Chattanooga Air Toxics Study

Final Risk Assessment Report

Prepared for: *U.S. EPA Region 4*

by: *Cambridge Environmental Inc.*

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Contents

Glossary of Terms

ACGIH	American Council of Governmental Industrial Hygienists
acute health effect	An adverse effect on a person's health that occurs from short-term exposure to a chemical. The period of short-term exposure can range from minutes to days; in the CATS, a 24-hour period is examined.
AEGL	Acute Exposure Guideline Level, a toxicity standard developed by the U.S. Environmental Protection Agency
AIHA	American Industrial Hygiene Association
air toxics	chemicals in air that are potentially toxic to human health, including numerous hazardous air pollutants (HAPs) designated within the Clean Air Act.
average concentration	the concentration that is calculated by adding together all of the values and dividing this sum by the number of measurements
average daily dose ADD	the average level at which a person is exposed to a chemical over the period in which exposure occurs
ATSDR	Agency for Toxic Substances and Disease Registry
Cal. EPA	California Environmental Protection Agency
cancer potency slope factor CPS	a numerical estimate of the rate at which a chemical might cause cancer at a given level of exposure
cancer risk (incremental)	the additional risk of getting cancer from exposure to a chemical in air
CATS	Chattanooga Air Toxics Study
CEEL	Community Emergency Exposure Level, a toxicity standard developed by the National Research Council
CHCAPCB	Chattanooga-Hamilton County Air Pollution Control Bureau
COPC	chemical of potential concern
chronic health effect	an adverse effect on a person's health that occurs from repeated exposure to a chemical over a lengthy period of time (usually years)
CSF	cancer slope factor

CT	Central Tendency
detection limit	the lowest concentration of a chemical that can be distinguished from the normal “noise” of an analytical instrument or method. Non-detects refers to chemicals that are not detected in a particular sample above a certain limit, usually the quantitation limit for the chemical in that sample.
DOE	Department of Energy
dose	a measure of exposure to a chemical that is calculated as the rate of a person’s chemical intake divided by the individual’s body weight
dose-response assessment	the process of quantitatively evaluating toxicity information and characterizing the relationship between the dose of a contaminant administered or received and the incidence of adverse health effects in the exposed population.
EPC (exposure point concentration)	concentration of a chemical in air that could be contacted by a person, derived from measurements in the CATS
ERPG	Emergency Response Planning Guideline, a toxicity standard published by the American Industrial Hygiene Association
exposure assessment	the evaluation of the magnitude, frequency, duration, and route of exposure.
hazard quotient (HQ) hazard index (HI)	estimate of non-cancer health risk, calculated as a person’s level of exposure to a chemical divided by a safe level of exposure; a hazard index is two or more hazard quotients of different chemicals added together
HEAST	Health Effects Assessment Summary Tables, a U.S. EPA chemical toxicity database
HHRA	Human Health Risk Assessment
IDLH	Immediately Dangerous to Life and Health
IRIS	Integrated Risk Information System, a U.S. EPA chemical toxicity database
IUR	Inhalation Unit Risk (for cancer assessments)
lifetime average daily dose LADD	the average level at which a person is exposed to a chemical over his/her lifetime, including periods in which exposure both occurs and does not occur
LOAEL	Lowest Observed Adverse Effects Level

lognormal distribution	a statistical pattern followed by many environmental datasets in which most values are contained in a limited range of values, and a few values are outside and usually greater than the core range
MRL	Minimum Risk Level, a non-cancer toxicity standard for a chemical developed by the Agency for Toxic Substance and Disease Registry (ATSDR)
measurement	the sample analytical results of a chemical in air, including instances in which the chemical was found, in which it was not found, and in which analytical problems or other factors invalidated the attempted measurement
monitor monitoring location	one of the six places in the Chattanooga area at which measurements of various air pollutants were taken in the CATS
NAAQS	National Ambient Air Quality Standards
NAS EEGLs	National Academy of Science Emergency Exposure Guidance Levels
NIOSH	National Institute of Occupational Safety and Health
NOAEL	No Observed Adverse Effects Level
normal distribution	a symmetric bell-shaped statistical pattern that characterizes some datasets, in which most values are found in a limited range, and a few values are both higher and lower than the core range
NRC	National Research Council
OAQPS	the United States Environmental Protection Agency's Office of Air Quality Planning and Standards
OEHHA	California Environmental Protection Agency's Office of Environmental Health Hazard Assessment
OSHA	Occupational Safety and Health Administration
PELs	Permissible Exposure Limits
PUF/XAD	a high air volume sampler consisting of a glass fiber filter with a polyurethane foam (PUF) and XAD (a proprietary resin) backup absorbent cartridge
Quality Assurance Project Plan QAPP	the checking procedures that are used to make sure that risk assessment calculations and database management are performed completely and correctly

reference concentration or dose RfC or RfD_i	a concentration or dose that represents a safe level of exposure, such that exposure will not cause adverse noncancer effects on health
risk assessment	a quantitative and qualitative assessment used to assess whether chemicals present in air present a risk of harm to people's health
REL	Reference Exposure Level, a toxicity standard developed by the California Environmental Protection Agency
Risk Assessment Workplan RAWP	the document which describes the procedures that were used in the CATS to develop this risk assessment
RME	Reasonable Maximum Exposure
SPEGL	Short-term Public Emergency Guidance Level, a toxicity standard developed by the National Research Council
standard deviation	a mathematical measure of the variability among a group of measurements
STELs	Short-Term Exposure Limits
SVOCs	Semi-Volatile Organic Compounds
TEEL	Temporary Emergency Exposure Level, a toxicity standard developed by the Department of Energy
TICs	Tentatively Identified Compounds
TLV-C	Threshold Limit Value - Ceiling
TLV-TWA	Threshold Limit Value - Time Weighted Average
TRI	Toxics Release Inventory
uncertainty analysis	the process of evaluating the quality and reliability of risk estimates
unit risk factor	a numerical estimate of the rate at which a chemical might cause cancer at a given concentration in air
upper confidence limit UCL 95% UCL	an estimate of the average concentration of a group of measurements that is biased high to account for uncertainty. The 95^{th} percentile upper confidence limit of the mean (95% UCL) is the value that, with 95% probability, is the highest that the true average concentration of the data could possibly be.
U.S. EPA	United States Environmental Protection Agency
VOC	Volatile Organic Compounds

Chapter 1 Executive Summary

The United States Region 4 Environmental Protection Agency (U.S. EPA) and the Chattanooga-Hamilton County Air Pollution Control Bureau (CHCAPCB) are jointly conducting an air toxics study of the Chattanooga Area in response to concerns from residents about exposure to toxic air pollutants associated with nearby industrial facilities. The primary objective of the Chattanooga Air Toxics Study (CATS) is to determine if residents of the Chattanooga area are being exposed to airborne concentrations of air pollutants that may pose unacceptable risks to health. The overall goal of the risk assessment was to provide information on risk that stakeholders can utilize in sound decision-making.

This risk assessment provides quantitative and qualitative estimates of long-term risk posed to human health through exposure to target air pollutants. A further objective of this phase was to evaluate risks to human health from acute exposure to airborne contaminants.

The risk assessment focused on direct inhalation of contaminants measured in outdoor air at six monitoring locations in Chattanooga between November 12, 1998 and October 29, 1999. Approximately 30 composite samples were collected over a 24-hour period approximately every 12 days from four different types of monitors at each of six locations. Samples were analyzed for volatile organic compounds (VOCs), semivolatile organic compounds (SVOCs), formaldehyde, and metals. Air sampling results were used to estimate risks of chronic health effects (both cancer and non-cancer) and acute non-cancer health effects due to inhalation of ambient air by residents. Chronic health effects may occur after long term exposure to relatively low levels of pollution. Acute health effects may occur after short term exposure to relatively high levels of pollution.

In the chronic non-cancer health risk assessment, the hazard quotients for three individual chemicals were at or above a value of one at one or more locations based on reasonable maximum exposure (RME) parameters (manganese, formaldehyde, and cobalt). A hazard quotient greater than one indicates that exposure is higher than a presumed "safe concentration" and that adverse non-cancer health effects may occur. Total hazard indices (HIs), based on RME parameters, and calculated by summing the hazard quotients for individual chemicals at each sampling location, ranged from a value of 2 to a value of 3 for an adult and from a value of 4 to a value of 8 for a child resident.

In the assessment of incremental lifetime risks of cancer, estimated risks of cancer for the RME scenario were at or above a level of one excess case of cancer in one million people exposed (1×10^{-6}) for a number of individual chemicals. Formaldehyde, chromium, chloroform, benzene,

carbon tetrachloride, arsenic, chloromethane, and tetrachloroethylene each posed an incremental cancer risk of greater than 1×10^{-6} at each of the six monitoring locations. Further, benzo(a)pyrene, bromodichloromethane, 1,4-dichlorobenzene, trichloroethylene, benzo(b)fluoranthene, nickel, 1,1,2-trichloroethane, and hexachloro-1,3-butadiene each posed an incremental risk of cancer greater than 1×10^{-6} at one or more of the locations. Total RME incremental risks of cancer summed over all chemicals at the six sampling locations ranged from 7×10^{-5} to 1×10^{-4} (for a 30 year exposure).

Hazard quotients at or above a value of 1 and incremental cancer risks at or above a value of 1×10^{-6} at each monitoring location are summarized in Table 1.1.

To evaluate potential impacts of short-term acute exposure to airborne contaminants, sample data collected from the six monitoring locations were compared to selected acute screening level toxicity data. Of all the detected constituents at all sampling locations, no contaminant concentration exceeded an acute screening level criterion.

The risk assessment is organized into seven chapters. Chapter 1 (this chapter) provides an executive summary and introduction to the analysis. Chapter 2, Background information, presents an overview of the study area. Chapter 3, Data evaluation, discusses data collection, data analysis, data quality assurance, and data management. Chapter 4, Human health risk assessment, presents the results of the quantitative human health risk assessment. Chapter 5, Human health acute effects analysis, presents the results of the screening-level evaluation of potential acute health effects from short-term exposure to airborne contaminants. Chapter 6 presents the conclusions of the report. Chapter 7 lists references used in the risk assessment.

Chapter 2 Background information

2.1 Introduction and purpose

Chattanooga is a city with a population of about 156,000 people and is located in Hamilton county in southeastern Tennessee. It is located adjacent to the Tennessee River and near the Tennessee/Georgia state line. It is bounded on the western side by a ridge that includes Lookout Mountain. Chattanooga currently has one of the largest numbers of industries of any city in Tennessee.

Within the last few years several citizens groups have expressed concerns that airborne pollutants may be causing increased instances of cancer and other illnesses in the area. For example, the citizens groups Stop Toxic Pollution and the Community Advisory Group have raised concerns about air toxics associated with the Alton Park/Piney Woods industrial complex.

According to the 1999 Toxics Release Inventory (TRI) reports for industrial facilities located in Hamilton County, approximately 2.4 million pounds of chemicals were released into the air by stack or fugitive emissions. In addition to TRI sources, a number of other sources contribute to air pollution in the Chattanooga area, including cars and trucks, natural sources, small businesses, heating (*e.g.*, woodsmoke), and dust.

In response to concerns about the potential for adverse health effects from air pollution in the Chattanooga area, the United States Region 4 Environmental Protection Agency (U.S. EPA) and the Chattanooga-Hamilton County Air Pollution Control Bureau (CHCAPCB) jointly conducted an air toxics monitoring study of the Chattanooga area between November 12, 1998, and October 29, 1999. The data were assessed to determine if residents of the Chattanooga area are being exposed to airborne concentrations of air pollutants that may pose unacceptable risks to health. This document presents an assessment of the potential risks posed by the chemicals found in that monitoring study.

2.2 Study area description

Six sites were identified as appropriate ambient air toxics monitoring locations for this study. The six sites were at the Emma Wheeler Homes, the Bethlehem Community Center, the 20th Street Fire Station, the Cellular One Tower site, the East Brainerd Fire Station, and the River Park site. These sites, and the rationale for including each of these sites in the study, are summarized in Table 2.1. A duplicate set of monitors were deployed at the Bethlehem Community Center for a total of seven sets of monitoring equipment at six sites. A full discussion of the monitoring study is provided in U.S. EPA Region 4 (1999). The monitoring locations are also depicted in Figure 2.1, a map of the Chattanooga area.

While this study attempts to assess air quality indicators across the Chattanooga area, several of the monitoring locations are concentrated in the Alton Park/Piney Woods neighborhoods in south Chattanooga. According to 2000 census data, 99% of the residents of South Chattanooga are African American and more than 65% of the residents live below poverty level. The Alton Park/Piney Woods neighborhoods in South Chattanooga include approximately 6,500 residents, is heavily concentrated with industry, and is located in close proximity to the Chattanooga Creek Superfund site (U.S. EPA Region 4, 2001a).

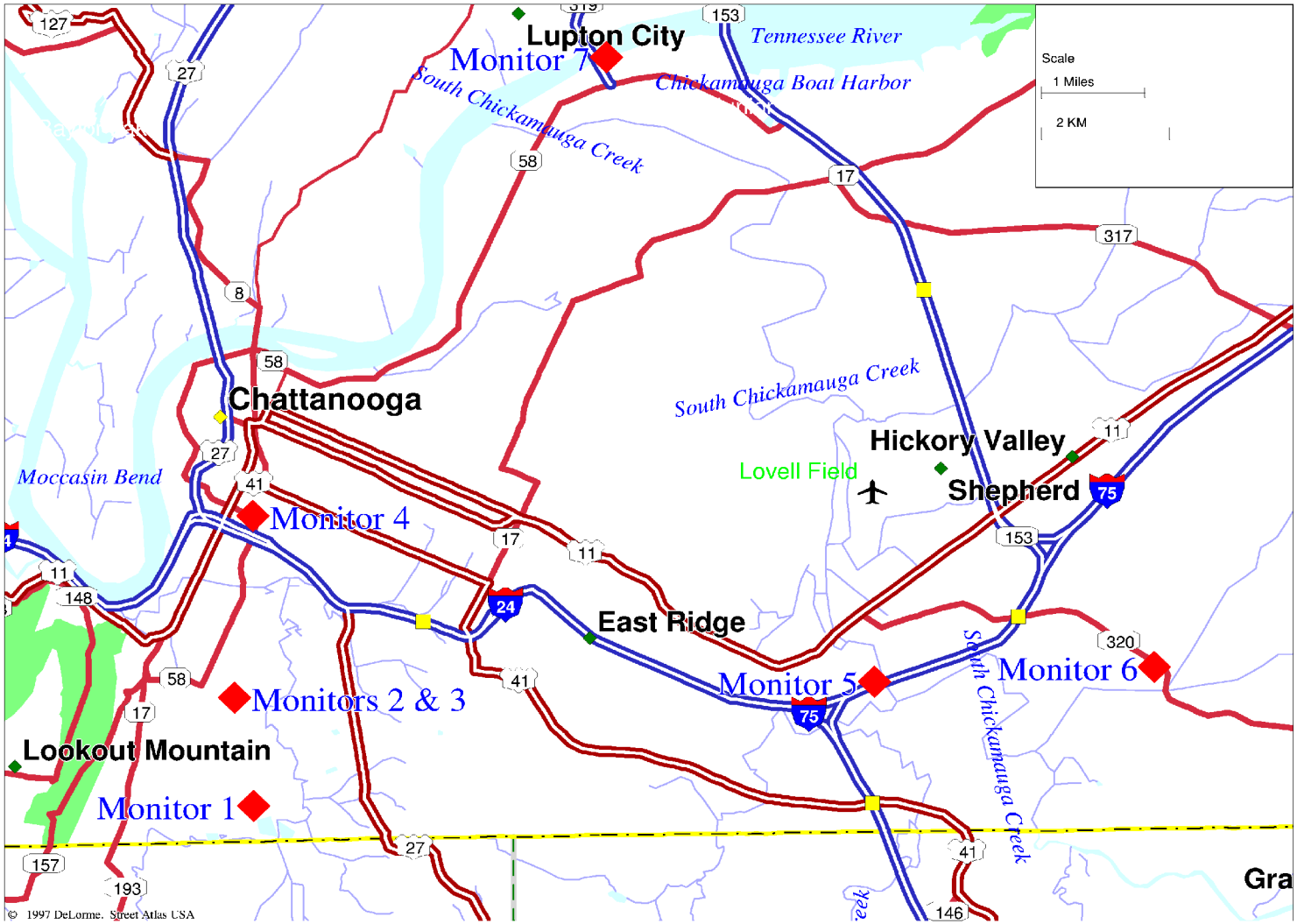


Figure 2.1 CATS monitoring locations (indicated by red diamonds)

Chapter 3 Data collection

This section discusses the data collection and data management aspects of the CATS air risk assessment.

3.1 Data collection

Data included in the risk assessment were collected by EPA Region 4 and the CHCAPCB. The monitoring study carried out the plans set forth in the Chattanooga Air Toxics Quality Assurance Project Plan (U.S. EPA Region 4, 1999). Air samples were analyzed by the EPA Region 4 Science and Ecosystem Support Division in Athens, Georgia for VOCs, SVOCs, and metals. Formaldehyde samples were analyzed by Eastern Research Group of Research Triangle Park, North Carolina. Laboratory data were summarized in a database and provided to Cambridge Environmental Inc. for use in the risk assessment. All data were validated as specified in the CATS Quality Assurance Project Plan (U.S. EPA Region 4, 1999). U.S. EPA Region 4 maintains the original database on which the risk assessment is based. A summary of the data collected at each of the six monitoring locations is provided in Tables 4.1 through 4.6.

The six sampling locations, as discussed in Chapter 2, were the Emma Wheeler Homes, the Bethlehem Community Center, the 20th Street Fire Station, the Cellular One Tower site, the East Brainerd Fire Station, and the River Park site. Where possible, air monitors were located 2 meters above ground level, a height selected to be representative of human exposure. Air samples were collected over a 24-hour period. Chemical concentrations, therefore, represent daily averages. Samples at each location were taken approximately every 12 days. All samples (with the exception of those removed based on quality assurance problems - see below) were used in both the chronic risk analysis and the acute risk analysis.

VOCs were collected in six-liter Silcosteel canisters using flow controllers to allow the pre-evacuated canister to fill slowly over a 24-hour period. The sampling was initiated by an electronic timer that opened a solenoid valve to allow air to flow into the canister. At the end of the 24-hour sampling interval, the timer closed the solenoid valve, sealing the cylinder. The flowrate was adjusted to allow approximately 5100 cc[1] of air to be collected in the 6000 cc canister during the 24-hour period. The sampling conformed to method TO-15 of the EPA Compendium of Methods for the Determination of Toxic Organic Compounds in Ambient Air (U.S. EPA, 1999c).

[1] cc indicates cubic centimeters (cm^3).

SVOCs were collected by the high volume PUF/XAD method. A high volume PUF/XAD sampler consisting of a glass fiber filter with a polyurethane foam (PUF) and XAD (a proprietary resin) backup absorbent cartridge were used. Approximately 300 m^3 of air were sampled during the 24-hour period. The sampling conformed to TO-13A of the EPA Compendium of Methods for the Determination of Toxic Organic Compounds in Ambient Air (U.S. EPA, 1999c).

Formaldehyde was collected on dinitro-phenylhydrazine saturated silica-gel Sep-Paks (DNPH cartridges). Approximately 1440 liters of air were sampled through the DNPH cartridges. The sampling conformed to Method TO-11A of the EPA Compendium of Methods for the Determination of Toxic Organic Compounds in Ambient Air (U.S. EPA, 1999c).

Suspended particulate (for the measurement of metals) was collected by the High Volume Particulate Method. The sampling conformed to the methods recommended in Volume 40 of the Code of Federal Regulations, Part 50, Appendix G.

3.2 National Ambient Air Quality Standards

The Clean air Act of 1970 mandated that the U.S. EPA establish air quality standards for pollutants that may harm the public health and welfare. The agency currently has set National Ambient Air Quality Standards (NAAQS) for six major pollutants, called criteria pollutants. One analyte detected in the monitoring program for this study, lead, is regulated under the NAAQS. The primary health standard for lead is a maximum quarterly average concentration of 1.5 $\mu g/m^3$.

The NAAQS for lead was not exceeded by any sample collected in the CATS air monitoring study. The maximum concentration of lead detected in any 24-hour sample was 0.062 $\mu g/m^3$, a factor of 24 less than the *quarterly* NAAQS (1.5 $\mu g/m^3$) [2]. Given this low concentration of lead in air, this chemical is not evaluated further in this risk assessment.

[2] Chattanooga is currently in compliance with the lead NAAQS.

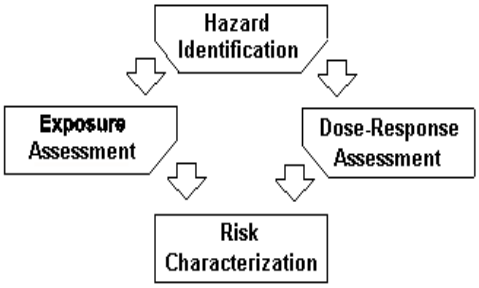
Chapter 4 Human health risk assessment

4.1 Introduction

Risk assessment is a tool that can be used to evaluate the likelihood that pollution will cause adverse effects on human and environmental health. For example, the CATS focuses on risks resulting from the inhalation of airborne chemicals by residents. A risk assessment combines information about the toxic potential of a pollutant, with the results of studies that evaluate the level of exposure people have to the pollutant, to derive quantitative and qualitative statements about the potential for the estimated exposure to result in adverse public health outcomes.

While the outcome of risk assessments only provide estimates of risk, they do help scientists evaluate the likelihood that environmental contaminants may cause harm to nearby populations. Using risk estimates and other information, stakeholders can make more informed decisions about the need to reduce exposures to toxic pollutants and thereby reduce the risk of possible health problems.

A risk assessment consists of four interrelated steps, as shown in the following diagram.



The general framework of each of these basic steps of risk assessment is described briefly below. The CATS risk assessment treats each of these steps in differing levels of detail. For example, the hazard identification step is not directly evaluated within the CATS, but rather has been developed from the collective knowledge of risk assessment science. The dose-response step also draws on methods and information developed outside of the CATS. As such, the CATS risk assessment focuses on the exposure assessment and risk characterization steps as they apply to the specific air toxics data collected in the Chattanooga area and simply draws on already developed information for the hazard identification and dose-response steps.

- ***Hazard Identification***

  Hazard identification identifies chemicals in the environment that might be hazardous to people's health. The process of hazard identification involves determining whether exposure to a specific chemical can cause an increase in the incidence of a particular adverse health effect, such as cancer or neurological disorders, and whether the adverse health effect is likely to occur in humans, or only in laboratory animals. This step also evaluates the nature and strength of the evidence that a chemical causes an adverse effect. The hazard identification stage of risk assessment is to some extent a cumulative process, since it in part depends on the body of information that scientists have developed over the years concerning environmental hazards associated with various chemicals. This collective knowledge has helped to develop the basic lists of chemicals considered in environmental investigations.

- ***Dose-Response Assessment***

  Once a chemical is determined to have potentially harmful effects, an evaluation is made of how different levels of exposure to the chemical result in different levels of response. In both the hazard identification and dose-response evaluations, information on a chemical's effects in humans (when available) and data from studies in laboratory animals are used to derive a mathematical relationship that relates levels of exposure to potential effects on health.

- ***Exposure Assessment***

  An exposure assessment is performed to evaluate who potentially contacts chemicals in the environment, how chemicals enter their bodies (for example, by inhalation), how often and how long they are exposed, and how much of a pollutant they are exposed to. Special attention is often given to sensitive groups such as children and the elderly. Estimates of exposure can be based on actual data (for example, from monitoring levels of chemicals in the air, as in the CATS) or from estimates developed using mathematical models and scientific judgement.

- ***Risk Characterization***

  In the final step of the risk assessment, the results of the dose-response assessment are combined with the results of the exposure assessment to provide estimates of the likelihood that adverse health effects may occur in a population (*e.g.*, residents in the CATS study area). An evaluation of the uncertainty in the overall risk estimate is also presented in this step to help interpret the meaning and robustness of risk estimates.

In this CATS risk assessment, the risk assessment process outlined above has been refined to consist of five major components:

- Data Evaluation (Section 4.2) — Discusses data usability, data quality, and selection of contaminants of potential concern (COPC).
- Exposure Assessment (Section 4.3) — Discusses potentially exposed populations and exposure pathways through which people may come in contact with contaminants at each monitoring location. Equations and exposure input parameters used to estimate chemical intakes are also provided in this section.
- Toxicity Assessment (Section 4.4) — Presents the chemical-specific dose-response data for use in quantifying potential human risks.
- Risk Characterization (Section 4.5) — Provides the calculated noncarcinogenic and carcinogenic risks for each exposed person. Total risks are also summarized by location.
- Uncertainty Analysis (Section 4.6) — Presents a discussion of uncertainties and factors that affect the reliability of risk estimates.

Chapter 4 focuses on chronic exposure (*i.e.*, long term exposure to relatively low levels of pollutants). A discussion of the risk assessment process for acute exposures (*i.e.*, short term exposures to relatively high levels of pollutants) is provided in Chapter 5.

The majority of the tables included in this section are analogous to the standard tables recommended by recent EPA guidance (U.S. EPA, 1998). These tables include the majority of data fields specified in the guidance, which also defines the structure of this risk assessment report.

4.1.1 Methodology

To assess potential public health risks, three major aspects of chemical contamination and exposure must be considered: 1) The presence of chemicals with toxic characteristics; 2) The existence of pathways through which people may contact such chemicals; and 3) The actual presence of an exposed population. The absence of any of these three aspects would result in an incomplete exposure pathway and nullify the calculation of risk.

An overview of the media, pathways and potentially exposed individuals evaluated in the CATS risk assessment is provided in Table 4.7. Subsequent sections provide justification and additional detail for each data element presented in this table.

The HHRA was performed using guidelines specified in the following documents:

- *Risk Assessment Guidance for Superfun*d, Volumes I, Part A (U.S. EPA, 1989);
- Office of Solid Waste and Emergency Response Directive 9355.7-04 Memorandum (U.S. EPA, 1995a);

- *Standard Default Exposure Factors* (U.S. EPA, 1991);
- *Exposure Factors Handbook* (U.S. EPA, 1997a); and
- *Supplemental Guidance to RAGS: Calculating the Concentration Term* (U.S. EPA, 1992b).

In addition, a project specific workplan was developed in support of this risk assessment. The *Chattanooga Air Toxics Study; Risk Assessment Workplan and Quality Assurance Project Plan* (U.S. EPA Region 4, 2001b) was developed to provide guidance specifically for the CATS air risk assessment project.

4.2 Data evaluation

The goal of the data evaluation step of a human health risk assessment is to develop a list of COPCs for each environmental medium under consideration. This process involves determining what data are available, assessing whether the existing data are of suitable quality and quantity to be included in a risk assessment, and identifying all chemicals, using this verified data set, that were detected at least once at a monitoring location. Any chemical detected even once out of the approximately 30 sampling events at a monitoring location was considered a COPC at that monitoring location. However, chemicals detected at one monitoring location are not necessarily COPCs at all monitoring locations. Thus, the list of COPCs may differ at each of the monitoring locations.

4.2.1 Data usability

Data were validated and selected for inclusion in the risk assessment in accordance with the CATS Risk Assessment Workplan (U.S. EPA Region 4, 2001b). For one monitoring location, the Bethlehem Community Center, air samples were collected in duplicate. The results of the duplicate samples were compared and found to be similar. These results indicate that the methods used for the collection of air samples were precise and reflective of high quality, usable data. Since the duplicate samples at the Bethlehem Community Center represent a single location, the results were averaged for each sampling date to develop an estimate of exposure point concentration (EPC). The EPC is the concentration of a chemical in air that could be inhaled by a person. COPCs were chemicals detected in either duplicate sample. If a chemical was detected in one of the duplicate samples, but not the other, the detected concentration was averaged with one-half the detection limit in the second sample. If the chemical was detected in neither of the duplicate samples on a given sampling date, but was detected on other sampling dates, the chemical was assumed to be present at the average of one-half the detection limits.

4.2.2 Data summary

Summary tables for each monitoring location were prepared in accordance with U.S. EPA guidance (U.S. EPA,1989). Recommended procedures for data use adopted in the CATS risk assessment include:

1. Results qualified as rejected (R-qualified) or not analyzed (NA-qualified or NAI-qualified) were not included in the data summary. Data qualified as estimated (J-qualified), as an average of several analytical results (A-qualified), or as tentatively identified compounds or TICs (N-qualified) were used in the risk assessment at reported values (with no modification to the presented sample result).

2. For all analyses that include non-detected results (U-qualified and UJ-qualified), a value of one-half the sample specific quantitation limit was used as a surrogate value.

3. No information on laboratory duplicates was available. Therefore, laboratory duplicate results were not considered in this risk assessment. Duplicates were collected at the Bethlehem Community Center only. As described in section 4.2.1, data from the duplicate samplers were averaged for each sampling date to determine the concentration of each data point before the calculation of the EPCs.

A full discussion of data used in this risk assessment, including data rejected based on quality assurance concerns is provided in Appendix B.

Treatment of qualified data in the risk assessment

Footnote or data qualifier	Explanation	Use data in risk assessment?
NA	Not analyzed	No
NAI	Not analyzed due to interferences	
J	Reported value is estimated; identification of the compound is definitive, but the reported concentration is uncertain	Yes
U	Compound not detected, reported value is the analytical detection limit	Yes, one-half the reported value to be used in averaging in cases where other samples report detected values
UJ	Combination of U and J qualifiers, indicating the compound was not detected, and that the analytical detection limit was estimated	
A	Average of several analytical results	Yes
AJ	Similar to the A qualifier, but with additional uncertainty concerning the reported concentration	
N	Tentatively identified compound	Yes, but considered separately in risk calculations because of uncertainty about the chemical and concentration
JN	Equivalent in meaning to the N qualifier	
K	Actual value is known to be less than value given	Yes
L	Actual value is known to be greater than value given	
R	Rejected	No

4.2.3 Selection of Chemicals of Potential Concern

As noted previously, a simple process was used for identifying COPCs in the CATS: any chemical detected (even once) at a monitoring location becomes a COPC at that monitoring location. Identification of a COPC at one or more monitoring locations, however, does not make it a COPC at all monitoring locations. Thus, the list of COPCs differs at each of the monitoring locations.

Two inorganic analytes, iron and magnesium, are considered to be essential nutrients when ingested (U.S. EPA, 1995a; U.S. EPA, 1989), and are typically excluded from the quantitative analysis of risk for this pathway. Given the lack of inhalation reference doses for these chemicals, inhalation exposure for iron and magnesium was not evaluated in this risk assessment. The potential effect of omitting these chemicals from the quantitative risk analysis is discussed in the uncertainty section of the report.

TICs are also excluded from the quantitative analysis of risk in this chapter. TICs were generally detected in only a small number of samples, and toxicological data are frequently unavailable for these chemicals. Risk estimates due to TICs in ambient air are discussed quantitatively in section 4.6.6 of the risk assessment.

4.3 Exposure assessment

The purpose of the exposure assessment is to predict the magnitude and frequency of potential human exposure to each of the compounds included in the risk assessment. In accordance with U.S. EPA guidance (U.S. EPA, 1989) a person with reasonably high exposure (a reasonable maximum exposure or RME scenario) was evaluated in this risk assessment. The RME represents exposure to a highly exposed person (*i.e.*, above the 90^{th} percentile of exposures), but whose exposure is not higher than the most highly exposed person in the population. However, the RME is only one of many possible exposures in a population. In order to get some sense of the range of possible exposures, average exposures were also quantified in this assessment. Specifically, a central tendency (CT) evaluation was developed to provide an estimate of the risks associated with average exposure to COPCs at each monitoring location in question. In addition to assessing the distribution of possible exposures by evaluating the CT and RME exposures, other methods are available as well. This is described more fully in Section 4.6

4.3.1 Conceptual exposure model and potential exposure routes

This section discusses the conceptual exposure model for the CATS risk assessment. A conceptual model facilitates consistent and comprehensive evaluation of the potential risks to human health by creating a framework for identifying the pathways through which people may

come in contact with contaminated media resulting from the source area. It describes the relationships between the following elements, which are necessary for a complete exposure pathway to exist:

- Sources of contamination (i.e., contaminant release mechanisms)
- Contaminant transport pathways
- Exposure mechanisms and exposure routes
- Potentially exposed individuals

The conceptual exposure model for this risk assessment is provided in Figure 4.1. As shown in the model, hazardous air pollutants and their precursors are emitted to ambient air and transferred to adult or child-age residents. While the direct exposure pathway evaluated in this risk assessment is the inhalation pathway, other pathways for exposure to airborne contaminants potentially exist. An example includes the dermal contact pathway. As discussed in EPA guidance (U.S. EPA, 1989), dermal absorption of vapor phase chemicals is considered to be lower than inhalation intakes in most instances and generally is not considered in exposure assessments. However, airborne chemicals also pose a potential for human exposure though indirect pathways such as deposition to soil followed by the incidental ingestion of soils by people or uptake by plants and animals which are subsequently ingested by people. The resources to evaluate such indirect exposure pathways is beyond the scope of this analysis and was not evaluated in this assessment.

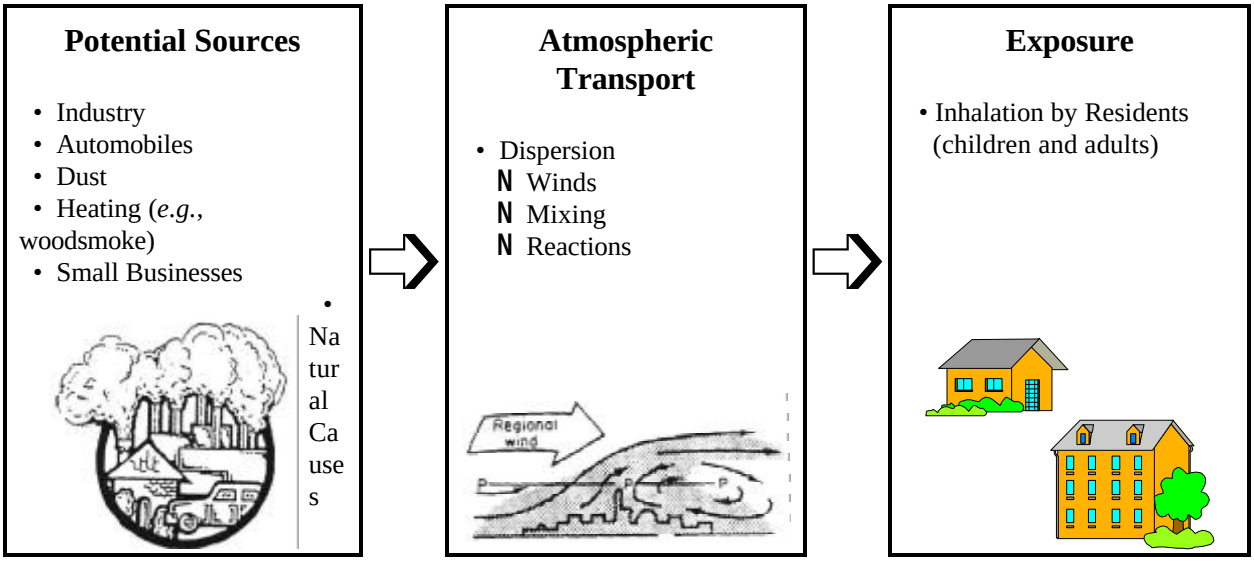


Figure 4.1 Conceptual exposure model

4.3.2 Potentially exposed individuals

Potentially exposed individuals evaluated in this study include both adults and children who currently live in general proximity to sampling locations and pollution sources. Residents were selected for evaluation as they represent the people who potentially receive the greatest amount of exposure to COPCs in non-occupational scenarios. Table 4.7 summarizes the potentially exposed individuals evaluated in this risk assessment.

4.3.3 Quantification of exposure

Estimates of exposure are based on EPCs of contaminants and on scenario-specific assumptions and intake parameters. EPCs are developed by analyzing and reducing the data identified as valid (as described above in Section 4.2). The models and equations used to quantify intakes are described in this section and have been obtained from a variety of U.S. EPA guidance documents, as cited in the sections that follow.

4.3.3.1 Data analysis and reduction

Data analysis and reduction were performed to:

- Reduce and summarize the data collected in the CATS monitoring study;
- Present the results of the monitoring study in an informative, understandable format; and
- Extract and generate the data needed for the risk assessment.

A large amount of data was collected in the CATS. The raw data are stored in electronic files, and each individual piece of data consists of a single measurement[3] of an individual chemical at a particular monitoring station. In addition, the database contains quality assurance and control data collected during the CATS. The three files that constitute the CATS database contain tens of thousands of measurements. Due to the complexity of the data, the files are not in a form that is easily accessible or comprehensible.

A computer program was written by Cambridge Environmental Inc. to analyze the raw data from the CATS database. The program was developed in the Delphi® programming environment, a Windows®-based application that implements the Pascal programming language. The data

[3] The term "measurement" refers to the sample analytical results of a chemical in air, and includes instances in which the chemical was found, in which it was not found, and in which analytical problems or other factors invalidated the attempted measurement.

analysis program was written in a manner to provide flexibility in pursuing alternative data analyses, and is adaptable to similar databases. EPA Region 4 is in possession of this program and the CATS analytical database.

The data analysis program sorts the measurements by both chemical and monitoring location. Statistical evaluations are developed separately for each monitoring location, since each location represents a different area of Chattanooga where people may contact air pollutants. For each combination of monitoring site and chemical, the statistical summary of measurements includes:

- Frequency of detection, which describes the number of valid measurements collected and the portion of those measurements in which the chemical was identified to be present;
- The range of concentrations detected, which consists of the highest and lowest concentrations at which the chemical was detected (including J-qualified data)[4];
- The range of detection limits; and
- An arithmetic average concentration, its standard deviation, and a 95th percentile upper confidence limit of the mean (calculated as a means of incorporating data uncertainty). Depending on the distribution of the underlying data, the 95th percentile upper confidence limit of the mean is the upper confidence limit on either a normal or a lognormal distribution.

Statistical analyses were conducted using standard methods developed for assessing sampling data (U.S. EPA, 1989). A description follows of the formulae that were used. Issues associated with data treatment (*e.g.*, non-detects, J-qualified data) were discussed in Section 4.2.2 above. The development of the data summaries proceeded according to the specific instructions provided in Appendix C of the *Chattanooga Air Toxics Study; Risk Assessment Workplan and Quality Assurance Project Plan* (U.S. EPA Region 4, 2001b).

4.3.3.2 Statistical methods

The arithmetic mean concentration was calculated in a standard manner as:

$$\bar{c} = \frac{\sum_{i=1}^{n} c_i}{n} \qquad \textbf{(4)}$$

[4] J-qualified concentrations are estimated concentrations less than the detection limit for the COPC.

where the terms are:

- $\bar{c}$ the arithmetic mean concentration;
- c_i an individual measurement of the concentration, designated by the subscript *i*; and
- n the total number of measurements.

The sample standard deviation *s* of the arithmetic mean concentration was calculated as:

$$s = \sqrt{\frac{\sum_{i=1}^{n} c_i - \bar{c}^{\,2}}{n-1}} \qquad \textbf{(4)}$$

where the terms are defined as above.

The U.S. EPA Office of Air Quality Planning and Standards (OAQPS) endorses the concept of using an upper confidence limit on the mean concentration for use in estimating exposure for air monitoring results (U.S. EPA Region 4, 2001c). Use of the upper confidence limit explicitly builds a statistical measure of uncertainty into the EPC. Some explanation of the rationale for using upper confidence limits is provided in the following paragraphs, followed by the equations and methods used in their calculation.

The arithmetic mean is constructed from discrete measurements taken over time. Constraints on resources, however, placed limits on the amount of sampling possible within the CATS (e.g., samples could not be collected every day). Instead, consistent with other air toxics studies, samples in the CATS were collected roughly one out of every twelve days. Statistically, the samples were collected in a manner to eliminate obvious sources of bias (*e.g.*, samples were not uniformly collected on the same day of the week, or only on weekdays or on weekends). In addition, collecting samples for a year allowed for potential evaluation of seasonal variability.

All factors being equal, one would expect the database to contain equal probabilities of sampling on days when pollutant concentrations may have been relatively high as on days when pollutant concentrations may have been relatively low. Since samples were not collected every single day, however, one cannot be absolutely certain that all possible conditions were sampled equally. The arithmetic mean concentration is thus subject to uncertainty due to a number of factors, including:

- Variability in concentrations;
- The ability to measure only a finite number of measurements from the distribution of concentrations;
- Potential inaccuracy in individual measurements of concentration; and
- The fact that the data are often censored (i.e., some of the measurements of concentration are non-detects, known to be between zero and an upper limit that corresponds to the quantitation limit of the analytical method used in the measurement).

Thus, use of the upper confidence limit is endorsed by OAQPS to explicitly account for the uncertainties introduced by discrete monitoring samples. The difference between the use of a straight mean value and its upper confidence limit can be described in terms of certainty and confidence. As the number of samples gets larger and larger, the difference between the mean and its upper confidence limit becomes smaller and smaller. Nevertheless, the mean calculated from averaging a finite number of samples from a distribution is only an estimate of the true mean. Selection of a second, different set of samples would likely result in a different, but no less valid, estimate of the true mean of the underlying distribution. If the straight mean is assumed for the exposure point concentration, chances are that the actual, true mean could be either lower or higher. Use of the 95th percentile upper confidence limit (95% UCL) of the mean, however, implies that there is a 95% likelihood that the true mean is lower, and that there is only a 5% chance that the true mean is higher. Therefore, use of the 95% UCL to represent the EPC is more conservative (in terms of overestimating, rather than underestimating potential exposure) than using a sample mean. As such, the use of the 95% UCL was adopted as the primary method for estimating the exposure point concentrations in this study. This is shown graphically for three hypothetical groups of samples at a monitor in Figure 4.2.[5]

[5]LCL = lower confidence limit.

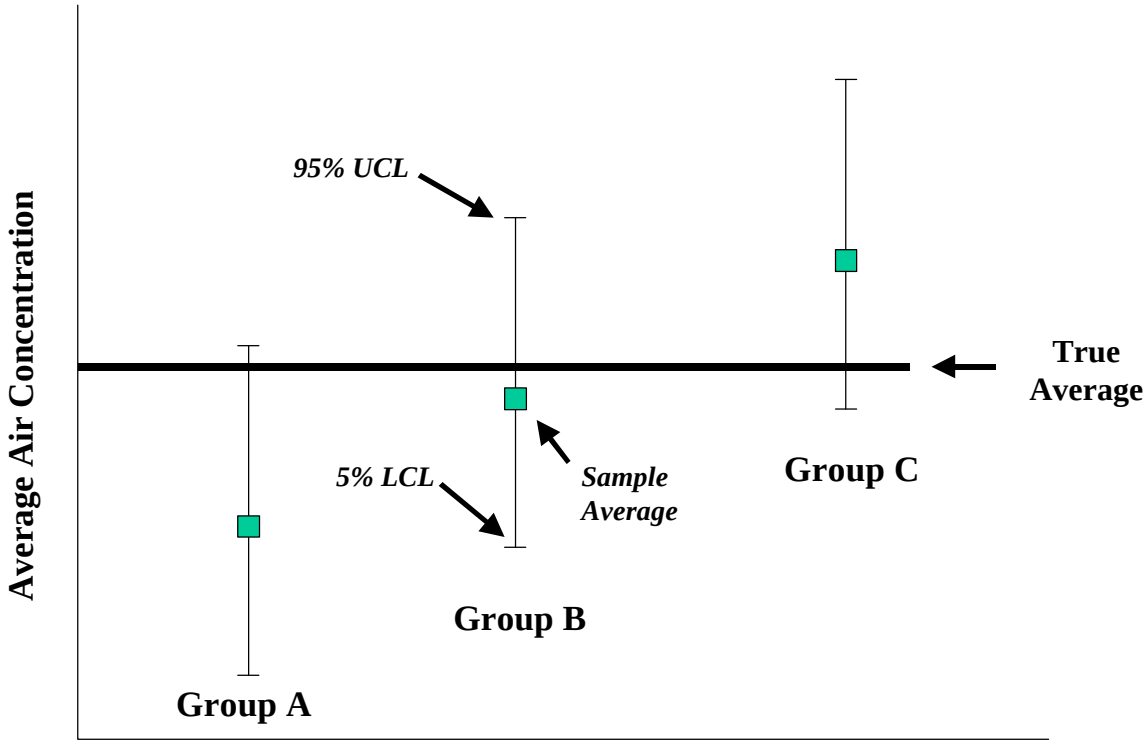


Figure 4.2 Use of the 95% UCL likely overestimates potential exposure

The method used to calculate the upper confidence limit depends upon the nature of the underlying data (U.S. EPA, 1992b). If the data are well-characterized by a normal distribution, the equation for the upper confidence limit is based on the statistical assumption of a normal distribution. If the data do not follow a normal distribution, the *de facto* assumption is made that the data are best represented by a log-normal distribution, and a different formula appropriate for a log-normal distribution is used to calculate the upper confidence limit. Default use of a log-normal distribution is consistent with the observation that many types of environmental data are log-normally distributed, including many air monitoring data sets (U.S. EPA, 1992b; U.S. EPA Region 4, 2000c).

Thus, in the CATS, the choice of statistical methods was made for each chemical based upon the distribution of the data. In calculating the upper confidence limit, the basic assumption was made that data were best characterized by a log-normal distribution unless an initial test of normality indicated that the data were normally distributed. The Shapiro-Wilks' test was used for this purpose. If the *p*-value for the Shapiro-Wilks' test was greater than 0.01, the null hypothesis that the data are normally distributed was accepted, and the equation that was used to calculate the 95^{th} percentile upper confidence limit of the mean concentration was (U.S. EPA, 1992b):

$$\bar{c}_{95} = \bar{c} + \frac{s t_{95}}{\sqrt{n}} \quad \textbf{(4)}$$

where the new terms are:

$\bar{c}_{95}$ the 95th percentile upper confidence limit of the arithmetic mean; and
t_{95} the one-sided student's *t* statistic (commonly tabulated) based on *n*–1 degrees of freedom.

Otherwise, the data were assumed to follow a log-normal distribution. A log-transformed data set was derived by taking the natural logarithms of the measured concentrations. The arithmetic mean and the standard deviation of the log-transformed data were calculated using Equations 4.1 and 4.2 above, and the 95th percentile upper confidence limit of the mean concentration was calculated as (U.S. EPA, 1992b):

$$\bar{c}_{95} = e^{\bar{c}_t + 0.5 s_t^2 + \left(\frac{s_t H}{\sqrt{n-1}} \right)} \quad \textbf{(4)}$$

where the new terms are:

$\bar{c}_t$ the arithmetic mean of the log-transformed data;
s_t the standard deviation of the log-transformed data; and
H the H-statistic (Gilbert, 1987).

In some cases the 95th percentile upper confidence limit of the mean exceeded the maximum detected concentration for a chemical. When this occurred, the maximum detected concentration of the chemical was used in place of the 95th percentile upper confidence limit. This procedure is consistent with EPA risk assessment guidance (U.S. EPA, 1992b). Additionally, if only one or two samples were available for a chemical, the maximum detected concentration was used. When only one or two samples are available for a chemical, the shape of the underlying distribution cannot be readily determined, and the maximum detected concentration of the chemical likely provides a reasonably conservative estimate of the concentration present in ambient air.

Statistical summaries of the data used in this risk assessment, as well as the EPCs calculated for all chemicals, are presented in Tables 4.1 through 4.6. As discussed previously, the duplicate samples collected at the Bethlehem Community Center were compared and found to be similar.

These results were first averaged for each sampling date to develop the data summary presented in Table 4.2. As per EPA federal and regional guidance, the EPCs used for the central tendency exposure evaluation were not varied from those used for the RME evaluation (U.S. EPA, 1992b; U.S. EPA, 1995a).

4.3.3.3 Exposure assessment

Standard risk assessment methods were used to estimate the rates at which people are exposed to the air pollutants measured in the CATS. Exposure assessment methods derive principally from exposure assessment guidelines (*e.g.*, U.S. EPA, 1992c), and are geared toward estimating different levels of exposure that people may experience. Exposure assessment primarily focuses on chronic exposure, in which a person is assumed to breath relatively low levels of air pollutants repeatedly over a lengthy period of time. Consideration of acute (short term) exposure to relatively high levels of air pollutants is also included in the CATS risk assessment (as described in Chapter 5), although methods for its evaluation are not as well developed as those for assessing chronic exposure.

Two categories of chronic exposure are assessed in the CATS risk assessment — central tendency (CT) exposure and reasonable maximum exposure (RME) — to attempt to give risk managers a sense of the distribution of risks in an exposed population.[6] The CT scenario attempts to assess the exposure to chemicals by a typical person in an exposed population. The goal of the RME scenario is to evaluate a level of exposure for a highly exposed person (greater than 90th percentile exposure) living in the study area, but not higher than the most highly exposed person. It is possible that a few people may be exposed to pollutants at higher levels than the RME estimate, but the majority of people are expected to be exposed to lower levels of pollutants than the RME. Consistent with this risk assessment methodology, the RME exposure assessment makes the following basic assumptions:

- A person lives, works, and otherwise stays near the monitoring location for the majority of a thirty-year period;
- This person spends 6 years of the 30 year period as a child and 24 years as an adult;
- The air that he/she breathes contains the same average concentrations of pollutants measured in the CATS (during the 12 month monitoring period) over a 30 year period.

These chronic, or long-term, estimates of pollutant exposure are quantified in terms of dose, a measurement that calculates the amount of pollutant exposure *per* an individual's body weight during a given time period (e.g., on a daily basis). Body weights and other exposure parameters

[6]In air toxics risk assessments, where modeling is employed, an alternative methodology is often used to evaluate risk distributions in a population. See Section 4.6 for a more full discussion of the various methods used to evaluate variation in risk.

that are used to estimate doses for adults and children are presented in Table 4.8. The formula used to calculate the average daily dose is:

$$ADD = \frac{EPC \times IR \times EF \times ED}{BW \times AT} \quad \textbf{(4)}$$

where the terms are:

ADD	average daily dose (in units of mg/kg-day);
EPC	exposure point concentration of the chemical in air that is breathed (in mg/m^3);
IR	inhalation rate (in m^3/day);
EF	the exposure frequency during the entire exposure period (days/year);
ED	exposure duration (years);
BW	body weight (kg); and
AT	averaging time (ED x 365 days/year).

Calculation of the *ADD* is relevant to the evaluation of chronic health effects other than cancer (*i.e.*, non-carcinogenic risks). In applying equation 4.5 to a non-carcinogenic chemical, the exposure duration (*ED*) in both the numerator and denominator are set equal to reflect the fact that the dose is averaged only over the period of time during which exposure occurs. *ADD* is calculated separately for adults and children but are not added, since it is presumed that once exposure stops, the risk stops (see below, Section 4.4, Toxicity Assessment).

A second measure — the lifetime average daily dose (*LADD*) — is used to evaluate the additional risk that a person might develop cancer from exposure to air pollutants, averaged over their lifetime. The lifetime average daily dose *LADD* is calculated with the same formula used for *ADD* (equation 4.5 above), except that the exposure is averaged over a person's lifetime (i.e., ED in the denominator is set equal to 70 years). Thus, the exposure duration term in the numerator corresponds to the portion of a person's life during which exposure is assumed to occur (*e.g.*, for reasonable maximum exposure, 6 years for a child, and 24 years for an adult), while the averaging time (*AT*) corresponds to the length of a person's life (70 years x 365 days/year). Performing the analysis in this fashion provides a lifetime average dose from a 30 year exposure. Unlike the analysis for non-carcinogens, the analysis for carcinogens is performed in this manner due to the presumption that, for most cancer causing agents, once the exposure stops, the risk continues (see Section 4.4, Toxicity Assessment). In estimating risks to carcinogenic chemicals, *LADD* values are calculated separately for adults and children, and then added together to give lifetime average risk estimates.

Both the average daily dose *ADD* and the lifetime average daily dose *LADD* are assumed to be proportional to the concentration of a chemical in air (*EPC*), which is based upon the detailed

measurements taken in the CATS. As described previously in the section on data analysis (section 4.3.3.2), the statistical metric used for the EPC is the 95th percentile upper confidence limit of the mean ($\overline{c}_{95}$). Discussions of the calculation and use of $\overline{c}_{95}$ are provided in section 4.3.3.2. Ramifications of the use of upper confidence limits for EPCs (as opposed to means or other statistical measures) are discussed in the uncertainty analysis.

4.4 Toxicity assessment

The toxicity assessment examines information concerning the potential human health effects associated with exposure to COPCs. The goal of the toxicity assessment is to provide, for each COPC, a quantitative estimate of the relationship between the magnitude and type of exposure and the likelihood of human health effects. The toxicity values presented in this section are integrated with the outputs of the exposure assessment to characterize the potential for the occurrence of adverse health effects (see Section 4.5, Risk Characterization).

The toxicity assessment involves the identification of cancer and noncancer health effects associated with each of the chemicals that have been selected as COPCs. It also provides the quantitative relationship between exposure and potential incidence of adverse health effects, also referred to as the dose-response relationship.

When developing a dose-response relationship for a chemical, toxicologists usually assess the entire toxicological database to develop cancer slope factors (CSFs) for carcinogenic effects and reference doses (RfDs) for noncarcinogenic effects. These data may include epidemiological studies, long-term animal bioassays, short-term tests, and comparisons of molecular structure. Data from these sources are reviewed to determine if a chemical is likely to be toxic to humans. Because of the general lack of available human studies, however, the majority of toxicity data used to derive CSFs and RfDs come from animal studies.

Inadequate toxicological data exist for a number of chemicals that were detected during CATS sampling activities. Specifically, non-cancer toxicological data are unavailable for 45 chemicals, while carcinogenic toxicological data are unavailable for 31 chemicals. Chemicals with no toxicity data were not quantitatively evaluated in this risk assessment, but are discussed in the evaluation of uncertainties.

4.4.1 Chronic noncarcinogenic effects

For chemicals that cause noncarcinogenic health effects, it is assumed that there exists a dose below which no adverse health effects will occur. Below this "threshold" dose, exposure to a chemical can be tolerated without adverse effects. Toxic effects are thought to only happen when physiologic protective mechanisms are overcome as exposure to a chemical exceeds its threshold level.

Systemic toxicity involves absorption and distribution of a toxicant from its entry point in the body to the site where it produces deleterious effects. The alternative to a systemic effect is a portal of entry effect, which may be produced at the site of first contact between the biological system and the toxicant. Some materials produce both systemic and portal of entry effects. In these cases, the toxicant can cause effects at the site of absorption and then be transported to another part of the body where it produces additional toxic effects. Substances that are considered systemic toxicants usually do not cause the same degree of toxicity in all parts of the body that are encountered, but instead elicit greater toxicity in one or a few organs or biological systems (called a "target organ" effect). Maternal and developmental endpoints are considered forms of systemic toxicity.

The potential for noncarcinogenic health effects resulting from exposure to chemicals is assessed by comparing an exposure estimate (intake or dose) to an RfD. RfDs are estimates (with uncertainty typically spanning an order of magnitude or greater) of a daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects. Chronic RfDs are specifically developed to be protective for long-term exposure to a compound (U.S. EPA, 1989).

The RfD is expressed in units of mg/kg/day, and represents an average daily intake of a contaminant per kilogram of body weight that is not sufficient to cause the threshold effect of concern. An RfD is specific to the chemical, the route of exposure, and the duration of time over which the exposure occurs. Separate RfDs generally exist for ingestion and inhalation pathways. When evaluating the noncarcinogenic effects, it is also necessary to identify the target organ for the critical effect used to develop the RfD. Specifically, RfDs are based upon the critical effect levels observed during human and animal studies, such as the lowest dose at which adverse health effects are observed. Uncertainty factors (UF) are then applied to (*i.e.*, divided into) the experimental doses. Depending on the number and types of studies and their quality, a number of different uncertainty factors may be included. The bases for application of different uncertainty factors are explained below:

- A UF of 10 may be used to account for variation in the general population and is intended to protect sensitive subpopulations, such as children and the elderly.

- A UF of 10 may be used when extrapolating from animal to humans. This factor is intended to account for the interspecies variability between humans and other mammals.

- A UF of 10 may be used when an adverse effects level derived from a subchronic study (instead of a chronic study) is used as the basis for the chronic RfD.

- A UF of 10 may be used when a Lowest Observed Adverse Effect Level (LOAEL) is used instead of a No Observed Adverse Effects Level (NOAEL). This factor is intended to account for the uncertainty associated with extrapolating from LOAELs to NOAELs.

A modifying factor (MF) may also be applied. A MF ranging from greater than zero to 10 is included to reflect a qualitative professional assessment of uncertainties in the critical study and in the entire database for the chemical not explicitly addressed by the UFs. The default value for the MF is 1 (U.S. EPA, 1989).

Inhalation noncancer reference values are typically expressed as a reference concentration (RfC) in units of mg/m^3. The RfC must be converted to an inhalation RfD_i to match the corresponding form of exposure estimates in the CATS. The conversion from an RfC to an RfD_i is based on an adult who inhales 20 m^3 of air per day and weighs 70 kg. The RfD_i is calculated by multiplying the RfC by 20 m^3/day and then dividing the calculated value by 70 kg. In the CATS assessment, such converted reference doses are referred to as a RfD_i to identify a noncarcinogenic reference dose for the inhalation route of exposure. [Note that this assessment does not use oral toxicity data to evaluate the inhalation route of exposure when inhalation data is missing for noncancer effects due to questions regarding portal of entry effects (for a more thorough description of the uncertainties associated with this subject, see Section 4.5, Uncertainty Analysis).]

A complete listing of the noncancer inhalation toxicity data used in the risk assessment is provided in Table 4.8. Toxicity data were obtained using the hierarchy of data sources and methodologies advocated by the U.S. EPA's OAQPS (U.S. EPA 2001b).

When evaluating toxicity data, EPA applied a consistent priority scheme to the universe of dose-response information as follows:

- EPA's Integrated Risk Information System (IRIS; U.S. EPA, 2001);
- Agency for Toxic Substances and Disease Registry (ATSDR) Minimum Risk Levels (MRLs; ATSDR, 2001);
- California EPA's Reference Exposure Levels (RELs) and unit risks (CAL EPA, 2001); and
- Reference concentrations and unit risks published in EPA's Health Effects Assessment Summary Tables (U.S. EPA, 1997c).

The most recent toxicity values from these databases are used in the CATS risk assessment.

4.4.2 Carcinogenic effects

Although a relatively small number of chemicals have been identified as proven human carcinogens, many other chemicals are suspected to cause carcinogenic effects. Similar to the evaluation of the toxicity of noncarcinogenic substances, the carcinogenic evaluation of chemicals includes both qualitative and quantitative analyses, including a weight-of-evidence measure of the likelihood that a chemical induces cancer in humans. This evaluation is based upon peer-reviewed scientific studies of humans and animals. The six weight-of-evidence classifications recognized by EPA are presented below (U.S. EPA, 1986).

- **Group A - Human Carcinogen:** Based on sufficient human data, the chemical is identified as a human carcinogen.

- **Group B1 - Probable Human Carcinogen:** Human data indicate that a causal association between the chemical and carcinogenic effects exists; however, alternative explanations can not be dismissed.

- **Group B2 - Probable Human Carcinogen:** Human data are insufficient to support a causal association, however, testing data in animals support a causal association.

- **Group C - Possible Human Carcinogen:** Human data are inadequate or lacking; however, animal data suggests a causal association. However, animal studies have deficiencies that limit their interpretation.

- **Group D - Not Classifiable as to Human Carcinogenicity:** Human and animal data are lacking or inadequate.

- **Group E - Evidence of Noncarcinogenicity to Humans:** Human data are negative or lacking, and adequate animal data indicate no association with cancer.

EPA assumes that thresholds generally do not exist for carcinogens; therefore, any exposure is associated with some quantifiable risks. Thus, the toxicity values for carcinogenicity, referred to as cancer slope factors (CSF), are a quantitative estimate of the probability of developing cancer (not a threshold dose, as is used for non-carcinogens). Specifically, a CSF is defined as a plausible upper-bound estimate of the probability of a response per unit intake of a chemical over a lifetime. The slope factor is used to estimate an upper-bound probability of an individual developing cancer as a result of a lifetime of exposure to a particular level of a potential carcinogen. Slope factors are derived from studies of carcinogenicity in humans and/or laboratory animals, and are typically calculated for compounds in Groups A, B1, and B2. Some Group C carcinogens also have slope factors.

Since the publication of the 1986 EPA cancer guidelines, there is a better understanding of the variety of ways in which carcinogens can operate. Today, many laboratories are moving toward adding new test protocols in their programs directed at mode of action questions. Based on this evolving science, EPA has proposed a new analytical framework for evaluating the carcinogenicity of chemicals that allows for the incorporation of all relevant biological information, a recognition of a variety of situations regarding cancer hazard, and flexibility to allow for consideration of future scientific advances (U.S. EPA, 1996). To the extent that such advances have been utilized by the CATS sources of toxicity data, the information was included in this assessment.

Slope factors are expressed in units of $(mg/kg\text{-}day)^{-1}$ for the oral route of exposure and as inhalation unit risks (IURs) in units of reciprocal $\mu g/m^3$ $(1/\mu g/m^3)$ for inhalation routes of exposure. Because cancer risk characterization requires an estimate of a reciprocal dose in units of $(mg/kg\text{-}day)^{-1}$ to match the forms of doses calculated in the CATS, IUR values must be converted to the mathematical equivalent of inhalation cancer slope factors, or risk per unit dose in (mg/kg-day). This is done by assuming that adult humans on average weigh 70 kg and inhale 20 m^3 of air per day [i.e., the inhalation unit risk $(1/\mu g/m^3)$ is divided by 20 m^3/day, multiplied by 70 kg, and multiplied by 1,000 $\mu g/mg$ to yield the mathematical equivalent of an inhalation slope factor in $(mg/kg\text{-}day)^{-1}$]. In the CATS assessment, such converted slope factors are referred to as a CSP_i to identify a carcinogenic slope factor for the inhalation route of exposure.

When determining the potential carcinogenic risk associated with airborne contaminants, the most preferable CSFs are those derived specifically for the inhalation route of exposure. However, in the absence of an inhalation IUR for a particular chemical, its oral CSF was used in the evaluation as a direct surrogate toxicity value.

For chromium compounds, the IRIS RfC for particulate hexavalent chromium was used in preference to the RfC for chromic acid mists and dissolved aerosols. Both the RfC and the IUR for hexavalent chromium were adjusted to reflect an assumption that 34% of all atmospheric chromium is hexavalent. This represents the best judgment of EPA staff, based on limited data on species of chromium emitted from five significant source categories. The total chromium mass in these emissions ranged from 0.4% to 70% hexavalent. Because the high end of the range was associated exclusively with electroplating sources, EPA chose 34%, the upper end of the range for utility boilers. It is likely that most sources of chromium missions in the US contain smaller amounts of hexavalent chromium (U.S. EPA, 2001b). The uncertainty of having proceeded in this manner is discussed in Section 4.6.

The IRIS unit risk for nickel inhalation was derived from evidence of the carcinogenic effects of insoluble nickel compounds in crystalline form. Soluble nickel species, and insoluble species in amorphous form, do not appear to produce genotoxic effects by the same toxic mode of action as insoluble crystalline nickel. Nickel speciation information for some of the largest nickel-emitting sources (including oil combustion, coal combustion, and others) suggests that at least 35% of

total nickel emissions may be soluble compounds. The remaining insoluble nickel emissions are not well-characterized, however. Consistent with this limited information, this analysis has conservatively assumed that 65% of emitted nickel is insoluble, and that all insoluble nickel is crystalline. On this basis, the IUR for nickel subsulfide (representing pure insoluble crystalline nickel) was multiplied by 0.65 and applied to all nickel compounds (U.S. EPA, 2001b). The uncertainty of having proceeded in this manner is discussed in Section 4.6.

A complete listing of oral and inhalation cancer toxicity data is provided in Table 4.9. The hierarchy of sources for this information is the same as that described for non-carcinogenic toxicity data in Section 4.4.1. IRIS data were given first priority, followed by California EPA data, and HEAST data (ATSDR does not provide CSPs).

4.5 Risk characterization

This section provides a characterization of the human health risks associated with the potential exposures to COPCs identified at the six monitoring stations in Chattanooga. Section 4.5.1 outlines the methods used to quantitatively estimate the type and magnitude of potential risks for exposed individuals. A summary of the risk assessment results is provided in Section 4.5.2.

4.5.1 Methodology for estimation of quantitative risks

Potential human health risks resulting from exposure to COPCs are estimated using algorithms established in U.S. EPA guidance (U.S. EPA, 1989). These methods are protective of human health since they likely overestimate (rather than underestimate) risk. The methodologies use specific algorithms to calculate risk as a function of chemical concentration, human exposure parameters, and toxicity.

Risks from airborne chemicals were calculated for either noncarcinogenic or carcinogenic effects. Some carcinogenic chemicals may also exhibit noncarcinogenic effects. In such cases, potential impacts were characterized for both types of health effects.

4.5.1.1 Noncarcinogenic effects

Non-cancer endpoints were evaluated for both a child and an adult. Since the child's average daily dose is greater than the adults due to the child's greater ratio of inhalation rate to body weight, exposure estimates for the child are greater than those for the adult, as expressed in terms of average daily dose. To estimate the risk of health effects other than cancer, the ADD for each chemical was compared to an inhalation reference dose (RfD_i, in units of mg/kg-d) that corresponds to a level of exposure that will likely not cause adverse health effects. The ratio between the ADD for a chemical and its RfD_i is called a hazard quotient (HQ). A hazard quotient

of less than one indicates that the chemical is unlikely to adversely affect human health. Specifically, the hazard quotient for each chemical was calculated as follows:

$$HQ = \frac{ADD}{RfD_i} \quad \textbf{(4)}$$

To estimate the aggregate non-cancer health risk associated with all chemicals detected in air at each monitoring location, a total hazard index (*HI*) was calculated by summing the hazard quotients for each chemical detected. A total hazard index of less than one indicates that the inhalation of all detected chemicals in ambient air is unlikely to adversely affect human health. However, a total hazard index of greater than one does not necessarily indicate a potential adverse risk to human health. Due to the health-protective method used to establish RfD_is, an HI, or even an individual hazard quotient, in excess of one may or may not reflect a likelihood of adverse health effects being manifested. In addition, different chemicals often affect different organs and systems in the human body (the "target organ effect"). Adding hazard quotients for chemicals with different effects may not accurately reflect the toxicologic effects of the two chemicals. (Note that for the non-cancer toxicity value, the source of the value usually lists the critical effect on a particular organ or bodily system upon which the toxicity value is based. While the toxicity value is based on a particular critical effect, the chemical may affect other organs and bodily systems.)

For any *HQ*s exceeding one, the critical effects upon which the toxicity values were based were identified. Target organs for chemicals having *HQ*s greater than one at each monitoring location are summarized in Table 4.31. (Note that a thorough analysis of the validity of disaggregating HIs $\geq$ 1 based on this "target organ effect" is a resource intensive effort that requires more than identifying just the critical effect for chemicals having, individually, a HQ $\geq$ 1. However, in a practical sense, this analysis may not even be necessary. See Section 4.6.5 for a more full discussion of how this issue was dealt with in this assessment.)

4.5.1.2 Carcinogenic effects

The risk of cancer is estimated by multiplying the LADD for each chemical by its cancer potency (CPS_i in units of kg-d/mg). As shown in the exposure parameters table (Table 4.10), the maximum reasonable lifetime average exposure assumes 6 years of exposure as a child and 24 years of exposure as an adult. The incremental cancer risk (*CR*) for each person (either a child or an adult) is calculated as follows:

$$CR = LADD \times CPS_i \quad \textbf{(4)}$$

As with non-cancer endpoints, a total incremental cancer risk is calculated by summing cancer risks for all of the chemicals detected at a given monitoring location. Incremental cancer risks are estimated for a child, adult, and then combined for a full 30 year exposure period.

4.5.2 Risk characterization summaries

This section contains a summary of the results of the risk characterization for the air risk assessment for the CATS. Separate risk characterization summaries are provided for the Emma Wheeler Homes, Bethlehem Community Center, 20th Street Fire Station, Cellular One Tower Site, East Brainerd Fire Station, and the River Park Site. Both noncarcinogenic hazards and carcinogenic risk estimates are described for each location. In summarizing these results, chemicals having a cancer risk greater than or equal to 1×10^{-6} or a hazard quotient greater than or equal to one are highlighted.

Noncarcinogenic risks are presented in Tables 4.11 through 4.16. Carcinogenic risks are presented in Tables 4.17 through 4.22. These tables present all chemicals that were detected at each monitoring location and for which toxicological data are available. Tables 4.29 and 4.30 present a summary of all total risk estimates. All risk and hazard index calculations presented in Tables 4.11 through 4.22 were performed using Reasonable Maximum Exposures (RME). Cancer risks and hazard indices for central tendency exposures are presented in summary form only in Tables 4.29 and 4.30. The principle findings at each location are described in narrative form in the following sections.

4.5.2.1 Emma Wheeler Homes

The non-cancer hazard index (HI) for an adult resident was 3 with the primary contributors being manganese and formaldehyde. The HI for a child resident was 8 with manganese and formaldehyde having individual hazard quotients greater than or equal to one.

Calculated cancer risks were 8×10^{-5} for an adult resident, 6×10^{-5} for a child resident, and 1×10^{-4} for a long-term (*i.e.*, 30-year) resident. Chemicals at this monitor that individually had risks in excess of 1×10^{-6} for the long-term resident were formaldehyde, chloroform, benzene, chromium, benzo(a)pyrene, carbon tetrachloride, bromodichloromethane, 1,4-dichlorobenzene, arsenic, chloromethane, nickel, tetrachloroethylene, trichloroethylene, and benzo(b)fluoranthene.

Table 4.11 summarizes RME HQs for the Emma Wheeler Homes. Table 4.17 summarizes incremental cancer risks. Table 4.23 summarizes all chemicals with an estimated HQ greater than or equal to 1 or an estimated cancer risk greater than or equal to 1×10^{-6}. Table 4.31 summarizes target organs for chemicals with a HQ greater than 1.

4.5.2.2 Bethlehem Community Center

As discussed previously, the duplicate samples collected at the Bethlehem Community Center on each date were combined prior to derivation of exposure point concentrations. The non-cancer hazard index (HI) for an adult resident was 2 with the primary contributor being manganese. The HI for a child resident was 6 with manganese having an individual hazard quotient greater than one.

Calculated cancer risks were 6×10^{-5} for an adult resident, 4×10^{-5} for a child resident, and 1×10^{-4} for a long-term (*i.e.*, 30-year) resident. Chemicals at this monitor that individually had risks in excess of 1×10^{-6} for the long-term resident were formaldehyde, benzene, chloroform, chromium, carbon tetrachloride, 1,4-dichlorobenzene, benzo(a)pyrene, arsenic, chloromethane, nickel, and tetrachloroethylene.

Table 4.12 summarizes RME HQs for the Bethlehem Community Center. Table 4.18 summarizes incremental cancer risks. Table 4.24 summarizes all chemicals with an estimated HQ greater than or equal to 1 or an estimated cancer risk greater than or equal to 1×10^{-6}. Table 4.31 summarizes target organs for chemicals with a HQ greater than 1.

4.5.2.3 20th Street Fire Station

The non-cancer hazard index (HI) for an adult resident was 2 with the primary contributor being manganese. The HI for a child resident was 6 with manganese having an individual hazard quotient greater than one.

Calculated cancer risks were 6×10^{-5} for an adult resident, 4×10^{-5} for a child resident, and 9×10^{-5} for a long-term (*i.e.*, 30-year) resident. Chemicals at this monitor that individually had risks in excess of 1×10^{-6} for the long-term resident were formaldehyde, chromium, benzene, chloroform, carbon tetrachloride, bromodichloromethane, arsenic, chloromethane, 1,1,2-trichloroethane, benzo(a)pyrene, nickel, tetrachloroethylene, and 1,4-dichlorobenzene.

Table 4.13 summarizes RME HQs for the 20th Street Fire Station. Table 4.19 summarizes incremental cancer risks. Table 4.25 summarizes all chemicals with an estimated HQ greater than or equal to 1 or an estimated cancer risk greater than or equal to 1×10^{-6}. Table 4.31 summarizes target organs for chemicals with a HQ greater than 1.

4.5.2.4 Cellular One Tower Site

The non-cancer hazard index (HI) for an adult resident was 2 with the primary contributors being cobalt and formaldehyde. The HI for a child resident was 4 with no chemical having an individual hazard quotient greater than one.

Calculated cancer risks were 4×10^{-5} for an adult resident, 3×10^{-5} for a child resident, and 7×10^{-5} for a lifetime resident. Chemicals at this monitor that individually had risks in excess of 1×10^{-6} for the long-term resident were formaldehyde, chromium, benzene, carbon tetrachloride, arsenic, bromodichloromethane, benzo(a)pyrene, chloroform, chloromethane, and tetrachloroethylene.

Table 4.14 summarizes RME HQs for the Cellular One Tower Site. Table 4.20 summarizes incremental cancer risks. Table 4.26 summarizes all chemicals with an estimated HQ greater than or equal to 1 or an estimated cancer risk greater than or equal to 1×10^{-6}. Table 4.31 summarizes target organs for chemicals with a HQ greater than 1.

4.5.2.5 East Brainerd Fire Station

The non-cancer hazard index (HI) for an adult resident was 2 with the primary contributor being cobalt. The HI for a child resident was 5 with cobalt having an individual hazard quotient greater than one.

Calculated cancer risks were 4×10^{-5} for an adult resident, 3×10^{-5} for a child resident, and 7×10^{-5} for a long-term (*i.e.*, 30-year) resident. Chemicals at this monitor that individually had risks in excess of 1×10^{-6} for the long-term resident were formaldehyde, chromium, benzene, carbon tetrachloride, bromodichloromethane, hexachloro-1,3-butadiene, chloroform, arsenic, chloromethane, 1,4-dichlorobenzene, and tetrachloroethylene.

Table 4.15 summarizes RME HQs for the East Brainerd Fire Station. Table 4.21 summarizes incremental cancer risks. Table 4.27 summarizes all chemicals with an estimated HQ greater than or equal to 1 or an estimated cancer risk greater than or equal to 1×10^{-6}. Table 4.31 summarizes target organs for chemicals with a HQ greater than 1.

4.5.2.6 River Park Site

The non-cancer hazard index (HI) for an adult resident was 2 with the primary contributors being cobalt, formaldehyde, and manganese. The HI for a child resident was 5 with cobalt having the only individual hazard quotient greater than or equal to one.

Calculated cancer risks were 4×10^{-5} for an adult resident, 3×10^{-5} for a child resident, and 7×10^{-5} for a long-term (*i.e.*, 30-year) resident. Chemicals at this monitor that individually had risks in excess of 1×10^{-6} for the long-term resident were formaldehyde, chromium, benzene, carbon tetrachloride, bromodichloromethane, arsenic, chloroform, chloromethane, nickel, and tetrachloroethylene.

Table 4.16 summarizes RME HQs for the River Park Site. Table 4.22 summarizes incremental cancer risks. Table 4.28 summarizes all chemicals with an estimated HQ greater than or equal to 1 or an estimated cancer risk greater than or equal to 1×10^{-6}. Table 4.31 summarizes target organs for chemicals with a HQ greater than 1.

4.5.2.7 Central tendency estimates

The results of the CT evaluation are summarized in Tables 4.29 and 4.30 for noncarcinogenic hazards and carcinogenic risks, respectively. Based on central tendency exposure estimates, adult HIs were 76% of RME HIs and child HIs were 69% of RME HIs. Overall hazard indices exceed one, ranging from 1 to 2 for the adult and 3 to 5 for the child at the six locations. Similar results were obtained for carcinogenic risks based on central tendency exposure estimates. Lifetime incremental risks of cancer for CT exposure were 45% of RME incremental risks, ranging in aggregate from 1×10^{-5} (East Brainerd Fire Station) to 2×10^{-5} (Emma Wheeler Homes). For most of the monitoring locations, at least one chemical with risks above 1×10^{-6} under RME exposure estimates no longer exceeded this level when central tendency exposure estimates were employed. These chemicals are indicated by italics on Tables 4.23 through 4.28.

4.6 Uncertainty analysis

This section discusses the uncertainties associated with this evaluation. The risk measures used in this evaluation are not fully probabilistic estimates of risk, but conditional estimates given a considerable number of assumptions about exposure and toxicity. Thus, it is important to consider the assumptions and uncertainties inherent in the risk assessment to place the risk estimates in proper perspective. Another use of uncertainty characterization can be to identify areas where additional data collection might significantly improve the basis for risk-based decision making (U.S. EPA, 1989).

Highly quantitative statistical uncertainty analysis is usually not practical or necessary for many risk assessments. As in all environmental risk assessments, it is already known that uncertainty about the numerical results is generally large (i.e., on the range of at least an order of magnitude or greater). Consequently, it is more important to identify the key variables and assumptions that contribute most to the uncertainty than to precisely quantify the degree of uncertainty in the risk assessment (U.S. EPA, 1989). The focus of this section is on qualitative information on the possible sources and magnitudes of uncertainty in the risk estimates and, where known, tools that may be used to reduce uncertainty.

There are uncertainties associated with each component of the risk assessment from data collection through risk characterization. For example, there is uncertainty in the initial selection of substances used to characterize exposures and risk on the basis of the sampling data. Other sources of uncertainty are inherent in the toxicity values for each substance and the exposure assessments used to characterize dose. Finally, additional uncertainties are incorporated in the risk assessment when exposures to several substances across multiple pathways are summed. In the following discussion, the main areas of uncertainty are discussed in the general context of the various risk assessment steps.

4.6.1 Chemicals evaluated in the risk assessment

The chemicals evaluated in this risk assessment were based on air sampling performed for the CATS project by U.S. EPA Region 4 and the CHCAPCB. The list of compounds included for analysis was extensive, but there is the possibility that some compounds of potential significance were not included in the list of compounds for analysis since routine methods are not available for a large number of known compounds. While this could lead to an underestimation of risk, it is not an area that can readily be reduced without access to additional resources and, in some cases, may be technically infeasible. Modeling is a practical alternative to monitoring for assessing the risk posed by additional chemicals.

Uncertainty may also be associated with the data evaluation process. Some data were rejected by the laboratory due to quality control issues. Excluding these data may result in an overestimation or underestimation of risks in the study area, depending on their influence on determining EPCs. Please see Appendix B for a full description of chemicals excluded from the analysis. Resampling, modeling, and perhaps advanced statistical evaluation data are possible tools that could be used to evaluate the affect of these excluded data (or to simply reduce the uncertainty) on risk estimates.

COPCs in this risk assessment were not compared to background concentrations. All COPCs were retained to provide a complete characterization of risk, even though some detected concentrations may be attributable to transport of chemicals into the Chattanooga region from sources external to the area. The use of this approach provides a more health protective assessment than assuming that some chemicals are attributable to external sources and excluding them from the evaluation.

4.6.2 Likelihood of the completed exposure pathways

There is little uncertainty that residents in the CATS study area are breathing air containing air pollutants. Many of the air monitoring locations used in this study are in very close proximity to occupied residential structures. However, indirect pathways such as deposition of airborne contaminants to soil or water with subsequent ingestion or dermal contact with airborne contaminants were not considered. Not having evaluated all possible exposure pathways will tend to underestimate the risk, but by an unknown amount. Reducing uncertainty in this area could require a significant modeling and/or monitoring effort.

4.6.3 Representativeness of risk estimates

The risk estimates presented in this report are based on a limited number of sampling points owing to resource constraints. Theoretically, risks in South Chattanooga should be reasonably similar to those calculated for the vicinity of the monitoring stations. However, the extent of the areal coverage represented by these few sampling points is unknown. It is also unknown how well the entire Chattanooga area is represented by just these six monitoring locations. A modeling effort would help to understand the temporal and spatial nature of air toxic concentrations in the Chattanooga area.

Other aspects of representativeness that are uncertain have to do with the amount of time that people are thought to be exposed and the range (or distribution) of exposure that may be occurring. These potential areas of uncertainty are discussed below.

4.6.3.1 Exposure duration

The risk estimates presented in this report presume that people remain in one specific residence in Chattanooga for no longer than 30 years and that, after that time, they may or may not remain in the Chattanooga area. Of course, people likely incur additional risks throughout their lives from breathing airborne pollutants; however, this assessment does not attempt to quantitate the entire risk that people incur over their full lifetime from breathing airborne pollutants in all their potential places of residency. In that sense, the risk estimates in this analysis very likely underestimate a persons true lifetime risk. Tools are, however, available to reduce this uncertainty. For example, there are sophisticated computer programs available that can be used to more realistically model the activity patterns people have as they go about working, resting, and playing (and the exposures they incur during such activities).[7] Ultimately, one may never know the true

[7] People do not usually stay in one place all day. Rather, they work, play, and may rest in different places. Such activities will affect their overall resulting exposures. In addition, indoor

(continued...)

distribution of risks for any population since detailed information on each of the individuals in the population would be required. However, stakeholders can reduce the uncertainty in risk estimates by applying these more advanced exposure assessment tools.

To get a sense of potential underestimation of risk that results from using only a 30 year exposure duration, the following simple analysis was performed:

For benzene at the 20th Street Fire Station, the RME cancer risk for a 30 year resident was calculated to be 1.32×10^{-5}. Had a 70 year (i.e., lifetime) exposure been presumed, the risk would have been calculated as the IUR times the EPC or:

$$(7.8 \times 10^{-6}\ m^3/\mu g)\ (3.03\ \mu g/m^3) = 2.36 \times 10^{-5}$$

Thus, the risk for a 70 year exposure is greater than that of a 30 year exposure by a factor of

$$2.36 \times 10^{-5} / 1.32 \times 10^{-5} = 1.8$$

Ultimately, all the 30 year risks presented in this assessment can be multiplied by a factor of 1.8 to get the risks for a population presumed to live in one residence for 70 full years. From this perspective, the impact on cancer risk values of having used only a 30 year exposure, rather than a 70 year exposure, is trivial (*i.e.*, there is little practical difference between a risk of 2×10^{-5} and 1×10^{-5}).[8]

It should be noted that, owing to the presumptions built into IURs and RfCs, it is arguably more robust to calculate risk by multiplying EPCs by unmodified toxicity factors (i.e., IUR and 1/RfC), rather than by estimating dose and multiplying by modified dose-equivalent forms of toxicity factors (i.e., CSF_is and $1/RfD_i$s) as was done in this assessment. However, as noted above, the practical implications of not having done so are negligible. The benefit of having performed the analysis as presented in this document is the potential additional insights one gains into the risks posed to important subpopulations in the community (*e.g.*, children).

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[7](...continued)
air may or may not be identical to outdoor air since indoor air is affected by indoor sources and may change the concentration of outdoor air as it enters the home. The ultimate effect of not having evaluated activity patterns and indoor air may over- or underestimate risk, depending on the individual in question.

[8] For non-cancer hazard, the difference between a 70 year exposure and an adult exposure of 24 years is even less (a factor of 1.05). Note that, unlike cancer, one may not combine adult and child hazards due to the threshold effect discussed in Section 4.4.1. As such, it is only relevant to compare the 24 year adult exposure (presumed in this assessment) to a 70 year exposure to gauge the effect of different exposure durations.

4.6.3.2 Distribution of risk in the population

In this analysis, the distribution or range of potential risks posed to the Chattanooga community was assessed by calculating risk posed to highly exposed and more average exposed children and adults *at each monitor*. This required the use of modified dose-forms of inhalation toxicity factors which, in an of itself, incorporates a certain (albeit, a relatively small) amount of uncertainty (see Section 4.6.3.1 above).

An alternate way of looking at risk distribution in a community (and, given sufficient information, the preferred way) is not to attempt to develop a range of risks at a particular monitor, but to assess the distribution of risks *across the entire study area*. While this is readily done with the information provided in this assessment by simply looking at the risks posed across monitors, performing the analysis in this fashion poses unique uncertainties, given that it is unknown how well only six monitors represent the entire Chattanooga area. This uncertainty could be readily reduced through the use of air dispersion modeling.

4.6.4 Parameter value uncertainty

During the course of a risk assessment, numerous parameter values such as breathing rate and body weight are included in the calculations of contaminant intake. An evaluation of certain key parameters is provided below.

The concentration term (the EPC) in this evaluation is relatively well-characterized given the number of samples collected over a one year period for each stationary sampling location and the number of stations included in the study. The values used to represent concentration are not likely to have underestimated the risk since a biased high-end estimate (the 95% UCL of the arithmetic mean) was used to estimate long-term time-averaged concentrations. Moreover, due to limited numbers of samples and the sensitivity of the 95% UCL for lognormally distributed contaminant concentrations (the most common assumption), maximum detected values were used in many cases as EPCs, a measure that biases high the long-term average values. If straight arithmetic average concentrations were used as EPCs, risk levels would be lower and would be equally likely to over- or underestimate risk. It should be noted that the use of one-half the detection limit as a surrogate for concentration for non-detected chemicals in any particular sample can influence the calculated 95% UCL value, possibly over- or underestimating the results (this is discussed more fully in Section 4.6.7). Nevertheless, the use of the 95% UCL should lead to an overestimation (and, thus, a conservative estimate) of risk.

Year-to-year variability is another source of potential uncertainty within EPCs. It is not known the extent to which the year during which these data were collected is representative of other years. Varying meteorological conditions, changing industrial processes and transportation habits, and a variety of other factors all influence the yearly average concentration of air pollutants. As

such, it is unknown how well the one year's worth of data collected in the CATS represents the other years of the presumed exposure duration. Reducing uncertainty in this area could require multi-year sampling, an evaluation of trends in emissions inventories, and modeling.

Values used for other exposure parameters used in this evaluation such as inhalation rate, exposure duration, and exposure frequency were obtained from a variety of sources. These parameter values were generally developed based on studies of activity patterns and physiology published in the primary, peer-reviewed literature. This risk assessment used both high-end (RME) and more average (CT) parameter values to evaluate risks. The RME values used are believed to be sufficiently conservative to overestimate risks for a typical member of the population (but not all people), while the CT parameters are likely to over and underestimate exposure levels for a number of people in the Chattanooga area (see Section 4.6.3.2 for a discussion of uncertainty in assessing variation in risk within a population).

4.6.5 Toxicity assessment uncertainty factors

Uncertainties associated with the toxicity assessment (determination of RfD_is and CSF_is and use of available criteria) are inherent in the risk assessment process. Uncertainty exists in the toxicological data base and in the methodology used to derive RfD_is and CSF_is (see also Section 4.6.3.1). However, an attempt has been made to compensate for this uncertainty through the use of uncertainty and modifying factors for RfD_is. For cancer effects, the uncertainty associated with dose-response factors is compensated for by assuming the 95 percent upper bound for the slope factor. While neither approach removes overall uncertainty, they do result in toxicity metrics that should lend towards overestimates (and thus conservative estimates) of risk. Both of these approaches impart a bias toward overestimating chemical toxicity, and hence, actual risk levels. In addition, oral CSFs were used in the inhalation pathway for COPCs lacking inhalation CSFs. This procedure will tend to reduce the underestimation of risks from this pathway (and may well tend to overestimate it).

Although lead was identified as a COPC in air, a quantitative risk assessment of lead has not been performed for non-carcinogenic health effects. The current standard for lead, measured as a quarterly standard, is 1.5 $\mu g/m^3$ (40 CFR Part 50). The NAAQS for lead was not exceeded at any of the study areas, nor were values close to the standard measured (the highest value was less than 5% of the standard). Exposure to airborne lead is probably not a significant source (or risk) compared to other chemicals.

Toxicity data were not available to evaluate approximately 30 of the 85 chemicals detected in the air samples, including the essential nutrients iron and magnesium. These chemicals were not evaluated in this risk assessment. Therefore, some uncertainty has been introduced into the risk assessment by omitting an evaluation of these chemicals, with the result that the risks presented are likely to be underestimated values. Reducing uncertainty for these chemicals would necessitate the development of an acceptable inhalation toxicity value for these chemicals.

Modifications to the toxicological data for nickel and chromium based on assumed forms of nickel and chromium in the atmosphere represent additional sources of uncertainty. The IUR used for nickel equaled the IUR for nickel subsulfide published in IRIS (U.S. EPA, 2001) multiplied by a factor of 0.65 to account for the presence of 35% of the nickel in non-carcinogenic forms. However, given the unknown nature of the nickel composition in the Chattanooga area, this may or may not be a conservative assumption. The RfD_i and IUR for chromium were adjusted to reflect data suggesting that most chromium in the atmosphere is not likely to be Chromium VI. While EPA's evaluation indicates that most sources of chromium emissions in the U.S. contain less than 34% hexavalent chromium, given the unknown nature of composition of chromium emissions in the Chattanooga area, this may or may not be a conservative assumption. An evaluation of the chromium and nickel sources in the Chattanooga area should help to shed some light on the veracity of these assumptions.

Other aspects of toxicity assessment uncertainty include:

- ***Use of Animal Data as a Surrogate for Human Exposures*** - A large amount of uncertainty exists due to the models used to extrapolate from animal data to humans, including the use of models to evaluate responses in the low dose portion of the experimental dose-response curve. Depending on the chemical in question, such assumptions may tend to over- or underestimate the resulting risk. IURs for chemicals with a weight-of-evidence cancer classification of A or B1 are based on human data. IURs for chemicals with a classification of B2 or C are based primarily on animal data. Weight-of-evidence cancer classifications for chemicals considered in the risk assessment were provided on Table 4.9.

- ***Multiple Substance Exposure Uncertainties*** - Uncertainties associated with summing risks or hazard quotients for several substances are of particular concern in the risk characterization step. The assumption of dose additivity ignores possible synergisms or antagonisms among chemicals, and assumes similarity in mechanisms of action and metabolism. Unfortunately, data to assess interactions quantitatively are generally lacking. In the absence of adequate information, this assessment has made the assumption that carcinogenic and non-carcinogenic risks should be treated as additive. These assumptions are made to help prevent an underestimation of cancer risk or potential noncancer health effects. For the CATS evaluation, the following statements regarding uncertainty in dose additivity may be made:

  - The idea that all carcinogens should be added together may overestimate the risk since it is only surmised that all carcinogens behave according to the linearized multistage model of cancer in which extrapolation to zero is assumed in the low dose portion of the dose-response curve. In fact, a number of carcinogens may actually behave more like classical non-carcinogens and exhibit a threshold effect. Conversely, neither antagonistic effects (which would cause a further

overestimation of risk) nor synergistic effects (which would cause the opposite) have been taken into account.

- All non-carcinogens were added together to arrive at a total hazard index. It is known, however, that many non-carcinogens target specific organs and other bodily systems. In some cases, it is appropriate to disaggregate the resulting hazard index based on a thorough knowledge of what is commonly referred to as the "target organ effect." Because the hazard quotient for at least one individual chemical exceeds a value of 1 at five of the six sampling locations, disaggregated hazard indices would exceed 1 for at least one target organ at most sampling locations (although the degree of exceedances would be smaller for target organ-based hazard indices) and, so, were not done. Similar to carcinogens, the role of unknown antagonistic and synergistic effects has also not been evaluated in this uncertainty analysis.

4.6.6 Tentatively identified compounds (TICs)

TICs were excluded from the risk calculations presented previously. By definition, both the identity and amount of these chemicals is uncertain. However, excluding these chemicals from the analysis likely results in an underestimate of risk estimates with respect to these chemicals. Of the 84 TICs reported by the laboratory, toxicological data of any kind are available for only 7 (acetaldehyde, benzyl chloride, butoxyethanol, hexane, isopropanol, methylcyclohexane, and propene). Toxicological data for those 7 chemicals are presented in Table 4.32. Hazard indices and cancer risks estimated for inhalation of these 7 TICs are presented in Tables 4.33 and 4.34 respectively. Of these 7 TICs, only the hazard indices estimated for acetaldehyde exceed 1. HQs for acetaldehyde range from 10 at the Bethlehem Community Center to 25 at the East Brainerd Fire Station. Estimated incremental cancer risks for both acetaldehyde and benzyl chloride, the only two TICs with carcinogenic toxicological data, exceed 1×10^{-6}. The maximum incremental cancer risk estimated for acetaldehyde is 1×10^{-4} at the East Brainerd Fire Station, and the maximum incremental cancer risk estimated for benzyl chloride is 1×10^{-6} at both the Emma Wheeler Homes and the Bethlehem Community Center.

While these data indicate possible HIs exceeding 1 and cancer risks exceeding 1×10^{-6}, it is important to keep in mind that these are *tentatively* identified compounds. For example, while the laboratory reports acetaldehyde as a TIC, present at concentrations ranging from 10 $\mu g/m^3$ to 200 $\mu g/m^3$, EPA recently estimated that the average acetaldehyde concentration in Tennessee is approximately 0.5 $\mu g/m^3$ and 1 $\mu g/m^3$ in Hamilton County (U.S. EPA, 2001a). Thus, additional analysis to confirm such analytical results and an assessment of the frequency with which these chemicals were detected (e.g., benzyl chloride detected in only one sample at Emma Wheeler homes) may be warranted prior to their use for scientifically supportable decision making purposes. No further analysis of chronic risks for these chemicals was performed in this assessment.

The maximum concentrations of TICs detected in samples were also compared to acute toxicological data. Acute toxicological data were only available for four chemicals: acetaldehyde, benzyl chloride, butoxyethanol, and isopropanol. The maximum detected concentrations of all four chemicals were less than acute toxicological data, as shown in Table 4.35, indicating that exposure to these chemicals is not likely to result in acute health effects.

4.6.7 Values below detection limits

When calculating the EPC, the way in which one includes the non-detects may have a potentially significant influence on the resulting average concentration. EPA's *Guidance for Data Quality Assessment, Practical Methods for Data Analysis* (the DQO Guidance; U.S. EPA, 2000a) provides recommendations for evaluating such sample sets in which chemicals are only detected in a fraction of the samples collected. The document states that there are a variety of ways to evaluate data sets that have detections and non-detects; however, the document goes on to state that there are no general procedures that are applicable to all cases.

In this CATS analysis, the standard assumption was made that in any given data set for a chemical at a monitor, the non-detected values were to be included in the EPC calculation at one-half of the sample quantitation limit, regardless of the frequency of detection of the chemical in question in the particular data set (with the exception of chemicals detected so infrequently that the underlying distribution of the data could not be readily determined - in such cases the maximum value found was used to represent the long term average). Subsequent refined analyses would then be carried out only for specific chemicals that appeared to contribute significantly to the overall risk and for which the initial assumption of using one-half the detection limit for the non-detects was thought to have possibly significantly impacted the magnitude of the resulting EPC. This Section provides this additional analysis, and is described below.

In the CATS risk analysis, most of the chemicals with a HQ $\geq$1 or a cancer risk $\geq$ $1x10^{-6}$ were either detected in the majority of samples collected at a monitor (i.e., greater than 85% of the time) or they were rarely detected in the samples collected at a monitor (i.e., less than 10% of the time). Only a few chemicals collected at a monitor had a frequency of detection that fell in the mid-range (i.e., between 10% and 85% of the time). For example, manganese, formaldehyde, benzene, chromium, carbon tetrachloride, arsenic, chloromethane, and nickel were detected in 85% or more of the samples collected at a monitor, while benzo(a)pyrene, bromodichloromethane, trichloroethene, benzo(b)fluoranthene, 1,1,2-trichloroethane, cobalt, and hexachloro-1,3-butadiene were detected in less than 15% of the samples. Chloroform, Tetrachloroethene, and 1,4-dichlorobenzene, on the other hand, had frequencies of detection that ranged between less than 15% up to greater than 50%, depending on the monitor.

The DQO guidance indicates that if a small proportion of samples in a dataset are not detected, these may be replaced with a small number, usually the detection limit divided by 2. Modification of this was done in the initial CATS assessment (a more conservative value, half the quantitation

limit, was used); therefore, no further analysis for frequently detected analytes is warranted. Conversely, assumptions made about how to utilize non-detects in EPC calculations for more infrequently detected chemicals may play a larger role in risk estimates.

As an initial test of the effect of non-detects on the EPCs for these more infrequently detected chemicals (i.e., chemicals with frequencies of detection <85%), the straight arithmetic means of the untransformed datasets were calculated with all non-detects set equal to zero. This should provide reasonable estimates of lower bound EPCs (i.e., a least conservative average value). When the risks and hazards were recalculated with these new numbers, all chemicals, with the exception of cobalt and tetrachloroethene, were found to equal or exceed an HQ of one (for cobalt) or a risk of $1x10^{-6}$ (for tetrachloroethene) for at least one monitor (see Tables 4.36 through 4.41 for this evaluation). It was thus concluded that the risks posed by most of the infrequently detected chemicals are likely present at a level of potential concern in at least some areas of Chattanooga. As such, the influence of censored data for these chemicals was not evaluated further and additional review was focused on only cobalt and tetrachloroethene.

For cobalt, this chemical was only detected in two samples over the course of the entire sampling period (in one sample at the River Park Site and in one sample at the East Brainerd Fire Station). The non-cancer hazard associated with cobalt was calculated to be an HQ of one for both locations for a highly exposed child receptor. Given this very low frequency of detection, and low apparent hazard, further evaluation of this chemical should probably await a determination of whether a credible source of cobalt can be identified. Confirmation samples for cobalt would also aid in clarifying whether this chemical is indeed present at levels that warrant further attention.

Unlike cobalt, tetrachloroethene was detected at every monitoring location at frequencies that ranged from seldom [*e.g.*, 1 out of 27 samples (4%) at the Cellular One Tower site] to relatively high [*e.g.*, 10 out of 29 samples (34%) at the 20th Street Fire Station]. At the other four monitoring locations, the frequency of detection ranged between 4% and 34% (*i.e.*, 13% at Emma Wheeler Homes, 21% at the Bethlehem Community Center, 17% at the East Brainerd Fire Station, and 26 % at the River Park Site). Given the common use of tetrachloroethene, such observations are not surprising — tetrachloroethene is probably often present at low levels, and the frequency of detection is simply indicative of the limitations of the analytical method.

To evaluate the potential impact of the relatively high number of non-detects on the tetrachloroethene EPC estimate, a maximum likelihood analysis was performed to obtain a better estimate of the average ambient air concentration of tetrachloroethene and the 95% UCL on the average. Maximum likelihood analysis is a statistical evaluation method used to estimate parameters that characterize a distribution by maximizing the probability or likelihood that the estimated parameter would produce the observed data. In this case, a likelihood function was defined that was proportional to the probability that a given estimate of the average tetrachloroethene concentration would produce the observed tetrachloroethene ambient air data. The likelihood function assumed the data were lognormally distributed. The likelihood function

was maximized to determine the most likely average concentration of tetrachloroethene in ambient air. Known characteristics of the likelihood function were then used to estimate the 95% UCL on the average using a technique known as the profile likelihood technique.

Maximum likelihood analysis was used to estimate an average and 95% UCL concentration at each monitoring location.[9] Table 4.42 shows the maximum likelihood estimates of the average and 95% UCL at each of the monitoring locations along with 30-year RME cancer risk estimates. At each of the five monitoring locations examined, 30-year RME cancer risk estimates based on both the maximum likelihood average and 95% UCL exceed 1×10^{-6}. This indicates that, even when more sophisticated statistical analyses are performed on the datasets, the chemical still appears to be present at numerous locations above a specified level (here 1×10^{-6}).

It should be noted that the detected concentrations of tetrachloroethylene at each location are well below the reported quantitation limits. Detected concentrations of tetrachloroethene ranged from 0.22 $\mu g/m^3$ to 0.7 $\mu g/m^3$ and were all estimated (*i.e.*, reported as J-values) below the quantitation limit, while quantitation limits ranged from 1.9 $\mu g/m^3$ to 5.6 $\mu g/m^3$. Given that these quantitation limits are relatively high with respect to the detected J-values, additional data with lower detection and quantitation limits would allow for better characterization of the tetrachloroethene EPC.

[9]The maximum likelihood estimation was not performed on the Cellular One Tower site data because tetrachloroethene was only detected in one sample.

Chapter 5 Human health acute effects analysis

The assessment presented in Chapter 4 evaluated the potential health risks resulting from long-term (chronic) exposure to relatively low levels of airborne toxicants. The risk analyses used an estimate of the annual average concentrations to which residents are exposed to make these risk estimates since the annual average is more representative of long term exposures than the individual sampling events from which the average is derived.

However, the health effects that persons may experience due to short-term (acute) exposures to elevated levels of 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 chemical that produces an increase in cancer rates after exposure to low concentrations for a long period of time (a chronic effect) might also cause immediate and severe eye irritation if present at high levels for a short period of time (an acute effect).

This portion of the risk assessment will evaluate the potential for adverse effects from acute exposures using the same monitoring results as used in the chronic evaluation. In this evaluation, however, samples will be evaluated on an individual basis rather than in a combined fashion as was done for the chronic human health assessment. Performing the analysis in this way will help avert the potential to "average out" spikes in concentration as is done in the chronic exposure analysis.

5.1 Introduction

Hazardous substances are routinely released to the environment as the result of predictable (planned) continuous and intermittent releases from point and mobile sources. However, facility and other non-facility releases (e.g., mobile source emissions from cars and trucks) may fluctuate considerably, with the result that daily and hourly maximum and minimum concentrations can vary widely. Due to these changing releases, surrounding populations may, at times, be exposed to relatively high concentrations of airborne toxicants even though the annual average of all these fluctuating concentrations may be relatively low. Since the health effects resulting from short term exposure to high concentration spikes can be considerably different from the health effects seen for exposures to relatively lower doses over a long period of time, it is necessary to evaluate the potential for acute health effects separately from chronic health effects. For the purpose of this evaluation, an acute exposure was defined as an independent, intermittent exposure event occurring up to no more than 24-hours in duration.

It should be noted that emergency or accidental releases may also present acute health risks to surrounding populations. However, this type of exposure is likely to be rare and to occur at much higher concentrations than would be expected due to the day-to-day fluctuations of sources in the CATS study area. As such, emergency/accidental releases were not explicitly evaluated in this assessment.

This evaluation focuses on the potential for acute health effects to occur from short-term exposure to elevated levels of airborne contaminants within the CATS study area. Specifically, the data collected from the six sampler locations were assessed for the potential to cause non-carcinogenic acute health effects for the locations and times sampled.

This evaluation does not consider carcinogenic effects resulting from acute exposures given the current limitations with such evaluations. For most chemicals, there are insufficient data to reasonably support these estimations in view of the many uncertainties related to extrapolation from long-term to short-term exposures and other factors such as mechanism of action, metabolism, promotional activity, and threshold effects (U.S. EPA, 1993b). Instead, the potential for chemicals detected in the CATS study area to result in cancer outcomes has been evaluated in the chronic exposure risk assessment (Chapter 4).

5.2 Methodology used in the acute evaluation

EPA has developed limited information for evaluating routine acute exposures to numerous potential airborne contaminants by the general population. In the absence of such information, this assessment relies on the data collected in the CATS monitoring program along with a reasonably conservative methodology that uses existing health-based criteria to evaluate the potential for acute adverse effects to occur in area residents. The data used in this evaluation and the methodology for evaluating these concentrations are described below.

5.2.1 General approach

To determine the potential for adverse health effects to occur from short-term exposure to elevated levels of airborne contaminants, each sample result collected in this study was compared to an acute health-based screening value, if available. Because this is a screening-level evaluation of potential acute health effects, it was assumed that if a contaminant exceeded the screening criteria then there was a potential for adverse human health effects.

5.2.2 Data used in the analysis

As discussed in Chapter 3, samples of ambient air were collected from the CATS study area during 1998 and 1999 in order to evaluate the potential for health effects to occur in area residents through inhalation of airborne toxicants. The data collected were captured by samplers located at 6 monitoring stations throughout the study area. These stationary samplers collected 24-hour composite samples approximately every twelve days. Duplicate samples at the Bethlehem Community Center were not averaged for this analysis.

5.2.3 Frequency and length of exposure

U.S. EPA views intermittent exposure as that lasting less than 24-hours and occurring no more frequently than monthly (U.S. EPA, 1994). This assumes that an acute exposure is at least 10 times higher than a monthly average and that individual exposures are independent of one another. U.S. EPA has also pointed out that very few chemicals will have enough data to determine a safe time period for an acute exposure. As such, each sample collected during this investigation was evaluated as a single, independent exposure. Samples collected were evaluated with the assumption that a person would be exposed to the detected concentration for no more than 24-hours.

5.2.4 Sources of acute health-based screening values

With few exceptions, there is no simple or widely accepted method for estimating the risks of routine short-term exposures to elevated concentrations of most toxic chemicals found in ambient air samples. As such, there are no uniformly accepted short-term air action levels for the majority of emissions from facilities and other common emission sources such as area sources. Instead, concentrations of chemicals protective of acute exposures have been established using a variety of differing methodologies. For example, occupational exposure limits are sometimes used to develop exposure values (acute and chronic) for protection of the general public. Such values are usually generated by dividing the occupational number by safety factors that can range from 4.2 to as great as 1,000 or more. The concept behind such safety factors is to account for differences between workers, for which the standards were developed, and residents, for which they were not (U.S. EPA, 1993b).

Some of the more widely used sources of toxicity values for acute effects analysis include (OEHHA, 1999):

State Guidelines

1. California Environmental Protection Agency Acute Reference Exposure Levels (California RELS) for Airborne Toxicants

Occupational Guidelines

1. American Conference of Governmental Industrial Hygienists (ACGIH) Threshold Limit Value-Time Weighted Average (TLV-TWA)

2. ACGIH Short-Term Exposure Limits (STELs)

3. Occupational Safety and Health Administration (OSHA) Permissible Exposure Limits (PELs)

4. National Institute of Occupational Safety and Health (NIOSH) Immediately Dangerous to Life and Health (IDLH) Values

5. ACGIH Threshold Limit Values — Ceiling (TLV-C)

6. NIOSH Recommended Exposure Limits (RELS)

Emergency Guidelines

1. National Academy of Science (NAS) Emergency Exposure Guidance Levels (EEGLs)

2. American Industrial Hygiene Association Emergency Response Planning Guidelines (ERPGs)

3. NAS Short-term Public Emergency Guidance Levels (SPEGLs)

4. U.S. EPA Acute Emergency Guidance Levels (AEGLs)

5. National Research Council Community Emergency Exposure Levels (CEELs)

Other Guidelines

1. U.S. EPA Air Pollution Warning Levels

2. U.S. EPA Levels of Concern

3. ATSDR Minimal Risk Levels (MRLs) for acute exposure

Table 5.1 provides a complete listing of the sources of airborne screening values that were considered for use in this evaluation. Included is a brief description of the intended use of each of these criteria and a summary of acceptance or rejection of the values for use in evaluating the CATS monitoring data.

5.2.5 Discussion of acute health-based screening values

Unlike the screening values developed to evaluate chronic exposures, only a limited number of benchmarks for acute inhalation exposures have been developed at this time for non-emergency acute exposures. The following sections contain brief descriptions of the acute screening values which were utilized in the acute exposure evaluation.

5.2.5.1 California Reference Exposure Levels

The California Environmental Protection Agency's (Cal EPA) Office of Environmental Health Hazard Assessment (OEHHA) has developed acute Reference Exposure Levels (RELs) for evaluating exposure to a limited number of airborne toxicants. OEHHA defines the acute REL as "an exposure that is not likely to cause adverse effects in a human population, including sensitive subgroups, exposed to that concentration for one hour on an intermittent basis" (OEHHA, 1998)[1]. RELs are based upon the most sensitive, relevant, adverse health effect reported in the medical and toxicological literature. RELs are designed to protect the most sensitive individuals in the population by the inclusion of margins of safety. Since margins of safety are incorporated to address data gaps and uncertainties, exceeding the REL does not automatically indicate an adverse health impact.

RELS were developed by first prioritizing chemicals to be evaluated. A literature search was then conducted of the selected contaminants. As part of the literature review, existing standards were identified and evaluated. If the existing standard was determined to be acceptable, it was adopted as the REL. However, most of the available standards were determined to be unacceptable, and the RELS were developed *de novo* from the medical and toxicological literature.

RELS were developed from the primary literature by selecting the best studies, with emphasis on those using human data. These studies were used to estimate a threshold level for no affect (a no observed adverse effect level or NOAEL). A NOAEL can be defined as "an exposure level with no biologically and/or statistically significant increase in the frequency or severity of adverse effects among an exposed population relative to a control group" (OEHHA 1998). Adjustments were made to the NOAELs to allow for comparison to one-hour exposures. Uncertainties in the data were then accounted for by applying safety factors to ensure that the REL values were

[1] Some acute RELs are derived for longer exposure periods, up to seven hours duration in some cases.

sufficiently conservative. For example, if the NOAEL is based upon a study involving animals, an uncertainty factor of 10 may be applied to account for the uncertainty of extrapolating toxicity data from the test species to humans. Similarly, an "interspecies" safety factor is typically used to account for the variability in sensitivity to a toxicant within the general human population. The acute exposure NOAEL is divided by these uncertainty factors to arrive at a derived acute screening value.

The RELS developed by the OEHHA are based upon one-hour exposure durations. This exposure duration was selected in order to be comparable to the sampling and modeling requirements of California regulations.

The available RELS have been developed specifically to evaluate the potential for human health effects from acute exposure of the general population, including sensitive individuals, to routine industrial air emissions. Therefore, the RELS developed by OEHHA were determined to be acceptable for use in this evaluation.

5.2.5.2 ATSDR Acute Minimum Risk Levels

The Agency for Toxic Substances and Disease Registry (ATSDR) has established airborne contaminant screening concentrations for acute exposure that are similar to the EPA's Reference Concentrations (RfCs) for chronic exposure. The values are derived by extensive review and evaluation of chemical-specific toxicological studies. Similar to CAL RELs, safety (or uncertainty) factors are applied to airborne contaminant concentrations from acute toxicology studies in which NOAELs have been established. Safety factors are applied to the NOAEL to minimize or correct for uncertainties in the development of the NOAEL values.

The screening concentrations derived by the ATSDR in this manner are called "Minimum Risk Levels" or MRLs, which are published in ATSDR's chemical-specific Toxicological Profiles. MRLs are derived for acute (1-14 days), intermediate (15-364 days), and chronic (365 days and longer). Only acute MRLs were selected for this screening level evaluation.

5.2.5.3 EPA Acute Exposure Guideline Levels (AEGLs)

The National Advisory Committee for Acute Exposure Guideline Levels for Hazardous Substances is developing AEGLs for short-term hazardous chemical exposure information (29 CFR 1910). These levels have been established for one time only exposures during emergency situations. Three levels of AEGLs have been developed. They are routinely used in evaluations involving the general public.

AEGL values are defined as follows:

AEGL-1 This is the airborne concentration (expressed as ppm or mg/m^3) of a substance at or above which it is predicted that the general population including "susceptible", but excluding "hyper susceptible" individuals, could experience notable discomfort. Airborne concentrations below AEGL-1 represent exposure levels that could produce mild odor, taste, or other sensory irritations.

AEGL-2 AEGL-2 is the airborne concentration (expressed as ppm or mg/m^3) of a substance at or above which it is predicted that the general population, including "susceptible", but excluding "hyper susceptible" individuals, could experience irreversible or other serious, long-lasting effects or impaired ability to escape. Airborne concentrations below the AEGL-2, but at or above AEGL-1, represent exposure levels that may cause notable discomfort.

AEGL-3 AEGL-3 is the airborne concentration (expressed as ppm or mg/m^3) of a substance at or above which it is predicted that the general population, including "susceptible", but excluding "hyper susceptible" individuals, could experience life-threatening effects or death. Airborne concentrations below AEGL-3, but at or above AEGL-2, represent exposure levels that may cause irreversible or other serious, long lasting effects or impair a person's ability to escape.

AEGL's have been established for four different exposure periods of 30 minutes, 1 hour, 4 hours and 8 hours for some chemicals. Where applicable, AEGL-1, AEGL-2 and AEGL-3 values have been used for contaminants of concern.

5.2.5.4 AIHA Emergency Response Planning Guidelines (ERPGs)

ERPGs are developed by the American Industrial Hygiene Association's Emergency Response Planning Committee (AIHA, 1999). Similar to the AEGLs, ERPGs are useful as guidelines for emergency planning involving release of chemical materials.

ERPG-1 The maximum concentration in air below which it is believed nearly all individuals could be exposed for up to one hour without experiencing other than mild transient adverse health effects or perceiving a clearly defined objectionable odor;

ERPG-2 The maximum concentration in air below which it is believed that nearly all individuals could be exposed for up to one hour without experiencing or developing irreversible or other serious health effects or symptoms that could impair their abilities to take protective action;

ERPG-3 The maximum concentration in air below which it is believed nearly all individuals could be exposed for up to one hour without experiencing or developing life-threatening health effects.

5.2.5.5 DOE Temporary Emergency Exposure Limits (TEELs)

TEELs are recently developed values based on statistical extrapolations from the literature on the toxicity of each chemical (http://www.scapa.bnl.gov). TEELs are interim values developed for use in the absence of ERPGs and are considered to be stop gap values to be used in emergency exposure situations.

DOE Temporary Emergency Exposure Limits (TEELs) are defined as follows:

TEEL-0 The threshold concentration below which most people will experience no appreciable risk of health effects;

TEEL-1 The maximum concentration in air below which it is believed nearly all individuals could be exposed without experiencing other than mild transient adverse health effects or perceiving a clearly defined objectionable odor.

TEEL-2 The maximum concentration in air below which it is believed nearly all individuals could be exposed without experiencing or developing irreversible or other serious health effects or symptoms that could impair their abilities to take protective action;

TEEL-3 The maximum concentration in air below which it is believed nearly all individuals could be exposed without experiencing or developing life-threatening health effects.

TEELs have been established utilizing existing toxicity data published for ERPGs, AEGLs and any other published toxicity data. Published data pertaining to TEELs recommends that TEELs be compared to peak 15-minute time weighted average concentrations.

5.2.5.6 NRC Community Emergency Exposure Levels (CEEL) & Short-Term Public Exposure Guidance Levels (SPEGL)

CEEL guidelines have been developed by the National Research Council Committee on Toxicology. The frame work has been established for developing these emergency response standards, but to date, no CEELs have been established (National Research Council, 1993).

The SPEGLs were also developed by the National Research Council Committee on Toxicology as public exposure guidelines for civilian populations near military bases. SPEGLs are not currently available for any contaminants detected in the CATS study.

5.2.6 Time adjustment of acute screening values

The data collected during the air sampling in Chattanooga consist of 24 -hour samples. For some acute screening values, the exposure time for which the value was derived may not match the 24-hour duration of the samples. In such cases it was necessary to adjust the screening values to match the sampling duration.

Haber's Law provides a method for extrapolating a contaminant concentration for a given time period to an equivalent concentration over a different time period. As described by ten Berge *et al.* (1986), Haber's Law states that the product of the concentration (C) and time of exposure (T) required to produce a specific physiologic effect is equal to a constant level or severity of response (K), or:

$$(C)(T) = K$$

When the duration of the experimental exposure concentration is different from the desired exposure duration, a modification of Haber's Law may be used to obtain an acute exposure level that is of a different duration from the experimental conditions (OEHHA, 1999):

$$(C^n)(T) = K$$

Where *n* describes the relationship between concentration and exposure duration in determining toxicity.

For example, in cases where *n* is equal to one, the toxicity of the chemical is equally dependent on concentration and exposure duration. However, when the value of *n* is less than one, the duration is a greater factor in determining toxicity than the concentration. Values of *n* that are greater than one indicate that the concentration is a greater determinant of toxicity than exposure duration.

OEHHA default values were derived by examining a range of chemicals for which specific *n* values were available. It was concluded that when extrapolating from a shorter exposure duration to a longer duration, a default value of one should be selected for *n* (OEHHA, 1999). It was not necessary to extrapolate from a longer to a shorter exposure duration for any chemicals in the risk assessment. For all of the chemicals evaluated in this study, default *n* values developed by OEHHA were used.

5.2.7 Screening value hierarchy

For comparison to 24-hour values, the following hierarchy was used. This hierarchy is based on discussions with an expert in acute toxicology at EPA's National Center for Environmental Assessment.

1. ATSDR Acute MRLs

2. CAL RELS adjusted to a 24-hour averaging period using Haber's Law for all chemicals regardless of evaluation endpoints

3. EPA AEGLs (using AEGL-1 which is the LOAEL) adjusted to a 24-hour averaging period using Haber's Law

4. AIHA ERPG's-1 and SPEGLs adjusted to a 24-hour averaging period using Haber's Law

This hierarchy should be read as follows: For evaluation of 24-hour samples, first attempt to use ATSDR Acute MRLs. If no MRL is available, then use CAL RELs adjusted to a 24-hour averaging period using Haber's Law. If there is no MRL or CAL REL for a chemical, then use the 8 hour AEGL-1 adjusted to a 24-hour averaging period using Haber's Law. Finally, if no AEGL is available, use ERPG-1, or SPEGLs adjusted to a 24-hour averaging period using Haber's Law.

DOE Temporary Emergency Exposure Limits (TEELs) were not used to compare to 24-hour samples. TEELs are interim values developed for use in the absence of ERPGs and are considered to be stop gap values to be used in emergency exposure situations. In addition, TEELs are recommended only for a 15 minute averaging time. Therefore, they were considered to be inappropriate for comparison to 24-hour data.

ATSDR Acute MRLs were considered to be the screening value of choice. ATSDR derives benchmark values for airborne chemicals that are protective of exposures lasting from 24-hours to 2 weeks. Since the minimum of the averaging time range matches the averaging time of the samples collected in the monitoring study, they were used preferentially for screening samples for acute effects.

The acute CAL RELs are derived benchmarks designed to be protective of residential exposure scenarios from routine emissions from industrial facilities. However, they are generally based on a 1-hour averaging time (sometimes longer) and must be converted to a 24-hour averaging time to be comparable to the samples evaluated in this acute evaluation. As such, they were given second priority.

EPA AEGLs are emergency planning guidelines and were given a higher priority than other emergency related values because they are more recent values. AEGLs for some chemicals are available for time periods longer than one hour. Where an AEGL was available for a longer time period, this value was used as the potential basis for calculating screening values to be compared with 24-hour values.

Based on these definitions AEGL-1 or ERPG-1 may be considered to represent a Lowest Observed Adverse Effect Level or LOAEL. To maintain consistency among the various potential screening values and as a conservative approach, LOAEL-equivalent values were used for all comparisons.

5.3 Summary and discussion of screening results

The detections for each sampling event were compared to their respective health-based screening levels for 24-hour exposures. Screening levels were available in the literature for 24 of the 86 chemicals of potential concern (28%). A complete list of the 24-hour acute screening values can be found in Table 5.2. When the maximum detected concentrations of a contaminant exceeded the selected acute exposure screening value, a ratio of the detected concentration to its screening value was calculated. This information is intended to quantify the magnitude of the exceedance. It should not be assumed that the magnitude of the potential acute effect, if any, from exposure to a chemical which exceeds its acute screening criterion is directly correlated with the screening value exceedance ratio. The equation for the calculated "acute hazard ratio" is presented below:

$$HR_{acute} = \frac{[chemical]}{[benchmark]_{acute}}$$

where [*chemical*] is the concentration of the chemical in outdoor air and $[benchmark]_{acute}$ is the acute exposure screening value (in the same units).

All of the maximum detected concentrations of contaminants at each of the six sampling locations were less than the identified acute screening levels.

5.4 Uncertainties

There are a number of contaminants that were detected for which there were no appropriate screening values identified. For comparison to 24-hour concentrations, 62 out of 86 chemicals of potential concern (72%) lacked acute screening values. Therefore, the potential human health effects of most contaminants could not be evaluated. This limitation in the screening methodology may have had the effect of underestimating the potential acute human health effects of the detected contaminants. Reducing this uncertainty will require either the development of additional toxicity values by the sources used in this assessment, the development of a new hierarchy of toxicity values, or the development of a new approach to assessing acute risks posed to residential receptors by routine emissions.

In acute toxicology experiments, the study design usually involves exposures of short duration to an otherwise unexposed animal. However, real world acute exposures typically occur intermittently, rather than as rare events in a lifetime. Thus, the typical ambient exposure scenario is not reflected in the standard acute toxicology experimental design. Based upon this limitation of experimental design, the possibility of cumulative effects from intermittent ambient exposures is not typically addressed in the development of the acute screening values. Therefore, the screening methodology used in this evaluation may underestimate the potential for human health risks. Reducing this type of uncertainty will require a more advanced understanding of acute toxicology and receptor exposures.

Haber's Law was used to adjust screening values from short periods of exposure to longer periods of exposure. Uncertainties exist in this procedure from several factors. The exponential "*n*" value has been empirically determined for relatively few chemicals. Therefore, assumptions are made regarding what value of "*n*" to use. The use of Haber's Law also assumes that the log function applied is valid for the chemicals in question. Although Haber's Law has been used extensively in the toxicological literature, uncertainties regarding its applicability are still present. Actual levels of concern may be either greater or less than the screening values calculated using Haber's Law.

The assumption that exposures of interest are independent of other exposures has several uncertainties. First, the possible additive, synergistic, or antagonistic effects of multiple exposures were not evaluated. This may underestimate or overestimate the potential impacts from simultaneous exposure to multiple chemicals depending on the contaminants.

The screening levels used in the acute exposure evaluation were taken from a variety of sources as described in Section 5.2.5. With the exception of CAL RELs, the screening levels were not derived for the explicit purposes of evaluating acute residential exposures from routine industrial emissions. For example, many of the screening values were derived for assessing releases in emergencies. Therefore, the screening values used may not be directly applicable to the potential

acute exposure scenarios presumed in this acute human health exposure analysis. As a result, the use of diverse screening values from multiple literature sources may underestimate or overestimate the potential for human health risks.

While reducing may of the types of uncertainty noted above will require a more advanced understanding of acute toxicology and receptor exposures, it is unlikely that addressing these uncertainties would affect the conclusions of the CATS acute risk assessment for many of the chemicals detected. Most measured ambient air concentrations were far below the available acute screening values. Only the maximum detected concentrations of formaldehyde (68% of the acute screening level), copper (60%), arsenic (29%), and nickel (15%) were within a factor of 10 of the screening level. All other maximum contaminant concentrations were a factor of 10 to 50,000 less than the acute screening level.

Chapter 6 Conclusions

This report presents the results of the human health risk assessment and acute effects evaluation performed using the air monitoring data developed for the CATS between November 12, 1998 and October 29, 1999. The air monitoring risk assessment focuses only on direct exposure to airborne contaminants through the inhalation pathway.

Sampling efforts between November 12, 1998 and October 29, 1999 included a year-long air monitoring program utilizing stationary air samplers, collecting approximately 30 composite samples for each chemical at each monitoring site over a 24-hour period every 12 days. Samples were analyzed for volatile organic compounds, semivolatile organic compounds, formaldehyde, and metals.

Air sampling results were used in two human health evaluations. Results were integrated into a quantitative human health risk assessment which focused on potential long-term, or chronic, impacts to human health from exposure to airborne contaminants. Air sampling results were also evaluated for potential acute impacts to human health through a screening-level comparison using established or estimated acute screening standards.

In the chronic human health risk assessment, hazard quotients for individual chemicals in excess of one for the child receptor were obtained for all sampling locations except the Cellular One Tower Site. Hazard quotients for manganese, formaldehyde, and cobalt all exceeded one at one or more sampling locations. HIs ranged from four to eight for the child receptor and two to three for the adult receptor (high end exposures).

For all locations, calculated chronic carcinogenic risks for at least one chemical were found to exceed the risk level of 1×10^{-6}. This risk level was exceeded at all sample locations by formaldehyde, chromium, chloroform, benzene, carbon tetrachloride, arsenic, chloromethane, and tetrachloroethylene. This risk level was also exceeded at several locations by benzo(a)pyrene, bromodichloromethane, 1,4-dichlorobenzene, and nickel. Total cancer risk ranged from 7×10^{-5} to 1×10^{-4} for a 30 year resident (high end exposure).

To evaluate potential impacts from acute exposure to airborne contaminants, sample data collected from stationary monitoring stations were compared to the selected chemical-specific acute screening criteria. Of all of the detected constituents from the six sample locations, no maximum measured concentration was found to exceed an acute screening level. However, acute

screening levels were available for only a relatively small portion of the chemicals detected in the monitoring study.

As for any risk assessment, it is important to consider sources of uncertainty that may result in an overestimate or underestimate of risks when evaluating the conclusions of the risk assessment. In general, an effort was made to overestimate risks associated with air toxics. The use of RME exposure parameters, 95% UCL EPCs, and conservatively derived toxicological data all contribute to overestimating risks. However, additional elements of uncertainty, such as chemicals not included in the analysis, exposure pathways in addition to direct inhalation of ambient air, and the exclusion of TICs from the analysis could result in underestimating risks.

Table 6.1 presents the results of the risk characterization. The table summarizes all chemicals with hazard quotients greater than or equal to one for a child receptor (high end exposure) and incremental risks of cancer greater than or equal to 1×10^{-6} for a 30-year resident (high end exposure) at each monitoring location.

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Chapter 1 Tables

Table 1.1 Summary of risk estimates at the six monitoring locations for reasonable maximum exposure

Location	Hazard quotients at or above 1 (child receptor)		Cancer risks at or above $1x10^{-6}$ (30 year exposure)	
Emma Wheeler Homes	MANGANESE	3	FORMALDEHYDE	6E-05
	FORMALDEHYDE	2	CHLOROFORM	1E-05
			BENZENE	1E-05
			CHROMIUM	9E-06
			BENZO-A-PYRENE	9E-06
			CARBON TETRACHLORIDE	7E-06
			BROMODICHLOROMETHANE	5E-06
			1,4-DICHLOROBENZENE	4E-06
			ARSENIC	4E-06
			CHLOROMETHANE	3E-06
			NICKEL	3E-06
			TETRACHLOROETHENE	2E-06
			TRICHLOROETHENE	1E-06
			BENZO(B)FLUORANTHENE	1E-06
Bethlehem Community Center	MANGANESE	2	FORMALDEHYDE	2E-05
			BENZENE	2E-05
			CHLOROFORM	2E-05
			CHROMIUM	1E-05
			CARBON TETRACHLORIDE	7E-06
			1,4-DICHLOROBENZENE	7E-06
			BENZO-A-PYRENE	6E-06
			ARSENIC	4E-06
			CHLOROMETHANE	3E-06
			NICKEL	2E-06
			TETRACHLOROETHENE	2E-06
20th Street Fire Station	MANGANESE	3	FORMALDEHYDE	2E-05
			CHROMIUM	1E-05
			BENZENE	1E-05
			CHLOROFORM	1E-05
			CARBON TETRACHLORIDE	7E-06
			BROMODICHLOROMETHANE	6E-06
			ARSENIC	4E-06
			CHLOROMETHANE	3E-06
			1,1,2-TRICHLOROETHANE	3E-06
			BENZO-A-PYRENE	2E-06
			NICKEL	2E-06
			TETRACHLOROETHENE	2E-06
			1,4-DICHLOROBENZENE	2E-06

Table 1.1 Summary of risk estimates at the six monitoring locations for reasonable maximum exposure

Location	Hazard quotients at or above 1 (child receptor)		Cancer risks at or above $1x10^{-6}$ (30 year exposure)	
Cellular One Tower Site	None		FORMALDEHYDE	2E-05
			CHROMIUM	1E-05
			BENZENE	9E-06
			CARBON TETRACHLORIDE	6E-06
			ARSENIC	6E-06
			BROMODICHLOROMETHANE	5E-06
			BENZO-A-PYRENE	3E-06
			CHLOROFORM	3E-06
			CHLOROMETHANE	3E-06
			TETRACHLOROETHENE	1E-06
East Brainerd Fire Station	COBALT	1	FORMALDEHYDE	2E-05
			CHROMIUM	1E-05
			BENZENE	9E-06
			CARBON TETRACHLORIDE	7E-06
			BROMODICHLOROMETHANE	6E-06
			HEXACHLORO-1,3-BUTADIENE	4E-06
			CHLOROFORM	3E-06
			ARSENIC	3E-06
			CHLOROMETHANE	3E-06
			1,4-DICHLOROBENZENE	2E-06
			TETRACHLOROETHENE	1E-06
River Park Site	COBALT	1	FORMALDEHYDE	2E-05
			CHROMIUM	1E-05
			BENZENE	8E-06
			CARBON TETRACHLORIDE	7E-06
			BROMODICHLOROMETHANE	5E-06
			ARSENIC	3E-06
			CHLOROFORM	3E-06
			CHLOROMETHANE	3E-06
			NICKEL	2E-06
			TETRACHLOROETHENE	2E-06

Chapter 2 Tables

Table 2.1 CATS monitoring locations

Site identification number	Name and location in Chattanooga	Rationale for inclusion of monitoring site
1	Emma Wheeler Homes 34°59.38' N, 85°18.57' W Located at Wilson Road and 51st St.	Potential maximum exposure site.
2 & 3 (co-located)	Bethlehem Community Center 35°00.33' N, 85°18.77' W Located at 200 West 38th St. and Kirkland Ave.	Downwind site of the Piney Woods/Alton Park industrial area. It is also a community impact site. It is located adjacent to Spencer-McCallie Homes (currently being demolished and rebuilt).
4	20th St Fire Station 35°01.93' N, 85°18.58' W Located on 20th St. near Interstate 24	Directly downwind of Foundry Row (U.S. Pipe and Foundry, Wheland, and two smaller foundries)
5	Cellular One Tower Site 35°00.47' N, 85°12.20' W Located at end of shopping center road at Cellular Tower Rd.	Located near the intersection where I-75 and I-24 split. It will measure the impact of mobile source emissions at the location of highest traffic flow in the city.
6	East Brainerd Fire Station #21 35°00.60' N, 85°9.33' W Located on East Brainerd Road on ridge out of the valley	Located in a location that will aid in assessing out of state transport of pollutants from sources in Georgia. The site is located 2 miles from the Georgia state line.
7	River Park Site 35°05.96' N, 85°14.95' W Located on Amnicola Highway, near River Industrial Park	Located near the second largest industrial complex in Chattanooga to assess exposure potential to population in this area of the city.

Chapter 4 Tables

Chapter 5 Tables

Table 5.1 Sources of acute exposure screening values evaluated

Agency or organization	Screening value	Intended use	Summary of Acceptance or Rejection of Screening Value
ATSDR	Acute Minimum Risk Levels (MRLs)	Created as a screening tool for the human health assessment of acute airborne concentrations of contaminants.	Accepted for use
Cal EPA, OEHHA	Reference Exposure Levels (RELs)	Used to evaluate a limited selection of airborne contaminants. Based on the most sensitive, relevant adverse health effect reported in toxicological literature.	Accepted for use
U.S. EPA	Acute Exposure Guidance Levels (AEGLs)	AEGLs were developed for evaluation of emergency releases.	Accepted for use
AIHA	Emergency Response Planning Guidelines (ERPGs)	ERPGs are used as guidelines for emergency planning involving release of chemical material.	Accepted for use
DOE	Temporary Emergency Exposure Limits (TEELs)	TEELs were developed for evaluation of emergency releases.	Determined to be inadequate for evaluation of 24-hour exposure
NRC	Community Emergency Exposure Levels (CEELs)	CEELs present a blueprint for developing emergency standards for the general public.	Determined to be inadequate
NRC	Short-Term Public Exposure Guidance Levels (SPEGLs)	SPEGLs are public exposure guidelines for civilian populations near military bases.	SPEGLs are not currently available for any contaminants detected in the CATS study.
OSHA	Permissible Exposure Limits (PELs)	Enforceable standards for occupational exposure (industrial workers) - carry the weight of law.	Determined to be inadequate
NIOSH	Recommended Exposure Levels (RELs), Short Term Exposure Limits (STELs)	Recommended exposure limits for occupational exposures - not enforceable.	Determined to be inadequate
ACGIH	Threshold Limit Values (TLVs), Short Term Exposure Limits (STELs)	Recommended exposure limits for occupational exposure derived through annual consensus of non-governmental organizations - not enforceable.	Determined to be inadequate

Notes:
ATSDR is the Agency for Toxic Substances and Disease Registry.
Cal EPA, OEHHA is the California EPA Office of Environmental Health Hazard Assessment.
AIHA is the American Industrial Hygiene Association.
DOE is the Department of Energy.
NRC is the National Research Council.
OSHA is the Occupational Safety and Health Administration.
NIOSH is the National Institute of Occupational Safety and Health.
ACGIH is the American Conference of Governmental Industrial Hygienists.

Table 5.2 Acute inhalation toxicity data for 24-hour exposure and maximum detected concentrations

Chemical of Potential Concern	CAS Number	Maximum detected concentration (mg/m^3)	24-hr Acute toxicity data (mg/m^3)	Reference for acute toxicity data
(3-AND/OR 4-)METHYLPHENOL	106-44-5	9.0e-05	NA	NA
(M- AND/OR P-)XYLENE	108-38-3	1.6e-02	9.2E-01	Cal EPA
1,1,1-TRICHLOROETHANE	71-55-6	2.3e-03	1.1E+01	ATSDR
1,1,2-TRICHLOROETHANE	79-00-5	3.2e-04	NA	NA
1,2,4-TRICHLOROBENZENE	120-82-1	5.0e-04	NA	NA
1,2,4-TRIMETHYLBENZENE	95-63-6	1.8e-02	NA	NA
1,2-DICHLOROBENZENE	95-50-1	2.8e-04	NA	NA
1,3,5-TRIMETHYLBENZENE	108-67-8	9.8e-04	NA	NA
1,4-DICHLOROBENZENE	106-46-7	1.3e-03	4.8E+00	ATSDR
2,4-DIMETHYLPHENOL	105-67-9	2.4e-05	NA	NA
2,4-DINITROTOLUENE	121-14-2	4.1e-06	NA	NA
2-METHYLNAPHTHALENE	91-57-6	4.4e-04	NA	NA
2-METHYLPHENOL	95-48-7	3.6e-05	NA	NA
2-NITROANILINE	88-74-4	1.9e-05	NA	NA
2-NITROPHENOL	88-75-5	8.7e-05	NA	NA
4-CHLOROANILINE	106-47-8	4.8e-04	NA	NA
4-NITROPHENOL	100-02-7	6.0e-05	NA	NA
ACENAPHTHENE	83-32-9	1.4e-04	NA	NA
ACENAPHTHYLENE	208-96-8	2.8e-05	NA	NA
ACETONE	67-64-1	6.0e-02	6.2E+01	ATSDR
ANTHRACENE	120-12-7	7.0e-06	NA	NA
ANTIMONY	7440-36-0	1.3e-05	NA	NA
ARSENIC	7440-38-2	9.2e-06	3.2E-05	Cal EPA
BARIUM	7440-39-3	7.8e-05	NA	NA
BENZENE	71-43-2	9.6e-03	1.6E-01	ATSDR
BENZO(A)ANTHRACENE	56-55-3	5.5e-06	NA	NA
BENZO(B)FLUORANTHENE	205-99-2	1.9e-05	NA	NA
BENZO(GHI)PERYLENE	191-24-2	1.8e-05	NA	NA
BENZO(K)FLUORANTHENE	207-08-9	1.3e-05	NA	NA
BENZO-A-PYRENE	50-32-8	1.4e-05	NA	NA
BERYLLIUM[1]	7440-41-7	5.0e-08	1.0E-03	AIHA, ERPG-2
BIS(2-ETHYLHEXYL) PHTHALATE	117-81-7	2.6e-04	NA	NA
BROMODICHLOROMETHANE	75-27-4	6.2e-04	NA	NA
BROMOMETHANE	74-83-9	1.0e-03	1.9E-01	ATSDR
CADMIUM	7440-43-9	2.7e-06	NA	NA
CARBON DISULFIDE	75-15-0	4.4e-02	1.6E+00	Cal EPA
CARBON TETRACHLORIDE	56-23-5	1.1e-03	1.3E+00	ATSDR
CHLOROFORM	67-66-3	2.9e-03	4.9E-01	ATSDR
CHLOROMETHANE	74-87-3	1.1e-02	1.0E+00	ATSDR
CHROMIUM	7440-47-3	3.2e-05	NA	NA
CHRYSENE	218-01-9	6.2e-05	NA	NA
COBALT	7440-48-4	4.4e-06	NA	NA
COPPER	7440-50-8	2.5e-03	4.2E-03	Cal EPA
DIBENZOFURAN	132-64-9	8.4e-05	NA	NA
DICHLORODIFLUOROMETHANE	75-71-8	2.6e-02	NA	NA
DIETHYL PHTHALATE	84-66-2	6.6e-05	NA	NA
DI-N-BUTYLPHTHALATE	84-74-2	5.3e-05	NA	NA
ETHYL BENZENE	100-41-4	5.2e-03	NA	NA
FLUORANTHENE	206-44-0	5.0e-05	NA	NA
FLUORENE	86-73-7	6.6e-05	NA	NA

Table 5.2 Acute inhalation toxicity data for 24-hour exposure and maximum detected concentrations

Chemical of Potential Concern	CAS Number	Maximum detected concentration (mg/m^3)	24-hr Acute toxicity data (mg/m^3)	Reference for acute toxicity data
FORMALDEHYDE	50-00-0	3.4e-02	4.9E-02	ATSDR
HEXACHLORO-1,3-BUTADIENE	87-68-3	2.9e-04	1.3E+00	AIHA, ERPG-1
INDENO (1,2,3-CD) PYRENE	193-39-5	1.1e-05	NA	NA
IRON	7439-89-6	3.5e-03	NA	NA
ISOPHORONE	78-59-1	5.9e-06	NA	NA
ISOPROPYLBENZENE	98-82-8	3.7e-04	NA	NA
LEAD	7439-92-1	1.4e-04	NA	NA
MAGNESIUM	7439-95-4	1.4e-03	NA	NA
MANGANESE	7439-96-5	2.3e-04	NA	NA
METHYL BUTYL KETONE	591-78-6	4.4e-04	NA	NA
METHYL ETHYL KETONE	78-93-3	8.4e-03	5.4E-01	Cal EPA
METHYL ISOBUTYL KETONE	108-10-1	6.3e-04	NA	NA
METHYLENE CHLORIDE	75-09-2	1.7e-02	2.1E+00	ATSDR
MOLYBDENUM	7439-98-7	6.7e-06	NA	NA
NAPHTHALENE	91-20-3	1.0e-03	NA	NA
NICKEL	7440-02-0	3.8e-05	2.5E-04	Cal EPA
N-PROPYLBENZENE	103-65-1	1.0e-03	NA	NA
O-CHLOROTOLUENE	95-49-8	2.8e-03	NA	NA
O-XYLENE	95-47-6	4.8e-03	9.2E-01	Cal EPA
P-CHLOROTOLUENE	106-43-4	1.0e-03	NA	NA
PENTACHLOROPHENOL	87-86-5	1.4e-05	NA	NA
PHENANTHRENE	85-01-8	1.4e-04	NA	NA
PHENOL	108-95-2	1.2e-03	2.4E-01	Cal EPA
PYRENE	129-00-0	4.1e-05	NA	NA
SELENIUM	7782-49-2	4.9e-06	NA	NA
STRONTIUM	7440-24-6	2.8e-05	NA	NA
STYRENE	100-42-5	1.7e-03	8.8E-01	Cal EPA
TERT-BUTYLBENZENE	98-06-6	2.0e-04	NA	NA
TETRACHLOROETHENE	127-18-4	7.0e-04	1.4E+00	ATSDR
THALLIUM	7440-28-0	5.0e-08	NA	NA
TIN	7440-31-5	5.0e-06	NA	NA
TITANIUM	7440-32-6	2.6e-05	NA	NA
TOLUENE	108-88-3	3.7e-02	3.8E+00	ATSDR
TRICHLOROETHENE	79-01-6	1.2e-03	1.1E+01	ATSDR
TRICHLOROFLUOROMETHANE	75-69-4	3.2e-03	NA	NA
ZINC	7440-66-6	3.3e-04	NA	NA

Notes:
NA - Not Available
AIHA - American Industrial Hygiene Association
ATSDR - Agency for Toxic Substances and Disease Registry
Cal. EPA - California Environmental Protection Agency
ERPG(1 or 2) - Emergency Response Planning Guidelines
[1] For beryllium, the ERPG-2 value is presented for information purposes since an ERPG-1 was not available.

Chapter 6 Tables

Table 6.1 Summary of risk estimates at the six monitoring locations for reasonable maximum exposure

Location	Hazard quotients at or above 1 (child receptor)		Cancer risks at or above $1x10^{-6}$ (30 year exposure)	
Emma Wheeler Homes	MANGANESE	3	FORMALDEHYDE	6E-05
	FORMALDEHYDE	2	CHLOROFORM	1E-05
			BENZENE	1E-05
			CHROMIUM	9E-06
			BENZO-A-PYRENE	9E-06
			CARBON TETRACHLORIDE	7E-06
			BROMODICHLOROMETHANE	5E-06
			1,4-DICHLOROBENZENE	4E-06
			ARSENIC	4E-06
			CHLOROMETHANE	3E-06
			NICKEL	3E-06
			TETRACHLOROETHENE	2E-06
			TRICHLOROETHENE	1E-06
			BENZO(B)FLUORANTHENE	1E-06
Bethlehem Community Center	MANGANESE	2	FORMALDEHYDE	2E-05
			BENZENE	2E-05
			CHLOROFORM	2E-05
			CHROMIUM	1E-05
			CARBON TETRACHLORIDE	7E-06
			1,4-DICHLOROBENZENE	7E-06
			BENZO-A-PYRENE	6E-06
			ARSENIC	4E-06
			CHLOROMETHANE	3E-06
			NICKEL	2E-06
			TETRACHLOROETHENE	2E-06
20th Street Fire Station	MANGANESE	3	FORMALDEHYDE	2E-05
			CHROMIUM	1E-05
			BENZENE	1E-05
			CHLOROFORM	1E-05
			CARBON TETRACHLORIDE	7E-06
			BROMODICHLOROMETHANE	6E-06
			ARSENIC	4E-06
			CHLOROMETHANE	3E-06
			1,1,2-TRICHLOROETHANE	3E-06
			BENZO-A-PYRENE	2E-06
			NICKEL	2E-06
			TETRACHLOROETHENE	2E-06
			1,4-DICHLOROBENZENE	2E-06

Table 6.1 Summary of risk estimates at the six monitoring locations for reasonable maximum exposure

Location	Hazard quotients at or above 1 (child receptor)		Cancer risks at or above $1x10^{-6}$ (30 year exposure)	
Cellular One Tower Site	None		FORMALDEHYDE	2E-05
			CHROMIUM	1E-05
			BENZENE	9E-06
			CARBON TETRACHLORIDE	6E-06
			ARSENIC	6E-06
			BROMODICHLOROMETHANE	5E-06
			BENZO-A-PYRENE	3E-06
			CHLOROFORM	3E-06
			CHLOROMETHANE	3E-06
			TETRACHLOROETHENE	1E-06
East Brainerd Fire Station	COBALT	1	FORMALDEHYDE	2E-05
			CHROMIUM	1E-05
			BENZENE	9E-06
			CARBON TETRACHLORIDE	7E-06
			BROMODICHLOROMETHANE	6E-06
			HEXACHLORO-1,3-BUTADIENE	4E-06
			CHLOROFORM	3E-06
			ARSENIC	3E-06
			CHLOROMETHANE	3E-06
			1,4-DICHLOROBENZENE	2E-06
			TETRACHLOROETHENE	1E-06
River Park Site	COBALT	1	FORMALDEHYDE	2E-05
			CHROMIUM	1E-05
			BENZENE	8E-06
			CARBON TETRACHLORIDE	7E-06
			BROMODICHLOROMETHANE	5E-06
			ARSENIC	3E-06
			CHLOROFORM	3E-06
			CHLOROMETHANE	3E-06
			NICKEL	2E-06
			TETRACHLOROETHENE	2E-06

Appendix A Toxicological profiles

Toxicological profiles included here are those for all chemicals with an estimated hazard index greater than 1 or an estimated incremental cancer risk greater than 1×10^{-6} at any of the six monitoring locations.

References:

ATSDR (1998). *Toxicological Profile for Arsenic (Update).* U.S. Department of Health and Human Services. Atlanta, GA. August 1998.

ATSDR (1997). *Toxicological Profile for Benzene (Update).* U.S. Department of Health and Human Services. Atlanta, GA. September 1997.

ATSDR (1989). *Toxicological Profile for Bromodichloromethane.* U.S. Public Health Service. Atlanta, GA. December 1989.

ATSDR (1994). *Toxicological Profile for Carbon Tetrachloride (Update).* U.S. Department of Health and Human Services. Atlanta, GA. May 1994.

ATSDR (1998). *Toxicological Profile for Chloromethane.* U.S. Department of Health and Human Services. Atlanta, GA. December 1998.

ATSDR (1997). *Toxicological Profile for Chloroform (Update).* U.S. Department of Health and Human Services. Atlanta, GA. September 1997.

ATSDR (1998). *Toxicological Profile for Chromium (Update).* U.S. Department of Health and Human Services. Atlanta, GA. August 1998.

ATSDR (1992). *Toxicological Profile for Cobalt.* U.S. Public Health Service. Atlanta, GA. July 1992.

ATSDR (1998). *Toxicological Profile for 1,4-Dichlorobenzene (Update).* U.S. Department of Health and Human Services. Atlanta, GA. December 1998.

ATSDR (1999). *Toxicological Profile for Formaldehyde.* U.S. Department of Health and Human Services. Atlanta, GA. July 1999.

ATSDR (1994). *Toxicological Profile for Hexachlorobutadiene.* U.S. Department of Health and Human Services. Atlanta, GA. May 1994.

ATSDR (1997). *Toxicological Profile for Manganese (Update).* U.S. Department of Health and Human Services. Atlanta, GA. September 1997.

ATSDR (1997). *Toxicological Profile for Nickel (Update).* U.S. Department of Health and Human Services. Atlanta, GA. September 1997.

ATSDR (1995). *Toxicological Profile for Polycyclic Aromatic Hydrocarbons (PAHS) (Update).* U.S. Department of Health and Human Services. Atlanta, GA. August 1995.

ATSDR (1997). *Toxicological Profile for Tetrachloroethylene (Update).* U.S. Department of Health and Human Services. Atlanta, GA. September 1997.

ATSDR (1989). *Toxicological Profile for 1,1,2-Trichloroethane.* U.S. Public Health Service. Atlanta, GA. December 1989.

ATSDR (1997). *Toxicological Profile for Trichloroethylene (Update).* U.S. Department of Health and Human Services. Atlanta, GA. September 1997.

Appendix B Development of data summary

Reprinted from the *Chattanooga Air Toxics Study Risk Assessment Workplan and Quality Assurance Project Plan* (EPA Region 4, 2001b):

INTRODUCTION

The contractor should provide a data summary for each chemical detected at each monitor in the CATS study area. There should be a separate summary table for each monitor. The values included in each table should be based solely on validated, field-collected environmental data at the monitor in question. Specifically, the tables should not included QA/QC data from the lab or from the field, with the exception of duplicate samples from the Bethlehem Community Center. The summary data for the duplicate samplers should be listed on separate tables and NOT combined with one another at this point. An example table is attached, with specific instructions for filling in the table. Tentatively Identified Compounds (TICs) should be included at the end of each summary table (one line for each TIC).

DATA TO OMIT FROM THE ANALYSIS

The following data should not be included in the data set used to develop the summary tables:

1. Due to metals contamination in one box of high volume filters, chromium, nickel, zinc, molybdenum, and barium results from any sampler from 9/9/99 through 10/27/99 are rejected.[1] Simply remove these results and base the summary statistics on the remaining dataset.

2. Sodium, potassium, calcium, and aluminum in any sample (USEPA 2000, op. cit.).

3. Formaldehyde results from site number 2 from 7/29/99 through 10/3/99 (USEPA 2000, op. cit.). These results are rejected. Simply remove these results and base the summary statistics on the remaining dataset.

[1]USEPA (2000), *Chattanooga CBEP Air Toxics Study Data*, memorandum from Danny France/USEPA Region 4 Air and Water Compliance Section to Linda Anderson-Carnahan, Chief/USEPA Region 4 Air Planning Branch, January 24.

4. For project 99-0225 (VOC samples only), the recommended holding times were exceeded for all VOC samples. As such, the lab marked all the VOC results from this sample date with the J flag, including the non-detects which have UJ flags. These data are rejected. Simply remove these VOC results and base the summary statistics on the remaining data set.

5. Projects # 99-0535 and 99-0519 (in the SVOC data set) are in fact QA projects where the PUF/XAD was checked. Simply remove these SVOC results and base the summary statistics on the remaining dataset (see "Blank" discussion below).

ADDITIONAL CORRECTIONS

1. For project 99-0478 (SVOC data) The STATION_ID mistakenly entered as 0S and should be 05S.

2. For project number 99-0662, sample ID 7379 (SVOC sample), the correct value is 0.07 ug/m^3 for 1-methylnapthalene.

3. Sample 1772 actually ran on 1/12/99 and was inadvertently left at the park site and picked up when the canister for 1/24/99 (number 1769) was installed. The only error was the sample date was not entered in the correct column on the custody sheet.

BLANKS

1. For analytical lab blanks (i.e., everything other than field blanks), the lab has already assessed any concentrations found in those blanks and adjusted the associated environmental sample results as necessary. Specifically, if the lab found a chemical in an analytical blank and in an associated environmental sample, they applied the "5-times" or "10-times" rule.[2] If the concentration in the environmental sample was determined to be blank contaminated, the lab reported that specific result as "not detect" and reported the result simply as an elevated quantitation limit. They did not mark such results as "B."

   Thus, for purposes of developing the data summary, the contractor should not have to worry about blank contaminated data (with one exception; see Blanks Number 2 below) and should simply include any non-detects as one-half the reported quantitation limit. It

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[2]See USEPA (1989), *Risk Assessment Guidance for Superfund, Volume I, Human Health Evaluation Manual (Part A), Interim Final*, Office of Emergency and Remedial Response (EPA/540/1-89/002, December; Section 5.5.

should also be noted that since the lab applied the 5X and 10X rules themselves, they should not have provided any of the analytical blank data in the information they sent to the contractor (with the exception of the one mistaken inclusion noted above). The contractor should identify to EPA any additional analytical blanks that they find to have been mistakenly included in the data set.

1. For field blanks, the lab did not apply the 5X of 10X rules. Rather, the lab simply reported these field blank samples in the data set provided to Cambridge. Cambridge should review these field blanks and apply the 5X/10X rules, in conjunction with EPA's consultation, to determine if any of the sample results should be rejected based on field blank data. It will be imperative that the contractor correctly identify which field blanks go with which set of environmental samples. The field blanks are generally marked "QASFLB," "QAVFLB," "QAFFLB," or "QAMFLB" (for semi-volatiles, volatiles, formaldehyde, or metals, respectively). There may also be instances where field blanks were included under an older naming scheme. For example, VOA Project 99-0199, ID/Station 08Blank is a VOA field blank. If the contractors finds samples marked ID/Station "8," this should be a field blank. The contractor should confirm any and all questionable entries with SESD (including any and all ID/Station = "08Blank").

DATA QUALIFIERS

Unless a value has been rejected for a previously stated reason, the following actions should be taken with regard to qualified data:

1. Values marked "J" should be used as is.

2. Values marked "U" should be included in the data set at one-half the specified quantitation limit value. So, for example, a result of "5U" would be included in an average as a value of 2.5. [Note: If all results for an analyte at a given sampler are all flagged "U," that analyte at that sampler is not included at all in the summary table for that sampler.]

3. Values marked "A" are an average of several analytical results and should be used as is (similar to J-flagged data).

4. Values marked "NA" were not analyzed and should not be included in any fashion.

5. Values marked "NAI" were not analyzed due to interferences and should not be included in any fashion.

6. Values marked "N" indicate presumptive evidence of the presence of the material. These are the tentatively identified compounds. They should be included as positive detections;

however, they should be included (as directed above) to separate lines at the end of the tables and marked clearly as TICS.

7. For "K" (actual value is known to be less than value given) and "L" (actual value is known to be greater than value given) qualified data, use the reported values as is (similar to a J-flag).

8. Data marked "R-qc" are rejected and should not be included in the data set.

9. There should be no data marked "C" since chlordane was not part of the analytical suite in this study.

10. Data marked "J" AND some other qualifier should be use as is according to the rules outlined above. For example, "UJ" means undetected at an estimated sample quantitation limit. Use ½ this estimated sample quantitation limit as a surrogate for concentration.

TABLE INSTRUCTIONS

1. Provide the name of each analyte detected in at least one sample along with the Chemical Abstract Number (no hyphens). Use the exact same name as that provided in the analytical data report.

2. Provide the maximum and minimum detected concentrations. If an analyte was only detected once, the max and min numbers will be the same.

3. Frequency of detection is the number of detections out of the number of NON-REJECTED samples. For example, if there were 30 samples, 5 of which were rejected, and 3 detections among the non-rejected data, the frequency of detection would be "3/25." If an analyte has no detections, it is not included in the table at all.

4. Range of quantitation limits includes the highest and lowest sample quantitation limit (SQL) among the NON-REJECTED samples. If the highest and lowest SQLs are the same number, the contractor should still specify the range (e.g., if 1 ug/m3 was the highest and lowest quantitation limit, the range would be reported as "1 to 1").

5. The arithmetic mean is the sum of the detected values (and ½ the SQL for non-detected samples) divided by the number of samples.

6. The 95% upper confidence limit (based on a normal distribution, if the data set tests out normal; otherwise, based on a presumption of lognormality).

At the end of each table, the contractor should provide one additional summary statistic and any necessary verbiage. Specifically, the contractor should identify how many of the planned samples were actually taken during the study as well as the number of samples (or analytes within samples) that were rejected or voided. The page should also list the specific reasons why they were rejected or voided. Ultimately, we want to be able to look at this page be able to tell two things, namely:

- What percentage of planned samples actually occurred; and
- Of those samples that actually occurred, how much of the data are useable data.

Table Number:
Site Name/Location:
Sampling Period:

CHEMICAL	CAS NUMBER	MAXIMUM CONC. (ug/m3)	MINIMUM CONC. (ug/m3)	FREQUENCY OF DETECTION	RANGE OF QUANTITATION LIMITS (ug/m3)	ARITHMETIC MEAN (ug/m3)	95% UCL (ug/m3)
VOAs (including formaldehyde)							
SVOAs							
Metals							
TICs							