52	0.49	30	100	0
Toluene	63.62	11.26	0	0	100
Dibromochloromethane	0.43	0.41	30	100	0

Swansea Site	**Summary Statistics (ug/m3)**		**Count of Non-Detects**		**Percentage of Samples In Which Compound Was Detected**
	Maximum	**Mean**	**Number**	**Percentage**	
1,2-Dibromoethane	0.42	0.41	30	100	0
n-Octane	1.68	0.54	10	33	67
Tetrachloroethylene	2.98	0.72	16	53	47
Chlorobenzene	0.23	0.21	30	100	0
Ethylbenzene	6.38	2.32	0	0	100
m,p - Xylene	14.68	7.18	0	0	100
Bromoform	0.67	0.64	30	100	0
Styrene	3.66	0.37	25	83	17
1,1,2,2 - Tetrachloroethane	0.65	0.62	30	100	0
o - Xylene	5.73	2.97	0	0	100
1,3,5-Trimethylbenzene	1.43	0.52	5	17	83
1,2,4-Trimethylbenzene	4.62	1.51	0	0	100
m - Dichlorobenzene	0.54	0.52	30	100	0
Chloromethylbenzene	0.36	0.35	30	100	0
p - Dichlorobenzene	0.45	0.45	30	100	0
o - Dichlorobenzene	0.51	0.50	30	100	0
1,2,4-Trichlorobenzene	0.59	0.43	30	100	0
Hexachloro-1,3-Butadiene	1.07	0.83	30	100	0

(Note: Acetonitrile VOID at Welby and Swansea due to contamination in sampler.)

Table 3.4, completed.

Percentage of Samples For Which Compound Was Detected

Tables 3.2 through 3.4 show the percentage of the samples in which each VOC was detected. Twelve of the compounds were detected in over 90 percent of the samples, at all three locations. These compounds are listed in Table 3.5. In contrast, 15 VOCs were never detected at all during the study. This is about one-fourth of the compounds that were sampled. Compounds never detected are listed in Table 3.6. It is interesting to note that vinyl chloride, which is considered to be very toxic, was not detected. Ethyl tert-butyl ether (ETBE) and tert-amyl methyl ether (TAME), which are added to automotive fuels to increase oxygen, were not detected. However, methyl tert-butyl ether (MTBE), which is another fuel additive, was detected at Welby, but not at CAMP or Swansea.

Comparing the two lists of compounds in Table 3.6 suggests that compounds which were not detected at one site, but were at another, are from local sources. At the CAMP station, acrylonitrile, 1,1-dichloroethene, trans-1,2-dichloroethylene, 1,2-dichloroethane, trichloroethylene, methyl methacrylate, and p-dichlorobenzene were likely from local sources. At Welby, bromomethane, trans-1,2-dichloroethylene, methyl tert-butyl ether, 1,2-dichloropropane, ethyl acrylate, bromodichloromethane, trichloroethylene, methyl methacrylate, cis-1,3-dichloropropene, trans-1,2-dichloropropene, 1,1,2-trichloroethane, dibromochloromethane, p-dichlorobenzene and 1,2-dibromoethane were probably local, as the other two sites did not consistently detect them. At Swansea, dichlorotetrafluoroethane, trans-1,2-dichloroethylene, and acrylonitrile were probably from local sources.

Table 3.5 - Volatile Organic Compounds Detected in Over 90 Percent of the Air Samples

Compounds Detected in Over 90 Percent of the Air Samples		
CAMP	**Welby**	**Swansea**
Acetylene	Acetylene	Acetylene
Propylene	Propylene	Propylene
Dichlorodifluoromethane	Dichlorodifluoromethane	Dichlorodifluoromethane
Chloromethane	Chloromethane	Chloromethane
Trichlorofluoromethane	Trichlorofluoromethane	Trichlorofluoromethane
Methylene Chloride		
Trichlorotrifluoroethane	Trichlorotrifluoroethane	Trichlorotrifluoroethane
Benzene	Benzene	Benzene
Toluene	Toluene	Toluene
Ethylbenzene	Ethylbenzene	Ethylbenzene
m,p - Xylene	m,p - Xylene	m,p - Xylene
o - Xylene	o - Xylene	o - Xylene
1,3,5-Trimethylbenzene		
1,2,4-Trimethylbenzene	1,2,4-Trimethylbenzene	1,2,4-Trimethylbenzene

Note: Methylene chloride was detected 79 percent of the time at Welby, and 77 percent of the time at Swansea.

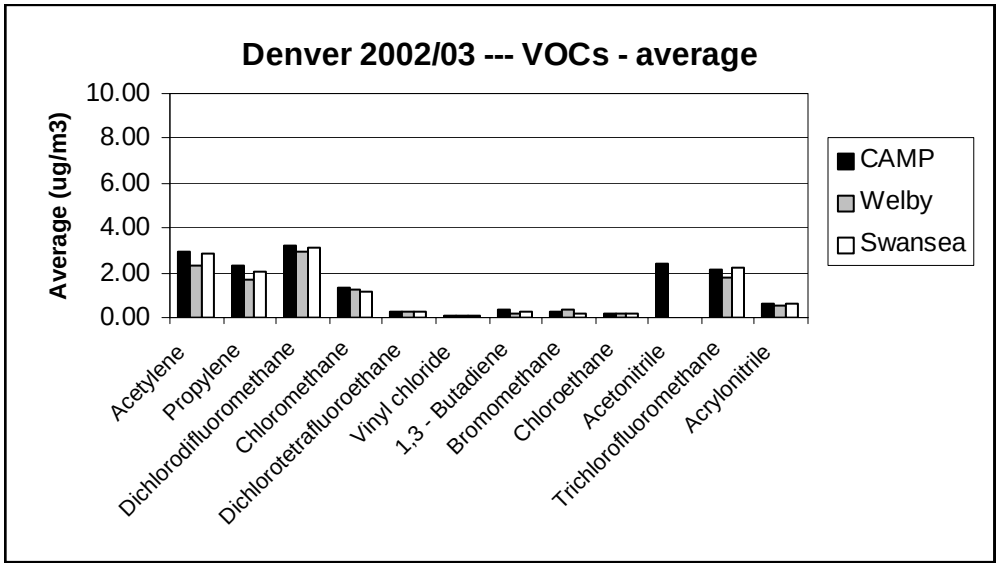
Table 3.6 - Volatile Organic Compounds Never Detected in the Air Samples

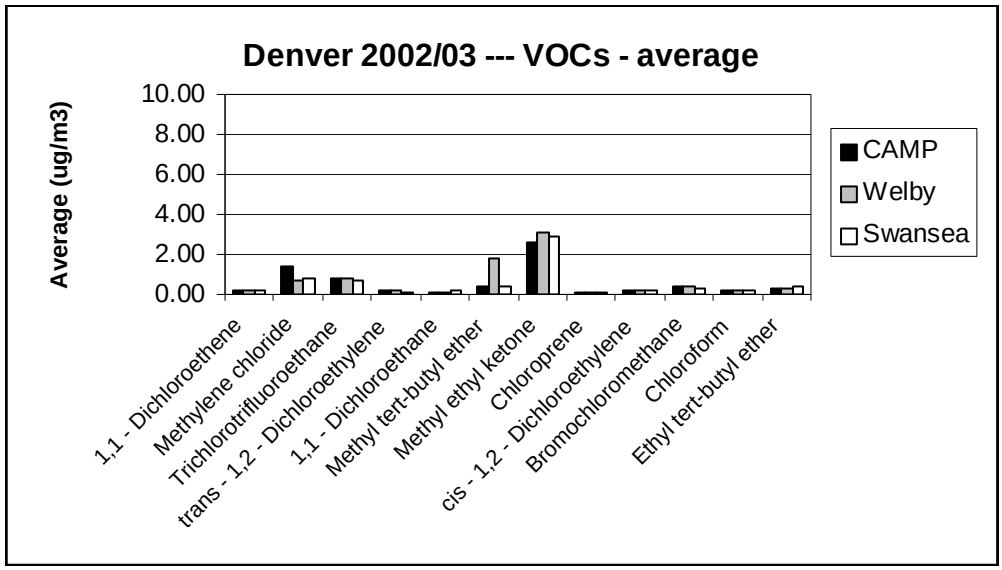
VOCs Never Detected in the Air Samples		
CAMP	**Welby**	**Swansea**
Dichlorotetrafluoroethane	Dichlorotetrafluoroethane	
Vinyl Chloride	Vinyl Chloride	Vinyl Chloride
Bromomethane		Bromomethane
Chloroethane	Chloroethane	Chloroethane
trans - 1,2 - Dichloroethylene		
	Acrylonitrile	
	1,1-Dichloroethene	1,1-Dichloroethene
	trans - 1,2 - Dichloroethylene	trans - 1,2 - Dichloroethylene
1,1 - Dichloroethane	1,1 - Dichloroethane	1,1 - Dichloroethane
Methyl tert-Butyl Ether		Methyl tert-Butyl Ether
Chloroprene	Chloroprene	Chloroprene
cis-1,2-Dichloroethylene	cis-1,2-Dichloroethylene	cis-1,2-Dichloroethylene
Bromochloromethane	Bromochloromethane	Bromochloromethane
Ethyl tert-Butyl Ether	Ethyl tert-Butyl Ether	Ethyl tert-Butyl Ether
	1,2 - Dichloroethane	1,2 - Dichloroethane
tert-Amyl Methyl Ether	tert-Amyl Methyl Ether	tert-Amyl Methyl Ether
1,2 - Dichloropropane		1,2 - Dichloropropane
Ethyl Acrylate		Ethyl Acrylate
Bromodichloromethane		Bromodichloromethane
		Trichloroethylene
		Methyl Methacrylate
cis -1,3 - Dichloropropene		cis -1,3 - Dichloropropene
trans - 1,3 - Dichloropropene		trans - 1,3 - Dichloropropene
1,1,2 - Trichloroethane		1,1,2 - Trichloroethane
Dibromochloromethane		Dibromochloromethane
1,2-Dibromoethane		1,2-Dibromoethane
Chlorobenzene	Chlorobenzene	Chlorobenzene
Bromoform	Bromoform	Bromoform
1,1,2,2 - Tetrachloroethane	1,1,2,2 - Tetrachloroethane	1,1,2,2 - Tetrachloroethane
m - Dichlorobenzene	m - Dichlorobenzene	m - Dichlorobenzene
Chloromethylbenzene	Chloromethylbenzene	Chloromethylbenzene
		p - Dichlorobenzene
o - Dichlorobenzene	o - Dichlorobenzene	o - Dichlorobenzene
1,2,4-Trichlorobenzene	1,2,4-Trichlorobenzene	1,2,4-Trichlorobenzene
Hexachloro-1,3-Butadiene	Hexachloro-1,3-Butadiene	Hexachloro-1,3-Butadiene

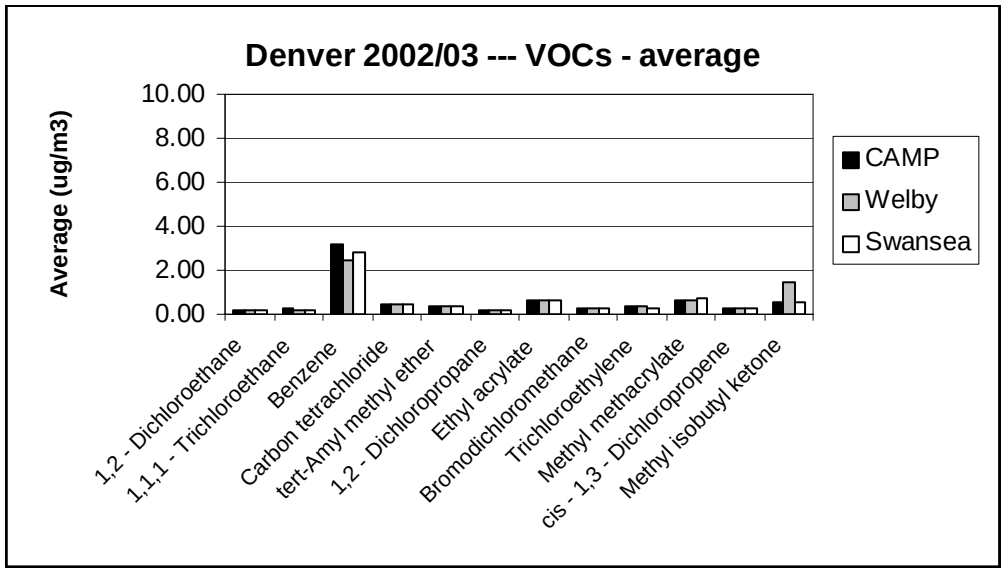
Graphs - Volatile Organic Compounds

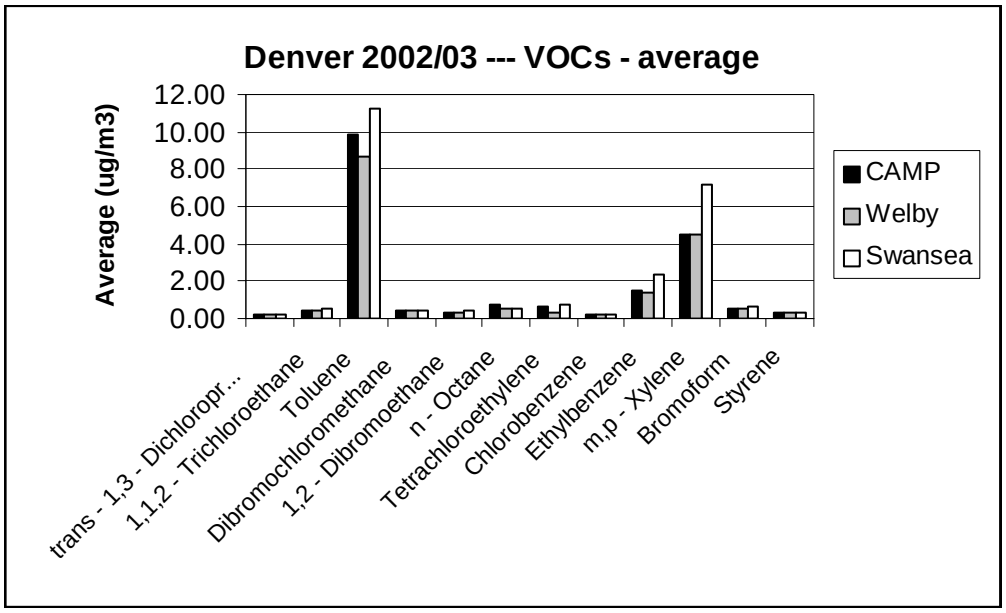
The following graphs show average and maximum concentrations for each compound, at the three monitoring locations. Almost all compounds had mean concentrations below 6 ug/m3. The only compounds with a mean above this were toluene (all 3 sites), and bromoform (Swansea site). The compounds showing the highest maximum values were acetonitrile, methylene chloride, methyl ethyl ketone, methyl isobutyl ketone, and toluene.

Figure 3.1 - Average VOCs






Denver 2002/03 --- VOCs - average
Average (ug/m3)
10.00
8.00
6.00
4.00
2.00
0.00
CAMP
Welby
Swansea
1,2 - Dichloroethane
1,1,1 - Trichloroethane
Benzene
Carbon tetrachloride
tert-Amyl methyl ether
1,2 - Dichloropropane
Ethyl acrylate
Bromodichloromethane
Trichloroethylene
Methyl methacrylate
cis - 1,3 - Dichloropropene
Methyl isobutyl ketone


Denver 2002/03 --- VOCs - average
Average (ug/m3)
12.00
10.00
8.00
6.00
4.00
2.00
0.00
CAMP
Welby
Swansea
trans - 1,3 - Dichloropr...
1,1,2 - Trichloroethane
Toluene
Dibromochloromethane
1,2 - Dibromoethane
n - Octane
Tetrachloroethylene
Chlorobenzene
Ethylbenzene
m,p - Xylene
Bromoform
Styrene

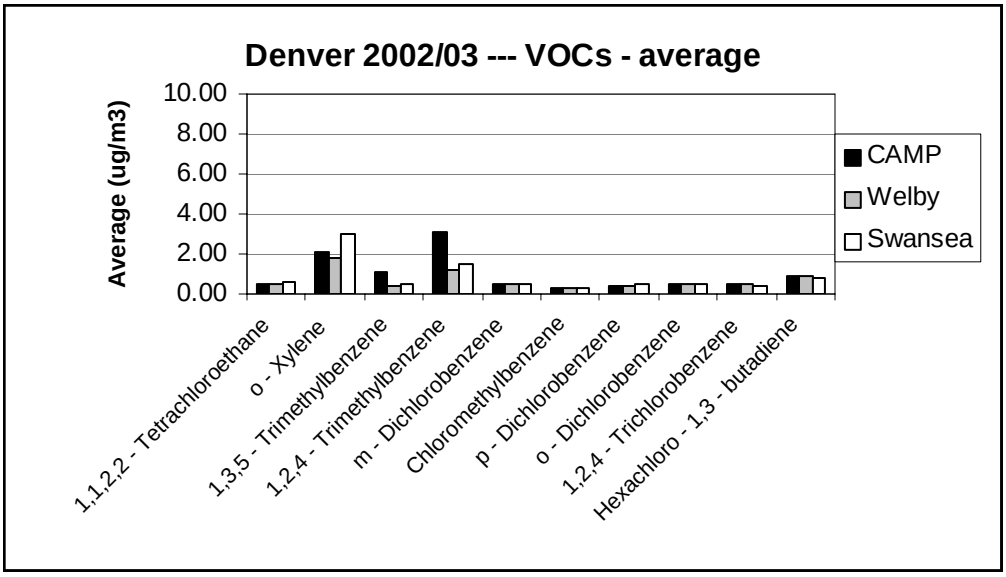
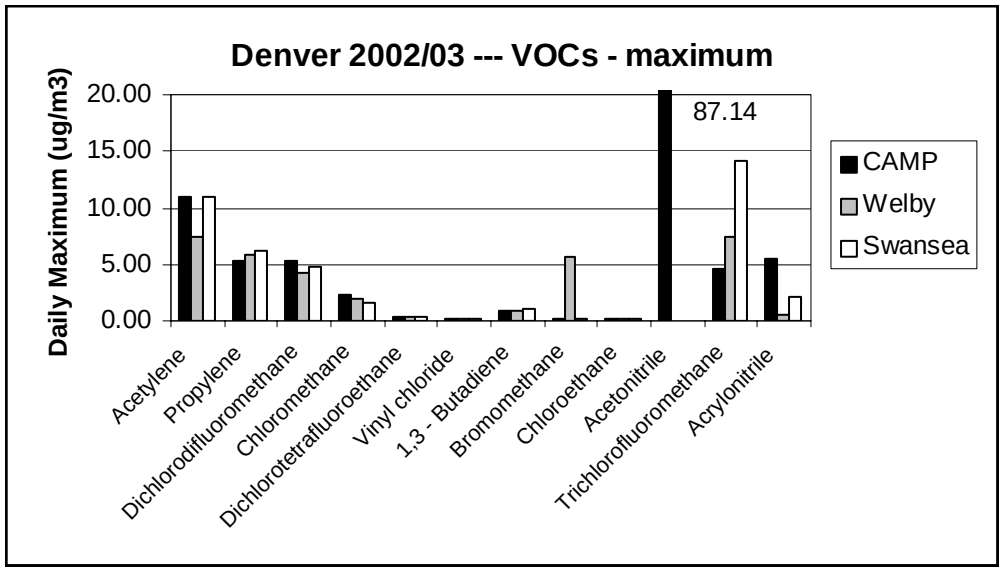
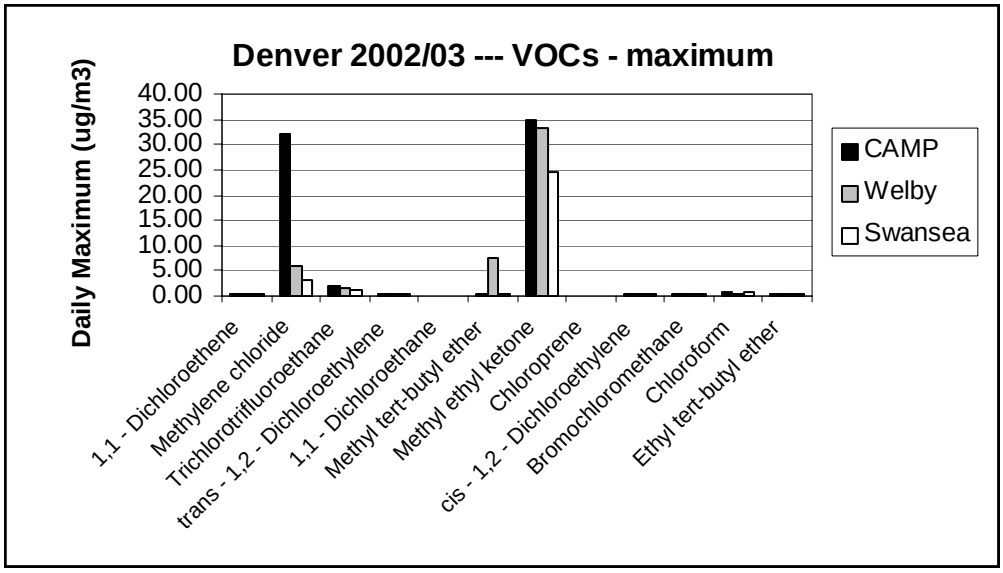
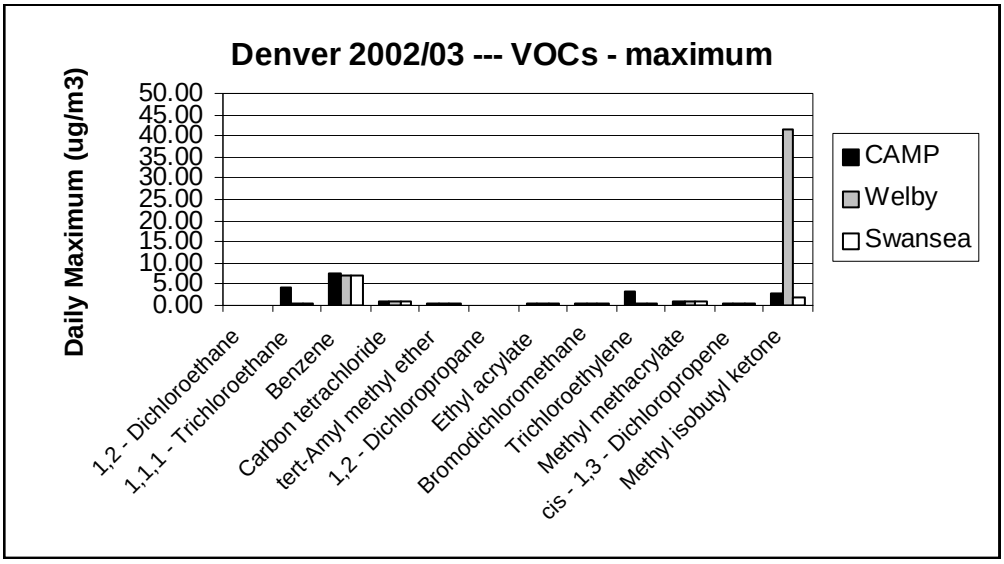
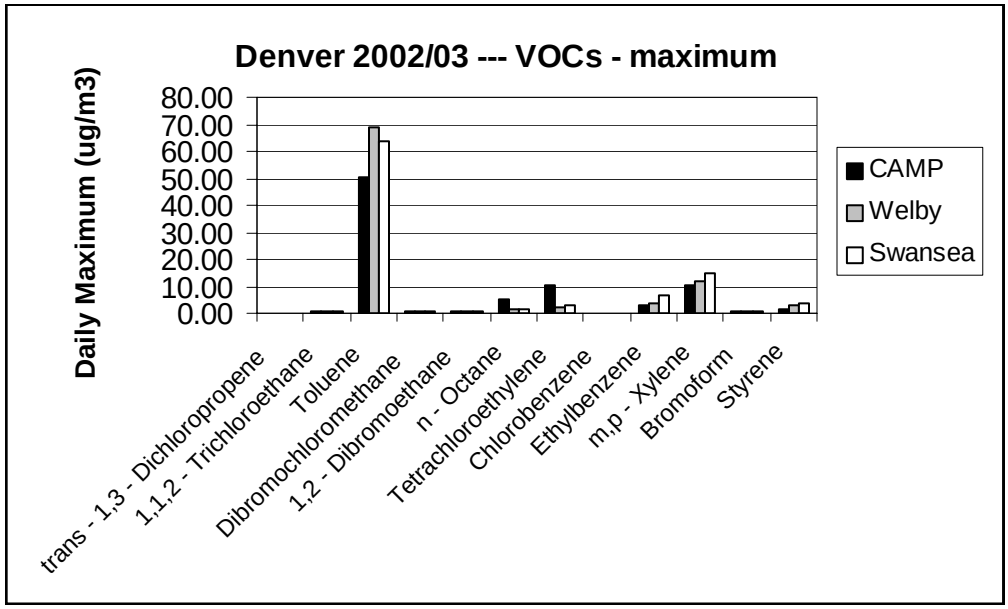

Denver 2002/03 --- VOCs - average
Average (ug/m3)
10.00
8.00
6.00
4.00
2.00
0.00
CAMP
Welby
Swansea
1,1,2,2 - Tetrachloroethane
o - Xylene
1,3,5 - Trimethylbenzene
1,2,4 - Trimethylbenzene
m - Dichlorobenzene
Chloromethylbenzene
p - Dichlorobenzene
o - Dichlorobenzene
1,2,4 - Trichlorobenzene
Hexachloro - 1,3 - butadiene

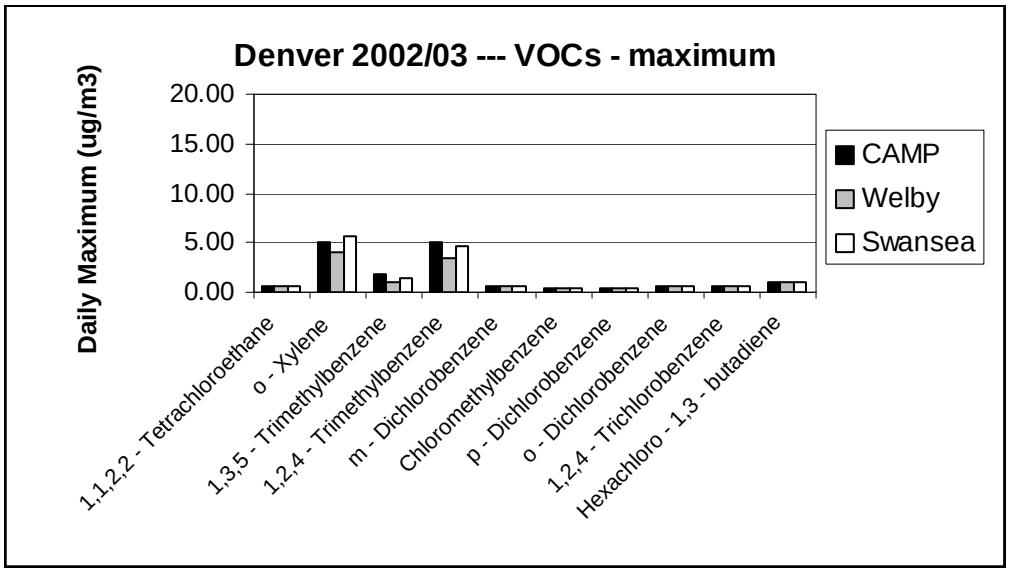
Figure 3.2 - Maximum VOCs








Denver 2002/03 --- VOCs - maximum
Daily Maximum (ug/m3)
80.00
70.00
60.00
50.00
40.00
30.00
20.00
10.00
0.00
CAMP
Welby
Swansea
trans - 1,3 - Dichloropropene
1,1,2 - Trichloroethane
Toluene
Dibromochloromethane
1,2 - Dibromoethane
n - Octane
Tetrachloroethylene
Chlorobenzene
Ethylbenzene
m,p - Xylene
Bromoform
Styrene


Denver 2002/03 --- VOCs - maximum
Daily Maximum (ug/m3)
20.00
15.00
10.00
5.00
0.00
CAMP
Welby
Swansea
1,1,2,2 - Tetrachloroethane
o - Xylene
1,3,5 - Trimethylbenzene
1,2,4 - Trimethylbenzene
m - Dichlorobenzene
Chloromethylbenzene
p - Dichlorobenzene
o - Dichlorobenzene
1,2,4 - Trichlorobenzene
Hexachloro - 1,3 - butadiene

Acetonitrile (Figure 3.3) data are available only for CAMP, as the other two samplers had contamination issues with this compound, and their data were invalidated. Unlike CAMP, the other two samplers had a common manifold for sampling carbonyls and VOCs. Evidently, acetonitrile used to prepare the DNPH cartridge can seep back through the manifold, and enter the canister. This problem, discovered at a remote site in Custer, South Dakota, led national contractor ERG to redesign their manifold system. Acetonitrile was only detected 18% of the time at CAMP. 1,3-Butadiene (Figure 3.4) was highest at CAMP, followed by Swansea and Welby. Benzene (Figure 3.5) is usually highest at CAMP, but sometimes the daily maxima occurred at Welby or Swansea. Carbon tetrachloride (Figure 3.6) showed an erratic pattern. This compound is probably emitted by local sources at each individual site. Chloroform (Figure 3.7) was rarely above detection limit, but peak days sometimes occurred simultaneously (see 9/29/02 graph point). p-Dichlorobenzene (Figure 3.8) was only seen during rare samples, when the laboratory temporarily had a lower detection limit. It was detected 5% of the time at CAMP, 4% at Welby, and was never detected at Swansea. Tetrachloroethylene (Figure 3.9) and trichloroethylene (Figure 3.10) were seen sporadically. 1,3,5-Trimethylbenzene (Figure 3.11) and 1,2,4-trimethylbenzene (Figure 3.12) tracked fairly well across the sites. The pattern of CAMP value highest, then Swansea, then Welby, suggests that motor vehicle traffic was the source of these trimethybenzene compounds.

Figure 3.3 - Acetonitrile

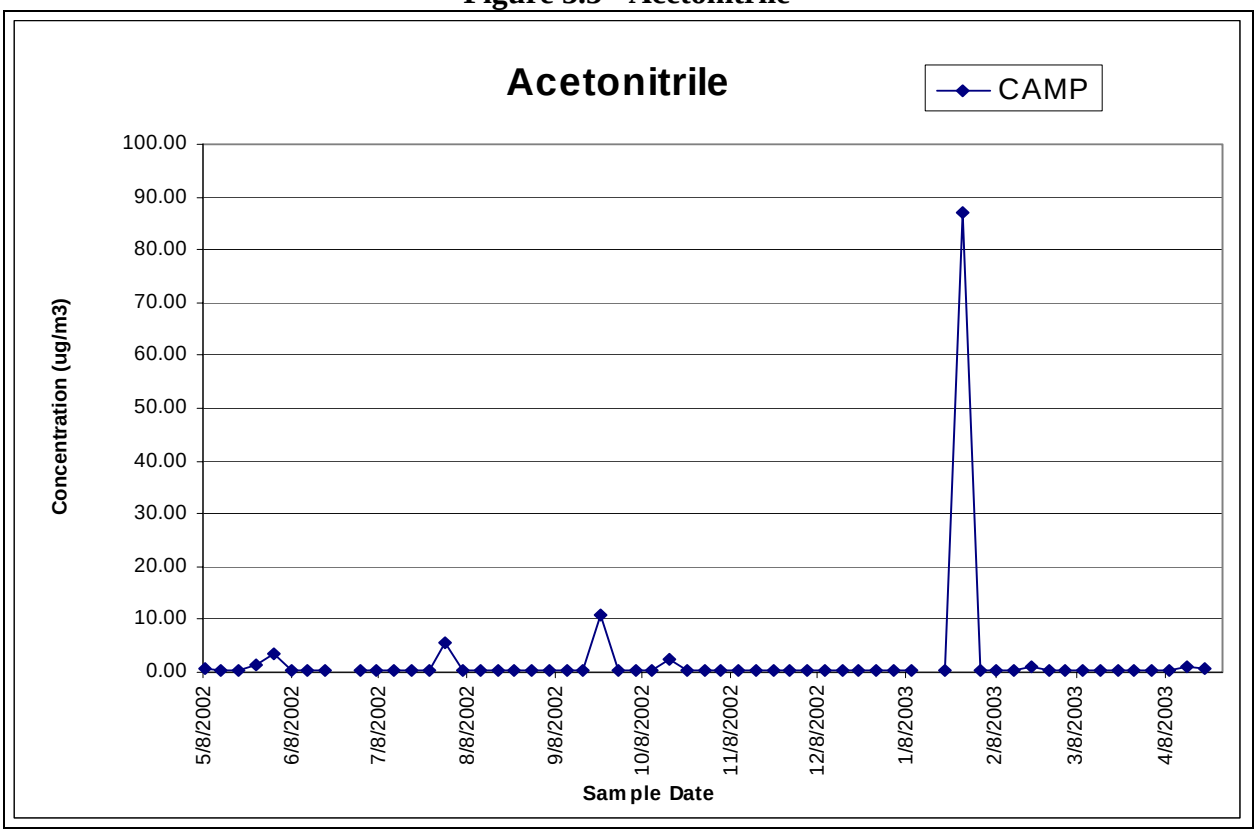


Figure 3.4 - 1,3-Butadiene

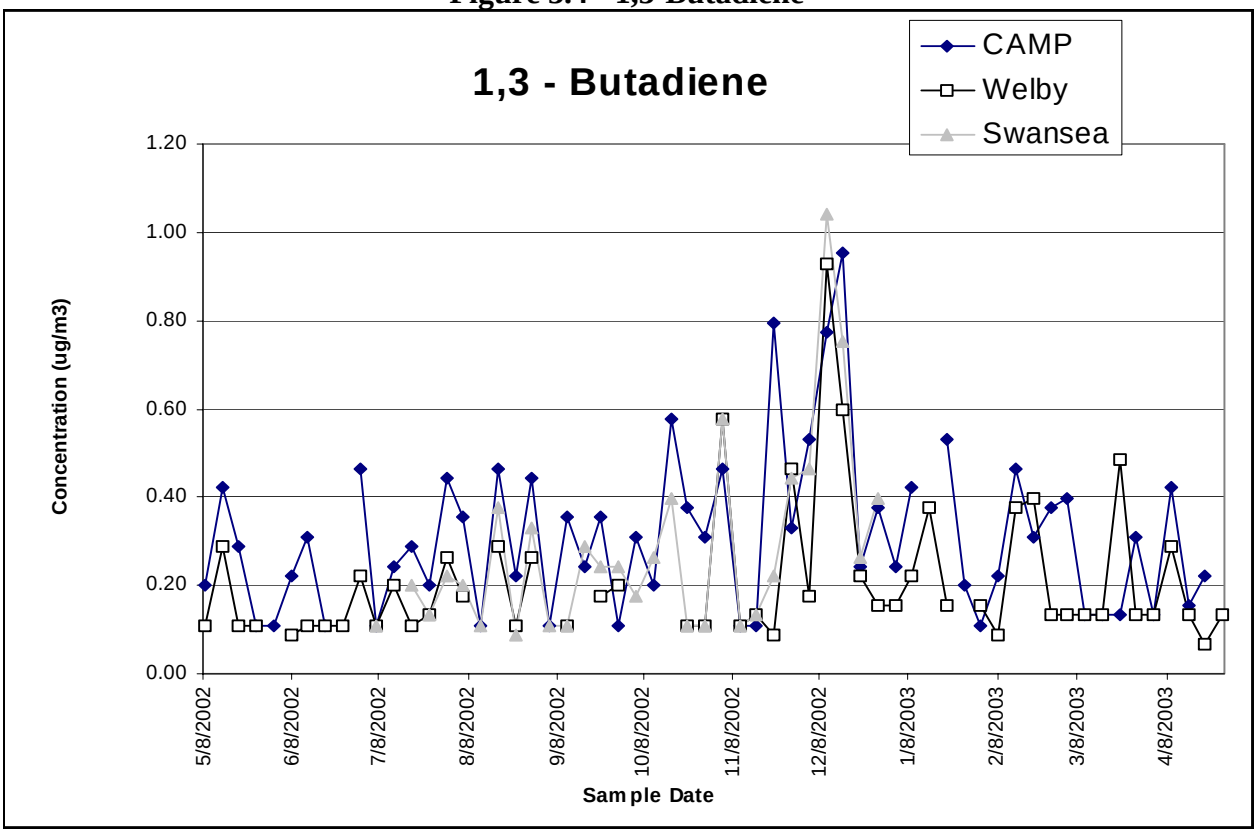


Figure 3.5 - Benzene

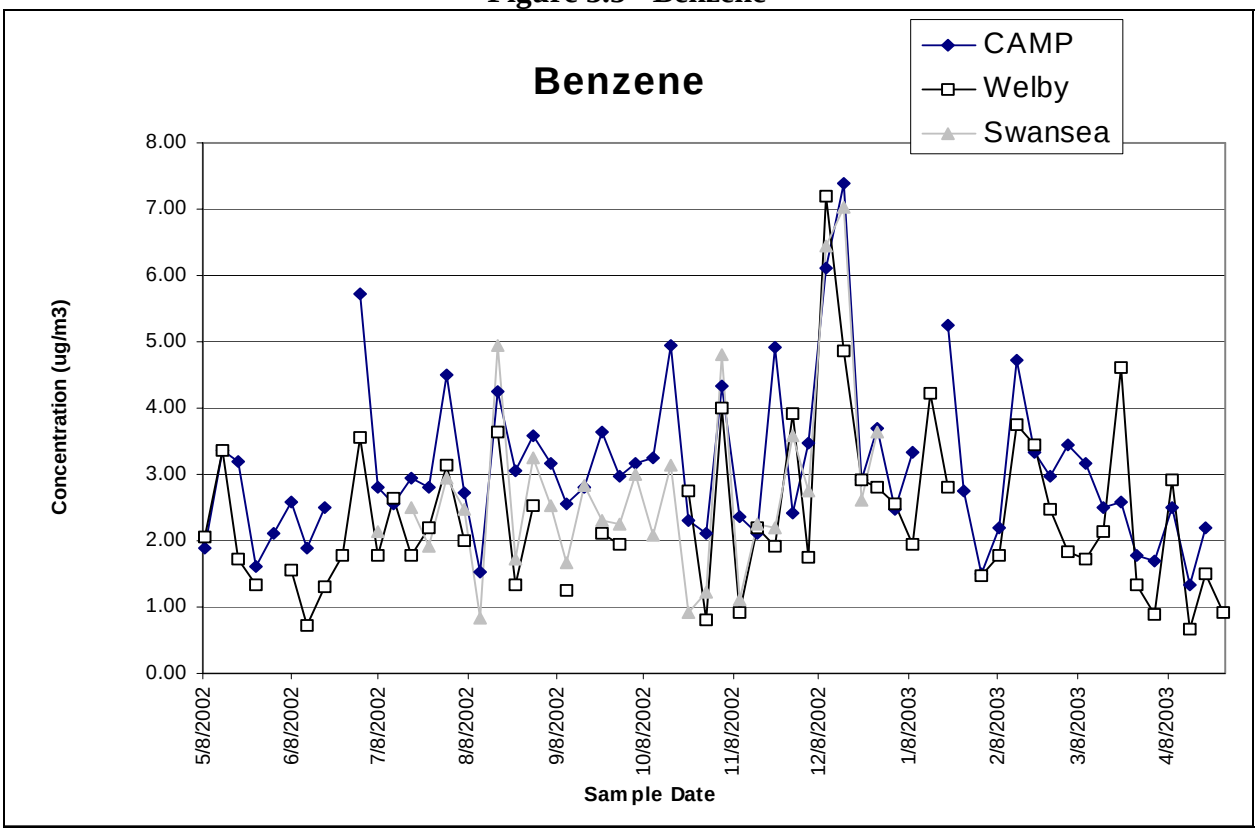


Figure 3.6 - Carbon Tetrachloride

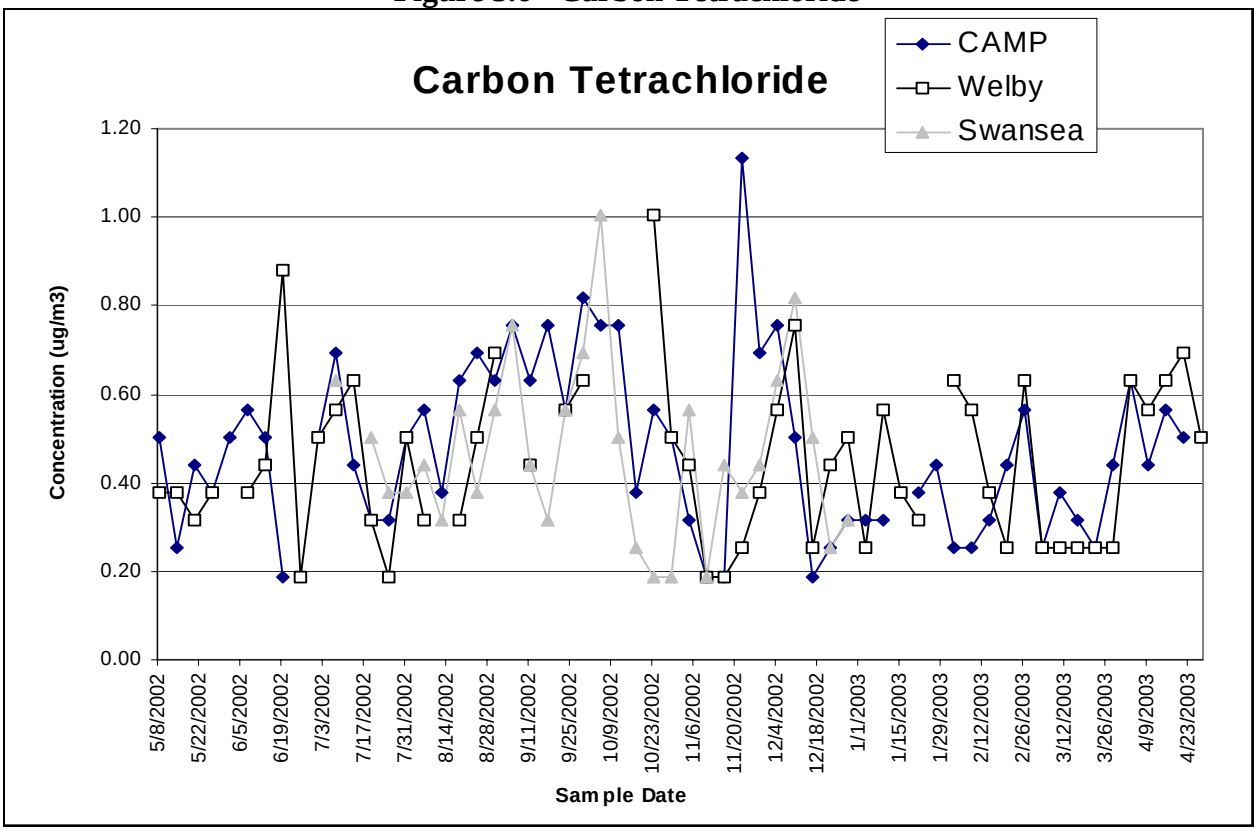


Figure 3.7 - Chloroform

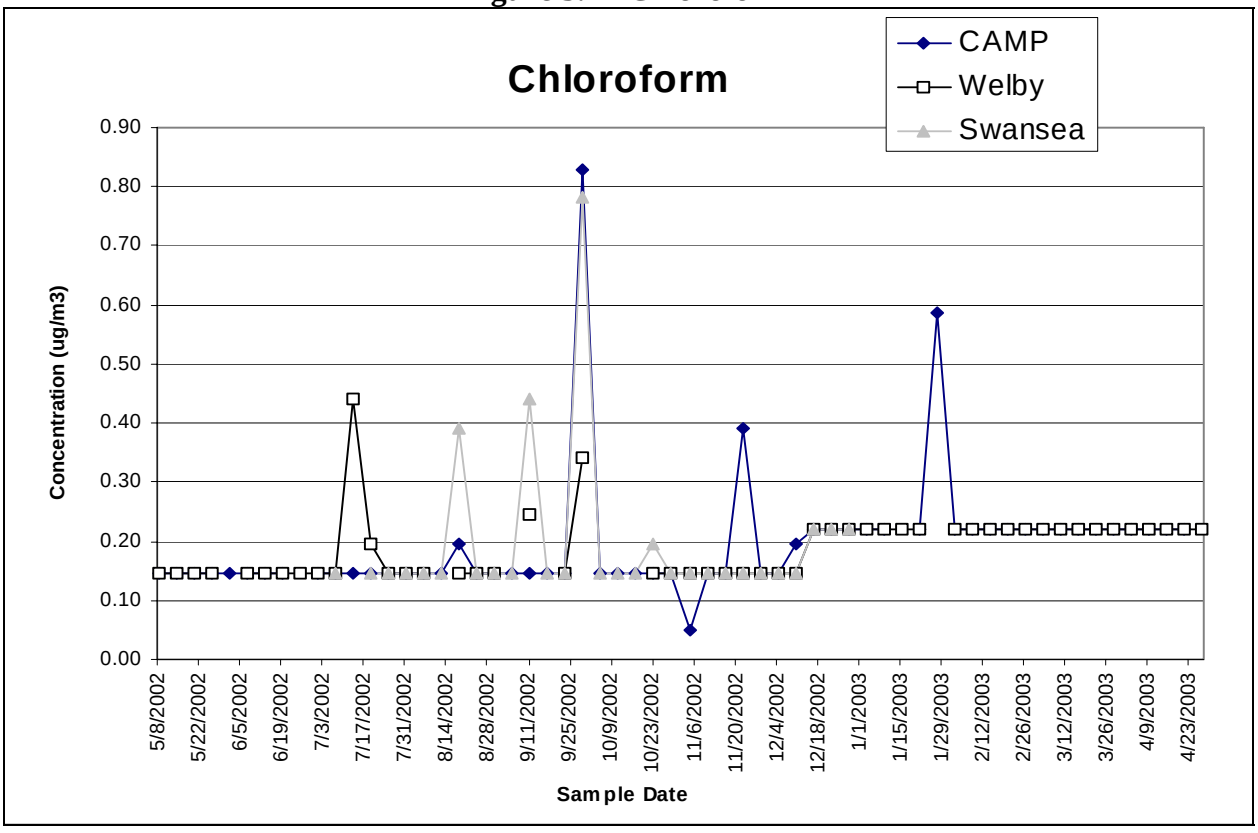


Figure 3.8 - p-Dichlorobenzene

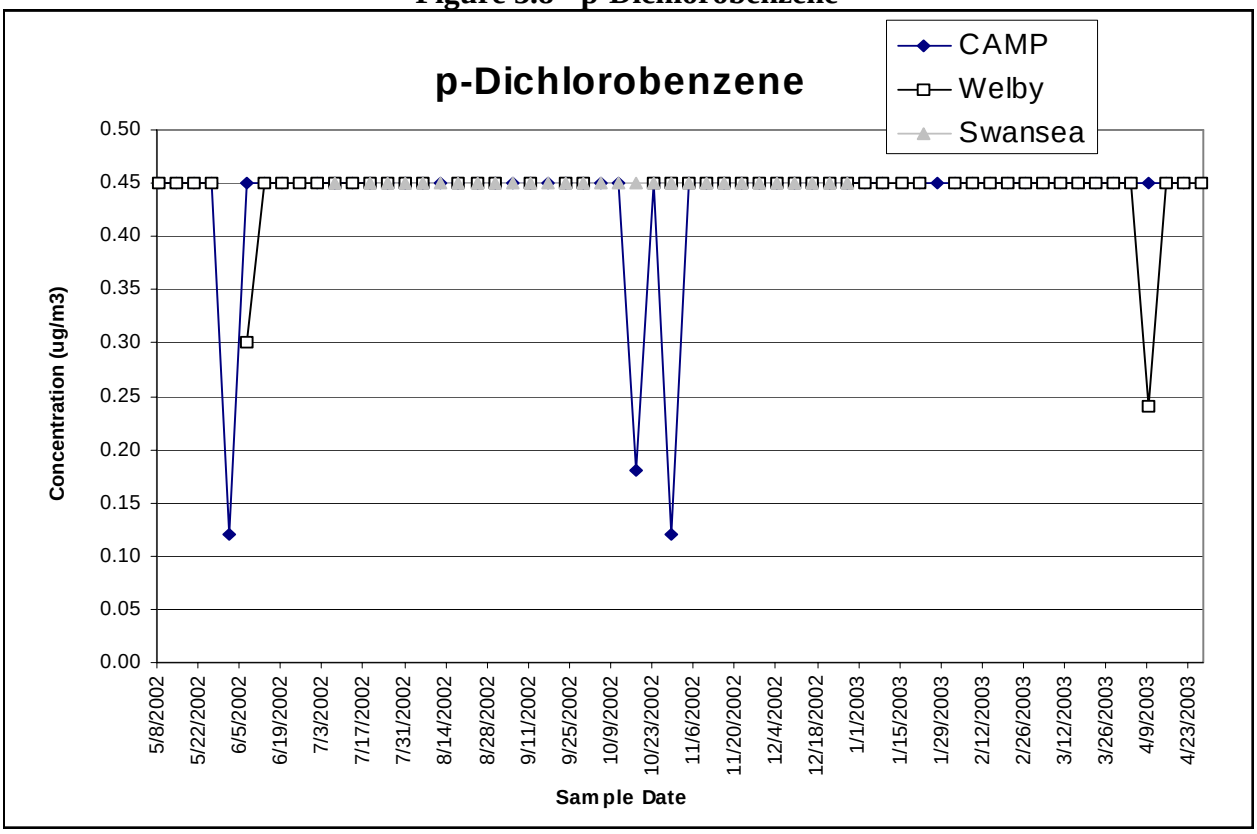


Figure 3.9 - Tetrachloroethylene

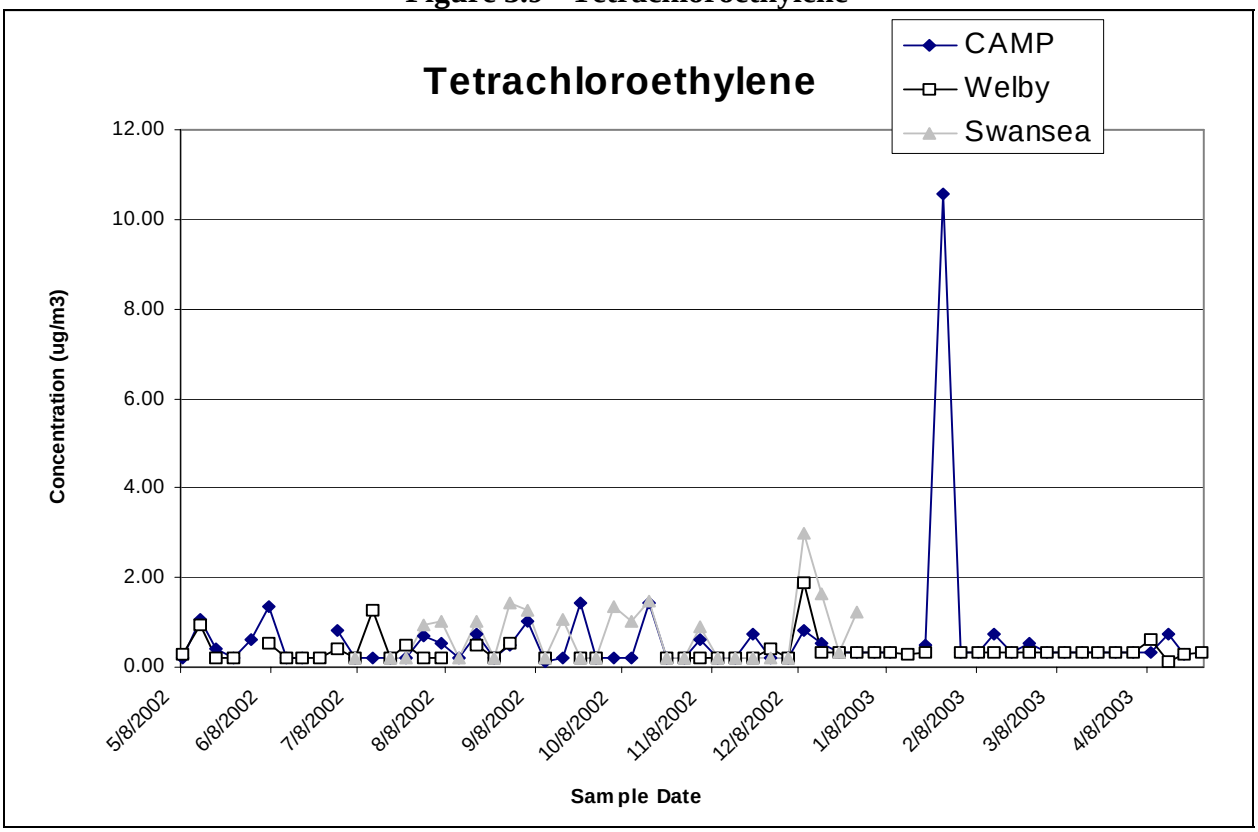


Figure 3.10 - Trichloroethylene

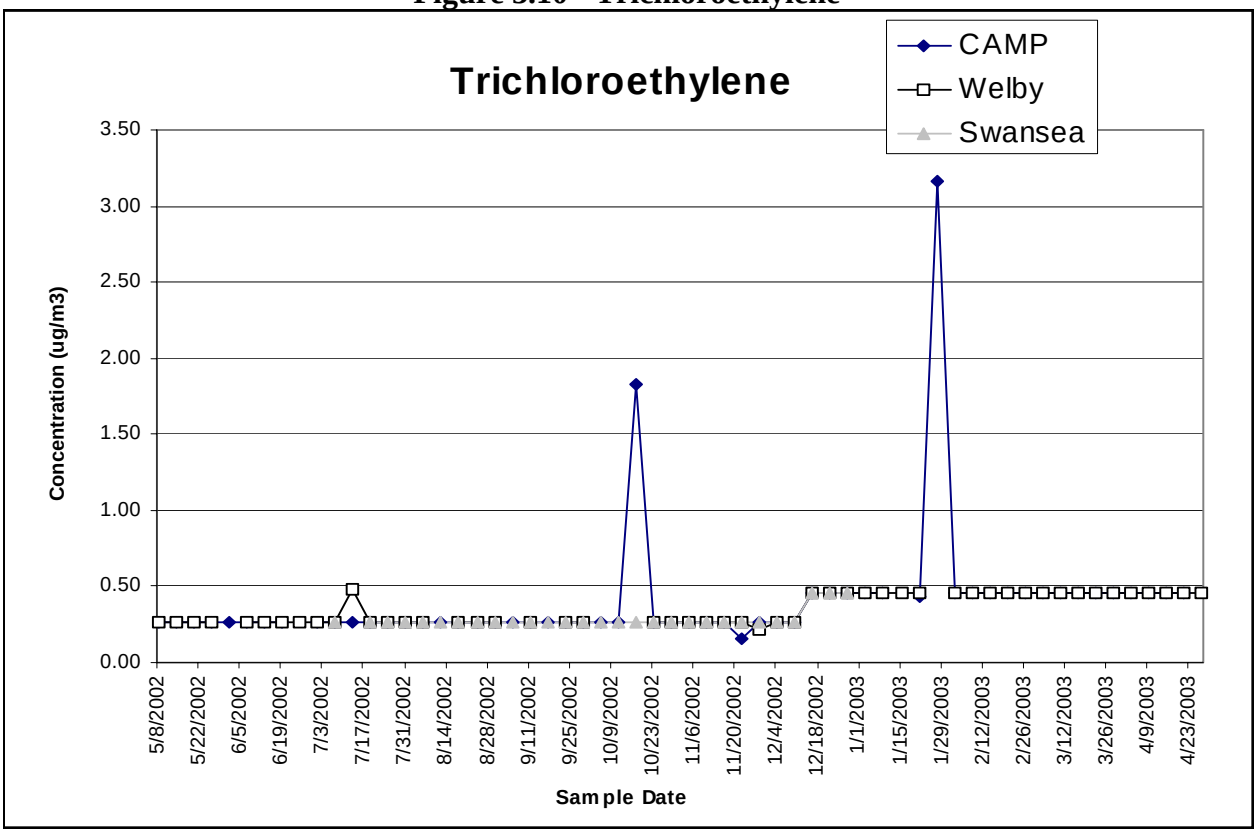


Figure 3.11 - 1,3,5-Trimethylbenzene

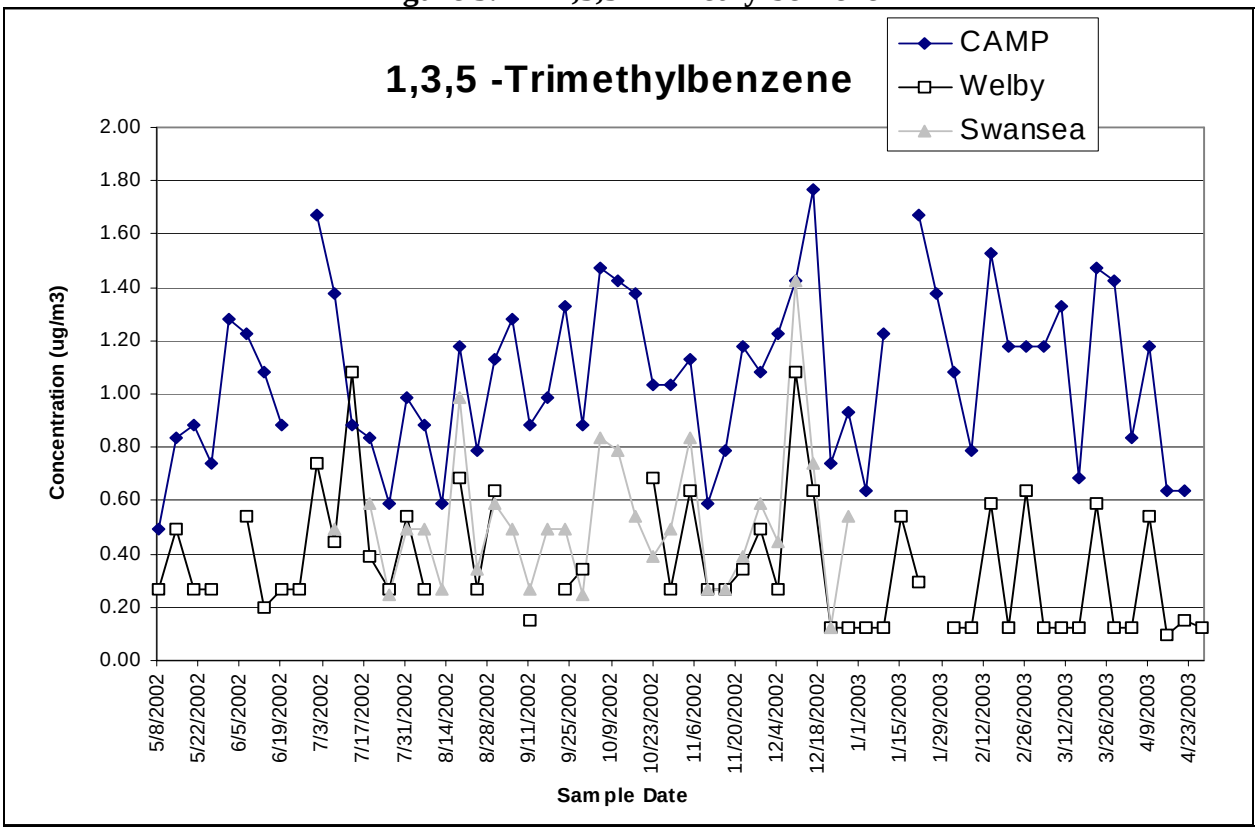
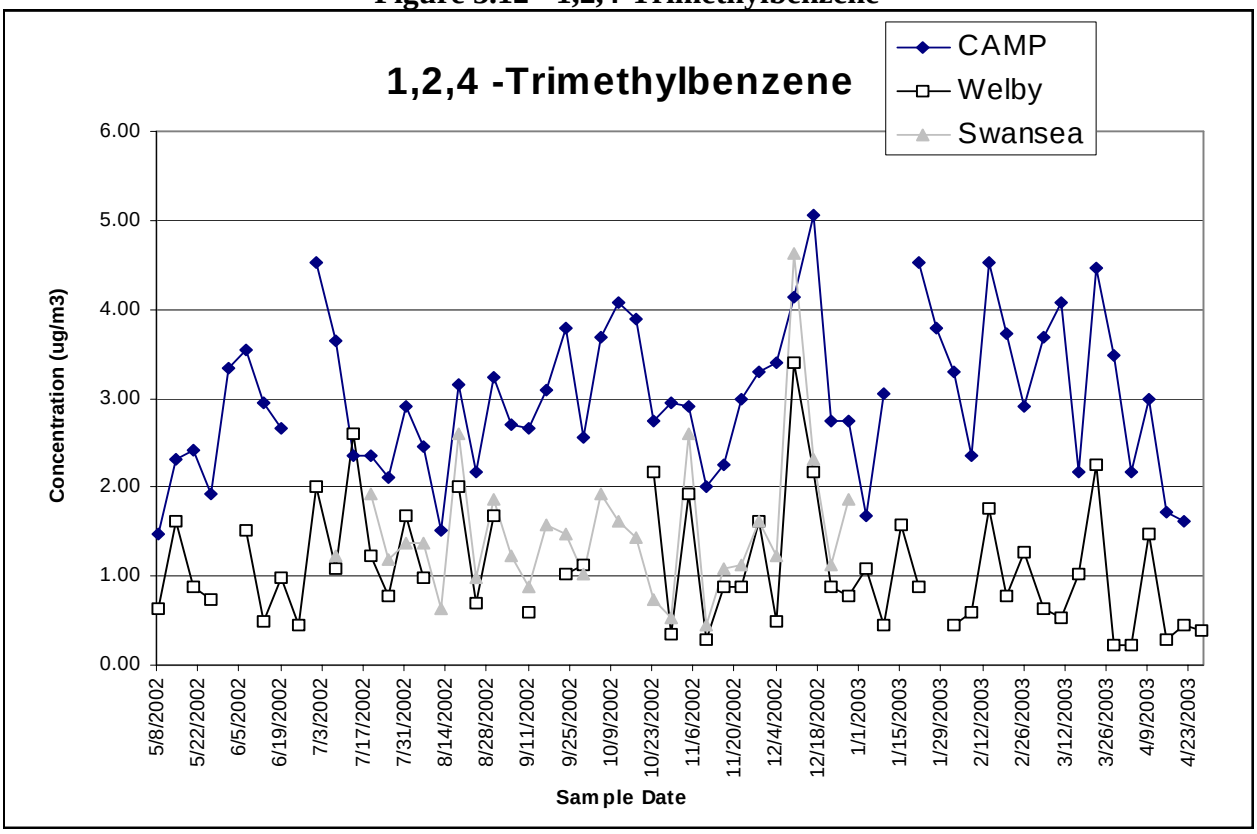


Figure 3.12 - 1,2,4-Trimethylbenzene



Precision of Sample Results - Volatile Organic Compounds

Periodically throughout the year, a second canister was sampled simultaneously with the main sample. These additional samples, known as duplicates, were collected in order to assess the precision (repeatability) of the canister sampling method. On the duplicate sampling dates, the laboratory also conducted a test of the precision of the analytical process by injecting two samples of each canister's air into the gas chromatograph/mass spectrometer. These samples are known as the laboratory replicates. Thus, this project collected two types of precision data – duplicate data, which assesses both sampling and analysis procedures, and replicate data, which assesses laboratory analytical method repeatability. Information regarding precision and accuracy results is available upon request to the Air Pollution Control Division.

Field Blanks - Volatile Organic Compounds

The volatile organic compound sampling method involves sampling in stainless steel canisters with specially-treated interior surfaces. The canisters are re-used. After a full canister is analyzed, it is pumped out repeatedly to a high vacuum. This procedure cleans it for the next use. Periodically, one canister from each cleaning batch is tested to make sure the method is performing adequately. The test canister is filled with ultra-pure air, and then analyzed. If it shows no contamination, the batch is released for use. If contamination is found, the entire batch is sent through the cleaning process for a second time. The canisters arrive in the field closed, and under 20 to 30 inches of vacuum. Therefore, field blanks are not used in this method. The canisters are "blanked" at the laboratory prior to shipping to the field.

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Section 4 - Metals at Denver Stations

May 2002 to April 2003

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Summary Statistics - Metals

Maximum and Mean - All Samples

Metals data collected at the three Denver stations from April 2002 through May 2003 are presented in this section. During the year-long period metals at CAMP and Welby, were sampled on a one-in-six day basis, for a total of 60 samples attempted. Of these, the laboratory successfully processed 93 and 95 percent from each site. The Swansea site recovered all scheduled samples in its six-month run (See Table 4.1).

Tables 4.2 through 4.4 summarize the annual maximum and mean concentrations for each of the metals measured during the study. Annual means were calculated by using one-half of the detection limit in place of the non-detect samples. Results show that manganese, lead, and chromium were the compounds with the highest mean concentrations in ambient air. Except for beryllium, all metals were detected in 100 percent of the samples taken.

Table 4.1 - Percentage Data Recovery For Metals Samples at Denver

Station	Samples Recovered	Sample Days Scheduled	Percentage Recovered
CAMP	56	60	93.3
Welby	57	60	95.0
Swansea	31	31	100.0

Table 4.2 - Annual Maximum and Mean Metals Concentrations at Denver - CAMP

CAMP	Summary Statistics (ug/m3)		Count of Non-Detects		Percentage of Samples in Which Compound Was Detected
	Maximum	Mean	Number	Percentage	
Antimony	0.0076	0.0027	0.0	0.0	100.0
Arsenic	0.0059	0.0013	0.0	0.0	100.0
Beryllium	0.0001	0.0000	24.0	42.9	57.1
Cadmium	0.0009	0.0004	0.0	0.0	100.0
Cobalt	0.0019	0.0010	0.0	0.0	100.0
Chromium (total)	0.0086	0.0042	0.0	0.0	100.0
Lead	0.0493	0.0155	0.0	0.0	100.0
Manganese	0.1362	0.0471	0.0	0.0	100.0
Mercury	0.0002	0.0001	0.0	0.0	100.0
Nickel	0.0045	0.0022	0.0	0.0	100.0
Selenium	0.0024	0.0010	0.0	0.0	100.0

Table 4.3 - Annual Maximum and Mean Metals Concentrations at Denver - Welby

Welby	Summary Statistics (ug/m3)		Count of Non-Detects		Percentage of Samples in Which Compound Was Detected
	Maximum	Mean	Number	Percentage	
Antimony	0.0079	0.0014	0	0.0	100.0
Arsenic	0.0041	0.0010	0	0.0	100.0
Beryllium	0.0002	0.0000	35	61.4	38.6
Cadmium	0.0007	0.0003	0	0.0	100.0
Cobalt	0.0128	0.0009	0	0.0	100.0
Chromium (total)	0.0063	0.0021	0	0.0	100.0
Lead	0.0598	0.0160	0	0.0	100.0
Manganese	0.1478	0.0469	0	0.0	100.0
Mercury	0.0002	0.0000	0	0.0	100.0
Nickel	0.0044	0.0020	0	0.0	100.0
Selenium	0.0024	0.0008	0	0.0	100.0

Table 4.4 - Annual Maximum and Mean Metals Concentrations at Denver - Swansea

Swansea	Summary Statistics (ug/m3)		Count of Non-Detects		Percentage of Samples in Which Compound Was Detected
	Maximum	Mean	Number	Percentage	
Antimony	0.0054	0.0021	0	0.0	100.0
Arsenic	0.0057	0.0010	0	0.0	100.0
Beryllium	0.0003	0.0000	16	51.6	48.4
Cadmium	0.0028	0.0005	0	0.0	100.0
Cobalt	0.0023	0.0008	0	0.0	100.0
Chromium (total)	0.0091	0.0032	0	0.0	100.0
Lead	0.0786	0.0239	0	0.0	100.0
Manganese	0.1236	0.0534	0	0.0	100.0
Mercury	0.0001	0.0000	0	0.0	100.0
Nickel	0.0053	0.0023	0	0.0	100.0
Selenium	0.0025	0.0010	0	0.0	100.0

Graphs - Metals

The metal compounds measured during the study are graphed in Figures 4.1 through 4.5. Figure 4.1 shows that lead and manganese were the metals measured at highest concentrations. Figure 4.2 indicates that maximum metals concentrations occurred at different locations for each metal. Thus, metals concentrations are believed to be related to local sources in the vicinity of each site. Arsenic (Figure 4.3) was lower in the late spring and summer, than in other parts of the year. Cobalt (Figure 4.4) tracked fairly consistently across sites. Manganese (Figure 4.5) had consistent high and low days across the three sites, but the site that gave each particular day's maximum varied.

Figure 4.1 - Average Metals at Sites

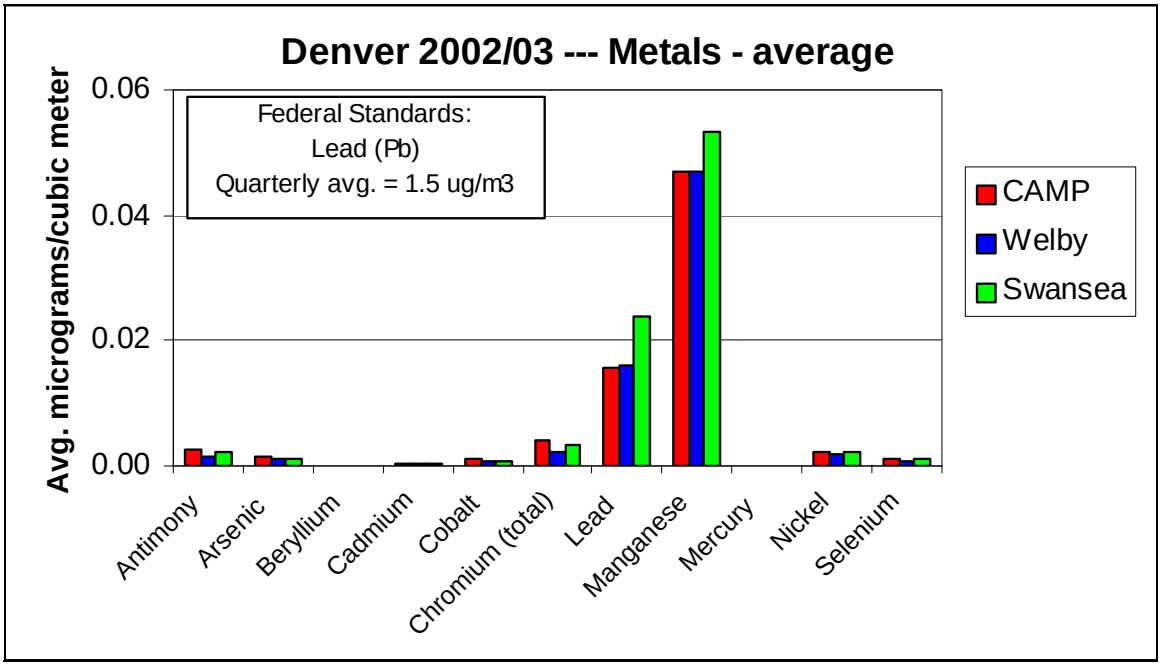


Figure 4.2 - Maximum Metals at Sites

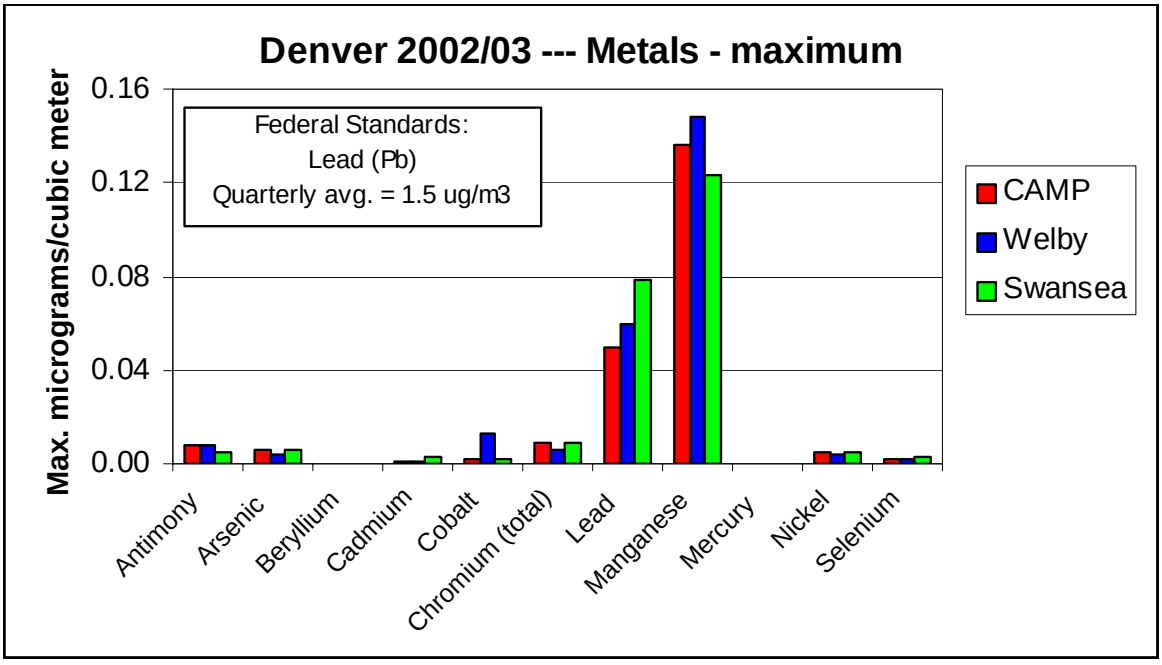


Figure 4.3 - Arsenic

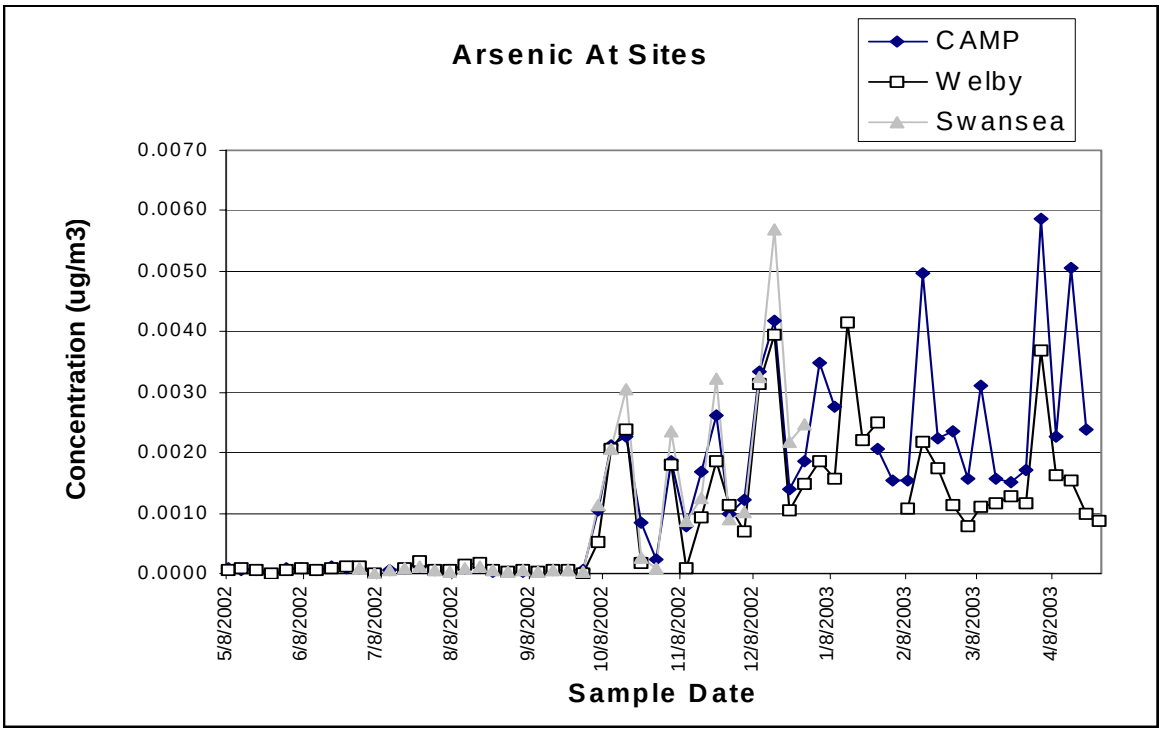


Figure 4.4 - Cobalt

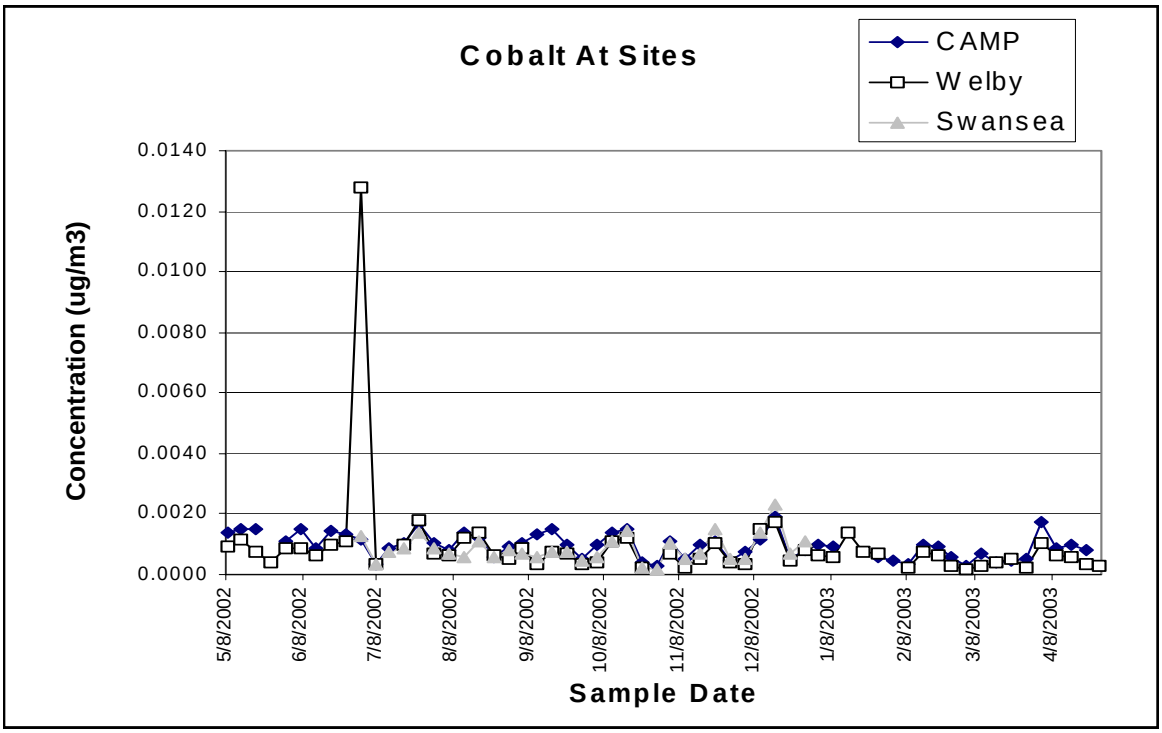
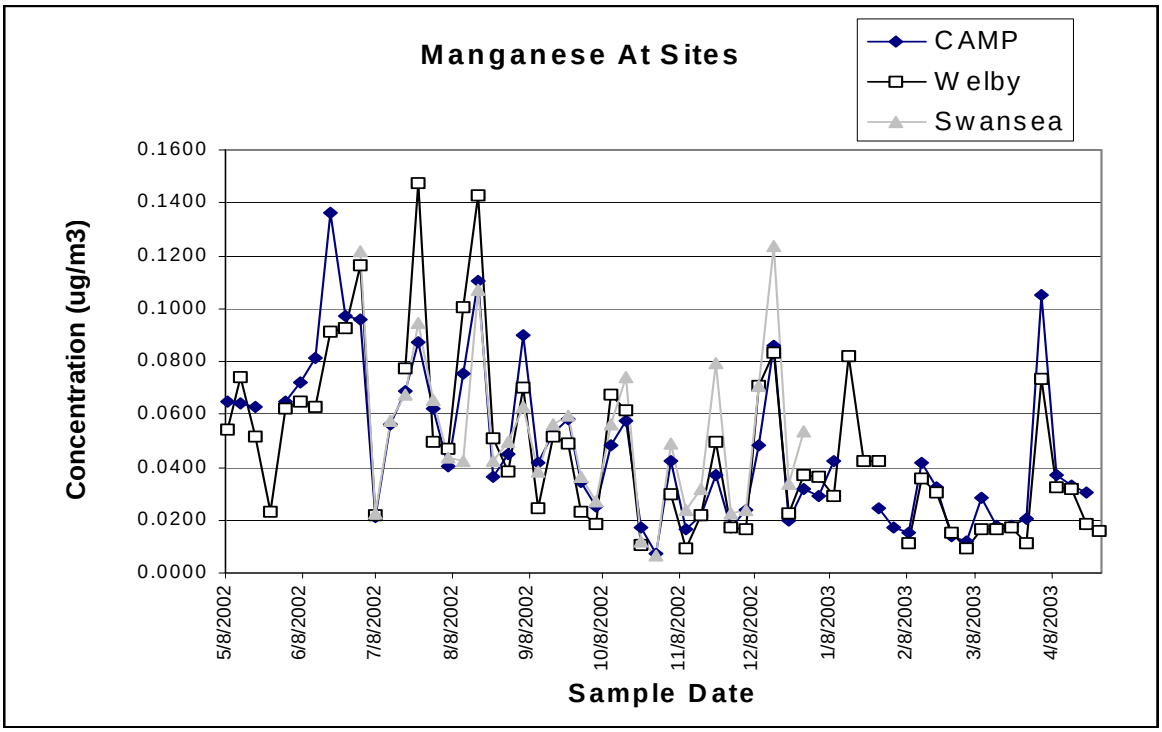


Figure 4.5 - Manganese



Precision of Sample Results - Metals Compounds

Once every 12 days, a second TSP sampler was run simultaneously with the main one at Welby. These additional samples, known as duplicates, were collected in order to assess the precision (repeatability) of the metals sampling method. In general, the duplicates showed good results, within the EPA goal of +/- 30 percent difference for individual samples.

Field Blanks - Metals Compounds

Occasionally, a filter was transported to the field, placed on a sampler, and immediately removed, without having any air passed through it. These "field blanks" are taken to assess whether contamination in the field or the sampling materials is significant. Antimony, beryllium, cadmium, cobalt, mercury and selenium showed little to no contamination in the blanks. Arsenic, lead, manganese, and nickel showed moderate levels of contamination, while chromium was the worst, averaging over 2500 ng/filter. These contamination findings are believed to be related to the use of metal knives in cutting individual filters from the giant sheets prepared at the factory. At the extremely low levels of metals in ambient air that the national air toxics network is assessing, such filter contamination is a concern. The project team for the nation-wide project plans to evaluate new filter materials and sampling methods in the future, in hopes of alleviating this problem. In any case, these "blank levels" were subtracted from the measured concentrations for each sample date, so that levels reported in air would not include filter contamination.

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Section 5 -Air Toxics Inhalation: Screening-Level Human Health Risk Assessment

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Introduction

This section presents the methodologies and findings of the risk evaluation for ambient air toxics monitoring conducted at three locations in Denver, Colorado from May 2002 through April 2003. The purpose of the evaluation was to determine if residents at any of these locations are being exposed to airborne concentrations of measured toxic air pollutants via inhalation that may pose unacceptable risks to human health.

Organization of This Section

In addition to this introduction, this chapter is organized into the following subsections:

Subsection 2 Selection Of Chemicals Of Potential Concern

This section discusses the chemicals of potential concern to human health, and provides a summary of the available data on the levels of these chemicals.

Subsection 3 Exposure Assessment

This section discusses how humans may be exposed to air toxics, now or in the future, and provides the approach for quantifying the level of exposure for those chemicals that are considered to be of potential significance.

Subsection 4 Toxicity Assessment

This section summarizes the characteristic cancer and non-cancer health effects of the chemicals of potential concern, and provides quantitative toxicity factors that can be used to calculate cancer and non-cancer risk levels.

Subsection 5 Risk Characterization

This section combines data on the level of exposure to chemicals of potential concern (Subsection 3) with information on the toxicity of each chemical (Subsection 4) to yield quantitative estimates of the risks of cancer and non-cancer effects in exposed humans.

Subsection 6 Uncertainties

This section reviews the sources of uncertainty in the risk estimates for humans, and evaluates which sources of uncertainty are likely to underestimate and which are likely to overestimate risk.

Subsection 7 Summary and Conclusions of the Screening-Level Risk Assessment

Subsection 8 References for Risk Assessment

This section provides full citations for EPA guidance documents and scientific publications referenced in the risk assessment.

Selection of Chemicals Of Potential Concern

This section selects chemicals of potential concern for further analysis in the risk assessment. Chemicals of Potential Concern (COPCs) are chemicals that exist in the environment at concentration levels of potential health concern to humans. For the risk assessment, each monitor location will be evaluated separately, so the data analysis and selection of COPCs is presented individually for each monitor.

Chemicals of potential concern (COPCs) are chemicals that a) are present, and b) occur at concentrations that are or might be of health concern to exposed humans. The U.S. Environmental Protection Agency (USEPA)

has derived a standard method for selecting COPCs, as detailed in Risk Assessment Guidance for Superfund: Human Health Evaluation Manual (Part A) (USEPA 1989a). Additionally, regional-specific guidance has been developed by USEPA Region 8 (1994) for use in the selection of COPCs. In brief, USEPA assumes that any chemical detected is a candidate for selection as a COPC, but identifies a number of methods that may be used for determining when a chemical is not of authentic concern and may be eliminated from further consideration. Each risk assessment may choose to apply some or all of the methods identified by USEPA to select COPCs, as appropriate. It is, however, important to note that the USEPA Risk Assessment Guidance, Part A (1989) superfund method is generally applied to other sites which are not part of the superfund process, because this method provides a selection process to reduce the number of contaminants of potential concern to a reasonable amount using the risk-based scientific approach.

Note that this approach is intended to be conservative. That is, it is considered likely that some of the chemicals retained for evaluation by this screening approach may pose little or no risk, but the intended outcome is that there will be no chemicals of authentic concern that escape this screen.

Step 1: Evaluation of Detection Frequencies

Chemicals having detection frequencies of less than 5% are generally not evaluated, quantitatively, as chemicals of potential concern. A contaminant with a detection frequency of greater than 5% is carried through to the toxicity/concentration screening process (Step 2).

The data analysis and selection of chemicals of potential concern (COPCs) are presented in the following sections for each individual monitoring location. Data are summarized for each location by identifying the chemicals that were analyzed for and calculating the frequency of detection. All chemicals that were detected in less than 5% of the samples were eliminated from further consideration as COPCs; however, risks associated with these chemicals are further evaluated in the uncertainty analysis to address potential cancer and non-cancer risks.

Step 2: Toxicity/Concentration Screen

Those chemicals that had detection frequencies greater than or equal to 5% were carried through a toxicity/concentration screen conducted in accord with USEPA (1994) guidance. This screening step involves comparing the maximum reported concentration of a chemical in a medium to an appropriate Risk-Based Concentration (RBC). RBCs are media-specific health-based levels that if exceeded, could indicate that there is a potential for adverse health effects to occur as a result of exposure. The screening benchmarks that were utilized in this step represent air concentrations of chemicals that are equivalent to either a hazard quotient of 0.1 or a cancer risk or 1E-07 (0.1 in a million). The objective of this screening step is to identify the chemicals in air that -- based on concentration and toxicity -- are most likely to contribute significantly to calculated risks, so that the risk assessment is focused on the "most significant" chemicals.

The maximum detected concentration of each individual chemical at a sample location was then compared to its respective benchmark value. If the maximum detected concentration of a given chemical at a monitoring station is less than its respective RBC, the chemical does not pose an unacceptable health risk and can be eliminated as a COPC.

Summary of COPCs

The following sections provide a summary of the frequency of detection screens and the toxicity/concentration screens for all chemicals positively identified during the monitoring program. Generally the chemicals detected during the monitoring program were found across all stations. The following sections summarize the COPCs for air selected for quantitative evaluation in the subsequent risk assessment.

CAMP Station

Table 5.1 summarizes the COPC screening process for the CAMP monitoring location. As seen, of the 81 chemicals that were initially screened, 33 were eliminated because they were found to have an overall frequency of detection of less than 5%. Of these 33 chemicals, 28 were never detected. An additional 15 chemicals (all non-carcinogens) were eliminated from further evaluation because the maximum detected concentration of each chemical was determined to be less than its respective toxicity screening value. These 15 non-carcinogenic chemicals, however, were evaluated separately for the potential hazards. It should be noted that individual hazard quotients for the 15 eliminated chemicals, using the maximum detected concentration, ranged from 0.0001 to 0.09, with a combined hazard index of 0.3 (i.e., significantly less than the acceptable level of 1). In addition, 12 chemicals were eliminated from further evaluation due to the lack of established toxicity values. A total of 21 chemicals were retained for the risk characterization at this monitoring location.

Welby Station

Table 5.2 summarizes the COPC screening process for the Welby monitoring location. As seen, of the 81 chemicals that were initially screened, 34 were eliminated because they were found to have an overall frequency of detection of less than 5%. Of these 34 chemicals, 30 were never detected. An additional 16 chemicals (all non-carcinogens) were eliminated from further evaluation because the maximum detected concentration of each chemical was determined to be less than its respective toxicity screening value. These 16 non-carcinogenic chemicals, however, were evaluated separately for the potential hazards. It should be noted that individual hazard quotients for the 16 eliminated chemicals, using the maximum detected concentration, ranged from 0.0001 to 0.06, with a combined hazard index of 0.2 (i.e., significantly less than the acceptable level of 1). In addition, 11 chemicals were eliminated from further evaluation due to the lack of established toxicity values. A total of 21 chemicals were retained for the risk characterization at this monitoring location.

Swansea Station

Table 5.3 summarizes the COPC screening process for the Swansea monitoring location. As seen, of the 81 chemicals that were initially screened, 35 were eliminated because they were found to have an overall frequency of detection of less than 5%. Of these 35 chemicals, 33 were never detected. An additional 16 chemicals (all non-carcinogens) were eliminated from further evaluation because the maximum detected concentration of each chemical was determined to be less than its respective toxicity screening value. These 16 non-carcinogenic chemicals, however, were evaluated separately for the potential hazards. It should be noted that individual hazard quotients for the 16 chemicals, using the maximum detected concentration, ranged from 0.0001 to 0.09, with a combined hazard index of 0.3 (i.e., significantly less than the acceptable level of 1). In addition, 11 chemicals were eliminated from further evaluation due to the lack of established toxicity values. A total of 19 chemicals were retained for the risk characterization at this monitoring location.

Exposure Assessment

The process that characterizes the route, duration, intensity, and frequency of contact with a chemical by a receptor is known as the exposure assessment. In this assessment, the receptors of interest are individuals that may reside within a monitoring area, and the principal exposure route of interest is inhalation. For this assessment, exposure to relatively low levels of pollutants repeatedly over a prolonged period of time is evaluated.

The following assumptions were made regarding exposure at the monitoring locations:

- A person lives, works, and otherwise stays near a given monitoring location for a 70-year time period.
- the air that the person breathes, both while indoors and outdoors, contains the same concentrations of pollutants measured in the study.
- Air quality, as reflected by the monitoring results, was assumed to remain relatively constant over the entire 70-year lifetime of a person living in the area.

Analytical data for COPCs were processed to derive exposure concentrations. The first step was to process all chemical results reported as non-detects. A non-detect indicates that the measurement equipment could not positively identify the chemical. This does not mean the chemical is not present; rather, if it is present it is at a concentration lower than the instrument can detect. As is standard practice in conducting risk assessments, all samples reported as non-detects were assigned a value of one-half the lowest concentration that the instrument can detect, known as the sample quantitation limit or SQL.

The concentration term used to assess risk from exposure is the arithmetic mean concentration of a contaminant, averaged over the location where exposure is presumed to occur during a specified time interval (USEPA 1989a). The location and size of the Exposure Point depends in part on human activity patterns and in part on the length of time that is required for a chemical to cause adverse effects.

Because the true mean concentration of a chemical within an Exposure Area cannot be calculated with certainty from a limited set of measurements, the USEPA recommends that the upper 95th confidence limit (UCL) of the arithmetic mean concentration be used as the Exposure Point Concentration (EPC) in calculating exposure and risk (USEPA 1992). If the calculated UCL is higher than the highest measured value, then the maximum value is used as the EPC instead of the UCL (USEPA 1992).

The method used to calculate the 95 percent UCL of a data set depends on whether the data are normal or lognormal in their distribution (USEPA 1992b). Two of the most important distributions involving environmental data are the normal distribution and the lognormal distribution. The graph of a normal distributed data is bell-shaped with the highest point located at the mean. A normal curve is symmetric about the mean, hence the part to the left of the mean is a mirror image of the part to the right. The graph of a lognormal distribution data exhibit frequency distributions that are non-negative and skewed. A simple logarithmic transformation will transform a lognormal distribution into a normal distribution. The equations for calculating the 95 percent UCL for normal distributions (Equation 1) or lognormal distributions (Equation 2), respectively, are as follows:

Equation 1:

$$UCL = \bar{x} + t\left(\frac{s}{\sqrt{n}}\right)$$

Equation 2:

$$UCL = e^{\left(\bar{x} + 0.5s^2 + \frac{sH}{\sqrt{n-1}}\right)}$$

where:

$\bar{x}$ = mean of the untransformed (equation 1) or natural log transformed (equation 2) data

UCL = upper confidence limit

e = constant (base of the natural log, equal to 2.718)

s = standard deviation of the untransformed (equation 1) or natural log transformed (equation 2) data

t = Student's t-statistic (for n-1 samples)

H = Land's H-statistic (Gilbert 1987)

n = number of samples.

The EPC values on a monitoring location basis are summarized in Tables 5.4 to 5.6.

Table 5.1 - COPC Selection for CAMP

Compound	CAS Number	CAMP site (DECO) 2105 Broadway, Denver, CO # of ND's	Detection Freq	Retain as COPC?	Maximum (ug/m3)	Screening Conc (ug/m3)	Retain as COPC?
Volatiles (57 samples)							
Acetylene	74-86-2	0	100.0%	yes	10.959	ND	ND
Propylene	115-07-1	0	100.0%	yes	5.266	ND	ND
Dichlorodifluoromethane	75-71-8	0	100.0%	yes	5.242	20	no
Chloromethane	74-87-3	0	100.0%	yes	2.272	9	no
Dichlorotetrafluoroethane	76-14-2	57	0.0%	no	--	ND	--
Vinyl chloride	75-01-4	57	0.0%	no	--	0.0113636	--
1,3 - Butadiene	106-99-0	10	82.5%	yes	0.951	0.0033333	yes
Bromomethane	74-83-9	57	0.0%	no	--	0.5	--
Chloroethane	75-00-3	57	0.0%	no	--	0.1206273	--
Acetonitrile	75-05-8	47	17.5%	yes	87.137	6	yes
Trichlorofluoromethane	75-69-4	0	100.0%	yes	4.663	70	no
Acrylonitrile	107-13-1	56	1.8%	no	--	0.0014706	--
1,1 - Dichloroethene	75-35-4	56	1.8%	no	--	0.5	--
Methylene chloride	75-09-2	4	93.0%	yes	31.892	0.2188184	yes
Trichlorotrifluoroethane	76-13-1	1	98.2%	yes	1.993	ND	ND
trans - 1,2 - Dichloroethylene	156-60-5	57	0.0%	no	--	7	--
1,1 - Dichloroethane	74-34-3	57	0.0%	no	--	49	--
Methyl tert-butyl ether	1634-04-4	57	0.0%	no	--	0.3846154	--
Methyl ethyl ketone	78-93-3	34	40.4%	yes	34.890	500	no
Chloroprene	126-99-8	57	0.0%	no	--	0.7	--
cis - 1,2 - Dichloroethylene	156-59-2	57	0.0%	no	--	3.5	--
Bromochloromethane	74-97-5	57	0.0%	no	--	ND	--
Chloroform	67-66-3	48	15.8%	yes	0.830	0.004329	yes
Ethyl tert-butyl ether	637-92-3	57	0.0%	no	--	ND	--
1,2 - Dichloroethane	107-06-2	56	1.8%	no	--	0.0038462	--
1,1,1 - Trichloroethane	71-55-6	45	21.1%	yes	4.147	100	no
Benzene	71-43-2	0	100.0%	yes	7.381	0.0128205	yes
Carbon tetrachloride	56-23-5	7	87.7%	yes	1.132	0.0066667	yes
tert-Amyl methyl ether	994-05-8	57	0.0%	no	--	ND	--
1,2 - Dichloropropane	78-87-5	57	0.0%	no	--	0.0052632	--
Ethyl acrylate	140-88-5	57	0.0%	no	--	0.0071429	--
Bromodichloromethane	75-27-4	57	0.0%	no	--	0.0056497	--
Trichloroethylene	79-01-6	53	7.0%	yes	3.168	0.000875	yes
Methyl methacrylate	80-62-6	56	1.8%	no	--	70	--
cis - 1,3 - Dichloropropene	10061-01-5	57	0.0%	no	--	ND	--
Methyl isobutyl ketone	108-10-1	55	3.5%	no	--	300	--
trans - 1,3 - Dichloropropene	10061-02-6	57	0.0%	no	--	ND	--
1,1,2 - Trichloroethane	79-00-5	57	0.0%	no	--	0.00625	--
Toluene	108-88-3	0	100.0%	yes	50.353	40	yes
Dibromochloromethane	124-48-1	57	0.0%	no	--	0.0041667	--
1,2 - Dibromoethane	106-93-4	57	0.0%	no	--	0.0001667	--
n - Octane	111-65-9	18	68.4%	yes	5.420	ND	ND

Compound	CAS Number	CAMP site (DECO) 2105 Broadway, Denver, CO # of ND's	Detection Freq	Retain as COPC?	Maximum (ug/m3)	Screening Conc (ug/m3)	Retain as COPC?
Tetrachloroethylene	127-18-4	34	40.4%	yes	10.581	0.0169492	yes
Chlorobenzene	108-90-7	57	0.0%	no	--	100	--
Ethylbenzene	100-41-4	0	100.0%	yes	3.126	100	no
m,p - Xylene	108-38-3 / 106-42-3	0	100.0%	yes	10.291	10.2	yes
Bromoform	75-25-2	57	0.0%	no	--	0.0909091	--
Styrene	100-42-5	31	45.6%	yes	1.448	100	no
1,1,2,2 - Tetrachloroethane	79-34-5	57	0.0%	no	--	0.0017241	--
o - Xylene	95-47-6	0	100.0%	yes	5.037	10.2	no
1,3,5 - Trimethylbenzene	108-67-8	0	100.0%	yes	1.770	0.6	yes
1,2,4 - Trimethylbenzene	95-63-6	0	100.0%	yes	5.064	0.6	yes
m - Dichlorobenzene	541-73-1	57	0.0%	no	--	10.5	--
Chloromethylbenzene	100-44-7	57	0.0%	no	--	0.0020408	--
p - Dichlorobenzene	106-46-7	54	5.3%	yes	0.451	0.0090909	yes
o - Dichlorobenzene	95-50-1	57	0.0%	no	--	20	--
1,2,4 - Trichlorobenzene	120-82-1	57	0.0%	no	--	20	--
Hexachloro - 1,3 - butadiene	87-68-3	57	0.0%	no	--	0.0045455	--
Inorganics (59 samples)							
Antimony	7440-36-0	0	100.0%	yes	0.008	ND	ND
Arsenic	7440-38-2	0	100.0%	yes	0.006	2.326E-05	yes
Beryllium	7440-41-7	24	57.1%	yes	0.000	4.167E-05	yes
Cadmium	7440-43-9	0	100.0%	yes	0.001	5.556E-05	yes
Chromium (total)	7440-47-3	0	100.0%	yes	0.009	0.01	no
Cobalt	7440-84-4	0	100.0%	yes	0.002	3.571E-05	yes
Lead	7439-92-1	0	100.0%	yes	0.049	1.5	no
Manganese	7439-96-5	0	100.0%	yes	0.136	0.005	yes
Mercury	7439-97-6	0	100.0%	yes	0.000	0.03	no
Nickel	7440-02-0	0	100.0%	yes	0.005	0.009	no
Selenium	7782-49-2	0	100.0%	yes	0.002	2	no
Carbonyls (56 samples)							
Formaldehyde	50-00-0	0	100.0%	yes	18.202	0.98	yes
Acetaldehyde	75-07-0	0	100.0%	yes	9.294	0.0454545	yes
Acetone	67-64-1	0	100.0%	yes	13.169	315	no
Propionaldehyde	123-38-6	0	100.0%	yes	1.342	ND	ND
Crotonaldehyde	123-73-9	0	100.0%	yes	0.374	0.0001842	yes
Butyr / Isobutyraldehyde	123-72-8 / 78-84-2	0	100.0%	yes	2.259	ND	ND
Benzaldehyde	100-52-7	1	98.2%	yes	1.060	35	no
Isovaleraldehyde	590-86-3	54	5.3%	yes	0.359	ND	ND
Valeraldehyde	110-62-3	0	100.0%	yes	1.221	ND	ND
Tolualdehydes (o-, m-, p-)	1334-78-7	1	98.2%	yes	1.178	ND	ND
Hexaldehyde	66-25-1	0	100.0%	yes	2.598	ND	ND
2,5-Dimethylbenzaldehyde	5779-94-2	52	8.8%	yes	0.194	ND	ND

Table 5.1, completed.

Table 5.2 - COPC Selection For Welby

Compound	CAS Number	Welby site (WECO) 78th Av. & Steele St., Denver, CO # of ND's	Detection Freq	Retain as COPC?	Maximum (ug/m3)	Screening Conc (ug/m3)	Retain as COPC?
Volatiles (57 samples)							
Acetylene	74-86-2	1	98.1%	yes	7.423	ND	ND
Propylene	115-07-1	0	100.0%	yes	5.817	ND	ND
Dichlorodifluoromethane	75-71-8	0	100.0%	yes	4.302	20	no
Chloromethane	74-87-3	0	100.0%	yes	1.982	9	no
Dichlorotetrafluoroethane	76-14-2	52	0.0%	no	--	ND	--
Vinyl chloride	75-01-4	52	0.0%	no	--	0.0113636	--
1,3 - Butadiene	106-99-0	21	59.6%	yes	0.929	0.0033333	yes
Bromomethane	74-83-9	50	3.8%	no	--	0.5	--
Chloroethane	75-00-3	52	0.0%	no	--	0.1206273	--
Acetonitrile	75-05-8		VOID			6	
Trichlorofluoromethane	75-69-4	0	100.0%	yes	7.473	70	no
Acrylonitrile	107-13-1	52	0.0%	no	--	0.0014706	--
1,1 - Dichloroethene	75-35-4	52	0.0%	no	--	0.5	--
Methylene chloride	75-09-2	11	78.8%	yes	5.767	0.2188184	yes
Trichlorotrifluoroethane	76-13-1	3	94.2%	yes	1.533	ND	ND
trans - 1,2 - Dichloroethylene	156-60-5	52	0.0%	no	--	7	--
1,1 - Dichloroethane	74-34-3	52	0.0%	no	--	49	--
Methyl tert-butyl ether	1634-04-4	28	46.2%	yes	7.463	0.3846154	yes
Methyl ethyl ketone	78-93-3	30	42.3%	yes	33.209	500	no
Chloroprene	126-99-8	52	0.0%	no	--	0.7	--
cis - 1,2 - Dichloroethylene	156-59-2	52	0.0%	no	--	3.5	--
Bromochloromethane	74-97-5	52	0.0%	no	--	ND	--
Chloroform	67-66-3	46	11.5%	yes	0.439	0.004329	yes
Ethyl tert-butyl ether	637-92-3	52	0.0%	no	--	ND	--
1,2 - Dichloroethane	107-06-2	52	0.0%	no	--	0.0038462	--
1,1,1 - Trichloroethane	71-55-6	43	17.3%	yes	0.491	100	no
Benzene	71-43-2	0	100.0%	yes	7.189	0.0128205	yes
Carbon tetrachloride	56-23-5	9	82.7%	yes	1.007	0.0066667	yes
tert-Amyl methyl ether	994-05-8	52	0.0%	no	--	ND	--
1,2 - Dichloropropane	78-87-5	52	0.0%	no	--	0.0052632	--
Ethyl acrylate	140-88-5	52	0.0%	no	--	0.0071429	--
Bromodichloromethane	75-27-4	52	0.0%	no	--	0.0056497	--
Trichloroethylene	79-01-6	50	3.8%	no	--	0.000875	--
Methyl methacrylate	80-62-6	52	0.0%	no	--	70	--
cis - 1,3 - Dichloropropene	10061-01-5	52	0.0%	no	--	ND	--
Methyl isobutyl ketone	108-10-1	46	11.5%	yes	41.539	300	no
trans - 1,3 - Dichloropropene	10061-02-6	52	0.0%	no	--	ND	--
1,1,2 - Trichloroethane	79-00-5	52	0.0%	no	--	0.00625	--
Toluene	108-88-3	0	100.0%	yes	68.821	40	yes
Dibromochloromethane	124-48-1	52	0.0%	no	--	0.0041667	--
1,2 - Dibromoethane	106-93-4	52	0.0%	no	--	0.0001667	--
n - Octane	111-65-9	25	51.9%	yes	1.402	ND	ND

Compound	CAS Number	Welby site (WECO) 78th Av. & Steele St., Denver, CO # of ND's	Detection Freq	Retain as COPC?	Maximum (ug/m3)	Screening Conc (ug/m3)	Retain as COPC?
Tetrachloroethylene	127-18-4	38	26.9%	yes	1.899	0.0169492	yes
Chlorobenzene	108-90-7	52	0.0%	no	--	100	--
Ethylbenzene	100-41-4	3	94.2%	yes	3.517	100	no
m,p - Xylene	108-38-3 / 106-42-3	0	100.0%	yes	12.072	10.2	yes
Bromoform	75-25-2	52	0.0%	no	--	0.0909091	--
Styrene	100-42-5	43	17.3%	yes	2.599	100	no
1,1,2,2 - Tetrachloroethane	79-34-5	52	0.0%	no	--	0.0017241	--
o - Xylene	95-47-6	1	98.1%	yes	4.038	10.2	no
1,3,5 - Trimethylbenzene	108-67-8	26	50.0%	yes	1.082	0.6	yes
1,2,4 - Trimethylbenzene	95-63-6	3	94.2%	yes	3.392	0.6	yes
m - Dichlorobenzene	541-73-1	52	0.0%	no	--	10.5	--
Chloromethylbenzene	100-44-7	52	0.0%	no	--	0.0020408	--
p - Dichlorobenzene	106-46-7	50	3.8%	no	--	0.0090909	--
o - Dichlorobenzene	95-50-1	52	0.0%	no	--	20	--
1,2,4 - Trichlorobenzene	120-82-1	52	0.0%	no	--	20	--
Hexachloro - 1,3 - butadiene	87-68-3	52	0.0%	no	--	0.0045455	--
Inorganics (59 samples)							
Antimony	7440-36-0	0	100.0%	yes	0.008	ND	ND
Arsenic	7440-38-2	0	100.0%	yes	0.004	2.326E-05	yes
Beryllium	7440-41-7	24	57.9%	yes	0.000	4.167E-05	yes
Cadmium	7440-43-9	4	93.0%	yes	0.001	5.556E-05	yes
Chromium (total)	7440-47-3	0	100.0%	yes	0.006	0.01	no
Cobalt	7440-84-4	0	100.0%	yes	0.013	3.571E-05	yes
Lead	7439-92-1	0	100.0%	yes	0.060	1.5	no
Manganese	7439-96-5	0	100.0%	yes	0.148	0.005	yes
Mercury	7439-97-6	0	100.0%	yes	0.000	0.03	no
Nickel	7440-02-0	0	100.0%	yes	0.004	0.009	no
Selenium	7782-49-2	0	100.0%	yes	0.002	2	no
Carbonyls (56 samples)							
Formaldehyde	50-00-0	0	100.0%	yes	7.455	0.98	yes
Acetaldehyde	75-07-0	0	100.0%	yes	6.398	0.0454545	yes
Acetone	67-64-1	0	100.0%	yes	8.413	315	no
Propionaldehyde	123-38-6	1	98.1%	yes	0.894	ND	ND
Crotonaldehyde	123-73-9	1	98.1%	yes	0.282	0.0001842	yes
Butyr / Isobutyraldehyde	123-72-8 / 78-84-2	0	100.0%	yes	0.999	ND	ND
Benzaldehyde	100-52-7	0	100.0%	yes	0.508	35	no
Isovaleraldehyde	590-86-3	52	1.9%	no	--	ND	--
Valeraldehyde	110-62-3	0	100.0%	yes	0.352	ND	ND
Tolualdehydes (o-, m-, p-)	1334-78-7	0	100.0%	yes	0.803	ND	ND
Hexaldehyde	66-25-1	0	100.0%	yes	0.435	ND	ND
2,5-Dimethylbenzaldehyde	5779-94-2	50	5.7%	yes	0.260	ND	ND

Acetonitrile VOID at Welby and Swansea, due to contamination problem.

Table 5.2, completed.

Table 5.3 - COPC Selection For Swansea

Compound	CAS Number	Swansea site (SWCO) 4650 Columbine St., Denver, CO # of ND's	Detection Freq	Retain as COPC?	Maximum (ug/m3)	Screening Conc (ug/m3)	Retain as COPC?
Volatiles (57 samples)							
Acetylene	74-86-2	1	96.6%	yes	10.917	ND	ND
Propylene	115-07-1	0	100.0%	yes	6.282	ND	ND
Dichlorodifluoromethane	75-71-8	1	96.6%	yes	4.747	20	no
Chloromethane	74-87-3	1	96.6%	yes	1.652	9	no
Dichlorotetrafluoroethane	76-14-2	29	0.0%	no	--	ND	--
Vinyl chloride	75-01-4	29	0.0%	no	--	0.0113636	--
1,3 - Butadiene	106-99-0	7	75.9%	yes	1.040	0.0033333	yes
Bromomethane	74-83-9	29	0.0%	no	--	0.5	--
Chloroethane	75-00-3	29	0.0%	no	--	0.1206273	--
Acetonitrile	75-05-8		VOID			6	
Trichlorofluoromethane	75-69-4	1	96.6%	yes	14.215	70	no
Acrylonitrile	107-13-1	28	3.4%	no	--	0.0014706	--
1,1 - Dichloroethene	75-35-4	29	0.0%	no	--	0.5	--
Methylene chloride	75-09-2	7	75.9%	yes	3.092	0.2188184	yes
Trichlorotrifluoroethane	76-13-1	1	96.6%	yes	1.379	ND	ND
trans - 1,2 - Dichloroethylene	156-60-5	29	0.0%	no	--	7	--
1,1 - Dichloroethane	74-34-3	29	0.0%	no	--	49	--
Methyl tert-butyl ether	1634-04-4	29	0.0%	no	--	0.3846154	--
Methyl ethyl ketone	78-93-3	19	34.5%	yes	24.715	500	no
Chloroprene	126-99-8	29	0.0%	no	--	0.7	--
cis - 1,2 - Dichloroethylene	156-59-2	29	0.0%	no	--	3.5	--
Bromochloromethane	74-97-5	29	0.0%	no	--	ND	--
Chloroform	67-66-3	25	13.8%	yes	0.781	0.004329	yes
Ethyl tert-butyl ether	637-92-3	29	0.0%	no	--	ND	--
1,2 - Dichloroethane	107-06-2	29	0.0%	no	--	0.0038462	--
1,1,1 - Trichloroethane	71-55-6	25	13.8%	yes	0.273	100	no
Benzene	71-43-2	0	100.0%	yes	7.029	0.0128205	yes
Carbon tetrachloride	56-23-5	4	86.2%	yes	1.007	0.0066667	yes
tert-Amyl methyl ether	994-05-8	29	0.0%	no	--	ND	--
1,2 - Dichloropropane	78-87-5	29	0.0%	no	--	0.0052632	--
Ethyl acrylate	140-88-5	29	0.0%	no	--	0.0071429	--
Bromodichloromethane	75-27-4	29	0.0%	no	--	0.0056497	--
Trichloroethylene	79-01-6	29	0.0%	no	--	0.000875	--
Methyl methacrylate	80-62-6	29	0.0%	no	--	70	--
cis - 1,3 - Dichloropropene	10061-01-5	29	0.0%	no	--	ND	--
Methyl isobutyl ketone	108-10-1	26	10.3%	yes	1.884	300	no
trans - 1,3 - Dichloropropene	10061-02-6	29	0.0%	no	--	ND	--
1,1,2 - Trichloroethane	79-00-5	29	0.0%	no	--	0.00625	--
Toluene	108-88-3	0	100.0%	yes	63.620	40	yes
Dibromochloromethane	124-48-1	29	0.0%	no	--	0.0041667	--
1,2 - Dibromoethane	106-93-4	29	0.0%	no	--	0.0001667	--
n - Octane	111-65-9	10	65.5%	yes	1.682	ND	ND
Tetrachloroethylene	127-18-4	16	44.8%	yes	2.984	0.0169492	yes

Compound	CAS Number	Swansea site (SWCO) 4650 Columbine St., Denver, CO # of ND's	Detection Freq	Retain as COPC?	Maximum (ug/m3)	Screening Conc (ug/m3)	Retain as COPC?
Chlorobenzene	108-90-7	29	0.0%	no	--	100	--
Ethylbenzene	100-41-4	0	100.0%	yes	6.383	100	no
m,p - Xylene	108-38-3 / 106-42-3	0	100.0%	yes	14.677	10.2	yes
Bromoform	75-25-2	29	0.0%	no	--	0.0909091	--
Styrene	100-42-5	25	13.8%	yes	3.664	100	no
1,1,2,2 - Tetrachloroethane	79-34-5	29	0.0%	no	--	0.0017241	--
o - Xylene	95-47-6	0	100.0%	yes	5.732	10.2	no
1,3,5 - Trimethylbenzene	108-67-8	5	82.8%	yes	1.426	0.6	yes
1,2,4 - Trimethylbenzene	95-63-6	0	100.0%	yes	4.621	0.6	yes
m - Dichlorobenzene	541-73-1	29	0.0%	no	--	10.5	--
Chloromethylbenzene	100-44-7	29	0.0%	no	--	0.0020408	--
p - Dichlorobenzene	106-46-7	29	0.0%	no	--	0.0090909	--
o - Dichlorobenzene	95-50-1	29	0.0%	no	--	20	--
1,2,4 - Trichlorobenzene	120-82-1	29	0.0%	no	--	20	--
Hexachloro - 1,3 - butadiene	87-68-3	29	0.0%	no	--	0.0045455	--
Inorganics (59 samples)							
Antimony	7440-36-0	0	100.0%	yes	0.005	ND	ND
Arsenic	7440-38-2	0	100.0%	yes	0.006	2.326E-05	yes
Beryllium	7440-41-7	16	48.4%	yes	0.000	4.167E-05	yes
Cadmium	7440-43-9	2	93.5%	yes	0.003	5.556E-05	yes
Chromium (total)	7440-47-3	0	100.0%	yes	0.009	0.01	no
Cobalt	7440-84-4	0	100.0%	yes	0.002	0.00	yes
Lead	7439-92-1	0	100.0%	yes	0.079	1.5	no
Manganese	7439-96-5	0	100.0%	yes	0.124	0.005	yes
Mercury	7439-97-6	1	96.8%	yes	0.000	0.03	no
Nickel	7440-02-0	0	100.0%	yes	0.005	0.009	no
Selenium	7782-49-2	0	100.0%	yes	0.003	2	no
Carbonyls (56 samples)							
Formaldehyde	50-00-0	0	100.0%	yes	9.418	0.98	yes
Acetaldehyde	75-07-0	0	100.0%	yes	5.545	0.0454545	yes
Acetone	67-64-1	0	100.0%	yes	8.369	315	no
Propionaldehyde	123-38-6	0	100.0%	yes	0.727	ND	ND
Crotonaldehyde	123-73-9	0	100.0%	yes	0.137	0.0001842	yes
Butyr / Isobutyraldehyde	123-72-8 / 78-84-2	0	100.0%	yes	0.835	ND	ND
Benzaldehyde	100-52-7	0	100.0%	yes	1.009	35	no
Isovaleraldehyde	590-86-3	28	3.4%	no	--	ND	--
Valeraldehyde	110-62-3	0	100.0%	yes	0.289	ND	ND
Tolualdehydes (o-, m-, p-)	1334-78-7	0	100.0%	yes	0.541	ND	ND
Hexaldehyde	66-25-1	0	100.0%	yes	0.331	ND	ND
2,5-Dimethylbenzaldehyde	5779-94-2	24	17.2%	yes	0.269	ND	ND

Acetonitrile VOID at Welby and Swansea, due to contamination problem.

Table 5.3, completed.

Table 5.4 - Exposure Point Concentrations for CAMP Monitoring Location

	Chemical	Max Value (ug/m3)	Min Value (ug/m3)	GM (ug/m3)	AM (ug/m3)	Stdev (ug/m3	UCL95 Norm (ug/m3)	UCL95 LogNorm (ug/m3)	EPC (ug/m3)
Volatiles	1,3-Butadiene	9.5E-01	1.1E-01	2.6E-01	3.1E-01	1.8E-01	3.5E-01	3.6E-01	0.36
	Acetonitrile	8.7E+01	3.6E-01	5.4E-01	2.3E+00	1.2E+01	4.9E+00	1.2E+00	4.89
	Methylene Chloride	3.2E+01	1.2E-01	7.5E-01	1.5E+00	4.2E+00	2.4E+00	1.6E+00	2.43
	Chloroform	8.3E-01	4.9E-02	1.8E-01	2.0E-01	1.1E-01	2.2E-01	2.1E-01	0.22
	Benzene	7.4E+00	1.3E+00	2.9E+00	3.1E+00	1.2E+00	3.4E+00	3.4E+00	3.37
	Carbon Tetrachloride	1.1E+00	1.9E-01	4.4E-01	4.7E-01	1.9E-01	5.2E-01	5.3E-01	0.53
	Trichloroethylene	3.2E+00	1.6E-01	3.5E-01	4.1E-01	4.3E-01	5.1E-01	4.3E-01	0.51
	Toluene	5.0E+01	2.9E+00	8.1E+00	9.4E+00	6.8E+00	1.1E+01	1.1E+01	10.93
	Tetrachloroethylene	1.1E+01	1.4E-01	3.9E-01	6.2E-01	1.4E+00	9.3E-01	6.4E-01	0.93
	m,p - Xylene	1.0E+01	1.6E+00	4.0E+00	4.3E+00	1.8E+00	4.7E+00	4.8E+00	4.79
	1,3,5-Trimethylbenzene	1.8E+00	4.9E-01	1.0E+00	1.1E+00	3.1E-01	1.1E+00	1.2E+00	1.15
	1,2,4-Trimethylbenzene	5.1E+00	1.5E+00	2.9E+00	3.0E+00	8.5E-01	3.2E+00	3.2E+00	3.22
	p - Dichlorobenzene	4.5E-01	1.2E-01	4.2E-01	4.3E-01	7.0E-02	4.5E-01	4.7E-01	0.45
Carbonyls	Formaldehyde	1.8E+01	4.4E+00	7.5E+00	8.2E+00	3.8E+00	9.0E+00	8.9E+00	9.00
	Acetaldehyde	9.3E+00	2.2E+00	3.9E+00	4.1E+00	1.6E+00	4.5E+00	4.5E+00	4.50
	Crotonaldehyde	3.7E-01	3.5E-02	9.1E-02	1.2E-01	9.6E-02	1.4E-01	1.4E-01	0.14
Inorganics	Arsenic	5.9E-03	2.1E-05	4.6E-04	1.3E-03	1.5E-03	1.7E-03	4.9E-03	4.9E-03
	Beryllium	1.3E-04	6.7E-06	2.0E-05	2.9E-05	2.9E-05	3.6E-05	3.8E-05	3.8E-05
	Cadmium	9.2E-04	9.3E-05	3.2E-04	3.5E-04	1.6E-04	3.9E-04	3.9E-04	3.9E-04
	Cobalt	1.9E-03	2.8E-04	8.6E-04	9.5E-04	4.1E-04	1.0E-03	1.1E-03	1.1E-03
	Manganese	1.4E-01	7.2E-03	3.9E-02	4.7E-02	2.9E-02	5.4E-02	5.7E-02	5.7E-02

All non-detects were evaluated at 1/2 the detection limit

GM = Geometric Mean
AM = Arithmetic Mean
STDEV = Standard Deviation

Table 5.5 - Exposure Point Concentrations for Welby Monitoring Location

	Chemical	Max Value (ug/m3)	Min Value (ug/m3)	GM (ug/m3)	AM (ug/m3)	Stdev (ug/m3)	UCL95 Norm (ug/m3)	UCL95 LogNorm (ug/m3)	EPC (ug/m3)
Volatiles	1,3-Butadiene	9.3E-01	6.6E-02	1.7E-01	2.1E-01	1.6E-01	2.5E-01	2.4E-01	0.25
	Acetonitrile	VOID							
	Methylene Chloride	5.8E+00	1.2E-01	4.2E-01	6.7E-01	8.9E-01	8.8E-01	8.6E-01	0.88
	Methyl tert-Butyl Ether	7.5E+00	2.9E-01	8.4E-01	1.6E+00	2.0E+00	2.1E+00	2.5E+00	2.51
	Chloroform	4.4E-01	1.5E-01	1.8E-01	1.9E-01	5.5E-02	2.0E-01	2.0E-01	0.20
	Benzene	7.2E+00	6.7E-01	2.1E+00	2.4E+00	1.2E+00	2.6E+00	2.7E+00	2.72
	Carbon Tetrachloride	1.0E+00	1.9E-01	4.1E-01	4.5E-01	1.9E-01	4.9E-01	5.0E-01	0.50
	Toluene	6.9E+01	1.5E+00	6.2E+00	8.2E+00	9.4E+00	1.0E+01	9.8E+00	10.37
	Tetrachloroethylene	1.9E+00	1.4E-01	3.1E-01	3.6E-01	2.9E-01	4.3E-01	4.0E-01	0.43
	m,p - Xylene	1.2E+01	7.8E-01	3.4E+00	4.2E+00	2.6E+00	4.8E+00	5.2E+00	5.19
	1,3,5-Trimethylbenzene	1.1E+00	9.8E-02	2.9E-01	3.6E-01	2.4E-01	4.2E-01	4.4E-01	0.44
	1,2,4-Trimethylbenzene	3.4E+00	2.2E-01	9.0E-01	1.1E+00	6.9E-01	1.3E+00	1.3E+00	1.35
Carbonyls	Formaldehyde	7.5E+00	1.2E+00	3.2E+00	3.5E+00	1.4E+00	3.8E+00	3.9E+00	3.88
	Acetaldehyde	6.4E+00	1.3E+00	2.7E+00	2.9E+00	1.1E+00	3.1E+00	3.1E+00	3.14
	Crotonaldehyde	2.8E-01	1.2E-02	5.9E-02	7.7E-02	6.1E-02	9.2E-02	9.7E-02	0.10
Inorganics	Arsenic	4.1E-03	1.3E-06	3.5E-04	9.9E-04	1.1E-03	1.2E-03	4.0E-03	4.0E-03
	Beryllium	1.5E-04	6.0E-06	1.9E-05	3.0E-05	3.3E-05	3.7E-05	3.8E-05	3.8E-05
	Cadmium	6.7E-04	1.2E-05	1.9E-04	2.5E-04	1.6E-04	2.9E-04	3.8E-04	3.8E-04
	Cobalt	1.3E-02	1.6E-04	6.4E-04	9.2E-04	1.6E-03	1.3E-03	1.0E-03	1.3E-03
	Manganese	1.5E-01	9.3E-03	3.7E-02	4.7E-02	3.2E-02	5.4E-02	5.8E-02	5.8E-02

All non-detects were evaluated at 1/2 the detection limit

GM = Geometric Mean
AM = Arithmetic Mean
STDEV = Standard Deviation

Acetonitrile VOID at Welby and Swansea, due to contamination problem.

Table 5.6 - Exposure Point Concentrations for Swansea Monitoring Location

	Chemical	Max Value (ug/m3)	Min Value (ug/m3)	GM (ug/m3)	AM (ug/m3)	Stdev (ug/m3)	UCL95 Norm (ug/m3)	UCL95 LogNorm (ug/m3)	EPC (ug/m3)
Volatiles	1,3-Butadiene	1.0E+00	8.8E-02	2.3E-01	2.8E-01	2.2E-01	3.5E-01	3.7E-01	0.37
	Acetonitrile	VOID							
	Methylene Chloride	3.1E+00	1.2E-01	4.6E-01	7.8E-01	8.5E-01	1.1E+00	1.3E+00	1.32
	Chloroform	7.8E-01	1.5E-01	1.8E-01	2.0E-01	1.3E-01	2.4E-01	2.2E-01	0.24
	Benzene	7.0E+00	8.3E-01	2.5E+00	2.8E+00	1.5E+00	3.3E+00	3.4E+00	3.38
	Carbon Tetrachloride	1.0E+00	1.9E-01	4.3E-01	4.7E-01	2.0E-01	5.3E-01	5.5E-01	0.55
	Toluene	6.4E+01	1.7E+00	8.7E+00	1.1E+01	1.1E+01	1.5E+01	1.5E+01	14.84
	Tetrachloroethylene	3.0E+00	2.0E-01	4.7E-01	7.2E-01	6.7E-01	9.3E-01	1.1E+00	1.10
	m,p - Xylene	1.5E+01	1.0E+00	6.3E+00	7.2E+00	3.5E+00	8.3E+00	9.2E+00	9.18
	1,3,5-Trimethylbenzene	1.4E+00	1.2E-01	4.6E-01	5.2E-01	2.7E-01	6.1E-01	6.3E-01	0.63
	1,2,4-Trimethylbenzene	4.6E+00	4.4E-01	1.3E+00	1.5E+00	8.1E-01	1.8E+00	1.8E+00	1.81
Carbonyls	Formaldehyde	9.4E+00	1.5E+00	4.6E+00	5.0E+00	1.9E+00	5.6E+00	5.9E+00	5.86
	Acetaldehyde	5.5E+00	1.4E+00	3.1E+00	3.3E+00	1.1E+00	3.6E+00	3.7E+00	3.67
	Crotonaldehyde	1.4E-01	1.7E-02	5.0E-02	5.7E-02	2.9E-02	6.6E-02	6.8E-02	0.07
Inorganics	Arsenic	5.7E-03	1.5E-07	2.2E-04	9.9E-04	1.4E-03	1.4E-03	1.3E-02	5.7E-03
	Beryllium	2.5E-04	6.3E-06	2.1E-05	4.6E-05	6.0E-05	6.5E-05	8.9E-05	8.9E-05
	Cadmium	2.8E-03	1.3E-05	3.6E-04	5.1E-04	4.8E-04	6.5E-04	9.2E-04	9.2E-04
	Cobalt	2.3E-03	1.7E-04	7.4E-04	8.4E-04	4.4E-04	9.7E-04	1.0E-03	1.0E-03
	Manganese	1.2E-01	6.5E-03	4.5E-02	5.3E-02	2.9E-02	6.2E-02	7.0E-02	7.0E-02

All non-detects were evaluated at 1/2 the detection limit

GM = Geometric Mean
AM = Arithmetic Mean
STDEV = Standard Deviation

Acetonitrile VOID at Welby and Swansea, due to contamination problem.

Toxicity Assessment

Overview

The basic objective of a toxicity assessment is to identify what adverse health effects a chemical causes, and how the appearance of these adverse effects depends on dose. In addition, the toxic effects of a chemical frequently depend on the route of exposure (oral, inhalation, dermal), duration of exposure (subchronic, chronic or lifetime), age, sex, diet, family traits, lifestyle, and state of health. Thus, a full description of the toxic effects of a chemical includes a listing of what adverse health effects the chemical may cause, and how the occurrence of these effects depends upon dose, route, duration of exposure, age, sex, diet, family traits, lifestyle, and state of health.

The toxicity assessment process is usually divided into two parts: the first characterizes and quantifies the cancer effects of the chemical, while the second addresses the non-cancer effects of the chemical. This two-part approach is employed because there are typically major differences in the risk assessment methods used to assess cancer and non-cancer effects. For example, cancer risks are expressed as a probability of suffering an adverse effect (cancer) during a lifetime and non-cancer hazards are expressed, semi-quantitatively, in terms of the hazard quotient (HQ), defined as the ratio between an individual's estimated exposure and the reference concentration (RfC). However, both cancer risks and hazard quotients estimate population risks and not an individual's personal risk A brief summary of the potential toxic effects of some major contaminants is provided in Attachment 1.

Cancer Effects

For cancer effects, the toxicity assessment process has two components. The first is a qualitative evaluation of the weight of evidence that the chemical does or does not cause cancer in humans. Typically, this evaluation is performed by the EPA, using the system summarized in the table below:

Category	Meaning	Description
A	Known human carcinogen	Sufficient evidence of cancer in humans.
B1	Probable human carcinogen	Suggestive evidence of cancer incidence in humans.
B2	Probable human carcinogen	Sufficient evidence of cancer in animals, but lack of data or insufficient data from humans.
C	Possible human carcinogen	Suggestive evidence of carcinogenicity in animals.
D	Cannot be evaluated	No evidence or inadequate evidence of cancer in animals or humans.

For chemicals which are classified in Group A, B1, B2, or C, the second part of the toxicity assessment is to describe the carcinogenic potency of the chemical. This is done by quantifying how the number of cancers observed in exposed animals or humans increases as the dose increases. Typically, it is assumed that the dose response curve for cancer has no threshold, arising from the origin, and increasing linearly until high doses are reached. Thus, the most convenient descriptor of cancer potency is the slope of the dose-response curve at low dose (where the slope is still linear). This is referred to as the Slope Factor (SF), which has dimensions of risk of cancer per unit dose. Conversely, the inhalation unit risk (IUR) is defined as the upper-bound excess lifetime cancer risk estimated to result from continuous exposure to an agent at a concentration of 1 ug/m3 in air.

Estimating the cancer SF and/or IUR is often complicated by the fact that observable increases in cancer incidence usually occur only at relatively high doses, frequently in the part of the dose-response curve that is no longer linear. Thus, it is necessary to use mathematical models to extrapolate from the observed high dose data to the desired (but not measurable) slope at low dose. In order to account for the uncertainty in this extrapolation process, EPA typically chooses to employ the upper 95th confidence limit of the slope as the Slope Factor. That is, there is a 95% probability that the true cancer potency is lower than the value chosen for the Slope Factor. This approach ensures that there is a margin of safety in cancer as well as non-cancer risk estimates.

Non-Cancer Effects

Essentially all chemicals can cause adverse health effects if given at a high enough dose. However, when the dose is sufficiently low, no adverse effect is observed. Thus, in characterizing the non-cancer effects of a chemical, the key parameter is the threshold dose or concentration at which an adverse effect first becomes evident. Exposures below the threshold are considered to be safe, while exposures above the threshold are likely to cause an effect.

The threshold dose is typically estimated from toxicological data (derived from studies of humans and/or animals) by finding the highest dose that does not produce an observable adverse effect, and the lowest dose that does produce an effect. These are referred to as the "No-observed-adverse-effect-level" (NOAEL) and the "Lowest-observed-adverse-effect-level" (LOAEL), respectively. The threshold is presumed to lie in the interval between the NOAEL and the LOAEL. However, in order to be conservative (protective), non-cancer risk evaluations are not based directly on the threshold exposure level, but on a value referred to as the Reference Dose (RfD) or Reference Concentration (RfC). The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure (dose) to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. The RfC has a similar definition, but represents the continuous inhalation concentration that is likely to be without an appreciable risk of deleterious effects during a lifetime. A RfC is reported in milligrams of pollutant per cubic meter of air (mg/m^3).

The RfD or RfC value is derived from the NOAEL (or the LOAEL if a reliable NOAEL is not available) by dividing by an "uncertainty factor". If the data are from studies in humans, and if the observations are considered to be very reliable, the uncertainty factor may be as small as 1.0. However, the uncertainty factor is normally at least 10, and can be much higher if the data are limited. The effect of dividing the NOAEL or the LOAEL by an uncertainty factor is to ensure that the toxicity value is not higher than the threshold level for adverse effects. Thus, there is always a "margin of safety" built into an RfD or RfC, and doses equal to or less than these toxicity values are nearly certain to be without any risk of adverse effect. Exposures higher than the RfD or RfC may carry some risk, but because of the margin of safety, an exposure above the RfD or RfC does not mean that an effect will necessarily occur.

Toxicity Values

The following hierarchy was used to compile a list of cancer and non-cancer toxicity values for this report. To start, inhalation values established specifically by the State of Colorado (e.g., trichloroethlyene, 1,1-dichloroethylene) were given priority over all other sources of toxicity values. The second source used to identify relevant toxicity values was EPA's Air Toxics Website (http://www.epa.gov/ttn/atw/toxsource/summary.html). This website contains a relatively comprehensive listing of chronic inhalation-based toxicity values for a wide range of chemicals. These values are currently utilized in the National-Scale Air Toxics Assessment (NATA) and were selected so that results of this current assessment would be comparable to others conducted across the nation. If values were not available from Colorado or the Air Toxics Website, an effort was made to fill these data gaps using (in order of preference) IRIS (EPA's Integrated Risk Information System), PPTRVs (EPA's Provisional Peer-Reviewed Toxicity Values), and other secondary (e.g., California EPA) sources, as applicable.

For some substances that lack inhalation-specific toxicity values, values were derived from oral toxicity estimates. Although conversion of oral dose-response information to inhalation exposure is not optimal risk assessment practice, the alternative would be to omit these substances altogether from any quantitative inhalation risk estimates. For this screening-level risk assessment it was regarded that the use of route-extrapolated toxicity values were preferable to the assumption of "zero" risk for these analytes. However, it is acknowledged that there is considerable uncertainty surrounding this approach and that results should be evaluated accordingly. For example, one consideration is that in some cases oral exposures may underestimate the toxicity from inhalation exposures since absorption may not be as fast or complete by the oral route

Available toxicity values derived from these sources for the chemicals of potential concern at this site are presented in Table 5.7.

Cancer Effects

Only those substances that are known or suspected human carcinogens were considered in calculating incremental cancer risks (USEPA WOE (Weight of evidence) groups).

A cancer toxicity criterion is a health assessment value that can be matched with environmental exposure data to estimate health risk. For carcinogens, toxicity measurements are generally expressed as a risk per unit concentration (e.g., an inhalation UR (unit risk) in units of risk per mg/m3) or as a risk per daily intake (e.g., an oral carcinogenic potency slope factor, or CPSo, in units of risk per mg/kg–day).

Inhalation URs were used if available. The inhalation UR represents an estimate of the increased cancer risk from a lifetime (assumed to be 70 years) of continuous exposure to a concentration of one unit of exposure.

If no inhalation UR was available for a known or suspected human carcinogen, the oral slope factor was converted to an inhalation UR by the following equation:

$$IUR = (SFo * IR)/BW$$

where:
IUR = inhalation unit risk estimate (1/mg/m3)
SFo = oral carcinogenic potency slope factor, equal to risk per mg/kg-day
IR = standard inhalation rate for an adult, equal to 20 m3/day; and
BW = standard assumption for average adult body weight, equal to 70 kg.

Table 5-7 contains the inhalation unit risk values for the COPCs.

Non-cancer Effects

For non-cancer effects, toxicity benchmarks are generally expressed as a concentration in air (e.g., an inhalation reference concentration or RfC in units of mg/m3 air) or as a daily intake (e.g., an oral reference dose or RfD in units of mg/kg–day).

RfCs are generally used for evaluating the inhalation route of exposure and were given preference for this study. The reference concentration is an exposure that is believed to be without significant risk of adverse non-cancer health effects in a chronically exposed population, including sensitive individuals. If no RfC was available, RfDs were converted to RFCs using the following equation:

$$RfC = (RfD * BW)/IR$$

where:
RfC = Inhalation reference concentration (mg/m3);
RfD = Oral reference dose (mg/kg–day);
IR = Standard inhalation rate for an adult, equal to 20 m3/day; and
BW = Standard assumption for average adult body weight, 70 kg.

Table 5.7 contains the reference concentration values for the COPCs.

Table 5.7 - Cancer and Non-cancer Toxicity Values

Compound	CAS Number	Final Chronic Risk Factor (non-cancer) ug/m3	Source	Final Chronic Risk Factor (cancer) 1/(ug/m3)	Source
VOCs					
Acetylene	74-86-2				
Propylene	115-07-1				
Dichlorodifluoromethane	75-71-8	*200.0*	*Heast-9*		
Chloromethane	74-87-3	*90.0*	*ATW -Iris*		
Dichlorotetrafluoroethane	76-14-2				
Vinyl chloride	75-01-4	*100.0*	*ATW -Iris*	*8.80E-06*	*ATW-Iris*
1,3 - Butadiene	106-99-0	*2.0*	*ATW -Iris*	*3.00E-05*	*ATW-Iris*
Bromomethane	74-83-9	*5.0*	*ATW -Iris*		
Chloroethane	75-00-3	*10000.0*	*ATW -Iris*	*8.29E-07*	*route - IRIS-9*
Acetonitrile	75-05-8	*60.0*	*ATW -Iris*		
Trichlorofluoromethane	75-69-4	*700.0*	*Heast-9*		
Acrylonitrile	107-13-1	*2.0*	*ATW -Iris*	*6.80E-05*	*ATW-Iris*
1,1 - Dichloroethene	75-35-4	*5.0*	*CDPHE*		
Methylene chloride	75-09-2	*1000.0*	*ATW-ATSDR*	*4.57E-07*	*IRIS-9*
Trichlorotrifluoroethane	76-13-1				
trans - 1,2 - Dichloroethylene	156-60-5	*70.0*	*route ext - IRIS-9*		
1,1 - Dichloroethane	74-34-3	*490.0*	*NCEA -9*		
Methyl tert-butyl ether	1634-04-4	*3000.0*	*ATW-Iris*	*2.60E-07*	*ATW-Cal*
Methyl ethyl ketone	78-93-3	*5000.0*	*ATW-Iris*		
Chloroprene	126-99-8	*7.0*	*ATW-Heast*		
cis - 1,2 - Dichloroethylene	156-59-2	*35.0*	*route ext - PPRTV-9*		
Bromochloromethane	74-97-5				
Chloroform	67-66-3	*98.0*	*ATW-ATSDR*	*2.31E-05*	*IRIS-9*
Ethyl tert-butyl ether	637-92-3				
1,2 - Dichloroethane	107-06-2	*2400.0*	*ATW-ATSDR*	*2.60E-05*	*ATW-Iris*
1,1,1 - Trichloroethane	71-55-6	*1000.0*	*ATW-Cal*		
Benzene	71-43-2	*30.0*	*ATW-Iris*	*7.80E-06*	*ATW-Iris*
Carbon tetrachloride	56-23-5	*190.0*	*ATW-D-ATSDR*	*1.50E-05*	*ATW-Iris*
tert-Amyl methyl ether	994-05-8				
1,2 - Dichloropropane	78-87-5	*4.0*	*ATW-Iris*	*1.90E-05*	*ATW-ConvOral*
Ethyl acrylate	140-88-5			*1.40E-05*	*ATW-ConvOral*
Bromodichloromethane	75-27-4	*70.0*	*route ext - IRIS-9*	*1.77E-05*	*route ext - IRIS-9*
Trichloroethylene	79-01-6	*600.0*	*ATW-Cal*	*1.14E-04*	*CDPHE*
Methyl methacrylate	80-62-6	*700.0*	*ATW-Iris*		
cis - 1,3 - Dichloropropene	10061-01-5				
Methyl isobutyl ketone	108-10-1	*3000.0*	*ATW-Iris*		
trans - 1,3 - Dichloropropene	10061-02-6				
1,1,2 - Trichloroethane	79-00-5	*400.0*	*ATW-P-Cal*	*1.60E-05*	*ATW-Iris*
Toluene	108-88-3	*400.0*	*ATW-Iris*		
Dibromochloromethane	124-48-1	*70.0*	*route ext - IRIS-9*	*2.40E-05*	*route ext - IRIS-9*
1,2 - Dibromoethane	106-93-4	*9.0*	*ATW-Iris*	*6.00E-04*	*ATW-Iris*
n - Octane	111-65-9				

Compound	CAS Number	Final Chronic Risk Factor (non-cancer) ug/m3	Source	Final Chronic Risk Factor (cancer) 1/(ug/m3)	Source
Tetrachloroethylene	127-18-4	*270.0*	*ATW-ATSDR*	*5.90E-06*	*ATW-Cal*
Chlorobenzene	108-90-7	*1000.0*	*ATW-Cal*		
Ethylbenzene	100-41-4	*1000.0*	*ATW-Iris*		
m,p - Xylene	108-38-3 / 106-42-3	*102.0*	*IRIS-9 All Xylenes*		
Bromoform	75-25-2	*70.0*	*route ext - IRIS-9*	*1.10E-06*	*ATW-Iris*
Styrene	100-42-5	*1000.0*	*ATW-Iris*		
1,1,2,2 - Tetrachloroethane	79-34-5	*210.0*	*route ext - PPRTV-9*	*5.80E-05*	*ATW-Iris*
o - Xylene	95-47-6	*102.0*	*IRIS-9 All Xylenes*		
1,3,5 - Trimethylbenzene	108-67-8	*6.0*	*PPRTV-9*		
1,2,4 - Trimethylbenzene	95-63-6	*6.0*	*PPRTV-10*		
m - Dichlorobenzene	541-73-1	*105.0*	*route ext - NCEA-9*		
Chloromethylbenzene	100-44-7			*4.90E-05*	*ATW-Cal*
p - Dichlorobenzene	106-46-7	*800.0*	*ATW-Iris*	*1.10E-05*	*ATW-Cal*
o - Dichlorobenzene	95-50-1	*200.0*	*Heast-9*		
1,2,4 - Trichlorobenzene	120-82-1	*200.0*	*ATW-Heast*		
Hexachloro - 1,3 - butadiene	87-68-3	*90.0*	*ATW-P-Cal*	*2.20E-05*	*ATW-Iris*
Carbonyls					
Formaldehyde	50-00-0	*9.8*	*ATW-ATSDR*	*5.50E-09*	*ATW-OAQPS*
Acetaldehyde	75-07-0	*9.0*	*ATW-IRIS*	*2.20E-06*	*ATW-Iris*
Acetone	67-64-1	*3150.0*	*route ext - Iris-9*		
Propionaldehyde	123-38-6				
Crotonaldehyde	123-73-9			*5.43E-04*	*route ext-HEAST-9*
Butyr / Isobutyraldehyde	123-72-8 / 78-84-2				
Benzaldehyde	100-52-7	*350.0*	*route ext - Iris-9*		
Isovaleraldehyde	590-86-3				
Valeraldehyde	110-62-3				
Tolualdehydes (o-, m-, p-)	1334-78-7				
Hexaldehyde	66-25-1				
2,5-Dimethylbenzaldehyde	5779-94-2				
Inorganics					
Antimony	7440-36-0				
Arsenic	7440-38-2	*0.0*	*ATW-Cal*	*4.30E-03*	*ATW-Iris*
Beryllium	7440-41-7	*0.0*	*ATW-Iris*	*2.40E-03*	*ATW-Iris*
Cadmium	7440-43-9	*0.0*	*ATW-Cal*	*1.80E-03*	*ATW-Iris*
Chromium (total)*	7440-47-3	*0.1*	*ATW-Iris*		
Cobalt	7440-84-4	*0.1*	*ATW-ATSDR*	*2.80E-03*	*PPRTV-9*
Lead	7439-92-1	*1.5*	*ATW-OAQPS*		
Manganese	7439-96-5	*0.1*	*ATW-Iris*		
Mercury	7439-97-6	*0.3*	*ATW-Iris*		
Nickel	7440-02-0	*0.1*	*ATW-D-ATSDR*		
Selenium	7782-49-2	*20.0*	*ATW-Cal*		

IRIS = EPA's Integrated Risk Information System
CDPHE = Colorado Department of Public Health and Environment
HEAST = Health Effects Assessment Summary Tables
CAL = State of California
PPRTV = EPA's Provisional Peer-Reviewed Toxicity Values
9, Reg 9 = EPA Region 9
10, Reg 10 = EPA Region 10
Route Ext = Route Extrapolation
ConvOral = Converted from Oral Route

Risk Characterization

The risk characterization integrates the information from the exposure assessment and the toxicity assessment to provide an estimate of the magnitude of potential risks, and the strength of the conclusions based on the uncertainty in the information used to generate these estimates. For this risk assessment the risk characterization means combining the exposure concentrations with the toxicity data to provide a quantitative estimate of the potential health impacts. Both cancer and non-cancer health effects are evaluated in this risk characterization. A brief summary of the potential sources and toxic effects of major contributors to the total cancer and non-cancer risks is provided in Attachment 1.

Cancer Risk Estimates

In this assessment, risk estimates for COPCs with a cancer endpoint were expressed in terms of the probability of contracting cancer from a lifetime of continuous exposure (70 year lifespan) to a constant air concentration of the COPC. The lifetime cancer risk for each COPC at each monitoring location was derived by multiplying the 95th percent upper confidence limit on the mean of the monitored ambient air concentrations by the respective IUR value, as shown in the following equation. The resulting products are added to estimate the total risk for the site. This summation is based upon the principle that the addition of each risk produces a combined total risk estimate.

$$Riskx = EPC * IURx$$

Where:
Riskx = the risk of the Xth COPC at a monitor:
EC = the exposure point concentration of the substance (i.e., most stringent of the 95% UCL or maximum air concentration); and
IURx = the inhalation unit risk of the substance.

Estimates of cancer risk were expressed as a probability, represented in scientific notation as a negative exponent of 10. For example, an additional lifetime risk of contracting cancer of 1 chance in 1,000,000 (or one additional person in 1,000,000) is written as lx10-6 or lE–06.

The level of cancer risk that is of concern is a matter of individual, community and regulatory judgment. However, the USEPA typically considers risks below 1E-06 to be so small as to be negligible (USEPA 1991b).

Non-Cancer Risk Estimates

In contrast to cancer risks, non-cancer hazards are not expressed as a probability of an individual suffering an adverse effect. Instead, non-cancer hazard to individuals is expressed in terms of the hazard quotient (HQ), defined as the ratio between an individual's estimated exposure and the Reference Concentration (RfC). For a given air toxic, exposures below the reference level (HQ less than1) are not likely to be associated with adverse health effects. With exposures increasingly greater than the reference concentration, the potential for adverse effects increases. HQs were calculated as follows:

$$HQ_x = EPC_x/RfC_x$$

Where:
HQx = the hazard quotient of the Xth COPC at a monitor:
EPC_x = the exposure point concentration of the substance (i.e., most stringent of the 95% UCL or maximum air concentration); and
RfCx = the reference concentration of the substance.

When used in the assessment of non-cancer risks, the hazard quotient is commonly reported to one significant figure (EPA 1989a). For example, a hazard quotient of 0.13 is rounded to 0.1, and a hazard quotient of 1.6 is rounded to 2.

Hazard quotient calculations for an individual chemical estimate the potential for adverse effects if a receptor is exposed to only that chemical. If there are multiple chemicals to which the receptor may be exposed then the consequences of the multiple exposures can be quantified, within some limitations. Because different pollutants may cause similar adverse health effects, it is often appropriate to combine hazard quotients associated with different substances. For non-carcinogenic chemicals, the hazard quotients for each exposure pathway can be summed to develop a hazard index (HI) for that exposure pathway.

For screening purposes, it is acceptable to sum all HQ values in order to derive an HI value. If the resulting HI is less than one, no further evaluation is necessary and it can be concluded that no unacceptable risks are present. If the HI is greater than unity as a consequence of summing several hazard quotients of similar value, it would be appropriate to segregate the compounds by effect and by mechanism of action and to derive separate hazard indices for each group. Table 5.8 presents a summary of critical effects associated with each of the chemicals carried through to the risk characterization.

Table 5.8 - Summary Of Critical Effects

			Critical Effect								
Compound	**CAS Number**	**Basis**	**Neurologic**	**Respiratory**	**Immunological**	**Systemic**	**Circulatory**	**Dermal/Ocular**	**Developmental**	**Reproductive**	**Mortality**
VOCs											
1,3 - Butadiene	106-99-0	*Ovarian Atrophy*								x	
Acetonitrile	75-05-8	*Mortality*									x
Acrylonitrile	107-13-1	*Degeneration and inflammation of nasal respiratory epithelium; hyperplasia of mucous secreting cells*		x							
Methylene chloride	75-09-2	*Hepatic*				x					
Methyl tert-butyl ether	1634-04-4	*Increased absolute and relative liver and kidney weights and increased severity of spontaneous renal lesions (females), increased prostration (females), and swollen periocular tissue (males and females)*				x					
Chloroform	67-66-3	*Hepatic*				x					
Benzene	71-43-2	*Decreased lymphocyte count*			x						
Carbon tetrachloride	56-23-5	*Hepatic*				x					
Trichloroethylene	79-01-6	*Nervous System, eyes*	x					x			
Toluene	108-88-3	*Neurological effects (other effect: Degeneration of nasal epithelium)*	x	x							
Tetrachloroethylene	127-18-4	*Neurologic*	x								
m,p - Xylene	108-38-3 / 106-42-3	*Impaired motor coordination (decreased rotarod performance)*	x								

Compound	CAS Number	Basis	Critical Effect								
			Neurologic	Respiratory	Immunological	Systemic	Circulatory	Dermal/Ocular	Developmental	Reproductive	Mortality
o - Xylene	95-47-6	*Impaired motor coordination (decreased rotarod performance)*	x								
1,3,5 - Trimethylbenzene	108-67-8	*Vertigo, headaches, drowsiness, anemia, altered bloodclotting, chronic asthma-like bronchitis*	x	x	x						
1,2,4 - Trimethylbenzene	95-63-6	*Vertigo, headaches, drowsiness, anemia, altered bloodclotting, chronic asthma-like bronchitis*	x	x	x						
p - Dichlorobenzene	106-46-7	*Increased liver weights in P1 males*				x					
Carbonyls											
Formaldehyde	50-00-0	*Respiratory*		x							
Acetaldehyde	75-07-0	*Degeneration of olfactory epithelium*		x							
Inorganics											
Arsenic	7440-38-2	*Development; Cardiovascular System; Nervous System*	x				x		x		
Beryllium	7440-41-7	*Beryllium sensitization and progression to CBD*		x	x						
Cadmium	7440-43-9	*Kidney; Respiratory System*		x		x					
Chromium (total)*	7440-47-3	*Lactate dehydrogenase in bronchioalveolar lavage fluid*		x	x						
Cobalt	7440-84-4	*Respiratory*		x							
Manganese	7439-96-5	*Impairment of neurobehavioral function (other effect: Impairment of neurobehavioral function.)*	x								

Table 5.8, completed.

Summary of Cancer and Non-Cancer Risk Estimates

Tables 5.9 to 5.12 summarize the cancer and non-cancer risk estimates at the monitoring studies evaluated in this investigation. A description of the findings for each station is presented below.

CAMP Station

Twenty-one chemicals from the CAMP monitoring station were carried through for evaluation in the risk characterization. Of these chemicals, fifteen were evaluated for cancer risks and twenty were evaluated for non-cancer risks.

As shown in Table 5.9, cancer risks for the individual chemicals ranged from 9E-08 to 8E-05, with a combined total risk of 2E-04. Although each individual chemical was determined to have a cancer risk below the upper end of EPA's generally acceptable range of 1E-06 to 1E-04, the cumulative total cancer risk somewhat exceeds this level. The largest individual contributors to the total risk estimate are crotonaldehyde (8E-05 or 38% of the total risk), trichloroethylene (6E-05 or 29% of the total risk), benzene (3E-05 or 13% of the total risk), and arsenic (2E-05 or 10% of the total risk).

Individual hazard quotients for non-cancer risks ranged from 0.001 to 1, with a combined hazard index of 4 (Table 5.9). None of the individual hazard quotients exceeded a value of one. Because the combined hazard index was greater than one, it was necessary to group hazard quotients according to critical effect and recalculate target specific hazard indices.

Table 5.12 shows the recalculated hazard indices for all monitoring stations. As seen, after summarizing hazard indices by critical effect, only two categories of effect (respiratory and neurologic) at the CAMP monitoring station had hazard indices exceeding a value of one.

A hazard index of 2 was calculated for respiratory effects at the CAMP monitoring station. Chemicals which contribute to this effect are toluene (HQ=0.03), 1,3,5-trimethylbenzene (HQ=0.2), 1,2,4-trimethylbenzene (HQ=0.5), formaldehyde (HQ=0.9), acetaldehyde (HQ=0.5), beryllium (HQ=0.002), cadmium (HQ=0.02) and cobalt (HQ=0.01). As seen, the largest contributors to respiratory risk at this monitoring station are formaldehyde, acetaldehyde and 1,2,4-trimethylbenzene. Although each individual chemical does not exceed a hazard quotient of 1, the cumulative hazard index of 2 indicates that there may be an increased potential for respiratory effects based on continuous exposure at this location.

A hazard index of 2 was calculated for neurologic effects at the CAMP monitoring station. Chemicals which contribute to this effect are trichloroethylene (HQ=0.0008), toluene (HQ=0.03), tetrachloroethylene (HQ=0.003), m,p-xylene (HQ=0.05), 1,3,5-trimethylbenzene (HQ=0.2), 1,2,4-trimethylbenzene (HQ=0.5), arsenic (HQ=0.2) and manganese (HQ=1). As seen, the largest contributors to neurologic risk at this monitoring station are manganese and 1,2,4-trimethylbenzene. Although each individual chemical does not exceed a hazard quotient of 1, the cumulative hazard index of 2 indicates increased potential for neurologic effects based on continuous exposure at this location.

Table 5.9 - Risk Estimates - CAMP Monitoring Station

	Hazard Index	Cancer Risk
Total Risk =	**4**	**2E-04**

Compound	CAS Number	EPC 95UCL ug/m3	RfC ug/m3	Air Unit Risk 1/(ug/m3)	Non Cancer Risk	Cancer Risk	% Contrib. Cancer
Volatiles (57 samples)							
1,3 - Butadiene	106-99-0	0.364	2	3.00E-05	0.2	1E-05	5.5%
Acetonitrile	75-05-8	4.887	60	--	0.1	--	--
Methylene chloride	75-09-2	2.430	1000	4.57E-07	0.002	1E-06	0.6%
Chloroform	67-66-3	0.221	98	2.31E-05	0.002	5E-06	2.6%
Benzene	71-43-2	3.367	30	7.80E-06	0.1	3E-05	13.1%
Carbon tetrachloride	56-23-5	0.529	190	1.50E-05	0.003	8E-06	4.0%
Trichloroethylene	79-01-6	0.506	600	1.14E-04	0.001	6E-05	28.9%
Toluene	108-88-3	10.929	400	--	0.03	--	--
Tetrachloroethylene	127-18-4	0.932	270	5.90E-06	0.003	5E-06	2.7%
m,p - Xylene	108-38-3 / 106-42-3	4.789	102	--	0.05	--	--
1,3,5 - Trimethylbenzene	108-67-8	1.152	6	--	0.2	--	--
1,2,4 - Trimethylbenzene	95-63-6	3.223	6	--	0.5	--	--
p - Dichlorobenzene	106-46-7	0.451	800	1.10E-05	0.001	5E-06	2.5%
Inorganics (59 samples)							
Arsenic	7440-38-2	0.004904	0	4.30E-03	0.2	2E-05	10.5%
Beryllium	7440-41-7	3.77E-05	0.02	0.0024	0.002	9E-08	0.0%
Cadmium	7440-43-9	0.000393	0.02	1.80E-03	0.02	7E-07	0.4%
Cobalt	7440-84-4	0.001093	0.10	2.80E-03	0.01	3E-06	1.5%
Manganese	7439-96-5	0.057342	0.05	--	1	--	--
Carbonyls (56 samples)							
Formaldehyde	50-00-0	9.00	10	5.50E-09	0.9	5E-08	0.0%
Acetaldehyde	75-07-0	4.50	9	2.20E-06	0.5	1E-05	4.9%
Crotonaldehyde	123-73-9	0.14	--	5.43E-04	--	8E-05	37.6%

Welby Station

Nineteen chemicals from the Welby monitoring station were carried through for evaluation in the risk characterization. Of these chemicals, fourteen were evaluated for cancer risks and eighteen were evaluated for non-cancer risks.

As shown in Table 5.10, cancer risks for the individual chemicals ranged from 9E-08 to 5E-05, with a combined total risk of 1E-04. Each individual chemical was determined to have a cancer risk below the upper end of EPA's generally acceptable range of 1E-06 to 1E-04, and the cumulative total cancer risk was not found to exceed the upper end of this range.

Individual hazard quotients for non-cancer risks ranged from 0.001 to 1, with a combined hazard index of 3 (Table 5.10). None of the individual hazard quotients exceeded a value of one. Because the combined hazard index was greater than one, it was necessary to group hazard quotients according to critical effect and recalculate target specific hazard indices.

Table 5.12 shows the recalculated hazard indices for all monitoring stations. As seen, after summarizing hazard indices by critical effect, only the category of neurologic effects at the Welby monitoring station had a hazard index exceeding a value of one.

A hazard index of 2 was calculated for neurologic effects at the Welby monitoring station. Chemicals which contribute to this effect are toluene (HQ=0.03), tetrachloroethylene (HQ=0.002), m,p-xylene (HQ=0.05), 1,3,5-trimethylbenzene (HQ=0.07), 1,2,4-trimethylbenzene (HQ=0.2), arsenic (HQ=0.1) and manganese (HQ=1). As for the CAMP station, the largest contributors to neurologic risk at this monitoring station are manganese and 1,2,4-trimethylbenzene. Although each individual chemical does not exceed a hazard quotient of 1, the cumulative hazard index of 2 indicates increased potential for neurologic effects based on continuous exposure at this location.

Table 5.10 - Risk Estimates - Welby Monitoring Station

	Hazard Index	Cancer Risk
Total Risk =	**3**	**1E-04**

Compound	CAS Number	EPC 95UCL ug/m3	RfC ug/m3	Air Unit Risk 1/(ug/m3)	Non Cancer Risk	Cancer Risk	% Contrib. Cancer
Volatiles (57 samples)							
1,3 - Butadiene	106-99-0	0.247	2	3.00E-05	0.1	7E-06	5.7%
Methylene chloride	75-09-2	0.881	1000	4.57E-07	0.001	4E-07	0.3%
Methyl tert-butyl ether	1634-04-4	2.505	3000	2.60E-07	0.001	7E-07	0.5%
Chloroform	67-66-3	0.203	98	2.31E-05	0.002	5E-06	3.6%
Benzene	71-43-2	2.717	30	7.80E-06	0.09	2E-05	16.3%
Carbon tetrachloride	56-23-5	0.500	190	1.50E-05	0.003	7E-06	5.8%
Toluene	108-88-3	10.366	400	--	0.03	--	--
Tetrachloroethylene	127-18-4	0.428	270	5.90E-06	0.002	3E-06	1.9%
m,p - Xylene	108-38-3 / 106-42-3	5.188	102	--	0.05	--	--
1,3,5 - Trimethylbenzene	108-67-8	0.440	6	--	0.07	--	--
1,2,4 - Trimethylbenzene	95-63-6	1.346	6	--	0.2	--	--
Inorganics (59 samples)							
Arsenic	7440-38-2	0.0040391	0.03	4.30E-03	0.1	2E-05	13.4%
Beryllium	7440-41-7	3.812E-05	0.02	2.40E-03	0.002	9E-08	0.1%
Cadmium	7440-43-9	0.0003837	0.02	0.0018	0.02	7E-07	0.5%
Cobalt	7440-84-4	0.0012901	0.10	2.80E-03	0.01	4E-06	2.8%
Manganese	7439-96-5	0.0581002	0.05	--	1	--	--
Carbonyls (56 samples)							
Formaldehyde	50-00-0	3.88	10	5.50E-09	0.4	2E-08	0.0%
Acetaldehyde	75-07-0	3.14	9	2.20E-06	0.3	7E-06	5.3%
Crotonaldehyde	123-73-9	0.10	--	5.43E-04	--	5E-05	40.6%

Swansea Station

Eighteen chemicals from the Swansea monitoring station were carried through for evaluation in the risk characterization. Of these chemicals, thirteen were evaluated for cancer risks and seventeen were evaluated for non-cancer risks.

As shown in Table 5.11, cancer risks for the individual chemicals ranged from 3E-08 to 4E-05, with a combined total risk of 1E-04. Although each individual chemical was determined to have a cancer risk below the upper end of EPA's generally acceptable range of 1E-06 to 1E-04, the cumulative total cancer risk somewhat exceeds this level. The largest individual contributors to the total risk estimate are crotonaldehyde (4E-05 or 29% of the total risk), benzene (3E-05 or 20% of the total risk) and arsenic (2E-05 or 19% of the total risk).

Individual hazard quotients for non-cancer risks ranged from 0.001 to 1, with a combined hazard index of 4 (Table 5.11). None of the individual hazard quotients exceeded a value of one. Because the combined hazard index was greater than one, it was necessary to group hazard quotients according to critical effect and recalculate target specific hazard indices.

Table 5.12 shows the recalculated hazard indices for all monitoring stations. As seen, after summarizing hazard indices by critical effect, only two categories of effect (respiratory and neurologic) at the Swansea monitoring station had hazard indices exceeding a value of one.

A hazard index of 2 was calculated for respiratory effects at the Swansea monitoring station. Chemicals which contribute to this effect are toluene (HQ=0.04), 1,3,5-trimethylbenzene (HQ=0.1), 1,2,4-trimethylbenzene (HQ=0.3), formaldehyde (HQ=0.6), acetaldehyde (HQ=0.4), beryllium (HQ=0.004), cadmium (HQ=0.05) and cobalt (HQ=0.01). As seen, the largest contributors to respiratory risk at this monitoring station are formaldehyde, acetaldehyde and 1,2,4-trimethylbenzene. Although each individual chemical does not exceed a hazard quotient of 1, the cumulative hazard index of 2 indicates increased potential for respiratory effects based on continuous exposure at this location.

A hazard index of 2 was calculated for neurologic effects at the Swansea monitoring station. Chemicals which contribute to this effect are toluene (HQ=0.03), tetrachloroethylene (HQ=0.004), m,p-xylene (HQ=0.09), 1,3,5-trimethylbenzene (HQ=0.1), 1,2,4-trimethylbenzene (HQ=0.3), arsenic (HQ=0.2) and manganese (HQ=1). As for the CAMP and Welby stations, the largest contributors to neurologic risk at this monitoring station are manganese and 1,2,4-trimethylbenzene. Although each individual chemical does not exceed a hazard quotient of 1, the cumulative hazard index of 2 indicates increased potential for neurologic effects based on continuous exposure at this location.

Table 5.11 - Risk Estimates - Swansea Monitoring Station

	Hazard Index	Cancer Risk
Total Risk =	**4**	**1E-04**

Compound	CAS Number	EPC 95UCL ug/m3	RfC ug/m3	Air Unit Risk 1/(ug/m3)	Non Cancer Risk	Cancer Risk	% Contrib. Cancer
Volatiles (57 samples)							
1,3 - Butadiene	106-99-0	0.37	2	3.00E-05	0.2	1E-05	8.4%
Methylene chloride	75-09-2	1.32	1000	4.57E-07	0.001	6E-07	0.5%
Chloroform	67-66-3	0.24	98	2.31E-05	0.002	6E-06	4.2%
Benzene	71-43-2	3.38	30	7.80E-06	0.1	3E-05	20.3%
Carbon tetrachloride	56-23-5	0.55	190	1.50E-05	0.003	8E-06	6.3%
Toluene	108-88-3	14.84	400	--	0.04	--	--
Tetrachloroethylene	127-18-4	1.10	270	5.90E-06	0.004	6E-06	5.0%
m,p - Xylene	108-38-3 / 106-42-3	9.18	102	--	0.09	--	--
1,3,5 - Trimethylbenzene	108-67-8	0.63	6	--	0.1	--	--
1,2,4 - Trimethylbenzene	95-63-6	1.81	6	--	0.3	--	--
Inorganics (59 samples)							
Arsenic	7440-38-2	0.005702	0.03	4.30E-03	0.2	2E-05	18.9%
Beryllium	7440-41-7	8.87E-05	0.02	2.40E-03	0.004	2E-07	0.2%
Cadmium	7440-43-9	0.000918	0.02	1.80E-03	0.05	2E-06	1.3%
Cobalt	7440-84-4	0.001029	0.1	0.0028	0.01	3E-06	2.2%
Manganese	7439-96-5	0.070402	0.05	0.00E+00	1	--	--
Carbonyls (56 samples)							
Formaldehyde	50-00-0	5.86	10	5.50E-09	0.6	3E-08	0.0%
Acetaldehyde	75-07-0	3.67	9	2.20E-06	0.4	8E-06	6.2%
Crotonaldehyde	123-73-9	0.07	--	5.43E-04	--	4E-05	28.6%

Table 5.12 - Hazard Indices for All Sites

Critical Effect	CAMP	Welby	Swansea
Neurologic	**2**	**2**	**2**
Respiratory	**2**	**1**	**2**
Immunological	0.8	0.4	0.5
Systemic	0.03	0.03	0.05
Circulatory	0.16	0.1	0.2
Dermal/Ocular	0.0008	--	--
Developmental	0.2	0.1	0.2
Reproductive	0.2	0.1	0.2
Mortality	0.08	--	--

NOTE: bolded cells have HI values exceeding 1

Uncertainties

Quantitative evaluation of the risks to humans from environmental contamination is frequently limited by uncertainty (lack of knowledge) regarding a number of important exposure and toxicity factors. This lack of knowledge is usually circumvented by making estimates based on whatever limited data that are available, or by making assumptions based on professional judgment when no reliable data are available. Because of these assumptions and estimates, the results of risk calculations are themselves uncertain, and it is important for risk managers and the public to keep this in mind when interpreting the results of a risk assessment. The following sections review the main sources of uncertainty in the risk calculations summarized in this report.

Uncertainties in Monitoring

One uncertainty in this study was the use of monitoring data to estimate the potential human health exposures and risks. The uncertainty stems from the inability to realistically monitor continuously at all places of interest. Thus a decision is made to monitor a portion of the time and in specific locations and apply the results to a broader situation.

The monitoring data at each station reflects a single year or less of chemical concentrations in air. It is uncertain how well this dataset reflects the lifetime exposure assumed in this risk assessment as changes in meteorology and chemical emissions could lead to lower or higher concentrations in air from year-to-year. To reduce this uncertainty would require monitoring over several years, or modeling based on changes in meteorology and chemical emissions.

Monitoring locations may or may not be representative of air concentrations to which an individual could be exposed 24 hours a day for a lifetime. As discussed previously, several of these monitoring locations were placed in areas with mixed industrial use or heavier traffic patterns. Potential health impacts associated with contaminant concentrations at these locations could over estimate the true risk since they may not reflect the actual long-term residential exposure concentration. Additionally, they could underestimate true risk to people living near sources of high concentrations of contaminant emissions.

A large number of chemicals were selected for monitoring, but still, limiting the number of chemicals analyzed in the monitoring program can result in an underestimation of risk, which could in some cases be reduced by monitoring for a larger group of chemicals. Many of the chemicals selected for monitoring were based on an assessment of the likely or known chemicals in air at the Denver monitoring locations.

One potentially significant limitation of the current National Air Toxics Monitoring Network is the lack of monitoring for the chemical acrolein. Modeling estimates suggest that this chemical may be present in amounts that could present a significant risk to the public. However, there is currently no EPA-approved method for monitoring for acrolein. Thus, a chemical that is one of the largest contributors to risk from computer modeling simulations cannot currently be measured to determine actual levels in the air. Therefore, acrolein risks may be underestimated. EPA is in the process of developing a method for measuring acrolein.

Uncertainties in COPC Selection

The frequency at which positively identified chemicals in the dataset were detected at a monitor was calculated and used as a means to focus the risk assessment on the most frequently detected chemicals. Any chemical that was not detected in at least 5% of the samples reported for a location was removed from further analysis in the risk assessment. Application of this 5% rule for each monitor location led to the selection of the chemicals of potential concern (COPCs) for evaluation in the risk assessment. Eliminating chemicals that were infrequently detected leads to an underestimate of the health impacts. Therefore, chemicals detected at greater than 0% and less than 5% frequency are further discussed here to evaluate the potential for underestimation of risk.

At the CAMP monitoring location, five chemicals were found to have the detection frequency of greater than 0% and less than 5%: Acrylonitrile (1.8%); 1,1-dichloroethene (1.8%); 1,2-Dichloroethane (1.8%); Methyl methacrylate (1.8%); and Methyl isobutyl ketone (3.5%). Of these chemicals, acrylonitrile and 1,2-dichloroethane exceed the cancer and/or noncancer acceptable risk levels. For example, the estimated cancer risk for acrylonitrile is 400 in a million (4E-04) and the noncancer HQ is 3.0, based on the maximum concentration of 5.6 ug/m3. These risk estimates will be 10-fold lower if based on the average concentration of 0.6 ug/m3. The estimated cancer risk for 1,2-dichloroethane is 6 in a million (6E-06), based on the maximum as well as average concentration of 0.2 ug/m3. At the Welby monitoring location, bromomethane (3.8%) and p-dichlorobenzene (3.8%) were found to have the detection frequency of greater than 0% and less than 5%. The noncancer HQ for bromomethane is 1.1, based on the maximum concentration of 5.7 ug/m3. This noncancer hazard is 10-fold lower at the average concentration of 0.4 ug/m3. The estimated cancer risk for p-dichlorobenzene is 5 in a million (5E-06), based on the maximum as well as average concentration of 0.45 ug/m3. At the Swansea monitoring location, only acrylonitrile (3.4%) was found to have the detection frequency of greater than 0% and less than 5%. The estimated cancer risk for acrylonitrile is 140 in a million (1.4E-04) and the noncancer HQ is 1.0, based on the maximum concentration of 2.1 ug/m3. The estimated cancer risk for acrylonitrile is 40 in a million based on the average concentration of 0.6 ug/m3.

Additionally, chemicals were eliminated from further evaluation if the maximum detected concentration of a chemical was less than its respective toxicity screening values. This limited the data set for evaluation to those chemicals that were assumed to contribute most significantly to the calculated risks. Although risk drivers are clearly carried through using this approach, elimination of these lesser significant chemicals leads to an underestimate of the health impacts.

Uncertainties in Concentration Estimates

Evaluation of human health risk at any particular location requires accurate information on the average concentration level of a COPC at that location. However, concentration values may vary from sample to sample, so the USEPA recommends that the 95% upper confidence limit (95% UCL) of the mean be used in evaluation of exposure and risk. This approach typically ensures that the risk estimates are likely higher than if means were used.

In deriving the exposure point concentration (i.e., 95% UCL) for this report, all non-detects were assigned a value of ½ the detection limit. When a chemical is reported as non-detectable, it does not mean the chemical is not present; rather, it may be present at a concentration lower than the instrument can detect. The true value of a non-detected chemical may range from not being present (i.e., zero concentration) to being present at a concentration just under the detection limit. The use of ½ the detection limit may over- or under-estimate the true concentration, but is considered to generally be a conservative approach.

This risk assessment has not quantitatively evaluated cancer risk for hexavalent chromium (Cr6) because no data are available for Cr6 concentration in air samples. Only total chromium was analyzed. Therefore, risks due to Cr6 can only be estimated as total chromium based on an assumed 1:6 ratio of Cr6: Cr3 (trivalent chromium) as per the EPA IRIS file on the toxicity values for Cr6. Due to the uncertainties associated with the actual concentration of Cr6 in air, risk estimates due to Cr 6 are discussed semi-quantitatively. Based on an assumed 1:6 ratio of Cr6: Cr3, for example, cancer risk estimates are 100 in a million, 70 in a million, and 100 in a million for the CAMP, Welby, and Swansea monitoring stations, respectively. Thus, the total cancer risks are likely to be underestimated in this risk assessment, due to the lack of information regarding hexavalent chromium concentrations in air. These risk estimates indicate the need for monitoring of Cr6 and this method is currently being developed by the USEPA.

Uncertainties in Human Exposure

There is usually wide variation between different individuals with respect to the level of contact they may have to chemicals in the environment. This introduces uncertainty as to the most appropriate values to use for exposure parameters.

Once released to the atmosphere, some air toxics can transfer to other media such as water, soil and vegetation. Air toxics with low vapor pressure are typically present in atmospheric particles which may deposit to the terrestrial environment by dry deposition processes. Besides being deposited, some of these can also accumulate, to a significant degree, in soil and vegetation, as well as bioaccumulate in living organisms and biomagnify in food chains. As a result, exposure to these air toxics can occur through multiple exposure pathways, including inhalation (breathing), dermal contact (touching), and ingestion (eating and drinking). The majority of risk for this evaluation is assumed to be a consequence of inhalation exposure, with other pathways contributing to a much lesser extent. However, by focusing on inhalation only, total risks are underestimated.

Another source of uncertainty is the risk for children, because children generally are expected to have some exposures that differ (higher or lower) from those of adults due to differences in size, physiology, and behavior. For example, children exposed to the same concentration of a chemical in air as adults may receive a higher dose because of greater lung surface area to body weight ratios, and higher ventilation rate per kilogram of body weight. EPA has recently concluded that cancer risks of mutagenic carcinogens generally are higher from early-life exposures than from similar exposure durations later in life. It is, however, important to note that when exposures are fairly uniform over a lifetime exposure of 70 years, the effect of child adjustments on the estimated lifetime cancer risk is relatively small. These adjustments are more important when estimating the cancer risks from less than 70 years of exposure duration. In addition, children are more at risk because of the availability of a longer latency period for the development of cancer.

Uncertainty in Risk Estimates due to Multiple Contaminants

Both carcinogenic and noncarcinogenic risks for multiple contaminants are assumed to be additive, in accordance with the EPA guidance for health risk assessment of chemical mixtures. This assumption, however, is associated with several limitations and, therefore, there is potential for under- or over-estimation of risk. For example, the assumption of additivity of risk does not account for synergistic or antagonistic chemical interactions.

Uncertainties in Toxicity Values

One of the most important sources of uncertainty in a risk assessment is in the RfC values used to evaluate non-cancer risk and in the inhalation unit risk values used to quantify cancer risk. In many cases, these values are derived from a limited toxicity database, and this can result in substantial uncertainty, both quantitatively and qualitatively. In order to account for these and other uncertainties associated with the evaluation of toxicity data, both RfCs and IURs are derived by the USEPA in a way that is intentionally conservative; that is, risk estimates based on these RfCs and IURs are more likely to overestimate risk.

For some substances that lack inhalation-specific toxicity values, values were derived from oral toxicity estimates. Although conversion of oral dose-response information to inhalation exposure is not optimal risk

assessment practice, the alternative would be to omit these substances altogether from any quantitative inhalation risk estimates. For this screening-level risk assessment, the use of route-extrapolated toxicity values was preferred to the assumption of "zero" risk for these analytes. However, it is acknowledged that there is considerable uncertainty surrounding this approach and that results should be evaluated accordingly. For example, crotonaldehyde was evaluated using a cancer toxicity factor derived from oral exposure data. For the cancer risk estimates, crotonaldehyde contributes to a majority of the total risk at each monitoring station, with risk estimates for this chemical ranging from 4E-05 to 8E-05. Additionally, the uncertainty (or potential underestimation of risk) can be introduced due to the lack of toxicity values for some chemicals. For example, no non-cancer toxicity value is available for crotonaldehyde. In this screening level assessment, toxicity values were not available for about 12 chemicals at each station, which may contribute to underestimating risk levels.

Crotonaldehyde is classified as a Group C, possible human carcinogen. This rating was assigned based on only one available animal carcinogenicity study that was limited by the use of only one sex of one species. Fewer tumors were observed in the high-dose group than in the low- dose group. It is unclear as to whether these effects would be anticipated following inhalation exposure to crotonaldehyde. Given the substantial uncertainty surrounding this toxicity value, the total cancer risk estimates were recalculated for the monitoring stations without crotonaldehyde. The total cancer risks without crotonaldehyde (and with crotonaldehyde) are noted below and shown in Figure 5.1:

- CAMP 1E-04 (2E-04 with crotonaldehyde)
- Welby 8E-05 (1E-04 with crotonaldehyde)
- Swansea 9E-05 (1E-04 with crotonaldehyde)

As seen, only one of the monitoring stations, CAMP, had total cancer risks that exceeded the EPA's upper acceptable limit of 1E-04. It should, however, be noted that the total cancer risks at all stations remain at the EPA upper acceptable limit of 1E-04 (or 100 in a million) even when crotonaldehyde is excluded.

Summary and Conclusions of the Screening-Level Risk Assessment

A screening-level risk assessment of the potential human health impacts from inhalation of air toxics has been conducted, in accordance with the Tier-1 of EPA's Air Toxic Risk Assessment Library (EPA, 2004), using data collected from air monitoring stations in Denver, Colorado. In general, this risk assessment can be considered a conservative estimate on the basis of the exposure assessment. For example, the chronic risk estimates are based on an individual that is exposed to the monitored concentrations over 70 years, for 24 hours per day. The potential human health implications of these exposures were characterized for both cancer and non-cancer health effects.

Cancer Risks

Total cancer risks were found to range from 1E-04 (100 excess cancers per 1 million individuals) to 2E-04 (200 excess cancers per 1 million individuals) across the various monitoring locations. These total risk estimates are based on all carcinogenic chemicals including crotonaldehyde, which as discussed in Section 6 was deemed to have a highly uncertain cancer toxicity value. Crotonaldehyde is one of the major contributors to the total risk at each monitoring station, with risk estimates for this chemical ranging from 4E-05 to 8E-05.

A range of "acceptable" health risk values for carcinogens has been historically proposed by U.S. EPA. Acceptability ranges from one in one million (1×10^{-06}) to one hundred per million (1×10^{-04}). Figure 5.1 shows a comparison of cancer risks across monitoring stations. As seen, the cancer risks are similar across all areas. Total cancer risks for the CAMP monitoring location slightly exceeds the upper end of EPA's acceptable risk range. Estimated total cancer risks for the CAMP monitoring location are 1E-04 without crotonaldehyde and 2E-04 with crotonaldehyde.

Non-Cancer Risks

Non-cancer risks were assessed at all monitoring stations by comparing the location specific exposure point concentration to a concentration that is considered to be without an appreciable risk of deleterious effects during a

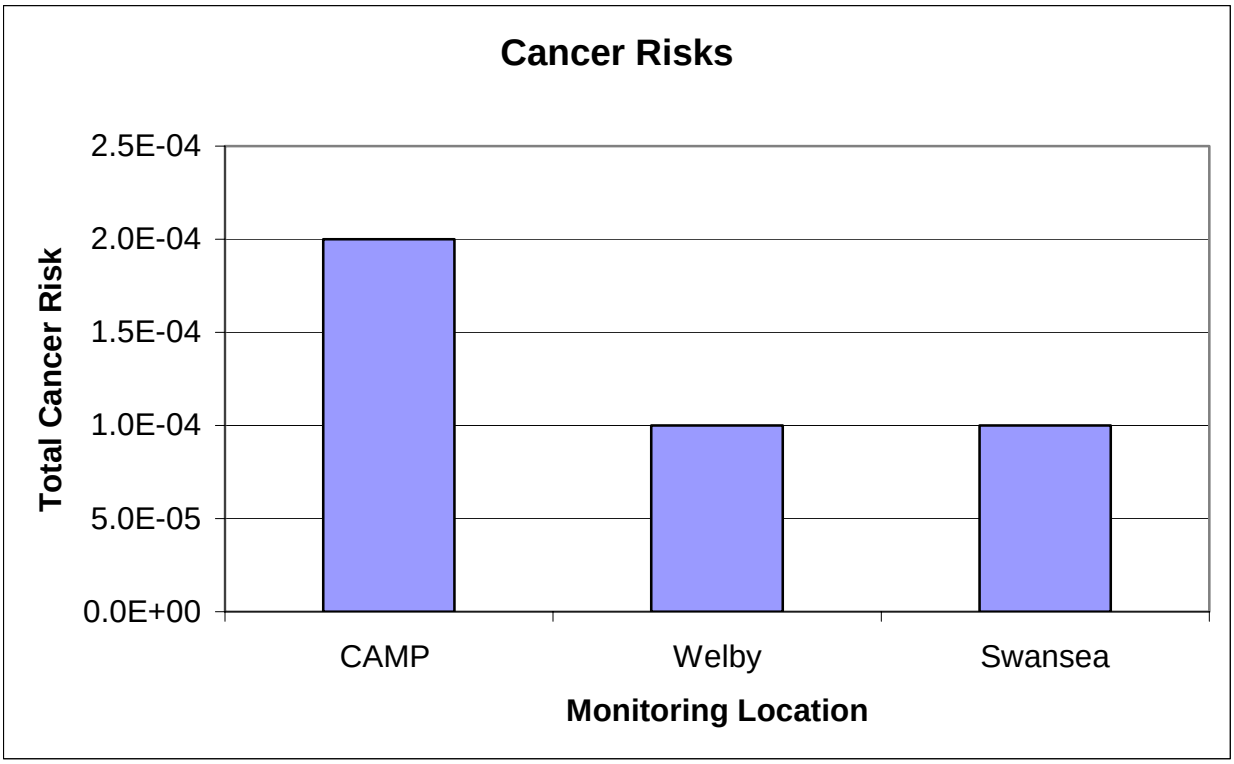
lifetime, for even the most sensitive individual. None of the individual chemicals that were assessed at any monitoring location were found to have a hazard quotient exceeding a value of one.

Hazard indices for each monitoring station were calculated by summing hazard quotients of individual chemicals that contribute to specific categories of known critical effects. For all critical effects other than respiratory and neurologic, hazard indices did not exceed a level of one at any monitoring location. For noncarcinogenic estimation of Hazard Indexes, an individual calculated index below one is generally regarded as an acceptable (or "safe") level of exposure.

Hazard indices of two were seen for respiratory effects at the CAMP and Swansea monitoring stations. The largest chemical contributors to the hazard indices at each of these locations were formaldehyde, acetaldehyde and 1,2,4-trimethylbenzene.

Hazard indices of two were also seen for neurologic effects at the CAMP, Welby, and Swansea monitoring stations. The largest chemical contributor to the hazard indices at each of the monitoring locations was manganese, which contributed to approximately 50% of the neurologic risk at each location. These elevated hazard indices indicate increased potential for respiratory and/or neurologic effects to occur in an individual exposed for seven or more years (i.e., 7 years to a lifetime) to air concentrations measured at several of the monitoring locations. These elevated hazard indices of 2 for respiratory and neurological effects indicate an important area for air monitoring and source apportionment.

Figure 5.1 - Cancer Risks



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Section 6 - Overall Summary and Conclusions

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Summary and Conclusions

Three carbonyls were present in all samples at all sites. These are formaldehyde, acetaldehyde, and acetone. However, levels of acetone were well below the non-cancer screening concentrations, so it was not retained in the risk analysis. Crontonaldehyde was present over 98% of the time, and is significant in the risk analyses. However, this crotonaldehyde significance is based on the use of a toxicity number based on an oral dose, not an inhalation dose. The inhalation health effects may be quite different. Automobiles are believed to be the largest emission source for formaldehyde, crotonaldehyde, and acetaldehyde, either as direct emissions, or as compounds forming from photochemical reactions. The impacts from aldehydes are difficult to control, because they can form as hydrocarbons emitted from automobiles and industrial processes react in the presence of sunlight. Analysis of results from the EPA national Urban Air Toxics Network indicates that acetaldehyde and acetone are problems on a nationwide scale. Levels of crotonaldehyde observed at the three sites are well within the normal range, as listed in the 2003 Urban Air Toxics Monitoring Program: Final Report. Thus, the situation in Denver is typical of most American cities.

Highest VOCs at Each Site

CAMP	Welby	Swansea
acetylene	acetylene	acetylene
propylene		propylene
toluene	toluene	toluene
m,p - xylenes	m,p - xylenes	m,p - xylenes
o-xylenes		o-xylene
Methyl ethyl ketone	methyl ethyl ketone	methyl ethyl ketone
dichlorodifluoromethane	dichlorodifluoromethane	dichlorodifluoromethane
acetonitrile		
trichlorofluoromethane		trichlorofluoromethane
benzene	benzene	benzene
1,2,4-trimethylbenzene		
		ethylbenzene

The highest volatile organic compounds at each site are shown in the table above. Except for methyl ethyl ketone and acetonitrile, these compounds were detected 100 percent of the time, at all three monitoring locations. Methyl ethyl ketone detections varied from site-to-site, suggesting local influences. Results from EPA's national network indicate that 1,3-butadiene, benzene, tetrachloroethylene and carbon tetrachloride are also a problem on a nationwide scale. 1-3 butadiene and benzene are believed to result from automobile emissions, while carbon tetrachloride and tetrachloroethylene are industrially-emitted compounds. Some other VOCs were present on a more localized basis, appearing at one site, but less often at the other. These are likely emitted from local industrial operations.

Tetrachloroethylene, or perchloroethylene, occurred at CAMP and Welby, but less than 7 percent of the time. It was never detected at Swansea. Concentrations suggest that this compound, used in dry cleaning, presents a greater than one-in-a-million risk of cancer. These results are consistent with EPA's national analyses, which indicate that levels of tetrachloroethylene are of concern in urban areas throughout the United States. p-Dichloro-benzene occurred less than 5 percent of the time at CAMP and Welby, and was never detected at Swansea. Although annual averages calculated indicate this compound may be a concern, the use of ½ the detection limit for all the non-detect days make these results highly uncertain. Unlike many of the others discussed, this compound appears to be a local problem.

Almost every metals sample had very low, but measurable, levels. Except for beryllium, all metals were detected in 100 percent of the samples. Lead and manganese were the metals detected at the highest concentrations. However, lead levels were well below the standards of 1.5 $\mu g/m^3$, as a monthly (Colorado standard) or a quarterly (federal standard) average. The levels of arsenic detected were low, were typical of other cities in Colorado, and were similar to other national air toxics monitoring sites. Manganese levels are speculated to be related to historic smelting activity, or may be naturally-occurring background levels.

In conclusion, a number of compounds related to vehicular emissions are present in Denver air. These are formaldehyde, acetaldehyde, benzene, and 1,3-butadiene. Carbon tetrachloride and tetrachloroethylene, which are from industrial sources, also may be a concern. Except for formaldehyde, these compounds appear to be at problem levels throughout the urban areas of the United States. Arsenic and manganese may also be of concern. Arsenic and chromium are present at low levels, while manganese may be from natural or industrial sources.

This report also presents the methodologies and findings of the risk evaluation for ambient air toxics monitoring conducted at three locations in Denver, Colorado from May 2002 through April 2003. The purpose of the evaluation was to determine if residents at any of these locations are being exposed to airborne concentrations of toxic air pollutants via inhalation that may pose unacceptable risks to human health. In general, this risk assessment can be considered a conservative estimate on the basis of the exposure assessment. For example, the chronic risk estimates are based on an individual that is exposed to the monitored concentrations over 70 years, for 24 hours per day. The potential human health implications of these exposures were characterized for both cancer and non-cancer health effects.

Total cancer risks were found to range from 1E-04 (100 excess cancers per 1 million individuals) to 2E-04 (200 excess cancers per 1 million individuals) across the various monitoring locations. These total risk estimates are based on all carcinogenic chemicals in this study, including crotonaldehyde, which was deemed to have a highly uncertain toxicity value. A range of "acceptable" health risk values for carcinogens has been historically proposed by U.S. EPA. Acceptability ranges from one in one million (1×10^{-06}) to one hundred per million (1×10^{-04}). As seen, the cancer risks are fairly comparable across all areas. Total cancer risks for the CAMP monitoring location slightly exceeds the upper end of EPA's acceptable risk range with estimated total cancer risks of 2E-04. Most large urban areas in the United States exhibit aggregate or total carcinogenic risks in the 10^{-04} to 10^{-05} range.

Non-cancer risks were assessed at all monitoring stations by comparing the location specific exposure point concentration to a concentration that is considered to be without an appreciable risk of deleterious effects during a lifetime, for even the most sensitive individual. None of the individual chemicals that were assessed at any monitoring location were found to have a hazard quotient exceeding a value of one. Hazard indices for each monitoring station were calculated by summing hazard quotients of individual chemicals that contribute to specific categories of known critical effects. For all critical effects other than respiratory and neurologic, hazard indices did not exceed a level of one at any monitoring location. For noncarcinogenic estimation of hazard indices, an individual calculated index below one is generally regarded as a "safe" level of exposure.

Hazard indices of two were seen for respiratory effects at the CAMP and Swansea monitoring stations. The largest chemical contributors to the hazard indices at each of these locations were formaldehyde, acetaldehyde and 1,2,4-trimethylbenzene. Hazard indices of two were also seen for neurologic effects at the CAMP, Welby, and Swansea monitoring stations. The largest chemical contributor to the hazard indices at each of the monitoring locations was manganese, which contributed to approximately 50% of the neurologic risk at each location. It is important to recognize that concentrations of manganese may be naturally occurring and investigations have not been conducted to determine if manganese represents background concentrations.

These elevated hazard indices indicate that there may be a potential for respiratory and/or neurologic effects to occur in an individual exposed for 7 or more years (i.e., 7 years to a lifetime) to air concentrations measured at several of the monitoring locations.

It should be noted that the results of this study and screening analysis are subject to some significant uncertainties. For example, EPA believes that acrolein contributes significantly to overall cancer risk. However, no monitoring method currently exists for acrolein in air. EPA is working to develop one for the future. Another uncertainty lies in the fact that polycyclic aromatic hydrocarbons (PAHs) were not monitored. Since diesel vehicles emit these compounds, an important vehicular air pollution source is absent from this analysis. Additionally, the study is based on one year of data collected at fixed monitoring stations. These fixed points may not adequately characterize exposure of a mobile population in a major metropolitan area. Most importantly, science is currently unable to assess exposures to multiple air toxics, simultaneously.

Calculations in this report use CDPHE and EPA's most recent, best estimates of a health risk values for each chemical compound. However, these health risk concentrations, as well as actual concentrations of chemicals in the air, change over time. Therefore, this study is best viewed as a "snapshot" in time.

A major goal of the study was to determine whether there are toxic compounds in air that are unique to Denver. If there are compounds that are significant locally, but not at the national level, then the EPA National Air Toxics Strategy may not be adequate to reduce air toxic cancer and non-cancer health risk in Colorado. The study results indicate that the compounds measured in Denver are the ones that EPA is focusing on nationally. For example, acetaldehyde and formaldehyde, two of the most important aldehydes monitored, are on the EPA list of Mobile Source Air Toxics (MSATs). The MSAT list of compounds contains 21 air toxics upon which the Environmental Protection Agency is focusing its control strategies. Benzene, 1,3-butadiene, toluene, and xylenes are also on this list. The two highest concentration metals observed in Denver, lead and manganese, are on the list. Acrolein and diesel exhaust, two air toxics this study did not address, are also MSAT targets.

It should be noted that many of the compounds observed in Denver are also on EPA's list of 33 Urban Hazardous Air Pollutants (HAPs). These compounds are believed to be the most important contributors to inhalation risk from outdoor air. Acetaldehyde, formaldehyde, benzene, 1,3 – butadiene, lead and manganese are on this list, just as they are on the MSAT list above. Tetrachloroethylene, while not an MSAT, is on this list of Urban HAPs. Crotonaldehyde is not on the EPA MSAT or HAPs lists, but Denver levels are within the range cited in national results for the Urban Air Toxics Monitoring Network in 2003. EPA's failure to list this compound is probably due to the fact that it lacks inhalation reference doses or cancer risk factors. However, strategies directed against acetaldehyde and formaldehyde will likely be effective in reducing crotonaldehyde. Thus, the study did not reveal any compounds that were of local-only significance.

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ATTACHMENT 1
Compounds Contributing to Cancer and Non-cancer Risks: Overview of Sources and Health Effects

Chemicals can be released to the environment as a result of their use and manufacture. Some chemicals may also form, as other chemicals react with sunlight and one another in outdoor air. A brief summary of the potential sources and health effects of cancer and non-cancer risk driving chemicals in the ambient air is provided below. This information is adopted from the following main sources: EPA Air Toxic Website, EPA Office of Pollution Prevention and Toxics (OPPT), EPA Integrated Risk Information System (IRIS), Agency for Toxic Substances and Disease Registry (ATSDR), New Jersey Department of Health and Senior Services, Occupational Safety and Health Administration (OSHA), National Institute of Occupational Safety and Health (NIOSH), and the California Air Resources Board (CARB).

Carbonyls

Three of the twelve carbonyl compounds sampled showed some toxicological significance. For purposes of this report, "significance" is defined as having a hazard quotient greater than 0.1, or contributing more than 1% to the total cancer risk at the monitoring location. (See Section 5 for the calculations of non-cancer and cancer risk at each site). Formaldehyde, acetaldehyde and crotonaldehyde are the compounds that are "significant". Information regarding the nature and sources of each of these compounds is given below.

Acetaldehyde

Acetaldehyde is a hydrocarbon with the formula CH_3CHO. It is thus closely related to formaldehyde, HCHO. Like formaldehyde, it exists in the atmosphere as a gas with a pungent odor. Acetaldehyde is ubiquitous in the ambient environment. It is mainly used as an intermediate in the synthesis of other chemicals, such as acetic acid, acetic anhydride, chloral, and glyoxal. It is employed in the food processing industry as a food and fish preservative, a flavoring agent, and in gelatin fibers. The tanning and paper industries use acetaldehyde, as do the perfume and dye manufacturers (CARB Acetaldehyde Fact Sheet).

Acetaldehyde can be released to the environment as a product of incomplete combustion in fireplaces and wood stoves, forest and wild fires, pulp and paper production, stationary internal combustion engines and turbines, vehicle exhaust, and petroleum refineries. Waste water processing is also a source. It is important to note that residential fireplaces and woodstoves are the two highest sources of emissions, followed by various industrial emissions.

Although it is used in industry, the California Air Resource Board believes that the largest sources in outdoor air are combustion and production from photochemical reactions (CARB Acetaldehyde Fact Sheet). Acetaldehyde itself can break down in these complex photochemical reaction pathways, forming formaldehyde.

The health effects of acetaldehyde are very similar to those of its chemical relative formaldehyde. It irritates the eyes and mucous membranes. It can paralyze the respiratory muscles, act as a narcotic to prevent coughing, and speed up pumping of the heart. Exposure can lead to headaches and sore throat. (Kirk Othmer, Vol 1, page 107). It should be noted that most of these health effects have been observed in factory workers, who are exposed to acetaldehyde concentrations thousands of times greater than those occurring in outdoor air. Acetaldehyde is believed to be a probable human carcinogen, leading to cancer of the nose and throat. Acetaldehyde has been shown to cause birth defects in animals, but no human research is available. (CARB Acetaldehyde Fact Sheet).

EPA's Technology Transfer Network Air Toxic Website provides information on the potential health effects of acetaldehyde. According to this source , the primary acute effects of acetaldehyde is irritation of the eyes, skin, and respiratory tract in humans. At higher exposure levels, erythema, coughing, pulmonary edema, and necrosis may happen. Chronic toxicity symptoms in humans resemble those of alcoholism.

The EPA has established a Reference Concentration (RfC) for inhalation exposure to acetaldehyde based on degeneration of the olfactory epithelium in rats. No information is available on the reproductive and developmental effects of acetaldehyde in humans. Animal studies data indicate that acetaldehyde may be a potential developmental toxin. EPA has classified acetaldehyde as a Group B2, probable human carcinogen, based on increased incidence of nasal tumors in male and female rats and laryngeal tumors in male and female hamsters after inhalation exposure.

The California Air Resources Board observed an annual mean of 1.33 ppb acetaldehyde in its state-wide network during 1996 (CARB Acetaldehyde Fact Sheet). The means observed in this Denver study, 2.30 ppb (CAMP), 1.59 ppb (Welby) and 1.81 ppb (Swansea) are a bit above the California data, but acetaldehyde in Denver occurs at levels typical of large urban areas. The annual report of the 2003 nationwide UATMP results gives a range of 0.02 – 9.44 ppbv observed throughout the network. The range for the Denver sites was 0.78- 3.08 ppbv. Acetaldehyde levels are therefore a national problem related primarily to the use of motor vehicles.

Crotonaldehyde

Crotonaldehyde, with the chemical formula of C_4H_6O, is also known as propylene aldehyde, beta-methylacrolein, crotinin aldehyde and butenal. Crotonaldehyde is a colorless liquid with a pungent, suffocating odor.

Crotonaldehyde can be emitted to the environment from the combustion of gasoline, the burning of wood, paper, cotton, plastic, and tobacco. It can also be released through industrial use. It is found naturally in emissions of some vegetables and volcanoes.

According to the ATSDR Medical Management Guidelines inhaled crotonaldehyde is highly toxic. It is irritating to the upper respiratory tract even at low concentrations. Crotonaldehyde vapor is heavier than air. Therefore, higher levels of crotonaldehyde vapors would be found nearer to the ground. The mechanism of toxicity of crotonaldehyde is not known, but it is highly reactive. Crotonaldehyde is also a skin irritant and can cause eye irritation and damage to the cornea. After an acute, relatively high concentration exposure, people may become sensitized to crotonaldehyde. Except for rare cases of sensitization, no health effects have been reported in humans exposed to relatively low concentrations of crotonaldehyde. No studies have been found that address reproductive or developmental edffects of crotonaldehyde in humans. The compound has been shown to cause degeneration of spermatocytes in mice. No teratogenic effects from acute exposures have been reported.

The Department of Health and Human Services has determined that crotonaldehyde may be a possible carcinogen. The EPA IRIS has classified crotonaldehyde as a possible carcinogen based on the fact that while there is no human data, there is an increased incidence of hepatic tumors in male rats. The possible carcinogenicity of crotonaldehyde is supported by genotoxic activity and the expected reactivity of croton oil and aldehyde. The EPA IRIS, however, has not derived a cancer toxicity value for the compound. The EPA HEAST (Health Effects Summary Tables) has established an oral cancer toxicity value for crotonaldehyde. The Agency For Research on Cancer has determined that crotonaldehyde is not classifiable as to its carcinogenicity to humans.

The annual report of the 2003 nationwide UATMP results gives a range of non-detect – 1.44 ppbv observed throughout the network. The range for the Denver sites was non-detect - 0.13 ppbv.

Formaldehyde

Formaldehyde is a hydrocarbon compound with the formula HCHO. It exists in the atmosphere as a colorless gas with a pungent odor. It is used in the manufacture of urea-formaldehyde resins which are used in particleboard and plywood products. Therefore, high levels of airborne formaldehyde can also be found in indoor air as a result of release from various consumer products such as building materials and home furnishings. Another source of formaldehyde in indoor air is smoking. It is also employed in chemical manufacturing of pharmaceuticals, herbicides, and sealants. Textile finishes, such as used for “permanent press” clothes, contain formaldehyde (Kirk-Othmer, Vol 11, pages 245 - 246).

EPA's Technology Transfer Network Air Toxic Website provides information on the potential sources and health effects of formaldehyde. According to this source, the major sources of formaldehyde emissions to the ambient air include power plants, manufacturing facilities, incinerators, forest and wild fires, stationary internal combustion engines and turbines, pulp and paper plants, petroleum refineries, and automobile traffic. In urban areas, combustion of automotive fuel is the dominant source for much of the year. However, formaldehyde can also form photochemically in the air, as other hydrocarbons and oxides of nitrogen from automobile traffic break down to form ozone. Complicating the situation is the fact that the complex ozone-producing atmospheric reactions may both create and destroy formaldehyde, as the chains of chemical reactions proceed along various pathways.

The Agency for Toxic Substances and Disease Registry (ATSDR), lists a number of possible health effects that may occur from inhalation of formaldehyde. Formaldehyde is an irritant. The major acute toxic effects via inhalation exposure are eye, nose, and throat irritation and effects on the nasal cavity. At 0.4 – 3 ppm, it may cause the eyes to tear. Other effects observed in humans from exposure to high levels of formaldehyde are coughing, wheezing, chest pain, and bronchitis (EPA's Technology Transfer Network Air Toxic Website). Formaldehyde is believed to be carcinogenic (cancer-causing) to humans. However, the body can quickly break down formaldehyde, so it does not accumulate in fatty tissue. Currently, ATSDR believes that formaldehyde does not cause birth defects in humans (ATSDR Toxicological Profile for Formaldehyde). Thus, the main concerns with this compound are its irritant properties and its potential ability to cause cancer of the nose and throat.

Chronic inhalation exposure to formaldehyde in humans has been associated with respiratory symptoms and eye, nose, and throat irritation. EPA has not established an inhalation Reference Concentration (RfC) for formaldehyde. However, the ATSDR has established an inhalation reference concentration called a Minimal Risk Level (MRL) for formaldehyde based on respiratory effects in humans. Developmental effects, such as birth defects, have not been observed in animal studies. EPA has classified formaldehyde as a Group B1, probable human carcinogen, based on limited evidence in humans and sufficient evidence in animals. Occupational studies have shown statistically significant increases in incidence of lung and nasopharyngeal cancer. This evidence is considered limited because of possible exposure to other agents. Animal studies have reported an increased incidence of nasal squamous cell carcinoma by inhalation exposure. Please see EPA IRIS for a detailed discussion on the carcinogenicity of formaldehyde.

ATSDR states that typical levels of formaldehyde in urban air are 10 – 20 ppb. ATSDR cites concentrations of 0.2 ppb for rural areas, and 2-6 ppb for suburban areas (ATSDR Toxicological Profile for Formaldehyde). The mean levels observed in Denver during this study, 6.63 ppb (CAMP), 2.84 ppb (Welby) and 4.07 ppb (Swansea) are within the "suburban" range. The annual report of the 2003 nationwide UATMP results gives a range of 0.11 – 40.00 ppbv observed throughout the network. The range for the Denver sites was 1.24 – 7.66 ppbv.

Volatile Organic Compounds

About ten of the fifty-eight volatile organic compounds sampled contributed to cancer and/or noncancer risks. For purposes of this report, compounds having a hazard quotient greater than 0.1, or contributing more than 1 % to the total cancer risk at the monitoring location are discussed as major and minor contributors to the total risk. (See Section 5 for the calculations of non-cancer and cancer risk at each site). At CAMP, the volatile organic compounds 1,3-butadiene, acetonitrile, benzene, 1,3,5-trimethylbenzene, and 1,2,4-trimethylbenzene had hazard quotients greater than or equal to 0.1. These same VOC compounds, except acetonitrile, had hazard indices above 0.1 at Swansea. (Acetonitrile data was invalidated at Swansea and Welby, due to sample contamination problems.) At Welby, the only VOCs with hazard quotients above 0.1 were 1,3-butadiene and 1,2,4-trimethylbenzene.

Volatile organic compounds contributing more than 1 % of the cancer risk at CAMP included 1,3-butadiene, chloroform, benzene, carbon tetrachloride, trichloroethylene, tetrachloroethylene, and p-dichlorobenzene. Welby and Swansea showed the same contributors, except for trichloroethylene and p-dichlorobenzene. Some health summary and source information regarding the ten significant compounds is given below.

Acetonitrile

Acetonitrile is a volatile organic compound with the formula CH_3CN. In the atmosphere, it exists as a gas. Acetonitrile is used in the chemical industry for making acrylic fibers, nitrile rubber, perfumes and pharmaceuticals. (CARB Fact Sheet on Acetonitrile). It is often used as a solvent.

Emissions from automobiles and manufacturing operations are the main atmospheric sources of acetonitrile. The California Air Resources Board indicates that coating, engraving, and allied services are the main stationary sources of the compound in California (CARB Fact Sheet on Acetonitrile).

Acetonitrile, also known as methyl cyanide, is metabolized to hydrogen cyanide in the human body (EPA OPPT Chemical Fact Sheet on Acetonitrile). As this metabolism takes time, health reactions to an exposure to acetonitrile may be delayed. Acetonitrile is an irritant to the skin, eyes, and lungs. Very high exposures can affect the nervous system, leading to drooling, nausea, vomiting, confusion, headache, and convulsions. Levels greater than 500 ppm can cause death (New Jersey Hazardous Substance Fact Sheet on Acetonitrile). It should be noted that many of these health effects are observed to occur at concentrations thousands of times higher than those usually found in outdoor air. Studies have indicated that acetonitrile can cause birth defects in animals, but generally only at levels where the mother is experiencing obvious symptoms. The EPA has established a Reference Concentration for inhalation exposure to acetonitrile based on mortality in mice. It is not known whether acetonitrile can cause cancer. Due to a lack of studies in this area EPA considers it not classifiable as to carcinogenic status.

EPA's OPPT chemical fact sheet on acetonitrile cites air concentration information in the Hazardous Substance Data Bank (HSDB). According to this source, levels in rural and urban US areas range from 2 to 7 ppb. The annual mean of 1.46 ppb at CAMP is a bit below the normal range, and the downtown Denver site detected acetonitrile only 18 percent of the time. (Acetonitrile data were not available from Swansea or Welby, due to sampler contamination problems at these locations). The annual report of the 2003 nationwide UATMP results gives a range of non-detect – 147.76 ppbv observed throughout the network. The range for the CAMP site was 0.46 – 56.83 ppbv.

Benzene

Benzene is a hydrocarbon compound with the formula C_6H_6. It exists in the atmosphere as a colorless gas with a sweet odor. It is used in chemical manufacturing of medicines, detergents, explosives, shoes, dyes, leather, resins, paints, plastics and inks (CARB Fact Sheet on Benzene). It is also present in gasoline.

The largest sources of benzene in ambient air are automobiles, gasoline service stations, refineries, and chemical plants. Burning of vegetative matter in forest fires and woodstoves is also a source. In ambient air, benzene reacts with hydroxyl (OH^-) radicals within a few hours. Since hydroxyl radicals are common in outdoor air, this chemical transformation prevents the build-up of large concentrations of benzene.

Benzene is a serious concern from a toxicological standpoint. Unlike many of the compounds discussed here, benzene is a proven human carcinogen. It damages the blood-forming capacity of the body, leading to anemia or leukemia. Like the other volatile organic compounds, breathing large amounts can cause lightheadedness, headache, vomiting, convulsions, coma and death. It also irritates the skin and eyes, exerting a drying effect. However, these health effects are usually seen in workplaces, where levels are thousands of times higher than those in outdoor air. Experiments with laboratory animals suggest that benzene exposure may be associated with numerous cancers. It may cause bone marrow damage and bone formation problems for a developing fetus (ATSDR Toxicological Profile for Benzene). Thus, EPA has had concern about whether levels of benzene in outdoor air are associated with cancer and leukemia. While no link with outdoor air concentrations has been unequivocally proven, EPA has acted to reduce air concentrations of this pollutant.

The EPA has established a Reference Concentration for inhalation exposure to benzene based on decreased lymphocyte count in an occupational epidemiologic study. Benzene is classified as a "known" human carcinogen

for all routes of exposure by the EPA IRIS based on the increased incidence of leukemia in epidemiologic and case studies.

The Agency for Toxic Substances and Disease Registry (ATSDR) cites national 1984 to 1986 data from 300 cities, which indicate an average benzene level of 1.8 ppb for urban and suburban areas (ATSDR Toxicological Profile for Benzene). The CAMP site mean of 0.99 ppb observed in this study is somewhat lower. Welby had a mean of 0.77, while the Swansea site had a mean of 0.87 ppbv. This suggests that benzene concentrations are fairly consistent in central Denver. The annual report of the 2003 nationwide UATMP results gives a range of 0.03 – 2.69 ppbv observed throughout the network. The range for the Denver sites was 0.21 – 2.20 ppbv.

1,3-Butadiene

1,3-Butadiene is a hydrocarbon compound with the formula C_4H_6. It exists in the atmosphere as a colorless gas with an odor similar to gasoline. It is used in making rubber and plastics. The most important use is in tire production. It is also used in the production of chemicals such as 1,4-hexadiene (NIOSH Current Intelligence Bulletin 41).

According to the California Air Resources Board, most emissions of 1,3-butadiene come from combustion of fuels in diesel and gas-powered motor vehicles. Other sources that they list include petroleum refining, tire wear, residential wood heating, and forest fires. Rubber and chemical production plants also have emissions. Breathing of cigarette smoke is another source of 1,3-butadiene exposure (ATSDR Fact Sheet).

1,3-Butadiene is of concern toxicologically because it is characterized as carcinogenic to humans based on the new EPA guidelines for cancer risk assessment and it also has adverse effects on reproduction and fetal development. Exposure to high concentrations can cause irritation and central nervous system effects such as eye irritation, cough, sore throat, headache, drowsiness, nausea, unconsciousness, and death. Rats and mice exposed to this compound in laboratory tests developed multiple cancers within single individuals. The animals had damaged testes and ovaries, and offspring of the animals had skeletal problems. Other effects seen in animals at low levels of inhalation exposure for one year include kidney and liver disease, and damaged lungs (ATSDR Fact Sheet). Generally, the acute health effects have not been seen at concentrations existing in outdoor air. However, EPA considers that the levels of 1,3-butadiene in air may represent a significant portion of the cancer risk related to ambient airborne chemicals.

The EPA has established a Reference Concentration for inhalation exposure to 1,3-butadiene based on ovarian atrophy in mice. The EPA has characterized 1,3-butadiene as carcinogenic to humans by inhalation based on the following total evidence: sufficient evidence from epidemiologic studies showing increased lymphohemato-poietic cancers and leukemia; tumors at multiple sites in animal studies, and strong evidence suggesting that the carcinogenic effects are mediated by genotoxic metabolites of 1,3-butadiene.

ATSDR estimates that urban and suburban areas have an average concentration of 0.3 ppb 1,3-butadiene, while rural areas have 0.1 ppb (ATSDR Toxicological Profile for 1,3-Butadiene). The annual average at CAMP is 0.14 ppbv, Welby's average is 0.10 ppbv, and Swansea's is 0.13 ppbv. The compound was detected 60 – 80 percent of the time. The annual report of the 2003 nationwide UATMP results gives a range of non-detect – 0.65 ppbv observed throughout the network. The range for the Denver sites was non-detect – 0.26 ppbv.

Carbon Tetrachloride

Carbon tetrachloride, also known as tetrachloromethane or methane tetrachloride, is a chlorinated hydrocarbon with the formula CCl_4. It exists in the atmosphere as a gas. It has a sweet odor. The primary uses of carbon tetrachloride were as a dry cleaning solvent, a grain fumigant, as a refrigerant, and as an aerosol propellant. Carbon tetrachloride has a long atmospheric half-life, so it can travel to the higher reaches of the atmosphere and damage the earth's ozone layer. Due to its toxicity and ozone-damaging qualities, most uses of carbon tetrachloride have been banned. It is still in use in industrial settings for producing refrigerants.

Carbon tetrachloride is emitted to the air from industrial sources and from petroleum refineries (California Air Resources Board Toxic Air Contaminant Identification List Summary for Carbon Tetrachloride). Carbon tetrachloride is also a common indoor air contaminant due to releases from building materials and products, such as cleaning agents, used in homes (Air Toxic Website). There are no natural sources of carbon tetrachloride; it is produced by man (ATSDR Toxicological Profile for Carbon Tetrachloride).

As is true for many of the chlorinated hydrocarbons, breathing large concentrations of carbon tetrachloride has central nervous system effects including lightheadedness, coma, convulsions, double vision, intoxication, and death. It can also cause vomiting. In animal studies, it had effects on the liver and kidney. Male rats exposed to carbon tetrachloride had lower sperm production. Female rats exposed to it had stunted offspring with birth defects. These health effects are generally observed in occupational settings, where people had exposure to very high levels over a number of years.

EPA has not established a Reference Concentration for carbon tetrachloride. The CalEPA has established a Reference Exposure Level for carbon tetrachloride based on liver effects in guinea pigs. Carbon tetrachloride has been associated with liver and kidney cancer in animals. EPA considers it a Class B2 Carcinogen (probable human carcinogen) based on liver tumors in animals.

The California Air Resources Board has monitored carbon tetrachloride at a number of locations, and found a mean value of 0.078 ppb (California Air Resources Board Toxic Air Contaminant Identification List Summary for Carbon Tetrachloride). The 0.07 ppb annual mean observed at all three sites in this study is at the same level. The annual report of the 2003 nationwide UATMP results gives a range of non-detect – 0.19 ppbv observed throughout the network. The range for the Denver sites was non-detect – 0.16 ppbv.

Chloroform

Chloroform, also known as trichloromethane or methane trichloride, is a chlorinated hydrocarbon with the formula $CHCl_3$. It exists in the atmosphere as a gas. It has a pleasant odor. The primary use of chloroform is in the production of chlorodifluoromethane (HCFC-22), which is a refrigerant.

Chloroform is emitted to the air from sources such as swimming pools, chlorinated water, and pulp and paper plants. It is also emitted from wastewater treatment plants. Some bacteria can produce chloroform under anaerobic conditions, but most emissions are man-made (ATSDR Toxicological Profile for Chloroform).

As is true for many of the hydrocarbons, breathing large concentrations of chloroform has central nervous system effects including lightheadedness, vomiting, coma, convulsions, double vision, intoxication, and death. In animal studies, chloroform had effects on the liver, spleen, and kidney. Male rats exposed to chloroform had lower sperm production. Female rats exposed to chloroform had stunted offspring with birth defects.

In humans, chronic exposure to chloroform through inhalation is associated with effects on the liver, including hepatitis and jaundice, and central nervous system effects, such as depression, and irritability (EPA Air Toxic Website). EPA has not established a Reference Concentration for chloroform. The CalEPA has established a Reference Exposure level (0.3 mg/m3) based on kidney and liver effects in rats. The ATSDR has established an inhalation Minimal Risk Level (MRL) (0.1 mg/m3) based on liver effects in humans. Chloroform has been associated with liver and kidney cancer in animals, but EPA considers it a Class B2 Carcinogen (probable human carcinogen). Chloroform is likely to be carcinogenic to humans under high-exposure conditions that lead to cytotoxicity and regenerative hyperplasia. The current inhalation unit risk factor from EPA IRIS is based on the 1987 evaluation and does not incorporate newer data or the new EPA cancer risk assessment guidelines. EPA is currently working to revise the assessment for inhalation exposure.

The Environmental Protection Agency has monitored at 104 locations, and found a mean value of 0.55 ppb (California Air Resources Board Toxic Air Contaminant Identification List Summary for Chloroform). The 0.04 ppb mean observed at all three sites in this Denver study is an order of magnitude below that. The annual report of the 2003 nationwide UATMP results gives a range of non-detect – 0.56 ppbv observed throughout the network. The range for the Denver sites was non-detect – 0.16 ppbv.

p-Dichlorobenzene

1,4-Dichlorobenzene, also known as para-dichlorobenzene, is a chlorinated hydrocarbon with the formula $C_6H_4Cl_2$. It exists in the atmosphere as a gas. It has a mothball-like odor. The primary uses of 1,4-dichlorobenzene are for mothballs, insecticide, or as a dry solid room/trash bin/toilet deodorant.

Most emissions of 1,4-dichlorobenzene in air come from its household uses as an insecticide and deodorant, or from factories that produce these household products. Industrial operations producing polyphenylene sulfide may also emit it, as 1,4-dichlorobenzene is used in the production process. There are no natural sources of 1,4-dichlorobenzene; it is produced by man (ATSDR Toxicological Profile for 1,4-Dichlorobenzene).

As is true for many of the chlorinated hydrocarbons, breathing large concentrations of 1,4-dichlorobenzene has central nervous system effects including lightheadedness, coma, convulsions, double vision, intoxication, and death. It also can cause vomiting. In animal studies, it had effects on the liver and kidney. 1,4-dichlorobenzene also effects the blood, leading to anemia and possibly, leukemia. (New Jersey Hazardous Substance Fact Sheet for 1,4-Dichlorobenzene). However, these health effects are generally observed in occupational settings. 1,4-dichlorobenzene has been associated with liver and kidney cancer in animals.

The EPA has established a Reference Concentration for inhalation exposure to 1,4-dichlorobenzene based on liver effects in rats. The EPA IRIS has not conducted a complete evaluation and determination for evidence of human carcinogenic potential. However, the International Agency for Research on Cancer (IARC) has classified p-dichlorobenzene as B2, possibly carcinogenic to humans. The CalEPA has derived an inhalation unit risk factor for cancer using an oral cancer toxicity value based on hepatic tumors in male mice.

The Environmental Protection Agency has monitored 1,4-dichlorobenzene at a number of locations, and found a mean value of 0.17 ppb during 1976 – 1986 (California Air Resources Board Toxic Air Contaminant Identification List Summary for 1,4-Dichlorobenzene). An 0.07 ppb mean was observed at CAMP and Welby, but the compound was detected less than 5 percent of the time. There were no levels above detection limit at Swansea. The annual report of the 2003 nationwide UATMP results gives a range of non-detect – 0.71 ppbv observed throughout the network. The range for the Denver sites was non-detect – 0.04 ppbv.

Tetrachloroethylene

Tetrachloroethylene, also known as perchloroethylene, is a chlorinated hydrocarbon with the formula C_2Cl_4. It exists in the atmosphere as a gas. It has a "chloroform-like" odor (NIOSH Pocket Guide to Chemical Hazards, Tetrachloroethylene). The primary uses of tetrachloroethylene are as a dry cleaning solvent, metal cleaning solvent, or for chemical production. Tetrachloroethylene is used in paints, inks, aerosols, glues, polishes, silicones and rubber products (CARB Fact Sheet on Tetrachloroethylene and EPA OPPT Chemical Fact Sheet on Tetrachloroethylene).

Most emissions of tetrachloroethylene come from degreasing, dry cleaning, or chemical production facilities. There are microorganisms that can produce tetrachloroethylene (ATSDR Toxicological Profile For Tetrachloroethyelene).

As is true for many of the chlorinated hydrocarbons, breathing large concentrations of tetrachloroethylene has central nervous system effects including lightheadedness, coma, convulsions, double vision, intoxication, and death. It also can cause vomiting. In animal studies, it had effects on the liver and kidney. It also is an irritant to eyes, lungs, and skin. However, many of these health effects were observed in occupational settings, where exposure is much higher than in outdoor air. Some animal studies suggest that tetrachloroethylene exposure may lead to leukemia (NIOSH Registry of Toxic Effects of Chemical Substances Information for Tetrachloroethylene). Tetrachloroethylene has been associated with liver and kidney cancer in animals.

The ATSDR has established a Minimal Risk Level (MRL) based on nervous system effects in humans. It is important to note that EPA is currently re-evaluating the toxic potential of tetrachloroethylene, including its carcinogenicity, and therefore no relevant information is available in IRIS. In the interim, EPA recommends the use

of CalEPA toxicity values as provisional values. The CalEPA cancer toxicity value is derived by considering data on liver tumors in male and female mice and mononuclear cell leukemia in male and female rats. EPA is currently working to revise the toxicity assessment for tetrachloroethylene.

The California Air Resources Board has monitored tetrachloroethylene at a number of locations within their state, and found a mean value of 0.019 ppb during 1996 (California Air Resources Board Toxic Air Contaminant Identification List Summary for Tetrachloroethylene). The annual mean at CAMP was 0.07 ppbv. Welby had a mean of 0.07 ppb, while Swansea had 0.08 ppb. These levels are greater than the network-wide mean value for California. However, this compound was detected less than half the time, for all three sites. The annual report of the 2003 nationwide UATMP results gives a range of non-detect – 768.10 ppbv observed throughout the network. The range for the Denver sites was non-detect – 1.63 ppbv.

Trichloroethylene

Trichloroethylene, also known as trichloroethene or acytylene trichloride, is a chlorinated hydrocarbon with the formula C_2HCl_3. It exists in the atmosphere as a gas. It has a sweet odor. The primary use of trichloroethylene is as a metal degreasing solvent. It is also used in paints, glues, and cleaning solvents. The chemical production industry also uses trichloroethylene.

Emission sources of trichloroethylene include automobile repair and metal fabrication shops, wastewater treatment plants, chemical plants, and landfills. There are marine algae that can produce trichloroethylene (ATSDR Toxicological Profile For Trichloroethyelene), but it is predominantly a man-made chemical.

As is true for many of the chlorinated hydrocarbons, breathing large concentrations of trichloroethylene has central nervous system effects including lightheadedness, coma, convulsions, double vision, intoxication, and death. It also can cause vomiting. In animal studies, it had effects on the liver and kidney. It also is an irritant to eyes, lungs, and skin. In workplace exposure situations, it has been associated with skin rashes and permanent nerve damage to facial muscles.

The toxic potential of TCE has been re-evaluated by the EPA and the draft toxicity assessment report was made available in 2001, but the final recommendations on the EPA IRIS will be available after the National Academy of Science recommendations. In summary, EPA classified TCE as a "possible-to-probable human carcinogen" in the late 1980's. The EPA completed the re-evaluation of the toxic potential of TCE in 2001 and classified TCE as a "highly likely human carcinogen" in the draft toxicity assessment report. This finding is supported by observations of increased risk of kidney, liver, lymphatic-hematopoietic, prostate, and cervical cancers in workers exposed to TCE. TCE has significant noncancer toxicity affecting the central nervous system and immune system, liver, kidney, and endocrine system following inhalation exposure. In 2004, the CDPHE adopted the new draft toxicity assessment as an interim policy.

The California Air Resources Board has monitored trichloroethylene at a number of locations within their state, and found a mean value of 0.033 ppb during 1996 (California Air Resources Board Toxic Air Contaminant Identification List Summary for Trichloroethylene). The .08 ppb annual mean observed at CAMP, and the value of 0.05 ppb at Swansea and 0.06 ppb at Welby, are significantly higher than the California state-wide average. Evidently, concentrations of this chemical in air are very localized, due to its emission sources in small-scale auto repair or metal shops. The annual report of the 2003 nationwide UATMP results gives a range of non-detect – 20.38 ppbv observed throughout the network. The range for the Denver sites was non-detect – 0.59 ppbv.

1,3,5-Trimethybenzene and 1,2,4-Trimethylbenzene

1,3,5-trimethylbenzene and 1,2,4-trimethylbenzene are isomers of the hydrocarbon formula C_9H_{12}. In pure form they are colorless liquids. They are used in chemical manufacturing of medicines, detergents, dyes, paints and inks. Trimethylbenzenes are a large component of distilled petroleum. They are also used as gasoline additives.

The largest sources of trimethylbenzenes in ambient air are likely to be automobiles, gasoline service stations, refineries, and chemical plants. In ambient air, trimethylbenzenes have a half-life of less than a day (EPA OPPT Chemical Summary For 1,2,4-Trimethylbenzene).

Health effects of trimethylbenzenes are similar to those of benzene. It damages the blood-clotting capacity of the body. Like the other volatile organic compounds, breathing large amounts can cause lightheadedness, headache, vomiting, convulsions, coma and death. It also irritates the skin and eyes, exerting a drying effect. Long-term exposure can lead to cough, reduced lung capacity, and bronchitis. However, these health effects are usually seen in workplaces, where levels are thousands of times higher than those in outdoor air. It is not known whether these compounds are carcinogenic. Some animal experiments suggest that they may cause bone formation problems for a developing fetus (EPA OPPT Chemical Summary For 1,2,4-Trimethylbenzene).

The Environmental Protection Agency cites national data indicating that average atmospheric concentrations of 1,2,4-trimethylbenzene are 0.58 ppb in rural areas, and 1.20 ppb in cities (EPA OPPT Chemical Summary For 1,2,4-Trimethylbenzene). The three Denver sites all had mean values of 0.06 ppb. As the EPA citation is for 1988, it is likely that concentrations have gone down in recent years. The annual report of the 2003 nationwide UATMP results gives a 1,3,5-trimethylbenzene range of non-detect – 2.69 ppbv observed throughout the network. The range for the Denver sites was non-detect – 0.34 ppbv. For 1,2,4-trimethylbenzene the national range was non-detect – 5.02 ppbv, and the Denver sites' range was 0.06 – 0.94 ppbv.

Metals

About four of the eleven metals sampled contributed to cancer and/or noncancer risks. For purposes of this report, compounds having a hazard quotient greater than 0.1, or contributing more than 1 % to the total cancer risk at the monitoring location are discussed as minor and major contributors to the total risk. (See Section 5 for the calculations of non-cancer and cancer risk at each site). These compounds are discussed below.

Arsenic

Arsenic is a metal-like element that occurs naturally in the earth's crust. Its chemical symbol is As. It exists in the atmosphere as particulate matter, in compounds formed from combination with other atoms such as oxygen, chlorine, and sulfur (ATSDR Public Health Statement for Arsenic). In the past, arsenic was used as a pesticide for orchard crops. Today, the chief use is in chromated copper arsenate (CCA) used to "pressure-treat" wood, to preserve it from decay in marine or in-ground usage. It is also used in metal alloy, glass-making, and electrical semi-conductors.

Emission sources of arsenic include smelters, coal-fired power plants, wood-burning, metals operations, mining operations, and incinerators. Arsenic occurs naturally in many soils, so wind-blown dusts from exposed land can contain it. Mine tailings piles generally contain enriched levels of arsenic, resulting in emissions of arsenic in the particulate emissions that occur under windy conditions. Soils contaminated by smelter fall-out can also be a source of emissions during high winds. Burning wood treated with CCA also leads to arsenic emissions.

Arsenic's toxicity has led to its use as a poison. Orally ingesting large amounts can be fatal. The effects of inhalation are similar to the oral effects. Arsenic disturbs the gastro-intestinal system, leading to abdominal pain, vomiting, and diarrhea. It affects the central nervous system, leading to nerve damage in the legs and arms. It can damage the liver and kidney. Arsenic also has effects on the skin, causing dark patches (hyperpigmentation), and skin cancer. Arsenic also irritates the eyes, lungs, and skin. These effects have been observed in situations of occupational exposure that are significantly higher than concentrations seen in outdoor air. Exposure can lead to effects in the blood, such as anemia.

EPA has not established a Reference Concentration for arsenic. The Cal EPA has established a chronic reference level based on the developmental effects in mice; and other target organs include the cardiovascular system and the nervous system. Arsenic exposure is known to cause lung cancer. EPA classifies arsenic in Group A, the known human carcinogens, based on an increased lung cancer mortality in multiple human populations exposed primarily through inhalation.

The Agency for Toxic Substances and Disease Registry (ATSDR) states that remote areas have concentrations of 0.001 to 0.003 µg/m3 arsenic in air, while urban locations range from 0.020 to 0.100 µg/m3 (ATSDR Toxicological Profile on Arsenic). The mean levels at the three Denver sites fall within the rural range. The annual report of the 2003 nationwide UATMP results gives a range of 0.10 – 1267.10 ppbv observed throughout the network. The range for the Denver sites was 0.10- 6.48 ppbv.

Cadmium

Cadmium is a metal-like element that occurs naturally in the earth's crust. Its chemical symbol is Cd. It exists in the atmosphere as particulate matter, in compounds formed from combination with other atoms such as oxygen, chlorine, and sulfur (ATSDR Public Health Statement for Cadmium). Cadmium is used in metal plating, in battery production, as a stabilizer in plastic products, in automobile tires and radiators, in printing, in fabric dyes, and in the electronics industry (CARB Toxic Air Contaminant Fact Sheet for Cadmium).

Emission sources of cadmium include smelters, coal-fired power plants, wood-burning, metals operations, mining operations, and incinerators. Cadmium occurs naturally in many soils, so wind-blown dusts from exposed land can contain it. Mine tailings piles generally contain enriched levels of cadmium, resulting in emissions of cadmium in the particulate emissions that occur under windy conditions. Soils contaminated by smelter fall-out can also be a source of emissions during high winds. Forest fires and volcanoes are natural emission sources of cadmium. As cadmium is often a trace element in fertilizers, fertilizer application is also an emissions source.

Cadmium disturbs the gastro-intestinal system, leading to abdominal pain, vomiting, and diarrhea. It affects the central nervous system, leading to nerve damage in the legs and arms. It can damage the liver and kidney, leading to kidney problems such as proteinuria and kidney stones. Long-term exposure has been associated with lung cancer in workers exposed to high levels. Lung irritation, bronchiolitis and emphysema may also occur. Cadmium can replace calcium in the body, leading to weakening of the bones. It is not known whether cadmium can affect infant development, but offspring of pregnant animals exposed to it have shown lower infant birth weight, bone malformations, and learning disabilities. Animals exposed to it have also shown damage to the testes and ovaries. EPA classifies cadmium in Group B1, the probable human carcinogens.

The Agency for Toxic Substances and Disease Registry (ATSDR) states that remote areas have concentrations of less than 0.001 ug/m3 cadmium in air, while urban locations range from 0.003 to 0.040 ug/m3 (ATSDR Toxiological Profile on Cadmium). The mean levels at the three Denver sites fall within the rural range. This may be due to decreased emissions in recent years.

Cobalt

Cobalt is a metal that occurs naturally in the earth's crust. Its chemical symbol is Co. It exists in the atmosphere as particulate matter, in compounds formed from combination with other atoms. Cobalt is used as an additive in metal processing, and also as a pigment in paints and plastics. Cobalt is used to produce jet engines, tools, magnets, grinding tools, and in replacement hip and knee joints for the body.

Emission sources of cobalt include metal producers, coal-fired power plants, wood-burning, mining operations, and incinerators. Cobalt also occurs in vehicular exhaust. Cobalt occurs naturally in some soils, so wind-blown dusts from exposed land can contain it. Soils contaminated by smelter fall-out can also be a source of emissions during high winds.

In small amounts, cobalt is believed to be an essential micronutrient in the human body. It is an important constituent of the vitamin B_{12}. EPA has not classified cobalt with regard to carcinogenicity, due to lack of information. Cobalt has demonstrated health effects including lung irritation, fibrosis, allergic dermatitis, induction of asthma, and irritation of the nasal passages. It also irritates the gastro-intestinal tract. Studies in animals suggest that it can damage sperm, and decrease fertility. It also can damage the kidneys, heart, thyroid, lungs and blood. Effects on blood are not completely understood (NIOSH Occupational Hazard Assessment: Criteria For Controlling Exposure To Cobalt). Cobalt has been used in the past to treat anemia, due to its ability to stimulate production of red blood cells. It should be noted that the adverse health effects have been observed in workers with long-term

exposure to cobalt compounds in industrial settings. These exposures were to concentrations occurring at levels hundreds or thousands of times higher than cobalt levels in outdoor air.

EPA has not established a Reference Concentration for cobalt. The CalEPA has established a chronic reference level based on respiratory effects in animals.

The California Air Resources Board monitored cobalt in 1996. They report a network-wide average of 0.008 ug/m3. The 0.0008 through 0.0010 annual means measured at the three Denver sites are an order of magnitude below the California results. The annual report of the 2003 nationwide UATMP results gives a range of 0.01 – 92.12 ppbv observed throughout the network. The range for the Denver sites was 0.16- 2.59 ppbv.

Manganese

Manganese is a metal that occurs naturally in the earth's crust. Its chemical symbol is Mn. It exists in the atmosphere as particulate matter, in compounds formed from combination with other atoms. Manganese is used as an additive in metal processing and steel production. It is also used in ceramics, matches, glass, dyes, batteries, and as a pigment in paints (California Air Resources Board Fact Sheet on Manganese). It is also employed in wood preservatives. Organic forms of manganese are used as pesticides and for disease prevention in crops such as fruits, vegetables, and cotton.

Emission sources of manganese include petroleum refineries, steel producers, cement producers, coal-fired power plants, wood-burning, metals operations, mining operations, and incinerators. Manganese occurs naturally in some soils, so wind-blown dusts from exposed land can contain it. Soils contaminated by smelter fall-out can also be a source of emissions during high winds.

Manganese is considered an essential micronutrient in the human body. The body tends to regulate manganese concentrations, so oral exposure to small amounts naturally present in food is rarely a problem. Exposure of manganese by inhalation can lead to health effects. Manganese health effects on the respiratory system include lung irritation, chemical pneumonia, cough, and bronchitis. Manganese may damage the central nervous system. The disease known as "manganism", which results from manganese poisoning, includes psychological and nervous system damage. Individuals with manganism have a mask-like face, depression, uncontrollable laughter, and lethargy. The central nervous system effects include trouble with tremors, balance and walking that is similar to that of Parkinson's disease. Central nervous system damage can occur at exposure levels below those that lead to manganism. Examples are decreases in visual reaction time, hand steadiness, and eye-hand coordination. Manganese also affects the gastro-intestinal tract and the kidneys. However, it should be noted that these health effects have been observed in workers with long-term exposure to manganese fumes and dusts in industrial settings. These exposures were at levels hundreds or thousands of times higher than manganese levels in outdoor air.

EPA classifies manganese as Group D, unclassifiable as to carcinogenic potential. This is because there is little evidence to link it to cancer health effects. EPA has established a Reference Concentration for manganese based on an impairment of neurobehavioral function in humans in occupational exposure studies.

The California Air Resources Board monitored manganese in 1996. They report a network-wide average of 0.0212 µg/m3 total manganese (CARB Fact Sheet on Manganese). The 0.047 to 0.053 annual means measured at the Denver sites are about twice the level of the California results. The annual report of the 2003 nationwide UATMP results gives a range of 0.01 – 5138.55 ppbv observed throughout the network. The range for the Denver sites was 6.60- 139.80 ppbv.

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