# FINAL REPORT

# WEST LOUISVILLE AIR TOXICS STUDY

# **RISK ASSESSMENT**

**Prepared for:** 

Metro Louisville Air Pollution Control District and West Jefferson County Community Task Force Louisville, KY

> Prepared by: Sciences International, Inc. 1800 Diagonal Road, Suite 500 Alexandria, VA 22314

> > October 2003

# **TABLE OF CONTENTS**

EXECUTIVE SUMMARY	. vi
1.0 INTRODUCTION	1
1.1 Description of the Monitoring Program	1
1.2 Local Meteorology	
1.3 Organization of This Report	3
2.0 DATA ANALYSIS AND SELECTION OF CHEMICALS OF POTENTIAL	
CONCERN	13
2.1 Louisville Police Firearms Training: WLATS Site 1	14
2.1.1 VOCs	14
2.1.2 SVOCs	
2.1.3 Reactive Aerosols and Metals	15
2.1.4 Pesticides/PCBs	
2.2 Ralph Avenue & Campground Road: WLATS Site 2	
2.2.1 VOCs	16
2.2.2 SVOCs	
2.2.3 Reactive Aerosols and Metals	17
2.2.4 Pesticides/PCBs	
2.3 Old Lake Dreamland Fire Department: WLATS Site 3	19
2.3.1 VOCs	19
2.3.2 SVOCs	19
2.3.3 Reactive Aerosols and Metals	20
2.3.4 Pesticides/PCBs	
2.4 St. Stephen Baptist Church: WLATS Site 4	
2.4.1 VOCs	20
2.4.2 SVOCs	21
2.4.3 Reactive Aerosols and Metals	
2.4.4 Pesticides/PCBs	22
2.5 University of Louisville Shelby Campus: WLATS Site 5	
2.5.1 VOCs	
2.5.2 SVOCs	
2.5.3 Reactive Aerosols and Metals	23
2.5.4 Pesticides/PCBs	24
2.6 Otter Creek Park: WLATS Site 6	24
2.6.1 VOCs	
2.6.2 SVOCs	
2.6.3 Reactive Aerosols and Metals	
2.7 Park DuValle:Southwick Community Center: WLATS Site 7	25
2.8 Farnsley Middle School: WLATS Site 8	
2.9 Chickasaw Park (Private Residence): WLATS Site 9	
2.10 New Lake Dreamland Fire Department: WLATS Site 10	
2.11 M.L. King Elementary School: WLATS Site 11	
2.12 Cane Run Elementary School: WLATS Site 12	29

2.13 SUMMARY OF COPCS	
3.0 EXPOSURE ASSESSMENT	
3.1 Chronic Exposures	
3.2 Acute Exposures	
4.0 HAZARD IDENTIFICATION AND DOSE-RESPONSE ASSESSMENT	
4.1 Chronic Toxicity	
4.1.1 Cancer Effects	
4.1.2 Non-cancer Effects	
4.2 Acute Toxicity	
5.0 RISK CHARACTERIZATION	77
5.1 Risk Characterization for Chronic Exposures	
5.1.1 Louisville Police Firearms Training: WLATS Site No. 1	79
5.1.2 Ralph Avenue & Campground Road: WLATS Site No. 2a	80
5.1.3 Ralph Avenue & Campground Road: WLATS Site No. 2b	80
5.1.4 Old Lake Dreamland Fire Department: WLATS Site No. 3	81
5.1.5 St. Stephen Baptist Church: WLATS Site No. 4	
5.1.6 University of Louisville Shelby Campus: WLATS Site No. 5	82
5.1.7 Otter Creek Park: WLATS Site No. 6	82
5.1.8 Park DuValle:Southwick Community Center: WLATS Site No. 7	82
5.1.9 Farnsley Middle School: WLATS Site No. 8	83
5.1.10 Chickasaw Park (Private Residence): WLATS Site No. 9	
5.1.11 New Lake Dreamland Fire Department: WLATS Site No. 10	84
5.1.12 M.L.King Elementary School: WLATS Site No. 11	85
5.1.13 Cane Run Elementary School: WLATS Site No. 12	
5.2 Acute Risk Characterization	
5.3 Risk Characterization Summary	
6.0 UNCERTAINTY ANALYSIS	
6.1 Monitoring Program	121
6.2 Data Analysis and Selection of Chemicals of Potential Concern	
6.3 Exposure Assessment	123
6.4 Hazard Identification and Dose-Response Assessment	
6.5 Risk Characterization	
7.0 CONCLUSIONS	
7.1 Chronic Risk Characterization	
7.2 Acute Risk Characterization	
8.0 REFERENCES	
GLOSSARY	
Appendix A Applytical Data As Reported	

- Appendix A Analytical Data As Reported
- Appendix B Sampling Events
- Appendix C Data Summaries
- Appendix D Risk Tables
- Appendix E Graphs of Risk Estimates for Risk Drivers
- Appendix F Toxicological Profiles

# LIST OF TABLES

Table 1-1.	WLATS Monitoring Site Locations	4
Table 1-2.	List of Analytes for the WLATS Monitoring Program	5
Table 2-1.	% Detection for Positively Identified Chemicals	31
Table 2-2.	COPCs by Monitor	37
Table 4-1.	Cancer Toxicity Values for COPCs	51
Table 4-2.	Non-cancer Toxicity Values for COPCs	57
Table 4-3.	Non-cancer Critical Effect	63
Table 4-4.	Acute Toxicity Values for COPCs	71
Table 5-1.	Median Cancer Risk Exceedances and WOE	89
Table 5-2.	Median Cancer Risk Exceedances and WOE-VOC Only	92
Table 5-3.	95% UCL Cancer Risk Exceedances and WOE	95
Table 5-4.	95% UCL Cancer Risk Exceedances and WOE-VOC Only	98
Table 5-5.	Median Non-cancer Risk Exceedances and Critical Effect	.101
Table 5-6.	Median Non-cancer Risk Exceedances and Critical Effect-VOC Only	.104
Table 5-7.	95% UCL Non-cancer Risk Exceedances and Critical Effect	.107
Table 5-8.	95% UCL Non-cancer Risk Exceedances and Critical Effect-VOC Only	.110

# LIST OF FIGURES

Figure 1-1.	Locations of all WLATS Monitoring Sites	9
Figure 1-2.	Locations of WLATS Monitoring Sites in the Metropolitan Area	10
Figure 1-3.	Wind Rose Diagram for the Period from April 2000 to December 2000	11
Figure 1-4.	Wind Rose Diagram for the Period from January 2001 to April 2001	12
Figure 5-1.	Median Exposure Case Cancer Risk-All COPCS	113
Figure 5-2.	Median Exposure Case Cancer Risk-VOCs Only	114
Figure 5-3.	95% UCL Exposure Case Cancer Risk-All COPCs	115
Figure 5-4.	95% UCL Exposure Case Cancer Risk-VOCs Only	116
Figure 5-5.	Median Exposure Case Non-cancer Hazard Index-All COPCs	117
Figure 5-6.	Median Exposure Case Non-cancer Hazard Index-VOCs Only	118
Figure 5-7.	95% UCL Exposure Case Non-cancer Hazard Index-All COPCs	119
Figure 5-8.	95% UCL Exposure Case Non-cancer Hazard Index-VOCs Only	120

# **EXECUTIVE SUMMARY**

Between April 2000 and April 2001, the Air Pollution Control District of Jefferson County, now known as the Metro Louisville Air Pollution Control District (MLAPCD), the United States Environmental Protection Agency (USEPA), the Commonwealth of Kentucky, and others worked with the West Jefferson County Community Task Force (WJCCTF) to conduct an air monitoring study of a large number of toxic air pollutants at twelve communities in the West Louisville, Kentucky, area. Designated the West Louisville Air Toxics Study (WLATS), the purpose of the study was to determine if residents of the area were being exposed to airborne concentrations of toxic air pollutants via inhalation that may pose unacceptable risks to human health. Sciences International, Inc. (Sciences) conducted a risk assessment of the air monitoring data collected in the WLATS, and this report details the methods and findings of this assessment.

Analytical data were summarized and chemicals of potential concern (COPCs) were selected at each monitoring location for detailed evaluation in the risk assessment. To be a COPC at a location, a chemical had to be detected in at least 10% of the samples collected at the monitor. All of the monitors in the network included analysis of volatile organic chemicals (VOCs) and a portion of these were selected as COPCs at every monitoring location. At several monitor locations, analysis was also conducted for other chemicals, specifically, semi-volatile organic chemicals (SVOCs), metals, pesticides and PCBs. With the exception of pesticides and PCBs, COPCs were selected from these additional chemicals if the frequency of detection at a location was at least 10%. The pesticides and PCBs were sampled on a single day and were not considered in the chronic risk assessment because the single sample may not be representative of the true chronic exposures at a location.

Only exposures via inhalation were evaluated, with risks calculated on a location-specific basis for individuals that may reside within each of the WLATS monitoring areas. Both chronic (long-term) and acute (short-term) exposures were evaluated. The chronic exposures assumed an individual was exposed to the air concentrations continuously for 24 hours per day over a 70 year period. Two exposure cases were evaluated in the chronic risk assessment. The median exposure case was based on using the median chemical concentration in air for each of the COPCs. The 95% UCL exposure case was based on using the 95% UCL on the mean of the chemical concentrations in air at a given monitor, or the maximum chemical concentration in air if it was less than the 95% UCL on the mean. For the acute risk assessment, maximum air concentrations at each monitor location were compared to the toxicity criteria to determine the potential human health impact. Toxicity criteria were derived from toxicology reviews conducted by the USEPA and other state, federal and international agencies and organizations.

The results for the chronic risk assessment indicated that all of the monitors in the WLATS monitoring program, including the background monitors, exceeded a  $1 \times 10^{-6}$  lifetime cancer risk. The State of Kentucky uses a  $1 \times 10^{-6}$  threshold to identify

acceptable risks. A total of 15 chemicals exceeded this threshold under the median exposure case, and a total of 17 chemicals exceeded this threshold for the 95% UCL exposure case. For the median exposure case, when looking at VOCs only, the cancer risks ranged from a high of  $1.1 \times 10^{-4}$ , at Site 3, to a low of  $1.4 \times 10^{-5}$  at Site 6. The median cancer risks for all COPCs ranged from a high of  $1.8 \times 10^{-4}$  at Site 2b to a low of  $3.8 \times 10^{-5}$  at Site 11. For the 95% UCL exposure case, the cancer risks for VOCs only ranged from a high of  $6.0 \times 10^{-4}$  at Site 2b, to a low of  $1.8 \times 10^{-5}$  at Site 6. When looking at all COPCs, the cancer risk for the 95% UCL exposure case ranged from a high of  $6.9 \times 10^{-4}$  at Site 2b, to a low of  $7.6 \times 10^{-5}$  at Site 6.

The non-cancer health impacts were evaluated by calculating a hazard quotient (HQ) for each COPC, and then summing the HQs at a location to determine the overall impact in the form of a Hazard Index (HI). If the value of the HQ is less than 1, then an adverse health impact from the exposure is unlikely. Similarly, if the HI for a monitor location is below a value of 1, then the cumulative impact from all of the COPCs is unlikely to result in an adverse health impact. For the median exposure case, non-cancer HIs ranged from a high of 1.09 at Site 2b to a low of 0.07 at Site 6, for VOCs only. For all COPCs, the median exposure case HI ranged from a high of 1.73 at Site 2b, to a low of 0.19 at Site 11. For the 95% UCL exposure case, the HIs for VOCs only ranged from a high of 8.58 at Site 2b, to a low of 0.09 at Site 6. For all COPCs, the HI for the 95% UCL exposure case ranged from a high of 9.43 at Site 2b, to a low of 0.5 at Site 6. While several HIs exceeded a value of 1, these instances were due to the concentrations of 1,3-butadiene, which was the only COPC to have an HQ that exceeded a value of 1, and only for the 95% UCL exposure case. Thus there is a potential for adverse health impacts based on the air concentrations of 1,3-butadiene for the 95% UCL exposure case.

For the residential monitors, the VOC only cancer risk for the median exposure case ranged from  $1.1 \text{ x}^{-4}$  at Site 3, to  $3.8 \text{ x}^{-5}$  at Site 11. For the VOC only case, the median cancer risk at all residential monitors were higher than for both background monitors. When looking at residential monitors for all COPCs, the median cancer risks ranged from  $1.7 \times 10^{-4}$  at Site 3 to  $3.8 \times 10^{-5}$  at Site 11. The median exposure case cancer risk estimates at four of the residential monitors was lower than for the background monitors, however, these four residential monitors did not include VOC analysis, whereas the background monitors did include risks from SVOCs and metals. For the 95% UCL exposure case cancer risks for VOCs at residential monitors only ranged from  $3.9 \times 10^{-4}$  at Site 3 to  $1.3 \times 10^{-4}$  at Sites 9 and 10. The residential risk range for all COPCs was from  $4.0 \times 10^{-4}$  at Site 3, to  $1.3 \times 10^{-4}$  at Sites 9 and 10. The median cancer risk estimates for all residential monitors were higher than either background monitor when looking at both VOCs only and for all COPCs.

For the median non-cancer health impacts at residential monitors, the HI for VOCs only ranged from 0.67 at Site 3 to 0.19 at Site 11, while the HI for all COPCs ranged from 1.17 at Site 3 to 0.19 at Site 11. When looking at VOCs only, the median exposure case HI for all residential monitors was greater than for the background monitors. When looking at all COPCs, the median exposure case HI for several residential monitors was below the background monitors, but unlike the background monitors, these residential monitors had

data for VOCs only. The results of the median non-cancer risk evaluation for the residential monitors indicate that adverse health impacts are unlikely. For the 95% UCL exposure case the HI ranged for residential monitors ranged from a high of 4.82 at Site 7 to a low of 0.47 at Site 4 when looking at VOCs only. For all COPCs, the 95% UCL exposure case HI at residential monitors ranged from a high of 4.82 at Site 7 to a low of 1.06 at Site 9. A comparison of the 95% UCL exposure case HI for the background monitors versus the residential monitors shows that the HIs at all residential monitors exceeds the HIs for the background monitors when looking at both VOCs only and for all COPCs. The results of the 95% UCL exposure case for non-cancer health impacts indicate a potential for adverse health impacts at three monitors location (Sites 3, 7 and 8) due to exposure to 1,3-butadiene. It should be noted that the 95% UCL exposure case will likely overestimate the true risk to the general population for reasons that are discussed in the risk assessment.

The acute risk characterization was conducted by calculating an HQ for each of the COPCs at a monitor location. The calculation of an HI for each monitor location by summing the individual HQs is not appropriate for the acute analysis. The HQs for the acute risk characterization did not exceed a value of 1 for any of the COPCs, indicating that an adverse health impact is not likely for the acute exposures.

# **1.0 INTRODUCTION**

Between April 2000 and April 2001, the Air Pollution Control District of Jefferson County, now known as the Metro Louisville Air Pollution Control District (MLAPCD), the United States Environmental Protection Agency (USEPA), the Commonwealth of Kentucky, and others worked with the West Jefferson County Community Task Force (WJCCTF) to conduct an air monitoring study of a large number of toxic air pollutants in a number of communities in the West Louisville, Kentucky, area. Designated the West Louisville Air Toxics Study (WLATS), the purpose of the study was to determine if residents of the area were being exposed to airborne concentrations of toxic air pollutants via inhalation that may pose unacceptable risks to human health.

Sciences International, Inc. (Sciences) conducted a risk assessment of the air monitoring data collected in the WLATS, using as primary guidance the WLATS Risk Assessment Work Plan and Quality Assurance Project Plan (WLATS Work Plan) provided to Sciences. This report presents the methodologies and findings of the risk assessment.

# 1.1 Description of the Monitoring Program

The WLATS monitoring program was designed to collect ambient air data that characterized the airborne concentrations of toxic air pollutants in residential areas of West Louisville. Twelve monitoring sites were selected for the study. Table 1-1 identifies the name, location and other characteristics of the monitoring locations in this study, and Figure 1-1 depicts their general geographic location throughout the study area. Figure 1-2 provides a close-up of the locations of the monitors in the West Louisville area. Each of the 12 monitoring sites was selected to represent a different, unique area at which exposure to airborne chemicals can occur to residents. Residential locations were selected for monitoring because exposure estimates for residential populations are typically greater than those in non-residential populations due to the fact that residents could potentially be present and exposed at a given location 24 hours per day. Responsibility for the monitors and the laboratory analysis was split between the USEPA Region IV, and the University of Louisville.

A total of 15 monitors were used in the WLATS network. At 11 of the 12 monitoring locations, a single monitor was used. At the Ralph Avenue & Campground Road location, two sets of duplicate monitors (i.e., four monitors in total) were used. The University of Louisville was responsible for one set of collocated monitors, and the USEPA was responsible for the other. Monitoring at all locations was conducted over a one-year period. To account for potential seasonal and temporal variability in air concentrations, the monitoring program was designed to collect 24-hour samples every twelfth day, resulting in approximately 30 sampling events at each location.

A complete list of all the chemicals that were included in the monitoring program is presented in Table 1-2. Volatile organic chemicals (VOCs) were the principal chemicals of interest in the WLATS, and thus were analyzed for at all monitoring locations and

during every monitoring event. Additionally, at the six monitor locations operated by the USEPA (i.e., WLATS site numbers 1 through 6), semi-volatile organic compounds (SVOCs), metals, and reactive aerosols were also routinely monitored. In addition, a single sampling event was conducted for pesticides and polychlorinated biphenyls (PCBs) at five of the USEPA-operated monitoring locations (i.e., WLATS site numbers 1 through 5) August 28, 2000. Additionally, in the laboratory an attempt was made to identify other organic chemicals present in air that are not included in the standard lists of VOCs and SVOCs. These chemicals are termed tentatively identified compounds (TICS).

# **1.2 Local Meteorology**

The local meteorology in the Louisville area has a significant influence in determining where chemicals in the atmosphere are carried and their airborne concentrations. Wind speed and wind direction are two of the most important meteorological factors. Airborne chemicals are carried along in the direction that the wind is blowing. In general, as wind speeds increase the airborne concentrations will decrease due to more air being available to mix with the chemicals and dilute their concentrations. In a developed area like the location of the WLATS monitors, the vertical obstacles to air flow such as buildings and trees increase the mixing in the atmosphere as the wind goes over and around the obstacles. The atmospheric stability is another critical meteorological factor in determining the amount of mixing that can occur in the air. On hot sunny days, the sun's energy warms the air in contact with the earth's surface and this warm air then rises upwards, leading to what is called an unstable atmosphere. The rising air causes increased motion in the atmosphere, which increases the amount of air that is available to dilute chemicals in the atmosphere. In contrast, on cloudless nights with low winds, the air near the earths surface cools faster than the air above, which leads to a stable atmospheric condition where vertical motion and resulting dilution of chemicals in the atmosphere is limited.

A wind rose provides a graphical display of the percentage of times the wind blows in a particular direction, and the range of wind speeds in each direction. This information is useful to interpreting the results of a monitoring program as it the shows the direction from which chemicals found in a monitor may have come. However, it contains no information about the atmospheric stability and thus the wind rose cannot be used alone as a means to identify the source of chemicals in a particular monitor. Typically, this is done using air modeling that includes among other things, both the information displayed in the wind rose, as well as information on the atmospheric stability. For informational purposes, a wind rose has been developed for the Louisville area for the time during the monitoring program (April 2000 to April 2001). This wind rose was based on data collected at the National Weather Service (NWS) station at the Louisville International Airport. The NWS station is approximately five miles or less to the southeast of the WLATS community monitoring locations. The western portion of the Louisville area, where the NWS station, the community monitors and much of the local industry is located, is relatively flat. Thus the wind directions and speeds seen at the NWS station

are likely to be the same as what was seen in the area of the WLATS community monitors and the industrial areas.

The wind rose for the Louisville area during the period of April 2000 to December 2000 is presented in Figure 1-3. The wind rose for the period from January 2001 through April 2001 is presented in Figure 1-4. Overall the winds generally are from the west, meaning that airborne chemicals will move from west to east across the WLATS monitoring grid.

# **1.3 Organization of This Report**

The remainder of this report is organized into six principal sections:

- Section 2 presents an analysis of the monitoring data and selects chemicals of potential concern (COPCs) for evaluation in the risk assessment.
- Section 3 outlines the assumptions and methods used to calculate exposure concentrations at each monitoring location.
- Section 4 characterizes the types of health effects potentially associated with each of the COPCs and identifies the toxicity criteria used to assess risks.
- Section 5 summarizes and discusses the risk assessment results for each of the monitoring locations.
- Section 6 summarizes important sources of uncertainty in this assessment and their potential impact on the risk estimates.
- Section 7 presents the conclusions of the risk assessment.

References are provided in Section 8. A Glossary follows the References. The appendices provide supporting detail for the risk assessment, including the validated analytical data, detailed monitor-specific data summaries and risk calculation results, graphical presentations of risk estimates and brief toxicity profiles for chemicals that were risk drivers.

Site Number	Name and Location	
		Comments
1	Louisville Police Firearms Training 4201 Algonquin Parkway Louisville, KY 40211 38° 13' 37.0" N; 85° 49' 24.0" W	Maximum impact site; fenceline monitoring for oil terminals. Co- located with weather station.
2	Ralph Avenue & Campground Road 4211 Campground Road Louisville, KY 40216 38° 12' 40.3" N; 85° 50' 26.2" W	Maximum impact and neighborhood population exposure site. QA/QC site.
3	Old Lake Dreamland Fire Department 4603 Campground Road Louisville, KY 40216 38° 12' 19.1" N; 85° 51' 9.5" W	Neighborhood population exposure site.
4	St. Stephen Baptist Church 1008 S. 15 <sup>th</sup> Street Louisville, KY 40210 38° 14' 32.0" N; 85° 46' 39.8" W	Neighborhood population exposure site.
5	University of Louisville, Shelby Campus 9001 Shelbyville Road Louisville, KY 40222 38° 13' 9.0" N; 85° 34' 59.0" W	Anthropogenic (i.e., man made) urban activity control site near major traffic corridor, considered an urban background monitor.
6	Otter Creek Park (Meade County) 850 Otter Creek Park Road Brandenburg, KY 40108 37° 56' 51.5" N; 85° 2' 34.8" W	Background site in public park 25 miles southwest of study area in predominantly upwind direction.
7	Park DuValle/Southwick Community Center 3621 Southern Avenue Louisville, KY 40211 38° 13' 49.6" N; 85° 49' 11.8" W	Neighborhood population exposure site.
8	Farnsley Middle School 3400 Lees Lane Louisville, KY 40216 38° 11' 3.2" N; 85° 50' 45.8" W	Neighborhood population exposure site.
9	Chickasaw Park (private residence) 942 S. 47 <sup>th</sup> Street Louisville, KY 40211 38° 14' 44.7" N; 85° 49' 58.1" W	Neighborhood population exposure site.
10	New Lake Dreamland Fire Department 4603 Cane Run Road Louisville, KY 40216 38° 11' 35.5" N; 85° 50' 45.8" W	Neighborhood population exposure site.
11	M.L. King Elementary School 4325 Vermont Avenue Louisville, KY 40210 38° 15' 26.3" N; 85° 49' 46.8" W	Neighborhood population exposure site.
12	Cane Run Elementary School 3951 Cane Run Road Louisville, KY 40222 38° 12' 34.8" N; 85° 49' 20.9" W	Neighborhood population exposure site.

Table 1-1WLATS Monitoring Site Locations

# Table 1-2List of Analytes for the WLATS Monitoring Program

#### Volatile Organic Compounds (VOCs) (Analyzed by EPA and U of L)

(m - and/or p-)Xylene
1,1,1,2-Tetrachloroethane
1,1,1-Trichloroethane
1,1,2,2-Tetrachloroethane
1,1,2-Trichloro-1,2,2-Trifluoroethane (Freon 113)
1,1,2-Trichloroethane
1,1-Dichloroethane
1,1-Dichloroethene (1,1-Dichloroethylene)
1,1-Dichloropropene
1,2,3-Trichlorobenzene
1,2,3-Trichloropropane
1,2,4-Trichlorobenzene <sup>1</sup>
1,2,4-Trimethylbenzene
1,2-Dibromo-3-Chloropropane (DBCP)
1,2-Dibromoethane (EDB)
1,2-Dichlorobenzene
1,2-Dichloroethane
1,2-Dichloropropane
1,2-Dichlorotetrafluoroethane (Freon 114)
1,3,5-Trimethylbenzene
1,3-Butadiene
1,3-Dichlorobenzene
1,3-Dichloropropane
1,4-Dichlorobenzene
2,2-Dichloropropane
2-Chloro-1,3-Butadiene (Chloroprene)
Acetone
Acrylonitrile
Benzene
Bromobenzene
Bromochloromethane <sup>2</sup>
Bromodichloromethane
Bromoform
Bromomethane
Butyl Acrylate
Carbon Disulfide
Carbon Tetrachloride
Chlorobenzene
Chlorodifluoromethane (Freon 22)
Chloroethane
Chloroform

Chloromethane Cis-1.2-Dichloroethene Cis-1,3-Dichloropropene Cyclohexane Dibromochloromethane Dibromomethane Dichlorodifluoromethane (Freon 12) Ethyl Acrylate Ethyl Benzene Hexachloro-1,3-Butadiene Hexane Isopropylbenzene (Cumene) Methyl Acetate Methyl Butyl Ketone Methyl Ethyl Ketone Methyl Isobutyl Ketone Methyl Methacrylate Methyl t-Butyl Ether (MTBE) Methylcyclohexane Methylene Chloride Naphthalene<sup>1</sup> n-Butylbenzene n-Propylbenzene o-Chlorotoluene o-Xylene p-Chlorotoluene p-Isopropyltoluene Sec-Butylbenzene Styrene Tert-Butylbenzene Tetrachloroethene (Tetrachloroethylene) Toluene Trans-1,2-Dichloroethene Trans-1,3-Dichloropropene Trichloroethene (Trichloroethylene) Trichlorofluoromethane (Freon 11) Vinyl Chloride

<sup>1</sup> Also included on SVOC list. EPA analyzed these compounds by both methods. SVOC data used in the risk assessment.

 $^{\rm 2}$  Not analyzed by U of L

# Table 1-2 (con't)List of Analytes for the WLATS Monitoring Program

#### Semivolatile Organic Compounds (SVOCs) (Analyzed by EPA Only)

Formaldehyde (3-and/or 4-)Methylphenol 1,1-Biphenyl 1,2,4-Trichlorobenzene 2,3,4,6-Tetrachlorophenol 2,4,5-Trichlorophenol 2,4,6-Trichlorophenol 2,4-Dichlorophenol 2,4-Dimethylphenol 2,4-Dinitrophenol 2,4-Dinitrotoluene 2,6-Dinitrotoluene 2-Chloronaphthalene 2-Chlorophenol 2-Methyl-4,6-Dinitrophenol 2-Methylnaphthalene 2-Methylphenol 2-Nitroaniline 2-Nitrophenol 3,3'-Dichlorobenzidine 3-Nitroaniline 4-Bromophenyl Phenyl Ether 4-Chloro-3-Methylphenol 4-Chloroaniline 4-Chlorophenyl Phenyl Ether 4-Nitroaniline 4-Nitrophenol Acenaphthene Acenaphthylene Acetophenone Anthracene Atrazine Benzaldehyde Benzo(a)anthracene Benzo(b)fluoranthene Benzo(ghi)perylene Benzo(k)fluoranthene Benzo-a-pyrene Benzyl Butyl Phthalate Bis(2-Chloroethoxy)Methane Bis(2-Chloroethyl) Ether

Bis(2-Chloroisopropyl) Ether Bis(2-Ethylhexyl) Phthalate Caprolactam Carbazole Chrysene Dibenzo(a,h)anthracene Dibenzofuran Diethyl Phthalate **Dimethyl Phthalate** Di-n-butylphthalate Di-n-octylphthalate Fluoranthene Fluorene Hexachlorobenzene (HCB) Hexachlorobutadiene Hexachlorocyclopentadiene (HCCP) Hexachloroethane Indeno (1,2,3-cd) Pyrene Isophorone Naphthalene Nitrobenzene N-Nitrosodi-n-Propylamine N-Nitrosodiphenylamine/diphenylamine Pentachlorophenol Phenanthrene Phenol Pyrene

# Table 1-2 (con't)List of Analytes for the WLATS Monitoring Program

Analyzed by EPA Only)	
analyzeu by El A Only)	
luminum	
ntimony	
rsenic	
arium	
eryllium	
admium	
alcium	
hromium	
obalt	
opper	
on	
ead	
Iagnesium	
Ianganese	
Iolybdenum	
ïckel	
otassium	
elenium	
ilver	
odium	
trontium	
hallium	
in	
itanium	
anadium	
ttrium	
inc	

#### Reactive Aerosols (Analyzed by EPA Only)

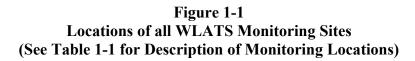
HCL (Calculated from Cl) HF (Calculated from F)

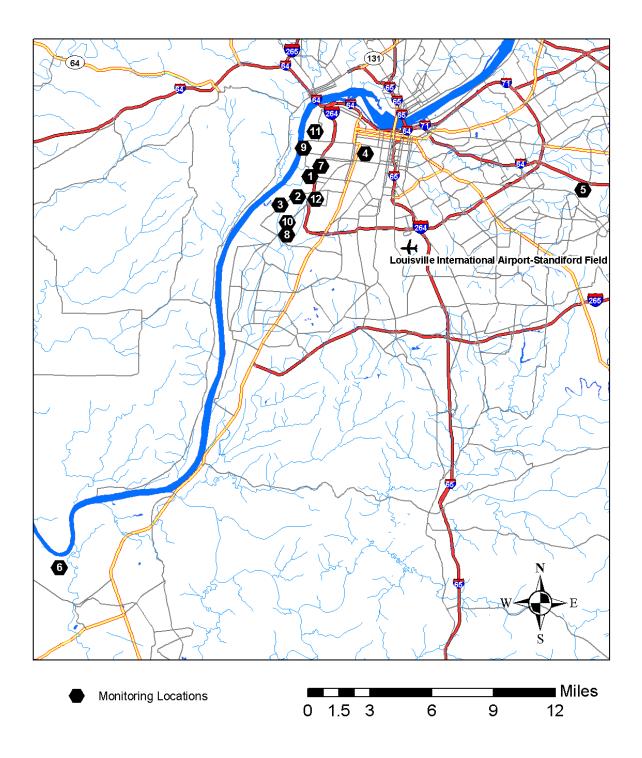
# Table 1-2 (con't)List of Analytes for the WLATS Monitoring Program

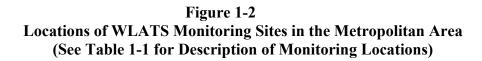
(Analyzed by EPA Only)		
4,4'-DDD (p,p'-DDD)	PCB Congener #201	
4,4'-DDE (p,p'-DDE)	PCB Congener #203	
4,4'-DDT (p,p'-DDT)	PCB Congener #206	
Aldrin	PCB Congener #208	
Alpha-BHC	PCB Congener #209	
Alpha-Chlordane	PCB Congener #28	
Alpha-Chlordene	PCB Congener #52	
Beta-BHC	PCB Congener #60	
Beta-Chlordene	PCB Congener #66	
Chlordene	PCB Congener #74	
Cis-Nonachlor	PCB Congener #77	
Delta-BHC	PCB Congener #81	
Dieldrin	PCB Congener #99	
Endosulfan I (alpha)	PCB-1016 (Aroclor 1016)	
Endosulfan II (beta)	PCB-1221 (Aroclor 1221)	
Endosulfan Sulfate	PCB-1232 (Aroclor 1232)	
Endrin	PCB-1242 (Aroclor 1242)	
Endrin Ketone	PCB-1248 (Aroclor 1248)	
Gamma-BHC (Lindane)	PCB-1254 (Aroclor 1254)	
Gamma-Chlordane	PCB-1260 (Aroclor 1260)	
Gamma-Chlordene	PCB-1268 (Aroclor 1268)	
Heptachlor	Toxaphene	
Heptachlor Epoxide	Trans-Nonachlor	
Methoxychlor		
Oxychlordane (Octachlorepoxide)		
PCB Congener #101		
PCB Congener #105		
PCB Congener #118		
PCB Congener #126		
PCB Congener #138		
PCB Congener #153		
PCB Congener #156		
PCB Congener #163		
PCB Congener #169		
PCB Congener #170		
PCB Congener #180		
PCB Congener #183		
PCB Congener #187		
PCB Congener #194		
PCB Congener #195		

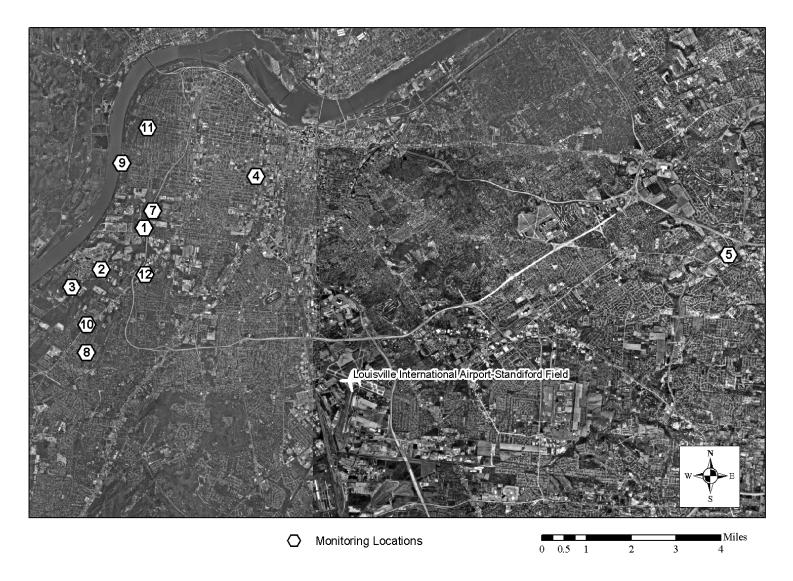
PCB Congener #196

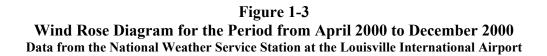
Pesticides/PCBs

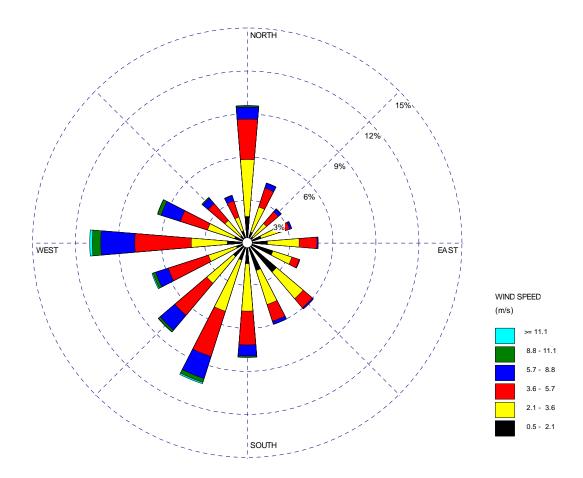


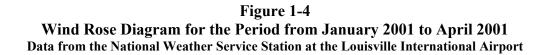


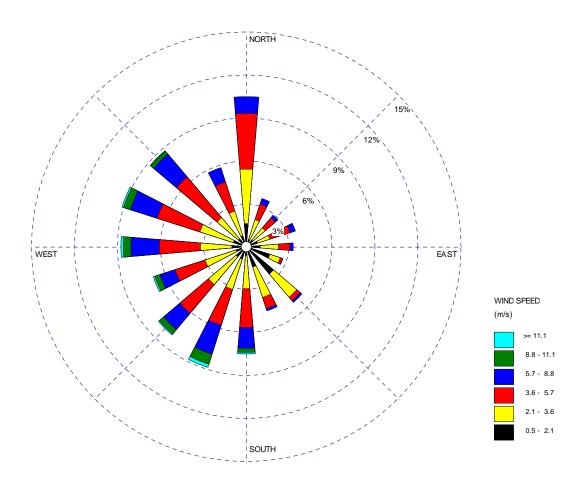












# 2.0 DATA ANALYSIS AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN

This Section summarizes the analytical data collected during the WLATS monitoring program and selects chemicals of potential concern for detailed analysis in the risk assessment. For the risk assessment, each monitor location will be evaluated separately, so the data analysis and selection of chemicals of potential concern (COPCs) is presented individually for each monitor. Data are summarized for each monitor by identifying the chemicals that were detected at least once at a level above the sample quantitation limit (SQL) and calculating the frequency of detection. Consistent with the protocols outlined in the WLATS Work Plan, all chemicals that were detected in 10% or more of the samples were selected as COPCs. The COPCs are then carried forward for further evaluation in the risk assessment. No tentatively identified compounds (TICs) were selected as COPCs given the uncertainties in the identity of these compounds. Appendix C summarizes the data for TICs.

The remainder of this Section provides a description of the monitoring activities at each of the WLATS monitoring program sites and identifies the chemicals that were detected and the associated detection frequency. Data for WLATS Site 1 is presented first, followed by Sites 2 through 12. For the collocated monitors at the Ralph Avenue & Campground Road location, only the data for the USEPA monitors was included in the analysis, as per the Work Plan. The analytical results for samples obtained by the USEPA at the collocated monitors are discussed separately, rather than as a combined average result. This has been done to preserve the ability to characterize the uncertainty in the monitoring program, and to ultimately determine if the two monitors would yield differences in the exposure point concentrations or COPCs. In all subsequent discussions, these two monitors will be designated as 2a and 2b, where the 2 identifies the Ralph Avenue & Campground Road location, following the designations provided by WLATS.

Data summaries for VOCs are presented first, followed by the data for non-VOC chemicals for the monitors that had evaluated a larger number of chemicals. The data from the single monitoring event conducted for pesticides and PCBs are not representative of chronic exposures that were the focus of this risk assessment, but are nevertheless summarized here for completeness and to support the evaluation of acute exposures to the extent possible.

The Section concludes with a summary of the COPCs selected for this assessment, followed by two tables. Table 2-1 presents a list of all chemicals that were detected at least once in any of the 13 WLATS monitors evaluated. Table 2-2 provides a list of the COPCs for each of the WLATS monitors evaluated. The difference between Table 2-1 and Table 2-2 is that chemicals that were detected in less than 10% of the samples collected at a monitor were not selected as a COPC for the monitor.

Appendix C presents detailed summaries of the analytical data for each monitoring site.

# 2.1 Louisville Police Firearms Training: WLATS Site 1

This site was defined as a maximum impact site for the monitoring program. In addition to sampling for VOCs, samples also were collected for SVOCs, metals, reactive aerosols and pesticides/PCBs analysis.

#### 2.1.1 VOCs

Sample results were provided for 29 of the 32 sampling dates during the monitoring period. However, only 28 sample dates were reported for some chemicals. The missing sampling events are unlikely to affect the ability of the monitor to reflect chronic conditions given that every month had at least one sample date reported. A total of 36 VOCs were detected in at least one sample date from this monitor. Twenty-two of the chemicals detected were found in at least 50% of the samples collected.

In general, the chemicals detected at the site were also found in the majority of the other monitors throughout the network. This is especially the case for chemicals that were detected at a high frequency at this site. For example, six chemicals detected in every sample date reported for this monitor were also detected in all other monitors as well. Similarly, eight other chemicals detected in all but one of the sample dates reported for this site also were detected in at least eleven of the WLATS monitors.

This monitor yielded the highest recorded concentrations in the monitoring program for the following four chemicals: methyl ethyl ketone; methylcyclohexane; methyl isobutyl ketone; and, 1,3,5-trimethylbenzene. This monitor also had detections of naphthalene as a VOC during the monitoring program, but these data will be disregarded in this risk assessment in favor of naphthalene SVOC analysis, which yielded a lower analytical detection limit.

Of the 36 VOCs detected at this site, only six chemicals were detected in less than 10% of the valid sample dates reported for this monitor location. Thus, a total of 30 VOCs were selected as COPCs for the site.

#### 2.1.2 SVOCs

SVOC results were reported for 26 sampling dates during the monitoring period. The number of samples dates reported for individual chemicals ranged from 23 to 26. The missing sample dates are unlikely to affect the ability of the monitor results to reflect chronic exposures as every month, except April 2000, had at least one sample date reported. A total of 21 SVOCs were detected at least once at this site. All but two of these 21 chemicals were detected in at least four of the seven monitors used for SVOC sampling. Just over half of the chemicals detected at this site were found in more than 50% of the samples reported.

Three chemicals were detected in all valid samples reported for this site (i.e., 2-methylnaphthalene, formaldehyde, and naphthalene). These three chemicals also were detected in all of the other SVOC monitors; however, the highest concentrations recorded during the monitoring program occurred at this site. Four other chemicals also exhibited their maximum concentrations at this site during the monitoring program (i.e., dibenzofuran, fluorene, acenapthene, and 4-nitrophenol).

Of the 21 SVOCs detected at this monitoring location, only five chemicals were detected in less than 10% of the valid samples. Thus, a total of 16 SVOCs were selected as COPCs for this site. The chemicals detected in less than 10% of the samples reported were detected in at least two and typically most of the other SVOC monitor locations.

#### 2.1.3 Reactive Aerosols and Metals

Monitoring for metals was reported for 25 sampling dates during the program; however, data for any one chemical was reported for only 24 samples dates. A total of 25 metals were detected at this site, and 16 of these were detected in more that 50% of the samples. All of the metals detected at this site also were detected in four or more of the other monitoring sites. Eight metals were found in all of the samples and three more chemicals were found in all but one of the samples. Nine of the metals detected during the program exhibited their highest concentration at this site (i.e., antimony, barium, calcium, iron, lead, potassium, strontium, thallium, and titanium), with strontium also detected at the maximum value at monitor 2a. Only one chemical of those detected, silver, was found in less than 10% of the sample dates reported for this site. Thus, a total of 24 metals were selected as COPCs for this monitoring location.

HCl was detected in a single sample and was the only reactive aerosol detected at this monitoring site. Across all sites, a total of five monitors at four different sites had a positive detection for HCl. Given that HCl was detected in less than 10% of the samples, it was not selected as a COPC for this monitoring location.

#### 2.1.4 Pesticides/PCBs

This is one of six monitors where pesticide and PCB samples were collected and analyzed from a single monitoring event. A total of 16 chemicals were detected, three of which were PCB congeners. All of these chemicals also were detected at another monitor, with ten of them found in at least four of the other monitors. The maximum concentrations for six of the pesticides/PCBs detected during the WLATS monitoring program were found at this site (i.e., 4,4'-DDT, alpha-BHC, endosulfan, heptachlor, heptachlor epoxide, and PCB congener # 60). These six chemicals were also found in at least four monitors, except for alpha-BHC, which was only found here and the monitor at the University of Louisville Shelby Campus.

# 2.2 Ralph Avenue & Campground Road: WLATS Site 2

This monitor was situated in what was hypothesized to be an area of maximum impact. It also served as a neighborhood exposure site. As discussed earlier, this site served a quality assurance/quality control (QA/QC) purpose by collecting duplicate samples from monitors located side-by-side. To support this QA/QC role, the sampling results for each class of chemical will be discussed first for one monitor (designated 2a), and then the other (designated 2b). Both the USEPA and the University of Louisville operated collocated monitors at this site, however as per the Risk Assessment Work Plan, only the data reported by the USEPA was evaluated.

### 2.2.1 VOCs

Monitoring results for 2a were available for all 32 sampling events in the monitoring program. A total of 33 VOCs were detected at this monitor, with almost two-thirds of the chemicals detected in more than 50% of the sampling events. Four chemicals were found in all of the samples at this site, and two more chemicals were found in all but one of the reported samples. The chemicals found at site 2a were not unique to the monitoring network, with 26 of the 33 VOCs detected at this site also found in 11 or more of the monitors. Two of the VOCs detected here, 2-chloro-1,3-butadiene (chloroprene), and chlorodifluoromethane (Freon 22), were at the highest concentrations found in the monitoring program, with chlorodifluoromethane also detected at maximum concentration at monitor 2b. Three chemicals were found in less than 10% of the site samples; however, they were detected in six of the monitors. A total of 30 VOCs were selected as COPCs for this monitoring location.

Monitor 2b had a total of 30 sampling events reported during the monitoring program; however, a maximum of 29 samples dates were reported for any single chemical detected. The missing sample dates should not affect the ability of this monitor to reflect chronic exposures given that all months had at least one sample date reported for each month during the monitoring period. A total of 40 VOCs were detected at monitor 2b, with just over half (23) detected in more than 50% of the samples reported. Six chemicals were found in all of the samples reported. For VOCs detected in multiple monitors, including both USEPA monitors at this site, 12 of these chemicals exhibited their maximum concentration at monitor 2b (i.e., 1,3-butadiene, acetone, acrylonitrile, bromodichloromethane, butyl acrylate, chlorodifluoromethane, chloroform, ethyl acrylate, methyl methacrylate, methyl t-butyl ether (MTBE), sec-butylbenzene, and trans-1,3-dichloropropene), with chlorodifluoromethane also detected at the same maximum concentration at monitor 2a. A total of ten chemicals were detected in less than 10% of the samples reported; seven of these were not detected in monitor 2a. Three of these seven chemicals were detected only at monitor 2b (i.e., bromodichloromethane, butyl acrylate, and trans-1,3-dichloropropene). For VOCs at site 2b, a total of 30 COPCs were selected for evaluation in the risk assessment.

A comparison of the VOC results for monitors 2a and 2b shows that they are in general agreement despite the disparity in the number of sampling events available for the

monitoring period. Although seven more chemicals were detected in monitor 2b, again, three of these chemicals were not detected at any other monitors in the network.

# 2.2.2 SVOCs

There were 30 sampling events reported for SVOCs at monitor 2a during the monitoring program. The two missing events were on the first two dates of the monitoring program, and should not affect the ability of the monitor results to reflect chronic conditions. A total of 25 SVOCs were detected; however, only nine of these chemicals were detected in more than 50% of the samples, and just two chemicals were detected in all of the samples. Although the frequency of detection was low for most of the chemicals, they were not unique to this monitor as most were found in at least five or more of the other SVOC monitors. The exception was diethyl phthalate, which was detected only at monitor 2a, and in only one of the 30 samples. Three other chemicals were detected in less than 10% of the samples for monitor 2a; however, they were also detected in at least one other monitor. Six other SVOCs besides diethyl phthalate were found at their highest concentration in this monitor (i.e., acetophenone, bis(2-ethylhexyl) phthalate, di-nbutylphthalate, nitrobenzene, pentachlorophenol, and pyrene), with pyrene also at the maximum concentration at monitor 2b. All six of these chemicals were detected at other monitoring sites as well. A total of 21 SVOCs were selected as COPCs for this monitoring location.

Monitor 2b had 25 SVOC sampling events reported. As was the case for monitor 2a, the first two dates of the monitoring program were missing. After that, the missing dates are preceded and followed by a valid measurement. Because at least one sample was reported for every month during the monitoring period, the results should provide a reasonable assessment of the chronic exposure. Twenty-three SVOCs were detected at this monitor, with most (14) detected in less than 50% of the samples. All but two of the SVOCs detected at this monitor were found in four or more other monitors. Only three of the SVOCs were detected in all of the samples and one more was found in all but one of the samples. For SVOCs found at more than one monitor, five of them exhibited their highest concentration at monitor 2b (i.e., 4-chloroaniline, acenaphthylene, anthracene, carbazole and pyrene), with pyrene a maximum at monitor 2a also. Six of the SVOCs at this monitor were detected in all of the samples. For these six infrequently detected chemicals, four were detected in at least four other monitors, one was found only at monitors 2a and 2b (i.e., anthracene), and carbazole was found only in monitor 2b. A total of 17 SVOCs were selected as COPCs for this monitoring location.

## 2.2.3 Reactive Aerosols and Metals

For site 2a, a total of 27 sampling events were reported; however, no more than 26 sample dates were reported for a single chemical. A total of 26 metals were detected at the monitor, with 18 of these detected in more than 50% of the samples, and nine detected in all of the samples. Most of the metals detected at this monitor were also found in all of the other monitors (i.e., 21 of 26) and all 26 were found in at least four monitors. Five of the metals were detected at the highest concentration found in the

monitoring program (i.e., chromium, magnesium, selenium, sodium, and strontium), with strontium also a maximum at monitor 1. Only two of the 26 metals detected at this monitor were found in less than 10% of the samples, leaving 24 metals as COPCs for further evaluation in the risk assessment.

Of the reactive aerosols included in the monitoring program, only HCl was detected, being reported in three samples. A total of five monitors at four different sites had at least one positive detection for HCl.

For monitor 2b, a total of 25 sampling events were reported. The first two dates of the monitoring program are missing, then a sample is reported and then two more sampling dates in a row were missing. The other three missing dates are preceded and followed by a valid sample. Despite the missing sampling dates, the monitor results should provide a reasonable estimate of the chronic exposure given that all months of the monitoring program had at least one sample reported. A total of 26 metals were detected at this site, with 17 of the 26 detected in more than 50% of the samples, with nine found in 100% of the samples, and 5 more found in all but one of the samples. All but five of the metals detected at this monitor were found in all of the other monitors used for metals analysis, and all of them were detected in at least four monitors. Four of the chemicals detected at this monitor were at the highest concentration found in the monitoring program (i.e., aluminum, beryllium, tin, and vanadium). Two of the 26 metals were selected as this site were found in less than 10% of the samples, thus a total of 24 metals were selected as COPCs for monitor 2b.

Again, HCl was the only reactive aerosol detected and was reported in only one sample. A total of five monitors at four different sites had at least one positive detection for HCl.

### 2.2.4 Pesticides/PCBs

Monitor 2a was one of six monitors where pesticide and PCB samples were collected and analyzed from a single monitoring event. A total of 12 chemicals were detected, one of which was a PCB. All of these chemicals also were detected at another monitor, with six of them found in at least five of the other monitors. The exceptions were PCB congener #101, which was found only at three monitors, and dieldrin, which was detected in monitor 2a and monitor 4 (St. Stephen Baptist Church).

For monitor 2b, 14 chemicals were detected, two of which were PCBs. In general, the chemicals detected at this monitor were also detected in the rest of the monitors, with ten of these chemicals found in at least four other monitors. Four of the pesticides/PCBs found at this monitor were at the highest concentrations found in the monitoring network (i.e., alpha-chlordane, beta-BHC, gamma-chlordane, PCB congener #52).

## 2.3 Old Lake Dreamland Fire Department: WLATS Site 3

This monitor was selected to represent a neighborhood exposure site. Sampling was conducted for VOCs, SVOCs, metals, reactive aerosols, and pesticides/PCBs.

### 2.3.1 VOCs

Sampling for VOCs was conducted on all 32 dates in the monitoring program; however, the number of sample dates reported for any detected VOCs at this site ranged from 27 to 30. The limited number of missing sample dates should not affect the ability of this monitor to provide a reasonable estimate of the chronic exposure. A total of 32 VOCs were detected at this site, with 22 chemicals detected in more than 50% of the samples and five detected in all of the samples. The VOCs detected at this site were also found throughout the monitoring network. Specifically, 26 of the 32 VOCs detected were found in 11 or more monitors, and all 32 VOCs were found in at least four monitors. None of the VOCs detected at this site exhibited a maximum concentration for the monitoring program. Three of the 32 VOCs detected at this site were found in less than 10% of the samples reported. A total of 29 VOCs were selected as COPCs for this monitoring location.

### 2.3.2 SVOCs

A total of 30 sampling events were reported for this monitor. The number of sample dates reported for an individual chemicals ranging from 25 to 28. The number of sample dates reported for the monitoring period should be sufficient to provide a reasonable estimate of the chronic exposure. Overall, 27 SVOCs were detected at this site, with only eight of these chemicals detected in more than 50% of the samples, and only formaldehyde was detected in all samples. Although the frequency of detection was low, all but six of these chemicals were found in four or more monitors in the network. The primary exception was hexachlorobutadiene, which was detected only at this monitor, and in only one sample. A total of seven SVOCs detected at this site, including hexachlorobutadiene, were the highest concentrations found in the monitoring program (i.e., (3-and/or 4-)methylphenol, 2-methyl-4,6-dinitrophenol, 2-methylphenol, 2nitrophenol, benzo(b)fluoranthene, hexachlorobutadiene, and phenol). Four of these chemicals were detected in all of the other SVOC monitors, while 2-methyl-4,6dinitrophenol, and benzo(b)fluoranthene, were found in only two monitors and hexachlorobutadiene was detected only at this site. Ten of the 27 SVOCs detected at this location were found in less than 10% of the samples reported. A total of 17 SVOCs were selected as COPCs for this monitoring location.

#### 2.3.3 Reactive Aerosols and Metals

A total of 29 sampling events were reported for metals analysis at this site; however, only 28 samples dates were reported for any individual chemicals. No data was reported for the first two dates in the monitoring program, then a valid sample was reported, after which the last missing event occurred for this site. The limited number of missing sample dates should not affect the ability of the monitor to produce a reasonable estimate of the chronic exposure. Twenty-five metals were detected at this site, with 15 of the metals detected in over 50% of the samples and seven detected in all of the samples. The metals detected at this site were not unique as all of them were detected in at least four other monitors and 21 were detected in all seven of the monitors used for metal analysis. Three of the metals at this site occurred at the highest concentrations found in the monitoring program (i.e., arsenic, cadmium, and manganese). Three metals were detected in less than 10% of the samples reported for this site. A total of 22 VOCs were selected as COPCs for this monitoring location.

Of the reactive aerosols included in the monitoring program, only HCl was detected at this monitoring site, and only in two samples. A total of five monitors at four different sites had at least one positive detection for HCl.

### 2.3.4 Pesticides/PCBs

The pesticide/PCB monitoring at this site yielded a total of 12 chemicals detected, two of which were PCB congeners. All of the chemicals detected here were found in at least three monitors. One chemical, detected at this site (4,4'-DDE) was at the highest concentration found in the monitoring network.

# 2.4 St. Stephen Baptist Church: WLATS Site 4

This site was identified as a general population exposure site. Sampling was conducted at this site for VOCs, SVOCs, metals, reactive aerosols and pesticides/PCBs.

### 2.4.1 VOCs

A total of 31 sampling events were reported for this site. For individual chemicals, the number of samples dates reported ranged from 28 to 30, which should provide sufficient data to evaluate a chronic exposure. Thirty-eight VOCs were detected at this site, with 18 of these chemicals detected in more than 50% of the samples, but none detected in all of the samples. Ten chemicals were found in less than 10% of the samples. Although the frequency of detection was low at this monitor, the chemicals detected were generally detected throughout the monitoring network. All but two of the compounds were found in five or more of the monitors and 26 of the chemicals were found in 11 or more monitors. The exceptions to this were 1,2-dichloroethane and bromomethane, which

were detected only at this site, with each detected in one of 30 samples. Naphthalene was detected using a VOC analytical technique at this location, but, as discussed earlier, this detection will be disregarded in preference for the SVOC analytical measurements. Therefore, please see Section 2.4.2, which discusses SVOC detections at this site, for details about naphthalene. This monitor yielded the highest detection found for dichlorodifluoromethane (Freon 12), which was detected in all of the other monitors in the network. Because 10 of the 38 VOCs detected at this site were found in less than 10% of the samples reported, a total of 28 VOCs were selected as COPCs for this location.

## 2.4.2 SVOCs

SVOC monitoring at this site consisted of 28 sampling events over the monitoring period. Only once did the missing samples occur back-to-back, which was for first two dates in the monitoring period. The other missing events were preceded and followed by valid measurements. All of the individual SVOCs detected at the site were reported for 28 sample dates, with the exception of formaldehyde, which was reported for only 24 sample dates. The limited number of missing sample dates should not affect the ability of the monitor results to be used for a chronic risk assessment. A total of 22 SVOCs were detected at this site; however, only nine were detected in more than 50% of the samples, and only two were detected in all samples (i.e., 2-methlynaphthalene and formaldehyde).

Although the frequency of detection was generally below 50% at this site, all but two of the SVOCs detected at this site (i.e., isophorene and 2,4,6- trichlorophenol) were found in at least five monitors. Four SVOCs detected at this site were at their highest concentrations found in monitoring network. However, with the exception of 2,4,6- trichlorophenol, the other three SVOCs (i.e., fluoranthene, isophorene, and phenanthrene) were detected in at least one other monitor. Isophorene was detected at this site and the Old Lake Dreamland Fire Department site. Fluoranthene and phenanthrene were detected in six and seven monitors, respectively. Two chemicals were detected in less than 10% of the samples, including 2,4,6-trichlorophenol, which was detected only at this monitor and in just a single sample. A total of 20 SVOCs were selected as COPCs for evaluation in the risk assessment.

### 2.4.3 Reactive Aerosols and Metals

A total of 26 sampling events were reported for the metals sampling at this site. The first two dates in the monitoring program were missed, followed by a valid sample, then another missing event. A second occurrence of two missed events in a row occurred in October of 2000, after which sampling occurred on the remaining scheduled dates. Individual chemicals detected at this site were reported to have 25 valid measurements. Because all months during the monitoring period, except April 2000, had a sample reported for at least one day, the missing dates should not affect the ability of the monitor to produce a reasonable estimate of the chronic exposure at the location. Twenty-seven metals were detected at this site, with 16 detected in more than 50% of the samples, and

ten detected in all of the samples. The metals found at this monitoring site were not unique as all but one were found in at least four monitors and 21 of the 27 were found in all of the monitors used for metals. The exception to this was yttrium, which was detected only at this monitor and in only 2 of 25 samples. In addition to yttrium, four other metals exhibited their maximum concentrations for the monitoring program at this site (i.e., cobalt, molybdenum, nickel, and zinc.). All four of these metals were detected in six or more samples from this site, with zinc in all but one sample, and nickel detected in every sample. Of the 27 metals detected at this site, only three were found in less than 10% of the samples, leaving 24 metals as COPCs for this location.

Of the reactive aerosols included in the monitoring program, only HCl was detected at this monitoring site, and only in one sample. A total of five monitors at four different sites had at least one positive detection for HCl.

## 2.4.4 Pesticides/PCBs

The pesticide/PCB monitoring at this site yielded a total of 19 chemicals detected. Of these 19, eight were PCB congeners. This was the most PCB congeners found at any site used for PCB monitoring. Additionally, this site yielded the highest concentrations detected in the monitoring program for 11 different pesticides/PCBs. Five of these chemicals were detected only at this site (i.e., PCB Congener #153, PCB Congener #163, PCB Congener #201, PCB Congener #209, and toxaphene). For the other six chemicals, dieldrin and PCB Congener #28 were detected in one other monitor (i.e., Ralph Avenue & Campground Road for dieldrin, and Louisville Police Firearms Training site for PCB Congener #28), while alpha-endosulfan, gamma-BHC (lindane), and oxychlordane (octachlorepoxide) were detected in at least three other monitors.

# 2.5 University of Louisville Shelby Campus: WLATS Site 5

This monitoring location was selected to represent an urban site near a major traffic corridor, but not in the immediate vicinity of any manufacturing facilities, and was thus considered an urban background monitor. During the monitoring program samples of VOCs, SVOCs, metals, reactive aerosols, and pesticides/PCBs, were collected at this site. Although sample data were not reported for all 32 sampling dates scheduled during the monitoring program, in all cases at least one sample is reported for every month. Thus, the missing sample dates should not affect the ability of the monitoring data to be used to evaluate a chronic exposure for this risk assessment.

## 2.5.1 VOCs

VOC samples were collected at this site for all of the scheduled monitoring dates in the program. Individual VOCs detected at the site had results for between 28 and 31 sample dates, meaning that every month of the monitoring period was well covered and the data should reflect a chronic exposure. A total of 32 VOCs were detected at this site, with

half of the chemicals detected in more than 50% of the samples, and only three chemicals detected in all of the samples. Thirteen of the chemicals were detected in less than 10% of the samples. Although the frequency of detection for most VOCs was relatively low at this site, the chemicals detected are not unique to this site as all of the chemicals were detected in at least four other monitors, and 18 were found at all of the other monitoring sites. Two of the VOCs detected at this site yielded their respective highest concentrations for the monitoring program (i.e., hexane and methyl acetate). Hexane was detected in 27 of 28 samples and methyl acetate was detected in 9 of 29. A total of 19 VOCs were found in more than 10% of the samples reported and were selected as COPCs for this monitor location.

### 2.5.2 SVOCs

A total of 30 SVOC sampling events were reported for this site during the monitoring program. Only the first two dates in the monitoring program were missed at this site, thus the data should be appropriate for evaluating a chronic exposure. A total of 19 SVOCs were detected at this site, with 14 of these chemicals detected in less than 50% of the samples. Only formaldehyde was detected in all of the SVOC samples for this site. Five of the SVOCs detected at this site were found in less than 10% of the samples. Two of these chemicals (i.e., 2,4,5-trichlorophenol and caprolactam) were detected only at this site, while the other three were detected in at least four monitors. Three SVOCs detected at this site exhibited maximum concentrations for the monitoring program, including the two chemicals that were detected only at this site. The third chemical was benzaldehyde, which was detected in all of the other monitors used for SVOC analysis. Of the 19 SVOCs detected at this monitor, 14 were found in more than 10% of the samples reported and were retained for evaluation as COPCs in the risk assessment.

### 2.5.3 Reactive Aerosols and Metals

Metals sampling at this site include a total of 25 sampling events. The first two dates in the monitoring program were missed, and on two other occasions early in the program, back-to-back sampling events were missed. Although 25 sampling events were reported for this monitor, individual metals have a maximum of 22 valid samples reported. Given that every month in the sampling period, except April 2000, had data for at least one sample date, the missing sample dates should not affect the use of the data for evaluation of a chronic exposure. A total of 22 metals were detected with 16 of the metals found in more than 50% of the samples, and seven metals found in all samples. Only two metals, silver and thallium, were detected in less than 10% of the samples; however, both of these metals were detected in all of the monitors used for analysis of metals. Two of the detected metals at this site were at their maximum concentrations for the monitoring program (i.e., copper and silver). Unlike silver, which was detected infrequently at this site, copper was detected in all of its valid samples. For metals at this monitor, of the 22 metals detected, 20 were found in more than 10% of the samples and were selected as COPCs.

Neither of the reactive aerosols included in the monitoring program was detected at this site.

#### 2.5.4 Pesticides/PCBs

A total of 10 chemicals were detected in the pesticide/PCB monitoring for this site. None of the PCBs were detected. The pesticides detected here were also found in at least four other monitors, except for alpha-BHC, which was found here and at the Louisville Police Firearms Training location. None of the chemicals detected at this site were maximum concentrations for the monitoring program.

# 2.6 Otter Creek Park: WLATS Site 6

This site was selected to represent a background location that is generally upwind of the study area and unlikely to be impacted by emissions released inside the study area or to a relatively lesser extent than the other monitors in the network. The purpose of a background monitor is to identify airborne chemicals that may be moving into the Louisville area from other regions. The background data can also be used to determine the potential impacts to residents from chemicals released outside the study area. During the monitoring program samples of VOCs, SVOCs, metals and reactive aerosols were collected. Although monitoring results were not reported for all of the sampling dates scheduled for the monitoring program, there was data for every chemical for at least one day of every month in the period. The limited number of missing sample dates should not affect the ability of this monitoring data to be used in a risk assessment of chronic exposures.

### 2.6.1 VOCs

A total of 32 sampling events were reported for this monitor; however, for individual chemicals the number of sample dates with results ranged from between 26 and 29, which provides sufficient sampling over the year to reflect a chronic exposure. Twenty-three VOCs were detected at this site, with 11 chemicals detected in more than 50% of the samples, and three of these found in all of the samples reported. Although the frequency of detection was relatively low at this site, all of the chemicals identified were also found in at least six monitors, including 18 that were found in all of the monitors. None of the VOCs detected at this site were at the highest concentrations found in the monitoring program. Of the 23 VOCs detected at this site, four were detected in less than 10% of the reported samples, leaving a total of 19 as COPCs for this site.

### 2.6.2 SVOCs

The SVOC sampling at this site was reported for 29 sampling events. The formaldehyde sampling results indicate that the first two dates in the sampling program were missed,

with another back-to-back period of missing samples in the summer of 2000. The missing dates for the rest of the SVOCs were preceded and followed by valid samples. For every month of the monitoring program there was at least one sample date with results for any individual chemical, therefore the missing sample dates should not affect the use of the data for evaluation of a chronic exposure.

A total of 16 SVOCs were detected at this site, with 13 detected in less than 50% of the samples, including six chemicals that were detected in less than 10% of the samples. The SVOCs detected at this site generally are not unique as all but two of the chemicals were found in five monitors and 12 were found in all of the SVOC monitors. The exception to this was for 2,4-dinitrophenol and n-nitrosodi-n-propylamine, which were only detected in this monitor. Apart from these two chemicals, 1,1- biphenyl was the only other SVOC that yielded a maximum concentration for the monitoring program at this site; however, it was detected in 4 of 28 samples at this site, and positively identified in all of the monitors used for the SVOC monitoring. A total of 10 SVOCs were selected as COPCs for this monitoring site.

### 2.6.3 Reactive Aerosols and Metals

A total of 26 sampling events were reported for metals sampling at this site, with only one period of back-to-back missing dates. The number of sampling dates with monitoring results provides sufficient coverage of the monitoring program for the data to be used to evaluate chronic exposures. Twenty-two metals were detected at this site, with half found in more than 50% of the samples, and only four metals detected in less than 10% of the samples. All of the metals detected at this site were found in all of the monitors used for metals analysis, except for one metal that was found in all but one. None of the detected metals were found at their maximum concentrations for the monitoring program. A total of 18 metals were selected as COPCs for this monitoring location.

Neither of the reactive aerosols included in the monitoring program was detected at this site.

# 2.7 Park DuValle:Southwick Community Center: WLATS Site 7

This monitor was selected to represent a neighborhood population exposure. During the monitoring program samples were collected at this site for VOC analysis only.

A total of 14 sampling events were reported for this site during the monitoring period. These sampling events occurred during the first nine months of the sampling program (i.e., April 2000 to December 2000), and include only one period where back-to-back samples were missed. However, no samples were reported after the December 2, 2000 sample through the end of April 2001, when the sampling period for the monitoring program ended. The range of sample dates reported for individual chemicals detected at this site was between 8 and 14. The chronic airborne concentrations used in the risk

assessment for this monitor may not have the same accuracy as do other monitors with more sample results reported for the monitoring period. For example, if airborne concentrations change with the seasons, this effect would have been missed by this monitor for the winter when no sample results are available.

The number of chemicals that were investigated at this monitor started at 38, then increased to 51 in the next five sampling events reported, and then rose to 79 for the last eight reported events. In total, 36 VOCs were detected at this monitor, with 16 chemicals detected in 50% or more of the samples, and another nine detected in between one-third to one-half of the samples. Most of the chemicals detected at this site were also found in 11or more of the monitors in the network. Five of the VOCs detected at this site exhibited the highest concentrations found for these chemicals in the monitoring program (i.e., bromoform, carbon disulfide, chloroethane, chloromethane, and trichloroethene). Chloroethane was only detected in three monitors in the network, and in only one sample at this site. Bromoform was also detected infrequently at this site (i.e., 2 of 13 samples), but was found in a total of six monitors in the network. The other three chemicals with maximums at this site were detected in more than 40% of the valid samples at this site, and were found in eight or more of the monitors in the program. Of the 36 VOCs detected at this smonitoring location, 35 were found in more than 10% of the samples reported and were selected as COPCs for this site.

# 2.8 Farnsley Middle School: WLATS Site 8

This monitor was selected to represent a neighborhood population exposure. During the monitoring program samples were collected at this site for VOC analysis only.

A total of 19 sampling events were reported for this monitoring site. No data were reported for the first six dates in the sampling program (i.e., April through May 2000). After that, samples were reported regularly for about four months during the summer and fall of 2000, with three missing dates, but valid samples reported before and after the missing events, so that each month had results for at least one sample date. A period with three consecutive missing dates occurred in November and early December of 2000, followed by a period with 11 reported sampling events out of the 12 scheduled. On an individual chemical basis, the majority of the VOCs detected had a total of 19 valid samples reported, and 26 chemicals had 16 valid samples reported.

Although there are several missing sampling dates, the seasons of the year appear to be adequately covered. Thus any changes in airborne concentrations following a change of season should have been detected in this monitor. Further, there is a sample result available for every month during the monitoring period, except for the months of April and May 2000 and November 2000. Therefore, the results available for this monitor should provide a reasonable estimate of the chronic exposure. However, the chronic airborne concentrations used in the risk assessment for this monitor may not have the same accuracy as do other monitors with more sample results reported for the monitoring period.

The number of VOCs investigated at this site began at 50 for the first reported sample, then rose to 51 for the following two reported samples, and then increased to 79 for the remaining 16 reported sampling events. A total of 44 VOCs were detected at this site, with 31 of these chemicals detected in less than 50% of the samples, including 11 chemicals that were detected in less than 10% of the samples. Although the frequency of detection was relatively low at this site, 34 of the 44 VOCs detected at this site were found in six or more monitors in the program, including 26 VOCs that were detected in 11 or more monitors. This monitoring location exhibited the highest concentrations detected in the program for the following 19 chemicals: (m- and/or p-)xylene; 1,1dichloroethene; 1,2,4-trimethylbenzene; 1,3-dichloropropane; 1,4-dichlorobenzene; benzene; cyclohexane; chlorobenzene; ethyl benzene; isopropylbenzene (cumene); methylene chloride; n-propylbenzene; naphthalene; o-xylene; styrene; tetrachloroethene; toluene; cis-1,2-dichloroethene; and, tert-butylbenzene. Tetrachloroethene was also found at a maximum concentration at monitor 9. Of the 44 VOCs detected at this site, a total of 33 were found in more than 10% of the samples reported, and were selected as COPCs.

# 2.9 Chickasaw Park (Private Residence): WLATS Site 9

This monitor was selected to represent a neighborhood population exposure. During the monitoring program samples were collected at this site for VOC analysis only.

A total of 11 sampling events were reported for this site during the monitoring period. The first three dates in the monitoring program were not reported, followed by eight valid sampling events between May 24, 2000 and August 28, 2000, with only one missing date during the period. For the remainder of the monitoring program (i.e., September 2000 to April 2001) there are only three reported sampling events out of 20 scheduled events. Although there are a significant number of dates missing for this monitor, there is a report for each of the season of the year. The majority of the sample dates reported (i.e., seven of the 11 dates reported) were for the summer of 2000. Thus, if there were a seasonal variation to the airborne concentrations, the effect seen in summer would tend to dominate an average concentration calculated from the data for this monitor. The chronic airborne concentrations used in the risk assessment for this monitor may not have the same accuracy as do other monitors with more sample results reported for the monitoring period.

The number of VOCs investigated in the first five reported sampling events was 50 for one event and 51 for the other four. This number rose to 79 VOCs for the remaining six reported sampling events. For individual chemicals detected at the site, the number of dates with samples reported ranged from 6 to 11. A total of 34 chemicals were detected at this site, with 24 of these chemicals detected in less than half of the samples, including 10 VOCs that were detected in less than 10% of the samples. Although the majority of the chemicals were detected infrequently at this site, for the most part they are not unique to this monitor. For example, 24 of the detected VOCs were found in 11 or more of the monitors, and all but four of the VOCs found here were also found in five or more of the monitoring sites. These four chemicals were found in less than 20% of the samples at this site, including two VOCs detected in only 10% of the samples. In addition, none of these four chemicals were detected in more than two other monitors, including one chemical, 1,2,3-trichlorobenzene, which was detected only at this monitor, and only in one of six samples. Two other VOCs detected at this site, besides 1,2,3-trichlorobenzene, exhibited the highest concentrations measured in the monitoring program (i.e., 1,1,2-trichloroethane, and 1,2,3-trichlorobenzene). Tetrachloroethene was also found at a maximum concentration at monitor 8. Both of these chemicals were detected in no more than 2 of 11 samples, but they were detected in 11 or more of the monitors in the network. Of the 34 VOCs detected at this site, 24 were found in more than 10% of the samples reported, and were retained as COPCs for evaluation in the risk assessment.

# 2.10 New Lake Dreamland Fire Department: WLATS Site 10

This monitor was selected to represent a neighborhood population exposure. During the monitoring program samples were collected at this site for VOC analysis only.

A total of 15 sampling events were reported for this monitor. The first five sampling dates in the monitoring period are missing, followed by nine sampling events reported out of 12 scheduled events. During the last six months of the sampling program, from November 2000 through April 2001, there were six sampling events reported out of 15 scheduled events. The monitoring data available does provide at least two sampling dates for each of the seasons in the year. As was the case for WLATS Site 9, the summer months have more samples reported than any other month, with six summer dates out of 15 total dates reported. However, the summer months would not be as dominant at this monitor, as winter has four reported dates, and fall has three. Ultimately, the chronic airborne concentrations used in the risk assessment for this monitor may not have the same accuracy as other monitors with more sample results reported for the monitoring period.

The monitoring at this site reported 51 VOCs in the first three samples and then rose to 79 VOCs for the remaining 12 samples. A total of 30 VOCs were detected at this site, half of which were detected in 50% or more of the samples. Six VOCs were detected in all of the samples and seven were detected in less than 10% of the samples. The VOCs detected at this site were typical of the chemicals found in the monitoring program, with 25 of the VOCs detected in 11 or more monitors and all but one of the chemicals found in at least six monitors. The exception was Freon 114 which was detected only at this monitor and in only one of fifteen samples. No other VOCs detected at this site were the maximum concentrations detected in the monitoring program. Of the 30 VOCs detected at this site, 23 were found in more than 10% of the samples reported, and were selected as COPCs for this monitoring location.

# 2.11 M.L. King Elementary School: WLATS Site 11

This monitor was selected to represent a neighborhood population exposure. During the monitoring program samples were collected at this site for VOC analysis only.

There were18 sampling events reported for this monitor. Sample results were not reported for the first five scheduled dates in the monitoring program. For the remainder of the monitoring program there were reports for 18 of the 27 scheduled events. There are at least two samples dates for each season of the year, and with the exception of April and May of 2000, there is at least one sample date reported for every month. Thus, although there are many missing sample dates, the reported sample dates should provide a reasonable approximation of the chronic exposure at this location. The chronic airborne concentrations used in the risk assessment for this monitor may not have the same accuracy as do other monitors with more sample results reported for the monitoring period.

The first four sampling events reported for this site evaluated 51 VOCs. This number was increased to 79 for the remaining fourteen events. A total of 30 VOCs were detected at this monitor, with 13 detected in more than 50% of the samples, and only two chemicals detected in less than 10% of the samples. All of the VOCs detected at this site were found in three or more monitors, and 25 were detected in at least 11 monitors. Four VOCs detected at this monitor were at their highest concentrations found during the monitoring program (i.e., 1,1,1-trichloroethane, 1,1,2-trichloro-1,2,2-trifluoroethane [Freon 113], carbon tetrachloride, and trichlorofluoromethane [Freon 11]). All four of these chemicals were detected throughout the monitoring network. In addition, they were detected in at least 11 of the 18 samples at this site, with the exception of 1,1,1-trichloroethane, which was detected in just 2 of the 18 samples. Of the 30 VOCs detected at this monitoring location, 28 were found in at least 10% of the samples reported, and were selected for evaluation in the risk assessment.

# 2.12 Cane Run Elementary School: WLATS Site 12

This monitor was selected to represent a neighborhood population exposure. During the monitoring program samples were collected at this site for VOC analysis only.

A total of 21 sampling events were reported for this monitor. The first three scheduled dates were missing, followed by regular reporting over the next six months, from May through October of 2000, which provided data for 13 of the 14 scheduled sampling dates. The remainder of the period the monitoring reports were not as regular, with samples reported for eight of the 15 scheduled events. Overall, monitoring results were available for multiple sample dates during every season of the year. Except for the months of April and November, 2000, every month in the monitoring period had sample results for at least one date. The sample dates provided for this monitor should provide a reasonable estimate of the potential chronic exposure at this site; however, the concentrations used in the risk assessment for this monitor may not have the same accuracy as monitors with more sample results reported for the monitoring period.

The first six sampling events reported evaluated 51 VOCs, which then rose to 79 VOCs for the remainder of the year. For the individual chemicals detected at the site, the majority had 21 sample dates reported; however, four chemicals had only 15 sample

dates reported. A total of 28 VOCs were detected at this site, with half detected in more than 50% of the samples reported, including seven chemicals that were detected in all of their valid samples. Conversely, seven of the VOCs detected were in less than 10% of the samples. All but one of the VOCs were detected at this site were found in six or more monitors, and 23 were detected in 11 or more monitors. Two VOCs detected at this site exhibited the highest concentrations found for this chemical throughout the monitoring network (i.e., vinyl chloride, and 1,2,4-trichlorobenzene). Both chemicals were detected in less than 10% of the samples at this site, but were detected in at least three monitors for the case of 1,2,4-trichlorobenzene and eight monitors for vinyl chloride. Of the 28 VOCs detected at this site, 21 were found in at least 10% of the samples reported, and were selected as COPCs for this monitoring location.

# 2.13 SUMMARY OF COPCS

Table 2-1 provides a summary of the frequency of detection for all chemicals positively identified during the WLATS monitoring program. Generally the chemicals detected during the monitoring program were found throughout the network. For the VOCs, there were 55 different chemicals identified and only 20 of these were found in less than six monitors, including nine that were found in only one monitor. Naphthalene was also found in very few monitors; however, when analyzed as an SVOC with a lower limit of detection, naphthalene was detected in seven monitors. Similarly, SVOCs detected in the WLATS monitoring program were widespread with 21 of the 35 chemicals found in more than four of the seven monitors used for SVOC analysis and only eight detected in just one monitor. HCl was detected in five of the seven monitors used for analysis, but on an infrequent basis. Metals were the most ubiquitous of the chemicals detected in the monitoring program with all but one of the 27 metals detected found in four or more of the seven monitors used for analysis. The exception was yttrium, which was found in a single monitor, and in only two of 25 samples collected at this monitor. Finally, the pesticides/PCBs were the least prevalent chemicals with half of the chemicals detected in three or less of the six monitors in the network used for detection of pesticides/PCBs.

	Table 2-1													
	% Detection for Positive	ly Identi	fied Cho	emicals	(COPO	$C if \ge 10$	% Freq	uency o	f Detect	ion)				
Chemical Class	Chemical Name					%	Detectio	on at Ea	ch Moni	tor				
		1	2a	2b	3	4	5	6	7	8	9	10	11	12
VOC	(M- AND/OR P-)XYLENE	100.0	80.6	89.7	86.7	90.0	77.4	41.4	92.9	36.8	36.4	86.7	33.3	81.0
VOC	1,1,1-TRICHLOROETHANE	51.7	45.2	58.6	50.0	60.0	58.1	48.3	21.4	15.8	18.2	13.3	11.1	4.8
VOC	1,1,2-TRICHLORO-1,2,2- TRIFLUOROETHANE (FREON 113)	100.0	100.0	100.0	100.0	96.4	100.0	100.0	14.3	73.7	18.2	53.3	61.1	61.9
VOC	1,1,2-TRICHLOROETHANE	6.9	0.0	0.0	3.3	3.3	0.0	0.0	0.0	5.3	9.1	0.0	0.0	0.0
VOC	1,1-DICHLOROETHENE	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	5.3	9.1	0.0	0.0	0.0
VOC	1,2,3-TRICHLOROBENZENE	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	16.7	0.0	0.0	0.0
VOC	1,2,4-TRIMETHYLBENZENE	96.6	67.7	79.3	66.7	76.7	54.8	10.3	28.6	10.5	0.0	13.3	11.1	0.0
VOC	1,2,4-TRICHLOROBENZENE	NU	NU	NU	NU	NU	NU	NU	14.3	0.0	18.2	0.0	0.0	4.8
VOC	1,2-DICHLOROETHANE	0.0	0.0	0.0	0.0	3.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
VOC	1,3,5-TRIMETHYLBENZENE	24.1									0.0			
VOC	1,3-BUTADIENE	55.2	61.3	72.4	60.0	43.3	9.7	6.9	69.2	63.2	27.3	53.3	27.8	81.0
VOC	1,3-DICHLOROPROPANE	0.0	0.0	0.0	0.0	0.0	0.0	0.0	37.5	12.5	0.0	0.0	7.1	0.0
VOC	1,4-DICHLOROBENZENE	0.0	0.0	3.4	0.0	20.0	3.2	0.0	21.4	10.5	9.1	0.0	22.2	14.3
VOC	2-CHLORO-1,3-BUTADIENE (CHLOROPRENE)	60.7	60.7	66.7	51.9	17.9	7.1	0.0	37.5	6.3	0.0	41.7	21.4	20.0
VOC	ACETONE	82.1	67.9	66.7	70.4	64.3	67.9	61.5	76.9	78.9	45.5	93.3	88.9	85.7
VOC	ACRYLONITRILE	3.6	0.0	3.7	0.0	0.0	0.0	0.0	25.0	43.8	66.7	16.7	21.4	53.3
VOC	BENZENE	100.0	90.3	89.7	86.7	90.0	83.9	82.8	100.0	94.7	90.9	100.0	94.4	100.0
VOC	BROMODICHLOROMETHANE	0.0	0.0	3.6	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
VOC	BROMOMETHANE	0.0 0.0 0.0 0.0 3.3 0.0 0.0 0.0 0.0 0.0									0.0			
VOC	BUTYL ACRYLATE	0.0	0.0	3.7	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
VOC	BROMOFORM	0.0	0.0	0.0	0.0	0.0	0.0	0.0	15.4	5.3	9.1	6.7	5.6	4.8
VOC	CARBON DISULFIDE	0.0	9.7	6.9	3.3	6.7	3.2	0.0	53.8	52.6	36.4	60.0	55.6	42.9
VOC	CARBON TETRACHLORIDE	96.6	90.3	93.1	90.0	90.0	90.3	89.7	50.0	73.7	63.6	80.0	77.8	66.7
VOC	CHLORODIFLUOROMETHANE (FREON 22)	100.0	100.0	100.0	100.0	96.4	100.0	100.0	100.0	93.8	100.0	100.0	100.0	100.0
VOC	CHLOROETHANE	0.0	0.0	3.4	0.0	0.0	0.0	0.0	7.1	10.5	0.0	0.0	0.0	0.0

	Table 2-1													
	% Detection for Positivel	y Identi	fied Cho	emicals	(COP	C if ≥ 10	% Freq	uency o	f Detect	ion)				
Chemical Class	Chemical Name					%	Detectio	n at Ea	ch Moni	itor				
		1	2a	2b	3	4	5	6	7	8	9	10	11	12
VOC	CHLOROFORM	51.7	67.7	75.9	63.3	16.7	9.7	0.0	42.9	21.1	27.3	40.0	22.2	19.0
VOC	CHLOROMETHANE	96.6	93.5	93.1	90.0	90.0	90.3	89.7	92.9	100.0	100.0	100.0	100.0	100.0
VOC	CYCLOHEXANE	42.9	10.7	14.8	14.8	7.1	3.6	0.0	30.8	15.8	18.2	6.7	11.1	0.0
VOC	CHLOROBENZENE	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	5.3	0.0	0.0	0.0	0.0
VOC	DICHLORODIFLUOROMETHANE (FREON 12)	96.4	96.7	100.0	100.0	96.6	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
VOC	ETHYL ACRYLATE	3.6	20.7	33.3	11.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
VOC	ETHYL BENZENE	96.6	74.2	85.7	73.3	66.7	51.6	17.2	42.9	21.1	9.1	26.7	16.7	9.5
VOC	FREON 114	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	6.7	0.0	0.0
VOC	HEXANE	96.4	96.4	100.0	96.3	89.3	96.4	88.5	100.0	52.6	72.7	80.0	72.2	76.2
VOC	ISOPROPYLBENZENE (CUMENE)	6.9	0.0	6.9	0.0	0.0	0.0	0.0	0.0	12.5	0.0	0.0	0.0	0.0
VOC	METHYL ACETATE	21.4	35.7	33.3	40.7	21.4	31.0	30.8	62.5	31.3	33.3	50.0	57.1	46.7
VOC	METHYL ETHYL KETONE	85.7	82.1	81.5	81.5 81.5 92.9 78.6 53.8 53.8 42.1 27.3 40.0							40.0	38.9	42.9
VOC	METHYL ISOBUTYL KETONE	35.7	0.0	0.0	0.0	7.1	3.6	0.0	0.0	5.3	9.1	0.0	0.0	0.0
VOC	METHYL METHACRYLATE	28.6	64.3	70.4	51.9	7.1	7.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
VOC	METHYL T-BUTYL ETHER (MTBE)	100.0	100.0	100.0	100.0	96.4	85.7	15.4	53.8	36.8	27.3	46.7	16.7	47.6
VOC	METHYLCYCLOHEXANE	60.7	14.3	18.5	40.7	32.1	7.1	0.0	37.5	12.5	0.0	0.0	0.0	0.0
VOC	METHYLENE CHLORIDE	69.0	51.6	58.6	66.7	60.0	45.2	24.1	92.9	94.7	81.8	86.7	83.3	100.0
VOC	N-PROPYLBENZENE	24.1	25.8	24.1	0.0	16.7	3.2	0.0	0.0	6.3	0.0	0.0	0.0	0.0
VOC	NAPHTHALENE	NU	NU	NU	NU	NU	NU	NU	0.0	6.3	0.0	0.0	0.0	0.0
VOC	O-XYLENE	96.6	77.4	79.3	76.7	66.7	48.4	13.8	28.6	15.8	9.1	6.7	11.1	0.0
VOC	SEC-BUTYLBENZENE	0.0	6.5	6.9	3.3	6.7	6.5	6.9	0.0	0.0	0.0	0.0	0.0	0.0
VOC	STYRENE	20.7	38.7	51.7	20.0	20.0	0.0	0.0	35.7	21.1	18.2	6.7	11.1	4.8
VOC	TETPACHI ODOETHENE		20.0	40.0	9.7	3.4	14.3	21.1	9.1	0.0	0.0	9.5		
VOC	TOLUENE	100.0	100.0	100.0	100.0	96.7	96.8	82.8	100.0	94.7	90.9	100.0	100.0	100.0
VOC	TRANS-1,3-DICHLOROPROPENE	0.0	0.0	3.4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
VOC	TRICHLOROETHENE (TRICHLOROETHYLENE)	6.9	0.0	0.0	0.0	3.3	0.0	6.9	42.9	21.1	9.1	6.7	11.1	0.0
VOC	TRICHLOROFLUOROMETHANE (FREON 11)	96.6	90.3	93.1	90.0	90.0	90.3	89.7	100.0	94.7	100.0	100.0	100.0	100.0

				Table 2	-1									
	% Detection for Positiv	vely Identi	fied Cho	emicals	(COP	C if $\geq 10$	% Freq	uency o	f Detect	ion)				
Chemical Class	Chemical Name					%	Detectio	on at Ea	ch Moni	itor				
		1	2a	2b	3	4	5	6	7	8	9	10	11	12
VOC	VINYL CHLORIDE	3.4	9.7	6.9	0.0	3.3	0.0	0.0	35.7	5.3	0.0	6.7	0.0	9.5
VOC	CIS-1,2-DICHLOROETHENE	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	5.3	9.1	0.0	0.0	0.0
VOC	TERT-BUTYLBENZENE	0.0	0.0	0.0	0.0	0.0	0.0	0.0	12.5	18.8	0.0	0.0	0.0	0.0
SVOC	(3-AND/OR 4-)METHYLPHENOL	46.2	46.7	32.0	39.3	39.3	25.9	14.3	NA	NA	NA	NA	NA	NA
SVOC	1,1-BIPHENYL	92.3	76.7	84.0	75.0	78.6	37.0	14.3	NA	NA	NA	NA	NA	NA
SVOC	2,4,5-TRICHLOROPHENOL	0.0	0.0	0.0	0.0	0.0	3.7	0.0	NA	NA	NA	NA	NA	NA
SVOC	2,4,6-TRICHLOROPHENOL	0.0	0.0	0.0	0.0	3.6	0.0	0.0	NA	NA	NA	NA	NA	NA
SVOC	2,4-DINITROPHENOL	0.0	0.0	0.0	0.0	0.0	0.0	3.6	NA	NA	NA	NA	NA	NA
SVOC	2-METHYL-4,6-DINITROPHENOL	7.7	0.0	0.0	3.6	0.0	0.0	0.0	NA	NA	NA	NA	NA	NA
SVOC	2-METHYLNAPHTHALENE	100.0	100.0	100.0	96.4	100.0	96.3	85.7	NA	NA	NA	NA	NA	NA
SVOC	2-METHYLPHENOL	34.6	30.0	36.0	21.4	17.9	11.1	7.1	NA	NA	NA	NA	NA	NA
SVOC	2-NITROPHENOL	26.9	20.0											NA
SVOC	4-CHLOROANILINE	0.0	10.0	8.0	3.6	0.0	3.7	0.0	NA	NA	NA	NA	NA	NA
SVOC	4-NITROPHENOL	11.5	6.7	16.0	17.9	14.3	0.0	0.0	NA	NA	NA	NA	NA	NA
SVOC	ACENAPHTHENE	84.6	50.0	52.0	39.3	57.1	33.3	0.0	NA	NA	NA	NA	NA	NA
SVOC	ACENAPHTHYLENE	3.8	10.0	8.0	3.6	10.7	3.7	0.0	NA	NA	NA	NA	NA	NA
SVOC	ACETOPHENONE	15.4	20.0	28.0	17.9	10.7	14.8	14.3	NA	NA	NA	NA	NA	NA
SVOC	ANTHRACENE	0.0	13.3	8.0	0.0	0.0	0.0	0.0	NA	NA	NA	NA	NA	NA
SVOC	BENZALDEHYDE	3.8	3.3	8.0	7.1	7.1	7.4	7.1	NA	NA	NA	NA	NA	NA
SVOC	BENZO(B)FLUORANTHENE	3.8	0.0	0.0	3.6	0.0	0.0	0.0	NA	NA	NA	NA	NA	NA
SVOC	BIS(2-ETHYLHEXYL) PHTHALATE	0.0	46.7	4.0	21.4	35.7	0.0	3.6	NA	NA	NA	NA	NA	NA
SVOC	CAPROLACTAM	0.0	0.0	0.0	0.0	0.0	3.7	0.0	NA	NA	NA	NA	NA	NA
SVOC	CARBAZOLE	0.0	0.0	4.0	0.0	0.0	0.0	0.0	NA	NA	NA	NA	NA	NA
SVOC	DI-N-BUTYLPHTHALATE	0.0	6.7	0.0	3.6	0.0	0.0	0.0	NA	NA	NA	NA	NA	NA
SVOC	DIBENZOFURAN	92.3	63.3	72.0	75.0	75.0	63.0	17.9	NA	NA	NA	NA	NA	NA
SVOC	DIETHYL PHTHALATE	0.0	3.3	0.0	0.0	0.0	0.0	0.0	NA	NA	NA	NA	NA	NA
SVOC	FLUORANTHENE	73.1	46.7	36.0	25.0	53.6	25.9	0.0	NA	NA	NA	NA	NA	NA
SVOC	FLUORENE	88.5	63.3	56.0	53.6	67.9	44.4	0.0	NA	NA	NA	NA	NA	NA
SVOC	FORMALDEHYDE	100.0	100.0	100.0	100.0	100.0	100.0	100.0	NA	NA	NA	NA	NA	NA
SVOC	HEXACHLOROBUTADIENE	0.0	0.0	0.0	3.6	0.0	0.0	0.0	NA	NA	NA	NA	NA	NA

	Table 2-1													
	% Detection for Positive	ly Identi	fied Cho	emicals	(COP	C if $\geq 10$	% Freq	uency o	f Detect	tion)				
Chemical Class	Chemical Name					%	Detectio	on at Ea	ch Moni	itor				
		1	2a	2b	3	4	5	6	7	8	9	10	11	12
SVOC	ISOPHORONE	0.0	0.0	0.0	7.1	10.7	0.0	0.0	NA	NA	NA	NA	NA	NA
SVOC	N-NITROSODI-N-PROPYLAMINE	0.0	0.0	0.0	0.0	0.0	0.0	3.6	NA	NA	NA	NA	NA	NA
SVOC	NAPHTHALENE	100.0	96.7	100.0	96.4	96.4	92.6	92.9	NA	NA	NA	NA	NA	NA
SVOC	NITROBENZENE	0.0	10.0	0.0	3.6	0.0	0.0	0.0	NA	NA	NA	NA	NA	NA
SVOC	PENTACHLOROPHENOL	3.8	30.0	20.0	3.6	10.7	0.0	3.6	NA	NA	NA	NA	NA	NA
SVOC	PHENANTHRENE	88.5	96.7	96.0	92.9	92.9	85.2	17.9	NA	NA	NA	NA	NA	NA
SVOC	PHENOL	57.7	56.7	56.0	67.9	42.9	29.6	35.7	NA	NA	NA	NA	NA	NA
SVOC	PYRENE	50.0	40.0	28.0	28.6	35.7	0.0	0.0	NA	NA	NA	NA	NA	NA
Р	4,4'-DDE (P,P'-DDE)	100.0	100.0	100.0	100.0	0.0	100.0	0.0	NA	NA	NA	NA	NA	NA
Р	4,4'-DDT (P,P'-DDT)	100.0	100.0	100.0	100.0	0.0	100.0	0.0	NA	NA	NA	NA	NA	NA
Р	ALPHA-BHC	100.0	0.0	0.0	0.0	0.0	100.0	0.0	NA	NA	NA	NA	NA	NA
Р	ALPHA-CHLORDANE /2	100.0	100.0	100.0									NA	NA
Р	BETA-BHC	100.0	0.0	100.0	0.0	0.0	0.0	0.0	NA	NA	NA	NA	NA	NA
Р	DIELDRIN	0.0	100.0	0.0	0.0	100.0	0.0	0.0	NA	NA	NA	NA	NA	NA
Р	ENDOSULFAN I (ALPHA)	100.0	100.0	100.0	100.0	100.0	100.0	0.0	NA	NA	NA	NA	NA	NA
Р	GAMMA-BHC (LINDANE)	100.0	100.0	100.0	0.0	100.0	100.0	0.0	NA	NA	NA	NA	NA	NA
Р	GAMMA-CHLORDANE /2	100.0	100.0	100.0	100.0	100.0	100.0	0.0	NA	NA	NA	NA	NA	NA
Р	HEPTACHLOR	100.0	100.0	100.0	100.0	100.0	100.0	0.0	NA	NA	NA	NA	NA	NA
Р	HEPTACHLOR EPOXIDE	100.0	100.0	100.0	100.0	100.0	100.0	0.0	NA	NA	NA	NA	NA	NA
Р	OXYCHLORDANE (OCTACHLOREPOXIDE) /2	100.0	0.0	100.0	100.0	100.0	0.0	0.0	NA	NA	NA	NA	NA	NA
Р	PCB CONGENER #101	100.0	100.0	0.0	0.0	100.0	0.0	0.0	NA	NA	NA	NA	NA	NA
Р							0.0	0.0	NA	NA	NA	NA	NA	NA
Р	PCB CONGENER #163	0.0	0.0	0.0	0.0	100.0	0.0	0.0	NA	NA	NA	NA	NA	NA
Р	PCB CONGENER #201	0.0	0.0	0.0	0.0	100.0	0.0	0.0	NA	NA	NA	NA	NA	NA
Р	PCB CONGENER #209	0.0	0.0	0.0	0.0	100.0	0.0	0.0	NA	NA	NA	NA	NA	NA
Р	PCB CONGENER #28	100.0	0.0	0.0	0.0	100.0	0.0	0.0	NA	NA	NA	NA	NA	NA
Р	PCB CONGENER #52	0.0	0.0	100.0	100.0	100.0	0.0	0.0	NA	NA	NA	NA	NA	NA
Р	PCB CONGENER #60	100.0	0.0	100.0	100.0	100.0	0.0	0.0	NA	NA	NA	NA	NA	NA
Р	TOXAPHENE	0.0	0.0	0.0	0.0	100.0	0.0	0.0	NA	NA	NA	NA	NA	NA

	Table 2-1													
	% Detection for Posi	itively Identi	fied Cho	emicals	(COP	C if $\geq 10$	% Freq	uency o	f Detect	tion)				
Chemical Class	Chemical Name					%	Detectio	on at Eac	ch Mon	itor				
		1	2a	2b	3	4	5	6	7	8	9	10	11	12
Р	ALPHA-CHLORDENE /2	100.0	100.0	100.0	100.0	100.0	0.0	0.0	NA	NA	NA	NA	NA	NA
Р	BETA-CHLORDENE /2	100.0	100.0	100.0	100.0	100.0	100.0	0.0	NA	NA	NA	NA	NA	NA
RA	HCL (CALCULATED FROM CL)	4.2	10.7	4.3	6.9	3.7	0.0	0.0	NA	NA	NA	NA	NA	NA
М	ALUMINUM	91.7	100.0	100.0	92.9	92.0	95.5	80.8	NA	NA	NA	NA	NA	NA
М	ANTIMONY	54.2	46.2	44.0	35.7	48.0	50.0	34.6	NA	NA	NA	NA	NA	NA
М	ARSENIC	100.0	100.0	100.0	100.0	100.0	100.0	92.3	NA	NA	NA	NA	NA	NA
М	BARIUM	100.0	100.0	100.0	100.0	100.0	100.0	84.6	NA	NA	NA	NA	NA	NA
М	BERYLLIUM	45.8	53.8	48.0	42.9	28.0	13.6	0.0	NA	NA	NA	NA	NA	NA
М	CADMIUM	45.8	53.8	60.0	60.7	36.0	54.5	38.5	NA	NA	NA	NA	NA	NA
М	CALCIUM	100.0	100.0	100.0	100.0	100.0	100.0	100.0	NA	NA	NA	NA	NA	NA
М	CHROMIUM	100.0											NA	
М	COBALT	0.0	3.8	8.0	3.6	24.0	0.0	0.0	NA	NA	NA	NA	NA	NA
М	COPPER	100.0	100.0	100.0	100.0	100.0	100.0	96.2	NA	NA	NA	NA	NA	NA
М	IRON	100.0	100.0	100.0	96.4	100.0	90.9	57.7	NA	NA	NA	NA	NA	NA
М	LEAD	50.0	53.8	56.0	35.7	56.0	40.9	30.8	NA	NA	NA	NA	NA	NA
М	MAGNESIUM	87.5	92.3	96.0	92.9	100.0	68.2	23.1	NA	NA	NA	NA	NA	NA
М	MANGANESE	100.0	100.0	100.0	100.0	100.0	100.0	96.2	NA	NA	NA	NA	NA	NA
М	MOLYBDENUM	20.8	23.1	16.0	14.3	52.0	0.0	0.0	NA	NA	NA	NA	NA	NA
М	NICKEL	100.0	100.0	100.0	100.0	100.0	100.0	100.0	NA	NA	NA	NA	NA	NA
М	POTASSIUM	16.7	11.5	12.0	10.7	24.0	0.0	7.7	NA	NA	NA	NA	NA	NA
М	SELENIUM	29.2	38.5	32.0	28.6	36.0	36.4	34.6	NA	NA	NA	NA	NA	NA
М	SILVER	4.2	3.8	8.0	7.1	8.0	9.1	7.7	NA	NA	NA	NA	NA	NA
М	SODIUM	91.7	96.2	96.0	96.4	96.0	90.9	96.2	NA	NA	NA	NA	NA	NA
М	STRONTIUM	95.8	96.2	96.0	92.9	100.0	72.7	26.9	NA	NA	NA	NA	NA	NA
М	THALLIUM	20.8	23.1	24.0	7.1	12.0	4.5	7.7	NA	NA	NA	NA	NA	NA
М	TIN	20.8	11.5	16.0	0.0	8.0	0.0	0.0	NA	NA	NA	NA	NA	NA
М	TITANIUM	95.8	96.2	96.0	92.9	96.0	81.8	34.6	NA	NA	NA	NA	NA	NA
М	VANADIUM	33.3	57.7	56.0	21.4	20.0	13.6	3.8	NA	NA	NA	NA	NA	NA
М	YTTRIUM	0.0	0.0	0.0	0.0	8.0	0.0	0.0	NA	NA	NA	NA	NA	NA
М	ZINC	95.8	96.2	96.0	92.9	96.0	86.4	84.6	NA	NA	NA	NA	NA	NA

VOC = Volatile Organic Compound SVOC = Semivolatile Organic Compound P = Pesticide/PCB RA = Reactive Aerosol M = Metal 0.0% = Analyzed but not detected NU = VOC data available but not used (See text Sec 2.13) NA = Not analyzed

		Tal	ole 2-2											
		COPCs	by Mor	nitor										
Chemical Class	Chemical Name						]	Monito	r					
		1	2a	2b	3	4	5	6	7	8	9	10	11	12
VOC	(M- AND/OR P-)XYLENE	Х	X	Х	X	Х	X	X	Х	Х	Х	Х	Х	Х
VOC	1,1,1-TRICHLOROETHANE	Х	Х	Х	X	Х	X	Х	Х	Х	Х	X	Х	
VOC	1,1,2-TRICHLORO-1,2,2-TRIFLUOROETHANE (FREON 113)	Х	X	Х	Х	X	X	X	X	X	Х	Х	Х	X
VOC	1,2,3-TRICHLOROBENZENE										Х			
VOC	1,2,4-TRIMETHYLBENZENE	Х	X	Х	Х	Х	X	X	Х	Х		Х	Х	
VOC	1,2,4-TRICHLOROBENZENE								Х		Х			
VOC	1,3,5-TRIMETHYLBENZENE	Х	Х	Х	Х	Х								
VOC	1,3-BUTADIENE	Х	Х	Х	Х	Х			Х	Х	Х	Х	Х	Х
VOC	1,3-DICHLOROPROPANE								Х	Х				
VOC	1,4-DICHLOROBENZENE					Х			Х	Х			Х	Х
VOC	2-CHLORO-1,3-BUTADIENE (CHLOROPRENE)	Х	Х	Х	Х	Х			Х			Х	Х	Х
VOC	ACETONE	Х	Х	Х	Х	Х	Х	Х	Х	Х	Х	Х	Х	Х
VOC	ACRYLONITRILE								X	X	X	X	X	X
VOC	BENZENE	Х	X	X	X	X	X	X	X	X	Х	X	X	Х
VOC	BROMOFORM								X					
VOC	CARBON DISULFIDE								X	X	Х	X	X	Х
VOC	CARBON TETRACHLORIDE	Х	X	X	Х	X	X	X	X	X	Х	X	X	Х
VOC	CHLORODIFLUOROMETHANE (FREON 22)	Х	X	Х	X	X	X	X	X	X	X	X	Х	Х
VOC	CHLOROETHANE									Х				
VOC	CHLOROFORM	Х	X	X	Х	X			X	X	Х	X	Х	Х
VOC	CHLOROMETHANE	Х	X	X	Х	Х	X	X	X	X	Х	X	X	X
VOC	CYCLOHEXANE	Х	X	X	Х				X	X	X		X	
VOC	DICHLORODIFLUOROMETHANE (FREON 12)	Х	X	X	Х	X	X	X	X	X	Х	X	Х	X
VOC	ETHYL ACRYLATE		Х	X	Х									
VOC	ETHYL BENZENE	Х	X	Х	Х	X	Х	X	X	Х		X	X	
VOC	HEXANE	Х	X	X	Х	X	X	X	X	X	Х	X	Х	Х
VOC	ISOPROPYLBENZENE (CUMENE)									Х				
VOC	METHYL ACETATE	Х	X	X	X	X	Х	X	Х	X	Х	X	Х	Х

#### WEST LOUISVILLE AIR TOXICS STUDY

		Tat	ole 2-2											
	C	<b>OPCs</b>	by Mor	nitor										
Chemical Class	Chemical Name		•				1	Monito	r					
		1	2a	2b	3	4	5	6	7	8	9	10	11	12
VOC	METHYL ETHYL KETONE	Х	X	Х	Х	Х	X	X	X	Х	X	X	X	X
VOC	METHYL ISOBUTYL KETONE	Х												
VOC	METHYL METHACRYLATE	Х	X	Х	Х									
VOC	METHYL T-BUTYL ETHER (MTBE)	Х	X	Х	Х	Х	Х	X	Х	Х	X	Х	Х	X
VOC	METHYLCYCLOHEXANE	Х	X	X	Х	X			Х	Х				
VOC	METHYLENE CHLORIDE	Х	Х	Х	Х	Х	Х	X	Х	Х	Х	Х	Х	X
VOC	N-PROPYLBENZENE	Х	X	X		X								
VOC	O-XYLENE	Х	X	X	Х	X	X	X	Х	Х			Х	
VOC	STYRENE	Х	Х	X	Х	X			Х	Х	X		Х	
VOC	TETRACHLOROETHENE (TETRACHLOROETHYLENE)	Х	Х	Х	Х	Х			Х	Х				
VOC	TOLUENE	X X X X X X X X X X X X X X X												
VOC	TRICHLOROETHENE (TRICHLOROETHYLENE)													
VOC	TRICHLOROFLUOROMETHANE (FREON 11)	Х	X	X	Х	X	X	X	Х	Х	X	X	Х	X
VOC	VINYL CHLORIDE								Х					
VOC	TERT-BUTYLBENZENE								Х	Х				
SVOC	(3-AND/OR 4-)METHYLPHENOL	Х	X	X	Х	X	X	X						
SVOC	1,1-BIPHENYL	Х	Х	X	Х	X	X	X						
SVOC	2-METHYLNAPHTHALENE	Х	Х	X	Х	X	X	X						
SVOC	2-METHYLPHENOL	Х	Х	X	Х	Х	X							
SVOC	2-NITROPHENOL	Х	Х	X	Х	X	X	X						
SVOC	4-CHLOROANILINE		Х											
SVOC	4-NITROPHENOL	Х		Х	Х	Х								
SVOC	ACENAPHTHENE	Х	X	X	Х	X	X							
SVOC	ACENAPHTHYLENE		Х			Х								
SVOC	ACETOPHENONE	Х	X	X	Х	Х	Х	X						
SVOC	ANTHRACENE		Х											
SVOC	BIS(2-ETHYLHEXYL) PHTHALATE		Х		Х	Х								
SVOC	DIBENZOFURAN	Х	Х	Х	Х	Х	Х	Х						
SVOC	FLUORANTHENE	Х	Х	Х	Х	Х	Х							
SVOC	FLUORENE	Х	Х	Х	Х	Х	Х							

#### WEST LOUISVILLE AIR TOXICS STUDY

		Tat	ole 2-2											
		COPCs	by Mor	nitor										
Chemical Class	Chemical Name		-				]	Monito	r					
		1	2a	2b	3	4	5	6	7	8	9	10	11	12
SVOC	FORMALDEHYDE	Х	Х	Х	Х	Х	X	Х						
SVOC	ISOPHORONE					Х								
SVOC	NAPHTHALENE	Х	Х	Х	Х	Х	X	Х						
SVOC	NITROBENZENE		Х											
SVOC	PENTACHLOROPHENOL		Х	Х		Х								
SVOC	PHENANTHRENE	Х	X	Х	X	Х	X	Х						
SVOC	PHENOL	Х	Х	Х	Х	Х	X	Х						
SVOC	PYRENE	Х	Х	Х	X	Х								
Р	4,4'-DDE (P,P'-DDE)	Х	Х	Х	Х		X							
Р	4,4'-DDT (P,P'-DDT)	Х	X	X	X		Х							
Р	ALPHA-BHC	X X I I I I I I I I I I I I I I I I I I												
Р	ALPHA-CHLORDANE /2	Х	X	X	X	Х	Х							
Р	BETA-BHC	Х		X										
Р	DIELDRIN		Х			Х								
Р	ENDOSULFAN I (ALPHA)	Х	Х	Х	Х	Х	X							
Р	GAMMA-BHC (LINDANE)	Х	X	X		Х	Х							
Р	GAMMA-CHLORDANE /2	Х	X	X	X	Х	Х							
Р	HEPTACHLOR	Х	X	X	X	Х	Х							
Р	HEPTACHLOR EPOXIDE	Х	X	X	X	Х	Х							
Р	OXYCHLORDANE (OCTACHLOREPOXIDE) /2	Х		X	X	Х								
Р	PCB CONGENER #101	Х	X			Х								
Р	PCB CONGENER #153					Х								
Р	PCB CONGENER #163					Х								
Р	PCB CONGENER #201					Х								
Р	PCB CONGENER #209					Х								
Р	PCB CONGENER #28	Х		1		Х								
Р	PCB CONGENER #52			Х	Х	Х								
Р	PCB CONGENER #60	Х		Х	Х	Х								
Р	TOXAPHENE					Х								
Р	ALPHA-CHLORDENE /2	Х	Х	Х	Х	Х								

#### WEST LOUISVILLE AIR TOXICS STUDY

		Tal	ole 2-2											
		COPCs	by Mor	nitor										
Chemical Class	Chemical Name						I	Monito	r					
		1	2a	2b	3	4	5	6	7	8	9	10	11	12
Р	BETA-CHLORDENE /2	Х	Х	Х	Х	Х	Х							
RA	HCL (CALCULATED FROM CL)		Х											
М	ALUMINUM	Х	Х	Х	Х	Х	Х	Х						
М	ANTIMONY	Х	Х	Х	Х	Х	Х	Х						
М	ARSENIC	Х	Х	Х	Х	Х	Х	Х						
М	BARIUM	Х	X	Х	Х	Х	X	Х						
М	BERYLLIUM	Х	Х	Х	X	Х	Х							
М	CADMIUM	Х	Х	Х	Х	Х	Х	Х						
М	CALCIUM	Х	X	X	X	Х	X	X						
М	CHROMIUM	X X X X X X X X												
М	COBALT													
М	COPPER	Х	X	X	X	Х	X	X						
М	IRON	Х	Х	Х	Х	Х	Х	Х						
М	LEAD	Х	Х	Х	Х	Х	Х	Х						
М	MAGNESIUM	Х	Х	Х	Х	Х	Х	Х						
М	MANGANESE	Х	Х	Х	Х	Х	Х	Х						
М	MOLYBDENUM	Х	Х	Х	Х	Х								
М	NICKEL	Х	Х	Х	Х	Х	Х	Х						
М	POTASSIUM	Х	Х	Х	Х	Х								
М	SELENIUM	Х	Х	Х	Х	Х	Х	Х						
М	SODIUM	Х	Х	Х	Х	Х	Х	Х						
М	STRONTIUM	Х	Х	Х	Х	Х	Х	Х						
М	THALLIUM	Х	Х	Х		Х								
М	TIN	Х	Х	Х										
М	TITANIUM	X	X	X	Х	X	X	X						
М	VANADIUM	X	X	X	Х	X	X							
М	ZINC	Х	X	Х	X	X	X	X						

## **3.0 EXPOSURE ASSESSMENT**

Exposure assessment is the process that characterizes the route, duration, intensity, and frequency of contact with a chemical by a receptor. In this assessment, the receptors of interest are individuals that may reside within the WLATS monitoring area, and the principal exposure route of interest is inhalation. Two exposure durations are evaluated: chronic and acute. For chronic scenarios, exposure to relatively low levels of pollutants repeatedly over a prolonged period of time is evaluated. For acute scenarios, one-time exposure to the highest concentration is assumed to occur.

## **3.1 Chronic Exposures**

In this assessment, chronic exposure was evaluated based on the median and 95<sup>th</sup> % Upper Confidence Limit (UCL) estimates of the long–term average concentration for each COPC, as per the requirements of the WLATS Risk Assessment Work Plan. The median value was selected to provide an estimate of the central tendency, while the 95% UCL was selected to reflect a conservative estimate of chronic exposure. The following conservative assumptions were used in the assessment of exposure at the median and 95<sup>th</sup> % UCL:

- A person lives, works, and otherwise stays near a given monitoring location for a 70year lifetime.
- The air that the person breathes, both while indoors and outdoors, contains the same concentrations of pollutants measured in the WLATS study.
- Air quality, as reflected by the WLATS 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 per the WLATS Work Plan guidance, and standard practice in conducting risk assessments, all samples reported as non-detects were assigned a value of ½ the lowest concentration that the instrument can detect, known as the sample quantitation limit or SQL. Although the Work Plan directed that results from the two collocated monitors at Ralph Avenue (WLATS 2a and 2b) be averaged for analysis as a single monitor point, it was agreed that we should keep the data from the two monitors separate and evaluate them independently as a means to evaluate the uncertainty in the monitoring program.

After treatment of non-detects was completed, descriptive statistics were calculated for each monitor. For each chemical reported at a monitor, the following information was determined:

- the frequency at which the chemical was detected at the monitor;
- the maximum and minimum detected concentrations;
- for chemicals with non-detects at the monitor (i.e., frequency of detection less than 100%), the range of SQLs was determined; and,
- the arithmetic mean, median and standard deviation of the chemical data was calculated as follows.

The arithmetic mean was calculated as:

$$\overline{c} = \frac{\sum_{i=1}^{n} c_i}{n}$$
 Equation 3-1

where:

 $\overline{c}$  = the arithmetic mean concentration;

 $c_i$  = an individual sample measurement; and

n = the total number of sample measurements.

The standard deviation was calculated as:

$$s = \sqrt{\frac{\sum_{i=1}^{n} (c_i - \overline{c})^2}{n-1}}$$
 Equation 3-2

where:

s = the standard deviation of the concentration data;

 $\overline{c}$  = the arithmetic mean concentration;

 $c_i$  = an individual sample measurement; and

n = the total number of sample measurements.

As per the WLATS Risk Assessment Work Plan, a median value was calculated for each chemical and monitor. The median value reflects the midpoint of the data; that is half of the values are above the median and half of the values are below. This value was selected to represent the central tendency of concentrations a person may be exposed to via inhalation. The median concentration was calculated by first arranging the sampling results for a given chemical at a single monitor in order from the smallest to the largest value. If the number of samples being evaluated was an even number, then the media was calculated as the arithmetic average of the two middle values. For example, if the sample values were 3, 6, 8, and 11, the median would be calculated as (6 + 8)/2, which gives a median value of 7. When the number of samples was odd, the median value is in the middle. For example, the median value for the numbers 1, 3, 5, 7, and 9 is 5.

In addition to these summary statistics, the data analysis also included various statistical calculations typically used in risk assessments. First, a statistical test was conducted to determine the distribution of the chemical data for a monitoring location. Following USEPA guidance, the Shapiro-Wilke test was conducted to test the hypothesis that the data were normally distributed. If this hypothesis proved true according to the test results, then the arithmetic mean and standard deviation calculated above were used to calculate the 95<sup>th</sup> percentile upper confidence limit, typically abbreviated as the 95<sup>th</sup> UCL. The 95<sup>th</sup> UCL is typically used as a conservative estimate of the true average concentration. Theoretically, the 95<sup>th</sup> UCL provides a value that 95% of the time equals or exceeds the true mean of the data. The 95% UCL value for normally distributed data was calculated using the following formula:

$$\overline{c}_{95} = c + \frac{s \bullet t_{95}}{\sqrt{n}}$$
 Equation 3-2

where:

 $\overline{c}_{95} = 95^{\text{th}}$  percentile upper confidence limit on the mean;  $\overline{c} =$  the arithmetic mean concentration; s = the standard deviation of the concentration data; t<sub>95</sub> = student's t statistic based on n-1 degree of freedom; and n = the total number of sample measurements.

If the Shapiro-Wilke test did not indicate that the data were normally distributed, then the assumption was made that the data were lognormal and a different equation was used to calculate the 95% UCL. First the data was log transformed by calculating the natural logarithm of each of the sample values. Then equations 1 and 2 above were used to compute the mean and standard deviation of the log transformed values. Finally, the 95% UCL was calculated using the following equation as per USEPA (1992a) Guidance.

$$\overline{c}_{95} = e^{\overline{c}_i + 0.5s_i + \left(\frac{s_i H}{\sqrt{n-1}}\right)}$$
Equation 3-3

where:

 $\overline{c}_{95} = 95^{\text{th}}$  percentile upper confidence limit on the mean;

- $\overline{c}_t$  = the arithmetic mean concentration of the log transformed values;
- $s_t =$  the standard deviation of the log transformed concentration data;

H = the H statistic (See Gilbert 1987); and

n = the total number of sample measurements.

Additional testing was done to determine if a lognormal distribution was appropriate in cases where a normal distribution was rejected on the basis of the Shapiro-Wilke test.

Cases where neither the normal or lognormal distribution was appropriate for a dataset were noted for evaluation in the uncertainty analysis. The results of the distribution testing are presented in Appendix C.

# **3.2 Acute Exposures**

Acute (short-term) health effects from air pollutants are also possible if concentrations are sufficiently high. Health effects that persons may experience due to short-term exposures to airborne contaminants can vary significantly from those experienced after long-term exposure to low doses, depending on the contaminant and its concentration. For example, a substance that produces an increase in cancer rates after exposure to low concentrations for a long period of time might also cause immediate and severe eye irritation if present at sufficiently high levels for a short period of time.

Methods to assess acute health effects, however, are not well established. As a conservative approach for this study, the highest concentrations of pollutants measured (during one year of monitoring) at each location were compared to acute benchmark concentrations. Reliance on maximum measured concentrations to evaluate the potential for adverse effects from acute exposures, as opposed to upper confidence limits of means, treats each sample independently, and thus averts the potential to "average out" spikes in concentration.

## 4.0 HAZARD IDENTIFICATION AND DOSE-RESPONSE ASSESSMENT

Generally recognized definitions of risk assessment include two components that depend on the core risk assessment disciplines of toxicology and epidemiology. These two components are known as hazard identification and dose-response assessment. Hazard identification is the process of determining whether exposure to a chemical can cause an increase in the incidence of an adverse health consequence in humans. Dose-response assessment is the process of characterizing the relationship between exposure and the incidence of an adverse health effect in the exposed population.

This assessment utilized toxicology reviews conducted by the USEPA, the Agency for Toxic Substances and Disease Registry (ATSDR), the State of California, the International Agency for Research on Cancer (IARC), and other government bodies to describe both the hazard and dose-response characteristics of the COPCs. These evaluations were conducted separately for health effects that could be caused following chronic (or long-term) exposures and those that could be caused following acute (one-time or short-term) exposures. Toxicity benchmarks, or levels of pollutants that at or below a value are assumed not to cause harmful effects to human health, were developed for chronic and acute exposures by various government bodies. These distinctions were made due to the uniqueness of the toxic response that can occur following short-term exposure to relatively high concentrations compared to that which can occur following long-term, lower-level exposure.

The remainder of this section describes the general types of health effects that were considered, details the type and source of the dose-response criteria that were utilized, and provides summary tables of the quantitative toxicity criteria that were used in the risk assessment.

# 4.1 Chronic Toxicity

Two distinct types of chronic health effects were considered in this assessment: cancer and non-cancer effects. To evaluate health effects and establish toxicity benchmarks in this study, the general hierarchy of data sources and methodologies outlined in the Risk Assessment Work Plan, and advocated by the USEPA's Office of Air Quality Planning and Standards in the National Air Toxics Assessment (NATA) were used. Wherever available, USEPA inhalation unit risk estimates (URE) for cancer and USEPA reference concentrations (RfCs) for non-cancer effects were used as benchmark concentrations. When these values were not available, other values were used as benchmark concentrations in the following hierarchical preference: (i) minimal risk levels (MRLs) developed by ATSDR, (ii) California EPA inhalation unit risks and reference exposure levels (RELs), and (iii) USEPA's health effects assessment summary tables (HEAST). Some toxics currently lack inhalation assessments from these sources and, therefore, oral potency estimates were used to calculate the inhalation toxicity values. The equations used are described in Sections 4.1.1 and 4.1.2. The same toxicity data source hierarchy outlined above was used to select oral toxicity values. The use of oral toxicity values as the basis of inhalation values introduces uncertainty into the overall risk assessment. This approach was nevertheless adopted here to provide some evaluation of the degree of potential risk from chemical exposure, instead of excluding a detected chemical completely from the evaluation. The uncertainty section of the risk assessment documents the use of oral data to represent inhalation risks and discusses the effect on the overall risk estimates.

Established toxicity data were not available for all COPCs and, therefore, no risk estimates were generated for these compounds. The potential consequences of this to the overall risk estimate are discussed in the uncertainty analysis.

#### 4.1.1 Cancer Effects

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 URE in units of risk per mg/m<sup>3</sup>) or as a risk per daily intake (e.g., an oral carcinogenic potency slope factor, or CPS<sub>o</sub>, in units of risk per mg/kg–day).

In hazard identification of carcinogens under the 1986 USEPA guidelines, human data, animal data, and supporting evidence are combined to characterize the weight–of–evidence (WOE) regarding the agent's potential as a human carcinogen into one of several categories:

- Group A Carcinogenic to Humans: Agents with adequate human data to demonstrate the causal association of the agent with human cancer (typically epidemiological data).
- Group B Probably Carcinogenic to Humans: Agents with sufficient evidence (i.e., indicative of a causal relationship) from animal bioassay data, but either limited (i.e., indicative of a possible causal relationship, but not exclusive of alternative explanations) human evidence (Group B1), or with little or no human data (Group B2).
- Group C Possibly Carcinogenic to Humans: Agents with limited animal evidence and little or no human data.
- Group D Not Classifiable as to Human Carcinogenicity: Agents without adequate data either to suggest or refute the suggestion of human carcinogenicity.

Group E – Evidence of Non–carcinogenicity for Humans: Agents that show no evidence for carcinogenicity in at least two adequate animal tests in different species or in both adequate epidemiologic and animal studies.

Weight-of-evidence determinations for carcinogenicity developed by the International Agency for Research on Cancer (IARC) were used for carcinogens not characterized by USEPA. Carcinogens are categorized by IARC as Group 1 (agents carcinogenic to humans), Group 2A (probable human carcinogen), and Group 2B (possible human carcinogen).

Only those substances that are known or suspected human carcinogens were considered in calculating incremental cancer risks (USEPA WOE groups A, B, or C, or IARC classifications of 1, 2A or 2B).

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

If no URE was available for a known or suspected human carcinogen,  $CPS_os$  were converted to a URE by the following equation:

$$URE = \frac{CPS_0 x IR}{BW}$$
 Equation 4-1

where:

URE	=	unit risk estimate (1/mg/m <sup>3</sup> )
$CPS_o$	=	oral carcinogenic potency slope factor, equal to risk per mg/kg-day
IR	=	standard inhalation rate for an adult, equal to 20 m <sup>3</sup> /day; and
BW	=	standard assumption for average adult body weight, equal to 70 kg.

Table 4-1 contains the chronic carcinogenic toxicity values for the COPCs.

The USEPA is currently revising the Guidelines for Carcinogenic Risk Assessment. Based on a review of the final draft version released for public comment (USEPA, 2003) there are several areas where the Guidance has been updated. One area is the use of default options applied when critical information about the human health effects of a substance is lacking. For example, if no information is available regarding the human health effects of a substance, then a common default option is to assume that adverse health effects seen in animals from exposure to a substance have the potential to occur in humans as well. The revised Guidance provides greater detail on the EPA's policy for using the default options. The weight-of-evidence approach to characterizing the potential for a substance to be a human carcinogen has been retained, but a more complete narrative summary of the available evidence and the uncertainties and default assumptions used is recommended. The new Guidelines also stress the importance of understanding the effects that a substance may cause in the body and how they might lead to the development of cancer. This information can be useful in determining the potency of a chemical as a carcinogen, the potential effects at low doses, who may be more susceptible to the substance, and whether animal studies are reliable indicators of potential effects in humans. The Guidelines have placed particular emphasis on the potential for increased vulnerability on childhood exposures. Due to the draft nature of the Guidelines revision, the new recommendations it contains were not adopted for this risk assessment. Rather, the 1999 version of the document was used, as it remains the EPA's operative guidance until the current draft is finalized.

#### 4.1.2 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  $mg/m^3$  air) or as a daily intake (e.g., an oral reference dose or RfD<sub>o</sub> 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, RfD<sub>o</sub>s were converted to RFCs using the following equation:

$$RfC = \frac{RfD \times BW}{IR}$$
 Equation 4-2

where:

RfC	=	Inhalation reference concentration (mg/m <sup>3</sup> );
RfD	=	Oral reference dose (mg/kg–day);
IR	=	Standard inhalation rate for an adult, equal to $20 \text{ m}^3/\text{day}$ ; and
BW	=	Standard assumption for average adult body weight, 70 kg.

Table 4-2 contains the chronic non-carcinogenic toxicity values for the COPCs.

Table 4-3 contains the chronic non-carcinogenic target organ information for the COPCs.

## 4.2 Acute Toxicity

In addition to long-term toxicity data, the potential for short-term acute effects from exposure to airborne COPCs also was evaluated. There is no simple or widely accepted method for estimating the risks from routine short-term exposures to the concentrations of most toxic substances found in ambient air samples. As such, there are no uniformly

accepted short-term air concentration benchmarks for emissions from facilities and other common emission sources such as area and mobile sources.

In addition, acute benchmarks cover a wide spectrum of potential health effects, ranging from mild irritation to life threatening conditions. Several acute benchmarks may be available for the same substance to address different short–term effects on health. Consistent with the screening level nature of acute health effects assessment, the lowest, or most conservative, acute benchmark was chosen for a given substance to evaluate all possible short–term health effects (generally, levels protective of mild effects).

Methods to develop acute benchmarks are ongoing in the USEPA, and the most recent recommendations from USEPA Region IV were used in the WLATS. The following sources of benchmarks were recommended as most appropriate for this analysis (listed in their order of preference):

ATSDR Acute Minimum Risk Levels (MRLs) - ATSDR derives benchmark values for airborne substances that are protective of exposures lasting from 24 hours to 14 days. Since this period includes the 24-hour averaging time of the samples collected in the WLATS, MRLs were used, assuming a 24 hour averaging time, for screening samples for potential acute health effects (ATSDR, 2002).

*California Acute Reference Exposure Levels (RELs)* - The acute RELs are recently derived benchmarks designed to be protective of a resident's short-term exposure to routine emissions from industrial facilities. RELs are generally derived for a 1-hour averaging period and were adjusted (using Haber's Law – see below) to match the 24-hour averaging period of the WLATS measurements (CalEPA, 2002).

*Acute Exposure Guideline Levels (AEGLs)* - AEGLs, developed by the National Advisory Committee of the USEPA Office of Pollution Prevention and Toxics (USEPA, 1997d), may correspond to exposure periods of 1/2, 1, 4, or 8 hours. AEGLs are currently under review, and were used with discretion.

*ERPGs* – The Department of Energy Subcommittee on Consequence Assessment and Protective Action (SCAPA) has developed Emergency Response Planning Guidelines (ERPGs). These acute benchmarks are designed to evaluate the potential consequences of accidental, catastrophic releases of chemicals. ERPGs are derived for a 1-hour averaging period and were adjusted to a 24-hour averaging period using Haber's Law (see Equation 4-3).

Based on this hierarchy of sources of acute benchmarks, AEGLs were not used in this risk assessment.

Some time periods that correspond to particular acute benchmarks required adjustment to the 24-hour averaging time of the WLATS measurements. Where necessary, acute benchmarks (AB) were adjusted using Haber's Law (ten Berge *et al.*, 1986) which states:

$$AB_{24} = AB\tau \ge \left(\frac{\tau}{24}\right)^{\frac{1}{n}}$$
 Equation 4-3

where:

$AB_{24}$	=	acute benchmark concentration based on a 24-hour averaging period
		(appropriate for use in the WLATS risk assessment);
$AB_{\tau}$	=	acute benchmark derived for a time–averaging period of $\tau$ hours;
τ	=	the averaging period (in hours) that corresponds to the acute
		benchmark concentration $AB_{\tau}$ ; and
п	=	an empirical exponent assumed to have a value of 1 for this risk
		assessment (values typically range from 1 to 2.5).

In applying the above equation, the acute benchmark concentrations  $AB_{24}$  and  $AB_{\tau}$  must be expressed in the same units. Also, a value of 1 was assumed for the coefficient n. If there were multiple time values to choose from, the value protective of the mildest effect and most closely matching the desired 24–hour averaging time was used as a starting point for extrapolation.

Given the uncertainties associated with performing acute risk analysis, a continuing evaluation of acute assessment methodologies was undertaken during the course of this risk assessment. However, no updated or alternate acute benchmarks or methodologies were identified that were found to be more appropriate than those identified above.

Table 4-4 contains the acute toxicity values for the COPCs.

		Oral CSF			Inh URE			
COMPOUND	CAS NO.	(1/mg/kg-d)	Source	Date	(1/mg/m3)	Source	Date	WOE $^1$
ACENAPHTHENE	83329							
ACENAPHTHYLENE	208968							D
ACETALDEHYDE	75070				2.2E-03	IRIS	01/01/91	B2
ACETONE	67641							D
ACETOPHENONE	98862							D
ACRYLONITRILE	107131	5.4E-01	IRIS	01/01/91	6.8E-02	IRIS	01/01/91	B1
ALUMINUM	7429905							
ANTHRACENE	120127							D
ANTIMONY	7440360							
ARSENIC	7440382	1.5E+00	IRIS	04/10/98	4.3E+00	IRIS	04/10/98	А
BARIUM	7440393							D
BENZALDEHYDE	100527							
BENZENE	71432	5.5E-02	IRIS	01/19/00	7.8E-03	IRIS	01/19/00	Α
BENZO(B)FLUORANTHENE	205992	1.2E+00	CAL EPA	12/01/02	1.1E-01	CAL EPA	12/01/02	B2
BENZOIC ACID	65850							D
BERYLLIUM	7440417	8.4E+00	CAL EPA	12/01/02	2.4E+00	IRIS	04/03/98	B1
BHC, ALPHA-	319846	6.3E+00	IRIS	07/01/93	1.8E+00	IRIS	07/01/93	B2
BHC, BETA-	319857	1.8E+00	IRIS	07/01/93	5.3E-01	IRIS	07/01/93	С
BHC, GAMMA-	58899	1.1E+00	CAL EPA	12/01/02	3.1E-01	CAL EPA	12/01/02	2B
BIPHENYL, 1,1-	92524							D
BIS(2-ETHYLHEXYL) PHTHALATE	117817	1.4E-02	IRIS	02/01/93	2.4E-03	CAL EPA	12/01/02	B2
BROMODICHLOROMETHANE	75274	6.2E-02	IRIS	03/01/93	1.8E-02	Calc		B2
BROMOFORM	75252	7.9E-03	IRIS	01/01/91	1.1E-03	IRIS	01/01/91	B2
BROMOMETHANE	74839							D
BUTADIENE, 1,3-	106990	6.0E-01	CAL EPA	12/01/02	3.0E-02	IRIS	11/05/02	B2
BUTANOL	71363							D
BUTYL ACRYLATE	141322							3
BUTYLBENZENE, N-	104518							
BUTYLBENZENE, SEC-	135988							
BUTYLBENZENE, TERT-	98066							
CADMIUM	7440439	1.5E+01	CAL EPA	12/01/02	1.8E+00	IRIS	06/01/92	B1

 Table 4-1
 Cancer Toxicity Values for COPCs

		Oral CSF			Inh URE			
COMPOUND	CAS NO.	(1/mg/kg-d)	Source	Date	(1/mg/m3)	Source	Date	WOE $^1$
CALCIUM	7440702							
CAPROLACTAM	105602							4
CARBAZOLE	86748	2.0E-02	HEAST	07/01/97	5.7E-03	Calc		B2
CARBON DISULFIDE	75150							
CARBON TETRACHLORIDE	5.62E+04	1.3E-01	IRIS	06/01/91	1.5E-02	IRIS	06/01/91	B2
CHLORDANE, ALPHA-	5103719	3.5E-01 <sup>a</sup>	IRIS	02/07/98	1.0E-01 <sup>a</sup>	IRIS	02/07/98	B2
CHLORDANE, GAMMA-	5103742	3.5E-01 <sup>a</sup>	IRIS	02/07/98	1.0E-01 <sup>a</sup>	IRIS	02/07/98	B2
CHLORDENE	3734483							
CHLORDENE, ALPHA-	NA							
CHLORDENE, BETA-	NA							
CHLORO-1,3-BUTADIENE, 2-	126998							2B
CHLOROANILINE, 4-	106478							2B
CHLOROBENZENE	108907							D
CHLORODIFLUOROMETHANE	75456							3
CHLOROETHANE	75003							3
CHLOROFORM	67663	1.9E-02	CAL EPA	12/01/02	2.3E-02	IRIS	10/19/01	B2
CHLOROMETHANE	74873							D
CHLOROPRENE	126998							
CHROMIUM (as VI)	18540299	4.2E-01	CAL EPA	12/01/02	1.2E+01	IRIS	09/03/98	Α
COBALT	7440484							2B
COPPER	7440508							D
CYCLOHEXANE	110827							
DDE, 4,4'-	72559	3.4E-01	IRIS	08/22/88	9.7E-02	Calc		B2
DDT, 4,4'-	50293	3.4E-01	IRIS	05/01/91	9.7E-02	IRIS	05/01/91	B2
DIBENZOFURAN	132649							D
DIBROMO-3-CHLOROPROPANE, 1,2-	96128	7.0E+00	CAL EPA	12/01/02	2.0E+00	CAL EPA	12/01/02	2B
DIBROMOCHLOROMETHANE	124481	8.4E-02	IRIS	01/01/92	2.4E-02	Calc		C
DIBROMOMETHANE	74953							
DIBROMOMETHANE, 1,2-	106934							2A
DICHLOROBENZENE, 1,4-	106467	4.0E-02	CAL EPA	12/01/02	1.1E-02	CAL EPA	12/01/02	2B
DICHLORODIFLUOROMETHANE	75718							

## Table 4-1Cancer Toxicity Values for COPCs

		Oral CSF			Inh URE			
COMPOUND	CAS NO.	(1/mg/kg-d)	Source	Date	(1/mg/m3)	Source	Date	WOE <sup>1</sup>
DICHLOROETHANE, 1,1-	75343							С
DICHLOROETHANE, 1,2-	107062	9.1E-02	IRIS	01/01/91	2.6E-02	IRIS	01/01/91	B2
DICHLOROETHENE, 1,1-	75354							С
DICHLOROETHENE, CIS-1,2-	156592							D
DICHLOROPROPANE, 1,2-	78875	6.8E-02	HEAST	07/01/97	1.9E-02	Calc		B2
DICHLOROPROPANE, 1,3-	142289							
DICHLOROPROPANE, 2,2-	594207							
DICHLOROPROPENE, 1,1-	563586							
DICHLOROPROPENE, TRANS-1,3-	10061026	1.0E-01 <sup>b</sup>	IRIS	05/25/00	4.0E-03 <sup>b</sup>	IRIS	05/25/00	B2
DICHLOROTETRAFLUOROETHANE,								
1,2-	76142							
DIELDRIN	60571	1.6E+01	IRIS	07/01/93	4.6E+00	IRIS	07/01/93	B2
DIETHYL PHTHALATE	84662							D
DI-N-BUTYLPHTHALATE	84742							D
DINITROPHENOL, 2,4-	51285							
ENDOSULFAN I	959988							
ETHYL ACRYLATE	140885	4.8E-02	HEAST	07/01/97	1.4E-02	Calc		B2
ETHYL BENZENE	100414							D
FLUORANTHENE	206440							D
FLUORENE	86737							D
FORMALDEHYDE	50000	2.1E-02	CAL EPA	12/01/02	1.3E-02	IRIS	05/01/91	B1
HCL (CALCULATED FROM CL)	7647010							3
HEPTACHLOR	76448	4.5E+00	IRIS	07/01/93	1.3E+00	IRIS	07/01/93	B2
HEPTACHLOR EPOXIDE	1024573	9.1E+00	IRIS	07/01/93	2.6E+00	IRIS	07/01/93	B2
HEPTANE	142825							
HEXACHLOROBUTADIENE	87683	7.8E-02	IRIS	04/01/91	2.2E-02	IRIS	04/01/91	С
HEXANE	110543							
HF (CALCULATED FROM F)	7664393							
IRON	7439896							
ISOPHORONE	78591	9.5E-04	IRIS	11/01/92	2.7E-04	Calc		С
ISOPROPYLBENZENE	98828							D

Table 4-1Cancer Toxicity Values for COPCs

		Oral CSF			Inh URE			
COMPOUND	CAS NO.	(1/mg/kg-d)	Source	Date	(1/mg/m3)	Source	Date	WOE $^1$
LEAD	7439921	8.5E-03	CAL EPA	12/01/02	1.2E-02	CAL EPA	12/01/02	B2
LIMONENE	5989275							3
MAGNESIUM	7439954							
MANGANESE	7439965							D
METHYL ACETATE	79209							
METHYL BUTYL KETONE	591786							
METHYL ETHYL KETONE	78933							D
METHYL ISOBUTYL KETONE	108101							
METHYL METHACRYLATE	80626							Е
METHYL T-BUTYL ETHER	1634044							3
METHYL-4,6-DINITROPHENOL, 2-	534521							
METHYLCYCLOHEXANE	108872							
METHYLENE CHLORIDE	75092	7.5E-03	IRIS	02/01/95	4.7E-04	IRIS	02/01/95	B2
METHYLNAPHTHALENE, 2-	91576							
METHYLPHENOL, 2-	95487							С
METHYLPHENOL, 3- (M-CRESOL)	108394							С
METHYLPHENOL, 4- (P-CRESOL)	106445							С
MOLYBDENUM	7439987							
NAPHTHALENE	91203							С
NICKEL	7440020	9.1E-01	CAL EPA	12/01/02	4.8E-01 <sup>c</sup>	IRIS	01/01/91	А
NITROBENZENE	98953							D
NITROPHENOL, 2-	88755							
NITROPHENOL, 4-	100027							
NITROSODI-N-PROPYLAMINE, N-	621647	7.0E+00	IRIS	07/01/93	2.0E+00	CAL EPA	12/01/02	B2
OXYCHLORDANE	27304138							
PCB Congener #101 (PCBs)	1336363	2.0E+00	IRIS	06/01/97	1.0E-01	IRIS	06/01/97	B2
PCB Congener #153 (PCBs)	1336363	2.0E+00	IRIS	06/01/97	1.0E-01	IRIS	06/01/97	B2
PCB Congener #163 (PCBs)	1336363	2.0E+00	IRIS	06/01/97	1.0E-01	IRIS	06/01/97	B2
PCB Congener #201 (PCBs)	1336363	2.0E+00	IRIS	06/01/97	1.0E-01	IRIS	06/01/97	B2
PCB Congener #209 (PCBs)	1336363	2.0E+00	IRIS	06/01/97	1.0E-01	IRIS	06/01/97	B2
PCB Congener #28 (PCBs)	1336363	2.0E+00	IRIS	06/01/97	1.0E-01	IRIS	06/01/97	B2

## Table 4-1Cancer Toxicity Values for COPCs

		Oral CSF			Inh URE			
COMPOUND	CAS NO.	(1/mg/kg-d)	Source	Date	(1/mg/m3)	Source	Date	WOE <sup>1</sup>
PCB Congener #52 (PCBs)	1336363	2.0E+00	IRIS	06/01/97	1.0E-01	IRIS	06/01/97	B2
PCB Congener #60 (PCBs)	1336363	2.0E+00	IRIS	06/01/97	1.0E-01	IRIS	06/01/97	B2
PENTACHLOROPHENOL	87865	1.2E-01	IRIS	07/01/93	5.1E-03	CAL EPA	12/01/02	B2
PHENANTHRENE	85018							D
PHENOL	108952							D
POTASSIUM	7440097							
PROPYLBENZENE, N-	103651							
PYRENE	129000							D
SELENIUM	7782492							D
SILVER	7440224							D
SODIUM	7440235							
STRONTIUM	7440246							
STYRENE	100425							2B
TETRACHLOROETHANE, 1,1,1,2-	630206	2.6E-02	IRIS	01/01/91	7.4E-03	IRIS	01/01/91	С
TETRACHLOROETHENE	127184	5.1E-02	CAL EPA	12/01/02	5.9E-03	CAL EPA	12/01/02	2A
THALLIUM	7440280							
TIN	7440315							
TITANIUM	7440326							
TOLUENE	108883							D
TOXAPHENE	8001352	1.1E+00	IRIS	01/01/91	3.2E-01	IRIS	01/01/91	B2
TRICHLORO-1,2,2-								
TRIFLUOROETHANE, 1,1,2-	76131							
TRICHLOROBENZENE, 1,2,3-	87616							
TRICHLOROBENZENE, 1,2,4-	120821							D
TRICHLOROETHANE, 1,1,1-	71556							D
TRICHLOROETHANE, 1,1,2-	79005	5.7E-02	IRIS	02/01/94	1.6E-02	IRIS	02/01/94	C
TRICHLOROETHENE	79016	1.5E-02	CAL EPA	12/01/02	2.0E-03	CAL EPA	12/01/02	2A
TRICHLOROFLUOROMETHANE	75694							
TRICHLOROPHENOL, 2,4,5-	95954							
TRICHLOROPHENOL, 2,4,6-	88062	1.1E-02	IRIS	02/01/94	3.1E-03	IRIS	02/01/94	B2
TRICHLOROPROPANE, 1,2,3-	96184	7.0E+00	HEAST	07/01/97	2.0E+00	Calc		B2
TRIMETHYLBENZENE, 1,2,4-	95636							

## Table 4-1Cancer Toxicity Values for COPCs

		Oral CSF			Inh URE			
COMPOUND	CAS NO.	(1/mg/kg-d)	Source	Date	(1/mg/m3)	Source	Date	WOE <sup>1</sup>
TRIMETHYLBENZENE, 1,3,5-	108678							
VANADIUM	7440622							
VINYL CHLORIDE	75014	1.5E+00	IRIS	08/07/00	8.8E-03	IRIS	08/07/00	Α
XYLENE, m-	108383							D
XYLENE, o-	95476							D
XYLENE, p-	106423							D
YTTRIUM	7440655							
ZINC	7440666							D

 Table 4-1
 Cancer Toxicity Values for COPCs

<sup>a</sup> Values for Technical Chlordane CAS# 12789036

<sup>b</sup> Values for 1,3-Dichloropropene CAS# 542756

<sup>c</sup> URE value for Nickel Subsulfide CAS#12035722

<sup>1</sup> EPA / IARC WOE Codes: Weight-of-Evidence

A / 1 Known human carcinogen

B1, B2 / 2A Probable human carcinogen

C / 2B Possible human carcinogen

D/3 Not classifiable

E / 4 Evidence of noncarcinogenicity / probably not carcinogenic

		Oral RfD			Oral RfD	Inh RfC			Inh RfC
COMPOUND	CAS NO.	(mg/kg-d)	Source	Date	UF / MF $^{\rm 1}$	(mg/m3)	Source	Date	UF / MF $^1$
ACENAPHTHENE	83329	6.0E-02	IRIS	04/01/94	3000 / 1	2.1E-01	Calc		
ACENAPHTHYLENE	208968								
ACETALDEHYDE	75070					9.0E-03	IRIS	10/01/91	1000 / 1
ACETONE	67641	1.0E-01	IRIS	08/01/93	1000 / 1	3.1E+01	ATSDR	05/01/94	100
ACETOPHENONE	98862	1.0E-01	IRIS	01/01/89	3000 / 1	3.5E-01	Calc		
ACRYLONITRILE	107131					2.0E-03	IRIS	12/01/91	1000 / 1
ALUMINUM	7429905								
ANTHRACENE	120127	3.0E-01	IRIS	07/01/93	3000 / 1	1.1E+00	Calc		
ANTIMONY	7440360	4.0E-04	IRIS	02/01/91	1000 / 1	1.4E-03	Calc		
ARSENIC	7440382	3.0E-04	IRIS	02/01/93	3 / 1	3.0E-05	CAL EPA	09/01/02	1000
BARIUM	7440393	7.0E-02	IRIS	01/21/99	3 / 1	2.5E-01	Calc		
BENZALDEHYDE	100527	1.0E-01	IRIS	09/07/88	1000 / 1	3.5E-01	Calc		
BENZENE	71432					6.0E-02	CAL EPA	09/01/02	10
BENZO(B)FLUORANTHENE	205992								
BENZOIC ACID	65850	4.0E+00	IRIS	07/01/93	1 / 1	1.4E+01	Calc		
BERYLLIUM	7440417	2.0E-03	IRIS	04/03/98	300 / 1	2.0E-05	IRIS	04/03/98	10 / 1
BHC, ALPHA-	319846	8.0E-03	ATSDR	07/01/99	100	2.8E-02	Calc		
BHC, BETA-	319857								
BHC, GAMMA-	58899	3.0E-04	IRIS	03/01/88	1000 / 1	1.1E-03	Calc		
BIPHENYL, 1,1-	92524	5.0E-02	IRIS	08/01/89	100 / 10	1.8E-01	Calc		
BIS(2-ETHYLHEXYL) PHTHALATE	117817	2.0E-02	IRIS	05/01/91	1000 / 1	7.0E-02	Calc		
BROMODICHLOROMETHANE	75274	2.0E-02	IRIS	03/01/91	1000 / 1	7.0E-02	Calc		
BROMOFORM	75252	2.0E-02	IRIS	03/01/91	1000 / 1	7.0E-02	Calc		
BROMOMETHANE	74839	1.4E-03	IRIS	07/01/91	1000 / 1	5.0E-03	IRIS	10/01/92	100 / 1
BUTADIENE, 1,3-	106990					2.0E-03	IRIS	11/05/02	1000 / 1
BUTANOL	71363	1.0E-01	IRIS	09/01/90	1000 / 1	3.5E-01	Calc		
BUTYL ACRYLATE	141322								
BUTYLBENZENE, N-	104518								
BUTYLBENZENE, SEC-	135988								
BUTYLBENZENE, TERT-	98066								

		Oral RfD			Oral RfD	Inh RfC			Inh RfC
COMPOUND	CAS NO.	(mg/kg-d)	Source	Date	UF / MF $^1$	(mg/m3)	Source	Date	UF / MF $^1$
CADMIUM	7440439	5.0E-04	IRIS	02/01/94	10 / 1	2.0E-05	CAL EPA	09/01/02	30
CALCIUM	7440702								
CAPROLACTAM	105602	5.0E-01	IRIS	09/07/88	100 / 1	1.8E+00	Calc		
CARBAZOLE	86748								
CARBON DISULFIDE	75150	1.0E-01	IRIS	09/01/90	100 / 1	7.0E-01	IRIS	08/01/95	30 / 1
CARBON TETRACHLORIDE	5.62E+04	7.0E-04	IRIS	06/01/91	1000 / 1	4.0E-02	CAL EPA	09/01/02	300
CHLORDANE, ALPHA-	5103719	5.0E-04 <sup>a</sup>	IRIS	02/07/98	300 / 1	7.0E-04 <sup>a</sup>	IRIS	02/07/98	1000 / 1
CHLORDANE, GAMMA-	5103742	5.0E-04 <sup>a</sup>	IRIS	02/07/98	300 / 1	7.0E-04 <sup>a</sup>	IRIS	02/07/98	1000 / 1
CHLORDENE	3734483								
CHLORDENE, ALPHA-	NA								
CHLORDENE, BETA-	NA								
CHLORO-1,3-BUTADIENE, 2-	126998								
CHLOROANILINE, 4-	106478	4.0E-03	IRIS	02/01/95	3000 / 1	1.4E-02	Calc		
CHLOROBENZENE	108907	2.0E-02	IRIS	07/01/93	1000 / 1	1.0E+00	CAL EPA	09/01/02	100
CHLORODIFLUOROMETHANE	75456					5.0E+01	IRIS	11/01/93	100 / 1
CHLOROETHANE	75003					1.0E+01	IRIS	04/01/91	300 / 1
CHLOROFORM	67663	1.0E-02	IRIS	10/19/01	1000 / 1	9.8E-02	ATSDR	09/01/97	100
CHLOROMETHANE	74873					9.0E-02	IRIS	07/17/01	1000 / 1
CHLOROPRENE	126998					7.0E-03	HEAST	07/01/97	300
CHROMIUM (as VI)	18540299	3.0E-03	IRIS	09/03/98	300 / 3	1.0E-04 <sup>b</sup>	IRIS	09/03/98	300 / 1
COBALT	7440484					1.0E-04	ATSDR	09/01/01	10
COPPER	7440508								
CYCLOHEXANE	110827								
DDE, 4,4'-	72559								
DDT, 4,4'-	50293	5.0E-04	IRIS	02/01/96	100 / 1	1.8E-03	Calc		
DIBENZOFURAN	132649								
DIBROMO-3-CHLOROPROPANE, 1,2-	96128					2.0E-04	IRIS	10/01/91	1000 / 1
DIBROMOCHLOROMETHANE	124481	2.0E-02	IRIS	03/01/91	1000 / 1	7.0E-02	Calc		
DIBROMOMETHANE	74953								
DIBROMOMETHANE, 1,2-	106934								
DICHLOROBENZENE, 1,4-	106467					8.0E-01	IRIS	11/01/96	100 / 1

		Oral RfD			Oral RfD	Inh RfC			Inh RfC
COMPOUND	CAS NO.	(mg/kg-d)	Source	Date	UF / MF $^{\rm 1}$	(mg/m3)	Source	Date	UF / MF $^1$
DICHLORODIFLUOROMETHANE	75718	2.0E-01	IRIS	11/01/95	100 / 1	7.0E-01	Calc		
DICHLOROETHANE, 1,1-	75343								
DICHLOROETHANE, 1,2-	107062					2.4E+00	ATSDR	09/01/01	90
DICHLOROETHENE, 1,1-	75354	5.0E-02	IRIS	08/13/02	100 / 1	2.0E-01	IRIS	08/13/02	30 / 1
DICHLOROETHENE, CIS-1,2-	156592	1.0E-02	HEAST	07/01/97	3000	3.5E-02	Calc		
DICHLOROPROPANE, 1,2-	78875					4.0E-03	IRIS	12/01/91	300 / 1
DICHLOROPROPANE, 1,3-	142289								
DICHLOROPROPANE, 2,2-	594207								
DICHLOROPROPENE, 1,1-	563586								
DICHLOROPROPENE, TRANS-1,3-	10061026	3.0E-02 °	IRIS	05/25/00	100 / 1	2.0E-02 °	IRIS	05/25/00	30 / 1
DICHLOROTETRAFLUOROETHANE,									
1,2-	76142								
DIELDRIN	60571	5.0E-05	IRIS	09/01/90	100 / 1	1.8E-04	Calc		
DIETHYL PHTHALATE	84662	8.0E-01	IRIS	02/01/93	1000 / 1	2.8E+00	Calc		
DI-N-BUTYLPHTHALATE	84742	1.0E-01	IRIS	08/01/90	1000 / 1	3.5E-01	Calc		
DINITROPHENOL, 2,4-	51285	2.0E-03	IRIS	07/01/91	1000 / 1	7.0E-03	Calc		
ENDOSULFAN I	959988	6.0E-03 <sup>d</sup>	IRIS	10/01/94	100 / 1	2.1E-02	Calc		
ETHYL ACRYLATE	140885								
ETHYL BENZENE	100414	1.0E-01	IRIS	06/01/91	1000 / 1	1.0E+00	IRIS	03/01/91	300 / 1
FLUORANTHENE	206440	4.0E-02	IRIS	07/01/93	3000 / 1	1.4E-01	Calc		
FLUORENE	86737	4.0E-02	IRIS	11/01/90	3000 / 1	1.4E-01	Calc		
FORMALDEHYDE	50000	2.0E-01	IRIS	09/01/90	100 / 1	9.8E-03	ATSDR	07/01/99	30
HCL (CALCULATED FROM CL)	7647010					2.0E-02	IRIS	07/01/95	300 / 1
HEPTACHLOR	76448	5.0E-04	IRIS	03/01/91	300 / 1	1.8E-03	Calc		
HEPTACHLOR EPOXIDE	1024573	1.3E-05	IRIS	03/01/91	1000 / 1	4.6E-05	Calc		
HEPTANE	142825								
HEXACHLOROBUTADIENE	87683	2.0E-04	HEAST	07/01/97	1000	7.0E-04	Calc		
HEXANE	110543	6.0E-02	HEAST	07/01/97	10000	2.0E-01	IRIS	07/01/93	300 / 1
HF (CALCULATED FROM F)	7664393								
IRON	7439896								
ISOPHORONE	78591	2.0E-01	IRIS	01/01/91	1000 / 1	2.0E+00	CAL EPA	09/01/02	30

		Oral RfD			Oral RfD	Inh RfC			Inh RfC
COMPOUND	CAS NO.	(mg/kg-d)	Source	Date	UF / MF $^{\rm 1}$	(mg/m3)	Source	Date	UF / MF $^1$
ISOPROPYLBENZENE	98828	1.0E-01	IRIS	08/01/97	1000 / 1	4.0E-01	IRIS	08/01/97	1000 / 1
LEAD	7439921								
LIMONENE	5989275								
MAGNESIUM	7439954								
MANGANESE	7439965	1.4E-01	IRIS	05/01/96	1 / 1	5.0E-05	IRIS	12/01/93	1000 / 1
METHYL ACETATE	79209	1.0E+00	HEAST	07/01/97	1000	3.5E+00	Calc		
METHYL BUTYL KETONE	591786								
METHYL ETHYL KETONE	78933	6.0E-01	IRIS	05/01/93	3000 / 1	1.0E+00	IRIS	07/01/92	1000 / 3
METHYL ISOBUTYL KETONE	108101	8.0E-02	HEAST	07/01/97	3000	2.8E-01	Calc		
METHYL METHACRYLATE	80626	1.4E+00	IRIS	03/02/98	100 / 1	7.0E-01	IRIS	03/02/98	10 / 1
METHYL T-BUTYL ETHER	1634044					3.0E+00	IRIS	09/01/93	100 / 1
METHYL-4,6-DINITROPHENOL, 2-	534521								
METHYLCYCLOHEXANE	108872					3.0E+00	HEAST	07/01/97	100
METHYLENE CHLORIDE	75092	6.0E-02	IRIS	03/01/88	100 / 1	1.0E+00	ATSDR	09/01/00	30
METHYLNAPHTHALENE, 2-	91576								
METHYLPHENOL, 2-	95487	5.0E-02	IRIS	09/01/90	1000 / 1	6.0E-01 <sup>e</sup>	CAL EPA	09/01/02	300
METHYLPHENOL, 3- (M-CRESOL)	108394	5.0E-02	IRIS	09/01/90	1000 / 1	6.0E-01 <sup>e</sup>	CAL EPA	09/01/02	300
METHYLPHENOL, 4- (P-CRESOL)	106445	5.0E-03	HEAST	07/01/97	1000	6.0E-01 <sup>e</sup>	CAL EPA	09/01/02	300
MOLYBDENUM	7439987	5.0E-03	IRIS	08/01/93	30 / 1	1.8E-02	Calc		
NAPHTHALENE	91203	2.0E-02	IRIS	09/17/98	3000 / 1	3.0E-03	IRIS	09/17/98	3000 / 1
NICKEL	7440020					2.0E-04	ATSDR	09/01/97	30
NITROBENZENE	98953	5.0E-04	IRIS	01/01/91	10000 / 1	1.8E-03	Calc		
NITROPHENOL, 2-	88755								
NITROPHENOL, 4-	100027								
NITROSODI-N-PROPYLAMINE, N-	621647								
OXYCHLORDANE	27304138								
PCB Congener #101 (PCBs)	1336363	2.0E-05	IRIS	11/01/96	300 / 1	7.0E-05	Calc		
PCB Congener #153 (PCBs)	1336363	2.0E-05	IRIS	11/01/96	300 / 1	7.0E-05	Calc		
PCB Congener #163 (PCBs)	1336363	2.0E-05	IRIS	11/01/96	300 / 1	7.0E-05	Calc		
PCB Congener #201 (PCBs)	1336363	2.0E-05	IRIS	11/01/96	300 / 1	7.0E-05	Calc		
PCB Congener #209 (PCBs)	1336363	2.0E-05	IRIS	11/01/96	300 / 1	7.0E-05	Calc		

		Oral RfD			Oral RfD	Inh RfC			Inh RfC
COMPOUND	CAS NO.	(mg/kg-d)	Source	Date	UF / MF $^{\rm 1}$	(mg/m3)	Source	Date	UF / MF $^1$
PCB Congener #28 (PCBs)	1336363	2.0E-05	IRIS	11/01/96	300 / 1	7.0E-05	Calc		
PCB Congener #52 (PCBs)	1336363	2.0E-05	IRIS	11/01/96	300 / 1	7.0E-05	Calc		
PCB Congener #60 (PCBs)	1336363	2.0E-05	IRIS	11/01/96	300 / 1	7.0E-05	Calc		
PENTACHLOROPHENOL	87865	3.0E-02	IRIS	02/01/93	100 / 1	1.1E-01	Calc		
PHENANTHRENE	85018								
PHENOL	108952	3.0E-01	IRIS	09/30/02	300 / 1	2.0E-01	CAL EPA	09/01/02	100
POTASSIUM	7440097								
PROPYLBENZENE, N-	103651								
PYRENE	129000	3.0E-02	IRIS	07/01/93	3000 / 1	1.1E-01	Calc		
SELENIUM	7782492	5.0E-03	IRIS	09/01/91	3 / 1	2.0E-02	CAL EPA	09/01/02	3
SILVER	7440224	5.0E-03	IRIS	12/01/96	3 / 1	1.8E-02	Calc		
SODIUM	7440235								
STRONTIUM	7440246	6.0E-01	IRIS	12/01/96	300 / 1	2.1E+00	Calc		
STYRENE	100425	2.0E-01	IRIS	09/01/90	1000 / 1	1.0E+00	IRIS	07/01/93	30 / 1
TETRACHLOROETHANE, 1,1,1,2-	630206	3.0E-02	IRIS	12/01/96	3000 / 1	1.1E-01	Calc		
TETRACHLOROETHENE	127184	1.0E-02	IRIS	03/01/88	1000 / 1	2.7E-01	ATSDR	09/01/97	100
THALLIUM	7440280								
TIN	7440315	6.0E-01	HEAST	07/01/97	100	2.1E+00	Calc		
TITANIUM	7440326								
TOLUENE	108883	2.0E-01	IRIS	04/01/94	1000 / 1	4.0E-01	IRIS	08/01/92	300 / 1
TOXAPHENE	8001352								
TRICHLORO-1,2,2-									
TRIFLUOROETHANE, 1,1,2-	76131	3.0E+01	IRIS	02/01/96	10 / 1	1.1E+02	Calc		
TRICHLOROBENZENE, 1,2,3-	87616								
TRICHLOROBENZENE, 1,2,4-	120821	1.0E-02	IRIS	11/01/96	1000 / 1	2.0E-01	HEAST	07/01/97	1000
TRICHLOROETHANE, 1,1,1-	71556								
TRICHLOROETHANE, 1,1,2-	79005	4.0E-03	IRIS	02/01/95	1000 / 1	1.4E-02	Calc		
TRICHLOROETHENE	79016					6.0E-01	CAL EPA	09/01/02	100
TRICHLOROFLUOROMETHANE	75694	3.0E-01	IRIS	08/01/92	1000 / 1	1.1E+00	Calc		
TRICHLOROPHENOL, 2,4,5-	95954	1.0E-01	IRIS	03/01/88	1000 / 1	3.5E-01	Calc		
TRICHLOROPHENOL, 2,4,6-	88062								
TRICHLOROPROPANE, 1,2,3-	96184	6.0E-03	IRIS	08/01/90	1000 / 1	2.1E-02	Calc		

		Oral RfD			Oral RfD	Inh RfC			Inh RfC
COMPOUND	CAS NO.	(mg/kg-d)	Source	Date	UF / MF $^1$	(mg/m3)	Source	Date	UF / MF $^1$
TRIMETHYLBENZENE, 1,2,4-	95636								
TRIMETHYLBENZENE, 1,3,5-	108678								
VANADIUM	7440622	7.0E-03	HEAST	07/01/97	100	2.5E-02	Calc		
VINYL CHLORIDE	75014	3.0E-03	IRIS	08/07/00	30 / 1	1.0E-01	IRIS	08/07/00	30 / 1
XYLENE, m-	108383	2.0E-01 <sup>f</sup>	IRIS	02/21/03	1000 / 1	1.0E-01 <sup>f</sup>	IRIS	02/21/03	300 / 1
XYLENE, o-	95476	2.0E-01 <sup>f</sup>	IRIS	02/21/03	1000 / 1	1.0E-01 <sup>f</sup>	IRIS	02/21/03	300 / 1
XYLENE, p-	106423	2.0E-01 <sup>f</sup>	IRIS	02/21/03	1000 / 1	1.0E-01 <sup>f</sup>	IRIS	02/21/03	300 / 1
YTTRIUM	7440655								
ZINC	7440666	3.0E-01	IRIS	10/01/92	3 / 1	1.1E+00	Calc		

 Table 4-2 Non-cancer Toxicity Values for COPCs

<sup>a</sup> Values for Technical Chlordane CAS# 12789036

<sup>1</sup> UF / MF Uncertainty Factor / Modifying Factor

<sup>b</sup> RfC Value for particulate Cr VI

<sup>c</sup> Values for 1,3-Dichloropropene CAS# 542756

<sup>d</sup> Value for Endosulfan CAS# 115297

<sup>e</sup> RfC value for Cresol Mixture CAS# 1319773

<sup>f</sup> Values for Xylene Mixture CAS# 1330207

#### Table 4-3 Non-cancer Critical Effect

			Target Organs for Other Inhalation Chronic Effects									
COMPOUND	Source	Critical Effect for Derivation of Toxicity Value	NEUR	RSP	CARD	DEV	SKIN	HEM	IMM	RPR	REN	HEP
ACENAPHTHENE												Í
ACENAPHTHYLENE												
ACETALDEHYDE	IRIS	Degeneration of olfactory epithelium						1				
ACETONE	ATSDR	Neurological endpoint	X									
ACETOPHENONE												
ACRYLONITRILE	IRIS	Degeneration and inflammation of nasal respiratory epithelium; hyperplasia of mucous secreting cells		X		X						
ALUMINUM												
ANTHRACENE					Ì			Ì			Ì	
ANTIMONY					Ì			Ì			Ì	
ARSENIC	CALEPA	Decreased fetal weight; increased incidences of intrauterine growth retardation and skeletal malformations in mice	X		X		X					
BARIUM								1				
BENZALDEHYDE								1				
BENZENE	CALEPA	Lowered red and white blood cell counts in occupationally exposed humans	X					X	X			
BENZO(B)FLUORANTHENE								1				
BENZOIC ACID												
BERYLLIUM	IRIS	Beryllium sensitization and progression to CBD		Х								
BHC, ALPHA-												
BHC, BETA-												
BHC, GAMMA-												
BIPHENYL, 1,1-												
BIS(2-ETHYLHEXYL) PHTHALATE												
BROMODICHLOROMETHANE												

#### Table 4-3 Non-cancer Critical Effect

			Target Organs for Other Inhalation C								nronic Effects			
COMPOUND	Source	Critical Effect for Derivation of Toxicity Value	NEUR	RSP	CARD	DEV	SKIN	HEM	IMM	RPR	REN	HEP		
BROMOFORM											X	X		
BROMOMETHANE	IRIS	Degenerative and proliferative lesions of the olfactory epithelium of the nasal cavity	X	Х										
BUTADIENE, 1,3-	IRIS	Ovarian atrophy		Х		Х	Ì	Ì	Ì	Х	Ì			
BUTANOL							Ì	Ì	Ì		Ì			
BUTYL ACRYLATE							Ì	Ì	Ì		Ì			
BUTYLBENZENE, N-							Ì	Ì	Ì		Ì			
BUTYLBENZENE, SEC-					1									
BUTYLBENZENE, TERT-					1									
CADMIUM	CALEPA	Kidney effects (proteinuria) and respiratory effects (reduction in forced vital capacity and reduction in peak expiratory flow rate) in occupationally exposed humans		Х							X			
CALCIUM			Ì		1		1	Ì	Ì		1			
CAPROLACTAM														
CARBAZOLE														
CARBON DISULFIDE	IRIS	Peripheral nervous system dysfunction	X		X	Х				Х				
CARBON TETRACHLORIDE	CALEPA	Increased liver weight and hepatic fatty infiltration in guinea pigs										X		
CHLORDANE, ALPHA-	IRIS	Hepatic effects			1			1				X		
CHLORDANE, GAMMA-	IRIS	Hepatic effects										X		
CHLORDENE														
CHLORDENE, ALPHA-														
CHLORDENE, BETA-														
CHLORO-1,3-BUTADIENE, 2-														
CHLOROANILINE, 4-														
CHLOROBENZENE	CALEPA	Increased liver weights, hepatocellular hypertrophy, renal degeneration and inflammation, and	X								X	X		

			Target Organs for Other Inhalation Chronic Effects									
COMPOUND	Source	Critical Effect for Derivation of Toxicity Value	NEUR	RSP	CARD	DEV	SKIN	HEM	IMM	RPR	REN	HEP
		testicular degeneration in rats										
CHLORODIFLUOROMETHANE	IRIS	Increased kidney, adrenal and										
	intio	pituitary weights (other effect:										
		Reduced maternal weight gain )										
CHLOROETHANE	IRIS	Delayed fetal ossification			Ì	Х						
CHLOROFORM	ATSDR	Hepatic endpoint	1		Ì							X
CHLOROMETHANE	IRIS	Cerebellar lesions	X		İ		İ				İ	
CHLOROPRENE			Ì		İ		İ				İ	
CHROMIUM (as VI)	IRIS	Nasal septum atrophy. Lactate dehydrogenase in bronchioalveolar lavage fluid		Х								
COBALT	ATSDR	Respiratory endpoint		Х								
COPPER												
CYCLOHEXANE												
DDE, 4,4'-												
DDT, 4,4'-												
DIBENZOFURAN												
DIBROMO-3-CHLOROPROPANE, 1,2-	IRIS	Testicular effects										
DIBROMOCHLOROMETHANE												
DIBROMOMETHANE												
DIBROMOMETHANE, 1,2-												
DICHLOROBENZENE, 1,4-	IRIS	Increased liver weights in P1 males										
DICHLORODIFLUOROMETHANE												
DICHLOROETHANE, 1,1-												
DICHLOROETHANE, 1,2-	ATSDR	Hepatic endpoint										
DICHLOROETHENE, 1,1-	IRIS	Liver toxicity (fatty change)									Х	Х
DICHLOROETHENE, CIS-1,2-												Х
DICHLOROPROPANE, 1,2-	IRIS	Hyperplasia of the nasal mucosa										Х
DICHLOROPROPANE, 1,3-												

			Target Organs for Other Inhalation Chronic Effects									
COMPOUND	Source	Critical Effect for Derivation of Toxicity Value	NEUR	RSP	CARD	DEV	SKIN	HEM	IMM	RPR	REN	HEP
DICHLOROPROPANE, 2,2-												
DICHLOROPROPENE, 1,1-												
DICHLOROPROPENE, TRANS-1,3-	IRIS	Hypertrophy/ hyperplasia of the nasal respiratory epithelium										
DICHLOROTETRAFLUOROETHANE, 1,2-												
DIELDRIN												
DIETHYL PHTHALATE												
DI-N-BUTYLPHTHALATE									1			
DINITROPHENOL, 2,4-									1			
ENDOSULFAN I												
ETHYL ACRYLATE												
ETHYL BENZENE	IRIS	Developmental toxicity		Х					1		Х	Х
FLUORANTHENE												
FLUORENE												
FORMALDEHYDE	ATSDR	Respiratory endpoint		Х								
HCL (CALCULATED FROM CL)	IRIS	Hyperplasia of nasal mucosa larynx and trachea										
HEPTACHLOR												
HEPTACHLOR EPOXIDE												
HEPTANE												
HEXACHLOROBUTADIENE												
HEXANE	IRIS	Neurotoxicity; electrophysiological alterations (other effect: Epithelial lesions in the nasal cavity )	X									
HF (CALCULATED FROM F)												
IRON												
ISOPHORONE	CALEPA	Developmental effects (reduced crown-rump length of female rat fetuses); teratogenicity (exencephaly in fetal rats and mice) in range finding study at 150 ppm				Х						

	Target Organs for Other Inhalation Chronic Effe							ects				
COMPOUND	Source	Critical Effect for Derivation of Toxicity Value	NEUR	RSP	CARD	DEV	SKIN	HEM	IMM	RPR	REN	HEP
ISOPROPYLBENZENE	IRIS	Increased kidney weights in female rats and adrenal weights in male and female rats										
LEAD												
LIMONENE												
MAGNESIUM												
MANGANESE	IRIS	Impairment of neurobehavioral function (other effect: Impairment of neurobehavioral function )	X									
METHYL ACETATE												
METHYL BUTYL KETONE												
METHYL ETHYL KETONE	IRIS	Decreased fetal birth weight										
METHYL ISOBUTYL KETONE												
METHYL METHACRYLATE	IRIS	Degeneration/ atrophy of olfactory epithelium (male rats)										
METHYL T-BUTYL ETHER	IRIS	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	
METHYL-4,6-DINITROPHENOL, 2-												
METHYLCYCLOHEXANE												
METHYLENE CHLORIDE	ATSDR	Hepatic endpoint										Х
METHYLNAPHTHALENE, 2-												
METHYLPHENOL, 2-		Decreased body weights and neurotoxicity (tremors, salivation, lacrimation, etc.)	X	Х								
METHYLPHENOL, 3- (M-CRESOL)	CALEPA	Decreased body weights and neurotoxicity (tremors, salivation, lacrimation, etc.)	x	Х								

				Tar	get Org	ans for	Other I	nhalatio	on Chro	nic Effe	ects	
COMPOUND	Source	Critical Effect for Derivation of Toxicity Value	NEUR	RSP	CARD	DEV	SKIN	HEM	IMM	RPR	REN	HEP
METHYLPHENOL, 4- (P-CRESOL)	CALEPA	Decreased body weights and neurotoxicity (tremors, salivation, lacrimation, etc.)	X	X								
MOLYBDENUM												
NAPHTHALENE	IRIS	Nasal effects: hyperplasia and metaplasia in respiratory and olfactory epithelium, respectively		X								
NICKEL	ATSDR	Respiratory endpoint		Х								
NITROBENZENE												
NITROPHENOL, 2-												
NITROPHENOL, 4-												
NITROSODI-N-PROPYLAMINE, N-												
OXYCHLORDANE												
PCB Congener #101 (PCBs)												
PCB Congener #153 (PCBs)												
PCB Congener #163 (PCBs)												
PCB Congener #201 (PCBs)												
PCB Congener #209 (PCBs)												
PCB Congener #28 (PCBs)												
PCB Congener #52 (PCBs)												
PCB Congener #60 (PCBs)												
PENTACHLOROPHENOL												
PHENANTHRENE												
PHENOL	CALEPA	Systemic effects including liver and nervous system effects	X		X						X	X
POTASSIUM												
PROPYLBENZENE, N-												
PYRENE												
SELENIUM	CALEPA	Clinical selenosis (liver, blood, skin, CNS)		Х								
SILVER												

				Taı	rget Org	ans for	Other I	nhalatio	on Chro	nic Effe	ects	
COMPOUND	Source	Critical Effect for Derivation of Toxicity Value	NEUR	RSP	CARD	DEV	SKIN	HEM	IMM	RPR	REN	HEP
SODIUM												
STRONTIUM												
STYRENE	IRIS	CNS effects	Х									
TETRACHLOROETHANE, 1,1,1,2-							1		1		1	
TETRACHLOROETHENE	ATSDR	Neurological endpoint	X									
THALLIUM												
TIN												
TITANIUM	Ì		İ		1		Ì		Ì		Ì	
TOLUENE	IRIS	Neurological effects (other effect: Degeneration of nasal epithelium)	X	Х								
TOXAPHENE												
TRICHLORO-1,2,2-	Ì		İ		1		Ì		Ì		Ì	
TRIFLUOROETHANE, 1,1,2-												
TRICHLOROBENZENE, 1,2,3-												
TRICHLOROBENZENE, 1,2,4-												
TRICHLOROETHANE, 1,1,1-												
TRICHLOROETHANE, 1,1,2-												ĺ
TRICHLOROETHENE	CALEPA	Drowsiness, fatigue, headache, and eye irritation	Х									
TRICHLOROFLUOROMETHANE												
TRICHLOROPHENOL, 2,4,5-												
TRICHLOROPHENOL, 2,4,6-												
TRICHLOROPROPANE, 1,2,3-												
TRIMETHYLBENZENE, 1,2,4-												
TRIMETHYLBENZENE, 1,3,5-												
VANADIUM												
VINYL CHLORIDE	IRIS	Liver cell polymorphism	X									Х
XYLENE, m-	CALEPA	Dose related increase in the prevalence of eye irritation, sore throat, floating sensation, and poor appetite.	x									

			Target Organs for Other Inhalation Chronic Effects									
COMPOUND	Source	Critical Effect for Derivation of Toxicity Value	NEUR	RSP	CARD	DEV	SKIN	HEM	IMM	RPR	REN	HEP
XYLENE, o-		Dose related increase in the prevalence of eye irritation, sore throat, floating sensation, and poor appetite.	X									
XYLENE, p-	CALEPA	Dose related increase in the prevalence of eye irritation, sore throat, floating sensation, and poor appetite.	x									
YTTRIUM												
ZINC												

Chronic Effects Codes: NEUR Neurological

RSPRespiratoryCARDCardiovascularDEVDevelopmentalSKINSkinHEMHematologicalIMMImmunologicalRPRReproductiveRENRenalHEPHepatic

		Inh RfC			Exposure	24 Hr RfC
COMPOUND	CAS NO.	(mg/m3)	Source	Date	Duration (hrs)	(mg/m3)
ACENAPHTHENE	83329					
ACENAPHTHYLENE	208968					
ACETALDEHYDE	75070	1.8E+01	ERPG-1	12/01/02	1	7.5E-01
ACETONE	67641	6.2E+01	ATSDR	05/01/94	24	6.2E+01
ACETOPHENONE	98862					
ACRYLONITRILE	107131	2.2E-01	ATSDR	12/01/90	24	2.2E-01
ALUMINUM	7429905					
ANTHRACENE	120127					
ANTIMONY	7440360					
ARSENIC	7440382	1.9E-04	CAL EPA	05/01/00	4	3.2E-05
BARIUM	7440393					
BENZALDEHYDE	100527					
BENZENE	71432	1.6E-01	ATSDR	09/01/97	24	1.6E-01
BENZO(B)FLUORANTHENE	205992					
BENZOIC ACID	65850					
BERYLLIUM	7440417	2.5E-02	ERPG-2 <sup>1</sup>	12/01/02	1	1.0E-03
BHC, ALPHA-	319846					
BHC, BETA-	319857					
BHC, GAMMA-	58899					
BIPHENYL, 1,1-	92524					
BIS(2-ETHYLHEXYL) PHTHALATE	117817					
BROMODICHLOROMETHANE	75274					
BROMOFORM	75252					
BROMOMETHANE	74839	1.9E-01	ATSDR	09/01/92	24	1.9E-01
BUTADIENE, 1,3-	106990	2.2E+01	ERPG-1	12/01/02	1	9.2E-01
BUTANOL	71363					
BUTYL ACRYLATE	141322	1.3E+02	ERPG-2 <sup>1</sup>	12/01/02	1	5.2E+00
BUTYLBENZENE, N-	104518					
BUTYLBENZENE, SEC-	135988					
BUTYLBENZENE, TERT-	98066					

 Table 4-4 Acute Toxicity Values for COPCs

		Inh RfC			Exposure	24 Hr RfC
COMPOUND	CAS NO.	(mg/m3)	Source	Date	Duration (hrs)	(mg/m3)
CADMIUM	7440439					
CALCIUM	7440702					
CAPROLACTAM	105602					
CARBAZOLE	86748					
CARBON DISULFIDE	75150	6.2E+00	CAL EPA	05/01/00	6	1.6E+00
CARBON TETRACHLORIDE	56235	1.3E+00	ATSDR	05/01/94	24	1.3E+00
CHLORDANE, ALPHA-	5103719					
CHLORDANE, GAMMA-	5103742					
CHLORDENE	3734483					
CHLORDENE, ALPHA-	NA					
CHLORDENE, BETA-	NA					
CHLORO-1,3-BUTADIENE, 2-	126998					
CHLOROANILINE, 4-	106478					
CHLOROBENZENE	108907					
CHLORODIFLUOROMETHANE	75456					
CHLOROETHANE	75003	4.0E+01	ATSDR	12/01/98	24	4.0E+01
CHLOROFORM	67663	4.9E-01	ATSDR	09/01/97	24	4.9E-01
CHLOROMETHANE	74873	1.0E+00	ATSDR	12/01/98	24	1.0E+00
CHLOROPRENE	126998					
CHROMIUM (as VI)	18540299					
COBALT	7440484					
COPPER	7440508	1.0E-01	CAL EPA	05/01/00	1	4.2E-03
CYCLOHEXANE	110827					
DDE, 4,4'-	72559					
DDT, 4,4'-	50293					
DIBENZOFURAN	132649					
DIBROMO-3-CHLOROPROPANE, 1,2-	96128					
DIBROMOCHLOROMETHANE	124481					
DIBROMOMETHANE	74953					
DIBROMOMETHANE, 1,2-	106934					
DICHLOROBENZENE, 1,4-	106467	4.8E+00	ATSDR	12/01/98	24	4.8E+00
DICHLORODIFLUOROMETHANE	75718					

 Table 4-4 Acute Toxicity Values for COPCs

		Inh RfC			Exposure	24 Hr RfC
COMPOUND	CAS NO.	(mg/m3)	Source	Date	Duration (hrs)	(mg/m3)
DICHLOROETHANE, 1,1-	75343					
DICHLOROETHANE, 1,2-	107062	2.0E+02	ERPG-1	12/01/02	1	8.3E+00
DICHLOROETHENE, 1,1-	75354					
DICHLOROETHENE, CIS-1,2-	156592					
DICHLOROPROPANE, 1,2-	78875	2.3E-01	ATSDR	12/01/89	24	2.3E-01
DICHLOROPROPANE, 1,3-	142289					
DICHLOROPROPANE, 2,2-	594207					
DICHLOROPROPENE, 1,1-	563586					
DICHLOROPROPENE, TRANS-1,3-	10061026					
DICHLOROTETRAFLUOROETHANE, 1,2-	76142					
DIELDRIN	60571					
DIETHYL PHTHALATE	84662					
DI-N-BUTYLPHTHALATE	84742					
DINITROPHENOL, 2,4-	51285					
ENDOSULFAN I	959988					
ETHYL ACRYLATE	140885	1.2E+02	ERPG-2 <sup>1</sup>	12/01/02	1	5.1E+00
ETHYL BENZENE	100414					
FLUORANTHENE	206440					
FLUORENE	86737					
FORMALDEHYDE	50000	4.9E-02	ATSDR	07/01/99	24	4.9E-02
HCL	7647010	2.1E+00	CAL EPA	05/01/00	1	8.8E-02
HEPTACHLOR	76448					
HEPTACHLOR EPOXIDE	1024573					
HEPTANE	142825					
HEXACHLOROBUTADIENE	87683	3.2E+01	ERPG-1	12/01/02	1	1.3E+00
HEXANE	110543					
HF	7664393	2.5E-02	ATSDR	09/01/01	24	2.5E-02
IRON	7439896					
ISOPHORONE	78591					
ISOPROPYLBENZENE	98828					
LEAD	7439921					

 Table 4-4 Acute Toxicity Values for COPCs

		Inh RfC			Exposure	24 Hr RfC
COMPOUND	CAS NO.	(mg/m3)	Source	Date	Duration (hrs)	(mg/m3)
LIMONENE	5989275					
MAGNESIUM	7439954					
MANGANESE	7439965					
METHYL ACETATE	79209					
METHYL BUTYL KETONE	591786					
METHYL ETHYL KETONE	78933	1.3E+01	CAL EPA	05/01/00	1	5.4E-01
METHYL ISOBUTYL KETONE	108101					
METHYL METHACRYLATE	80626					
METHYL T-BUTYL ETHER	1634044	7.2E+00	ATSDR	08/01/96	24	7.2E+00
METHYL-4,6-DINITROPHENOL, 2-	534521					
METHYLCYCLOHEXANE	108872					
METHYLENE CHLORIDE	75092	2.1E+00	ATSDR	09/01/00	24	2.1E+00
METHYLNAPHTHALENE, 2-	91576					
METHYLPHENOL, 2-	95487					
METHYLPHENOL, 3-	108394					
METHYLPHENOL, 4-	106445					
MOLYBDENUM	7439987					
NAPHTHALENE	91203					
NICKEL	7440020	6.0E-03	CAL EPA	05/01/00	1	2.5E-04
NITROBENZENE	98953					
NITROPHENOL, 2-	88755					
NITROPHENOL, 4-	100027					
NITROSODI-N-PROPYLAMINE, N-	621647					
OXYCHLORDANE	27304138					
PCB Congener #101	1336363					
PCB Congener #153	1336363					
PCB Congener #163	1336363					
PCB Congener #201	1336363					
PCB Congener #209	1336363					
PCB Congener #28	1336363					
PCB Congener #52	1336363					
PCB Congener #60	1336363					

 Table 4-4 Acute Toxicity Values for COPCs

		Inh RfC			Exposure	24 Hr RfC
COMPOUND	CAS NO.	(mg/m3)	Source	Date	Duration (hrs)	(mg/m3)
PENTACHLOROPHENOL	87865					
PHENANTHRENE	85018					
PHENOL	108952	5.8E+00	CAL EPA	05/01/00	1	2.4E-01
POTASSIUM	7440097					
PROPYLBENZENE, N-	103651					
PYRENE	129000					
SELENIUM	7782492					
SILVER	7440224					
SODIUM	7440235					
STRONTIUM	7440246					
STYRENE	100425	2.1E+01	CAL EPA	05/01/00	1	8.8E-01
TETRACHLOROETHANE, 1,1,1,2-	630206					
TETRACHLOROETHENE	127184	1.4E+00	ATSDR	09/01/97	24	1.4E+00
THALLIUM	7440280					
TIN	7440315					
TITANIUM	7440326					
TOLUENE	108883	3.8E+00	ATSDR	09/01/00	24	3.8E+00
TOXAPHENE	8001352					
TRICHLORO-1,2,2-TRIFLUOROETHANE, 1,1,2-	76131					
TRICHLOROBENZENE, 1,2,3-	87616					
TRICHLOROBENZENE, 1,2,4-	120821					
TRICHLOROETHANE, 1,1,1-	71556	1.1E+01	ATSDR	08/01/95	24	1.1E+01
TRICHLOROETHANE, 1,1,2-	79005					
TRICHLOROETHENE	79016	1.1E+01	ATSDR	09/01/97	24	1.1E+01
TRICHLOROFLUOROMETHANE	75694					
TRICHLOROPHENOL, 2,4,5-	95954					
TRICHLOROPHENOL, 2,4,6-	88062					
TRICHLOROPROPANE, 1,2,3-	96184	1.8E-03	ATSDR	09/01/92	24	1.8E-03
TRIMETHYLBENZENE, 1,2,4-	95636					
TRIMETHYLBENZENE, 1,3,5-	108678					
VANADIUM	7440622	2.0E-04	ATSDR	07/01/92	24	2.0E-04
VINYL CHLORIDE	75014	1.3E+00	ATSDR	09/01/97	24	1.3E+00

 Table 4-4 Acute Toxicity Values for COPCs

		Inh RfC			Exposure	24 Hr RfC
COMPOUND	CAS NO.	(mg/m3)	Source	Date	Duration (hrs)	(mg/m3)
XYLENE, M-	108383	4.3E+00 a	ATSDR	08/01/95	24	4.3E+00
XYLENE, O-	95476	4.3E+00 a	ATSDR	08/01/95	24	4.3E+00
XYLENE, P-	106423	4.3E+00 a	ATSDR	08/01/95	24	4.3E+00
YTTRIUM	7440655					
ZINC	7440666					

<sup>a</sup> Values for Xylene Mixture CAS# 1330207

<sup>1</sup> ERPG-1 not available

# **5.0 RISK CHARACTERIZATION**

The risk characterization integrates the information from the exposure assessment and toxicity assessment steps in the risk 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 chronic and acute toxicity data to provide a quantitative estimate of the potential health impacts. Both chronic and acute exposures are evaluated in this risk characterization. The chronic evaluation addresses both cancer and non-cancer health effects. The remainder of this section is divided into three subsections: one for details of the risk characterization for chronic exposure; another for the evaluation of acute exposures; and, a risk summary section. Within the discussion of chronic exposures the cancer estimates will be followed by the non-cancer evaluation for each monitoring site. A detailed assessment of the uncertainty in the risk characterization is provided in the Uncertainty Section (i.e., Section 6).

# 5.1 Risk Characterization for Chronic Exposures

The risk characterization for the chronic exposures was conducted by comparing the relevant toxicity criteria to the exposure concentration estimated from the WLATS monitoring data. Two different estimates of the potential risk were calculated: a central tendency case based on the median air concentration; and a 95% UCL exposure case selected to represent a conservative estimate of exposure based on the 95% UCL concentration of the COPC in air.

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. Cancer risk for each COPC at a monitoring location was derived as follows:

$$Risk_x = EC \times IUR_x$$
 Equation 5-1

Where:

<i>Risk</i> <sub>x</sub>	=	the risk of the X <sup>th</sup> COPC at a monitor:
EC	=	the exposure concentration of the substance (i.e., median or 95% UCL air
		concentration); and
$IUR_x$	=	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  $1 \times 10^{-6}$  or 1E–06.

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, defined as the ratio between the estimated exposure to an individual and the

Reference Concentration (RfC). For a given air toxic, exposures below the reference level (HQ<1) 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:

$$HQ_x = \frac{EC}{RfC_x}$$
 Equation 5-2

Where:

$HQ_x$	=	the hazard quotient of the X <sup>th</sup> COPC at a monitor;
EC	=	the exposure concentration of the substance (i.e., median or 95% UCL air
		concentration); and
$RfC_r$	=	the reference concentration of the substance.

When multiple noncarcinogens were present simultaneously, the individual HQs were summed to create a hazard index (HI), as:

$$HI = \sum_{x} \frac{EC}{RfC_x}$$
 Equation 5-3

Where:

HI = the hazard index of the X<sup>th</sup> COPCs at a monitor; EC = the exposure concentration of the individual substances; and  $RfC_x$  = the reference concentration of the individual substances.

The HI is a measure of the potential for an adverse health effect from all of the COPCs combined. Different pollutants, however, may cause completely different adverse health effects or act via completely different mechanisms of action, so it is often inappropriate to sum HQs associated with different endpoints (USEPA, 2001). Therefore, when the hazard index at a site exceeded a value of 1, the aggregate risk from exposure to multiple COPCs was assessed by adding the individual HQs for materials that act by a similar mechanism of action or the same target organ for the critical effect. Unless otherwise noted, the hazard indices presented in this Section are the sum of all hazard quotients for the COPCs at a monitor, which conservatively assumes that all of the COPCs have similarities in the mechanism of action or the target organ for the critical effect. Any deviations from this conservative hazard index will identify the similarity on which the hazard index was based and the COPCs that were included.

In the risk discussion for each monitor, the total cancer risk and HI will be presented based on all COPCs selected for the monitor. As discussed earlier, some monitors had data for VOCs, SVOCs, and metals, while others had analysis for VOCs only. For those monitors with analysis for more than VOCs, the total cancer risk and HI will be presented for all COPCs at the monitor and for the VOCs only. This will allow for a more balanced comparison of the potential impacts of the monitors in the WLATS network.

For each monitor, the risk drivers will be identified based on COPCs that exceed a cancer risk level of  $1 \times 10^{-6}$  or an HQ of 0.1. The use of risk drivers helps to focus the risk assessment on those COPCs with the greatest potential to impact human health. The use of a  $1 \times 10^{-6}$  threshold for the cancer risk level is consistent with the acceptable cancer risk threshold used in the State of Kentucky. An HQ of 0.1 for a COPC would indicate that adverse health effects are unlikely as a result of the exposure evaluated. However, using an HQ value of 0.1 as a threshold for the risk drivers provides a means to identify any COPCs that significantly contribute to an HI that exceeds a value of 1, at which there is a potential for an adverse health effect.

Summary tables have been provided at the end of this section that presents the risk estimates for all compounds that were considered risk drivers for the site. The tables provide the risk estimate for each of the chemicals at the monitors for which they were COPCs, and their percent contribution to the total risk at the monitor. Specifically: Tables 5-1 and 5-2 present the median exposure case cancer risk for all COPCs and for VOCs only, respectively; Tables 5-3 and 5-4 present the cancer risks for the 95% UCL exposure case for all COPCs and VOCs only, respectively; Tables 5-5 and 5-6 present the non-cancer HI for the median exposure case for all COPCs and VOCs only, respectively; and Tables 5-7 and 5-8 present the non-cancer HI for the 95% UCL exposure case for all COPCs and VOCs only, respectively. The tables are followed by a series of graphs that illustrate the total risk at each monitor in the WLATS network for a specific exposure case. These graphs provide an easy mechanism to compare the risks for the various monitor types (e.g., background, maximum impact, and neighborhood), along with an indication of the variability across the monitoring network. Figures 5-1 and 5-2 present the median case cancer risk for all COPCs, and for VOCS only, respectively. Figures 5-3 and 5-4 present the cancer risks for the 95% UCL exposure case for all COPCs and for VOCs only, respectively. Figures 5-5 and 5-6 present the median non-cancer HI for all COPCs and for VOCs only, respectively. Figures 5-7 and 5-8 present the HI for the 95% UCL exposure case for all COPCs and VOCs only, respectively.

A complete presentation of the risk calculations for all COPCs is provided in Appendix D. In addition, graphical displays for the risk drivers are provided in Appendix E that show the risk estimate for the chemical at every monitor in which it was evaluated along with an indication of the total risk at the monitor. This provides an easy means to see what contribution a risk driver made to the total risk at the monitor, the spatial characteristics of the chemicals concentration across the WLATS monitoring network, and the distribution of total risk across the network. This visualization can aid in planning by identifying hot-spots or trends, and the risk implications. For relevant monitors the graphs are provided to show the impacts for VOCs only, and for all COPCs.

#### 5.1.1 Louisville Police Firearms Training: WLATS Site No. 1

The median cancer risk for the site COPCs that were VOCs was  $5.1 \times 10^{-5}$ . The median cancer risk for all COPCs at the site was  $1.2 \times 10^{-4}$ . The risk drivers for the site were formaldehyde, 1-3, butadiene, benzene, carbon tetrachloride, chloroform, tetrachloroethene, arsenic, chromium, and nickel. For the 95% UCL exposure case, the cancer risk for the site VOCs that were COPCs

was 8 x  $10^{-5}$ . The 95% UCL exposure case had a cancer risk of 1.9 x  $10^{-4}$  for all site COPCs. The risk drivers for the 95% UCL exposure case were formaldehyde, 1-3, butadiene, benzene, carbon tetrachloride, chloroform, tetrachloroethene, arsenic, cadmium, chromium, and nickel.

For the site COPCs that were VOCs, the median case non-cancer HI was 0.33. For all COPCs the median exposure case the HI was 0.93. For site COPCs that were VOCs, the HI for the 95% exposure case was 0.79. For all site COPCs the 95% UCL exposure case HI was 1.9. The risk drivers for both the median and the 95% UCL exposure cases were 1,3-butadiene, manganese, and formaldehyde. Given the HI for both the median and 95% UCL exposure cases was below a value of 1, the potential for an adverse health impact as a result of this exposure is unlikely.

### 5.1.2 Ralph Avenue & Campground Road: WLATS Site No. 2a

The median cancer risk for the monitor COPCs that were VOCs was  $8.4 \times 10^{-5}$ . The median cancer risk for all site COPCs was  $1.5 \times 10^{-4}$ . The risk drivers for the median exposure case were formaldehyde, 1-3, butadiene, benzene, carbon tetrachloride, chloroform, ethyl acrylate, tetrachloroethene, arsenic, chromium, and nickel. For COPCs that were VOCs, the cancer risk for the 95% UCL exposure case was  $3.6 \times 10^{-4}$ . For all site COPCs the total cancer risk for the 95% UCL exposure case was  $4.6 \times 10^{-4}$ . The risk drivers for the 95% UCL exposure case were formaldehyde, 1-3, butadiene, benzene, carbon tetrachloride, chloroform, ethyl acrylate, tetrachloroethene, arsenic, cadmium, chromium, and nickel.

The median case non-cancer HI for site COPCs that were VOCs was 0.63. For the median case the HI for all site COPCs was 1.2. The risk drivers were formaldehyde, 1-3, butadiene, and manganese. For the median exposure case the HI was less than a value of 1 indicating that an adverse health effect is unlikely as a result of the exposure. For site COPCs that were VOCs, the non-cancer HI for the 95% UCL exposure case was 5.2. The HI for all site COPCs for the 95% UCL exposure case, the risk drivers were formaldehyde, 1-3, butadiene, toluene, and manganese. Of the site COPCs, only 1,3-butadiene had a HQ that exceeded a value of 1. Given that the HQ for 1,3-butadiene exceeded a value of 1, there is a potential for an adverse health effect as a result of the exposure evaluated.

## 5.1.3 Ralph Avenue & Campground Road: WLATS Site No. 2b

The median cancer risk for site COPCS that were VOCs was  $1.1 \times 10^{-4}$ . For all site COPCs the median case the cancer risk was  $1.8 \times 10^{-4}$ . For the site COPCs that were VOCs, the cancer risk for the 95% UCL exposure case was  $6.0 \times 10^{-4}$ . The 95% UCL exposure case cancer risk for all site COPCs was  $6.9 \times 10^{-4}$ . The risk drivers for both the median exposure case and the 95% UCL exposure case were formaldehyde, 1-3, butadiene, benzene, carbon tetrachloride, chloroform, ethyl acrylate, arsenic, cadmium, chromium, and nickel.

For the site COPCs that were VOCs, the non-cancer HI for the median exposure was 1.1. For all site COPCs the HI for the median exposure was 1.7. The risk drivers were formaldehyde, 1,3-butadiene and manganese. None of these risk drivers had an HQ above 1, and the HI based on

summing by similar critical effect (see Table 5-5 last column) also is less than 1 for the risk drivers. Therefore, an adverse health effect from this exposure is unlikely. For the 95% UCL exposure case, the HI for site COPCs that were VOCs was 8.6. For all site COPCs the HI for the 95% UCL case was 9.4. The risk drivers were formaldehyde, 1,3-butadiene, toluene and manganese. Of these 1,3-butadiene had an HQ above a value of 1, indicating a potential for an adverse health impact as a result of the exposure evaluated.

### 5.1.4 Old Lake Dreamland Fire Department: WLATS Site No. 3

The median cancer risk for site COPCs that were VOCs was  $1.1 \times 10^{-4}$ . The median cancer risk for all site COPCs was  $1.7 \times 10^{-4}$ . The 95% UCL exposure case cancer risk for the site COPCs that were VOCs was  $3.1 \times 10^{-4}$ . The total cancer risk for the 95% UCL exposure case for all site COPCs was  $4.0 \times 10^{-4}$ . The risk drivers for both the median and 95% UCL exposure case were formaldehyde, 1-3, butadiene, benzene, carbon tetrachloride, chloroform, ethyl acrylate, tetrachloroethene, arsenic, cadmium, chromium, and nickel.

The median exposure case non-cancer HI for site COPCs that were VOCs was 0.67. For all site COPCs the median case HI was 1.2. The HQ for all COPCs was less than a value of 1, and HIs based on summing HQs for risk drivers with similar critical effects (see Table 5-5 last column) did not result in an HI that exceeded a value of 1, therefore, adverse health impacts are unlikely for this exposure. For the 95% UCL exposure case, the HI for the site COPCs that were VOCs was 3.4. The HI for the 95% UCL exposure case for all site COPCs was 4.2. The risk drivers for both the median and 95% UCL exposure case were formaldehyde, 1-3, butadiene, and manganese. Of these risk drivers, only 1,3-butadiene had an HQ that exceeded a value of 1, and only for the 95% UCL exposure case. The fact that the HQ for 1,3-butadiene exceeded a value of 1 for the 95% UCL exposure case indicates a potential for an adverse health effect as a result of the exposure evaluated.

#### 5.1.5 St. Stephen Baptist Church: WLATS Site No. 4

The median case cancer risk for the site COPCs that were VOCs was  $5.4 \times 10^{-5}$ . For all of the site COPCs the median case cancer risk was  $1.3 \times 10^{-4}$ . The 95% UCL exposure case cancer risk for site COPCs that were VOCs was  $7.5 \times 10^{-5}$ . For all site COPCs, the 95% UCL exposure case cancer risk was  $1.8 \times 10^{-4}$ . The risk drivers for both the median and 95% UCL exposure cases were formaldehyde, 1-3, butadiene, 1,4-dichlorobenzene, benzene, carbon tetrachloride, chloroform, tetrachloroethene, arsenic, chromium, and nickel.

The median non-cancer HI for site COPCs that were VOCs was 0.26. For all site COPCs the HI for the median exposure case was 0.84. For the 95% UCL exposure case the HI for site COPCs that were VOCs was 0.47. For all site COPCs the 95% UCL exposure case HI was 1.4. The risk drivers for both the median and 95% UCL exposure cases were formaldehyde, 1-3, butadiene, and manganese, with no HQ exceeding a value of 1 for any COPC under either the median or 95% UCL exposure cases. The HI for all COPCs under the median case, and for VOCs only under the 95% UCL exposure case, are below 1 indicating that an adverse health effect is

unlikely. The HI for all site COPCs under the 95% UCL exposure case exceeds a value of 1, but an HI based on similar critical effects (see Table 5-7 last column) did not exceed a value of 1, therefore an adverse health effect as a result of the exposures evaluated is unlikely.

### 5.1.6 University of Louisville Shelby Campus: WLATS Site No. 5

For site COPCs that were VOCs, the median cancer risk was  $1.7 \times 10^{-5}$ . For all site COPCs the median cancer risk was  $6.7 \times 10^{-5}$ . The risk drivers for the median exposure case were formaldehyde, benzene, carbon tetrachloride, arsenic, chromium, and nickel. For the 95% UCL case the cancer risk for site COPCs that were VOCs was  $2.2 \times 10^{-5}$ . For all site COPCs the 95% UCL exposure case cancer risk for this site was  $8.5 \times 10^{-5}$ . The risk drivers for the 95% UCL exposure case were formaldehyde, benzene, carbon tetrachloride, arsenic, cadmium, chromium, and nickel.

The median exposure case non-cancer HI for the site COPCs that were VOCs was 0.08. For all site COPCs the median case HI was 0.43. The risk driver for the median case was manganese. The 95% UCL exposure case HI for site COPCs that were VOCs was 0.13. For all site COPCs the 95% UCL exposure case HI 0.72. The risk drivers for the 95% UCL exposure case were manganese and formaldehyde. The HI for the median and 95% UCL exposure cases did not exceed a value of 1 indicating that the potential for adverse health effects as a result of the exposures is unlikely.

## 5.1.7 Otter Creek Park: WLATS Site No. 6

The cancer risk for the median exposure case for site COPCs that were VOCs was  $1.4 \times 10^{-5}$ . For all site COPCs the median cancer risk was  $5.9 \times 10^{-5}$ . For the 95% UCL exposure case, the cancer risk for site COPCs that were VOCs was  $1.8 \times 10^{-5}$ . For all site COPCs the 95% UCL exposure case cancer risk was  $7.6 \times 10^{-5}$ . The risk drivers for both the median and 95% UCL exposure cases were formaldehyde, benzene, carbon tetrachloride, arsenic, chromium, and nickel.

The median exposure case non-cancer HI for site COPCs that were VOCs was 0.07. For all site COPCs the HI for the median exposure case was 0.31. None of the COPCs had an HQ that exceeded a value of 0.1 for the median exposure case. The 95% UCL exposure case HI for site COPCs that were VOCs was 0.09. For all site COPCs the HI for the 95% UCL exposure case was 0.5. For the 95% UCL exposure case, the risk driver was manganese. The fact that the HI for both the median and 95% UCL exposure cases is below a value of 1.0 indicates that non-cancer human health impacts are unlikely.

## 5.1.8 Park DuValle:Southwick Community Center: WLATS Site No. 7

At this monitoring location, analysis was conducted for VOCs only; therefore it is not necessary to provide VOC only and total COPC risks, as they are the same. The median cancer risk for site

COPCs was  $6.0 \ge 10^{-5}$ . The risk drivers were 1-3, butadiene, 1,4-dichlorobenzene, acrylonitrile, benzene, carbon tetrachloride, chloroform, methylene chloride, tetrachloroethene, and vinyl chloride. For the 95% UCL exposure the cancer risk for site COPCs was  $3.9 \ge 10^{-4}$ . The risk drivers were 1-3, butadiene, 1,4-dichlorobenzene, acrylonitrile, benzene, bromoform, carbon tetrachloride, chloroform, methylene chloride, tetrachloroethene, trichloroethene, and vinyl chloride. The cancer risk estimates for this monitor may have been impacted by the missing sample dates, given that a maximum of 14 samples dates was reported for any single COPC. The risk estimates for some of the risk drivers for the 95% UCL exposure case were based on the maximum detected concentration because the 95% UCL of the mean concentrations in air was greater than the maximum detected value. Although this introduces an uncertainty into the risk estimates, they are unlikely to underestimate, and are more likely to overestimate the true risks for this exposure case, as the maximum detected air concentration in air was used for the risk characterization.

The non-cancer HI for the median exposure case was 0.48. For the 95% UCL exposure case the HI was 4.8. The risk drivers for the 95% UCL exposure case were 1-3, butadiene, acrylonitrile, bromoform, carbon disulfide, and toluene. Of the risk drivers for the 95% UCL exposure case, only 1,3-butadiene had an HQ that exceeded a value of 1. For the 95% UCL exposure case, the maximum detected concentration of 1,3-butadiene at this monitor was used for the risk characterization as it was lower than the 95% UCL of the mean concentrations for this COPC. Thus although a potential exists for an adverse health effect based on the 95% UCL exposure case to 1,3-butadiene, this may well be an overestimate of the true potential for health impacts due to using the maximum detected concentration in air.

Appendix C provides a complete listing for this monitor of the COPC concentration in air used for the 95% UCL exposure case, and the basis for the value (i.e., maximum of 95% UCL on the mean).

## 5.1.9 Farnsley Middle School: WLATS Site No. 8

At this monitoring location, analysis was conducted for VOCs only; therefore it is not necessary to provide VOC only and total COPC risks, as they are the same. The median exposure case cancer risk for this site was  $4.8 \times 10^{-5}$ . The risk drivers for the median exposure case were 1-3, butadiene, 1,4-dichlorobenzene, acrylonitrile, benzene, carbon tetrachloride, chloroform, and tetrachloroethene. For the 95% UCL exposure case the cancer risk was  $2.5 \times 10^{-4}$ . The risk drivers were 1-3, butadiene, 1,4-dichlorobenzene, acrylonitrile, benzene, carbon tetrachloride, chloroform, and tetrachloroform, methylene chloride, tetrachloroethene, and trichloroethene.

The non-cancer HI for the median exposure case was 0.33, with only 1,3-butadiene exceeding an HQ of 0.1, indicating an adverse health impact is unlikely. For the 95% UCL exposure case the total non-cancer HI was 3.1. The risk drivers for the 95% UCL exposure case were 1-3, butadiene, acrylonitrile, and toluene. Of these three risk drivers, only 1,3-butadiene exceeded an HQ of 1 for the 95% UCL exposure case. Each of the three risk drivers for the 95% UCL exposure case has a different critical effect (see Table 5-7 last column), thus an HI based on similar critical effects would only exceed a value of 1 for 1,3-butadiene. The fact that HQ for

1,3-butadiene exceeds a value of 1 indicates a potential for an adverse health effect associated with this exposure.

#### 5.1.10 Chickasaw Park (Private Residence): WLATS Site No. 9

At this monitoring location, analysis was conducted for VOCs only; therefore it is not necessary to provide VOC only and total COPC risks, as they are the same. The median exposure case cancer risk was  $4.4 \times 10^{-5}$ . The risk drivers for the median exposure case were 1-3, butadiene, acrylonitrile, benzene, carbon tetrachloride, and chloroform. The cancer risk for this site under the 95% UCL exposure case was  $1.3 \times 10^{-4}$ . The risk drivers were 1-3, butadiene, acrylonitrile, benzene, carbon tetrachloride, and methylene chloride. The cancer risk estimates for this monitor may have been impacted by the missing sample dates, given that a maximum of 11 samples dates was reported for any single COPC. The risk estimates for methylene chloride for the 95% UCL exposure case was based on the maximum detected concentration because the 95% UCL of the mean concentrations in air was greater than the maximum detected value. Although this introduces an uncertainty into the risk estimates, they are unlikely to underestimate, and are more likely to overestimate the true risks for this exposure case, as the maximum detected concentration in air was used for the risk characterization. Of the risk drivers, the maximum COPC concentration in air was only used for methylene chloride.

The non-cancer HI for the median case exposure was 0.27, with only acrylonitrile above a hazard quotient of 0.1. For the 95% UCL exposure case the HI was 1.1. The risk drivers were 1,3-butadiene and acrylonitrile, and toluene, none of which had an HQ at or above a value of 1. Because all three of the risk drivers for the 95% UCL exposure case have a different critical effect (see Table 5-7 last column), an HQ for the risk drivers based on critical effect would not exceed a value of 1 for the risk drivers. Therefore, an adverse health effect is unlikely based on the exposures evaluated.

#### 5.1.11 New Lake Dreamland Fire Department: WLATS Site No. 10

At this monitoring location, analysis was conducted for VOCs only; therefore it is not necessary to provide VOC only and total COPC risks, as they are the same. The median exposure case cancer risk for this site was  $4.5 \times 10^{-5}$ . The risk drivers for the median case were 1-3, butadiene, acrylonitrile, benzene, carbon tetrachloride, and chloroform. For the 95% UCL exposure case the cancer risk was  $1.3 \times 10^{-4}$ . The risk drivers for the 95% UCL exposure case were 1-3, butadiene, acrylonitrile, benzene, carbon tetrachloride, chloroform, and methylene chloride. Although the number of sample dates available for this monitor was 15 or less, in no case was the maximum concentration in air used to evaluate any of the risk drivers. The risk drivers were detected in at least 40% of the samples reported for this monitor, with the exception of arcylonitrile were both greater than  $\frac{1}{2}$  the detection limit, which was used in calculating the concentrations used in the median and 95% UCL exposure cases; therefore, the results are not biased towards artificially high concentrations due to elevated detection limits.

For the median exposure case the HI was 0.33, with only 1,3-butadiene exceeding an HQ of 0.1. The 95% UCL exposure case HI was 1.3. For the 95% UCL exposure case the risk drivers were 1-3, butadiene, acrylonitrile, and toluene, all of which had HQ's that were less than 1. Because all three of the risk drivers for the 95% UCL exposure case have a different critical effect (see Table 5-7 last column), an HQ for the risk drivers based on critical effect would not exceed a value of 1 for the risk drivers. Therefore, an adverse health effect is unlikely based on the exposures evaluated.

### 5.1.12 M.L.King Elementary School: WLATS Site No. 11

At this monitoring location, analysis was conducted for VOCs only; therefore it is not necessary to provide VOC only and total COPC risks, as they are the same. The median exposure case cancer risk was  $3.8 \times 10^{-5}$ , with six chemicals exceeding a cancer risk of  $1 \times 10^{-6}$ . The risk drivers were 1-3, butadiene, 1,4-dichlorobenzene, acrylonitrile, benzene, carbon tetrachloride, and chloroform. For the 95% UCL case, the total cancer risk for this site was  $1.4 \times 10^{-4}$ , with eight chemicals exceeding a cancer risk of  $1 \times 10^{-6}$ . The risk drivers were 1-3, butadiene, 1,4-dichlorobenzene, acrylonitrile, benzene, carbon tetrachloride, and chloroform. For the 95% UCL case, the total cancer risk for this site was  $1.4 \times 10^{-4}$ , with eight chemicals exceeding a cancer risk of  $1 \times 10^{-6}$ . The risk drivers were 1-3, butadiene, 1,4-dichlorobenzene, acrylonitrile, benzene, carbon tetrachloride, chloroform, methylene chloride, and trichloroethene.

For the median exposure case the HI was 0.19, with none of the COPCs exceeding an HQ quotient of 0.1. For the 95% UCL exposure case the HI was 1.4. For this case the risk drivers were 1,3-butadiene, acrylonitrile, and toluene, none of which had an HQ that exceeded a value of 1. Because all three of the risk drivers for the 95% UCL exposure case have a different critical effect (see Table 5-7 last column), an HQ for the risk drivers based on critical effect would not exceed a value of 1 for the risk drivers. Therefore, an adverse health effect is unlikely based on the exposures evaluated.

## 5.1.13 Cane Run Elementary School: WLATS Site No. 12

At this monitoring location, analysis was conducted for VOCs only; therefore it is not necessary to provide VOC only and total COPC risks, as they are the same. For the median exposure case the cancer risk was  $6.3 \times 10^{-5}$ , with six chemicals above a cancer risk of  $1 \times 10^{-6}$ . The risk drivers in this case were 1-3, butadiene, 1,4-dichlorobenzene, acrylonitrile, benzene, carbon tetrachloride, and chloroform. For the 95% UCL exposure case the cancer risk for this site was  $1.4 \times 10^{-4}$ , with seven COPCs exceeding a cancer risk of  $1 \times 10^{-6}$ . The risk drivers for the 95% UCL exposure case were 1-3, butadiene, 1,4-dichlorobenzene, acrylonitrile, benzene, carbon tetrachloride, chloroform, and methylene chloride.

The median exposure case HI was 0.50 with two chemicals, 1-3, butadiene and acrylonitrile, above an HQ hazard quotient of 0.1. For the 95% UCL exposure case the hazard index was 1.3. Once again the risk drivers were drivers were 1,3-butadiene, and acrylonitrile, however, the HQ for neither of these two COPCs exceeded a value 1. Because the two risk drivers for the 95% UCL exposure case have a different critical effect (see Table 5-7 last column), an HI based on

critical effect would not exceed a value of 1 for the risk drivers, indicating that an adverse health effect at this site is unlikely for the exposures evaluated.

# 5.2 Acute Risk Characterization

Risks of acute health effects were estimated in much the same way as risks of non-cancer health effects. Maximum detected concentrations of each contaminant ( $CA_{max}$ ) were compared to acute benchmark concentrations (AB) through the calculation of acute hazard quotients (HQ<sub>acute</sub>):

$$HQ_{acute} = \frac{CA_{\max}}{AB}$$
(5.4)

where both  $CA_{max}$  and AB are expressed in the same units. Unlike chronic hazard quotients, however,  $HQ_{acute}$  values of individual substances were not added together because acute health effects vary widely from one chemical to another.

The acute toxicity characterization was based on a comparison of the maximum detected concentration for each COPC at a given monitor. As stated earlier, the assessment of acute risks is not as well developed as the chronic evaluation, leading to a relatively higher degree of uncertainty in the risk estimates. An HQ was calculated for each COPC at each monitor. None of the COPCs evaluated for any monitor had an HQ greater than 1. A value of 1 was used as the threshold for risk drivers for the acute risk assessment because the exposure is based on using the maximum COPC concentration in air, and not some measure of the mean concentration in air (i.e., median, or 95% UCL on the mean) as was done in the chronic risk assessment where an HQ of 0.1 was used as the risk driver threshold. A complete listing of the HQs for each COPC at every monitor location is presented in Appendix D, along with other relevant details on the acute risk assessment.

# 5.3 Risk Characterization Summary

The risk assessment evaluated the potential for adverse human health impacts from chronic and acute exposures to COPCs selected of each monitor in the WLATS program. For each monitor, the COPCs were chemicals that were found in at least 10% of the samples at the monitor. Approximately half of the monitors evaluated only VOCs (i.e., WLATS Sites 7 through 12). The remaining monitors (i.e., WLATS Sites 1 through 6) evaluated SVOCs and metals, in addition to VOCs. The difference in the number of chemicals evaluated at the various monitors makes a direct comparison of the total risk estimates across the network difficult if one is looking for the monitor location with the maximum impact. To aid in a comparative analysis of the risk estimates across the WLATS network, the following summary discusses the overall risks in terms of VOCs only, and all COPCs. The VOC only analysis provides the most direct means of comparing the monitors as the same group of chemicals were analyzed by all monitors in the WLATS network. The risk estimates for all COPCs includes the impacts from metals and SVOCs, which were not included in the analysis for some monitors. Because risk drivers were

identified for the metals and SVOCs at the monitors where they were analyzed, the risk estimates for all COPCs at these monitors will increase relative to the estimate for VOCs only. For monitors where only VOCs were analyzed, the risk estimate for VOCs only and for all COPCs will be the same. Thus, a comparison of the risk estimates across the WLATS monitors for all COPCs may lead to inappropriate conclusion of where the highest impacts occur, unless one considers that not all chemicals were analyzed at all of the monitors.

The evaluation of chromium and nickel in this risk assessment was based on the assumption that all of the concentration detected in air was the most toxic form of the metal. For chromium, this meant evaluating all of the chromium concentrations in air as if they were hexavalent chromium. For nickel, all of the concentrations in air were evaluated as if they were nickel subsulfide. It is likely that this treatment for chromium and nickel will tend to overestimate the potential health impacts at monitors where these metals were selected as COPCs. This issue is discussed in more detail in the Section 6.0.

For the chronic risk assessment, four risk estimates were calculated based on the median and 95% UCL exposure cases: a median exposure case cancer risk estimate: a 95% UCL exposure case cancer risk estimate; a median exposure case non-cancer hazard index; and, a 95% UCL exposure case non-cancer hazard index. For the acute exposure analysis, a hazard quotient was calculated for COPCs at a monitor based on the maximum concentration detected for the COPC at the monitor. The remainder of this section will discuss the risk estimates across the monitoring network for each of these cases. The risk estimates for risk drivers at each monitor are presented in Tables 5-1 through 5-8 for the median exposure case cancer risks, the 95% UCL exposure case cancer risks, the median exposure case non-cancer HI and chemical-specific HQs, and the 95% UCL exposure case non-cancer HI and chemical-specific HQs, respectively. The tables also indicate the total risk at the monitor for VOCs only and for all COPCs, and the contribution that each risk driver made to the total risks. A detailed listing of the risk estimates for all COPCs at a monitor are presented in Appendix D. Finally, graphical displays of the total cancer risk or non-cancer HI are shown in Figures 5-1 through 5-8. These figures display the risk at each monitor location across the WLATS network, with separate graphs of the median and 95% UCL exposure cases for the cancer and non-cancer estimates, based on VOCs only and all COPCs. Similar graphs are presented in Appendix F for all COPCs that were a risk driver for at least one monitor in the WLATS network.

For the median exposure case cancer risk estimate, the values across the WLATS network ranged from  $1.1 \times 10^{-4}$  to  $1.4 \times 10^{-5}$  for VOCs only,  $1.8 \times 10^{-4}$  to  $3.8 \times 10^{-5}$  for all COPCs. The maximum impact occurred at WLATS Site 3 for VOCs only. For all COPCs, the maximum impact occurred at Site 2b. For the residential monitors, the VOC only cancer risk for the median case ranged from  $1.1 \times ^{-4}$  at Site 3, to  $3.8 \times ^{-5}$  at Site 11. For the VOC only case, the median cancer risk at all residential monitors were higher than for both background monitors. When looking at residential monitors for all COPCs, the median cancer risks ranged from  $1.7 \times 10^{-4}$  at Site 3 to  $3.8 \times 10^{-5}$  at Site 11. The median exposure case cancer risk estimates at four of the residential monitors was lower than for the background monitors, however, these four residential monitors included only VOC analysis, whereas the background monitors did include risks from VOCs, SVOCs and metals.

The cancer risk estimates for the 95% UCL exposure case ranged from  $6.0 \times 10^{-4}$  to  $1.8 \times 10^{-5}$  for VOCs only, and from  $6.9 \times 10^{-4}$  to  $7.6 \times 10^{-5}$  for all COPCs. The maximum impact occurred at Site 2b for both the case of VOCs only and when looking at all COPCs. For the residential monitors, the 95% UCL exposure case cancer risks for VOCs only ranged from  $3.9 \times 10^{-4}$  at Site 7 to  $1.3 \times 10^{-4}$  at Sites 9 and 10. The residential risk range for all COPCs was from  $4.0 \times 10^{-4}$  at Site 3, to  $1.3 \times 10^{-4}$  at Sites 9 and 10. The 95% UCL cancer risk estimates for all residential monitors was higher than either background monitor when looking at both VOCs only and for all COPCs.

The non-cancer HI for the median exposure case across the WLATS network ranged from 1.09 to 0.07 for VOCs only, and from 1.73 to 0.19 for all COPCs. The maximum impact occurred at Site 2b for both VOCs only and for all COPCs. For residential monitors, the HI for VOCs only ranged from 0.67 at Site 3 to 0.19 at Site 11, while the HI for all COPCs ranged from 1.17 at Site 3 to 0.19 at Site 11. When looking at VOCs only, the median exposure case HI for all residential monitors was greater than for the background monitors. When looking at all COPCs, the median exposure case HI for several residential monitors was below the background monitors, but unlike the background monitors, these residential monitors had data for VOCs only.

For the 95% UCL exposure case, the non-cancer HIs for the WLATS network ranged from 8.58 to 0.09 for VOCs only, and from 9.43 to 0.50 for all COPCs. The maximum impact occurred at Site 2b for both VOCs only and for all COPCs. For residential monitors, the 95% UCL exposure case HI ranged from a high of 4.82 at Site 7 to a low of 0.47 at Site 4 when looking at VOCs only. For all COPCs, the 95% UCL exposure case HI at residential monitors ranged from a high of 4.82 at Site 7. A comparison of the 95% UCL exposure case HI for the background monitors versus the residential monitors shows that the HIs at all residential monitors exceeds the HIs for the background monitors when looking at both VOCs only and for all COPCs.

For the acute analysis, a hazard quotient was calculated for each COPC at a monitor. The HQ was based on a comparison of the maximum detected concentration of the COPC at the monitor versus the toxicity value for the COPC. Because it is not appropriate to sum the individual HQs for the acute analysis, no total estimate in the form of a HI, was calculated for the WLATS monitors. The results of the acute analysis shows that no COPC had an HQ that exceeded a value of 1 indicating that an adverse health effect in very unlikely as a result of the assumed exposure.

	WLAT EPA S			S Site 2a ite ID 2	WLATS EPA Si	S Site 2b ite ID 3				S Site 4 te ID 5
Compound	Cancer Risk	% Contribution to Cumulative Risk	Cancer Risk	% Contribution to Cumulative Risk	Cancer Risk	% Contribution to Cumulative Risk	Cancer Risk	% Contribution to Cumulative Risk	Cancer Risk	% Contribution to Cumulative Risk
FORMALDEHYDE	1.7E-05	13.9%	1.4E-05	9.6%	2.1E-05	11.7%	1.7E-05	9.7%	1.7E-05	13.3%
1,3-BUTADIENE 1,4-DICHLOROBENZENE ACRYLONITRILE	9.9E-06	8.1%	3.0E-05	19.9%	5.7E-05	31.1%	3.2E-05	18.4%	8.6E-06 7.7E-06	6.8% 6.1%
BENZENE	1.4E-05	11.5%	1.0E-05	6.8%	9.4E-06	5.1%	1.1E-05	6.2%	9.6E-06	7.5%
CARBON TETRACHLORIDE CHLOROFORM	9.3E-06 1.3E-05	7.6% 10.4%	1.0E-05 1.7E-05	6.9% 11.5%	9.5E-06 1.6E-05	5.2% 8.8%	1.0E-05 2.5E-05	5.8% 14.1%	9.5E-06 1.3E-05	7.4% 10.6%
ETHYL ACRYLATE			1.2E-05	7.8%	1.3E-05	7.3%	2.9E-05	16.9%		
METHYLENE CHLORIDE TETRACHLOROETHENE (TETRACHLOROETHYLENE) VINYL CHLORIDE	4.4E-06	3.6%	4.7E-06	3.2%			4.9E-06	2.8%	4.6E-06	3.6%
ARSENIC	6.0E-06	4.9%	6.5E-06	4.3%	5.6E-06	3.0%	4.9E-06	2.8%	4.7E-06	3.7%
CADMIUM CHROMIUM	4.6E-05	37.5%	4.1E-05	27.7%	1.1E-06 4.7E-05	0.6%	1.3E-06 3.7E-05	0.7%	4.8E-05	37.8%
NICKEL	4.6E-05 1.8E-06	1.5%	4.1E-03 2.2E-06	1.5%	4.7E-03 2.5E-06	1.4%	1.7E-05	1.0%	4.8E-03 2.5E-06	2.0%
ALL OTHER COMPOUNDS	1.0E-06	0.8%	1.4E-06	0.9%	5.4E-07	0.3%	4.0E-07	0.2%	1.4E-06	1.1%
CUMULATIVE RISK	1.2E-04		1.5E-04		1.8E-04		1.7E-04		1.3E-04	

TABLE 5-1MEDIAN CANCER RISK EXCEEDANCES AND WOE

	WLAT EPA Si	S Site 5 ite ID 6	WLAT EPA Si	S Site 6 ite ID 7	WLAT U of L S			S Site 8 ite ID M		S Site 9 Site ID I
Compound	Cancer Risk	% Contribution to Cumulative Risk	Cancer Risk	% Contribution to Cumulative Risk	Cancer Risk	% Contribution to Cumulative Risk	Cancer Risk	% Contribution to Cumulative Risk	Cancer Risk	% Contribution to Cumulative Risk
FORMALDEHYDE	1.2E-05	18.1%	7.9E-06	13.4%						
1,3-BUTADIENE 1,4-DICHLOROBENZENE					1.8E-05 3.4E-06	30.1% 5.6%	1.2E-05 3.4E-06	24.9% 6.9%	3.3E-06	7.4%
ACRYLONITRILE					7.5E-06	12.4%	7.5E-06	15.5%	1.9E-05	42.0%
BENZENE	7.3E-06	10.9%	5.2E-06	8.9%	1.1E-05	19.0%	5.5E-06	11.4%	6.0E-06	13.6%
CARBON TETRACHLORIDE	9.2E-06	13.7%	9.0E-06	15.3%	7.6E-06	12.7%	1.1E-05	23.6%	1.0E-05	23.6%
CHLOROFORM					5.6E-06	9.4%	5.6E-06	11.6%	5.6E-06	12.7%
ETHYL ACRYLATE										
METHYLENE CHLORIDE					2.2E-06	3.7%				
TETRACHLOROETHENE (TETRACHLOROETHYLENE)					2.0E-06	3.4%	2.0E-06	4.2%		
VINYL CHLORIDE					1.1E-06	1.9%				
ARSENIC	4.0E-06	5.9%	3.5E-06	5.9%						
CADMIUM										
CHROMIUM	3.2E-05	47.5%	3.1E-05	52.9%						
NICKEL	1.3E-06	2.0%	1.4E-06	2.4%						
ALL OTHER COMPOUNDS	1.2E-06	1.9%	7.4E-07	1.3%	1.1E-06	1.8%	8.4E-07	1.7%	2.8E-07	0.6%
CUMULATIVE RISK	6.7E-05		5.9E-05		6.0E-05		4.8E-05		4.4E-05	

TABLE 5-1 (con't)MEDIAN CANCER RISK EXCEEDANCES AND WOE

	WLATS U of L S	S Site 10 lite ID K	WLATS U of L S			S Site 12 Site ID F	EPA / IA	RC WOE
Compound	Cancer Risk	% Contribution to Cumulative Risk	Cancer Risk	% Contribution to Cumulative Risk	Cancer Risk	% Contribution to Cumulative Risk	A/1	B1, B2, C / 2A, 2B
FORMALDEHYDE								X
1,3-BUTADIENE 1,4-DICHLOROBENZENE ACRYLONITRILE	1.1E-05 7.5E-06	25.2%	3.3E-06 3.4E-06 7.5E-06	8.7% 8.8% 19.7%	1.8E-05 3.4E-06 1.6E-05	28.9% 5.3% 26.1%		X X X X
BENZENE	9.1E-06	20.0%	6.0E-06	15.9%	7.3E-06	11.6%	X	Λ
CARBON TETRACHLORIDE	1.1E-05	25.3%	1.1E-05	30.1%	1.1E-05	18.2%		Х
CHLOROFORM	5.6E-06	12.4%	5.6E-06	14.8%	5.6E-06	9.0%		Х
ETHYL ACRYLATE								X
METHYLENE CHLORIDE	_							X
TETRACHLOROETHENE (TETRACHLOROETHYLENE)								X
VINYL CHLORIDE							X	
ARSENIC							X	
CADMIUM								X
CHROMIUM							Х	
NICKEL							X	
ALL OTHER COMPOUNDS	2.8E-07	0.6%	8.1E-07	2.1%	5.1E-07	0.8%		

3.8E-05

TABLE 5-1 (con't)MEDIAN CANCER RISK EXCEEDANCES AND WOE

CUMULATIVE RISK

4.5E-05

6.3E-05

#### WEST LOUISVILLE AIR TOXICS STUDY

	WLAT EPA Si		WLATS EPA Si	S Site 2a ite ID 2	WLATS EPA Si			S Site 3 ite ID 4	WLAT EPA Si	S Site 4 te ID 5
Compound	Cancer Risk	% Contribution to VOC Only Risk	Cancer Risk	% Contribution to VOC Only Risk	Cancer Risk	% Contribution to VOC Only Risk	Cancer Risk	% Contribution to VOC Only Risk	Cancer Risk	% Contribution to VOC Only Risk
1,3-BUTADIENE	9.9E-06	19.6%	3.0E-05	35.3%	5.7E-05	54.0%	3.2E-05	28.6%	8.6E-06	16.1%
1,4-DICHLOROBENZENE									7.7E-06	14.4%
ACRYLONITRILE										
BENZENE	1.4E-05	27.8%	1.0E-05	12.1%	9.4E-06	8.9%	1.1E-05	9.7%	9.6E-06	17.8%
CARBON TETRACHLORIDE	9.3E-06	18.4%	1.0E-05	12.3%	9.5E-06	9.0%	1.0E-05	9.0%	9.5E-06	17.6%
CHLOROFORM	1.3E-05	25.0%	1.7E-05	20.5%	1.6E-05	15.3%	2.5E-05	22.0%	1.3E-05	25.1%
ETHYL ACRYLATE			1.2E-05	13.9%	1.3E-05	12.7%	2.9E-05	26.2%		
METHYLENE CHLORIDE										
TETRACHLOROETHENE (TETRACHLOROETHYLENE)	4.4E-06	8.8%	4.7E-06	5.6%			4.9E-06	4.3%	4.6E-06	8.5%
VINYL CHLORIDE										
ALL OTHER VOC COMPOUNDS	2.1E-07	0.4%	2.0E-07	0.2%	1.9E-07	0.2%	2.3E-07	0.2%	2.0E-07	0.4%
VOC RISK	5.1E-05		8.4E-05		1.1E-04		1.1E-04		5.4E-05	

 TABLE 5-2

 MEDIAN CANCER RISK EXCEEDANCES AND WOE - VOC ONLY

		S Site 5 ite ID 6		S Site 6 ite ID 7	WLAT U of L S			S Site 8 ite ID M	WLAT U of L S	
Compound	Cancer Risk	% Contribution to VOC Only Risk	Cancer Risk	% Contribution to VOC Only Risk	Cancer Risk	% Contribution to VOC Only Risk	Cancer Risk	% Contribution to VOC Only Risk	Cancer Risk	% Contribution to VOC Only Risk
1,3-BUTADIENE					1.8E-05	30.1%	1.2E-05	24.9%	3.3E-06	7.4%
1,4-DICHLOROBENZENE					3.4E-06	5.6%	3.4E-06	6.9%		
ACRYLONITRILE					7.5E-06	12.4%	7.5E-06	15.5%	1.9E-05	42.0%
BENZENE	7.3E-06	44.0%	5.2E-06	36.3%	1.1E-05	19.0%	5.5E-06	11.4%	6.0E-06	13.6%
CARBON TETRACHLORIDE	9.2E-06	55.0%	9.0E-06	62.5%	7.6E-06	12.7%	1.1E-05	23.6%	1.0E-05	23.6%
CHLOROFORM					5.6E-06	9.4%	5.6E-06	11.6%	5.6E-06	12.7%
ETHYL ACRYLATE										
METHYLENE CHLORIDE					2.2E-06	3.7%				
TETRACHLOROETHENE (TETRACHLOROETHYLENE)					2.0E-06	3.4%	2.0E-06	4.2%		
VINYL CHLORIDE					1.1E-06	1.9%				
ALL OTHER VOC COMPOUNDS	1.6E-07	1.0%	1.6E-07	1.1%	1.1E-06	1.8%	8.4E-07	1.7%	2.8E-07	0.6%
VOC RISK	1.7E-05		1.4E-05		6.0E-05		4.8E-05		4.4E-05	

TABLE 5-2 (con't)MEDIAN CANCER RISK EXCEEDANCES AND WOE - VOC ONLY

	WLATS U of L S		WLATS U of L S		WLATS U of L S		EPA / IA	RC WOE
Compound	Cancer Risk	% Contribution to VOC Only Risk	Cancer Risk	% Contribution to VOC Only Risk	Cancer Risk	% Contribution to VOC Only Risk	A / 1	B1, B2, C / 2A, 2B
1,3-BUTADIENE	1.1E-05	25.2%	3.3E-06	8.7%	1.8E-05	28.9%		Х
1,4-DICHLOROBENZENE			3.4E-06	8.8%	3.4E-06	5.3%		Х
ACRYLONITRILE	7.5E-06	16.5%	7.5E-06	19.7%	1.6E-05	26.1%		Х
BENZENE	9.1E-06	20.0%	6.0E-06	15.9%	7.3E-06	11.6%	X	
CARBON TETRACHLORIDE	1.1E-05	25.3%	1.1E-05	30.1%	1.1E-05	18.2%		Х
CHLOROFORM	5.6E-06	12.4%	5.6E-06	14.8%	5.6E-06	9.0%		Х
ETHYL ACRYLATE								Х
METHYLENE CHLORIDE								Х
TETRACHLOROETHENE								Х
(TETRACHLOROETHYLENE)								Δ
VINYL CHLORIDE							X	
ALL OTHER VOC COMPOUNDS	2.8E-07	0.6%	8.1E-07	2.1%	5.1E-07	0.8%		

TABLE 5-2 (con't)MEDIAN CANCER RISK EXCEEDANCES AND WOE - VOC ONLY

VOC RISK

4.5E-05

3.8E-05

6.3E-05

	WLAT EPA Si			S Site 2a ite ID 2	WLATS EPA Si		WLAT EPA Si		WLAT EPA Si	S Site 4 te ID 5
Compound	Cancer Risk	% Contribution to Cumulative Risk	Cancer Risk	% Contribution to Cumulative Risk	Cancer Risk	% Contribution to Cumulative Risk	Cancer Risk	% Contribution to Cumulative Risk	Cancer Risk	% Contribution to Curnulative Risk
FORMALDEHYDE	4.6E-05	23.7%	2.2E-05	4.8%	2.8E-05	4.1%	3.5E-05	8.8%	2.3E-05	13.2%
1,3-BUTADIENE 1,4-DICHLOROBENZENE ACRYLONITRILE	3.0E-05	15.6%	2.9E-04	63.4%	5.0E-04	72.0%	1.9E-04	47.5%	1.7E-05 8.8E-06	9.6% 5.0%
BENZENE	2.0E-05	10.1%	1.2E-05	2.5%	1.2E-05	1.7%	1.4E-05	3.6%	1.3E-05	7.2%
BROMOFORM										
CARBON TETRACHLORIDE	1.1E-05	5.5%	1.1E-05	2.5%	1.1E-05	1.6%	1.2E-05	2.9%	1.1E-05	6.3%
CHLOROFORM	1.6E-05	8.2%	3.1E-05	6.8%	4.5E-05	6.6%	7.7E-05	19.5%	1.9E-05	10.8%
ETHYL ACRYLATE			1.2E-05	2.6%	3.3E-05	4.9%	1.2E-05	2.9%		
METHYLENE CHLORIDE TETRACHLOROETHENE										
(TETRACHLOROETHYLENE)	3.5E-06	1.8%	5.6E-06	1.2%			3.2E-06	0.8%	4.9E-06	2.7%
TRICHLOROETHENE (TRICHLOROETHYLENE)										
VINYL CHLORIDE										
ARSENIC	8.0E-06	4.2%	1.1E-05	2.3%	7.0E-06	1.0%	8.6E-06	2.2%	6.6E-06	3.7%
CADMIUM	1.2E-06	0.6%	1.7E-06	0.4%	1.8E-06	0.3%	2.5E-06	0.6%	0.02.00	21170
CHROMIUM	5.6E-05	28.9%	6.0E-05	12.9%	5.2E-05	7.5%	4.2E-05	10.6%	6.6E-05	36.9%
NICKEL	2.1E-06	1.1%	2.4E-06	0.5%	2.9E-06	0.4%	2.1E-06	0.5%	6.4E-06	3.6%
ALL OTHER COMPOUNDS	5.7E-07	0.3%	1.0E-06	0.2%	7.3E-07	0.1%	7.7E-07	0.2%	1.9E-06	1.0%
CUMULATIVE RISK	1.9E-04		4.6E-04		6.9E-04		4.0E-04		1.8E-04	

TABLE 5-395% UCL CANCER RISK EXCEEDANCES AND WOE

	WLAT EPA Si	S Site 5 ite ID 6		S Site 6 ite ID 7	WLAT U of L S			S Site 8 ite ID M	WLAT U of L S	S Site 9 Site ID I
Compound	Cancer Risk	% Contribution to Cumulative Risk	Cancer Risk	% Contribution to Cumulative Risk	Cancer Risk	% Contribution to Cumulative Risk	Cancer Risk	% Contribution to Cumulative Risk	Cancer Risk	% Contribution to Cumulative Risk
FORMALDEHYDE	2.0E-05	23.7%	1.1E-05	15.2%						
1,3-BUTADIENE 1,4-DICHLOROBENZENE					1.9E-04 1.9E-05	47.9% 4.9%	1.1E-04 9.1E-06	45.1% 3.7%	3.2E-05	24.9%
ACRYLONITRILE					6.6E-05	16.9%	4.2E-05	17.0%	2.9E-05	22.2%
BENZENE	1.0E-05	12.3%	6.8E-06	9.0%	3.2E-05	8.3%	1.9E-05	7.9%	8.6E-06	6.7%
BROMOFORM		10.00			1.3E-05	3.2%				10.000
CARBON TETRACHLORIDE	1.1E-05	13.2%	1.1E-05	14.9%	1.2E-05	3.0%	1.3E-05	5.4%	1.3E-05	10.0%
CHLOROFORM	_				2.0E-05	5.0%	2.0E-05	8.1%	4.2E-05	32.1%
ETHYL ACRYLATE METHYLENE CHLORIDE					1.7E-05	4.5%	1.7E-05	6.7%	5.4E-06	4.2%
TETRACHLOROETHYLENE)					3.2E-06	0.8%	1.7E-05	4.9%	3.4E-00	4.2%
TRICHLOROETHENE (TRICHLOROETHYLENE)					1.6E-05	4.1%	3.0E-06	1.2%		
VINYL CHLORIDE					4.6E-06	1.2%				
ARSENIC	5.7E-06	6.7%	7.7E-06	10.2%						
CADMIUM	1.5E-06	1.8%								
CHROMIUM	3.4E-05	39.8%	3.4E-05	45.6%						
NICKEL	1.7E-06	2.0%	2.8E-06	3.8%						
ALL OTHER COMPOUNDS	4.4E-07	0.5%	1.0E-06	1.4%	0.0E+00	0.0%	0.0E+00	0.0%	0.0E+00	0.0%
CUMULATIVE RISK	8.5E-05		7.6E-05		3.9E-04		2.5E-04		1.3E-04	

TABLE 5-3 (con't)95% UCL CANCER RISK EXCEEDANCES AND WOE

	WLATS U of L S	Site 10 ite ID K	WLATS U of L S		WLATS U of L S		EPA / IA	RC WOE
Compound	Cancer Risk	% Contribution to Cumulative Risk	Cancer Risk	% Contribution to Cumulative Risk	Cancer Risk	% Contribution to Cumulative Risk	A/1	B1, B2, C / 2A, 2B
FORMALDEHYDE								Х
								Х
1,3-BUTADIENE	5.0E-05	39.3%	5.8E-05	41.7%	5.4E-05	39.5%		Х
1,4-DICHLOROBENZENE			6.6E-06	4.7%	6.3E-06	4.6%		Х
ACRYLONITRILE	2.4E-05	18.8%	2.3E-05	16.3%	4.1E-05	30.3%		Х
BENZENE	1.4E-05	10.8%	1.1E-05	8.0%	1.1E-05	8.0%	Х	
BROMOFORM								Х
CARBON TETRACHLORIDE	1.3E-05	10.0%	1.4E-05	10.0%	1.2E-05	8.8%		Х
CHLOROFORM	2.3E-05	18.3%	1.9E-05	13.4%	1.0E-05	7.4%		Х
ETHYL ACRYLATE								Х
METHYLENE CHLORIDE	3.4E-06	2.7%	5.3E-06	3.8%	2.0E-06	1.5%		Х
TETRACHLOROETHENE (TETRACHLOROETHYLENE)								Х
TRICHLOROETHENE (TRICHLOROETHYLENE)			3.1E-06	2.2%				Х
VINYL CHLORIDE							Х	
ARSENIC							X	
CADMIUM								Х
CHROMIUM							Х	
NICKEL							X	
ALL OTHER COMPOUNDS	0.0E+00	0.0%	0.0E+00	0.0%	0.0E+00	0.0%		
CUMULATIVE RISK	1.3E-04		1.4E-04		1.4E-04			

TABLE 5-3 (con't)95% UCL CANCER RISK EXCEEDANCES AND WOE

#### WEST LOUISVILLE AIR TOXICS STUDY

	WLATS Site 1 EPA Site ID 1		WLATS Site 2a EPA Site ID 2		WLATS Site 2b EPA Site ID 3		WLATS Site 3 EPA Site ID 4		WLATS Site 4 EPA Site ID 5	
Compound	Cancer Risk	% Contribution to VOC Only Risk	Cancer Risk	% Contribution to VOC Only Risk	Cancer Risk	% Contribution to VOC Only Risk	Cancer Risk	% Contribution to VOC Only Risk	Cancer Risk	% Contribution to VOC Only Risk
1,3-BUTADIENE	3.0E-05	37.7%	2.9E-04	80.2%	5.0E-04	83.0%	1.9E-04	61.5%	1.7E-05	22.9%
1,4-DICHLOROBENZENE									8.8E-06	11.9%
ACRYLONITRILE										
BENZENE	2.0E-05	24.4%	1.2E-05	3.2%	1.2E-05	1.9%	1.4E-05	4.6%	1.3E-05	17.3%
BROMOFORM										
CARBON TETRACHLORIDE	1.1E-05	13.3%	1.1E-05	3.1%	1.1E-05	1.9%	1.2E-05	3.8%	1.1E-05	15.1%
CHLOROFORM	1.6E-05	19.8%	3.1E-05	8.6%	4.5E-05	7.6%	7.7E-05	25.2%	1.9E-05	25.9%
ETHYL ACRYLATE			1.2E-05	3.2%	3.3E-05	5.6%	1.2E-05	3.8%		
METHYLENE CHLORIDE										
TETRACHLOROETHENE (TETRACHLOROETHYLENE)	3.5E-06	4.4%	5.6E-06	1.5%			3.2E-06	1.0%	4.9E-06	6.5%
TRICHLOROETHENE										
(TRICHLOROETHYLENE) VINYL CHLORIDE										
ALL OTHER VOC COMPOUNDS	2.8E-07	0.4%	3.0E-07	0.1%	2.8E-07	0.0%	4.4E-07	0.1%	3.3E-07	0.4%
VOC RISK	8.0E-05		3.6E-04		6.0E-04		3.1E-04		7.5E-05	

TABLE 5-495% UCL CANCER RISK EXCEEDANCES AND WOE - VOC ONLY

	WLATS Site 5 EPA Site ID 6		WLATS Site 6 EPA Site ID 7		WLATS Site 7 U of L Site ID D		WLATS Site 8 U of L Site ID M		WLATS Site 9 U of L Site ID I	
Compound	Cancer Risk	% Contribution to VOC Only Risk	Cancer Risk	% Contribution to VOC Only Risk	Cancer Risk	% Contribution to VOC Only Risk	Cancer Risk	% Contribution to VOC Only Risk	Cancer Risk	% Contribution to VOC Only Risk
1,3-BUTADIENE					1.9E-04	47.9%	1.1E-04	45.1%	3.2E-05	24.9%
1,4-DICHLOROBENZENE					1.9E-05	4.9%	9.1E-06	3.7%		
ACRYLONITRILE					6.6E-05	16.9%	4.2E-05	17.0%	2.9E-05	22.2%
BENZENE	1.0E-05	47.5%	6.8E-06	37.3%	3.2E-05	8.3%	1.9E-05	7.9%	8.6E-06	6.7%
BROMOFORM					1.3E-05	3.2%				
CARBON TETRACHLORIDE	1.1E-05	51.2%	1.1E-05	61.8%	1.2E-05	3.0%	1.3E-05	5.4%	1.3E-05	10.0%
CHLOROFORM					2.0E-05	5.0%	2.0E-05	8.1%	4.2E-05	32.1%
ETHYL ACRYLATE										
METHYLENE CHLORIDE					1.7E-05	4.5%	1.7E-05	6.7%	5.4E-06	4.2%
TETRACHLOROETHENE (TETRACHLOROETHYLENE)					3.2E-06	0.8%	1.2E-05	4.9%		
TRICHLOROETHENE (TRICHLOROETHYLENE)					1.6E-05	4.1%	3.0E-06	1.2%		
VINYL CHLORIDE					4.6E-06	1.2%				
ALL OTHER VOC COMPOUNDS	2.9E-07	1.3%	1.6E-07	0.9%	0.0E+00	0.0%	0.0E+00	0.0%	0.0E+00	0.0%
VOC RISK	2.2E-05		1.8E-05		3.9E-04		2.5E-04		1.3E-04	

TABLE 5-4 (con't)95% UCL CANCER RISK EXCEEDANCES AND WOE - VOC ONLY

	WLATS Site 10 U of L Site ID K		WLATS Site 11 U of L Site ID N		WLATS Site 12 U of L Site ID F		EPA / IARC WOE	
Compound	Cancer Risk	% Contribution to VOC Only Risk	Cancer Risk	% Contribution to VOC Only Risk	Cancer Risk	% Contribution to VOC Only Risk	A/1	B1, B2, C / 2A, 2B
1,3-BUTADIENE	5.0E-05	39.3%	5.8E-05	41.7%	5.4E-05	39.5%		Х
1,4-DICHLOROBENZENE			6.6E-06	4.7%	6.3E-06	4.6%		Х
ACRYLONITRILE	2.4E-05	18.8%	2.3E-05	16.3%	4.1E-05	30.3%		Х
BENZENE	1.4E-05	10.8%	1.1E-05	8.0%	1.1E-05	8.0%	Х	
BROMOFORM								Х
CARBON TETRACHLORIDE	1.3E-05	10.0%	1.4E-05	10.0%	1.2E-05	8.8%		Х
CHLOROFORM	2.3E-05	18.3%	1.9E-05	13.4%	1.0E-05	7.4%		Х
ETHYL ACRYLATE								Х
METHYLENE CHLORIDE	3.4E-06	2.7%	5.3E-06	3.8%	2.0E-06	1.5%		Х
TETRACHLOROETHENE (TETRACHLOROETHYLENE)								Х
TRICHLOROETHENE (TRICHLOROETHYLENE)			3.1E-06	2.2%				Х
VINYL CHLORIDE							Х	
ALL OTHER VOC COMPOUNDS	0.0E+00	0.0%	0.0E+00	0.0%	0.0E+00	0.0%		

TABLE 5-4 (con't)95% UCL CANCER RISK EXCEEDANCES AND WOE - VOC ONLY

VOC RISK

1.3E-04

1.4E-04

1.4E-04

	WLAT EPA Si		WLATS EPA Si	S Site 2a te ID 2	WLATS EPA Si			S Site 3 ite ID 4	WLAT EPA Si	S Site 4 ite ID 5
Compound	Non-cancer Risk	% Contribution to Cumulative Risk	Non-cancer Risk	% Contribution to Cumulative Risk	Non-cancer Risk	% Contribution to Cumulative Risk	Non-cancer Risk	% Contribution to Cumulative Risk	Non-cancer Risk	% Contribution to Cumulative Risk
FORMALDEHYDE	1.3E-01	14.2%	1.1E-01	9.0%	1.7E-01	9.7%	1.3E-01	11.4%	1.3E-01	15.8%
1,3-BUTADIENE ACRYLONITRILE	1.7E-01	17.7%	5.0E-01	39.8%	9.5E-01	54.8%	5.4E-01	46.1%	1.4E-01	17.1%
MANGANESE	3.0E-01	32.2%	3.1E-01	24.9%	3.0E-01	17.3%	2.1E-01	18.0%	2.8E-01	33.3%
ALL OTHER COMPOUNDS	3.3E-01	35.9%	3.3E-01	26.2%	3.2E-01	18.2%	2.8E-01	24.5%	2.8E-01	33.9%
CUMULATIVE RISK	9.3E-01		1.2E+00		1.7E+00		1.2E+00		8.4E-01	

 TABLE 5-5

 MEDIAN NON-CANCER RISK EXCEEDANCES AND CRITICAL EFFECT

		S Site 5 ite ID 6	WLAT EPA Si	S Site 6 ite ID 7	WLAT U of L S	S Site 7 lite ID D		S Site 8 Site ID M		S Site 9 Site ID I
Compound	Non-cancer Risk	% Contribution to Cumulative Risk	Non-cancer Risk	% Contribution to Cumulative Risk	Non-cancer Risk	% Contribution to Cumulative Risk	Non-cancer Risk	% Contribution to Cumulative Risk	Non-cancer Risk	% Contribution to Cumulative Risk
FORMALDEHYDE										
1,3-BUTADIENE					3.0E-01	62.6%	2.0E-01	60.1%		
ACRYLONITRILE									1.4E-01	51.7%
MANGANESE	1.4E-01	32.0%								
ALL OTHER COMPOUNDS	2.9E-01	68.0%	3.1E-01	100.0%	1.8E-01	37.4%	1.3E-01	39.9%	1.3E-01	48.3%
CUMULATIVE RISK	4.3E-01		3.1E-01		4.8E-01		3.3E-01		2.7E-01	

 TABLE 5-5 (con't)

 MEDIAN NON-CANCER RISK EXCEEDANCES AND CRITICAL EFFECT

		S Site 10 Site ID K		S Site 11 Site ID N		S Site 12 Site ID F	
Compound	Non-cancer Risk	% Contribution to Cumulative Risk	Non-cancer Risk	% Contribution to Cumulative Risk	Non-cancer Risk	% Contribution to Cumulative Risk	Critical Effect
FORMALDEHYDE							Respiratory
1,3-BUTADIENE	1.9E-01	56.9%			3.0E-01	59.9%	Reproductive
ACRYLONITRILE					1.2E-01	23.9%	Respiratory
MANGANESE							Neurological
ALL OTHER COMPOUNDS	1.4E-01	43.1%	1.9E-01	100.0%	8.2E-02	16.2%	

1.9E-01

## TABLE 5-5 (con't) MEDIAN NON-CANCER RISK EXCEEDANCES AND CRITICAL EFFECT

CUMULATIVE RISK

3.3E-01

5.0E-01

	WLAT EPA Si			S Site 2a ite ID 2	WLATS EPA Si	S Site 2b ite ID 3		S Site 3 ite ID 4	WLAT EPA S	S Site 4 ite ID 5
Compound	Non-cancer Risk	% Contribution to VOC Only Risk	Non-cancer Risk	% Contribution to VOC Only Risk	Non-cancer Risk	% Contribution to VOC Only Risk	Non-cancer Risk	% Contribution to VOC Only Risk	Non-cancer Risk	% Contribution to VOC Only Risk
1,3-BUTADIENE	1.7E-01	50.1%	5.0E-01	78.5%	9.5E-01	87.1%	5.4E-01	80.2%	1.4E-01	55.8%
ACRYLONITRILE										
ALL OTHER VOC COMPOUNDS	1.6E-01	49.9%	1.4E-01	21.5%	1.4E-01	12.9%	1.3E-01	19.8%	1.1E-01	44.2%
VOC RISK	3.3E-01		6.3E-01		1.1E+00		6.7E-01		2.6E-01	

# TABLE 5-6 MEDIAN NON-CANCER RISK EXCEEDANCES AND CRITICAL EFFECT - VOC ONLY

	WLAT EPA Si	S Site 5 ite ID 6	WLAT EPA S	S Site 6 ite ID 7		S Site 7 Site ID D		S Site 8 ite ID M	WLAT U of L S	
Compound	Non-cancer Risk	% Contribution to VOC Only Risk	Non-cancer Risk	% Contribution to VOC Only Risk	Non-cancer Risk	% Contribution to VOC Only Risk	Non-cancer Risk	% Contribution to VOC Only Risk	Non-cancer Risk	% Contribution to VOC Only Risk
1,3-BUTADIENE					3.0E-01	62.6%	2.0E-01	60.1%		
ACRYLONITRILE									1.4E-01	51.7%
ALL OTHER VOC COMPOUNDS	8.1E-02	100.0%	7.4E-02	100.0%	1.8E-01	37.4%	1.3E-01	39.9%	1.3E-01	48.3%
VOC RISK	8.1E-02		7.4E-02		4.8E-01		3.3E-01		2.7E-01	

 TABLE 5-6 (con't)

 MEDIAN NON-CANCER RISK EXCEEDANCES AND CRITICAL EFFECT - VOC ONLY

	WLATS U of L S	Site 10 ite ID K		S Site 11 Site ID N	WLATS U of L S	Site 12 Site ID F	
Compound	Non-cancer Risk	% Contribution to VOC Only Risk	Non-cancer Risk	% Contribution to VOC Only Risk	Non-cancer Risk	% Contribution to VOC Only Risk	Critical Effect
1,3-BUTADIENE	1.9E-01	56.9%			3.0E-01	59.9%	Reproductive
ACRYLONITRILE					1.2E-01	23.9%	Respiratory
ALL OTHER VOC COMPOUNDS	1.4E-01	43.1%	1.9E-01	100.0%	8.2E-02	16.2%	

## TABLE 5-6 (con't) MEDIAN NON-CANCER RISK EXCEEDANCES AND CRITICAL EFFECT - VOC ONLY

VOC RISK

3.3E-01

1.9E-01

5.0E-01

SCIENCES INTERNATIONAL, INC.

	WLAT EPA S	S Site 1 ite ID 1		S Site 2a ite ID 2	WLATS EPA Si	S Site 2b ite ID 3		S Site 3 ite ID 4		S Site 4 ite ID 5
Compound	Non-cancer Risk	% Contribution to Cumulative Risk	Non-cancer Risk	% Contribution to Cumulative Risk	Non-cancer Risk	% Contribution to Cumulative Risk	Non-cancer Risk	% Contribution to Cumulative Risk	Non-cancer Risk	% Contribution to Cumulative Risk
FORMALDEHYDE	3.6E-01	19.3%	1.7E-01	2.9%	2.2E-01	2.3%	2.7E-01	6.5%	1.8E-01	12.9%
1,3-BUTADIENE ACRYLONITRILE BROMOFORM	5.0E-01	27.0%	4.9E+00	80.7%	8.3E+00	87.7%	3.1E+00	74.2%	2.8E-01	19.8%
CARBON DISULFIDE										
TOLUENE			1.3E-01	2.2%	1.3E-01	1.4%				
MANGANESE	4.7E-01	25.0%	4.2E-01	6.9%	4.1E-01	4.4%	3.3E-01	7.8%	5.1E-01	35.8%
ALL OTHER COMPOUNDS	5.3E-01	28.6%	4.4E-01	7.3%	4.0E-01	4.2%	4.9E-01	11.5%	4.5E-01	31.5%
CUMULATIVE RISK	1.9E+00		6.0E+00		9.4E+00		4.2E+00		1.4E+00	

TABLE 5-795% UCL NON-CANCER RISK EXCEEDANCES AND CRITICAL EFFECT

		S Site 5 ite ID 6		S Site 6 ite ID 7	WLAT U of L S	S Site 7 ite ID D		S Site 8 Site ID M		S Site 9 Site ID I
Compound	Non-cancer Risk	% Contribution to Cumulative Risk	Non-cancer Risk	% Contribution to Cumulative Risk	Non-cancer Risk	% Contribution to Cumulative Risk	Non-cancer Risk	% Contribution to Cumulative Risk	Non-cancer Risk	% Contribution to Cumulative Risk
FORMALDEHYDE	1.6E-01	21.8%								
1,3-BUTADIENE ACRYLONITRILE					3.1E+00 4.8E-01	64.2% 10.0%	1.9E+00 3.1E-01	60.5% 10.1%	5.4E-01 2.1E-01	51.0% 20.0%
BROMOFORM CARBON DISULFIDE					1.6E-01 1.5E-01	3.4% 3.2%				
TOLUENE					5.1E-01	10.5%	5.8E-01	19.0%	1.5E-01	13.7%
MANGANESE	2.8E-01	38.7%	1.7E-01	34.1%						
ALL OTHER COMPOUNDS	2.9E-01	39.5%	3.3E-01	65.9%	4.2E-01	8.7%	3.2E-01	10.4%	1.6E-01	15.2%
CUMULATIVE RISK	7.2E-01		5.0E-01		4.8E+00		3.1E+00		1.1E+00	

TABLE 5-7 (con't)95% UCL NON-CANCER RISK EXCEEDANCES AND CRITICAL EFFECT

		S Site 10 Site ID K		S Site 11 Site ID N		S Site 12 Site ID F	
Compound	Non-cancer Risk	% Contribution to Cumulative Risk	Non-cancer Risk	% Contribution to Cumulative Risk	Non-cancer Risk	% Contribution to Cumulative Risk	Critical Effect
FORMALDEHYDE							Respiratory
1,3-BUTADIENE	8.4E-01	64.8%	9.7E-01	69.6%	8.9E-01	66.7%	Reproductive
ACRYLONITRILE	1.8E-01	13.7%	1.7E-01	12.0%	3.0E-01	22.6%	Respiratory
BROMOFORM							Hepatic
CARBON DISULFIDE							Neurological
TOLUENE	1.2E-01	9.7%	1.0E-01	7.5%			Neurological
MANGANESE							Neurological
ALL OTHER COMPOUNDS	1.5E-01	11.8%	1.5E-01	11.0%	1.4E-01	10.7%	

1.4E+00

# TABLE 5-7 (con't)95% UCL NON-CANCER RISK EXCEEDANCES AND CRITICAL EFFECT

CUMULATIVE RISK

1.3E+00

1.3E+00

	WLAT EPA Si			S Site 2a ite ID 2	WLATS EPA Si	S Site 2b ite ID 3		S Site 3 ite ID 4		S Site 4 ite ID 5
Compound	Non-cancer Risk	% Contribution to VOC Only Risk	Non-cancer Risk	% Contribution to VOC Only Risk	Non-cancer Risk	% Contribution to VOC Only Risk	Non-cancer Risk	% Contribution to VOC Only Risk	Non-cancer Risk	% Contribution to VOC Only Risk
1,3-BUTADIENE	5.0E-01	63.3%	4.9E+00	94.4%	8.3E+00	96.4%	3.1E+00	92.5%	2.8E-01	60.8%
ACRYLONITRILE										
BROMOFORM										
CARBON DISULFIDE										
TOLUENE			1.3E-01	2.6%	1.3E-01	1.6%				
ALL OTHER VOC COMPOUNDS	2.9E-01	36.7%	1.6E-01	3.0%	1.8E-01	2.1%	2.5E-01	7.5%	1.8E-01	39.2%
VOC RISK	7.9E-01		5.2E+00		8.6E+00		3.4E+00		4.7E-01	

# TABLE 5-8 95% UCL NON-CANCER RISK EXCEEDANCES AND CRITICAL EFFECT - VOC ONLY

		S Site 5 ite ID 6		S Site 6 ite ID 7	WLAT: U of L S			S Site 8 lite ID M		S Site 9 Site ID I
Compound	Non-cancer Risk	% Contribution to VOC Only Risk	Non-cancer Risk	% Contribution to VOC Only Risk	Non-cancer Risk	% Contribution to VOC Only Risk	Non-cancer Risk	% Contribution to VOC Only Risk	Non-cancer Risk	% Contribution to VOC Only Risk
1,3-BUTADIENE					3.1E+00	64.2%	1.9E+00	60.5%	5.4E-01	51.0%
ACRYLONITRILE					4.8E-01	10.0%	3.1E-01	10.1%	2.1E-01	20.0%
BROMOFORM					1.6E-01	3.4%				
CARBON DISULFIDE					1.5E-01	3.2%				
TOLUENE					5.1E-01	10.5%	5.8E-01	19.0%	1.5E-01	13.7%
ALL OTHER VOC COMPOUNDS	1.3E-01	100.0%	8.9E-02	100.0%	4.2E-01	8.7%	3.2E-01	10.4%	1.6E-01	15.2%
VOC RISK	1.3E-01		8.9E-02		4.8E+00		3.1E+00		1.1E+00	

## TABLE 5-8 (con't) 95% UCL NON-CANCER RISK EXCEEDANCES AND CRITICAL EFFECT - VOC ONLY

	WLATS U of L S	Site 10 ite ID K	WLATS U of L S	S Site 11 Site ID N		S Site 12 Site ID F	
Compound	Non-cancer Risk	% Contribution to VOC Only Risk	Non-cancer Risk	% Contribution to VOC Only Risk	Non-cancer Risk	% Contribution to VOC Only Risk	Critical Effect
1,3-BUTADIENE	8.4E-01	64.8%	9.7E-01	69.6%	8.9E-01	66.7%	Reproductive
ACRYLONITRILE	1.8E-01	13.7%	1.7E-01	12.0%	3.0E-01	22.6%	Respiratory
BROMOFORM							Hepatic
CARBON DISULFIDE							Neurological
TOLUENE	1.2E-01	9.7%	1.0E-01	7.5%			Neurological
ALL OTHER VOC COMPOUNDS	1.5E-01	11.8%	1.5E-01	11.0%	1.4E-01	10.7%	

## TABLE 5-8 (con't) 95% UCL NON-CANCER RISK EXCEEDANCES AND CRITICAL EFFECT - VOC ONLY

VOC RISK

1.3E+00

1.4E+00

1.3E+00

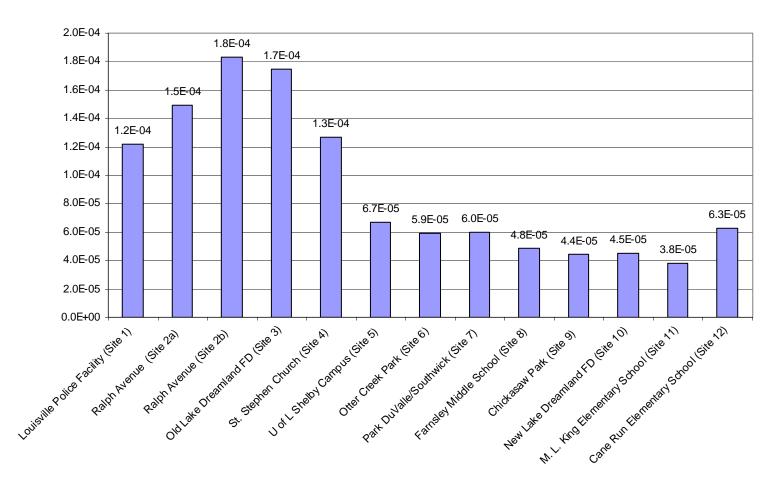


Figure 5-1 Median Exposure Case Cancer Risk - All COPCs

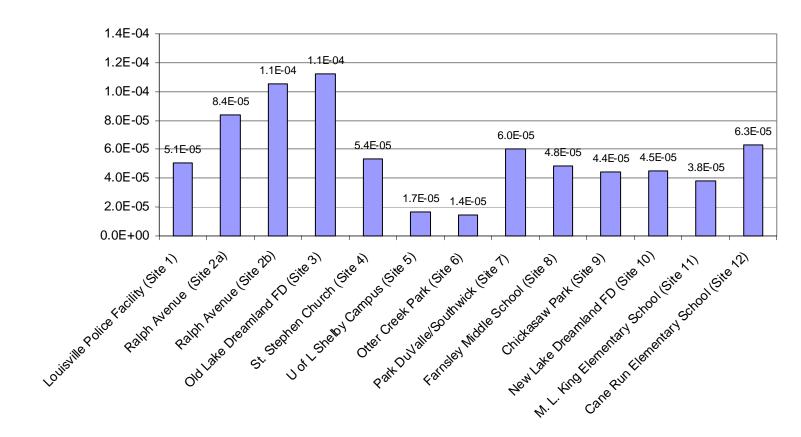
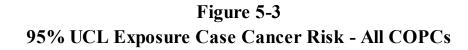
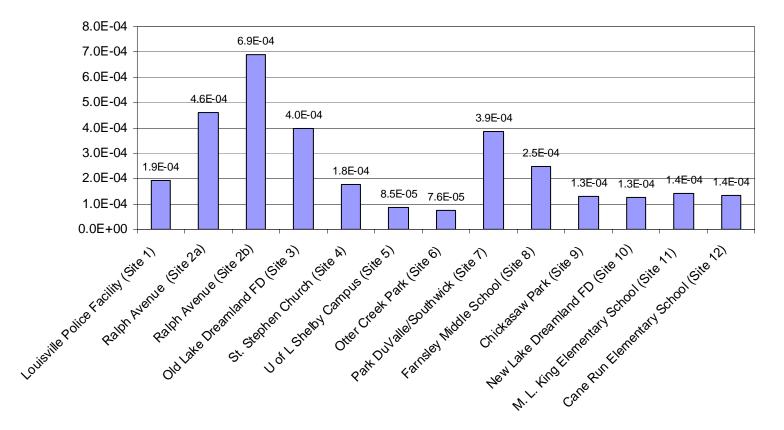


Figure 5-2 Median Exposure Case Cancer Risk - VOCs Only





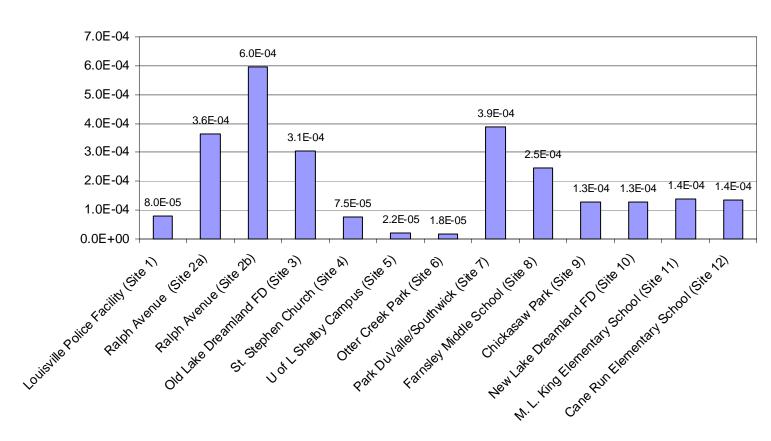
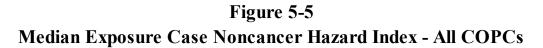
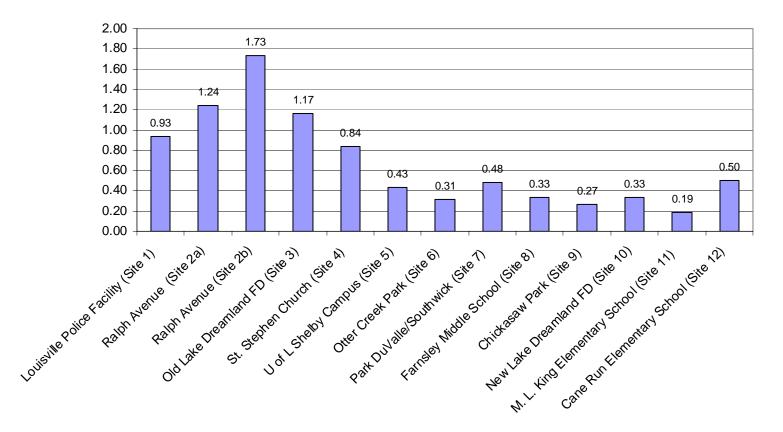
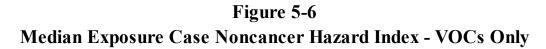
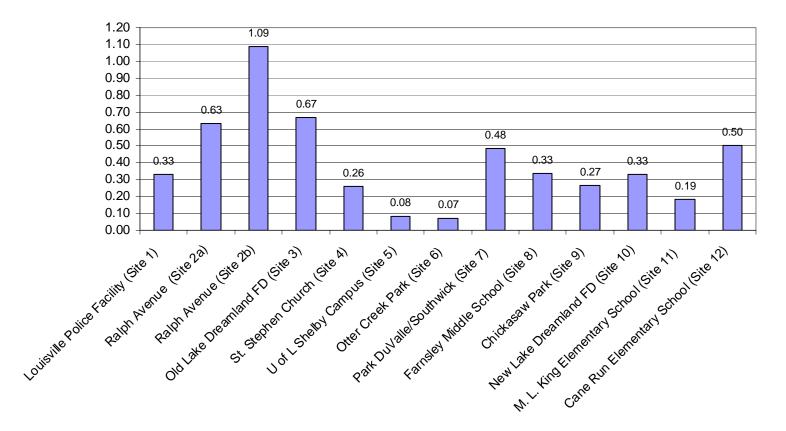


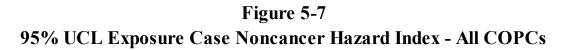
Figure 5-4 95% UCL Exposure Case Cancer Risk - VOCs Only

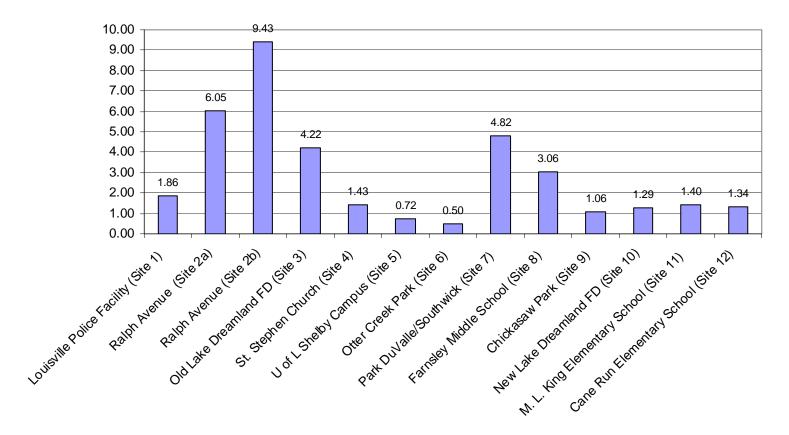


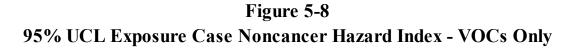


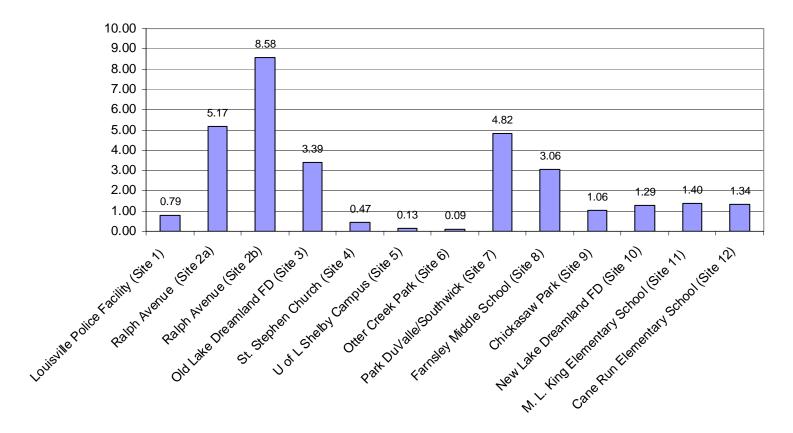












## 6.0 UNCERTAINTY ANALYSIS

All risk assessments involve the use of assumptions, judgment and incomplete data to varying degrees. The way in which these uncertainties are addressed and incorporated into the risk assessment can significantly affect the degree of conservatism in the risk assessment. An uncertainty analysis provides a means to review the factors that contribute to the risk assessment and gain a better perspective on the risk estimates that are presented. A logical way to discuss the uncertainty of the risk assessment is to go through each of the major components and identify the key variables and assumptions that have the most impact on the uncertainty in the risk assessment. Whenever possible, the discussion will present examples of how much the risk estimates could change with changes in the assumptions or methodology. Steps that could be taken to reduce the uncertainty are also identified when alternatives are available. The remainder of this discussion will follow the major Sections of the Risk Assessment presented thus far.

## 6.1 Monitoring Program

One of the primary uncertainties 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. One means to reduce the uncertainty in monitor placement is to conduct air modeling to identify relevant locations based on local meteorology and information about the sources of the airborne chemicals.

A large number of chemicals were selected for monitoring in the WLATS program. However, it is possible that important chemicals were not evaluated because of a lack of resources or because test procedures were not available for the chemicals. In the WLATS program, the impact of limiting the number of chemicals analyzed can be observed by looking at the difference in risk estimates for VOC only monitors versus monitors that analyzed VOCs, SVOCs, and metals. Clearly, limiting the number of chemicals analyzed in the monitoring program can result in an underestimation of risk, which could in some case 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 released into the air from major sources in the Louisville area, which helps to reduce the uncertainty associated when selecting the set of chemicals to be evaluated.

A thorough review of the equipment and quality assurance used for the monitoring program was not conducted as part of this investigation. However, it appears that standard equipment and methods were used for the monitoring program. The standard equipment and methods for sample collection and handling are typically tested to ensure that accurate and reliable results will be obtained. This would include discarding sampling data if the method was not followed closely enough. Rejecting data from the

monitoring program could lead to an over- or underestimation of the potential health impacts.

Duplicate monitors were placed at the Ralph Avenue and Campground Site (WLATS 2a and 2b). The data from the USEPA collocated monitors at this site were evaluated separately as a means to provide some insights into the variability and uncertainty in the results from the monitoring program. No attempt was made to conduct a statistical analysis of the differences in the results for monitors 2a and 2b. Rather, the direct impact on the risk estimates will be used as a means to discuss the uncertainty. Each monitor had analysis for VOCs, SVOCs, and metals. For the VOCs, monitor 2a had 33 chemicals detected from which 30 COPCs were selected, while monitor 2b had 40 chemicals detected, with the same 30 COPCs selected as for monitor 2a. For the SVOCs, monitor 2a had 25 detected chemicals, with 21 selected as COPCs, while monitor 2b had 23 chemicals detected with 16 of the 17 COPCs for monitor 2b also selected as COPCs for monitor 2a. Finally, for metals the monitors had the same 26 chemicals detected and the same 24 COPCs selected for evaluation in the risk assessment.

From a risk perspective the results indicate that the two monitors are in good general agreement. The risk estimates for monitor 2b were higher than for 2a in all cases for both cancer and non-cancer effects. The percent difference between the risk estimates for and COPCs at monitor 2a versus 2b is as follows: for the median case cancer risk the percent difference was 16%; for the 95% UCL exposure the percent difference in the cancer risks was 33%; for the median exposure case non-cancer HI the percent difference was 30%; and, for the 95% UCL exposure case the percent difference was 36%. These results tend to indicate that the monitoring results are generally reliable and will provide consistent results that vary by less than a factor of two from a risk perspective.

## 6.2 Data Analysis and Selection of Chemicals of Potential Concern

A validated data set of analytical results for the monitors in the WLATS program was provided as the basis for the risk assessment. This dataset contained the airborne concentrations for various chemicals detected during the year of monitoring. In some cases, the laboratory could not definitively determine the chemical responsible for the measured air concentration. The air concentrations were then assigned to a chemical that most closely matched characteristics of the unknown chemical, based on the laboratory tests. Because the chemical cannot be definitively determined, it is classified as a tentatively identified compound (TIC). Because of the uncertainty associated with the TICs, they were not evaluated in this risk assessment. This may lead to an underestimation of the health impacts. Conversely, evaluating a TIC as a more potent chemical for causing adverse health effects than may actually be the case would overestimate the potential health impacts. Additional monitoring or different techniques applied in the laboratory might reduce this uncertainty.

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

significant chemicals. Any chemical that was not detected in at least 10% of the samples reported for a location was removed from further analysis in the risk assessment. Application of this 10% rule for each monitor location led to the selection of the chemicals of potential concern (COPCs) for evaluation in the chronic and acute risk assessment. Eliminating chemicals that were infrequently detected could lead to an underestimate of the health impacts. A total of 26 chemicals positively detected in at least one monitor in the WLATS network were eliminated from the risk evaluation because they were found in less than 10% of the samples.

The potential to underestimate the health impacts might be reduced if all of the chemicals detected in the monitoring program were included in the risk assessment. Then a decision must be made whether to use only the detected concentrations to estimate what people could be exposed to, or to assume some value for all cases where the concentration was not detected and then combine these with the detected concentrations to calculate an exposure concentration using all of the samples. Using only the detected concentrations would tend overestimate the potential health impacts associated with the true exposure. Similarly, it could be argued that the true concentration of the chemical is overestimated when the data without detections is used with an assumed value if the true value is significantly lower than the assumed value because it is only there in extremely small concentrations most of the time.

### 6.3 Exposure Assessment

For this risk assessment, the exposure assessment consisted of conducting statistical tests on the dataset and then calculating exposure point air concentrations using the most appropriate method as defined by the test results. The statistical tests were used to determine the shape of the distribution. If the data for a chemical at a monitor were normally distributed, then the 95% UCL of the mean air concentration was calculated using normal statistics (see Equation 3-3). Otherwise a lognormal distribution was assumed, and the 95% UCL of the mean air concentration was calculated using lognormal statistics (see Equation 3-4).

If the assumption of lognormality was incorrect, this would introduce uncertainty into the risk estimates for the 95% UCL exposure case. As part of the risk assessment, the validity of the lognormal assumption was tested for all chemicals that were risk drivers and were not normally distributed. This analysis indicated that many of the risk drivers did not fit either a normal or a lognormal distribution. The impact of this uncertainty is unknown. The application of more advanced statistical techniques could reduce the uncertainty. Appendix D provides a complete listing of the distribution test results for all COPCs in the WLATS network.

In calculating the air concentrations for the median and 95% UCL exposure cases for COPCs, a value of <sup>1</sup>/<sub>2</sub> the detection limit was used for samples where the actual concentration was not detectable. As discussed in Section 6.2, the potential impact of

this uncertainty on the risk estimates could be to over- or underestimate the actual health impacts.

The use of a median and 95% UCL on the mean for the exposure point air concentrations was designed to reflect a central tendency and reasonably conservative estimate of the true exposure. While the median concentration is unlikely to overestimate the true exposure, the 95% UCL on the mean may in fact be an overestimate. By definition, the 95% UCL on the mean implies that there is a 95% probability that the true mean of the air concentration is lower, and only a 5% probability that the true mean is higher. Another conservative aspect of the 95% UCL exposure case occurred when the calculated 95% UCL on the mean was greater than the maximum detected air concentration for the monitor. In this case, the maximum air concentration for the monitor was used to calculate the risk estimates for the 95% UCL exposure case. Air modeling could be used to attempt to reduce the uncertainty in the exposure point air concentrations.

A standard component of an exposure assessment is analysis that determines all of the routes of exposure associated with a COPC. This risk assessment evaluated the inhalation exposure route for the airborne chemicals detected in the WLATS monitors. There is no doubt that people in the vicinity of the WLATS monitoring program are breathing the chemicals found in the air monitors. Therefore, this is an actual exposure pathway. There may be other exposure pathways that are also complete in the sense that people could actually be exposed to the chemicals by another means. For example, airborne chemicals could deposit onto soil or surface water and lead to exposures via dermal or ingestion pathways. The true health impacts may be underestimates by an unknown amount as a result of ignoring these pathways. A more thorough multi-pathway risk assessment based on additional monitoring and/or modeling data could reduce this uncertainty.

Another typical aspect of an exposure assessment is calculating the dose that an individual could receive as a result of the exposure evaluated. Factors such as the frequency and duration of the exposure are selected to match the behavior of the population being modeled. For this risk assessment, it was assumed that an individual was exposed for 24 hours per day, 365 day a year, for 70 years, as per the Risk Assessment Work Plan. These assumptions may underestimate potential health impacts if the air concentrations increase over time. More likely, they will overestimate potential health impacts for this exposure, at least for a large portion of the population, as their movements in and out of the area would reduce the exposure time. Conducting an analysis of the behaviors and activity patterns of the residents, and developing more site-specific values for the frequency and duration of the exposure could reduce this uncertainty.

## 6.4 Hazard Identification and Dose-Response Assessment

The Hazard Identification and Dose-Response portion of the risk assessment is designed to identify the potential health hazards associated with the COPCs selected for the risk

assessment, and to obtain a toxicity value which provides a numerical expression of the incidence of adverse health effects based on the dose received. The risk characterization step then combines the toxicity values with the dose estimates made for the receptors being evaluated in the risk assessment, to develop estimates of risk for public health. The primary source of the toxicity values used in this risk assessment was the EPA Integrated Risk Information System (IRIS). The nature of the uncertainties in the toxicity values from IRIS also apply to the other sources of toxicity data used in this assessment (i.e., CalEPA).

Uncertainties in the toxicity values used for this risk assessment stem from a number of sources. The first area of uncertainty is in the adequacy of the database available to assess the dose-response relationship. A number of techniques are available to derive a toxicity value using the dose-response relationship, and each imparts some level of uncertainty as well. An additional factor in this risk assessment was that toxicity values did not exist for some COPCs, which meant that the chemicals were either eliminated from further in the risk assessment, or surrogate data was used. Use of either alternative will introduce another degree of uncertainty into the risk estimates.

The need for an adequate toxicity database from which to develop the dose-relationship is essential for deriving a representative toxicity value. In most cases dose-response data is not available for human exposures. Therefore animal studies are used to represent the potential affects in humans. In addition, the number of studies available for a chemical may not be sufficient to provide a clear picture of the true dose-response, especially in the region where the exposure is to low doses. In many cases the toxicity studies are based on exposures to animals at high doses of the chemical, doses that are less than a chronic duration, or doses that do not reach a no-effect level. Regardless of whether the dose is in animals or humans, there is uncertainty regarding the effect on especially sensitive populations that are not considered in the dose-response relationship. For non-cancer toxicity values, uncertainty and modifying factors are used to account for these factors. For cancer toxicity values, which are estimates of the probability to develop cancer as a result of a given exposure, the value is based on a linear extrapolation to zero or the background dose, from a point of departure on the dose-response curve, or it is based on the use of uncertainty and modifying factors similar to the method used for noncarcinogens. The intent in both cases is to provide toxicity values that tend to overestimate the risks in the face of uncertainty in the derivation of the value.

An example of the uncertainty in the IRIS toxicity values is the case of acrylonitrile, which is a risk driver in this risk assessment. The current IRIS value for acrylonitrile was used in this risk assessment to assess the potential for cancer health effects, as per the Risk Assessment Work Plan guidance. The database of toxicity studies used for the IRIS acrylonitrile value was developed in 1983. A screening-level review conducted for IRIS by an EPA contractor in September of 2002 identified one or more significant new studies in the more recent toxicology literature pertinent to the cancer assessment for acrylonitrile (IRIS, 2003). The International Agency for Research on Cancer (IARC) conducted an evaluation of acrylonitrile in 1999 using more recent occupational exposure toxicity studies and concluded there was not a credible association between acrylonitrile and lung cancer (IARC, 1999). IARC stated that the new human studies corrected for actual or potential problems in the previous studies used to assess acrylonitrile carcinogenicity. As a result of including the new human epidemiology studies into the analysis of acrylonitrile carcinogenicity, IARC lowered their classification of acrylonitrile to a 2B (i.e., possibly carcinogenic in humans) indicating that there is inadequate evidence in humans. The previous IARC classification for acrylonitrile had been as a 2A, which is used for probable human carcinogens. IRIS currently classifies acrylonitrile as a probable human carcinogen (B1).

Toxicity data were not available for some chemicals detected in the WLATS monitoring program. In some cases, a surrogate value was used for these chemicals based on a similar chemical for which toxicity data was available. The chemicals for which surrogates were applied are noted in the summary tables provided in Section 4. Similarly, for some chemicals the only toxicity value available was related to exposures via oral ingestion rather than inhalation, which was the focus of this risk assessment. In these cases, an inhalation toxicity value was developed from the oral value using a route-to-route extrapolation. While the use of the surrogate data and route-to-route extrapolations to fill gaps in the toxicity database will introduce additional uncertainty into the risk estimates, this conservative approach will tend to overestimate the potential health impacts relative to ignoring chemicals without toxicity data entirely. Reducing this uncertainty would require additional toxicity research and studies.

The evaluation of chromium and nickel in the risk assessment was based on the assumption that both chemicals existed entirely in their most toxic forms. Specifically, for chromium, it was assumed that all chromium detected was hexavalent chromium particulates, and that all nickel was nickel subsulfide. In the National Air Toxics Assessment, EPA assumed that 34% of a total chromium measurement was in the toxic hexavalent chromium form (EPA, 2001). They added that it is likely that most sources of chromium emissions in the United States contain smaller amounts of hexavalent chromium. For nickel, the EPA assumed that 65% of the total nickel measurement was in the toxic nickel subsulfide form (EPA, 2001). The assumption that the measurements of total nickel and chromium from the WLATS monitors was all (i.e., 100%) in the most toxic forms is likely to overestimate the potential human health impacts. Additional monitoring that identifies the form of the chromium and nickel in the air at the WLATS monitors would reduce this uncertainty.

## 6.5 Risk Characterization

In the risk characterization, the toxicity and exposure assessments were combined to develop a quantitative description of the potential for adverse human health effects. Thus all of the uncertainties related to the steps in the exposure and toxicity assessments affect these risk estimates. In addition, there are uncertainties related to how the risk characterization is presented and interpreted. For example, for both carcinogenic and non-cancer risk estimates, the cancer risk and HQs for individual COPCs were added to obtain an indication of the total health impact at a monitor location. This assumption

ignores the potential for synergisms or antagonisms among chemicals, effectively assuming that all of the chemicals have a similar mechanism of action and metabolism in the human body. This assumption would tend to overestimate true risks if antagonistic effects occurred, and would underestimate risks if synergistic effects were to occur. Information to evaluate these effects for carcinogens is generally lacking. For noncarcinogens, it is possible to develop HIs that group together chemicals with similar target organs for the critical health effect. For this risk assessment, most HIs calculated without regard to target organ were less than 1, indicating that an adverse health effect was unlikely. Thus, summing the HI on the basis of target organ would not be useful. In the cases where the HI summed for all chemicals did exceed a value of 1, the primary contributor to the exceedance typically exceeded a value of 1 by itself, so that the prediction for the potential of an adverse health effect would not change.

The risk estimates for exposure to the airborne concentration found in the WLATS monitoring programs assumes that an individual is continuously exposed at the same location for 70 years. As discussed earlier, the actual behaviors and activities of the residents may result in lower exposures, in which case the risk estimates may overestimate the true risks. Information on the actual population of interest could reduce this uncertainty.

The WLATS monitoring data used in the risk assessment reflects a single year 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.

The risk estimates provided in this assessment were based on monitoring results from 12 locations throughout the Louisville area. It is not clear how well these locations represent any other receptors in the Louisville area. An inspection of the total risk graphs presented in Section 5 shows the variability across the monitoring network. Assuming that a monitor was representative of any location beyond where it was sited would introduce an uncertainty that my over- or underestimate the true health impacts at the unmonitored locations. The sites that were selected to represent maximum impact locations were in fact had either the highest (in the case of monitor 2b) or among the highest (in the case of monitors 2a and 1) risk estimates for the exposure evaluated in this assessment.

Another source of uncertainty in the risk estimates were the missing sample dates for a number of monitors. A detailed statistical analysis was not conducted to evaluate the impact of the missing sample dates on the risk estimates. However, a review of the risk drivers and risk estimates for the monitors can provide some insights into the potential impacts of the missing sample dates. Monitors seven through 12 were all analyzed for VOCs as part of the program operated by the University of Louisville. Within this group of monitors, Site 7 is noteworthy as it had between eight and 14 sample dates available for analysis, and it showed the highest or second highest risk impact for this group of

monitors for all exposure cases evaluated. With the exception of Site 9, which had between six and 11 sample dates available for analysis, all of the other monitors in this group had results for more sample dates than Site 7. Site 7 had the second highest values for the median exposure case cancer and non-cancer risk estimates. For the 95% UCL exposure case, Site 7 had the highest risk estimates for both the cancer and non-cancer cases. The risk estimates for Site 7 were approximately two to four times higher than for the other monitors. For several risk drivers at Site 7, the risk estimate for the 95% UCL exposure case was based on the maximum detected concentration due to an inability to compute a representative 95% UCL of the mean. This was not the case for the other monitors. In general, all of the monitors, including Site 7, had the same COPCs dominating the total risk estimate. In some cases, Site 7 had more COPCs as risk drivers than any of the other monitors, but these unique COPCs contributed less than 10% to the total risk estimate for Site 7. In summary, the missing sample dates may tend to lead to an overestimate of the true risks at the site, especially for the 95% UCL exposure case.

## 7.0 CONCLUSIONS

A risk assessment of the potential human health impacts from inhalation of air toxics has been conducted using data collected during the WLATS air-monitoring program in the Metropolitan Louisville, KY area. In general, this risk assessment can be considered a conservative estimate on the basis of the exposure assessment. For example, for the chronic risk estimates it was assumed that an individual would be exposed to the monitored concentrations over 70 years, for 24 hours per day. The potential human health implications of these chronic exposures were characterized for both cancer and non-cancer health effects. In addition, an acute risk characterization, representing a 24hour exposure to elevated concentrations in air, was performed by comparing the maximum concentrations measured at the monitor location to the relevant acute toxicity criteria. The remainder of this Section provides the conclusions of the chronic and then the acute risk assessments.

## 7.1 Chronic Risk Characterization

The cancer risks for the WLATS program exceeded a value of  $1 \times 10^{-6}$  for all WLATS monitors, including background monitors, for both the median and 95% UCL exposure cases. A cancer risk of  $1 \times 10^{-6}$  is used in the State of Kentucky as the threshold for acceptable risks. The maximum impact for the entire WLATS monitoring network occurred at Site 2b for all but the median exposure to VOCs only, in which case Site 3 was slightly higher than Site 2b. The cancer risks for all residential monitors exceeded the values for the two background monitors for both the median and 95% UCL exposure cases when looking at VOCs only. Because six of the eight residential monitors did not analyze metals or VOCs, it is difficult to compare the results for all COPCs to the background monitors where metals and SVOCs make a significant contribution to the risk estimates when looking at all COPCs. For the residential monitors, looking at VOCs only, the highest cancer impact was predicted for Site 3 for the median exposure case and Site 7 for the 95% UCL exposure case. The risk estimates for Site 7 were based on the maximum detected concentration in air for several of the risk drivers, and may overestimate risks as a result.

Once again, it may be misleading to compare the cancer risk estimates for all COPCs at the residential monitors because six of the eight monitors were analyzed for VOCs only. There were a total of 15 COPCs that exceeded a cancer risk of  $1 \times 10^{-6}$  for at least one monitor location for the median exposure case. For the 95% UCL exposure case, there were a total of 17 COPCs that exceeded a cancer risk of  $1 \times 10^{-6}$  for at least one monitor location. Tables 5-1 through 5-4 show the cancer risk estimates and percent contribution to the overall risk at a monitor for the COPCs that exceeded a cancer risk of exceeded a cancer risk of  $1 \times 10^{-6}$ . Figures 5-1 through 5-4 present a graphical display of the total cancer risk estimates for each of the monitoring locations in the WLATS network.

For the non-cancer health assessment, an HI of 1 was exceeded at three of 12 monitors for the median exposure case (i.e., Sites 2a, 2b and 3), and at all but the two background monitors for the 95% UCL exposure case. The maximum impact for the non-cancer health impacts occurred at Site 2b for all cases. The highest residential impacts occurred at Site 3 for the median exposure case and Site 7 for the 95% UCL exposure case. This analysis was based on the conservative assumption that all of the COPCs for a monitor location had the same critical effect. For the median exposure case, none of the HQs associated with an individual COPC at a monitor exceeded a value of 1. Furthermore, when HIs were calculated on the basis of similar critical effects, none of these exceeded a value of 1. This would indicate that under the median exposure case, adverse health impacts would not be likely. For the 95% UCL exposure case, only the HQ for 1,3-butadiene exceeded a value of 1, which occurred at five monitors (i.e., Sites 2a, 2b, 3, 7 and 8). This indicates that there is a potential for an adverse health impact associated with exposure to 1,3-butadiene at these monitor sites, based on the 95% UCL exposure case.

## 7.2 Acute Risk Characterization

The acute risk characterization indicates that none of the COPCs exceed their respective acute toxicity criteria. This result indicates that acute health impacts are not likely to occur in the area. However, this conclusion reflects considerable uncertainty as acute toxicity criteria were not available for all of the chemicals detected in the monitoring network. Furthermore, the risk characterization was based on the maximum detected air concentration over a 24-period, as measured every 12 days. If the actual maximum occurred on a day when monitoring was not conducted, the true maximum may in fact be underestimated.

### 8.0 REFERENCES

- AIHA (2001), 2001 AIHA ERPG/WEEL Handbook, American Industrial Hygiene Association. Fairfax, VA.
- ATSDR (2002), *Minimum Risk Levels*, URL: http://www.atsdr.cdc.gov/mrls.html., Agency for Toxic Substances and Disease Registry, Atlanta, GA.
- CalEPA (2002), Acute and Chronic RELs and Supporting Documentation URL: http://www.oehha.org/air/hot\_spots/index.html., California Environmental Protection Agency.
- Gilbert (1987), *Statistical Methods for Environmental Pollution Monitoring*, John Wiley & Sons, New York, NY.
- ten Berge, W.F., Zwart. A, and Appelman, L.M. (1986), *Concentration-Time Mortality Response Relationship of Irritant and Systemically Acting Vapours and Gases*, Journal of Hazardous Materials 13:301-309.
- NRC (1985), *Emergency and Continuous Exposure Guidance Levels for Selected Airborne Contaminants,* Vol. 5. Washington, DC: National Academy Press, Committee on Toxicology, Board on Toxicology and Environmental Health Hazards, Commission on Life Sciences, National Research Council.
- NRC (1993), Guidelines for Developing Community Emergency Exposure Levels for Hazardous Substances, Washington, DC: National Academy Press, Subcommittee on Guidelines for Developing Community Emergency Exposure Levels (CEELs) for Hazardous Substances, Committee on Toxicology, Board on Environmental Studies and Toxicology, Commission on Life Sciences, National Research Council.
- USEPA (1989), *Risk Assessment Guidance for Superfund, Volume I, Human Health Evaluation Manual (Part A)*, Washington, DC: Office of Emergency and Remedial Response (EPA/540/ 1–89/002).
- USEPA (1991), Risk Assessment Guidance for Superfund, Volume 1: Human Health Evaluation Manual, Supplemental Guidance, Standard Default Exposure Factors, Interim Final, Office of Emergency and Remedial Response (OSWER Directive 9285.6–03), March 25.
- USEPA (1992a), Supplemental Guidance to RAGS: Calculating the Concentration Term, Office of Solid Waste and Emergency Response (OSWER Directive 9285.7–08I), May.
- USEPA (1992b), "Guidelines for Exposure Assessment," Federal Register 57(104) 22888–22938, May 29.
- USEPA (1997a), *Exposure Factors Handbook, Volume I, General Factors*, Office of Research and Development (EPA/600/P–95/002Fa), August.
- USEPA (1997b), *The Lognormal Distribution in Environmental Applications*, Office of Solid Waste and Emergency Response (EPA/600/R–97/006), December.
- USEPA (1997c), *Health Effects Assessment Summary Tables: FY 1997 Annual*, Office of Research and Development.
- USEPA (1997d), National Advisory Committee for Acute Exposure Guideline Levels for Hazardous Substances, Notice, *Federal Register*, October 30, pp. 58839–58851.

- USEPA (1998a), Risk Assessment Guidance for Superfund: Volume I Human Health Evaluation Manual, Part D, Standardized Planning, Reporting, and Review of Superfund Risk Assessments, Interim, Office of Solid Waste and Emergency Response (OSWER Directive 9285.7–01D), 1998.
- USEPA (1999a), Ranking and Selection of Hazardous Air Pollutants for Listing Under Section 112(k) of the Clean Air Act Amendments of 1990. Technical Support Document, Office of Air Quality Planning and Standards, July 28,1999.
- USEPA (2001), National-Scale Air Toxics Assessment for 1996 -(Draft for USEPA Science Advisory Board Review), Office of Air Quality Planning and Standards, Research Triangle Park, N.C. USEPA-453/R-01-003.
- USEPA (2002), Integrated Risk Information Systems accessed at <u>http://www.epa.gov/ngispgm3/iris/index.html</u>.
- USEPA (2003), Draft Final Guidelines for Carcinogen Risk Assessment (External Review Draft, February 2003), Risk Assessment Forum, Washington, DC.
- USEPA Region IV (2000a), *The West Louisville Air Toxics Project, Louisville, Jefferson County, Kentucky, Air Monitoring Quality Assurance Project Plan,* (with the Jefferson County Air Pollution Control Bureau) July 19.

## GLOSSARY

AEGLs ATSDR BW CARD CAS COPCs CPS <sub>o</sub> DEV	Acute Exposure Guidance Levels Agency for Toxic Substances and Disease Registry Body weight Cardiovascular effects Chemical Abstract Service Chemicals of potential concern Oral cancer potency slope Developmental effects
ERPGs	Emergency Response Planning Guidelines
HCl	Hydrochloric acid
HEAST	Health Effects Assessment Summary Table
HEM	Hematological effect
HEP	Hepatic effect
HI	Hazard Index
HQ	Hazard Quotient
IARC	International Agency for Research on Cancer
IR	Inhalation rate
IMM	Immunological effect
MLAPCD	Metro Louisville Air Pollution Control Board
MRLs	Minimum Risk Levels
NATA	National Air Toxics Assessment
NEUR	Neurological effect
PCBs	Polychlorinated biphenyls
QA	Quality assurance
QC	Quality control
RELs	Reference Exposure Levels
RfC	Reference concentration
RfD <sub>o</sub>	Oral reference dose
RME	Reasonable Maximum Exposure
RPR	Reproductive effect
RSP	Respiratory effect
SCAPA	Subcommittee on Consequence Assessment and Protective
	Action
SKIN	Skin effect
SQL	Sample Quantitation Limit
SVOCs	Semi-volatile organic compounds
TICs	Tentatively identified compounds
UCL	Upper Confidence Limit
URE	Unit risk estimate
VOCs	Volatile organic compounds
WLATS	West Louisville Air Toxics Study
WJCCTF	West Jefferson County Community Task Force
WOE	Weight of Evidence