St. Louis Community Air Project
Air Toxics Risk Characterization

Air Pollution Control Program
Air Quality Analysis Section

June 2005

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Appendix

1.0 INTRODUCTION

During fall 1997, the U.S. Environmental Protection Agency (EPA) initiated a program for working with local communities to identify environmental issues and find solutions for them. Staff of EPA Region 7 and their partners conducted a Listening Tour in St. Louis City to learn from residents, workers, and business owners about their environmental concerns. The overwhelming majority of those citizens cited air pollution and its health effects as their most important environmental concern. In response to this concern, EPA Region 7 staff and other interested stakeholders created the St. Louis Community Air Project (CAP).

Subsequently in 1999, EPA devised an Urban Air Toxics Strategy (Strategy) to integrate the framework for addressing air toxics in urban areas. To achieve this goal, EPA conducts expanded air toxics monitoring and modeling to identify areas of concern, to prioritize efforts at reducing risks, and to track progress in those efforts. EPA informs stakeholders about the Strategy and seeks their input to program designs for implementing it. The CAP has become a model application of this Strategy that other urban air projects can emulate.

The St. Louis CAP is a broad-based collaborative effort to improve residents' health by identifying and reducing air pollutants in St. Louis urban areas. The CAP Partnership – over 40 community stakeholders that governed the project – was formed in July of 2000, and includes staff of EPA Region 7, Missouri Department of Natural Resources (MDNR), St. Louis City Air Pollution Control Division, St. Louis Association of Community Organizations (SLACO) as well as residents, businesses, community and environmental groups and educational institutions. The CAP Partnership, guided by its goal of healthier air for St. Louisans, has identified and prioritized air pollutants in St. Louis City, excluding pollutants regulated under the Clean Air Act's (CAA) National Ambient Air Quality Standards (NAAQS) such as ozone and particulate matter. This effort by the CAP will lead to the development of industry- or air pollutant-specific strategies to improve air quality. The CAP Partnership also measured ambient concentrations of diesel particulate matter in accordance with EPA's belief that it poses risks to the public as an air pollutant. EPA's *Health Assessment Document* for diesel particulate matter classified it as a probable human carcinogen (EPA 2002a).

This report focuses on the CAP's priority of identifying air pollutants in St. Louis City and then determining the pollutants of concern. It describes techniques used to monitor ambient air and protocols that identified five priority air pollutants of concern; acetaldehyde, arsenic compounds, benzene, chromium compounds, and formaldehyde, it also reviews potential concerns about diesel particle matter. The report aims to improve present and future urban air toxic assessments by comparing these ambient-air monitoring data and analyses to National-Scale Air Toxics Assessment (NATA) modeling results.

Materials and methods used for air sampling are discussed first in this report, followed by a discussion of the measurement results. Next the chemicals of concern are discussed individually followed by a comparison of the NATA. A discussion of the CAP toxic emission inventory is followed by the risk characterization section. Finally, conclusions and recommendations are offered.

2.0 MATERIALS AND METHODS

EPA and MDNR first determined the list of analytes that would be sampled and worked with the Partnership in locating suitable monitoring sites. The instrumentation and analytical methods used to collect air samples were then determined followed by determining the cancer and noncancer benchmark concentrations.

2.1 ANALYTE DETERMINATION

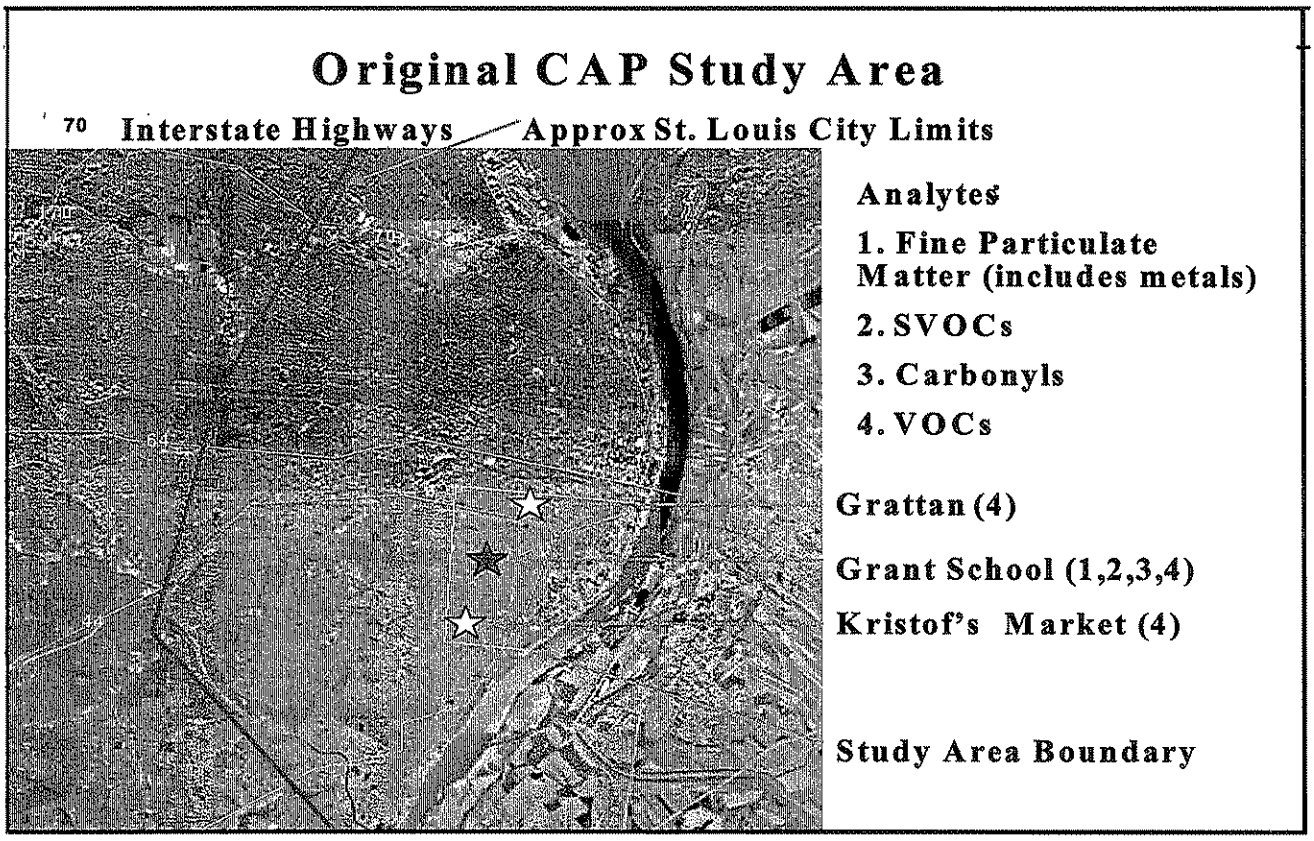
EPA and MDNR used Cumulative Exposure Project maximum modeled concentrations (Rosenbaum et al 1999) to estimate ambient concentrations of the 188 hazardous air pollutants (HAPs) defined by the Clean Air Act in the St. Louis metropolitan area. These estimated ambient concentrations were compared to a benchmark, also referred to as a screening value. At the time this report was initiated, the Cumulative Exposure Project's risk estimates were still under EPA review and not yet available to the public. The CAP Partnership thus chose to use Region 9 Preliminary Remediation Goals (PRGs) (EPA 1999a) as benchmark values, or the Missouri Risk Assessment Level (Giroir 1999) if no PRG benchmark value was available. PRG and Missouri Risk Assessment Level benchmarks are preferred as they are derived without having to extrapolate oral-dose response assessments to define inhalation benchmarks. Additionally, they are associated with cancer risk levels of 1 in 1,000,000 or 1×10^{-6}

Each HAP with an estimated ambient concentration greater than the screening value was placed on the analyte list. (Analytes are chemical compounds that are the subjects of a chemical analysis.) Chemicals on the Urban Air Toxics List (Federal Register 1999) and those chemicals that could be analyzed at no additional cost were also added to the analyte list. Lack of reliable analytical methods eliminated 21 chemicals from this list; polychlorinated biphenyls also were not considered because of high testing costs. The final analyte list included 113 analytes and diesel particulate matter (for which elemental carbon was measured as a surrogate). Of these 113 analytes, 104 were HAPs and 9 were organic compounds that were not HAPs. Within this group of 113 analytes, 61 had assigned cancer benchmarks and 51 had assigned noncancer benchmarks. Data were compared to these health-based benchmarks. Table A1-1 in Appendix A lists the 188 HAPs, indicates the 33 Urban Air Toxics, and shows the CAP analytes.

2.2 MONITORING NETWORK

Monitoring sites were installed in a network that represented the CAP study area of zip codes 63118 and 63104 as accurately as possible. The monitoring sites were not located adjacent or in close proximity to major pollution sources. Site placement was intended to represent "neighborhood" scale — larger individual point sources at some distance, and mobile source impacts generally representing the study area. Samplers were located at Grant School (3026 Minnesota Avenue, AIRS ID 29-510-0089), Kristof's Market (3217 Keokuk Street, AIRS ID 29-510-0091), and 1120 Grattan Street (AIRS ID 29-510-0090). Figure 2-1 depicts these monitoring site locations.

FIGURE 2-1 – SITE LOCATION MAP



Designated the core site, the Grant School site included the most complete sampling. It is in a residential and commercial location near the center of the study area. Several city thoroughfares carrying moderate to heavy traffic lay in close proximity. The Kristof's Market site, designed as a satellite site, is in a residential location at the southern extent of the study area. Significant inner-city traffic moves in close proximity to this site, although traffic volumes are smaller here than at the Grant School and Grattan sites. The Grattan site, also a satellite site, is at the northeast edge of the study area and is the closest to downtown out of the three sites. It is close to and equidistant from the intersection of Interstates 55 and 44 to the south, and Interstate 64 to the north. This area also is residential and commercial, with an area of point sources to the north.

At the Grant School site, monitors measured ambient concentrations of metals, volatile organic compounds, semi-volatile organic compounds, diesel particulate matter (elemental carbon as a surrogate), dioxin, and carbonyl compounds for a complete 24-hour period at a once-every-six-day frequency. Dioxin sampling was conducted for only one month due to the high cost of sampling. During this period, the sample results for dioxin came back as non-detectable (ND). The two satellite sites monitored solely for ambient concentrations of volatile organic compounds at a once-every-six-day frequency.

High ambient formaldehyde concentrations were measured at the Grant School site from December 2001 – October 2002. The CAP Partnership thus agreed to continue measuring formaldehyde, as well as other carbonyl compounds, at the Grant School site and to expand the monitoring network to other sites. This expansion is referred to as Phase II of the CAP Project. Changes included:

1. Sampling at the two satellite sites (Grattan and Kristof's Market) was discontinued in May 2002 (locations are indicated on Figure 2-1). The Grant School site continued operation until December 2003, sampling volatile organic compounds and carbonyls in an effort to enhance understanding of formaldehyde's temporal and spatial variation within the St. Louis area.

2. To investigate the distribution of formaldehyde and other analytes, the study area was broadened to include the north St. Louis area by establishing the Blair St. Site (AIRS ID 29-510-0085) as a National Air Toxics Trends Station and the Arnold fine particulate matter ($PM_{2.5}$) metals site. The Blair Street site- a residential/commercial site north of downtown St. Louis City- monitors for volatile organic compounds, carbonyls, black carbon (as a surrogate for diesel particulates), $PM_{2.5}$ metals, and in July 2003, particulate matter (PM_{10}) metals.

3. To provide further information on pollutant distributions and pollutant transport, carbonyl and speciated non-methane organic compound sampling was also conducted at the rural site near Bonne Terre Station, south of St. Louis (AIRS ID 29-186-0005). This monitoring site began operation in December 2002 and was shut down in December 2003.

4. Time-differentiated measurements of concentrations of formaldehyde and other species is being conducted at Washington University just west of St. Louis using an OPSIS ultraviolet differential optical absorption spectrometer (UV DOAS). This monitor measures ambient formaldehyde concentrations in 5-minute intervals, which will help provide temporal signatures indicating likely source profiles and clearer information about short-term exposures. It will also allow correlation with other time-resolved data, including meteorological data, and other air pollutant concentrations, especially ozone. Preliminary data from the OPSIS UV DOAS show somewhat higher ambient formaldehyde concentrations than those measured at Blair Street or Grant School during Phase II, but much lower ambient concentrations than those measured at Grant School during Phase I. Expectations are that final data from measurements at Washington University will be reported in 2005.

2.3 INSTRUMENTATION AND ANALYTICAL METHODS

At the core site, an Eastern Research Group model ERG:AT/C-3 sampler collected samples analyzed for volatile organic compounds and carbonyl compounds. Samples analyzed for semi-volatile organic compounds and dioxin were collected using a General Metals model GPS1 sampler equipped with a polyurethane foam filter followed by XAD resin. A MetOne model Super SASS sampler collected and measured metals and fine particulate matter ($PM_{2.5}$) samples.

At the satellite sites, Xon-Tech model 910A SUMMA canister samplers collected samples analyzed for volatile organic compounds.

Analysis for $PM_{2.5}$ included several types of laboratory techniques. EPA Method IO-3.3 (X-Ray Fluorescence) was used to determine and analyze $PM_{2.5}$ elements. Ion chromatography methods were used to analyze $PM_{2.5}$ ions. National Institute for Occupational Safety and Health 5040 Thermal Optical Analysis was used to analyze $PM_{2.5}$ carbon. This method is also referred to as Thermal Optical Transmittance (TOT) and is used to analyze for elemental and organic carbon fractions in $PM_{2.5}$. Gravimetric methods (rfps-0498-16) were used to determine $PM_{2.5}$ mass.

EPA Methods TO-9 and TO-11A were used to sample and analyze dioxin and carbonyl compounds, respectively. EPA Methods TO-13 and TO-15 were used to sample and analyze semi-volatile organic compounds and volatile organic compounds, respectively.

2.4 CANCER AND NONCANCER BENCHMARK CONCENTRATIONS

The methodology for establishing analyte health benchmarks is both specific to the St. Louis CAP and consistent with EPA guidelines for evaluating risk to public health. It is similar to other analyses EPA has conducted in support of section 112(k) of the Clean Air Act Amendments (EPA 1999b) and the NATA (EPA 2001a). EPA and MDNR staffs explained this methodology to the CAP Partnership via educational presentations—describing the methods used to assess toxicity, prescribed and generally accepted methods of conducting scientific studies, and the adverse human health impacts of certain pollutants. In the end, the St. Louis CAP Partnership learned accepted processes to establish both cancer and noncancer health benchmarks.

When establishing health benchmarks, the CAP Partnership found that the EPA did not have a complete toxicity database for establishing health benchmarks. In order to fill the gaps, additional data sources were prioritized according to the best available science and compliance with a consistent scheme. NATA prioritized the data sources according to (1) applicability, (2) conceptual consistency with EPA risk assessment guidelines, and (3) level of review.

The methodology for the St. Louis CAP also employed such a prioritization of data sources. Using existing toxicity information, the CAP developed separate health benchmarks for possible cancer and noncancer effects. If the CAP lacked sufficient toxicity information about a pollutant, it was unable to develop health benchmarks for that substance and so could not assess that pollutant's health risk to the community.

For the St. Louis CAP, the definition of a health benchmark was different than the definition found in the EPA's Integrated Risk Information System (IRIS). IRIS defines benchmark concentration as a concentration producing a predetermined change in response rate of an adverse effect, the benchmark response, compared to the background concentration. To make this definition easier to communicate and understand, the CAP Partnership defined health benchmark as the ambient air concentration of a single pollutant believed low enough not to significantly threaten public health if chronically inhaled (in units of parts per billion by volume [ppbv] for gases and micrograms per cubic meter [$\mu g/m^3$] for metals). Both cancer and

noncancer benchmarks were calculated for pollutants having both a unit risk estimate and an EPA Reference Concentration.

These health benchmarks reflect the ambient air concentration representing an upper bound excess lifetime cancer risk of 1 in 100,000 or 1 x 10^{-5} or a noncancer risk equal to EPA's Reference Concentration or equivalent. The CAP chose to establish the target level of an excess individual lifetime cancer risk at 1-in-100,000 (1 x 10^{-5}). If an air pollutant exceeded this level of risk, the CAP would develop and implement additional activities to reduce exposure. Although a target level of cancer risk of 1-in-1,000,000 reflects a more stringent criterion, analytical techniques cannot identify and quantify as many analytes at pollutant concentrations hypothesized to be associated with this target level as with a risk level of 1-in-100,000. The 1-in-100,000 target level of cancer risk therefore allowed the CAP Partnership to collect more useful data about more analytes and to identify air pollutants of potential concern. For those pollutants having cancer and noncancer benchmarks, except for aniline, the 70-year cancer benchmark was smaller than the noncancer benchmark.

For the cancer effects, the CAP preferred data source was EPA's IRIS (EPA 2002b), which contains the unit risk estimates (UREs) and EPA's weight-of-evidence determination. When the UREs were unavailable in IRIS, the California Environmental Protection Agency's (CalEPA) cancer potency values were used (CalEPA 2001a). CalEPA's information is considered a good source of surrogate information because its risk assessment practices are similar to EPA's practices and include a formal peer review process. Also, a formal comparison showed that in most cases, cancer toxicity values did not vary by more than five-fold between EPA and CalEPA (CalEPA 1996). The final source of data was the EPA's Health Effects Summary Tables (HEAST) (EPA 1997). HEAST consolidates cancer toxicity values from various EPA offices for chemicals of primary interest to the Superfund and Resource, Conservation, and Recovery Act programs. HEAST contains provisional toxicity values that have undergone some form of internal EPA review but not an Agency-wide peer review.

The International Agency for Research on Cancer (IARC 2002) also evaluates the evidence and issues judgments whether chemicals are potentially carcinogenic in human beings. IARC uses a similar weight-of-evidence approach to categorize compounds' carcinogenic potential in human beings. Though IARC does not establish quantitative estimates of carcinogenic potency, the CAP included IARC weight-of-evidence as supporting information.

From these sources, the St. Louis CAP identified 61health benchmarks for potentially carcinogenic substances. Cancer benchmarks were developed assuming inhalation exposures of 15, 30, and 70 years, based upon the variability of the population to re-locate and potentially reduce their long-term exposure to urban pollutants. The benchmark concentration was determined by dividing 1 x 10^{-5} by the inhalation URE. For the 15-, 30-, and 70- year exposure scenarios, the cancer benchmark (based on assuming lifetime exposure) was multiplied by the ratio of 70 years to exposure duration; that is, 70/15, 70/30, or 70/70. Table A1-2 in Appendix A presents the cancer benchmarks assuming continuous exposure for 15, 30, and 70 years, both the EPA and IARC weight-of-evidence classifications, and the data sources.

For noncancer effects, the preferred source of data was again IRIS, which contains Reference Concentrations (RfC). A RfC is an estimate of a lifetime continuous inhalation exposure to the human population (including sensitive subgroups) that poses no appreciable risk of deleterious noncancer health effects (EPA 1994). The RfC's uncertainty is estimated at an order of magnitude. When a RfC was not available in IRIS, the next preference was the Agency for Toxic Substances and Disease Registry's (ATSDR) Chronic Minimal Risk Levels (MRL) (ATSDR 2001). A MRL is an estimate of the daily exposure to a hazardous substance likely to pose no appreciable risk of adverse noncancer health effects over a specified exposure duration. Chronic MRLs have exposure durations of 365 days and longer. The third source of data was CalEPA's Chronic Reference Exposure Levels (RELs) (CalEPA 2001b). The final source of data was the HEAST database (EPA 1997). Table A1-3 in Appendix A lists noncancer benchmarks and their data sources.

As part of its study of the adverse human health effects of diesel particulate matter, CalEPA (1998) derived unit risk estimates for lung cancer based on a 1987 case-control study and a 1988 cohort study of U.S. railroad workers. Referencing these two studies, CalEPA derived a lowest lifetime risk estimate of 1.3 in 10,000 (1.3×10^{-4}) for every exposure to 1 $\mu g/m^3$ of diesel particulate matter and a highest lifetime risk estimate of 2.4 in 1,000 (2.4×10^{-3}) for every exposure to 1 $\mu g/m^3$. Yet in its review of CalEPA's derivations, EPA (2002a) found major limitations in both of these studies. For the cohort study, the Agency found a lack of quantitative data on exposure to diesel exhaust. For the case-control study, the Agency found possible over-estimates of cigarette smoking, use of job classification as a surrogate for exposure to diesel exhaust, lack of data on the contribution of unknown occupational or environmental exposures, and a sub-optimal latency period of 22 years. All in all, EPA found that these epidemiological data were too uncertain to derive a satisfactory unit risk estimate for diesel exhaust-induced lung cancer. EPA concluded that current available data were inadequate to derive a cancer unit risk estimate for diesel exhaust or its component, diesel particulate matter. For this reason, the CAP Partnership chose not to use the CalEPA lifetime risk estimates to characterize the cancer risk of exposure to ambient diesel particulate matter.

The CAP called for the Partnership to develop an action plan to reduce the community's exposure to any pollutant exceeding its 70-year health benchmark for cancer or any exceedance of the noncancer benchmarks for long-term exposure. For those pollutants detected at ambient concentrations equal to or greater than the ambient concentrations associated with a risk of 1×10^{-5}, the action plan aimed to reduce these pollutants' ambient concentration. To achieve this reduction, the action plan called for Partnership members to (1) share CAP's monitoring results and pollution solutions through public presentations and outreach, (2) work with community and school groups promoting the use of the Air Toxics educational materials that the Missouri Botanical Garden developed, and (3) join with the American Lung Association, school districts, school bus companies, and other interested stakeholders to find ways to reduce diesel particulate emissions. Appendix D provides more information on this action plan and those CAP activities that occurred in addition to what is detailed in this technically-focused report.

3.0 DISCUSSION OF MEASUREMENT RESULTS

This section discusses sampling and analysis results from the Grant School site from May 13, 2001 through December 29, 2003, and from the Grattan and Kristof's Market sites from May 13, 2001 through May 14, 2002. Because detected ambient concentrations were low, and the budget allowed short-term sampling, the Grant School site collected ambient data on semi-volatile organics from May 13, 2001, through July 31, 2002, and on metals from June 18, 2001, through June 25, 2002. A complete data set is available at the St. Louis CAP's website, http://www.stlcap.org/Chemicals-V4.asp, for those interested in conducting independent analyses. The data analysis flowchart that the Technical Team of the St. Louis CAP Partnership followed is shown in Figure 3-1.

Many analytes were not detected any or a portion of the time. To understand what these non-detects mean, one must examine the relationship between the analyte's Minimum Detection Limit (MDL) and it's noncancer and/or 70-year cancer (70-Bench) benchmark value. The MDL is defined as the minimum concentration of a substance that can be measured and reported with 99 percent confidence that the analytes' concentration is greater than zero. The MDL is determined from analysis of a sample in a given matrix containing the analyte. There is greater confidence in the analytical result when ambient concentrations are detected above the MDL.

Table 3-1 lists analytes that were not detected. Most of them have detection limits less than their noncancer benchmarks and/or 70-Bench values; therefore, values from these analytes indicate negligible risk. For analytes highlighted within Table 3-1, detection limits for the current methods exceed their 70-Bench values; therefore, non-detection of them via the methods used allows no conclusion about risks they may pose.

Table 3-2 lists analytes that have noncancer benchmarks and/or 70-Bench values and were detected less than half the time. Most of these analytes have detection limits less than their noncancer benchmarks and/or 70-Bench values. Detection less than half the time does not allow calculation of a meaningful average concentration, but in general the risk from these analytes is less than the 1 in 100,000 excess lifetime cancer risk defined by their 70-Bench values, and the noncancer risk is not appreciable. The highlighted analytes in the table have detection limits greater than their 70-Bench values. In these cases, non-detection (or only occasional detection) cannot be interpreted as negligible risk.

Table 3-3 lists analytes with averages less than half of their 70-Bench values (or noncancer benchmarks in cases where 70-Bench values were not available). Reported concentrations less than the minimum detection limit were included in the averages, because it was believed that these values, although semi-quantitative, provided a better estimate of the actual concentration than substitution of half the detection limit. Half the detection limit was used for non-detects in calculating averages; justification for this substitution is provided in Appendix B. Because average concentrations are less than half of 70-Bench or noncancer benchmark values, the risk from each of these analytes is less than the 1 in 100,000 excess lifetime cancer risk defined by its 70-Bench value, and the noncancer risk is not appreciable.

Table 3-4 lists analytes with averages greater than half of their 70-Bench values. The same procedure described in the preceding paragraph was used for reported concentrations less than

the detection limit and for non-detects. Analytes highlighted in yellow have concentrations greater than their 70-Bench values. The five analytes with concentrations greater than or equal to their 70-Bench values became the five CAP chemicals of concern due to the associated possibility for increased cancer risk.

FIGURE 3-1 – FLOWCHART FOR THE SELECTION OF THE FIVE CHEMICALS OF CONCERN

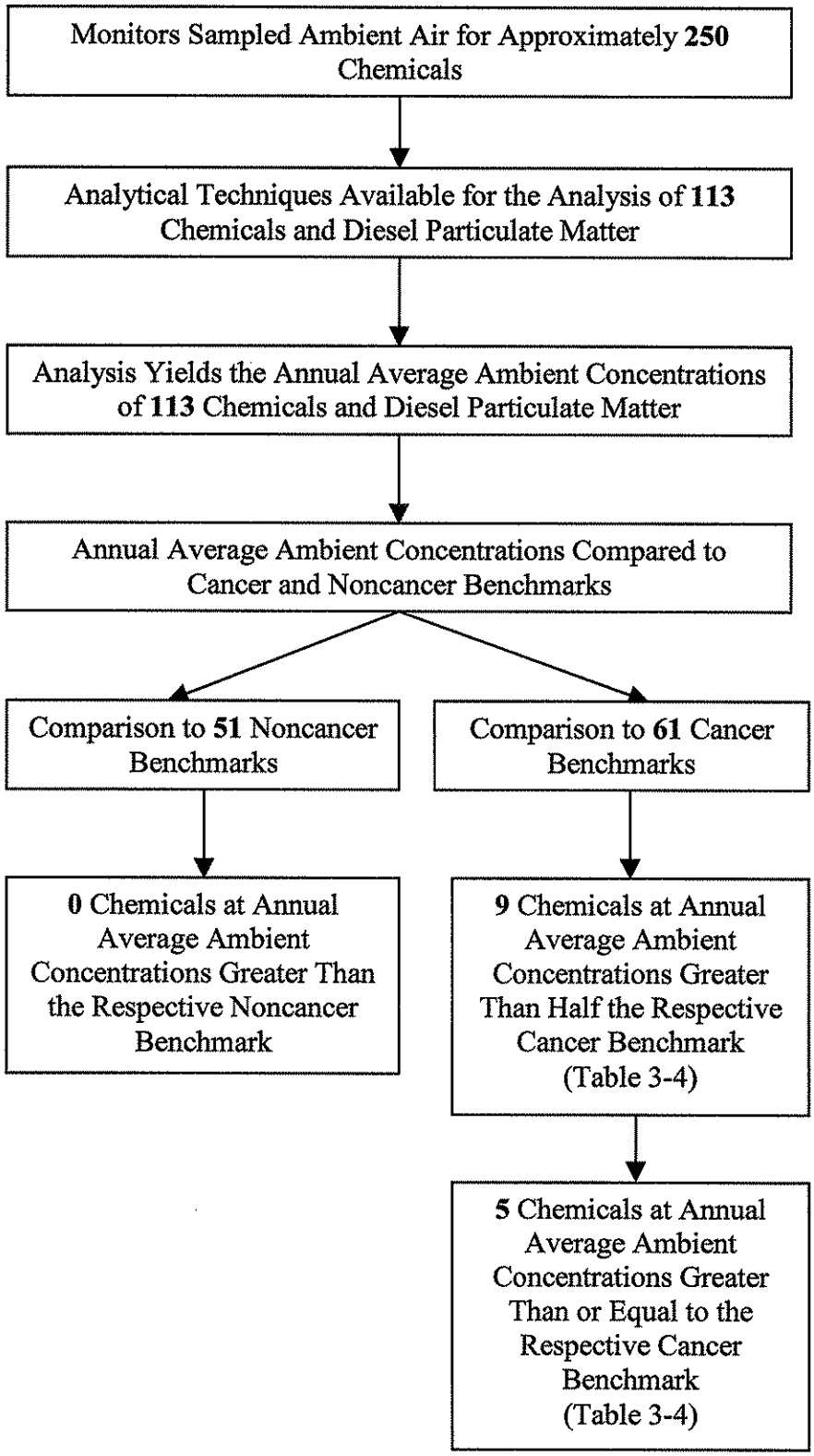


TABLE 3-1 – CAP ANALYTES NEVER DETECTED

						Sampling Location		
Analyte	**Noncancer**	**Source**	**70-Bench**	**Source**	**MDL**	**Grant**	**Grattan**	**Kristof's**
1,2-Dibromoethane	0.10	CAL-EPA	0.00600	EPA-IRIS	0.080	x	x	x
1,1,2 - Trichloroethane			0.12000	EPA-IRIS	0.060	x	x	x
1,1,2,2 - Tetrachloroethane			0.02000	EPA-IRIS	0.060	x	x	x
1,2 - Dichloroethane	593.00	ATSDR	0.09000	EPA-IRIS	0.080	x	x	x
1,2 - Dichloropropane	0.87	EPA-IRIS			0.070	x	x	x
1,2,4-Trichlorobenzene	27.00	HEAST			0.060	x	x	x
1,2,4-Trichlorobenzene	27.00	HEAST			0.060	x		
2,3,7,8-Tetrachlorodipenzo-p-Dioxin (sampled 2/02 only)			2.30E-08	EPA-ORD	7.59E-09	x		
2,4,6-Trichlorophenol			0.40000	EPA-IRIS	0.005	x		
2,4-Dinitrotoluene			0.01500	EPA-IRIS	0.007	x		
2-Acetylaminofluorene			0.07000	CAL-EPA	0.006	x		
2-Nitroaniline	0.04	HEAST			0.011	x		
3,3'-Dichlorobenzidine			0.00300	CAL-EPA	0.004	x		
3-Methylcholanthrene			0.01800	CAL-EPA	0.005	x		
4-Dimethylaminoazobenzene			0.00100	CAL-EPA	0.007	x		
7,12-Dimethylbenz(a)anthracene			0.00150	CAL-EPA	0.009	x		
Acetonitrile	36.00	EPA-IRIS			0.25		x	
Aniline	0.26	EPA-IRIS	1.70000	CAL-EPA	0.021	x		
Azobenzene			2.40000	EPA-IRIS	0.012	x		
Benzidine			0.00002	EPA-IRIS	0.000	x		
Benzo(a)anthracene			0.00970	CAL-EPA	0.004	x		
Benzo(a)pyrene			0.00090	CAL-EPA	0.009	x		
Benzo(b)fluoranthene			0.00880	CAL-EPA	0.009	x		
Benzo(k)fluoranthene			0.00880	CAL-EPA	0.012	x		
bis (2-Chloroethyl)ether			0.08000	CAL-EPA	0.010	x		
Bromodichloromethane			1.80000	CAL-EPA	0.060	x	x	x
Bromoform			0.88000	EPA-IRIS	0.080	x		x
Chlorobenzilate			1.70000	HEAST	0.007	x		
Chloroprene	1.93	HEAST			0.100	x	x	x
Chrysene			0.09750	CAL-EPA	0.006	x		
cis -1,3 - Dichloropropene	4.41	EPA-IRIS	0.55000	EPA-IRIS	0.100	x	x	x
Dibenz(a,h)anthracene			0.00070	CAL-EPA	0.007	x		
Hexachloro-1,3-Butadiene (Hexachlorobutadiene)			0.04200	EPA-IRIS	0.060	x	x	x
Hexachlorobenzene	0.26	CAL-EPA	0.00190	EPA-IRIS	0.006	x		
Hexachlorocyclopentadiene	0.20	EPA-IRIS			0.010	x		
Hexachloroethane			0.25800	EPA-IRIS	0.003	x		
Indeno(1,2,3-cd)pyrene			0.00810	CAL-EPA	0.006	x		
Methyl Methacrylate	171.00	EPA-IRIS			0.180	x		
Methyl methanesulfonate			1.60000	CAL-EPA	0.018	x		
N-Nitrosodibutylamine			0.00093	EPA-IRIS	0.017	x		
N-Nitrosodiethylamine			0.00005	EPA-IRIS	0.020	x		
N-Nitrosopyrrolidine			0.00500	EPA-IRIS	0.017	x		
o-Toluidine			0.05000	CAL-EPA	0.015	x		
Pentachlorophenol			0.18400	CAL-EPA	0.007	x		
Phenacetin			15.87000	CAL-EPA	0.011	x		
Safrole			1.05000	CAL-EPA	0.011	x		
Vinyl Chloride	39.00	EPA-IRIS	0.43000	EPA-IRIS	0.060	x	x	x

*All Concentrations in ppbv. Highlighted MDLs are greater than 70-Bench value.
*CAP - Community Air Project
*CAL-EPA - California Environmental Protection Agency
*EPA-IRIS - Environmental Protection Agency's Integrated Risk Information System as of September 2002
*EPA-ORD – Environmental Protection Agency's Office of Research and Development
*HEAST - Health Effects Summary Tables

Note: An "x" indicates that sampling occurred at this site. The highlighted analytes have detection limits greater than their 70-Bench values, so non-detection (or only occasional detection) cannot be interpreted as negligible risk. Non-highlighted analytes have detection limits below their 70-Bench values, so non-detection can be interpreted as negligible risk.

TABLE 3-2 – CAP ANALYTES DETECTED LESS THAN HALF THE TIME

						Sampling Location		
Analyte	**Noncancer**	**Source**	**70-Bench**	**Source**	**MDL**	**Grant**	**Grattan**	**Kristof's**
1,1,1 - Trichloroethane	183.00	CAL-EPA			0.060	x		
Acetonitrile	36.00	EPA-IRIS			0.250	x		x
Bromoform			0.88000	EPA-IRIS	0.080		x	
Bromomethane	1.29	EPA-IRIS			0.090	x	x	x
Chlorobenzene	217.00	CAL-EPA			0.060	x	x	x
Chloroethane	3790.00	EPA-IRIS			0.080	x	x	x
Chloroform	20.00	ATSDR	0.09000	EPA-IRIS	0.050	x	x	x
Methyl Isobutyl Ketone	20.00	HEAST			0.150	x	x	x
Methyl Methacrylate	171.00	EPA-IRIS			0.180		x	x
N-Nitrosodimethylamine			0.00023	EPA-IRIS	0.020	x		
N-Nitrosopiperidine			0.01700	CAL-EPA	0.019	x		
p - Dichlorobenzene	133.00	EPA-IRIS	0.15100	CAL-EPA	0.090	x		
Styrene	235.00	EPA-IRIS			0.070	x	x	x
Tetrachloroethylene	40.00	ATSDR	0.27000	CAL-EPA	0.060	x	x	x
Trichloroethylene	112.00	CAL-EPA	0.93000	CAL-EPA	0.060	x	x	x
Acrylonitrile	0.92	EPA-IRIS	0.06900	EPA-IRIS	0.210	x	x	x
Phenol	50.00	CAL-EPA			0.029	x		
Isophorone	354.00	CAL-EPA			0.019	x		
bis(2-Ethylhexyl)phthalate			0.26000	CAL-EPA	0.004	x		

*All Concentrations in ppbv. Highlighted MDLs are greater than 70-Bench value.
*CAP - Community Air Project
*ATSDR – Agency for Toxic Substances and Disease Registry
*CAL-EPA - California Environmental Protection Agency
*EPA-IRIS - Environmental Protection Agency's Integrated Risk Information System as of September 2002
*HEAST - Health Effects Summary Tables

Note: An "x" indicates sampling occurred at this sight. The highlighted analytes have detection limits greater than their 70-Bench values, so non-detect (or only occasional detection) cannot be interpreted as negligible risk. Non-highlighted analytes have detection limits below their 70-Bench values, so non-detection can be interpreted as negligible risk.

TABLE 3-3 – CAP ANALYTES LESS THAN HALF OF BENCHMARK CONCENTRATIONS

						Sampling Location & Ave. Ambient Concentration			
Analyte	**Noncancer**	**Source**	**70-Bench**	**Source**	**MDL**	**Grant**	**SNMOC**	**Grattan**	**Kristof's**
1,1,1 - Trichloroethane	183.00	CAL-EPA			0.060	--		0.038	0.040
Chloromethane	48.00	ATSDR	2.700	HEAST	0.060	0.617		0.601	0.605
Ethylbenzene	230.00	EPA-IRIS			0.040	0.537		0.144	0.162
Isopropylbenzene (Cumene)	81.00	EPA-IRIS			0.008		0.076		
m,p - Xylene	33.00	ATSDR			0.050	1.793	1.393	0.369	0.435
Methyl Ethyl Ketone	339.00	EPA-IRIS			0.150	0.771		1.508	0.718
Methyl tert-Butyl Ether	832.00	EPA-IRIS	139.000	CAL-EPA	0.180	0.502		0.427	0.613
Methylene Chloride	288.00	ATSDR	6.000	EPA-IRIS	0.060	1.025		0.470	0.581
Naphthalene (SVOC)	0.57	EPA-IRIS			0.015	0.054			
n-Hexane	57.00	EPA-IRIS			0.130		0.429		
o - Xylene	33.00	ATSDR			0.050	0.632	0.439	0.145	0.194
Propylene	1746.00	CAL-EPA			0.050	0.774		0.564	0.685
Styrene	235.00	EPA-IRIS			0.070		0.203		
Toluene	106.00	EPA-IRIS			0.060	1.836	1.580	0.835	1.043
Antimony (ug/m3)	0.20	EPA-IRIS			0.015	0.010			
Lead (ug/m3)	1.50	EPA-NAAQS	0.830	CAL-EPA	0.005	0.013			
Manganese (ug/m3)	0.05	EPA-IRIS			0.002	0.003			
Mercury (ug/m3)	0.30	EPA-IRIS			0.004	0.002			
Nickel (ug/m3)	0.20	ATSDR	0.032	CAL-EPA	0.001	0.001			

*All Concentrations in ppbv. Highlighted MDLs are greater than 70-Bench value.
*CAP - Community Air Project
*ATSDR - Agency for Toxic Substances and Disease Registry
*CAL-EPA - California Environmental Protection Agency
*EPA-IRIS - Environmental Protection Agency's Integrated Risk Information System as of September 2002
*EPA-NAAQS – Environmental Protection Agency's National Ambient Air Quality Standard
*HEAST - Health Effects Summary Tables
*SNMOC - Speciated non-methane organic compounds sampled at Grant Street

Note: Annual average concentrations were calculated by substituting half of the minimum detection limit for analytical results that were reported as non-detects. Reported concentrations less than the minimum detection limit were included in the averages (refer to Appendix B). The highlighted analytes have detection limits greater than their 70-Bench values, so non-detection (or only occasional detection) cannot be interpreted as negligible risk. Non-highlighted analytes have a lifetime cancer risk of less than 1 in 100,000 and negligible noncancer risk.

TABLE 3-4 – ANALYTES AT OR ABOVE HALF OF BENCHMARK CONCENTRATIONS

						Sampling Location & Ave. Ambient Concentration			
Analyte	**Noncancer**	**Source**	**70-Bench**	**Source**	**MDL**	**Grant**	**SNMOC**	**Grattan**	**Kristof's**
1,3-Butadiene	0.90	EPA-ORD	0.150	EPA-ORD	0.070	0.079	0.100	0.055	0.070
Acetaldehyde	5.00	EPA-IRIS	2.500	EPA-IRIS	0.007	2.668			
Benzene	19.00	EPA-ORD	0.410	EPA-IRIS	0.040	0.468	0.457	0.410	0.455
Carbon Tetrachloride	6.36	CAL-EPA	0.110	EPA-IRIS	0.080	0.086		0.102	0.101
Formaldehyde	7.98	ATSDR	0.627	EPA-IRIS	0.004	19.370			
p-Dichlorobenzene	133.00	EPA-IRIS	0.151	CAL-EPA	0.090			0.107	0.058
Arsenic, ug/m3	0.03	CAL-EPA	0.002	EPA-IRIS	0.002	0.002			
Cadmium, ug/m3	**0.02**	**CAL-EPA**	**0.006**	**EPA-IRIS**	**0.011**	**0.005**			
Chromium, ug/m3	0.29	EPA-IRIS	0.002	EPA-IRIS	0.002	0.002			

*All Concentrations in ppbv. Highlighted analytes are greater than or equal to 70-Bench value.
*CAP - Community Air Project
*ATSDR - Agency for Toxic Substances and Disease Registry
*CAL-EPA - California Environmental Protection Agency
*EPA-IRIS - Environmental Protection Agency's Integrated Risk Information System as of September 2002
*EPA-ORD - Environmental Protection Agency's Office of Research and Development
*HEAST - Health Effects Summary Tables

Note: Annual average concentrations were calculated by substituting half of the minimum detection limit for analytical results that were reported as non-detects. Reported concentrations less than the minimum detection limit were included in the averages (refer to Appendix B). Analytes highlighted in Table 3-4 have concentrations greater than or equal to their 70-Bench values and are the five CAP chemicals of concern. Chromium has an average concentration less than its 70-Bench value, but equal to its 70-Bench value when rounded to one significant figure. The cadmium MDL is bold because it exceeds its 70-Bench value.

Ambient levels of formaldehyde in the twelve months of sampling are considered not to be representative of true ambient air concentrations. Section 4.1 discusses the reasons for this conclusion.

4.0 CHEMICALS OF CONCERN AND EXPANDING THE CAP NETWORK

As mentioned in the preceding section, data collected within the CAP study area identified chemicals of concern exceeding community set health benchmarks for cancer. These chemicals of concern include formaldehyde, acetaldehyde, benzene, arsenic compounds, and chromium compounds. The CAP Partnership also included diesel particulate matter as a pollutant of concern based upon EPA identifying this pollutant as a probable human carcinogen at current ambient concentrations found in the environment. This section provides a discussion of an expanded network, Phase II, to answer questions of spatial variability, particularly of formaldehyde, and provides a summary of sampling and analytical results for the CAP Phase II monitoring network. The section also gives an overview of potential sources of these pollutants and emissions of these pollutants as reported to the 1996 National Toxics Inventory.

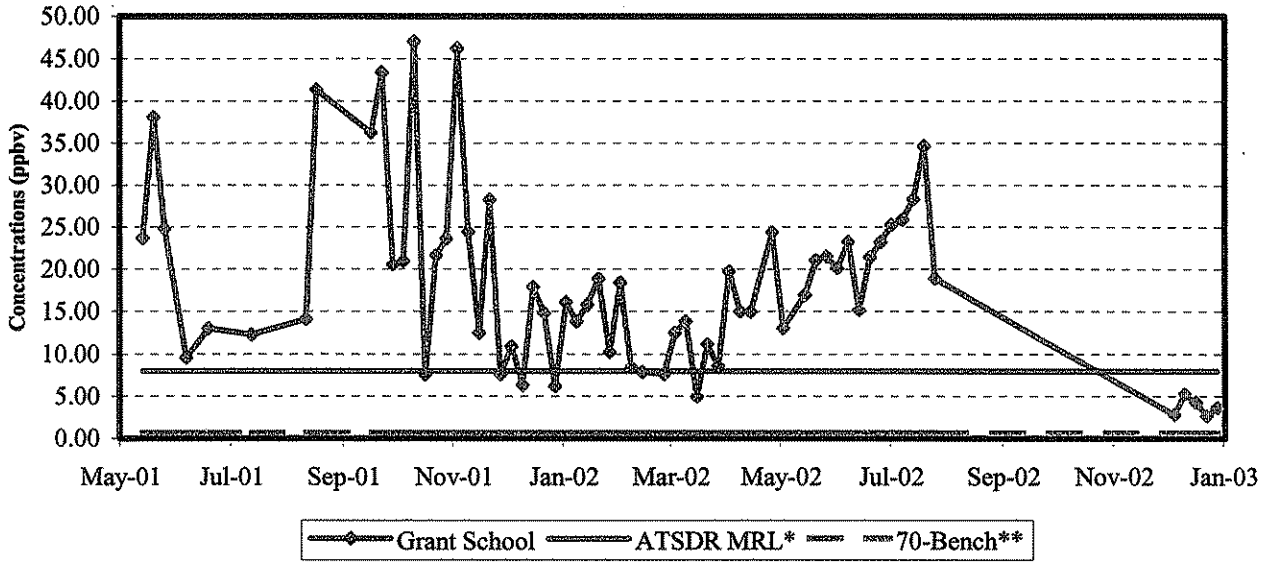
4.1 FORMALDEHYDE AND PHASE II MONITORING

During the first year of monitoring at Grant School, the annual average ambient formaldehyde concentration detected in the CAP study area was 19.368 ppbv. According to this concentration, formaldehyde exceeded its 70-Bench value by a factor of 31, while no other pollutant exceeded its 70-Bench by more than a factor of 1.0. At this ambient concentration, formaldehyde posed a risk of 31 additional cases of cancer in a human population of 100,000 for a 70-year exposure. Formaldehyde had an average concentration greater than its noncancer benchmark, potentially posing a noncancer risk assuming chronic exposure for at least one year.

A time series plot for formaldehyde showing both Phase I and Phase II data collected at Grant School is presented in Figure 4-1. Because monitors located at Washington University, Blair Street, and Bonne Terre were not operational during Phase I, ambient formaldehyde data from these monitors were not available for presentation in this time series plot.

FIGURE 4-1

TIME SERIES FOR FORMALDEHYDE



* ATSDR MRL = 7.98 ppbv
** 70-Bench = 0.627 ppbv

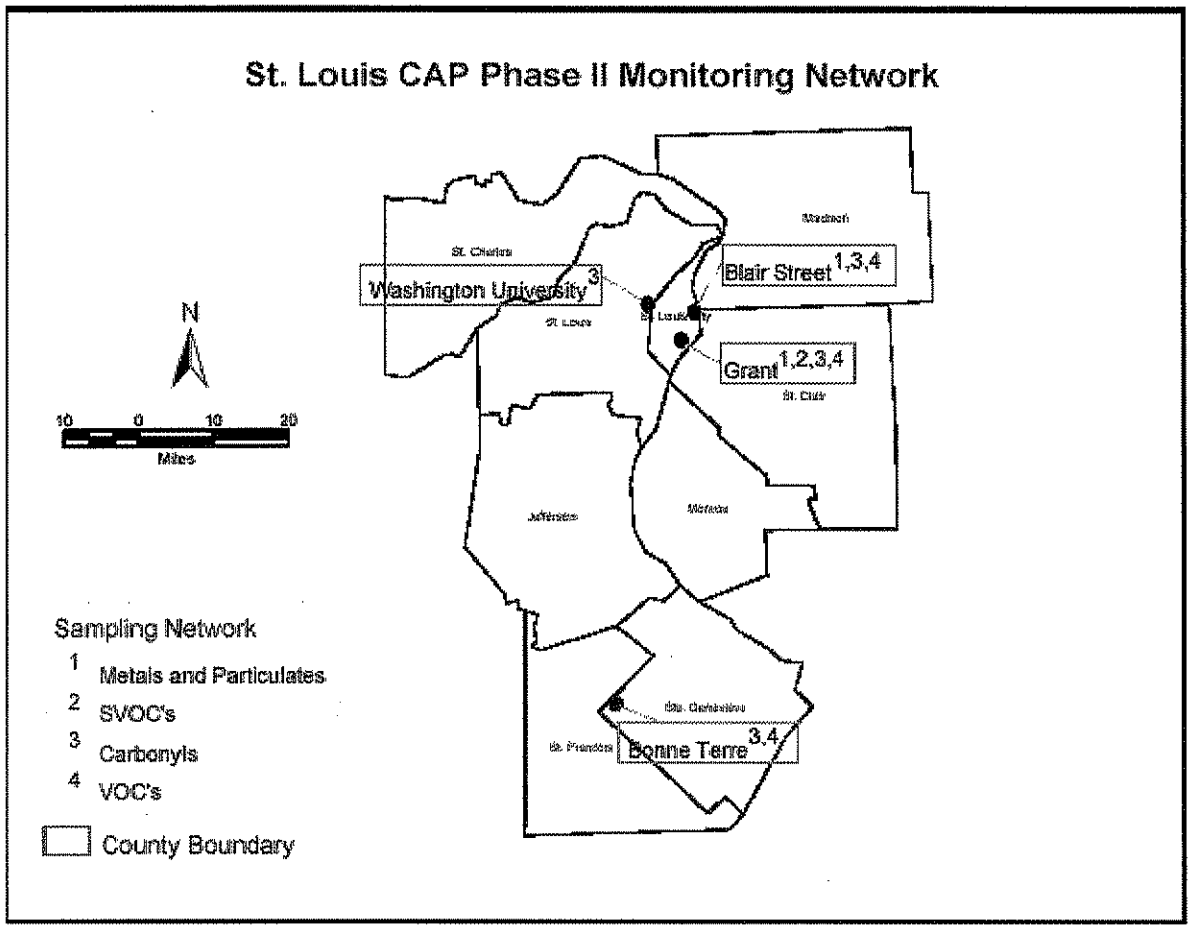
Between May and September 2001, and July and October 2002, carbonyl samples were voided due to moisture contamination. No sampling occurred in November 2002.

Formaldehyde results are generally higher in summer and early autumn and lower in spring and winter. During the summer of 2001 (May 31 – September 10) carbonyl sampling for acetaldehyde and formaldehyde resulted in 13 of the 18 samples being voided due to moisture contamination of the monitor. The source of the contamination was due to high levels of humidity in the air. To correct this error, Eastern Research Group revised the sampling train to include heating of the inlet tubing to prevent condensation.

The CAP was surprised that the annual average ambient formaldehyde concentration was 19.368 ppbv. To learn more about formaldehyde and its atmospheric formation, and provide information on concentrations of the chemicals of concern in other parts of the St. Louis area, the CAP Partnership agreed to continue operations at the Grant School site and to expand the formaldehyde monitoring network to other sites referred to as Phase II of the CAP.

The Phase II sites included, Grant School, Washington University, Blair Street, and a rural site near Bonne Terre, Missouri (Figure 4-2). At the Grant School, Blair Street, and Bonne Terre sites, carbonyl monitors operated for a complete 24-hour period at a once-every-six-day frequency beginning in December 2002 and ending in December 2003. A continuous formaldehyde sampler was located at Washington University. Data from the continuous sampler continue to undergo validation, and so are not presented here.

FIGURE 4-2 – PHASE II ST. LOUIS COMMUNITY AIR PROJECT MONITORING NEWORK FROM DECEMBER 2002 TO DECEMBER 2003



With implementation of the Phase II monitoring network, the CAP partnership was able to obtain a more representative dataset for the St. Louis urban area, and better characterize the relationship between the urban levels of chemicals of concern and more rural areas, represented by the Bonne Terre site. Following in Table 4-1 is a summary of the annual average results for 24-hour sampling at sites in the Phase II network. Sections following will provide greater detail on those results, and discuss some potential sources.

TABLE 4-1 – CAP PHASE II CHEMICALS OF CONCERN: AVERAGE OF 24-HOUR SAMPLING RESULTS

						Sampling Locations		
Analyte	**Noncancer**	**Source**	**70-Bench**	**Source**	**MDL**	**Blair**	**Grant**	**Bonne Terre**
Acetaldhyde, ppbv	5.00	EPA-IRIS	2.500	EPA-IRIS	0.007	2.014	2.551	1.092
Benzene, ppbv	19.00	EPA-ORD	0.410	EPA-IRIS	0.040	0.48	0.44	0.22
Formaldehyde, ppbv	7.98	ATSDR	0.627	EPA-IRIS	0.004	4.080	3.724	3.396
Arsenic, $\mu g/m^3$	0.03	CAL-EPA	0.002	EPA-IRIS	0.002	0.002	*	*
Chromium, $\mu g/m^3$	0.29	EPA-IRIS	0.002	EPA-IRIS	0.002	0.002	*	*

* Sampling for the chemical of concern did not occur at this location.
Sampling occurred between December 2002 and December 2003.

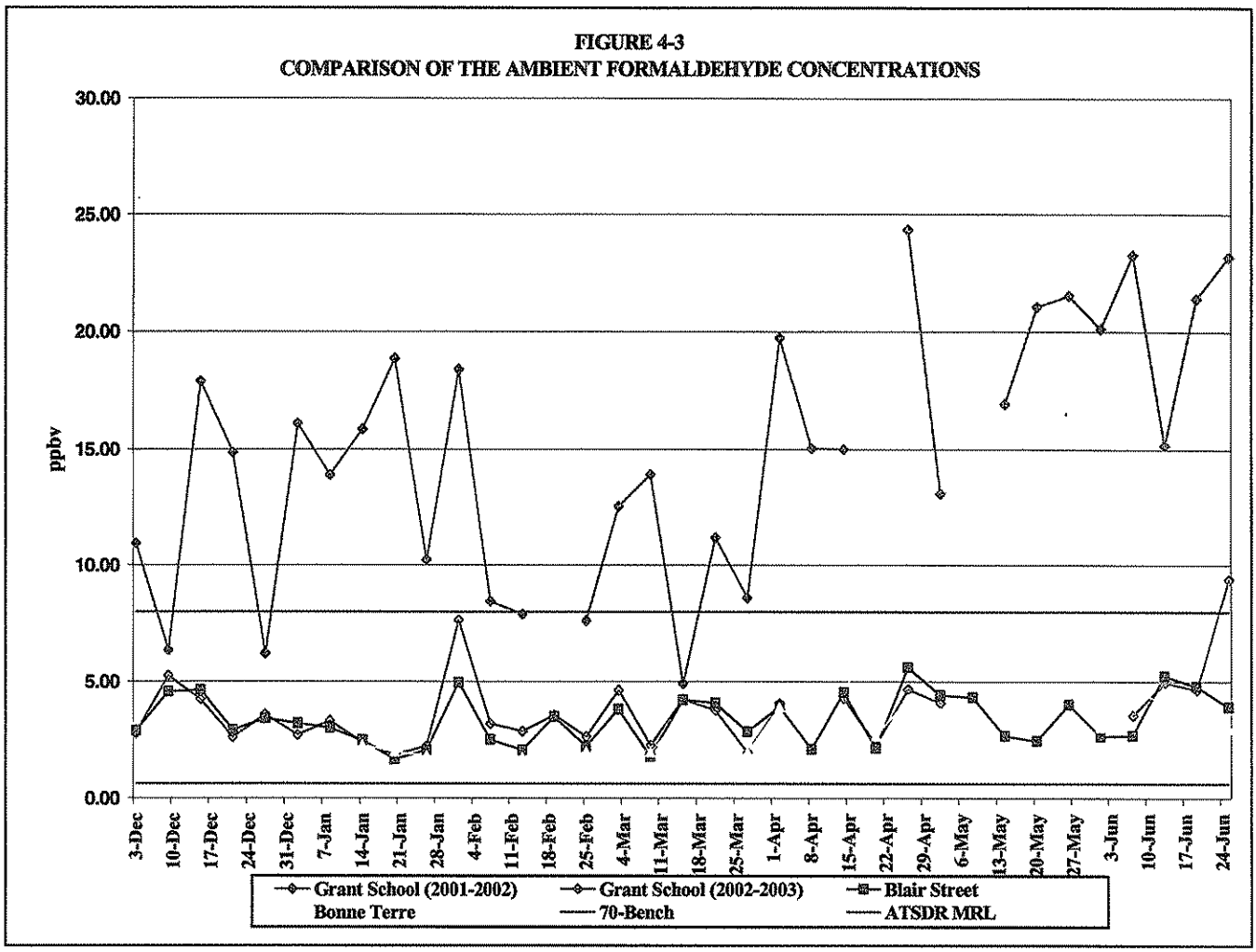
4.2 FORMALDEHYDE SOURCES AND TEMPORALLY RESOLVED DATA

Formaldehyde is both emitted directly into the atmosphere and formed secondarily as a byproduct from other chemical reactions occurring in the atmosphere. Mobile and industrial sources emit formaldehyde directly into the atmosphere. The 1996 National Toxics Inventory for St. Louis City indicates that approximately 144 tons per year of formaldehyde is released directly into the air each year, and mobile sources account for 92 percent of these emissions. Formaldehyde also forms secondarily as a volatile organic compound precursor to ozone formation. Previous monitoring studies indicate that most formaldehyde in the atmosphere traces to secondary formation from chemicals released by mobile sources.

Decreasing the amount of volatile organic compounds released into the air will reduce outdoor formaldehyde concentrations. Using vehicles that burn alternative fuels like ethanol, propane, compressed natural gas, and biodiesel will reduce the release of volatile organic compounds into the air. Additionally, driving less and purchasing fuel-efficient vehicles to reduce car emissions will reduce direct and indirect formations of outdoor formaldehyde concentrations—direct reduction from burning less fuel and indirect reduction from distribution of smaller amounts of gasoline and petroleum products.

Figure 4-3 compares the ambient formaldehyde data collected at the Grant School site between December 2001 and October 2002, and at the Grant School, Blair Street, and Bonne Terre sites between December 2002 and December 2003. In the second year of monitoring, the average ambient formaldehyde concentration at Grant School was 3.72 ppbv, considerably lower than the 19.368 ppbv of the first year. According to the second year results, the cancer risk reduces to 5.9 additional cases per 100,000 people versus the 31 cancer cases utilizing the results from the first year of monitoring in 2001. Also the 2002 noncancer risk is below the noncancer benchmark based upon annual average recorded values.

FIGURE 4-3
COMPARISON OF THE AMBIENT FORMALDEHYDE CONCENTRATIONS



This comparison shows that the ambient formaldehyde data collected at these three sites between December 2002 and June 2003 did not reproduce the same ambient formaldehyde data collected at the Grant School site between December 2001 and June 2002. During the first year of sampling, ambient formaldehyde data collected at Grant School between December 2001 and June 2002 indicated 27 of the 32 air samples with formaldehyde concentrations greater than its noncancer benchmark value. Between December 2002 and June 2003, although all sets of ambient formaldehyde data were greater than its 70-Bench value (0.627 ppbv), only 1 of the 29 ambient air samples collected at the Grant School site had formaldehyde concentrations greater than its long-term noncancer benchmark value (7.98 ppbv). Blair Street and Bonne Terre sites recorded no formaldehyde concentrations greater than the noncancer benchmark value over this same period. No significant difference was found between the ambient formaldehyde data collected at the Blair Street, Bonne Terre, and Grant School sites between December 2002 and December 2003.

Average ambient formaldehyde concentrations also reflect this difference in the data collected between December 2001 – October 2002 and December 2002 – December 2003. Between December 2001 and October 2002, the average ambient formaldehyde concentration was 16.75

ppbv. Between December 2002 and December 2003, however, the average ambient formaldehyde concentrations at the Grant School, Blair Street, and Bonne Terre sites were 3.72, 4.08, and 3.40 ppbv, respectively. Again, ambient formaldehyde data collected at the Blair Street and Bonne Terre sites between December 2002 and December 2003 are consistent with the ambient formaldehyde data collected at the Grant School site between December 2002 and December 2003. Because no meteorological conditions or specific point source influences have been identified to account for this difference in the ambient formaldehyde data sets between Phase I and Phase II monitoring, a sampling or an analytical error during Phase I may account for these variations. The formaldehyde monitor at Grant School was replaced by a somewhat different unit in December 2002. This earlier monitor may have been the cause of the sampling uncertainties for formaldehyde during calendar year 2001. For purposes of risk characterization the ambient formaldehyde data collected from December 2002 to December 2003 will be used to quantify the cancer and noncancer risks posed by this pollutant.

The Grant School monitoring results from Phase I are insufficient to conclude that an area and/or point sources contributed significantly to the ambient concentrations observed. During Phase I, the ambient formaldehyde concentration was measured at only a single location, Grant School, and no correlation with wind direction was apparent. For these reasons, no conclusions could be reached about the geographical distribution of formaldehyde or about the nature of sources. The additional monitoring from Phase II at Grant School, Blair Street, Washington University, and Bonne Terre indicate very uniform levels of formaldehyde throughout St. Louis and outlying areas. Based upon this uniformity it is appropriate to classify this pollutant as regional in nature. During most of the year, pollutant levels appear to be in the neighborhood of a few parts per billion (ppb). During the summer and early fall months, 24-hour levels sometimes reach 10 ppb, more generally 5-7 ppb. This increase is likely related to secondary formation of formaldehyde through photochemistry of combustion products of all types and some biogenic emissions. Additional investigation is needed to characterize the source contribution from biogenic, area, or point source influences.

Currently EPA's Science Advisory Board is re-evaluating the cancer risk posed by formaldehyde. The Environmental Health Subcommittee of the EPA's Science Advisory Board (SAB) previously reviewed the EPA's risk assessment for formaldehyde in 1991. In 2003, the EPA's Office of Research and Development (ORD) responded to recommendations of that review and considered updates reflecting the current state of the science and new studies. As of May 2005, ORD is preparing a toxicological review of formaldehyde and will submit this review to the SAB for external peer review.

The toxicological review will assess potential health effects from exposure to formaldehyde via oral and inhalation paths. The review will examine the physiochemical characteristics of formaldehyde, its mode of action, and dose-response analyses—and will develop dosimetry models and a hazard characterization to reach major risk characterization conclusions. These conclusions will include a health risk estimate for lifetime exposures using available data endpoints to address life stages (for example, reproductive) and developmental indices. The review also will describe major uncertainties and research needs, and their potential impacts on future iterations of the risk assessment.

Changes to the unit risk estimate and the EPA Reference Concentration derived for formaldehyde are likely outcomes of SAB's assessment of the toxicological review. The St. Louis CAP Partnership will further review the formaldehyde data should such a change occur.

4.3 ACETALDEHYDE

Burning organic materials such as gasoline, oil, natural gas, coal, wood, and trash directly releases acetaldehyde into the air. Car and other vehicle exhausts, as well as tobacco smoke, also are direct sources of acetaldehyde. Reactions of volatile organic compounds with sunlight in the atmosphere are secondary sources of acetaldehyde. Most acetaldehyde in the atmosphere originates secondarily, rather than via direct release by emitting sources, as a volatile organic compound precursor to ozone formation. The 1996 National Toxics Inventory for St. Louis City indicates direct release of approximately 64 tons of acetaldehyde into the atmosphere each year, and that mobile sources account for 95 percent of these direct emissions. This emission inventory for hazardous air pollutants was provided by local, state, and federal air programs and represents a comprehensive source profile for air emissions from industrial, mobile, and area sources.

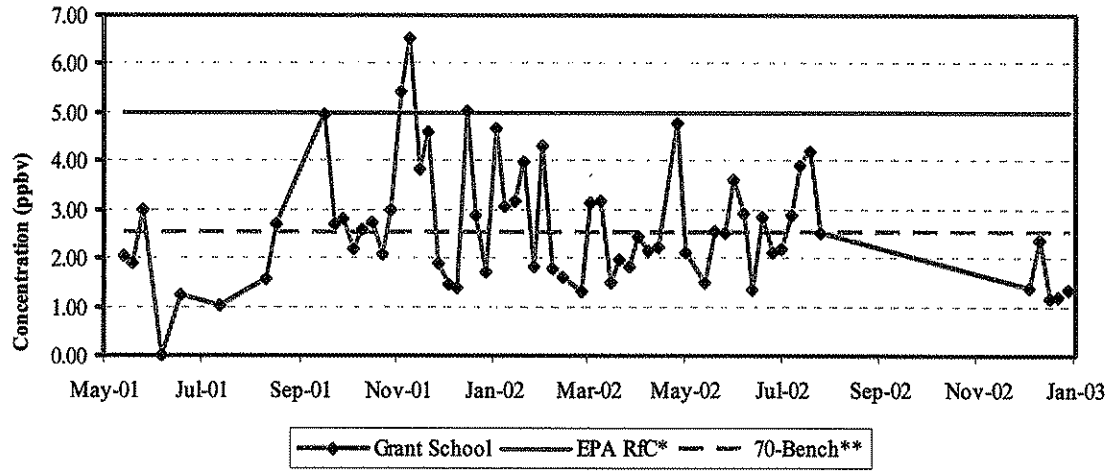
Decreasing the amount of volatile organic compounds released to the air will reduce the secondary formation of acetaldehyde and the associated cancer risk. Driving less and purchasing vehicles that pollute less will reduce vehicle emissions, which in turn will reduce the amount of volatile organic compounds released to the air.

During this first year of monitoring (Phase I) at Grant School from May 2001 to May 2002, the annual average ambient concentration of acetaldehyde detected in the CAP study area was 2.668 ppbv. Figure 4-4 shows the time series plot for acetaldehyde conducted during Phase I. During the summer of 2001 (May 31 – September 10) carbonyl sampling for acetaldehyde and formaldehyde resulted in 13 of the 18 samples being voided due to moisture contamination of the monitor. The source of the contamination was due to high levels of humidity in the air. To correct this error, Eastern Research Group installed a moisture trap to prevent fouling of the sampling tubes.

Monitoring for acetaldehyde was conducted at Grant School from December 2002 –2003 as part of a follow-up investigation to high ambient levels of formaldehyde (refer to Section 4-1). This follow-up monitoring for carbonyls is referred to as Phase II of the St. Louis CAP Project. The annual average ambient acetaldehyde concentration during Phase II monitoring was reported at 2.551 ppbv, comparable to the first year monitoring results of 2.668 ppbv.

FIGURE 4-4

TIME SERIES PLOTS FOR ACETALDEHYDE



* EPA RfC - EPA Reference Concentration
** 70-Bench - Cancer Benchmark

A time series plot for acetaldehyde showing both Phase I and Phase II data collected at Grant School is presented in Figure 4-4. Because monitors located at Washington University, Blair Street, and Bonne Terre were not operational during Phase I, ambient acetaldehyde data from these monitors were not available for presentation in this time series plot. Between July and October 2002, carbonyl samples were voided due to moisture contamination. No sampling occurred in November 2002.

A review of Phase I and II data identifies acetaldehyde's seasonal dependence. Ambient levels appear to be rise in late fall and decline in winter, then rise again in late spring. Secondary formation of acetaldehyde is greatly influenced by meteorological conditions such as temperature and solar radiation as well as photo-reactive decay. The average acetaldehyde concentration slightly exceeded its 70-Bench value, posing a cancer risk of one additional case of cancer in a human population of 100,000 with a 70-year exposure. Acetaldehyde's average ambient concentration was approximately half of its noncancer benchmark and does not pose a noncancer risk assuming a lifetime exposure.

Currently EPA's Science Advisory Board (SAB) is re-evaluating the cancer risk posed by acetaldehyde. In 2003, the EPA's Office of Research and Development (ORD) responded to recommendations of that review and considered updates reflecting the current state of the science and new studies. As of May 2005, ORD is preparing a toxicological review of acetaldehyde and will submit this review to the SAB for external peer review.

The toxicological review will assess potential health effects from exposure to acetaldehyde via oral and inhalation paths. The review will examine the physiochemical characteristics of acetaldehyde, its mode of action, dose-response analyses, and will develop dosimetry models and a hazard characterization to reach major risk characterization conclusions. These conclusions will include a health risk estimate for lifetime exposures using available data endpoints to address life stages (for example, reproductive) and developmental indices. The review also will describe major uncertainties and research needs, and their potential impacts on future iterations of the risk assessment. Changes to the unit risk estimate and RfC derived for acetaldehyde are possible outcomes of SAB's assessment of the toxicological review.

4.4 BENZENE

Unlike formaldehyde and acetaldehyde, benzene is not formed secondarily- it is only produced through direct release into the atmosphere. Burning organic material releases benzene into the air. Car and other vehicle exhausts, tobacco smoke, refineries, pharmaceutical companies, and chemical plants emit benzene into the air. Gasoline and petroleum-product distribution is a source of benzene—via transfer from trucks to tanks at gasoline stations and terminals. According to the 1996 National Toxics Inventory for St. Louis City, approximately 300 tons of benzene escapes into the atmosphere each year. Auto and truck exhausts in the St. Louis area account for approximately 88 percent of this. Industrial sources are responsible for less than 1 percent. The remaining 11 to 12 percent comes from distribution of gas and natural gas fuels.

Driving less by carpooling, using public transportation, properly maintaining vehicles to reduce fuel usage, and purchasing vehicles that pollute less will reduce vehicle emissions and the amount of benzene directly released to the air. Using vehicles that run on alternative fuels also promises additional direct reductions in benzene emissions. An indirect reduction would result from smaller distribution of gasoline and petroleum products. Together, these actions will reduce cancer risk from exposure to benzene.

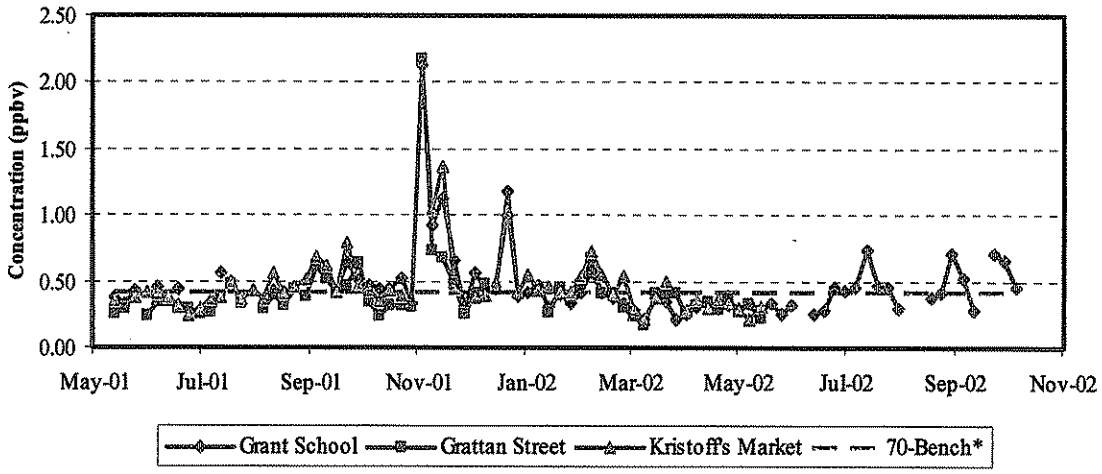
During this first year of monitoring, annual average ambient levels of benzene detected at Grant School, Grattan, and Kristof's were very comparable.

Grant School – 0.468 ppbv Grattan – 0.410 ppbv Kristof's – 0.455 ppbv

The annual average monitored benzene concentration from these three sites was 0.444 ppbv. This slightly exceeded its 70-Bench value, posing a cancer risk of one additional case of cancer in a human population of 100,000 for a 70-year exposure. From the data gathered, it does not appear to pose a noncancer risk assuming a lifetime exposure. Figure 4-5 shows time series plots for benzene concentrations in the study area during Phase I. Because Phase II ambient concentrations were similar to other ambient benzene concentrations measured in other urban areas of the country, these Phase II data were not presented in this time series plot. Benzene does not show seasonal dependence.

FIGURE 4-5

TIME SERIES PLOTS FOR BENZENE



* 70-Bench – Cancer

4.5 ARSENIC COMPOUNDS

Metal smelting operations, cement kilns, agricultural burning, and combustion engines are all sources of arsenic compounds. Other sources include tobacco smoke, wood burning (treated and untreated), gasoline, oil, coal, and use of arsenic-containing pesticides and herbicides. Multiple industrial and other area sources release small quantities of arsenic compounds that are difficult to estimate and include in existing emission inventories. The 1996 National Toxics Inventory for St. Louis City indicates an annual release of about 500 pounds of arsenic compounds, 94 percent from industrial sources.

Reducing the use of fossil fuels and installing additional air pollution control equipment at suspect facilities will reduce emissions of particulates containing arsenic compounds and the associated cancer risk from exposure to these arsenic compounds.

During the first year of monitoring, the annual average ambient concentration of arsenic compounds detected in the CAP study area was 0.002 $\mu g/m^3$. The ambient values for arsenic were detected right at the MDL. Confidence in the analytical result increases as ambient concentrations are detected above the MDL. Based upon the monitoring values for this pollutant it may be considered as having a high degree of uncertainty. Additional studies using more sensitive collection and analytical methods may be able to determine ambient arsenic compound concentrations more accurately.

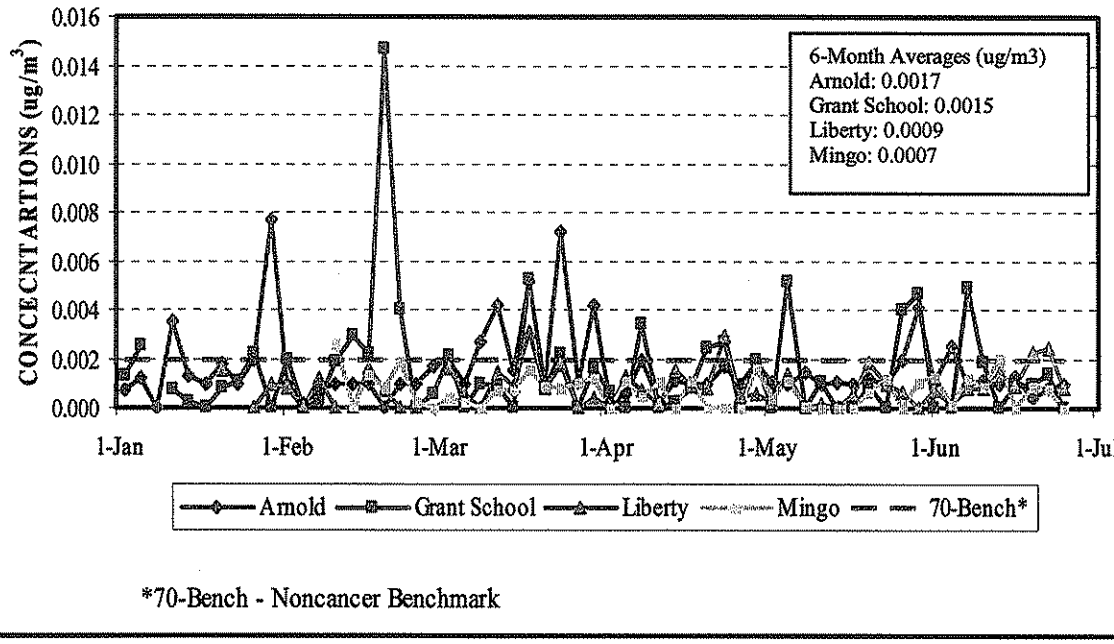
Arsenic is emitted to the air primarily as arsenic trioxide and is usually found in the atmosphere as a mixture of particulate arsenite and arsenate. This metal compound was evaluated as

inorganic arsenic for purposes of determining cancer and noncancer risks. Utilizing concentration levels of arsenic compounds as a (1:1) surrogate for inorganic arsenic the annual average value was slightly greater than the 70-Benchmark value for cancer. This corresponds to one additional case of cancer in a human population of 100,000 with a 70-year exposure. A lifetime exposure to this ambient arsenic compounds concentration however does not appear to pose a noncancer risk.

Figure 4-6 shows the time series plots for ambient arsenic concentrations at four $PM_{2.5}$ speciation sites in Missouri that were each collecting ambient arsenic data between January and June 2002. This plot shows a statewide comparison of the ambient arsenic data collected at these three other monitoring locations to the data collected at the Grant School location of the St. Louis CAP.

FIGURE 4-6

TIME SERIES FOR AMBIENT ARSENIC CONCENTRATIONS AT FOUR $PM_{2.5}$ SPECIATION SITES IN MISSOURI FROM JANUARY - JUNE 2002



*70-Bench - Noncancer Benchmark

The Mingo site at the Mingo Wildlife Refuge near Puxico is a characteristically rural site. The other three sites are characteristically urban. Figure 4-6 shows predominant peaks associated with the urban sites at Arnold and Grant School. However, no predominant peaks are associated with the other urban site at Liberty or with the rural site at Mingo. The six-month average ambient arsenic concentrations at the Arnold and Grant School sites are roughly twice as great as the averages at the Liberty and Mingo sites. These observations suggest local source(s) may be influencing the ambient arsenic concentrations in Arnold and St. Louis City.

4.6 CHROMIUM COMPOUNDS

Electroplaters, ore smelters and refiners, chemical and refractory processes, cement kilns, dye and pigment manufacturers, and wood product preservation processes release chromium compounds into the air. Multiple sources, predominately industrial, release small quantities of chromium compounds. Based on the 1996 National Toxics Inventory for St. Louis City, an estimated 3,000 pounds of chromium compounds enter the atmosphere each year. Ambient chromium compound concentrations may also be traced to sources outside of the St. Louis metropolitan area. Chromium compounds can be in fine particulate matter transported long distances via the wind before settling in the St. Louis metropolitan area.

Installing additional air pollution control equipment at suspect facilities will reduce emissions of chromium compounds and thereby reduce cancer risk.

During the first year of monitoring, the annual average ambient chromium concentration detected in the CAP study area was 0.002 $\mu g/m^3$. The ambient values for chromium were detected at the Minimum Detection Limit (MDL). The MDL is defined as the minimum concentration of a substance that can be measured and reported with 99 percent confidence that the analytes' concentration is greater than zero and is determined from analysis for a sample in a given matrix containing the analyte. As ambient concentrations are detected above the MDL the confidence in the analytical result increases. Based upon the monitoring values for this pollutant it maybe considered as having a high degree of uncertainty.

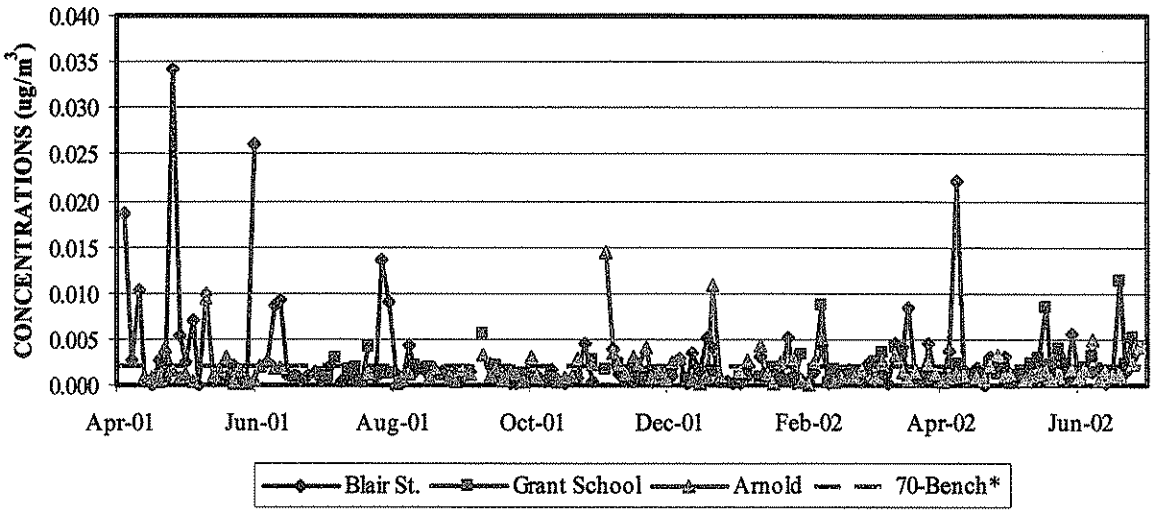
The cancer and noncancer benchmarks for chromium compounds were based upon the assumption that 34 percent of all atmospheric chromium was the hexavalent species. The source of this chromium speciation was a 1996 National-Scale Air Toxics Assessment (NATA) (EPA 2003b) recommendation. Within the NATA analysis of chromium and hexavalent chromium, the total chromium mass present as the hexavalent species ranged from 0.4 percent to 70 percent. Because the high end of the range was associated exclusively with electroplating sources, the EPA chose 34 percent, the upper end of the range for utility boilers. It is likely that most sources of chromium emissions in the U.S. contain smaller amounts of hexavalent chromium.

Based upon this review by EPA, the Technical Team of the CAP Partnership adjusted the IRIS RfC and URE for particulate hexavalent chromium to reflect this adjustment of 34 percent. Based upon these adjustments the annual average concentration value of chromium compounds was less than the 70-Bench value for cancer but equal to the 70-Bench value when rounded to one significant figure. This corresponds to a cancer risk of one additional case of cancer in a human population of 100,000 with a 70-year exposure. A lifetime exposure to this ambient chromium compounds concentration however does not appear to pose a noncancer risk.

Time series plots for ambient chromium concentrations at three $PM_{2.5}$ speciation sites that were each collecting ambient chromium data between April 2001 and June 2002 in St. Louis appear in Figure 4-7. This plot indicates possible spatial variation of ambient chromium. The average chromium concentration is higher at Blair Street in St. Louis City than at Grant School in St. Louis City and at Arnold, Missouri.

FIGURE 4-7

TIME SERIES FOR AMBIENT CHROMIUM CONCENTRATIONS AT THREE $PM_{2.5}$ SPECIATION SITES IN ST. LOUIS



* 70-Bench - Cancer

4.7 DIESEL PARTICULATE MATTER

Diesel particulate matter is released primarily from diesel engines in mobile sources (school buses, diesel trucks, and heavy-duty and off-road construction equipment). According to the 1996 National Toxics Inventory for St. Louis City, about 540 tons of diesel particulate matter is released into the air each year. Heavy-duty diesel trucks and construction equipment like bulldozers, tractors, and backhoes emit approximately 77 percent of this diesel particulate matter. The remaining 23 percent comes from on-road mobile sources such as diesel trucks, buses, and light-duty diesel engines.

Using alternative fuels like ethanol, propane, compressed natural gas, and biodiesel reduces ambient diesel particulate matter concentration. Reduction also results from retrofitting diesel engines using particle traps, catalysts, and other engine modifications. A diesel engine retrofitted with a particle trap can achieve a 60 to 90 percent reduction in particulate matter, volatile organic compounds, and carbon monoxide emission when used with ultra-low sulfur fuel.

EPA's Study "Diesel PM Model to Measurement Comparison Report" (October 2002, publication ID EPA420-D-02-004) develops a methodology for looking at elemental carbon readings as a surrogate to diesel particulate matter. From this study EPA determined that elemental carbon measurements from a $PM_{2.5}$ monitor utilizing the Thermal Optical Transmittance (TOT) detection method could be equated to diesel particulate matter measurements. The methodology looked at seasonal variations across geographical areas. The

adjustment for St. Louis would require elemental carbon values to be multiplied by 2.25 to represent ambient diesel particulate matter levels.

Utilizing the first year of monitoring results for elemental carbon collected at the Grant School site and the above study, the Technical Team of the CAP Partnership determined that the average annual ambient diesel particulate matter concentration ranged between 1.5 and 1.7 $\mu g/m^3$. This ambient level is well below the CAP noncancer benchmark. Excess cancer risk for this pollutant, as described in Section 2.4, is too uncertain to derive a satisfactory unit risk estimate for diesel exhaust-induced lung cancer. EPA concluded that current available data were inadequate to derive a cancer unit risk estimate for diesel exhaust or its component, diesel particulate matter. Despite these findings, the CAP Partnership still considered diesel particulate matter an important chemical pollutant to address because of the community's concern and ongoing debate and development of human health risk information.

EPA required new emission standards for on-road diesel engines in 2004, and will require more stringent standards in 2007. In April 2003, the EPA proposed new emission standards for non-road diesel engines used in construction, agricultural, and industrial operations. In this rulemaking, EPA also proposed a greater than 99-percent reduction in the sulfur content of fuel used in these engines. EPA will require petroleum companies to produce diesel fuel with a lower sulfur content in 2006. Taken together, these efforts will reduce significantly diesel particulate matter released from both on-road and off-road mobile sources.

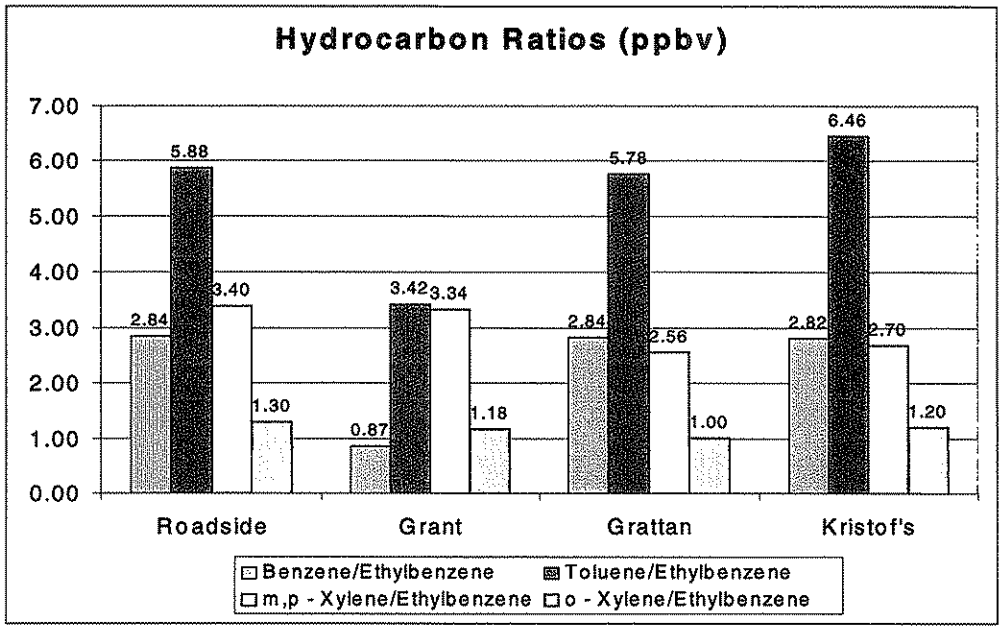
4.8 OTHER HYDROCARBON COMPOUNDS

The VOC data, specifically benzene, toluene, ethylbenzene, and xylene (BTEX) data were evaluated to determine potential sources. A 1990 field measurement project in Atlanta, conducted as a part of the Atlanta Ozone Precursor Monitoring Study, measured speciated volatile hydrocarbon emissions, including BTEX emissions, during heavy traffic at "roadside," at a tunnel-like underpass as well as evaporative emissions from gasoline (Conner et al., 1995). The Atlanta study provided detailed speciated hydrocarbon profiles of motor vehicle emissions. A recent analysis of data from other United States air toxics monitoring sites has shown concentration ratios of benzene, toluene, *m, p*-xylenes, and *o*-xylene, to ethylbenzene to be similar to ratios calculated from the results of the Atlanta study. This analysis suggests that these compounds as measured at multiple United States sites likely result largely from motor vehicle emissions (Eastern Research Group, 2003).

Figure 4-8 shows ratios of average concentrations of benzene, toluene, *m, p*-xylenes, and *o*-xylene, respectively, to ethylbenzene for the Atlanta roadside site and for the three CAP monitoring sites. As in the national study, the generally similar pattern of ratios suggests that these analytes result largely from motor vehicle exhaust sources. Ratios from measurements at the Grant School site do not fit the pattern as well as the ratios from the two satellite sites because of unique results for some hydrocarbons at the Grant School site, including ethylbenzene. Measured concentrations for ethylbenzene, toluene, and the xylenes showed higher concentrations at the Grant School site than at the satellite sites initially, with a general decrease over the duration of the project to concentrations similar to those measured at the other sites. This decreasing trend may result from a sampling artifact (that diminished over time), or may indicate a local source (whose emissions decreased with time) different from motor vehicle

exhaust. No matter the reason for this decreasing trend, the large measured concentrations of ethylbenzene at the Grant School site reduced the size of the ratios of the selected hydrocarbons, but did not alter the pattern of these ratios when compared to patterns of ratios measured at Grattan, Kristof's Market, and Roadside.

FIGURE 4-8 – RATIOS OF AVERAGE CONCENTRATIONS OF SELECTED HYDROCARBONS TO AVERAGE ETHYLBENZENE CONCENTRATIONS



Roadside represents speciated volatile hydrocarbon emissions measured during heavy traffic at a tunnel-like underpass in 1990 as part of the Atlanta Ozone Precursor Monitoring Study.

5.0 COMPARISON TO THE NATIONAL-SCALE AIR TOXICS ASSESSMENT

The National Air Toxics Assessment (NATA) is designed to improve EPA, state, local, tribal governments, and public comprehension of the air toxics problem in this country. EPA will apply results of this Assessment to help it:

- Identify air toxics of greatest potential concern
- Characterize the relative contributions to air toxics concentrations and population exposures of different types of air toxics emissions sources
- Set priorities for collecting additional air toxics data to improve estimates of air toxics concentrations and their potential public health impacts
- Establish a baseline to track trends over time in modeled ambient concentrations of air toxics
- Establish a baseline for measuring progress toward goals for inhalation risk reduction from ambient air toxics.

NATA includes four steps to assess a number of air toxics and diesel particulate matter across the country. Two of these steps are to inventory air toxics emissions and to estimate annual average ambient air toxics concentrations.

EPA compiled a National Toxics Inventory in 1999 of air toxics emissions from outdoor sources. Emissions sources in the inventory included major stationary sources, area and other sources, and on-road and non-road mobile sources. EPA then entered this 1999 emissions data into the Assessment System for Population Exposure Nationwide (ASPEN) air dispersion computer model. The ASPEN model estimated 1999 ambient concentrations of the air toxics across a census tract.

Acetaldehyde, benzene, formaldehyde, arsenic compounds, chromium compounds, and diesel particulate matter were among the air toxics that the 1999 NATA examined in St. Louis City. Table 5-1 compares the NATA modeled ambient concentrations predicted to occur in Tract 116500, the location of the Grant School monitor, to the St. Louis CAP's Grant School monitoring data for these air toxics.

TABLE 5-1 – COMPARISON OF THE NATIONAL-SCALE AIR TOXICS ASSESSMENT'S (NATA) MODELED AMBIENT CONCENTRATIONS TO THE ST. LOUIS COMMUNITY AIR PROJECT'S (CAP) MONITORED AMBIENT CONCENTRATIONS

Selected Air Toxic	NATA Modeled Annual Average Ambient Concentration in Tract 116500 ($\mu g/m^3$)[a]	St. Louis CAP's Grant School Monitored Annual Average Ambient Concentration ($\mu g/m^3$)
Diesel Particulate Matter	3.28176	1.5 to 1.7
Arsenic Compounds	0.000217	0.002
Chromium Compounds	0.000225	0.002
Acetaldehyde	2.2416335	4.8
Formaldehyde	2.2966552	4.58[b]
Benzene	2.207612	1.5

[a] $\mu g/m^3$ is micrograms per cubic meter.
b The formaldehyde concentration represents December 2002 through December 2003 data. All other concentrations represent May 2001 to May 2002 data.

The comparison shows that NATA *overestimated* the annual average ambient diesel particulate matter and benzene concentrations in St. Louis City. Yet NATA also *underestimated* annual average ambient concentrations of arsenic compounds (nine-fold), chromium compounds (nine-fold), acetaldehyde (two-fold), and formaldehyde (two-fold).

Though specific to St. Louis City, this comparison suggests that NATA may need to refine its process to improve estimations. The modeled results provided by NATA are constructed to answer questions about emissions, ambient air concentrations, exposures and risks across broad geographic areas (such as counties, states and the Nation) at a moment in time. As supported by the CAP findings and as stated by EPA, NATA should not be used to identify exposures and risks for specific individuals, or even to identify exposures and risks in small geographic regions such as a specific census tract. These limitations, or caveats, must always be kept in mind when interpreting the results and comparing these results to monitored values.

The modeling used in NATA is the ASPEN model, a computer simulation model used to estimate toxic air pollutant concentrations. The ASPEN model takes into account important determinants of pollutant concentrations, such as: rate of release, location of release, the height from which the pollutants are released, wind speeds and directions from the meteorological stations nearest to the release, breakdown of the pollutants in the atmosphere after being released (i.e., reactive decay), settling of pollutants out of the atmosphere (i.e., deposition), and transformation of one pollutant into another (i.e., secondary formation). The model estimates toxic air pollutant concentrations for every census tract in the continental United States, Puerto Rico and the Virgin Islands. The output for the model is presented at the county level whereas the ambient concentration estimates are at the local, neighborhood level. Despite this difference, it is still important to compare the model output to the ambient concentration estimates as a means to verify the ASPEN model estimates of the 1999 ambient concentrations of the air toxics across a census tract. This output at the county level may be significantly different when compared to ambient monitoring data. Ambient data may be influenced by specific area or point

sources (fence line monitoring). ASPEN-modeled county values may significantly underestimate local scale conditions.

6.0 TOXIC EMISSION INVENTORY

The CAP Partnership developed an emissions inventory report to help identify sources of toxic chemicals that pose the greatest potential heath risks. This emission inventory served as an indication of the HAPs present in the ambient air. To the extent that the emission inventory was complete, an accounting of emitted air pollutants was performed. The emissions inventory therefore included all 188 HAPs, not only those HAPs currently monitored.

The emissions inventory consisted of four major components: point sources, area sources, on-road mobile sources, and off-road mobile sources. This inventory compiled the best estimates of toxic emissions then available. The 1999 Missouri Emission Inventory Questionnaire served as the source of emissions data for point sources. For area sources, the 1996 National Toxics Inventory (NTI) provided the emissions data. The 1996 NTI also provided the emissions data for off-road mobile sources, and the computer model, MOBILE6, estimated the emissions of on-road mobile sources.

6.1 POINT SOURCES

A point source is any stationary source that emits or has potential to emit any of the following: 10 tons per year of any listed HAP; 25 tons or more of any combination of listed HAPs; or more than the *de minimis* amount of any criteria pollutant. Point-source data has been completed for St. Louis City and County for 1996 and 1999, and projected for 2001. Annual MDNR/APCP Emissions Inventory Questionnaires mailed to sources in the study area and on-site inspections of facilities during December 1999 served as sources for this data. Table 6-1 lists the top point-source polluters by company. The Clean Air Act requires these and other large point-source polluters to report all emissions to EPA. Table 6-2 lists the top toxic emissions from point sources in 1999 (Freebairn 2001a).

Table 6-1: Top-Ten St. Louis Point Source Air Toxics Emittors in 1999

Plant Name	Emissions (tons/yr)
AmerenUE	1,890.84
Ford Motor Co.	470.59
Decorative Surfaces	216.95
Chrysler Corp. North Plant	214.94
Anheuser-Busch Inc.	109.40
Mallinckrodt Inc.	106.54
Centerline Industries	46.15
Boeing Company	45.94
US Paint Div.	45.90
Dana Corporation	45.23

Table 6-2 Top Ten Total St. Louis Air Toxics Emitted in 1999

Toxic Chemical	Emissions (tons/yr)
Hydrogen Chloride	1,959
Xylene	333
Toluene	284
Perchloroethylene	189
Methyl Isobutyl Ketone	188
Methyl Ethyl Ketone	167
2-Butoxyethanol	138
Glycol Ethers	105
Methanol	98
Trichloroethylene	45

6.2 AREA SOURCES

Area sources are stationary sources that have the potential to emit less than 10 tons per year of any listed HAP or 25 tons per year of any combination of listed HAPs. The area source inventory compiled for the CAP study is based on 1996 National Toxic Inventory estimates of source categories present in zip codes 63104 and 63118. Table 6-3 lists the top air toxics emitting area-source categories (Freebairn 2001b).

Table 6-3 Top Ten Area Source Category Air Toxic Emittors- from the City of St. Louis 1996 NTI.

Category	Emissions (lb/yr)
Consumer Products Usage	892,419
Surface Coatings: Architectural	291,060
Autobody Refinishing Paint Application	177,940
Gasoline Distribution Stage I	137,727
Surface Coatings: Industrial Maintenance	81,760
Paints and Allied Products Manufacturing	74,751
Perchloroethylene Dry Cleaning	67,960
Structure Fires	49,040
Paint Stripping Operations	48,680
Asphalt Paving: Cutback Asphalt	27,901

6.3 ON- AND OFF-ROAD MOBILE SOURCES

Table 6-4 lists the most emitted mobile source air toxics for 1996, 1999, and 2001(Freebairn 2001a). The on-road source inventory estimates vehicle emissions from automobiles, trucks, buses, and other motor vehicles traveling on established roads and highways in the City of St. Louis. The off-road source inventory estimates emissions from vehicles such as construction equipment.

Table 6-4 Top Ten Mobile Source Air Toxics Emitted in St. Louis.

	Emissions (tons/yr)		
Toxic Chemical	1996	1999	2001
Toluene	597	730	764
Benzene	407	438	458
MTBE	407	438	458
Xylene	383	411	430
Acetaldehyde	217	233	244
Formaldehyde	217	233	244
Butadiene	217	233	244
Ethylbenzene	96	103	108
N-Hexane	50	53	56
Styrene	22	24	25

7.0 RISK CHARACTERIZATION

Effectively addressing community health concerns must include consideration of many factors that may influence community health—indoor air quality, diet, smoking, access to medical care, lifestyle, and genetics. Risk characterization quantitatively estimates potential adverse health effects by integrating conclusions about hazard and dose responses with those from the exposure assessments. It also expresses a level of confidence in these conclusions by evaluating uncertainties associated with each aspect of the assessment.

This risk characterization derives solely from ambient monitoring of toxic air pollutants in the St. Louis metropolitan area. The monitoring effort applied multiple analytical methods to characterize ambient conditions for 113 analytes plus diesel particulate matter. Of these pollutants, 104 were HAPS. Sixty-one of these analytes had assigned cancer benchmarks, and 51 had assigned noncancer benchmarks (refer to Appendix A, Tables A1-2 and A1-3, respectively).

This risk characterization focuses on the five pollutants that exceeded the community-set health benchmarks: acetaldehyde, arsenic compounds as inorganic arsenic, benzene, chromium compounds as hexavalent chromium, and formaldehyde. The risk characterization for formaldehyde does not include ambient data collected during the first year or Phase I monitoring—refer to Sections 4.1 & 4.2 for a detailed discussion on why the monitoring results for Phase I were excluded from this review.

This characterization also assumes that individuals are exposed continuously at the same monitored annual concentration for 70, 30, or 15 years. Scientific evidence indicates that human beings exposed to benzene, arsenic compounds, and chromium compounds are at risk to develop cancer. Evidence exists that animals exposed to acetaldehyde and formaldehyde are at risk to develop cancer (though no definitive evidence of cancer risk to humans from these substances has emerged up to now).

EPA's Science Advisory Board (SAB) now is reviewing the formaldehyde risk assessment with an evaluation of the unit risk estimate (URE). The current URE for formaldehyde is based on animal studies conducted in 1983 (Kerns 1983). The Environmental Health Subcommittee of the SAB previously reviewed the EPA's risk assessment for formaldehyde in 1991. In 2003, the EPA's Office of Research and Development (ORD) responded to recommendations of that review and considered updates reflecting the current state of science. The ORD is preparing a toxicological review of formaldehyde that it will submit to the SAB for external review. A similar review concerning acetaldehyde is also planned.

The toxicological review will assess possible health effects from exposure to formaldehyde via oral and inhalation pathways. The review will examine the physiochemical characteristics of formaldehyde, its mode of action, and dose-response analyses; it will develop dosimetry models and a hazard characterization to reach major risk characterization conclusions. These conclusions will include a health risk estimate for lifetime exposures using available data endpoints to address life stages (for example, reproductive and developmental indices).

The review also will describe major uncertainties and research needs, and their possible impacts on future iterations of the risk assessment. Changes to the URE and Reference Concentration (RfC) derived for formaldehyde are likely outcomes of SAB's assessment of this review.

Cancer risks vary slightly for arsenic and chromium exposures. Ambient average concentrations for these pollutants are found right at the detection limit of 0.002 ug/m^3, which also serves as the 70-year excess cancer risk value. This analytical uncertainty contributes to the overall uncertainty in estimating the excess cancer risk for these pollutants. Also, daily variability or changing patterns in the ambient concentrations reported at each monitor for these pollutants indicate potential for local scale industrial influences—further affecting estimates of long-term cancer risks from these pollutants for residents living in the St. Louis metropolitan area. Moreover, estimates of these cancer risks depend on surrogate estimates for hexavalent chromium and inorganic arsenic (actual concentrations of these pollutants are unknown), thus generating an additional level of uncertainty.

In part, risk characterization quantitatively estimates potential cancer and noncancer risks that exposure to the ambient concentration of an analyte poses to the public. A review of the monitoring results for each pollutant (its annual average ambient concentration) did not identify any pollutant exceeding its respective noncancer benchmarks.

To evaluate the potential health impacts of ambient concentrations of acetaldehyde, benzene, formaldehyde, arsenic compounds, and chromium compounds, the CAP Partnership examined three factors that directly impact potential risks: (1) duration of exposure, (2) target level of cancer risk, and (3) additivity of cancer risk.

7.1 DURATION OF EXPOSURE

The St. Louis CAP Partnership decided to estimate potential cancer risk as a result of 70-, 30-, and 15-year exposure intervals. Because residents of the CAP study area reside in their domiciles for varying periods of time — a few months to many years — the CAP Partnership believed this approach would help residents relate more readily to the data.

7.2 TARGET LEVEL OF CANCER RISK

The CAP Partnership chose to establish the target level of an excess individual lifetime cancer risk at 1-in-100,000 (1×10^{-5}). If an air pollutant exceeded this level of risk, the CAP would develop and implement additional activities to reduce exposure. Although a target level of cancer risk of 1-in-1,000,000 reflects a more stringent criterion, analytical techniques cannot identify and quantify as many analytes at pollutant concentrations hypothesized to be associated with this target level as with a risk level of 1-in-100,000. The 1-in-100,000 target level of cancer risk therefore allowed the CAP Partnership to collect more useful data about more analytes and to identify air pollutants of potential concern.

7.3 ADDITIVITY OF CANCER RISK

To evaluate possible additivity of cancer risk, the CAP Partnership decided to apply two methods. The first method adds the cancer risks of all compounds classified as known human

carcinogens (Group A) separately from all compounds classified as likely (Group B) and possible (Group C) human carcinogens, using EPA's weight of evidence classification. This approach separates cancer risks of the *known* human cancer from the *probable* human cancer-causing analytes and the *possible* human cancer causing analytes. The second method adds the cancer risks of all analytes regardless of weight-of-evidence classification. Examining the data following both methods allows the CAP to account for differences in the weight-of-evidence classification and to incorporate potential changes in the NATA approach for calculating cancer risk.

7.4 RISK CHARACTERIZATION RESULTS

Table 7-1 characterizes excess cancer risks for St. Louis residents exposed to acetaldehyde, benzene, formaldehyde, arsenic compounds, and chromium compounds by comparing the annual average ambient concentrations of these analytes with the ambient concentration associated with a target level of cancer risk of 1-in-100,000 for the specified duration of exposure.

TABLE 7-1 – EXCESS CANCER RISK CHARACTERIZATION FOR THE ST. LOUIS CAP

Analyte	Annual Average Ambient Concentration	Cancer Benchmarks and Risk Associated with Measured Concentrations (Risk in 100,000)		
		70-Year	30-Year	15-Year
Arsenic Compounds	0.002 $\mu g/m^3$	0.002		
Risk in 100,000		1		
Chromium Compounds	0.002 $\mu g/m^3$	0.002		
Risk in 100,000		1		
Acetaldehyde	2.668 ppbv	2.5 ppbv		
Risk in 100,000		1		
Benzene	0.444 ppbv	0.41 ppbv		
Risk in 100,000		1		
Formaldehyde	3.72 ppbv	0.627 ppbv	1.46	2.93
Risk in 100,000		5.9	2.5	1.3

$\mu g/m^3$ Micrograms per cubic meter
ppbv Parts per billion by volume

For arsenic compounds, chromium compounds, acetaldehyde, and benzene, a cancer risk of one additional case of cancer in a human population of 100,000 was associated only with a 70-year exposure to the annual average ambient concentrations quantified in the CAP study. For the annual average ambient formaldehyde concentration quantified in the CAP study, cancer risks of 5.9, 2.5, and 1.3 additional cases of cancer in a human population of 100,000 were associated with exposures of 70, 30, and 15 years, respectively.

TABLE 7-2 – ADDITIVITY OF CANCER RISK FOR THE ST. LOUIS CAP

Analyte	Weight-of-Evidence Classification	Cancer Risk Associated With 70-Year Exposure (Risk in 100,000)
Arsenic Compounds	A	1
Benzene	A	1
Chromium Compounds	A	1
Total		3
Acetaldehyde	B2	1
Formaldehyde	B1	5.9
Total		6.9

* Weight-of-evidence classifications derive from EPA IRIS. Cancer risks associated with 70-year exposures are taken from Table 7-1. Classification A = known human cancer-causing analyte; Classification B1 = probable human cancer-causing agent based on limited human carcinogenicity data; Classification B2 = possible human cancer-causing agent based on sufficient animal carcinogenicity data.

Table 7-2 shows additivity of excess cancer risk for residents of St. Louis, within the weight-of-evidence classifications of analytes (see discussion of this in Section 7.3) and assumption of 70-year exposures. Table 7-2 shows that a 70-year exposure to *known* human cancer-causing

analytes in the A group may pose a total cancer risk of 3 additional cancer cases in a human population of 100,000. This risk is evenly divided among the three analytes. A 70-year exposure to the *probable* human cancer-causing analytes in the B group poses a total cancer risk of 6.9 additional cases of cancer in a human population of 100,000 — roughly two times greater than that of exposure to the known human cancer-causing analytes. Formaldehyde is the primary driver of cancer risk (represents the higher risk) from exposure to the probable human cancer-causing analytes. Thus, for a 70-year exposure at these annual average ambient concentrations, all analytes may pose a total cancer risk of 9.9 additional cases of cancer in a human population of 100,000.

To summarize, at the time of this report, the annual average ambient concentrations of acetaldehyde, benzene, formaldehyde, arsenic compounds, and chromium compounds may pose for the residents of St. Louis a total excess cancer risk equal to 9.9 additional cases of cancer in a human population of 100,000 following 70 years of exposure. Of these analytes, formaldehyde is the primary driver of the cancer risk. An evaluation of the data did not identify any pollutants that exceeded long-term noncancer benchmarks for the CAP study area. This study thus did not identify a significant noncancer health threat.

8.0 UNCERTAINTIES

This study does not provide a complete evaluation of all airborne pollutants that may be present in the study area, and thus may underestimate risk for exposed populations. Air quality issues that may impact residents of this study area and are not fully addressed by this study include: (1) spatial coverage; (2) exposure to toxic metals from PM_{10} particles that represent an inhalation risk to the upper respiratory system; (3) ground-level ozone and particulate matter; (4) short-term peak concentrations of toxic pollutants; and (5) indoor air concentrations of toxic pollutants. This study does not fully address spatial coverage because the St. Louis CAP initially focused on a specific area in St. Louis City. Only later was it expanded to include a larger part of the St. Louis urban area. This study does not fully address exposure to toxic metals from PM_{10} metals because the resources of the $PM_{2.5}$ speciation trends network were available at the time. As a consequence, initial metals measurements were made of $PM_{2.5}$ rather than PM_{10}. Later, PM_{10} metals sampling and analysis were initiated at Blair Street as a part of the National Air Toxics Trends Station (NATTS) program. The remaining three air quality issues were beyond the scope of the St. Louis CAP.

Two areas of uncertainty in this assessment are (1) detection limits/laboratory measurements for pollutants, and (2) calculation of health-based benchmarks. An estimated uncertainty level of up to +/- 25 percent is associated with laboratory measurements of many toxic compounds. The +/- 25 percent uncertainty in analytical results applies primarily to analytes measured at concentrations (parts per billion) near the MDL, the detection limit of the instrumentation. For analytes measured at concentrations greater than the MDL, including all of the analytes with average ambient concentrations greater than benchmark values except arsenic, the relative analytical uncertainty is smaller. A non-detectable concentration was recorded at 50 percent of the instrument detection limit if the analyte was detected at least 50 percent of the time during the sampling period (refer to Appendix B). A number of compounds however, such as acrolein, which was not sampled nor analyzed, cannot be detected using available laboratory techniques.

Development of health benchmarks typically relies on controlled studies that demonstrate the health effects of air pollutants on human and/or animal subjects. Occupational exposure studies are the primary sources of human exposure data and are used frequently as bases for developing health benchmark values. When relevant occupational data are not available for a compound, risk assessors may use results from controlled animal studies to model potential adverse effects on humans. For many compounds, the lack of information regarding the actual dose-response relationship between a contaminant and an exposed human can generate uncertainty over the protectiveness of the benchmarks. To provide a protective level in developing health benchmarks, therefore, "uncertainty factors" are developed to improve accuracies of: (1) extrapolation of results from animals to human; (2) uncertainty that the most sensitive health effect was observed; and (3) extrapolation of results from adults to sensitive populations such as children and the elderly. Thus, accuracy of health-based benchmarks depends upon the number, type, and integrity of completed health studies.

9.0 SUMMARY OF CONCLUSIONS AND RECOMMENDATIONS

This phase of Community Air Project monitoring was successful. It achieved the primary objective of effectively monitoring the CAP pollutants and comparing their concentrations to health benchmarks developed by the Technical Team and accepted by the CAP Partnership. An added success of the CAP was improved communication among the community and the state and federal agencies—the EPA and the Missouri Department of Natural Resources communicated data and complex issues in a language that resonated with the community, while the community in turn communicated its concerns, first hand observations of air quality in St. Louis, and became comfortable with some of the technical aspects of air toxics.

The CAP monitoring sampled and analyzed ambient air toxics that included many HAPs. As discussed in Section 2.0, 113 analytes—including 104 HAPs and all but one of the 33 Urban Air Toxics—were analyzed during the first year of CAP sampling. Of these 113 analytes, no conclusion can be drawn about risk from 17 of them (see Section 3.0 and Appendix A): 14 were never detected but have a 70-Bench concentration less than the MDL, 3 were detected less than half the time and have a 70-Bench concentration less than the MDL. Ninety-one analytes were either not detected, were detected less than half the time at low concentrations, or had average concentrations less than either the 70-Bench or noncancer benchmark concentration if applicable.

Analysis of the first year of sampling utilized current health studies, EPA benchmarks, and led to the development of an enhanced formaldehyde monitoring network in the St. Louis metropolitan area. As described in greater detail in Section 7.0, the results identified five sampled analytes with average measured concentrations equal to or greater than the 70-Bench concentration, indicating an increased cancer risk greater than 1 in 100,000. The analytes identified were benzene, formaldehyde, acetaldehyde, arsenic compounds, and chromium compounds. Of these 5 analytes, only formaldehyde had an average concentration significantly greater than the cancer 70-Bench concentration. Revised monitoring was begun in December 2002 to examine formaldehyde levels in greater detail. The additional formaldehyde monitoring resulted in three monitors each recording much lower values than the previous 2001 single monitor recorded. As a result, the CAP Partnership has chosen to disregard the Phase I formaldehyde data and use data from Phase II (see Sections 4.1 & 4.2 for further discussion) for risk characterization. Even with the Phase II data, formaldehyde concentrations and the associated risk remain the greatest out of all CAP-monitored pollutants.

The CAP chemicals of concern were also found at significant levels nationally in urban areas, as indicated in the most recent Air Toxics monitoring Policy-Relevant White Paper for the Lake Michigan Air Director's Consortium (Sonoma Technology 2004). "The typical urban ambient data range exceeds the cancer benchmarks for acetaldehyde, formaldehyde, 1,3-butadiene, benzene, carbon tetrachloride, arsenic, and chromium." While 1,3-butadiene and carbon tetrachloride are not identified as CAP chemicals of concern, an excess cancer risk of one in a million was identified with monitored concentrations for those two pollutants in the CAP study area.

In addition to the community actions to reduce air toxics as outlined in Appendix D, from the data and information available now, we can make some recommendations for ongoing ambient air monitoring of toxics in the future:

1. The CAP Partnership's evaluation of pollutant sources of acetaldehyde, arsenic compounds, benzene, chromium compounds, diesel particulate matter, and formaldehyde has led them to consider actions that would help limit impacts of these pollutants of concern.

   Reorganization to initiate these actions has begun. Monitoring and data analysis to support this effort should continue along with evaluation of ambient air toxics in Missouri. Emission inventories for these pollutants of concern should improve locally and nationally. Ultimately, source apportionment methods must be applied to evaluate where ambient air toxics of concern are coming from.

2. A 24-hour time resolved sampling as in the CAP limits our ability to determine the sources and meteorology of observed pollutant concentrations. While the sampling indicates some general areas of emissions and characteristics, no clear determination of source-specific impacts is yet possible from the available data. New technologies developed for the $PM_{2.5}$ Supersites Program, however, may provide ambient air data that may be used to identify sources of arsenic and chromium. For this reason, integration of the national particulate matter monitoring program into the national air toxics monitoring program should continue.

3. Enhanced speciation monitoring methods (PM_{10}) for arsenic and chromium compounds (hexavalent chrome) are needed to refine our risk characterization through improved detection limits. This ability will improve our capability to identify local emission sources for this pollutant. Also, refined emission inventories for these pollutants should be developed to improve our ability to model local point and area sources.

Formaldehyde monitoring data collected at three locations (Grant School, Blair St., and Bonne Terre) in the CAP Phase II monitoring showed fairly consistent pollutant levels among the three sites—indicating not only urban scale but also even regional impacts of this pollutant. Given the need to more clearly evaluate concentrations related to the benchmark and evaluate short-term risk, monitoring in the next year will focus on the Blair St. NATTS area. This will provide solid evidence of formaldehyde levels affecting the region from all sources.

Considering EPA's approach to continued assessment of toxics, changes in monitoring methods may occur within the next few years along with alteration of our understanding of risks related to ambient air toxins. Toxics monitoring at the Blair St. National Air Toxics Trends Site and other future sampling may lead to refocus of issues. As we continue to increase our knowledge in science and apply new technology and health data, we will improve our risk characterization for the St. Louis area and the State of Missouri. In the meantime, the information provided by the CAP suggests closer examination, concern, and action to reduce levels of benzene, formaldehyde, acetaldehyde, arsenic compounds, chromium compounds, and diesel particulate matter is in order to protect the health of those living in St. Louis. By undertaking such actions as outlined in Section 4 and Appendix D, we may reduce the risk of cancer and improve the health of all those breathing the air in St. Louis.

10.0 REFERENCES

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APPENDIX A

Appendix A: Hazardous Air Pollutants and Cancer and Noncancer Benchmarks

Table A1-1 lists the 188 HAPs and indicates urban air toxics and CAP analytes. The HAPs in the lower part of the list and not marked as CAP analytes were not measured as a part of the CAP, so this project cannot draw conclusions about risks from these substances.

Five urban air toxics not included on the St. Louis CAP analyte list were acrolein, polychlorinated biphenyls, quinoline, beryllium compounds, and coke oven emissions. In general, these analytes are not part of the standard sets of analytes for the analytical methods used in the CAP. Methods for sampling and analyzing acrolein are however under development, and sampling and analyzing polychlorinated biphenyls was conducted during a limited period as part of the CAP. Adding these five analytes to these sets therefore would have made sampling and analysis particularly expensive.

Cancer and chronic noncancer benchmark values are listed in Table A1-2 and Table A1-3, respectively. The tables identify bases for calculation of the benchmarks (as discussed in Section 2.0).

Table A1-1. List of Hazardous Air Pollutants with Various Characteristics Indicated

CAS Number	Chemical Name	Urban Air Toxics	CAP Analyte	VOC	SVOC	Carbonyl	PM2.5
75070	Acetaldehyde	X	X			X	
75058	Acetonitrile		X	X			
98862	Acetophenone		X		X		
107131	Acrylonitrile	X	X	X			
92671	4-Aminobiphenyl		X		X		
62533	Aniline		X		X		
71432	Benzene	X	X	X			
92875	Benzidine		X		X		
117817	Bis(2-ethylhexyl)phthalate (DEHP)		X		X		
75252	Bromoform		X	X			
106990	1,3-Butadiene	X	X	X			
56235	Carbon tetrachloride	X	X	X			
108907	Chlorobenzene		X	X			
510156	Chlorobenzilate		X		X		
67663	Chloroform	X	X	X			
126998	Chloroprene		X	X			
98828	Cumene		X	X			
132649	Dibenzofurans		X		X		
84742	Dibutylphthalate		X		X		
106467	1,4-Dichlorobenzene(p)		X		X		
91941	3,3-Dichlorobenzidene		X		X		
542756	1,3-Dichloropropene	X	X	X			
60117	Dimethyl aminoazobenzene		X		X		
119937	3,3'-Dimethyl benzidine		X		X		
131113	Dimethyl phthalate		X		X		
51285	2,4-Dinitrophenol		X		X		
121142	2,4-Dinitrotoluene		X		X		
140885	Ethyl acrylate		X	X			
100414	Ethyl benzene		X	X			
107062	Ethylene dichloride (1,2-Dichloroethane)	X	X	X			
50000	Formaldehyde	X	X			X	
118741	Hexachlorobenzene	X	X		X		
87683	Hexachlorobutadiene		X		X		
77474	Hexachlorocyclopentadiene		X		X		
67721	Hexachloroethane		X		X		
110543	Hexane		X	X			
78591	Isophorone		X		X		
78933	Methyl ethyl ketone (2-Butanone)		X	X			
108101	Methyl isobutyl ketone (Hexone)		X	X			
80626	Methyl methacrylate		X	X			
1634044	Methyl tert butyl ether		X	X			
75092	Methylene chloride (Dichloromethane)	X	X	X			
91203	Naphthalene		X		X		
98953	Nitrobenzene		X		X		

Table A1-1. Continued

CAS Number	Chemical Name	Urban Air Toxics	CAP Analyte	VOC	SVOC	Carbonyl	PM2.5
100027	4-Nitrophenol		X		X		
62759	N-Nitrosodimethylamine		X		X		
82688	Pentachloronitrobenzene (Quintobenzene)		X		X		
87865	Pentachlorophenol		X		X		
108952	Phenol		X		X		
123386	Propionaldehyde		X			X	
78875	Propylene dichloride (1,2-Dichloropropane)	X	X	X			
100425	Styrene		X	X			
1746016	2,3,7,8-Tetrachlorodibenzo-p-dioxin	X	X		X		
79345	1,1,2,2-Tetrachloroethane	X	X	X			
127184	Tetrachloroethylene (Perchloroethylene)	X	X	X			
108883	Toluene		X	X			
95534	o-Toluidine		X	X			
120821	1,2,4-Trichlorobenzene		X	X			
79005	1,1,2-Trichloroethane		X	X			
79016	Trichloroethylene	X	X	X			
95954	2,4,5-Trichlorophenol		X		X		
88062	2,4,6-Trichlorophenol		X		X		
75014	Vinyl chloride	X	X	X			
1330207	Xylenes (isomers and mixture)		X	X			
95476	o-Xylenes		X	X			
108383	m-Xylenes		X	X			
106423	p-Xylenes		X	X			
0	Antimony Compounds		X				X
0	Arsenic Compounds (inorganic including arsine)	X	X				X
0	Cadmium Compounds	X	X				X
0	Chromium Compounds	X	X				X
0	Cobalt Compounds		X				X
0	Lead Compounds	X	X				X
0	Manganese Compounds	X	X				X
0	Mercury Compounds	X	X				X
0	Nickel Compounds	X	X				X
0	Polycylic Organic Matter4	X	X		X		
0	Selenium Compounds		X				X
60355	Acetamide						
53963	2-Acetylaminofluorene				X		
107028	Acrolein	X		X			
79061	Acrylamide			X			
79107	Acrylic acid			X			
107051	Allyl chloride			X			
90040	o-Anisidine						
1332214	Asbestos						
98077	Benzotrichloride			X			
100447	Benzyl chloride			X			
92524	Biphenyl				X		
542881	Bis(chloromethyl)ether			X			
156627	Calcium cyanamide						
133062	Captan						
63252	Carbaryl						
75150	Carbon disulfide			X			
463581	Carbonyl sulfide			X			

Table A1-1. Continued

CAS Number	Chemical Name	Urban Air Toxics	CAP Analyte	VOC	SVOC	Carbonyl	PM2.5
120809	Catechol			X			
133904	Chloramben						
57749	Chlordane						
7782505	Chlorine						X
79118	Chloroacetic acid			X			
532274	2-Chloroacetophenone						
107302	Chloromethyl methyl ether			X			
1319773	Cresols/Cresylic acid (isomers and mixture)			X			
95487	o-Cresol			X			
108394	m-Cresol				X		
106445	p-Cresol				X		
94757	2,4-D, salts and esters						
3547044	DDE						
334883	Diazomethane			X			
96128	1,2-Dibromo-3-chloropropane			X			
111444	Dichloroethyl ether (Bis(2-chloroethyl)ether)				X		
62737	Dichlorvos						
111422	Diethanolamine						
121697	N,N-Diethyl aniline (N,N-Dimethylaniline)			X			
64675	Diethyl sulfate			X			
119904	3,3-Dimethoxybenzidine						
79447	Dimethyl carbamoyl chloride			X			
68122	Dimethyl formamide			X			
57147	1,1-Dimethyl hydrazine			X			
77781	Dimethyl sulfate			X			
534521	4,6-Dinitro-o-cresol, and salts						
123911	1,4-Dioxane (1,4-Diethyleneoxide)			X			
122667	1,2-Diphenylhydrazine						
106898	Epichlorohydrin (l-Chloro-2,3-epoxypropane)			X			
106887	1,2-Epoxybutane			X			
51796	Ethyl carbamate (Urethane)			X			
75003	Ethyl chloride (Chloroethane)			X			
106934	Ethylene dibromide (Dibromoethane)	X		X			
107211	Ethylene glycol						
151564	Ethylene imine (Aziridine)			X			
75218	Ethylene oxide	X		X			
96457	Ethylene thiourea						
75343	Ethylidene dichloride (1,1-Dichloroethane)			X			
76448	Heptachlor						
822060	Hexamethylene-1,6-diisocyanate						
680319	Hexamethylphosphoramide						
302012	Hydrazine						
7647010	Hydrochloric acid						
7664393	Hydrogen fluoride (Hydrofluoric acid)						
123319	Hydroquinone						
58899	Lindane (all isomers)						
108316	Maleic anhydride						

Table A1-1. Continued

CAS Number	Chemical Name	Urban Air Toxics	CAP Analyte	VOC	SVOC	Carbonyl	PM2.5
67561	Methanol			X			
72435	Methoxychlor						
74839	Methyl bromide (Bromomethane)			X			
74873	Methyl chloride (Chloromethane)			X			
71556	Methyl chloroform (1,1,1-Trichloroethane)			X			
60344	Methyl hydrazine	X		X			
74884	Methyl iodide (Iodomethane)			X			
624839	Methyl isocyanate			X			
101144	4,4-Methylene bis(2-chloroaniline)						
101688	Methylene diphenyl diisocyanate (MDI)						
101779	4,4¬-Methylenedianiline						
92933	4-Nitrobiphenyl				X		
79469	2-Nitropropane			X			
684935	N-Nitroso-N-methylurea			X			
59892	N-Nitrosomorpholine			X			
56382	Parathion						
106503	p-Phenylenediamine						
75445	Phosgene			X			
7803512	Phosphine						
7723140	Phosphorus						X
85449	Phthalic anhydride						
1336363	Polychlorinated biphenyls (Aroclors)	X			X		
1120714	1,3-Propane sultone			X			
57578	beta-Propiolactone			X			
114261	Propoxur (Baygon)						
75569	Propylene oxide			X			
75558	1,2-Propylenimine (2-Methyl aziridine)						
91225	Quinoline	X					
106514	Quinone						
96093	Styrene oxide			X			
7550450	Titanium tetrachloride						X
95807	2,4-Toluene diamine						
584849	2,4-Toluene diisocyanate						
8001352	Toxaphene (chlorinated camphene)						
121448	Triethylamine			X			
1582098	Trifluralin						
540841	2,2,4-Trimethylpentane			X			
108054	Vinyl acetate			X			
593602	Vinyl bromide			X			
75354	Vinylidene chloride (1,1-Dichloroethylene)			X			
	Beryllium Compounds	X					X
	Coke Oven Emissions	X			X		
	Cyanide Compounds1			X			
	Glycol ethers2			X			
	Fine mineral fibers3				X		
	Radionuclides (including radon)5			X			

NOTE: For all listings above which contain the word "compounds" and for glycol ethers, the following applies: Unless otherwise specified, these listings are defined as including any unique chemical substance that contains the named chemical (i.e., antimony, arsenic, etc.) as part of that chemical's infrastructure.

1 X'CN where X = H' or any other group where a formal dissociation may occur. For example KCN or Ca(CN)2

2 Includes mono- and di- ethers of ethylene glycol, diethylene glycol, and triethylene glycol R-(OCH2CH2)n -OR' where n = 1, 2, or 3

R = alkyl or aryl groups
R′ = R, H, or groups, which when removed, yield glycol ethers with the structure: R-(OCH2CH2)n-OH. Polymers are excluded from the glycol ether category.

3 Includes mineral fiber emissions from facilities manufacturing or processing glass, rock, or slag fibers (or other mineral derived fibers) of average diameter 1 micrometer or less.

4 Includes organic compounds with more than one benzene ring, and which have a boiling point greater than or equal to 100º C.

5 A type of atom which spontaneously undergoes radioactive decay.

VOC = volatile organic compound

SVOC = semi-volatile organic compound

Carbonyl = compound containing carbonyl group; that is, a carbon atom double bonded to an oxygen atom

PM2.5 = particulate matter having an aerodynamic diameter smaller than 2.5 micrometers

Table A1-2. Cancer Health Benchmarks

		WEIGHT OF EVIDENCE		REFERENCE VALUES		RISK BASED CONCENTRATION (1:100,000)			
CHEMICAL NAME	CAS NO	EPA	IARC	Detection Limit ug/m^3	Unit Risk Estimate 1/(ug/m^3)	70 year exposure duration ug/m^3	30 year exposure duration ug/m^3	15 year exposure duration ug/m^3	SOURCE
Metals									
Arsenic compounds	7440-38-2	A	1	0.002	4.3E-03	0.002	0.005	0.011	EPA-IRIS
Cadmium compounds	7440-43-9	B1	1	0.011	1.8E-03	0.006	0.013	0.026	EPA-IRIS
Chromium compounds	7440-47-3	A	1	0.002	4.2E-03	0.002	0.006	0.011	EPA-IRIS
Lead compounds	7439-92-1	B2	2B	0.005	1.2E-05	0.830	1.94	3.87	CAL-EPA
Nickel compounds	7440-02-0	A	2B	0.001	3.1E-04	0.032	0.075	0.149	CAL-EPA
Toxicity Values Unavailable									
Metals									
Antimony compounds	7440-36-0	-	2B	0.015					
Cobalt compounds	7440-48-4	-	-	0.001					
Manganese compounds	7439-96-5	D	-	0.002					
Mercury compounds	7439-97-6	D	3	0.004					
Selenium compounds	7782-49-2	D	3	0.002					

		WEIGHT OF EVIDENCE		REFERENCE VALUES		RISK BASED CONCENTRATION (1:100,000)			
CHEMICAL NAME	CAS NO	EPA	IARC	Detection Limit ppb	Unit Risk Estimate 1/(ppb)	70 year exposure duration ppb	30 year exposure duration ppb	15 year exposure duration ppb	SOURCE
Polycyclic Aromatic Hydrocarbons (PAHs)									
Benzo(a)anthracene	56-55-3	B2	2A	0.004	1.0E-03	0.0097	0.023	0.045	CAL-EPA
Benzo(a)pyrene	50-32-8	B2	2A	0.009	1.1E-02	0.0009	0.002	0.004	CAL-EPA
Benzo(b)fluoranthene	205-99-2	B2	2B	0.009	1.1E-03	0.0088	0.021	0.041	CAL-EPA
Benzo(k)fluoranthene	207-08-9	B2	2B	0.012	1.1E-03	0.0088	0.021	0.041	CAL-EPA
Chrysene	218-01-9	B2	3	0.006	1.0E-04	0.0975	0.227	0.455	CAL-EPA
Dibenz(a,h)anthracene	53-70-3	B2	2A	0.007	1.4E-02	0.0007	0.002	0.003	CAL-EPA
7,12-Dimethylbenz(a)anthracene	57-97-6			0.009	6.7E-03	0.0015	0.004	0.007	CAL-EPA
Indeno(1,2,3-cd)pyrene	193-39-5	B2	2B	0.006	1.2E-03	0.0081	0.019	0.038	CAL-EPA
Organic Compounds									
Acetaldehyde	75-07-0	B2	2B	0.007	4.0E-06	2.5	5.8	11.7	EPA-IRIS
2-Acetylaminofluorene	53-96-3			0.006	1.4E-04	0.07	0.17	0.33	CAL-EPA
Acrylonitrile	107-13-1	B1	2A	0.210	1.4E-04	0.069	0.16	0.32	EPA-IRIS
Aniline	62-53-3	B2	3	0.021	6.0E-06	1.7	3.9	7.7	CAL-EPA
Azobenzene	103-33-3	B2		0.012	4.2E-06	2.40	5.60	11.20	EPA-IRIS
Benzene	71-43-2	A	1	0.040	2.5E-05	0.41	0.95	1.90	EPA-IRIS
Benzidine	92-87-5	A	1	0.000	5.0E-01	1.99E-05	4.64E-05	9.29E-05	EPA-IRIS
Bis(2-chloroethyl) ether	111-44-4	B2	3	0.010	1.2E-04	0.08	0.19	0.39	CAL-EPA
Bis(2-ethylhexyl)phthalate	117-81-7	B2	2B	0.004	3.8E-05	0.26	0.61	1.23	CAL-EPA
Bromodichloromethane	75-27-4	B2		0.060	5.5E-06	1.8	4.2	8.5	CAL-EPA
Bromoform	75-25-2	B2	3	0.080	1.1E-05	0.88	2.05	4.11	EPA-IRIS
1,3-Butadiene	106-99-0	A	2A	0.070	6.7E-05	0.15	0.35	0.70	EPA-ORD
Carbon tetrachloride	56-23-5	B2	2B	0.080	9.4E-05	0.11	0.25	0.50	EPA-IRIS
Chlorobenzilate	510-15-6	B2	3	0.007	5.8E-06	1.7	4	8	HEAST
Chloroform	67-66-3	B2	2B	0.050	1.1E-04	0.09	0.21	0.41	EPA-IRIS

Table A1-2 continued. Cancer Health Benchmarks

		WEIGHT OF EVIDENCE		REFERENCE VALUES		RISK BASED CONCENTRATION (1:100,000)			
CHEMICAL NAME	CAS NO	EPA	IARC	Detection Limit ppb	Unit Risk Estimate 1/(ppb)	70 year exposure duration ppb	30 year exposure duration ppb	15 year exposure duration ppb	SOURCE
Chloromethane	74-87-3	C	3	0.060	3.7E-06	2.7	6.3	12.7	HEAST
1,2-Dibromoethane	106-93-4	B2	2A	0.080	1.7E-03	0.006	0.014	0.027	EPA-IRIS
p-Dichlorobenzene	106-46-7	-	2B	0.090	6.6E-05	0.151	0.35	0.71	CAL-EPA
3,3'-Dichlorobenzidine	91-94-1	B2		0.004	3.6E-03	0.003	0.007	0.013	CAL-EPA
1,1-Dichloroethane	75-34-3	C		0.080	4.0E-07	25	58	117	CAL-EPA
1,2-Dichloroethane	107-06-2	B2	2B	0.060	1.1E-04	0.09	0.22	0.44	EPA-IRIS
cis-1,3-Dichloropropene	542-75-6	B2	2B	0.100	1.8E-05	0.55	1.29	2.57	EPA-IRIS
4-Dimethylaminoazobenzene	60-11-7	-	2B	0.007	1.2E-02	0.001	0.0019	0.0039	CAL-EPA
2,4-Dinitrotoluene	121-14-2	B2		0.007	6.8E-04	0.015	0.034	0.069	CAL-EPA
Formaldehyde	50-00-0	B1	2A	0.004	1.6E-05	0.627	1.46	2.93	EPA-IRIS
Hexachlorobenzene	118-74-1	B2	2B	0.006	5.3E-03	0.002	0.004	0.009	EPA-IRIS
Hexachloro-1,3-butadiene	87-68-3	C	3	0.060	2.4E-04	0.042	0.098	0.197	EPA-IRIS
Hexachloroethane	67-72-1	C	3	0.003	3.9E-05	0.258	0.602	1.20	EPA-IRIS
3-Methylcholanthrene	56-49-5			0.005	5.6E-04	0.018	0.042	0.084	CAL-EPA
Methyl methanesulfonate	66-27-3		2B	0.018	6.2E-06	1.6	3.76	7.51	CAL-EPA
Methyl tert-butyl ether	1634-04-4	-		0.180	7.2E-08	139.0	324	649	CAL-EPA
Methylene chloride	75-09-2	B2	2B	0.060	1.7E-06	6.0	14	28	EPA-IRIS
N-Nitrosodiethylamine	55-18-5	B2		0.020	2.1E-01	4.80E-05	1.1E-04	2.2E-04	EPA-IRIS
N-Nitrosodimethylamine	62-75-9	B2		0.043	4.3E-02	2.30E-04	5.37E-04	0.0011	EPA-IRIS
N-Nitroso-di-n-butylamine	924-16-3	B2		0.017	1.1E-02	9.3E-04	0.002	0.004	EPA-IRIS
N-Nitrosopiperidine	100-75-4		2B	0.019	5.9E-04	0.017	0.040	0.079	CAL-EPA
N-Nitrosopyrrolidine	930-55-2	B2		0.017	2.0E-03	0.005	0.012	0.023	EPA-IRIS
o-Toluidine	95-53-4		2B	0.015	2.2E-04	0.05	0.105	0.210	CAL-EPA
Pentachlorophenol	87-86-5	B2	2B	0.007	5.4E-05	0.184	0.43	0.86	CAL-EPA
Phenacetin	62-44-2		2A	0.011	6.3E-07	15.87	37	74	CAL-EPA
Safrole	94-59-7		2B	0.011	9.5E-06	1.05	2.45	4.90	CAL-EPA
2,3,7,8-Tetrachlorodibenzo-*p*-dioxin	1746-01-6	B2		7.59E-09	4.4E+02	2.3E-08	5.32E-08	1.06E-07	EPA-ORD
1,1,2,2-Tetrachloroethane	79-34-5	C	3	0.060	4.0E-04	0.02	0.06	0.12	EPA-IRIS
Tetrachloroethylene	127-18-4	B2-C	2B	0.060	3.8E-05	0.27	0.62	1.24	CAL-EPA
1,1,2-Trichloroethane	79-00-5	C	3	0.060	8.7E-05	0.12	0.27	0.54	EPA-IRIS
Trichloroethylene	79-01-6	B2-C	3	0.070	1.1E-05	0.93	2.17	4.34	CAL-EPA
2,4,6-Trichlorophenol	88-06-2	B2		0.005	2.5E-05	0.40	0.92	1.85	EPA-IRIS
Vinyl chloride	75-01-4	A	1	0.060	2.3E-05	0.43	1.00	2.01	EPA-IRIS
Toxicity Values Unavailable									
Polycyclic Aromatic Hydrocarbons (PAHs)									
Acenaphthene	83-32-9	-	-	0.005					
Acenaphthylene	208-96-8	D	-	0.007					
Anthracene	120-12-7	D	3	0.012					
Benzo(g,h,i)perylene	191-24-2	D	3	0.007					
Fluoranthene	206-44-0	D	3	0.009					
Fluorene	86-73-7	D	3	0.007					

Table A1-2 continued. Cancer Health Benchmarks

		WEIGHT OF EVIDENCE		REFERENCE VALUES		RISK BASED CONCENTRATION (1:100,000)			
CHEMICAL NAME	CAS NO	EPA	IARC	Detection Limit ppb	Unit Risk Estimate 1/(ppb)	70 year exposure duration ppb	30 year exposure duration ppb	15 year exposure duration ppb	SOURCE
Naphthalene	91-20-3	C	-	0.015					
Phenanthrene	85-01-8	D	3	0.007					
Pyrene	129-00-0	D	3	0.006					
Organic Compounds									
Acetonitrile	75-05-8	D	-	0.250					
Acetophenone	98-86-2			0.014					
4-Aminobiphenyl	92-67-1	-	1	0.007					
Bromomethane	74-83-9	D	3	0.090					
Chlorobenzene	108-90-7	D	-	0.060					
Chloroethane	75-00-3	B2	3	0.080					
Chloroprene	126-99-8	-	2B	0.100					
Dibenzofuran	132-64-9	D	-	0.007					
1,2-Dichloropropane	78-87-5	B2	3	0.070					
Dimethyl phthalate	131-11-3	D	-	0.006					
Di-n-Butyl phthalate	84-74-2	D		0.006					
2,4-Dinitrophenol	51-28-5	-	-	0.007					
Diesel particulates	NA	B	2A						
Ethyl acrylate	140-88-5	B2	2B	0.160					
Ethylbenzene	100-41-4	D	2B	0.040					
Hexachlorocyclopentadiene	77-47-4	E	-	0.010					
Isophorone	78-59-1	C	-	0.019					
Methyl ethyl ketone	78-93-3	D	-	0.150					
Methyl isobutyl ketone	108-10-1	-	-	0.150					
Methyl methacrylate	80-62-6	E	3	0.180					
2-Methylphenol	95-48-7			0.180					
4-Methylphenol	106-44-5			0.180					
n-Hexane	110-54-3	-	-	0.130					
2-Nitroaniline	88-74-4			0.011					
Nitrobenzene	98-95-3		2A	0.025					
4-Nitrophenol	100-02-7			0.006					
N-Nitrosodi-n-propylamine	621-64-7	B2	2B	0.020					
Pentachloronitrobenzene	82-68-8	-	3	0.004					
Phenol	108-95-2	D	3	0.029					
Propionaldehyde	123-38-6	-	-	0.001					
Propylene	115-07-1			0.050					
Styrene	100-42-5	-	2B	0.070					
Toluene	108-88-3	D	3	0.060					
1,2,4-Trichlorobenzene	120-82-1	D	-	0.060					
1,1,1-Trichloroethane	71-55-6	D	-	0.060					
2,4,5-Trichlorophenol	95-95-4	-	2B	0.004					
2,2,4-Trimethylpentane	540-84-1	-		0.108					
Xylenes (*m*,*p*)	1330-20-7	D	3	0.050					
Xylenes (*o*)	95-47-6	D	3	0.050					

No Toxicity Data

Acronyms:
EPA = Environmental Protection Agency
IARC = International Agency for Research on Cancer
IRIS = Integrated Risk Information System as of September 2002
CAL-EPA = California Environmental Protection Agency
ORD = Office of Research and Development
HEAST = Health Effects Assessment Summary Tables

Weight of Evidence:
Groups A and 1 = Known Human Carcinogen
Groups B and 2A = Probable Human Carcinogen
Groups C and 2B = Possible Human Carcinogen
Groups D and 3 = Not Classified as to Human Carcinogenicity
Groups E and 4 = Noncarcinogenic for Humans

Table A1-3. Noncancer Benchmarks			Reference Concentration (RfC) or Equivalent Value	
Chemical Name	CAS NO.	Detection Limit (ug/m^3)	(ug/m^3)	Source
Metals				
Antimony Compounds	7440-36-0	0.0150	0.2	EPA-IRIS
Arsenic Compounds	7440-38-2	0.0020	0.03	CAL-EPA
Cadmium Compounds	7440-43-9	0.0110	0.02	CAL-EPA
Chromium Compounds	7440-47-3	0.0020	0.29	EPA-IRIS
Lead Compounds	7439-92-1	0.0050	1.5	EPA-NAAQS
Manganese Compounds	7439-96-5	0.0020	0.05	EPA-IRIS
Mercury Compounds	7439-97-6	0.0040	0.3	EPA-IRIS
Nickel Compounds	7440-02-0	0.0010	0.2	ATSDR
Diesel Particulates			5	EPA-IRIS
Chemical Name	CAS NO.	Detection Limit (ppbv)	Reference Concentration (RfC) or Equivalent Value (ppbv)	Source
Polycyclic Aromatic Hydrocarbons (PAHs)				
Naphthalene	91-20-3	0.015	0.57	EPA-IRIS
Organic Compounds				
Acetaldehyde	75-07-0	0.007	5	EPA-IRIS
Acetonitrile	75-05-8	0.25	36	EPA-IRIS
Acrylonitrile	107-13-1	0.21	0.92	EPA-IRIS
Aniline	62-53-3	0.021	0.26	EPA-IRIS
Benzene	71-43-2	0.04	19	EPA-ORD
Bromomethane	74-83-9	0.09	1.29	EPA-IRIS
1,3-Butadiene	106-99-0	0.07	0.90	EPA-ORD
Carbon Tetrachloride	56-23-5	0.08	6.36	CAL-EPA
Chlorobenzene	108-90-7	0.06	217	CAL-EPA
Chloroethane	75-00-3	0.08	3790	EPA-IRIS
Chloroform	67-66-3	0.05	20	ATSDR
Chloromethane	74-87-3	0.06	48	ATSDR
Chloroprene	126-99-8	0.10	1.93	HEAST
1,2-Dibromoethane	106-93-4	0.08	0.10	CAL-EPA
p - Dichlorobenzene	106-46-7	0.09	133	EPA-IRIS
1,2-Dichloroethane	107-06-2	0.06	593	ATSDR
1,2-Dichloropropane	78-87-5	0.07	0.87	EPA-IRIS
cis-1,3-Dichloropropene	542-75-6	0.10	4.41	EPA-IRIS
Ethylbenzene	100-41-4	0.04	230	EPA-IRIS
Formaldehyde	50-00-0	0.004	7.98	ATSDR
Hexachlorobenzene	118-74-1	0.006	0.26	CAL-EPA
Hexachlorocyclopentadiene	77-47-4	0.010	0.20	EPA-IRIS
n-Hexane	110-54-3	0.130	57	EPA-IRIS
Isophorone	78-59-1	0.019	354	CAL-EPA
Methyl Ethyl Ketone	78-93-3	0.15	339	EPA-IRIS
Methyl Isobutyl Ketone	108-10-1	0.15	20	HEAST
Methyl Methacrylate	80-62-6	0.18	171	EPA-IRIS

2-Methylphenol	95-48-7	0.18	136	CAL-EPA
Methyl Tert-Butyl Ether	1634-04-4	0.18	832	EPA-IRIS
Methylene Chloride	75-09-2	0.06	288	ATSDR
2-Nitroaniline	88-74-4	0.011	0.035	HEAST
Phenol	108-95-2	0.029	50	CAL-EPA
Propylene	115-07-1	0.05	1746	CAL-EPA
Styrene	100-42-5	0.07	235	EPA-IRIS
Tetrachloroethylene	127-18-4	0.06	40	ATSDR
Toluene	108-88-3	0.06	106	EPA-IRIS
1,2,4-Trichlorobenzene	120-82-1	0.06	27	HEAST
1,1,1-Trichloroethane	71-55-6	0.06	183	CAL-EPA
Trichloroethylene	79-01-6	0.07	112	CAL-EPA
Vinyl Chloride	75-01-4	0.06	39	IRIS
Xylenes (m,p)	1330-20-7	0.05	33	ATSDR
Xylenes (o)	95-47-6	0.05	33	ATSDR
Toxicity Values Unavailable				
Metals				
Cobalt Compounds	7440-48-4	0.001		
Selenium Compounds	7782-49-2	0.002		
Polycyclic Aromatic Hydrocarbons (PAHs)				
Acenaphthene	83-32-9	0.005		
Acenaphthylene	208-96-8	0.007		
Acetophenone	98-86-2	0.014		
Anthracene	120-12-7	0.012		
Benzo(g,h,i)perylene	191-24-2	0.007		
Fluoranthene	206-44-0	0.009		
Benzo(a)anthracene	56-55-3	0.004		
Benzo(a)pyrene	50-32-8	0.009		
Benzo(b)fluoranthene	205-99-2	0.009		
Benzo(k)fluoranthene	207-08-9	0.012		
Chrysene	218-01-9	0.006		
Dibenz(a,h)anthracene	53-70-3	0.007		
7,12-Dimethylbenz(A)Anthracene	57-97-6	0.009		
Fluorene	86-73-7	0.007		
Indeno(1,2,3-cd)pyrene	193-39-5	0.006		
Phenanthrene	85-01-8	0.007		
Pyrene	129-00-0	0.006		
Organic Compounds				
2-Acetylaminofluorene	53-96-3	0.006		
4-Aminobiphenyl	92-67-1	0.007		
Benzidine	92-87-5	0		
bis(2-Chloroethyl)Ether	111-44-4	0.01		
bis(2-Ethylhexyl)Phthalate	117-81-7	0.004		
Bromodichloromethane	75-27-4	0.06		
Bromoform	75-25-2	0.080		

Chlorobenzilate	510-15-6	0.007		
Dibenzofuran	132-64-9	0.007		
3,3'-Dichlorobenzidine	91-94-1	0.004		
1,1-Dichloroethane	75-34-3	0.100		
Dimethyl Phthalate	131-11-3	0.006		
4-Dimethylaminoazobenzene	60-11-7	0.007		
Di-n-Butyl Phthalate	84-74-2	0.006		
2,4-Dinitrophenol	51-28-5	0.007		
2,4-Dinitrotoluene	121-14-2	0.007		
Ethyl Acrylate	140-88-5	0.16		
Hexachloro-1,3-Butadiene	87-68-3	0.060		
Hexachloroethane	67-72-1	0.003		
3-Methylcholanthrene	56-49-5	0.005		
Methyl Methanesulfonate	66-27-3	0.018		
4-Methylphenol	106-44-5	0.180		
Nitrobenzene	98-95-3	0.025		
4-Nitrophenol	100-02-7	0.006		
N-Nitrosodiethylamine	55-18-5	0.020		
N-Nitrosodimethylamine	62-75-9	0.043		
N-Nitrosodibutylamine	924-16-3	0.017		
N-Nitrosodipropylamine	621-64-7	0.020		
N-Nitrosopiperidine	100-75-4	0.019		
N-Nitrosopyrrolidine	930-55-2	0.017		
o-Toluidine	95-53-4	0.015		
Pentachloronitrobenzene	82-68-8	0.004		
Pentachlorophenol	87-86-5	0.007		
Phenacetin	62-44-2	0.011		
Propionaldehyde	123-38-6	0.001		
Safrole	94-59-7	0.011		
2,3,7,8-Tetrachlorodibenzo-p-Dioxin	1746-01-6	7.59E-09		
1,1,2,2-Tetrachloroethane	79-34-5	0.060		
1,1,2-Trichloroethane	79-00-5	0.060		
2,4,5-Trichlorophenol	95-95-4	0.004		
2,4,6-Trichlorophenol	88-06-2	0.006		
2,2,4-Trimethylpentane	540-84-1	0.108		

Acronyms:
EPA = Environmental Protection Agency
IRIS = Integrated Risk Information System as of 9/30/02
CAL-EPA = California Environmental Protection Agency
NAAQS = National Ambient Air Quality Standard
ATSDR = Agency for Toxic Substances and Disease Registry
ORD = Office of Research and Development
HEAST = Health Effects Assessment Summary Tables

APPENDIX B

Appendix B: Justification for Substitution of Half the Detection Limit for Nondetects

This appendix discusses the best approach to deal with data below the Method Detection Limit (MDL) or Non-Detects (NDs).

Five different methods of dealing with NDs were investigated by observing the behavior of relative bias of average concentrations of pollutants at different assumed ND percentages. The five methods were: (1) dropping NDs from the data set and (2-5) replacing NDs with MDL, MDL/2, %ND*MDL, or zero. CAP data obtained at Grant School, Kristof's Market, and Grattan monitoring sites from May 2001 through April 2002 were used. Only pollutants with half of their average concentration greater than the 70-year benchmark and with recorded NDs were included in the analysis — analytes 1,3-butadiene, chloroform, arsenic, p-dichlorobenzene, and tetrachloroethylene met these criteria.

The attached figures show percent differences between the actual average concentration for each compound and the simulated average concentration obtained from the data set incorporating the five methods. All values are in ppbv. According to the results in the figures, replacing NDs with MDL/2 values seem to reveal bias close to zero for almost all the selected compounds at the three sites. In addition, in all cases, as percent of data below MDL increases, the bias between actual and calculated average concentrations also increases. But regardless of this trend, the MDL/2 approach seems to indicate consistently less bias even at the highest assumed percent of values below MDL (in this case, 50 percent).

Though the MDL/2 approach appears best for dealing with NDs in a data set, replacing NDs with MDL seems also to perform well. The biases of other methods appear to increase or fluctuate steadily as the percent of data below MDL increases, with %ND*MDL as the most sensitive.

The compiled CAP data of these compounds through April 2002 show ND percentages between 12 and 70 percent. Reported NDs were fewest for arsenic and greatest for chloroform. According to this data, projection of bias with implementation of the MDL/2 approach would entail accepting bias in average concentrations of 20 percent at the least.

Analytical results are as follows.

FIGURE A2-1

PERCENT DATA BELOW METHOD DETECTION LIMIT FOR GRANT SCHOOL

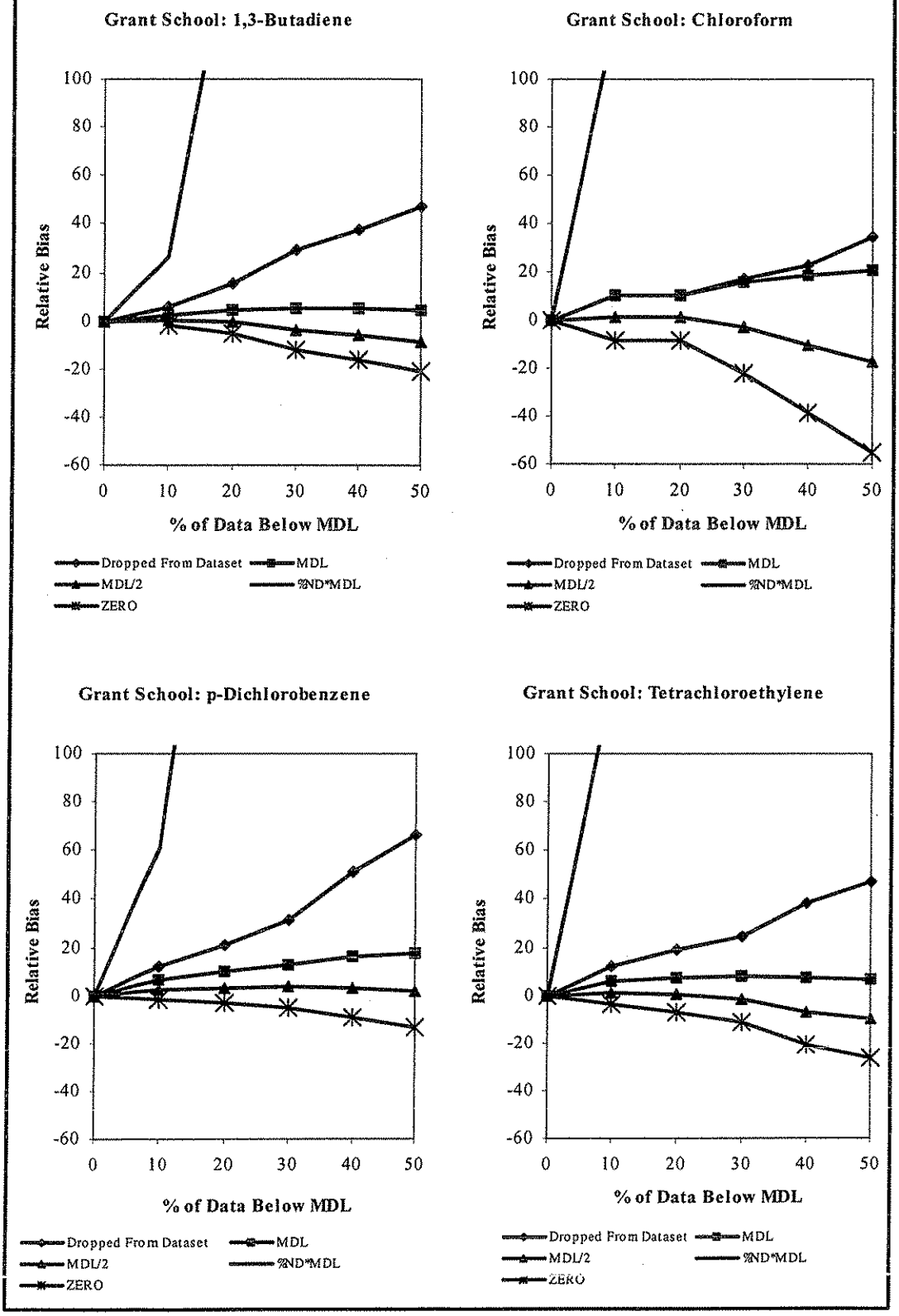


FIGURE A2-1

PERCENT DATA BELOW METHOD DETECTION LIMIT FOR GRANT SCHOOL

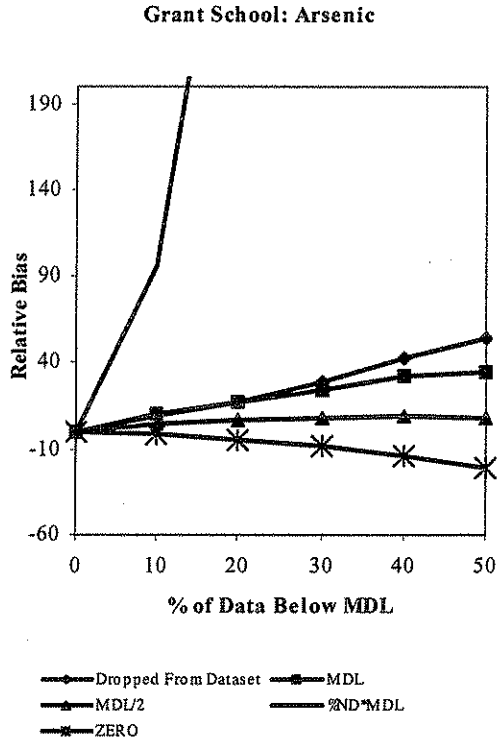


FIGURE A2-2

PERCENT DATA BELOW METHOD DETECTION LIMIT FOR GRATTAN ST.

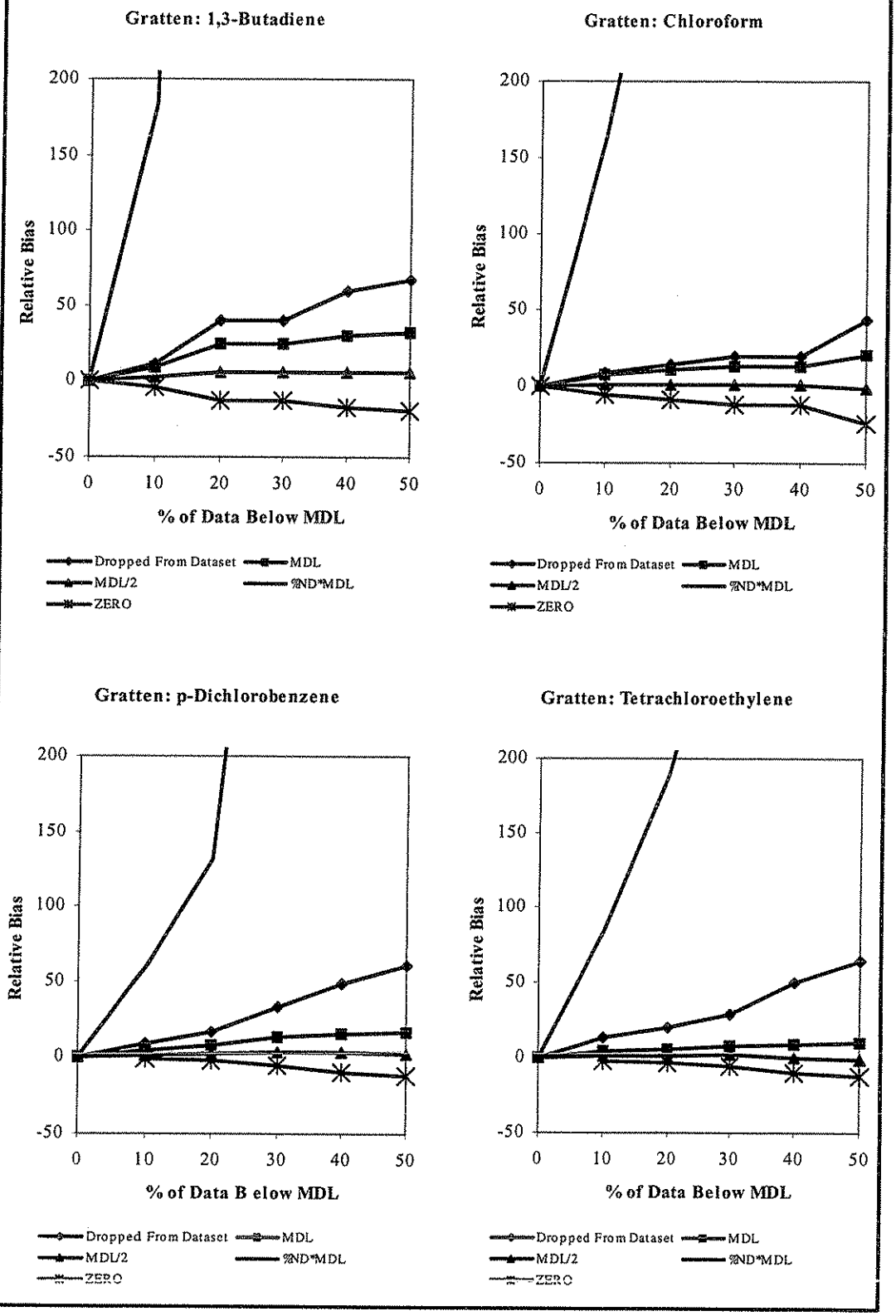
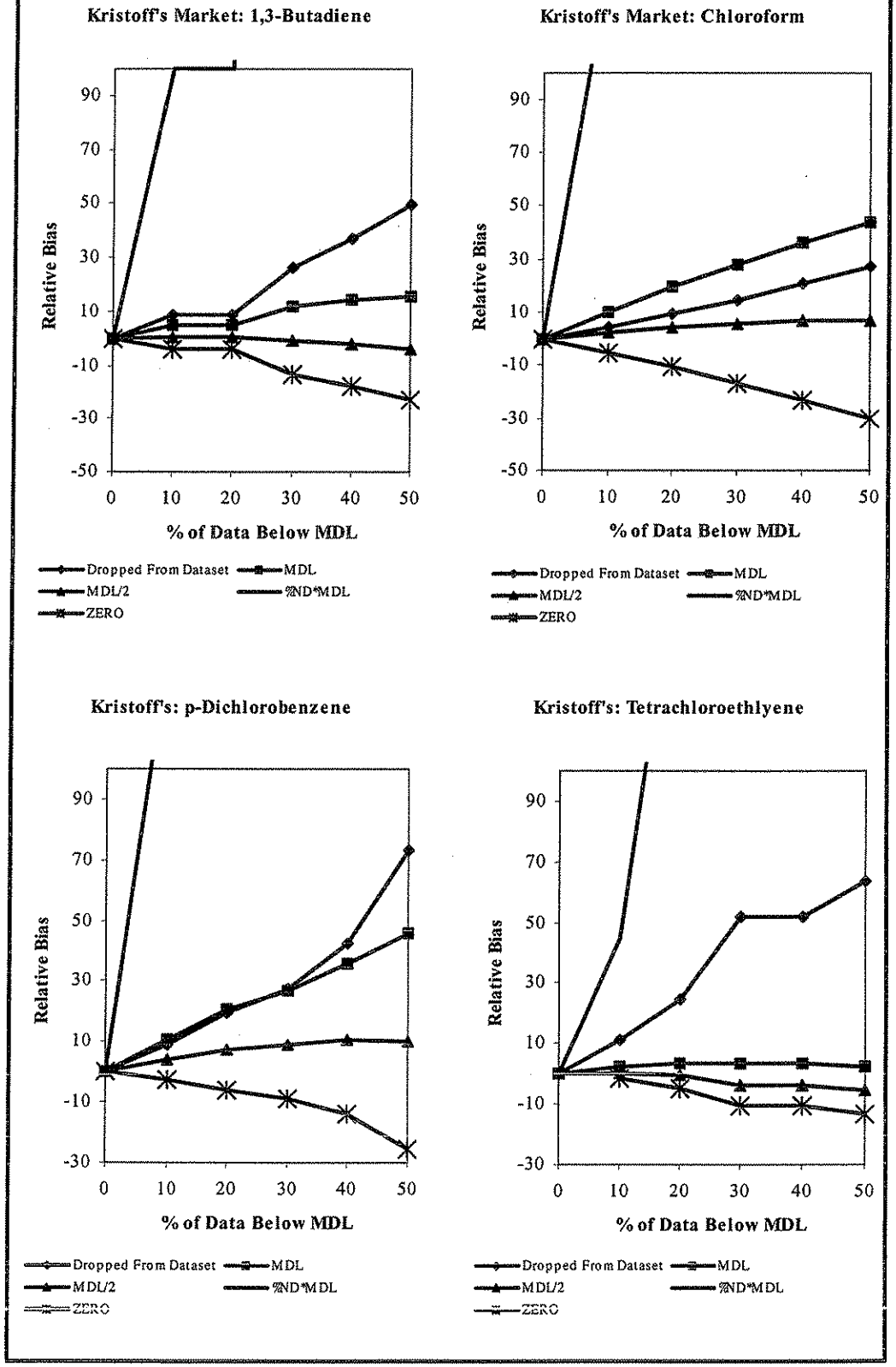


FIGURE A2-3

PERCENT DATA BELOW METHOD DETECTION LIMIT FOR KRISTOFF'S MARKET



APPENDIX C

Appendix C: Data Completeness and Quality Control Results

Table C3-1shows data completeness for each site and type of measurement.

Table C3-1. Data Completeness

Grant School VOC and SNMOC	81%
Grant School SVOC	89%
Grant School carbonyl	73%
Grattan	89%
Kristof's Market	92%

The study conducted duplicate sampling and replicate analyses on multiple sampling days for volatile organic compounds (VOCs), speciated non-methane organic compounds (SNMOCs), and carbonyls. Analytical results were reported as D1, R1, D2, and R2. For reporting purposes, R1 was assumed equivalent to the normal single sample and analysis, and the R1 value was used as the point of reference to calculate differences. Since samplers were equipped with capability to collect duplicate samples, one sampler was used to collect each set of duplicate samples.

For VOCs, for all analytes and all three sites, the average absolute deviations from R1 were 12 percent (D1), 15 percent (D2), and 13 percent (R2). In general, percent differences were smaller for higher reported concentrations and larger for smaller concentrations near the detection limit. For benzene (the only VOC with an average concentration greater than 70-Bench), average absolute percent deviations were 9 (D1), 8 (D2), and 6 (R2). If two anomalous results had been omitted from the calculation, percent deviations would have been only 5, 6, and 5, respectively. Figure C3-1 shows benzene results for duplicate and replicate samples.

For SNMOCs, for all analytes at the Grant School site, average percent absolute deviations were 13 (D1), 21 (D2), and 18 (R2).

For carbonyls, for all analytes at the Grant School site, average percent absolute deviations were 8 (D1), 18 (D2), and 17 (R2). The greater differences for D2 and R2, and examination of individual results, suggest in some cases a significant difference between the duplicate samples—perhaps in the amount of air that passed through the two sorbent cartridges. For formaldehyde, the average percent absolute deviations were 1 (D1), 21 (D2), and 21 (R2). For acetaldehyde, the average absolute deviations were 1 (D1), 13 (D2), and 14 (R2). These results are consistent with the above conclusion about differences between duplicate samples. Figures C3-2 and C3-3 show formaldehyde and acetaldehyde analytical results, respectively, for duplicate and replicate samples.

To evaluate precision of sampling and analysis results, duplicate samples with a completely different sampler were collected by temporarily relocating the Grattan VOC sampler to the Grant School site on December 15, 2001. Figure C3-4 shows ratios of Grattan sampler results to Grant School sampler results for each analyte quantified in both samples. The ratio for most analytes was near 1, indicating reasonable precision. However, a group of six analytes (methylene chloride, toluene, N-octane, ethylbenzene, *m*-, *p*-xylenes, and *o*-xylenes) showed ratios significantly less than 1. This appears consistent with results for these analytes at Grant School

that showed higher concentrations early in the sampling project and a decrease over time not consistent with results at Grattan and Kristof's Market. Possibly, a sampling artifact associated with the Grant School sampler was responsible for these results.

In addition to the field quality control described in this appendix, the analytical laboratory follows a quality assurance plan that specifies requirements for instrument calibration, quality control check sample analysis, canister cleaning, and so on. (Eastern Research Group, *Support for NMOC/NMOC, UATMP, and PAMS Networks, Quality Assurance Project Plan*, submitted to EPA, 2000).

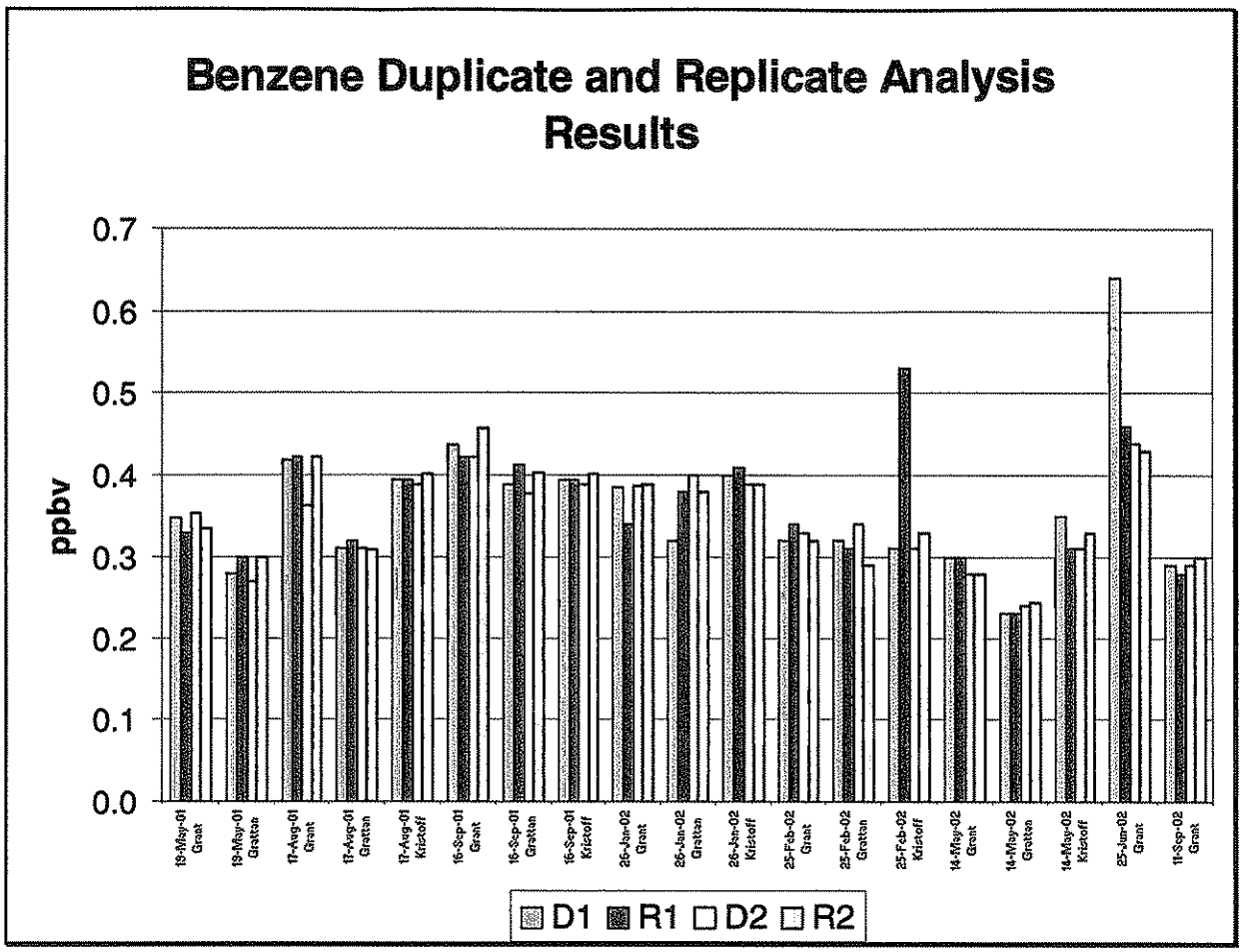


Figure C3-1. Benzene Duplicate and Replicate Analysis Results.

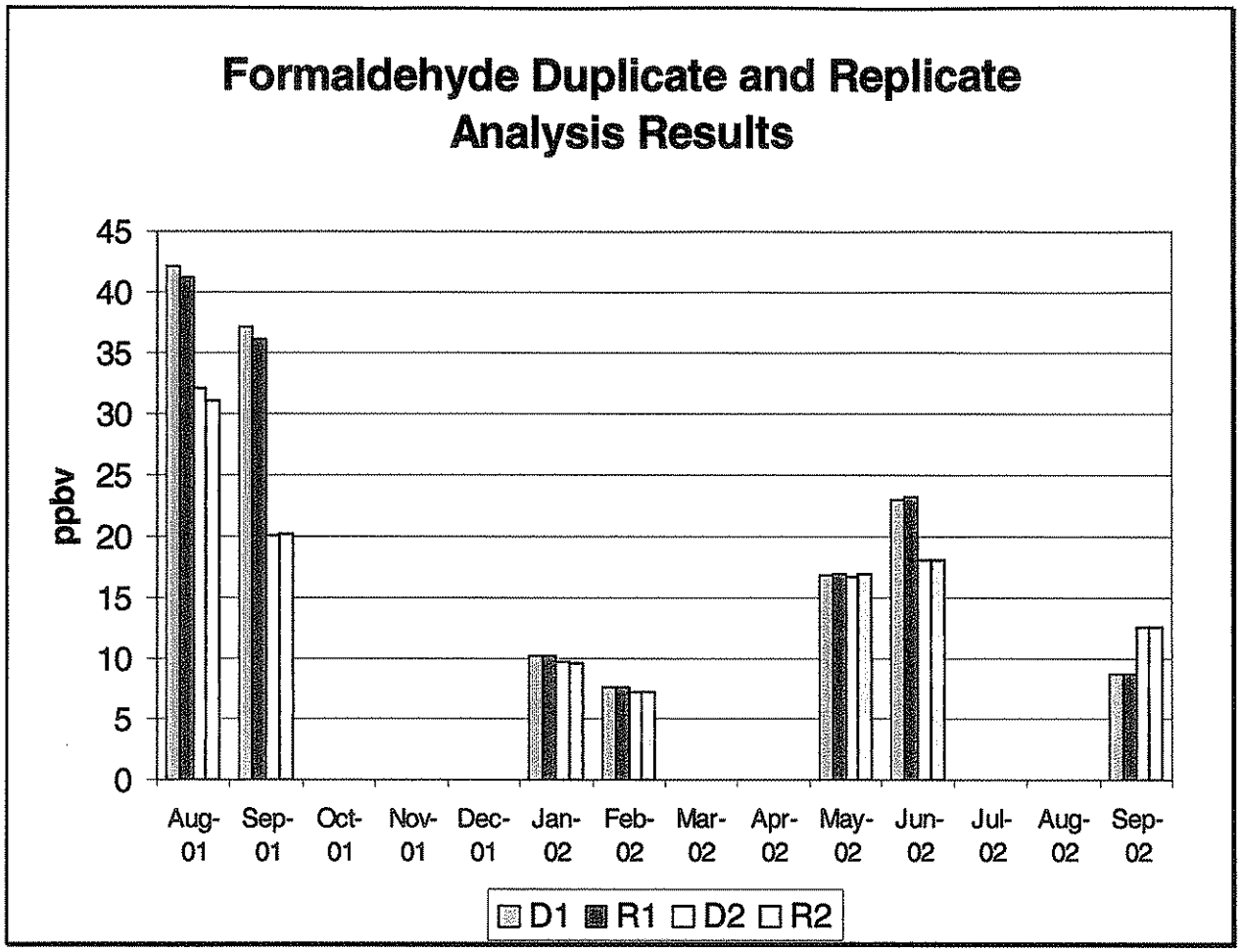


Figure C3-2. Formaldehyde Duplicate and Replicate Analysis Results.

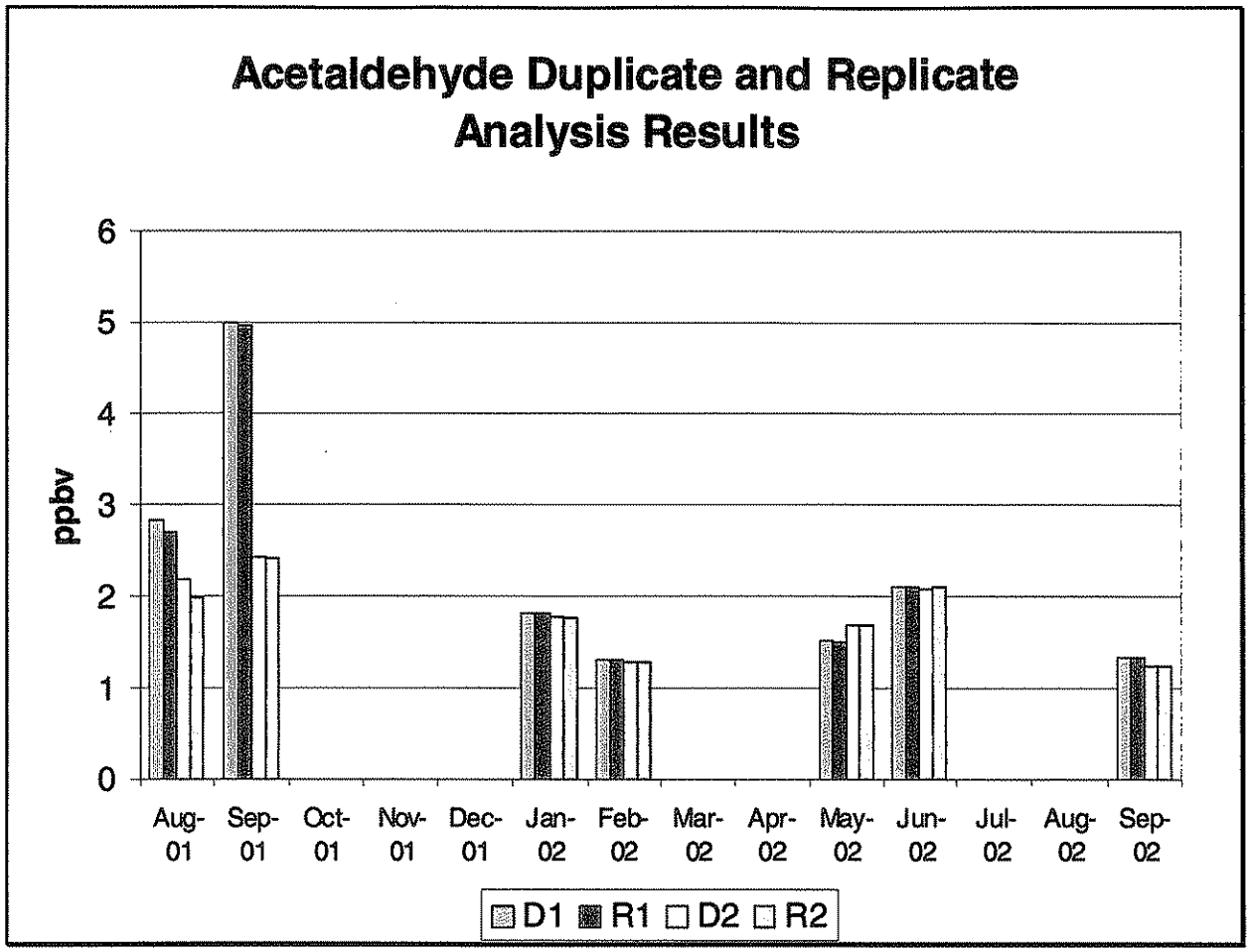
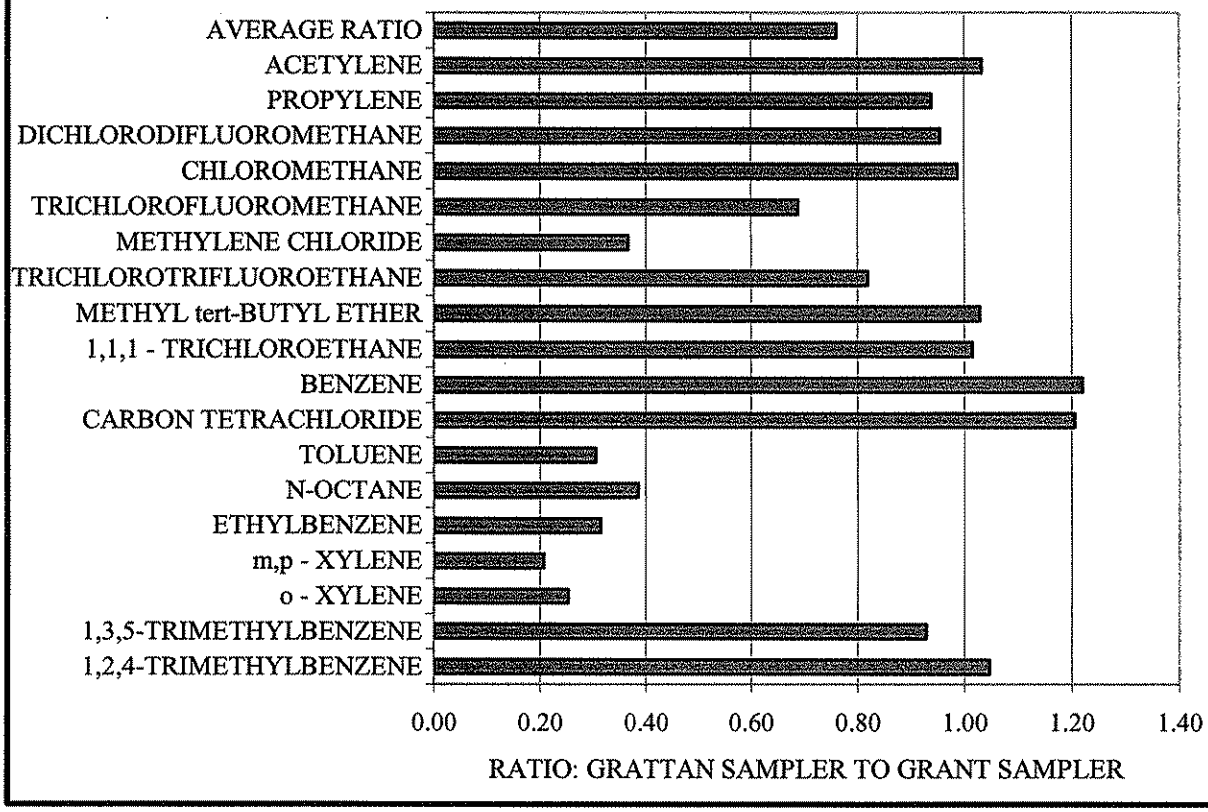


Figure C3-3. Acetaldehyde Duplicate and Replicate Analysis Results.

FIGURE C3-4

DECEMBER 15, 2001 PRECISION STUDY RESULTS



APPENDIX D

Appendix D: St. Louis Community Air Project Community Engagement and Education

The St. Louis Community Air Project (CAP) is a community-based environmental project established in response to St. Louisans' concerns about air pollution and their health. More than data collection and analysis, St. Louis CAP provides ways for community stakeholders to work with federal, state and local governments to address air pollution issues in non-traditional ways.

The CAP Partnership is a group of community stakeholders that began meeting in July 2000 to govern the project. Partners (see list below) include residents of the project area, neighborhood associations, service providers, universities, local businesses, environmental groups, and representatives of local, state and federal government. The St. Louis Association of Community Organizations (SLACO), a 25-year old coalition of neighborhood associations and churches working to improve the quality of life in St. Louis neighborhoods, is the managing CAP Partner. Bradley & Company facilitated monthly meetings. The CAP Partnership's diverse interests - coming together on a monthly basis to advise and direct the project – has made the St. Louis CAP unique among urban air toxics projects.

Because community stakeholders govern St. Louis CAP, outreach and education became an important part of the project. The main goal of the Partnership's outreach and education efforts is to help people better understand air pollution and how they can take an active role in protecting their health and the environment.

Since the Partnership's inception in July 2000, CAP Partners have engaged thousands of local residents through their education and outreach activities. In addition to informational presentations, classroom presentations, a website, regular mailings and e-mail, CAP has developed strong partnerships with several local institutions in order to better reach the public.

St. Louis CAP Resident Survey – CAP Partners worked with St. Louis University's School of Public Health to develop and distribute a survey to residents in the two project area zip codes. The survey was designed to help CAP Partners better understand residents' perceptions about air pollution and its sources. Eighteen hundred surveys were distributed in summer and fall of 2001 to residents in the two project area zip codes. Eleven percent of the surveys were returned. Results have since improved Partnership communication efforts.

Read About Our Air – Working closely with St. Louis CAP, the St. Louis Public Library developed air pollution kiosks at three branches and will soon expand to a fourth branch. Library patrons can check out youth- and adult-focused books, videos, CD-ROMs and state and federal publications about air pollution and environmental issues. This collection of library materials is intended to help people gain better access to environmental information.

The Importance of Clean Air is a 12-minute video conceived, created and produced by Roosevelt High School students and faculty. Starring the Clean Air Cowboy and the Clean Air Kid, the video teaches the importance of clean air and how they can impact it. Subsequent to producing the video, Roosevelt held its first all-school assembly in over five years to receive recognition from the EPA Regional Administrator for this accomplishment.

Clean Air Bookmark Contest – Building on an already strong partnership, St. Louis CAP and the St. Louis Public Library debuted the Clean Air Bookmark Contest in spring 2003. St. Louis Public Library staff give classroom presentations to encourage participation in the Contest. Presentations include showing Roosevelt's *The Importance of Clean Air* and inviting students to

create bookmarks to illustrate their best ideas for cleaner, healthier air. In 2004, over five hundred entries from more than seventy schools competed for the honor of published bookmarks.

In The Air: Tools for Learning About Airborne Toxics Across the Curriculum – Working with its Partners, the CAP Partnership became critically aware of the need to create educational units about air toxics. The Missouri Botanical Garden's EarthWays Center was brought on-board to develop *In the Air,* interdisciplinary, multi-media educational modules for five age blocks - Kindergarten through Adult. *In The Air* aims to increase knowledge about air pollution and to make connections between behaviors and air quality. Modules are available for free at www.InTheAir.org.

In the coming year, St. Louis CAP will continue its efforts towards healthier air with four action-oriented campaigns.

Idle-Free Schools - The American Lung Association of Missouri, the American Bottom Conservancy (in Illinois) and CAP will encourage idle reduction at schools. Turning off bus, truck and car engines will save money on fuel and keep kids from breathing harmful exhaust.

Detox Your Domicile* **Train the Trainer** - In spring 2005, CAP will host several training sessions on how to put on your own performance of *Detox Your Domicile*, the home improvement show that shows you how to put the GREEN into CLEAN. Participants will receive the scripts and prizes they need to stage the performance.
* *Detox Your Domicile* is the adult education module in *In The Air.*

Indoor Air Quality Toolkit for the Workplace - The Missouri Botanical Garden's EarthWays Center will use U.S. Green Building Council standards for indoor air quality to develop this toolkit. Presentations and toolkits for your workplace will be available in spring 2005.

Improve List of Air Pollution Sources for CAP Pollutants of Concern - With the help of the Missouri Department of Natural Resources and the City of St. Louis Air Pollution Control Division, this campaign will help us better understand where air pollution comes from. CAP will recruit volunteers to assist with collecting information on air pollution sources in their neighborhood.

To learn more, please contact Emily Andrews at (314) 533-9104 x 205 or emlandrews@hotmail.com or visit www.stlcap.org.

St. Louis CAP
COMMUNITY AIR PROJECT

Our Goal is Healthier Air!

St. Louis CAP Partners
(Past & Present)

Residents of the Project area
American Bottom Conservancy
American Lung Association of Missouri
Anheuser-Busch
The Boeing Company
Bradley & Company
Community Environmental Resource Program
East-West Gateway Council of Governments
501 Creative
Frauenhoffer & Associates
Grace Hill Settlement House
Laidlaw
Marine Villa Improvement Association
Metro
Missouri Botanical Garden – ECO-ACT Environmental Leadership Program
Missouri Botanical Garden's EarthWays Center
Missouri Coalition for the Environment
Missouri Department of Natural Resources
Mount Pleasant Neighborhood Association
North Side Neighborhood Clean Air Project
Regional Chamber and Growth Association
Rhodia,
RideFinders
Roosevelt High School
Alderman Craig Schmid
Sierra Club
Sigma Aldrich
Slay Transportation Company
Solutia
St. Alexius Hospital
St. Louis Association of Community Organizations (SLACO)
St. Louis City Air Pollution Control
St. Louis City Comptroller's Office
St. Louis City Neighborhood Stabilization Office
St. Louis Development Corporation
St. Louis Department of Health
St. Louis Housing Authority
St. Louis Metropolitan Sewer District
St. Louis Public Schools
St. Louis Public Library
St. Louis Regional Clean Air Project
St. Louis Regional Clean Cities Program
St. Louis University
Tetra Tech EM
Team Sweep Model Citizens Program
Tower Grove East Neighborhood Association
Tower Grove Health Watch
U.S. Environmental Protection Agency
U.S. Green Building Council – St. Louis Regional Chapter
Washington University
Wyman Community Connections. . . and growing

APPENDIX E

Appendix E: Comments Received and the Responses to Those Comments

The authors thank Emily Andrews, Don Simpson, Scott Clardy, Motria Caudill, Phuong Nguyen, George Bollweg, Ted Palma, Roy Smith, and David Guinnup for reviewing the draft version of this report and providing the following comments.

Comments from the St. Louis Association of Community Organizations

General Comments

Was consideration given to including a glossary?

Response: Yes, the authors considered including a glossary. The authors believe a glossary is not necessary however because of the small number of acronyms appearing in the text.

I am planning to work with the Partnership at our next meeting (October 26) to discuss the content of a CAP Partnership Final Report that would include activities outside of the technical sphere of the project, such as information on how we convened the Partnership, who the members are, how the monthly meetings worked, outreach & education efforts, our very successful partnerships with the Library & with Roosevelt High School, etc. I'd love to include something in the appendix about the plans for this or where one could go to get this information. I would imagine that we could have something in hand by the end of the year.

Response: The authors accepted this comment and added Appendix D, which contains this information, to the report.

Although I certainly understand that this study set out to measure levels of air toxics and compare them to long-term cancer and non-cancer benchmarks, I would like to see more discussion of short-term health effects. There are many chemicals for which we monitored that had no cancer benchmarks or no long-term benchmarks at all. Certainly all of these chemicals can have short-term health effects. The fact that we have some of the highest asthma rates in the country in the City of St. Louis makes this an important issue. And from CAP's experience with community surveys, presentations and educational outreach, this (asthma and short-term health effects from air pollution) is an issue that the community truly cares about and wants to know more about.

Response: The St. Louis Community Air Project was a monitoring effort that examined the chronic health effects of exposure to air pollution. The Technical Team of the Partnership made no effort to examine the acute health effects of exposure to air pollution. This comment is therefore outside the scope of the monitoring effort.

If possible, I'd love to include something about CAP next steps in the appendix (in terms of the new Community Based Air Toxics Grant for which SLACO has been recommended.) I think it would be helpful to show that we are planning on following up. I would be happy to provide a short write-up!

Response: The authors accepted this comment and added Appendix D, which contains this information, to the report.

Specific Comments

Figure 4-2. Can we explain the November 2001 spike in Benzene? Which also occur with Acetaldehyde and Formaldehyde. We regard the HCHO data as no good, but why not the other if the spike occurs around the same time? Especially because Acetaldehyde was measured by the same equipment as Formaldehyde, right?

Response: The authors can not explain the November 2001 spike in benzene, acetaldehyde, and formaldehyde ambient concentrations. The authors regard the ambient formaldehyde data as not representative, rather than as no good, because the ambient concentrations measured in Phase I were dramatically different from those measured in Phase II. The ambient acetaldehyde concentrations however did not show this dramatic change in concentration between the two phases. The average ambient acetaldehyde concentration measured at Grant School was 2.67 ppbv during Phase I and 2.55 ppbv during Phase II. The ambient acetaldehyde concentration data are therefore more representative of the true air quality than are the ambient formaldehyde concentration data.

Section 4.3, page 15. In the first paragraph, HCHO is described at a VOC precursor to ozone formation. Isn't Acetaldehyde also a precursor? This isn't mentioned in section 4.1. Also in the 3rd paragraph, we see non-cancer both with a hyphen and without in the same sentence.

Response: Yes, acetaldehyde is also a VOC precursor. The authors accepted these comments and corrected the text.

Section 4.3, page 17, 2nd paragraph. Saying "CAP Project" (last line) is redundant — should just be CAP. As it was presented in Partnership meetings, one of the reasons for expanded and continued monitoring was to learn more about HCHO and its formation AND to determine whether large oak forests in southeastern MO might be contributing to high HCHO levels in the St. Louis metro area. This isn't discussed here.

Response: The authors accepted these comments, corrected the text, and offered more discussion on this topic.

Section 4.3, page 17, 3rd paragraph. You mention four sites, but only list two — Grant School & Wash U are missing. When did monitors stop? This isn't mentioned here. Also, we haven't seen the word carbonyl yet in the report — previously aldehyde was used. If the two are interchangeable, I would choose only one and consistently use it.

Response: The authors accepted these comments, corrected the text, and offered more discussion on this topic.

Section 4.3, page 18, 2nd full paragraph. I think we need to say more here about WHY data from Phase 1 is insufficient in concluding an area or point source signature. Also, in the last line, should it be biogenic instead of bioorganic?

Response: The authors accepted these comments and revised the text.

Section 4-5 page 21, 2nd full paragraph. Please be careful when saying that the CAP Partnership did something like, "adjusted the IRIS RfC and URE for particulate hexavalent chromium. This was something, I believe, that the technical team decided and I don't recall it ever being discussed at a Partnership meeting. Could you revise this so that it accurately reflects that?

Response: The authors accepted these comments and revised the text.

Section 4.6, page 22. Could we include an explanation as to why diesel pm is considered a CAP Pollutant of Concern? Meaning, even though we didn't have a health benchmark for it, CAP Partners considered it to be an important pollutant to address because of the community's concern and because of other health risk information.

Response: The authors accepted this comment and included an explanation in the text.

Section 4.6, page 22, 5th paragraph. See comment above on chromium — I don't think this was something the Partnership determined or could have determined (the annual ambient diesel pm concentration). It was a technical team task then reported to the Partnership.

Response: The authors accepted this comments and revised the text.

Section 6.1, page 26. Did the CAP emission inventory of point source include sources in Illinois? Seems that we talked about working with EPA R5 and IEPA to get that info.

Response: Due to the extensive editing of this section, this comment is no longer relevant to the text.

Section 6.2, 2nd paragraph. I would replace laundromats with dry cleaners. I think they have very different connotations (i.e. a laundromat is somewhere you take your regular old laundry to wash and dry in a regular washer and dryer and a dry cleaner uses a chemical process to clean your clothes).

Response: Due to the extensive editing of this section, this comment is no longer relevant to the text.

Section 6.2, page 27, first full paragraph. Did volunteers really collect info on residential sources? We did talk about this and I know Mollie drafted a survey, but I don't think we ever really followed up — except with a few Partners.

Response: Due to the extensive editing of this section, this comment is no longer relevant to the text.

Section 6.3, page 27. First sentence indicates that this hasn't happened yet, however, reading further, it seems like it has. Also, was data only collected on mobile sources in the City of St. Louis? Nothing in St. Louis County or Illinois?

Response: Due to the extensive editing of this section, this comment is no longer relevant to the text.

Section 6.3 page 28, very last sentence — again with the residential survey. How many really went out? And how many were returned?

Response: Due to the extensive editing of this section, this comment is no longer relevant to the text.

Section 7.1 page 31, please include Partnership when talking about CAP here. This was very much a Partnership decision.

Response: The authors accepted this comment and corrected the text.

Section 8.0, page 33, second paragraph. Some numbers to go along with the indoor/outdoor air quality discussion would be helpful here. For example, I think I've heard that we spend something like 80 — 90% of our time indoors. And I've heard figures about how much more polluted the indoor air is. Or that perhaps the concentration of pollution is greater.

Response: The authors accepted this comment and added numerical data to text.

Section 8.0, page 33, third paragraph. The 25% uncertainty level mentioned here seems to almost qualify our data as useless from the perspective of a non-scientist. Is there a way to explain this better so that people don't think that we're afraid to say something about the data? Or that we've spent a lot of money on nothing?

Response: The authors accepted this comment and added a better explanation to the text.

Section 9.0, page 35. Any reason why Wash U data hasn't been included in the report? Or is there a timeline for when we'll be able to see this data?

Response: The authors accepted this comment and added a reason for not including the Washington University data in the report, as well as an estimated date for the release of these data, to the text.

Appendix A. Table Al -1. Any reasons for not including 5 of the urban air toxics in the CAP study? In particular Acrolein has come up as a pollutant of concern in other cities that have collected air toxics data.

Response: The authors accepted this comment and added reasons for not including these urban air toxics to the text.

Comments from the St. Louis City Air Pollution Control Division

At the CAP Draft Report meeting, I pulled Eric's graph which showed the "Comparisons of the ambient formaldehyde concentrations" out of the report and wrote "Check out" pointing to the 25 ug/m3 value at Grant School for early April 2003. I didn't want to say anything at the meeting, but I remembered that we only had one or two values above 10 ug/m3 (and those were like 10.5 or something) with the new ERG system. Knowing that Eric needed feedback comments before August 20 or so, I looked into this on Friday 7/23. My Field Data Sheet showed that I had set the sampler up incorrectly and it ran that way from midnight till 8 AM and that I was going to VOID the sample if the concentrations were atypical in any way. My ERG Lab April 2003 Carbonyl Data Report showed that I VOIDED 4/9/03 and sent that information to Bern. But I don't keep a copy of the Level 0/1 Data Validation Report I send to Bern. I keep the ERG Lab report on which I cross out and mark as VOID any bad field runs.

I checked with Bern first, just to make sure his Level 0/1 report from me showed the VOID and his Level 2/3 (?) which comes at a later evaluation showed the VOID also - Just so our records are all correct for any later investigation by anyone.

****The whole point being that the high formaldehyde data point for Grant School 4/9/03 needs to be removed from the CAP Draft Report****

Response: The authors accepted this comment and removed the data point from Figure 4-6.

Comments from the Missouri Department of Health and Senior Services

General Comments

The monitoring conducted by the CAP was consistent with current United States Environmental Protection Agency (EPA) guidance regarding quality assurance for air pollution measurement. The risk characterization appears consistent with the methodology set forth in the EPA National-Scale Air Toxics Assessment for 1996. The cancer benchmarks and risk characterization set forth in this document appear protective of public health.

Tables

Tables 3-1 (page 8), 3-2 (page 9), 3-3 (page 10), and 3-4 (page 11) utilize the incorrect acronym "HEIST" in the table key and incorrectly define the acronym "EPA IRIS". Please change "HEIST" to "HEAST" and please define "EPA IRIS" as the United States Environmental Protection Agency, Integrated Risk Information System. DHSS also recommends that the following acronyms used in the tables be defined to enhance the clarity and transparency of the tables: "EPA-ORD", "EPA-NAAQS", and "ATSDR".

Response: The authors accepted these comments and corrected the tables.

Table A1-2 spans pages A-6 through A-8. The headings of all three pages indicate that this table is "continued." Is there a page missing? Furthermore, Acetaldehyde, Arsenic, Benzene, and Chromium are missing from this table. DHSS recommends that any missing pages be included or that the heading be relabeled appropriately.

Response: The authors accepted these comments and included missing pages and analytes.

Additionally, DHSS recommends that IRIS citations include the month and year the relevant data is acquired from IRIS.

Response: The authors accepted this comment and included the month and year in the IRIS citations.

Comments from the U.S. Environmental Protection Agency's Region 5 Office

Motria Caudill's Comments

A few comments in order of sections:

1.0 Introduction. This section makes no mention of important NAAQS issues, i.e. ongoing problems and new standards for ozone and PM2.5. Understand that the CAP study addresses air toxics, but criteria pollutants should be noted as important health concerns.

Response: The St. Louis Community Air Project was an air toxics monitoring effort. Mentioning important NAAQS issues is outside the scope of this effort.

2.1. It would be interesting to mention a few of the other HAPs that were dropped from the list for lack of monitoring methods, e.g. acrolein and other important risk-drivers.

Response: The authors accepted this comment and added text stating acrolein was dropped from the analyte list due to the absence of a reliable analytical method to Section 2.1.

Figure 2.1. This map is not very informative and needs some other geographic or environmental features.

Response: The authors accepted this comment and replaced this map with an annotated aerial photograph.

4.0. There are numerous statements about data that "appear" to be higher in one season versus another, or data that "seem" to be higher at one site versus another. Such observations should be confirmed or refuted with a statistical test, such as ANOVA (analysis of variance) to show whether seasons are statistically different or a paired t-test.

Response: The St. Louis CAP Partnership decided early in this project that statistical analyses of the data were not an objective of this monitoring effort.

Phuong Nguyen's Comments

1) Section 2.2. The last sentence mentions about the meteorological data. I think more discussion is needed about what instrument was used to measure the data (Tower or SODAR), how the data was collected? How many months of met data were collected? How it was used? Etc… Such information is important since meteorology greatly influenced the formation of acetaldehyde, the peak of formaldehyde, and acetaldehyde.

Response: The authors accepted these comments but did not address them. Rather than addressing these comments, the authors removed all meteorological references from the text because the meteorological data were never presented in the report.

2) Section 4.4. Arsenic Compounds. Page 19 (paragraph 1) discussed the fact that Arsenic compounds can be in fine particular matter and transported long distance before settling in the St. Louis area. Page 20 said that the observation showed that local sources influence the ambient Arsenic concentrations in Arnold and St. Louis City. It was not clear to me (after reading this section) whether Arsenic compounds is a regional or local issue?

Response: Based upon the authors' data interpretation, the report suggests that arsenic compounds are a local issue. To clarify the report, the authors removed all references to transport from the text.

3) Section 4.8. The first sentence should be written as Meteorological conditions such as low temperature (low or high?), relative humidity, and (low or high?) barometric pressure affect the concentration of pollutants in the air. If unsure about the relative humidity, and barometric pressure, then perhaps should not be specific about temperature.

Response: The authors accepted these comments but did not address them. Rather than addressing these comments, the authors removed all meteorological references from the text because the meteorological data were never presented in the report.

4) Page 19. The fact that EPA's Science Advisory Board is re-evaluating the cancer risk posed by formaldehyde, is there any plan that the CAP partnership will further review the formaldehyde data as a result of new EPA finding?

Response: Yes, the CAP Partnership may further review the formaldehyde data due to the new U.S. EPA finding. The text now expresses this idea.

George Bollweg's General Comments

The report seems to contain a great deal of valuable technical information, but its presentation can be improved. A better description of the overall organization of the project and report (e.g. clear statement of project origin and process, goals, objectives, methods, results, interpretation) could help.

Response: Section 1 of the report already presents a satisfactory organization of the project and report.

The project's stakeholder involvement component gets much less attention than technical air monitoring/modeling results and methods. If the CAP partnership aspires to be "...a model application of this [Urban Air Toxics] Strategy that other urban air projects can emulate..." more description of the stakeholder interaction process would help.

Response: The authors accepted this comment and added Appendix D, which contains this information, to the report.

The report seems heavily oriented toward single pollutant outdoor air monitoring technical issues (e.g. air pollutant concentration data analysis and comparisons). The use of technical appendices

helps make the report more useful to the general reader, although even more information (e.g. section 2.3, Instrumentation and Analytical Methods) might belong in an appendix. Since the community's central environmental concern appears to have been air pollution and its health effects (per p. 2), the reason for emphasizing air monitoring rather than other relevant technical issues (e.g. formaldehyde toxicity) might be more clearly described.

Response: The authors did not accept this comment. Acceptance of this comment will require the authors to partake in a major rewriting of the report.

Since the report becomes focused on five pollutants, the selection of those pollutants should be made clearer, e.g. a simple flow chart or diagram showing the process at the end of section 3 or the beginning of section 4.

Response: The authors accepted this comment and added a flow chart to section 3.

Conclusions and recommendations should be more easily apparent. Readers interested only in the "bottom line" might read section 9 only (p. 34, Summary of Conclusions and Recommendations), which could be frustrating with the current draft. Although the first sentence concludes that "The first year of Community Air Project monitoring was successful", it seems more promotional than informational. There's mention in paragraph 2 of what *can't be* concluded, but what *can be* concluded should also be clearly stated. The "70-Bench concentration" jargon might be better replaced with something more understandable (e.g. "risk based comparison concentration") for readers who don't wish to search for the term's definition. One seemingly major conclusion seems to garble the distinction between cancer risk and air pollutant concentrations ("...This evaluation indicates that the five pollutants are widespread and common to all urban centers in the United States, and may be used to establish a base level for determining excess cancer risk from inhalation exposure to air toxics for the St. Louis metropolitan area..." paragraph 3, last sentence, p. 34). And although stand-alone recommendations are listed (p. 35), they could be stated more clearly. For example, recommendation 1 appears to suggest continuing analysis like that in the present report (plus source apportionment methods) without giving a rationale for continuing that analysis or identifying the issue(s) to be addressed with source apportionment methods.

Response: The authors accepted these comments and revised the text.

George Bollweg's Specific Comments

p. 2, second paragraph: USEPA's Urban Air Toxics Strategy (July 1999) is mentioned. Since the 1997 St. Louis initiative appears to have preceded the Urban Strategy, readers may wonder about the original motivation for the project. One way to improve this might be to reverse the order of the first and second paragraph.

Response: The authors accepted this comment and reversed the order of the first and second paragraphs.

p. 2, third paragraph (third to last sentence): this mentions "strategies to improve air quality". What are these strategies? Are they described somewhere?

Response: The authors accepted this comment and identified and described strategies to improve air quality in section 2.

p. 2, fourth paragraph describes "...techniques used to monitor ambient air and protocols that identified five priority air pollutants of concern; acetaldehyde, arsenic compounds, benzene, chromium compounds, and formaldehyde..." The "protocol" used to identify the five chosen priority pollutants should be presented more clearly.

Response: The authors did not accept this comment. The purpose of this paragraph is to list the subject matter in the subsequent report sections. Section 3 more appropriately presents the "protocol".

p. 3, section 2.1 (Analyte determination) includes information on "cancer benchmarks" that seems to belong in section 2.4 ("Cancer and noncancer benchmark concentrations").

Response: The authors did not accept this comment. The discussion in section 2.1 describes the use of human health benchmarks to identify the candidate analytes that will remain on the analyte list, and the number of these analytes that had cancer benchmarks associated with them. This section does not specifically discuss cancer benchmarks.

p. 3-4, section 2.2 (Monitoring network): Fig. 2.1 gives the impression that the monitors are "piled up" on one another and within only one of the boundaries (zip codes? counties?) in the figure. Can the areas within the boundaries be named? Is there a reason to show such a large area if only one was monitored? If not, maybe a smaller (higher-resolution) map with more local detail (e.g. roads) could be substituted for the present map. The superscripts on the three named sites in the Figure should include a legend like Figure 4-5, p. 16.

Response: The authors accepted these comments and replaced this map with an annotated aerial photograph.

p. 5, section 2.4, Cancer and noncancer benchmark concentrations: note that the term "benchmark concentration" has a specific USEPA IRIS definition (a concentration producing a predetermined change in response rate of an adverse effect [the benchmark response] compared to background) that's different from the definition presented in the third paragraph ("...the ambient air concentration of a single pollutant low enough not to threaten public health if chronically inhaled...").

Response: The authors accepted this comment and added text to section 2.4 to distinguish the St. Louis CAP Partnership's definition of benchmark from the U.S. EPA's definition of benchmark.

Terminology: the "70-Bench" descriptor should probably be defined in section 2.4 (it doesn't appear until p. 7, Discussion of Measurement Results) even though it is used broadly and is not conventional.

Response: The authors accepted this comment and defined 70-Bench in section 2.4.

p. 7, Section 3.0 Discussion of Measurement Results

The present version of the document is unclear as to how the "Pollutants of Concern" (which appear in section 4.0) were identified (section 3.0?) and I'm still not sure I understand. Amplifying the confusion is the last paragraph of section 3.0 (p. 7) that begins with the statement "Table 3-4 lists analytes with averages greater than half of their 70-Bench values" while Table 3-4 (p. 11) is titled "TABLE 3-4 .ANALYTES LESS THAN HALF OF BENCHMARK CONCENTRATIONS". Suggestion: to simplify the reader's job, how about a simple flow diagram showing the process and logic behind 188 initial analytes> 113 analytes> fewer analytes>...> five "Pollutants of Concern"?

Response: The authors accepted this comment and added a flow chart to section 3.

p. 26, Section 6.0 Toxic Emission Inventory

"The CAP partnership has developed an emissions inventory report to identify sources of toxic chemicals that pose the greatest potential health risks." (first sentence, p. 26). To avoid the potential misinterpretation that the author(s) intend to assess health risks with an emissions inventory, I suggest inserting the word "help" before the word "identify".

Response: The authors accepted this comment and inserted the word "help" as suggested.

Table 6-4 (p. 27) contains identical entries for 1996, 1999 and 2001 for acetaldehyde, formaldehyde and butadiene. Identical entries area also given for benzene and MTBE. Are these correct? It seems hard to believe that these values for these chemicals would remain constant between 1996 and 1999, and that the 2001 projection is also one of no change.

Response: The authors accepted this comment and corrected these entries.

On p. 28, the first complete sentence seems only partially true ("...To assess the level of diesel emissions in the St. Louis CAP area, the activity level of all diesel sources should be determined for each examined facility...."). Wouldn't a great deal of diesel emissions come from transient mobile sources not associated with particular local facilities?

Response: Due to the extensive editing of this section, this comment is no longer relevant to the text.

p. 29, Section 7.0 Risk Characterization: although the term "risk characterization" is part of the title of the document, USEPA's Handbook for Risk Characterization is not cited or mentioned.

Response: The authors did not accept this comment because this document was not a source of reference material.

p. 31, section 7.2 .the following statement should probably be reworded: "...Though a target level of cancer risk of 1-in-1,000,000 would protect human health to a greater extent, analytical techniques cannot identify and quantify as many analytes at this target level as at a target level of cancer risk of 1-in-100,000." *Suggested alternative wording to reflect apparent intent:* "...Although a target level of cancer risk of 1-in-1,000,000 reflects a more stringent criterion, analytical techniques cannot identify and quantify as many analytes at pollutant concentrations hypothesized to be associated with this target level as with a risk target level of 1-in-100,000."

Response: The authors accepted this comment and corrected the text as suggested.

p. 33, Section 8.0 Limitations

The first paragraph states that the lack of a complete evaluation of all airborne pollutants (e.g. ozone, PM, indoor air pollutants) "...may lead to an overestimation or underestimation of risk for exposed populations." It's difficult to understand how the lack of a complete evaluation could lead to risk overestimates. In addition, this section should probably contain some discussion of current controversies related to formaldehyde toxicity, or a reference to the report section that describes the issue.

Response: The authors accepted this comment accepted and deleted the words "...overestimation or..." from this paragraph.

Response: The authors did not accept this comment. This report will remain silent with respect to the controversies related to formaldehyde toxicity because U.S. EPA has not yet finished its investigation of formaldehyde toxicity.

p. 34, Section 9.0 Summary of Conclusions and Recommendations

The risk characterization-related conclusions seem absent; the text again focuses on outdoor air pollutant monitoring issues. The third paragraph on p. 34 finishes with the following sentences:

"...Ambient results for these [five] pollutants of concern are also very similar in concentration to monitoring values reported by EPA's Urban Air Toxics Monitoring Program (refer to Appendix D for a summary of results from this study). This evaluation indicates that the five pollutants are widespread and common to all urban centers in the United States, and may be used to establish a base level for determining excess cancer risk from inhalation exposure to air toxics for the St. Louis metropolitan area...."

I agree with the first part of the last sentence, but the second part seems garbled. Why/how/where should air pollutant concentration results be used to "establish a base level [of what?] for determining excess cancer risk from air toxics inhalation in St. Louis?"

Response: The authors did not accept this comment. The second and third paragraphs address risk characterization. To clarify section 9.0, the third paragraph now refers to section 7.0.

Response: The authors accepted this comment. The text now reads, "The average concentrations of these five pollutants in urban areas can be used to estimate of excess cancer risk from inhalation exposure to air toxics for the St. Louis metropolitan area."

p. A-4 appears to repeat p. A-2 (Table Al-I).

Response: The authors accepted this comment and corrected Table A-4.

Comments from the U.S. Environmental Protection Agency's Office of Air Quality Planning and Standards

Comments from Ted Palma, Roy Smith, and Dave Guinnup

Section 2.1. The basis for the PRGs and Missouri RALs should be explained, in particular, what risk levels they correspond to and whether they extrapolate inhalation benchmarks from oral dose-response assessments. It is also unclear why the CAP did not simply use the CEP's own risk estimates for planning purposes.

Response: The authors accepted these comments and revised the text in the first paragraph of section 2.1

Section 2.2. The report should note here that the network was later expanded to include formaldehyde.

Response: The authors accepted this comment and revised the text.

Fig 2-1. The scale of the map is too large, making the monitors appear to be clustered together. This figure should be either remapped at higher resolution, or have a higher-resolution inset added. The meaning of the superscript numbers should be explained.

Response: The authors accepted these comment and replaced this map with an annotated aerial photograph.

Section 2.4. Paragraph 3 should be modified to say benchmarks are set at a level **"believed low** enough not to significantly threaten" (bold indicates suggested additional words). These concepts should be defined in this paragraph (not at the end of the section) as corresponding to an individual cancer risk of 10 in a million or to EPA's RfC or equivalent.

Response: The authors accepted these comments and revised the text.

Paragraph 3 should also convey that both cancer and noncancer benchmarks were calculated for compounds having both a URE and RfC. A summary statement comparing cancer and noncancer benchmarks for the same substance (i.e., in all but 1 in case the cancer benchmark was lower) may also be worth adding.

Response: The authors accepted these comments and revised the text.

Section 2.4 also needs a fuller explanation of the potential risk management implications of these benchmarks, e.g., providing a summary explanation of the "action plan" referred to in paragraph 7 (e.g., what it is and who will develop it). This explanation should include a description of the process used to select the risk levels at which the benchmarks were set.

Response: The authors accepted this comment and revised the last paragraph of this section.

The section should discuss any extrapolation of HEAST (or other) oral dose-response values to inhalation exposure, e.g., specifically how the extrapolation was done and why extrapolation was preferred over other inhalation benchmarks that may have been available.

Response: The authors did not accept this comment because the authors did perform an extrapolation of HEAST, or other, oral dose-response values to inhalation exposure.

The decision to express all benchmarks in ppmv rather than their native mass/volume units has prevented us from double-checking them. We don't see any obvious errors, but we suggest that you confirm all these conversions.

Response: The authors accepted this comment and confirmed all conversions.

Section 3.0. We suggest revising the data analysis process to the following approach, which we think is easier to explain to lay readers and also more convincing:

1. Compare the maximum observed concentration with the benchmark value. Remove from the analysis each HAP whose maximum does not reach its benchmark(s), and tabulate the comparison.

2. Calculate the average concentration of each remaining HAP, with non-detects set at the MDL. Calculate the 95 percent upper confidence limit of the mean. Remove from the analysis each HAP whose 95 percent UCL does not reach its benchmark(s), and tabulate the comparison.

3. Examine in detail the remaining HAPs, addressing issues such as detection frequency, MDLs above the benchmark, degree of benchmark exceedance. As part of this, show the effect of assuming the MDL, half the MDL, or zero for non-detects.

The general principle behind this suggestion is to remove as many non-drivers as possible using a simple, clear, and conservative rationale before tackling the more difficult issues in step (3). Using the UCL rather than the mean in the analysis is an important part of this.

Response: The authors did not accept this comment. The Technical Team of the St. Louis CAP Partnership determined that the data analysis process presented in the report is easier to explain to lay readers and easier for lay readers to understand. For this reason, the authors will not analyze the data using the suggested process.

The phrases "no risk" and "zero risk" should be avoided. Instead, we suggest "insignificant," "negligible," "below CAP benchmarks," or something similar.

Response: The authors accepted this comment and revised the text.

It is not clear why "half the benchmark" was used as the cutoff between Tables 3-3 and 3-4. Tabulating values at or above the specified action plan levels would seem more appropriate.

Table 3-4 should say "Analytes greater than..." The Table 3-4 footnotes on highlighting do not seem to describe what is in the table. It's unclear what boldface means.

Response: The authors accepted these comments and revised the text. True, half the benchmark is a somewhat arbitrary cutoff. Yet it allows the inclusion of any analyte having an ambient concentration approaching a cancer or noncancer benchmark to be scrutinized.

The MDL concept should be defined in this section rather than later (it's currently described redundantly in most of the sections on individual pollutants).

Response: The authors accepted this comment and revised the text.

Section 4.0. The phrase, "the ambient levels for these pollutants are consistent with" UATMP levels should be clarified (e.g., within a factor of 2? *5?* 10?).

Response: The authors accepted this comment and revised the text.

Section 4.1. If there are plans to expand acetaldehyde monitoring, that should be mentioned here.

Response: The authors accepted this comment and revised the text.

The last sentence in the section should be revised to: "Changes in the unit risk estimate and RfC... are possible outcomes..."

Response: The authors accepted this comment and revised the text.

Section 4.3. We suggest deleting Figure 4-3, which adds little.

Response: The authors accepted this comment and deleted Figure 4-3.

Beyond the simple screening procedure described above, it is inappropriate to compare short-term data to a long-term benchmark (as done at the top of page 18). Since formaldehyde clearly is an important risk driver, only long-term concentrations should be compared with chronic dose-response values.

Response: The authors accepted this comment and revised the text.

The first two paragraphs on page 18 state that these data "are comparable, and they support the ambient formaldehyde data..." This would be better phrased as "are consistent with the ambient formaldehyde data..."

Response: The authors accepted this comment and revised the text.

In the 3rd paragraph, the phrase "many times its 70-Bench value" is too vague. This should be revised to reflect the degree of difference, e.g., formaldehyde exceeded its 70-bench value by a factor of five, while no other pollutant exceeded its 70-bench by more than a factor of 1.5."

(Note that these numbers are pulled out of the air, as examples.) The last sentence in the 3rd paragraph should say "greater than its noncancer benchmark."

Response: The authors accepted these comments and revised the text.

Section 4.4. The report mentions "an uncertain estimate drawing on emissions inventory" in the 1st paragraph, implying low confidence in the estimate for arsenic. Sections of the report discussing other substances should receive analogous statements of confidence in those inventories. As written, the report seems comfortable with those other inventories, but this apparent confidence needs to be justified. The second to last sentence in the 4th paragraph should say, "does not appear to pose a **significant** risk." This section should also mention plans, if any, to improve the detection limit (e.g., longer sample times, etc.).

Response: The authors accepted these comments. Because creating an emissions inventory was not a task of the St. Louis CAP, the authors removed the implication of low confidence in the estimate for arsenic from the text. Secondly, the Technical Team of the St. Louis CAP has no plans to improve the detection limit.

Section 4.5. The 4th paragraph should cite the 1996 NATA national scale assessment as the source for the chromium speciation recommendation. This is not general OAQPS policy. Also, referring to it as a "best judgment" is misleading. 34 percent was OAQPS 's estimate of a reasonable maximum, as the description at the end of the 4th paragraph makes clear.

Response: The authors accepted these comments and revised the text.

In the 5th paragraph, the last sentence should say "no significant risk". As with arsenic, any plans to improve the detection limits should be mentioned.

Response: The authors accepted these comments and revised the text. The Technical Team of the St. Louis CAP has no plans to improve the detection limit.

Section 4.6. It was not immediately clear to us where diesel PM was measured. In the 3rd paragraph, the statement that the EPA method is more robust than SCAQMD's method needs some support. Why is this so?

Response: The authors accepted these comments. The St. Louis CAP measured diesel PM at the Grant School site. The authors removed the statement referring to the U.S. EPA method as more robust than SCAQMD's method from the text.

In the 5th paragraph, revise to "This ambient level is well below the CAP noncancer benchmark." (And the RfC for diesel should be added to the appropriate table further forward in the report.)

Response: The authors accepted these comments and revised the text.

Section 4.7. This section lacks necessary detail. It should provide at least some summary information on how the Atlanta data were collected, why they are relevant to the question of

VOC sources in St. Louis, and why no other roadside data were included in this section. The suggestion that the pattern at the Grant station is a sampling artifact is not supported by the analysis. The idea should be to determine whether component distributions are similar or different than roadside data, and Grant appears to be different. The analysis does not provide any basis to rule out the existence of a different source.

Response: The authors accepted these comments and revised the text.

Section 4.8. This information seems to be introductory, and we suggest either moving it forward in the report or removing it. If it remains, the word "low" should be deleted from the first sentence.

Response: The authors accepted this comment and removed the text.

Section 5.0. The last paragraph suggests that the CAP worked only with NATA county summaries. If so, this is unfortunate, because EPA made the NATA tract-level exposure and risk outputs publicly available (http://www.epa.gov/ttnlatw/nataltedlexporisk.html), and would have provided the tract-level ambient concentration estimates if asked.

Response: The authors accepted this comment. The report now presents a comparison of the St. Louis CAP ambient concentrations of the five pollutants of concern to the 1999 NATA estimated tract-level ambient concentrations of these five pollutants.

Comparing the CAP monitored values with NATA county averages is clearly not valid, because the averages reflect every tract in the county (and only during 1996) and the monitors reflect single locations (during other periods). Even comparing monitor data with individual tract NATA results would not be valid because the times and locations would not be the same. We suggest redrafting this section in the form of a discussion of these points, which could then expand into a discussion of the different (and complementary) strengths and weakness of the CAP and the NATA approaches.

Response: The authors did not accept this comment. An expansive discussion of the different and complementary strengths and weakness of the St. Louis CAP and the NATA approaches is beyond the scope of this report.

Section 6.0. This section shows a lot of work, but still leaves some unanswered questions. How do the emissions square with the ambient measurements? What trends have been identified so far? What plans are there in this area for the future?

Response: Due to the extensive editing of this section, this comment is no longer relevant to the text.

Section 7.0. You may want to consider comparing individual pollutant risk estimates and cumulative risk estimates from the CAP to those from NATA using census tract data from the nearest census tracts. This information would support the discussion of uncertainties in Section 8, including possible future changes that would reduce specific uncertainties.

Response: The authors did not accept this comment because at the time of this report's release, the U.S. EPA has not yet made the 1999 NATA risk estimates and cumulative risk estimates publicly available.

Section 8.0. This section should be renamed "Uncertainties." In addition to the information already here, it should (1) mention that acrolein wasn't monitored (and why), (2) discuss the limited spatial coverage, and (3) discuss the lack of PM 10 metals analyses (and why).

Response: The authors accepted these comments and revised the text.

Section 9.0. In the 2nd paragraph, first sentence we suggest saying, "The CAP monitoring sampled and analyzed...." which removes the "most effective methods" wording. A legitimate claim of most effective methods would require additional justification (i.e., arguments that every alternative would have been less effective). This would be pointless: it's a good study, and it should stand on its own merits.

Response: The authors accepted this comment and revised the text.

Recommendation #2 on pg. 35 does not follow from the study, which focuses on chronic exposures.

Response: The authors accepted this comment and removed the text.