

Appendix D

Air Toxics

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Appendix D

Air Toxics

Introduction

In November 1999, the MPCA Staff Paper on Air Toxics cited 11 pollutants of concern because levels in Minnesota exceeded inhalation health benchmarks (levels below which health impacts are not expected) based on monitoring and/or modeling in some areas of Minnesota. After further analysis, here is the status of the four chemicals for which the MPCA recommended action:

- Benzene remains a pollutant of concern in the outdoor air despite the fact that levels are declining slightly. Other Minnesota studies show even higher levels of benzene indoors than outdoors.
- Formaldehyde levels remain above the current cancer health benchmark at most sites and are a concern, but the benchmark may increase based on improved toxicity information.
- Carbon tetrachloride levels have dropped below the inhalation health benchmark at all sites monitored since 1998 (the substance was banned from production in 1996).
- Chloroform exceeded the inhalation health benchmark at one monitoring site in International Falls, probably due to nearby paper mill emissions. The U.S. paper mill has been required to further reduce chloroform-forming chemicals. The Minnesota Department of Health reviewed the health benchmark information and recommended a revised inhalation health benchmark. Chloroform does not exceed this revised benchmark at any sites, including International Falls.

Diesel exhaust is a concern

Diesel exhaust was recommended for further study and the MPCA believes there is enough data to warrant preventative steps to reduce diesel exhaust emissions (see Appendix E, Diesel Exhaust) and to recommend additional research to better understand the problem in Minnesota. The MPCA is still improving its capacity to monitor the other six pollutants of concern recommended for further study.

Noncancer effects may be more important

For Minnesotans as a whole, mobile sources and area sources (small stationary sources) are estimated to be much bigger contributors to increased cancer risk from breathing outdoor air toxics compared with point sources (large stationary sources). While the MPCA continues to be concerned with the cancer risk associated with breathing air toxics, including diesel exhaust, the non-cancer effects may be even more important (partly because of the conservatism in the method used to derive the cancer inhalation health benchmarks). In addition, point source industrial facilities and numerous smaller sources may be responsible for elevated air pollution levels for people living nearby.

Scientific uncertainty calls for a precautionary approach

Scientific knowledge of the health and ecological effects caused by particular pollutants is improving, but the MPCA only monitors and models a fraction of the anthropogenic (human-caused) pollutants emitted into the air each day. These pollutants may have cumulative effects, but there is little research available on risk to public health from exposure to multiple pollutants. The inhalation health benchmarks are set for exposure to individual pollutants. Depending on the use of the inhalation health benchmarks, such as for environmental reviews of major point source emissions, risks may be described as the sum of the estimated risks from individual pollutants. In this report, MPCA is making individual comparisons of inhalation health benchmarks with ambient concentrations.

Regulatory approaches cannot keep up with scientific understanding; therefore a precautionary approach to reduce air toxics is appropriate. The action steps the MPCA is taking are described in the Action Steps-Mobile Sources (L) and Action Steps -Stationary Sources (M) Appendices.

1.0 Definitions

1.1 Pollutant and Source Definitions

Basic Air Toxic Definition

Toxic air pollutants, or air toxics, include a wide variety of different chemicals released to air that are known or suspected to cause serious harm to individuals exposed to high enough amounts.

Which Chemicals are Air Toxics?

While there is no single complete list of toxic air pollutants, several partial lists have been compiled to identify those of relatively greater concern. These lists were developed based on available, but often limited, information about their toxic effects, the amounts released to the air, and their measured ambient air concentrations. Considering that over ten thousand chemicals are listed for use in the U.S., clearly many chemicals have not been evaluated.

- Prior to the 1990 Clean Air Act Amendments Congress defined “hazardous air pollutants” (HAPs) as air pollutants which EPA believed cause, or contribute to, air pollution which may reasonably be anticipated to result in an increase in mortality or an increase in serious irreversible, or incapacitating reversible, illness. The 1990 Clean Air Act Amendments redefined HAPs to be 188 specific chemicals. The list is provided on EPA’s web site at <http://www.epa.gov/ttn/uatw/pollsour.html>.
- As required by Section 112(k) of the 1990 Clean Air Act Amendments, the U.S. Environmental Protection Agency assessed the amounts of chemicals released to urban air and developed a list of 33 Priority Air Toxics for the Integrated Urban Air Strategy. They are listed on EPA’s web site at <http://www.epa.gov/ttn/uatw/urban/list33.html>.

After this list was developed, EPA added diesel exhaust particulate matter as another pollutant of significant concern.

- The MPCA Staff paper on Air Toxics (MPCA, 1999) identified chemicals in Minnesota's air that were found, either through direct measurement or modeling efforts, to be present at concentrations greater than health-based benchmarks. An update on the MPCA Staff Paper on Air Toxics is provided in section 3.

These are not exhaustive lists. Additional chemicals may also be considered toxic air pollutants or "air toxics". For example, hydrogen sulfide can clearly be toxic, even lethal, at high enough concentrations in air, yet it does not appear on any of the lists described above. The criteria air pollutants (sulfur dioxide, ozone, particulate matter, nitrogen oxides, lead, and carbon monoxide) may also cause serious health effects and so may also be considered "toxic" air pollutants.

Several factors limit scientists' ability to identify all toxic air pollutants of concern.

- For most chemicals scientists lack comprehensive toxicity information and exposure data. Health-benchmarks for inhalation exposure are only available for a fraction of the chemicals released to air.
- Health-benchmarks are developed to be air concentrations likely to be without appreciable risk of harmful effects on humans. However, depending upon the chemical, the level that could cause harm may be slightly higher than or far above the inhalation health benchmark. Lower benchmarks indicate that the chemical is either relatively more dangerous or that it is a chemical for which little information is available.
- Current scientific methods have been unable to measure adverse health effects from inhalation exposures to most air toxics in the range of ambient outdoor air concentrations. It is uncertain whether this is because information is lacking and the epidemiology methods are insensitive or because health effects are not occurring even at levels above the benchmarks.
- People are exposed to mixtures of chemicals in the air they breathe, not individual chemicals. Information on the toxicity of these mixtures is lacking.
- With respect to a number of volatile organic compounds found in outdoor ambient air, an individual's personal exposures, from their daily activities such as pumping gas, using consumer products and spending time indoors, often lead to higher exposures to these chemicals than breathing the outdoor air.
- Recent efforts to identify priority toxic air pollutants have been limited to potential assessment of risks of breathing these chemicals in air. The environmental and human health impacts of air toxics that persist and accumulate in the environment and the food chain, such as mercury, dioxin, and certain pesticides, have not been considered in these assessments.

Crossover between air toxics and criteria pollutants

Although Congress defined the Hazardous Air Pollutants to increase their assessment and promote reduction efforts of chemicals not already regulated as criteria pollutants, there is crossover between these two regulatory definitions. For example, the criteria pollutant particulate matter (PM) is a complex and variable mixture. Depending on its emission source, it may include toxic compounds such as mercury, cadmium, vanadium, polycyclic aromatic hydrocarbons (PAHs), dioxins, furans, etc. Furthermore, lead is both a criteria pollutant and HAP. Similarly, Volatile Organic Compounds (VOCs) are a complex mixture of pollutants that fall under the criteria pollutant regulations due to their ability to participate in the chemical reactions that form ozone. At the same time, many individual VOCs are considered to be air toxics.

Sources

Toxic air pollutants are released to the air by manufacturing operations, cars, trucks, power plants, businesses, consumer products, and other combustion processes such as woodburning, burning trash, etc. Natural sources of toxic air pollutants include plants, volcanos, forest fires and microbes. When discussing emissions sources, sources are usually labeled as ‘point,’ ‘area,’ or ‘mobile’ sources. Point sources are usually large, permitted facilities. In the *Air Quality in Minnesota* report, point sources are referred to as “large stationary sources.” Area sources are smaller, ubiquitous sources such as gas stations and residential wood burning. In the *Air Quality in Minnesota* report, area sources are referred to as “small stationary sources.” Mobile sources are cars, trucks, locomotive, construction equipment, aircraft, and other small non-point sources.

1.2 Health Information Definitions

Effects

In general, high enough exposures to various toxic chemicals can lead to a range of serious health effects. Examples of serious health effects include: harm to a developing fetus or child, interference with successful reproduction, cancer, harm to specific organs, such as the liver, kidney, bones, heart, skin, etc., or to systems, such as the nervous, blood-forming, respiratory, and immune systems. The minimum chemical exposure that will result in a specific effect depends on the chemical and the particular effects. Some effects are very apparent, while others, such as behavioral changes or slight changes in blood chemistry, are more subtle and difficult to measure and may not be identified in toxicity tests. Hazards of high level exposures to a number of urban air toxics were described in the MPCA Staff Paper (MPCA, 1999). EPA has summarized the hazards associated with toxic air pollutants. These summaries can be found at the following web site:
<http://www.epa.gov/ttn/uatw/hapindex.html>.

Dose Response Concept in Toxicology

Chemicals differ in their abilities to harm people. This is why some chemicals are considered to be “poisonous” and other chemicals to be “safe”. In fact, essentially all chemicals, at high enough exposure levels, can cause harm. People who study poisons often describe this by the phrase “the dose makes the poison.” For some chemicals there are small exposure levels that most scientists believe cause no harm. These chemicals are considered to have a threshold. A threshold is an exposure level that causes no harm. For example, when diluted enough, an acid may be safe to touch. For other chemicals, including many that cause cancer, regulatory scientists assume that even the smallest amounts of exposure can slightly increase a person’s chance of getting cancer. Higher exposures increase their chance of getting cancer even more. This non-threshold phenomenon that has been ascribed to carcinogens may also apply to some effects of other chemicals. Current EPA guidance also recognizes that a threshold may exist for some carcinogens.

Properties and Exposures

Because the definition of toxic air pollutants is so broad, it includes chemicals from many classes of chemicals encompassing a broad range of physical chemical properties. Air toxics include both organic and inorganic chemicals, volatile and semi-volatile organic compounds, and metals. Once released to the environment, toxic air pollutants may occur as gases, vapors, or as liquid droplets or solid particles (aerosols). Some “persistent” chemicals remain in the environment for long periods of time, while others undergo chemical reactions and change to different, more or less hazardous, chemicals. Some occur singly while others occur as complex mixtures. Depending on their chemical physical properties and fate in the environment, some such as the volatile organic compounds remain primarily in the air. Some, which readily dissolve in water, may be found in rainwater, lakes and rivers. Others, such as semi-volatile organic compounds (chlorinated pesticides and dioxins) and some metals (mercury) tend to remain in the environment for long periods of time and can accumulate in the food chain. Thus, although air toxics are, by definition, initially released to the air, people can contact these chemicals different ways, through different routes of exposure, such as by breathing the air, drinking water, or eating food. More information on these bioaccumulative chemicals is included in [Appendix F](#), Persistent Bioaccumulative Toxics.

What are Inhalation Health Benchmarks?

An inhalation health benchmark is a concentration of an individual chemical in the air that, based on available information, is considered essentially safe for the public to breathe. The inhalation health benchmarks are lower concentrations extrapolated from higher concentrations that have been shown in animals or humans to cause adverse effects. Exposures to air concentrations somewhat higher than the inhalation health benchmarks may also be safe, but there is not enough information to know how much higher, if any, would be considered safe. Inhalation health benchmarks do not indicate at what higher

concentrations actual health impacts would likely occur. More information on inhalation health benchmarks and their development is available in section 4.0.

How do Benchmarks and National Ambient Air Quality Standards (NAAQS) Differ?

Different approaches are used in setting inhalation health benchmarks, also known as health risk values (HRVs) in Minnesota (see section 4.1 for more information on the HRVs), and the NAAQS. Inhalation health benchmarks and NAAQS are not derived by the same methods or applied in the same manner, nor are they designed to provide equivalent levels of protection. The HRVs were not developed with a specific intended application, while the NAAQS are chemical concentrations associated with monitoring requirements, specific averaging times, and enforcement consequences. Because the NAAQS are enforceable standards, setting them requires a much higher burden of proof to demonstrate health effects at levels of potential environmental concentrations than is used for setting the HRVs.

U.S. EPA attempts to set the NAAQS at concentrations with a margin of safety below clearly defined human health effects. Because there are economic consequences for exceeding an enforceable NAAQS, there is clear pressure not to set them farther below the human effect levels than necessary. In contrast, the HRVs are derived as protective levels that the Minnesota Department of Health (MDH) is confident are without appreciable harm. The HRV development process uses best available toxicity and epidemiology data, but where greater uncertainties exist, the resulting HRV concentrations are set in a health-protective manner.

EPA selected the 15 ug/m³ annual average PM_{2.5} NAAQS because it was slightly lower than the lowest level that was most likely to cause serious effects (e.g., death, asthma attacks, bronchitis). EPA used epidemiological studies that measured human health impacts including mortality and less severe effects to set the PM_{2.5} annual standard, and reported clear evidence of short-term PM_{2.5} health effects most evident at concentrations of 16 ug/m³ and higher. EPA also reported data showing measurable health effects from long term exposures at an average PM_{2.5} concentration of 18ug/m³. EPA concluded that 15 ug/m³ would provide a "margin of safety" and set the PM_{2.5} NAAQS at that concentration. EPA clearly stated that the required margin of safety did not mean that no health effects would occur to anyone at concentrations below the standard, or even that the new standard would eliminate all deaths from PM_{2.5}. EPA estimated that the standard, when enforced, would lead to about 15,000 fewer deaths in the U.S.

Members of the medical community petitioned EPA to adopt standards significantly lower than the current proposed fine particulate matter (PM_{2.5}) standards to protect the more vulnerable residents (62 FR 38651). EPA did not select a lower level because of the greater uncertainty in measuring adverse effects at the lower concentrations. The PM_{2.5} standard is currently under U.S. Supreme Court review.

2.0 Sources and Emissions

2.1 Chemicals Highlighted in MPCA's Staff Paper

A summary of emission contributions by principal source category (point, area, and mobile) to pollutants highlighted in the MPCA Staff Paper is shown in Table 1. In the *Air Quality in Minnesota report* point and area sources are referred to as “large stationary” and “small stationary” sources, respectively. Background contributions are also shown in this table as a percent of the modeled mean concentration. Background concentrations are made up of chemicals arriving in our air via long-range transport, resuspension of historical emissions, and emissions from natural sources. The analysis is based on data from the US EPA Cumulative Exposure Project (CEP) study (Woodruff et al, 1998; Caldwell et al, 1998; Rosenbaum et al, 1999; SAI, 1999) for Minnesota.

Table 1. Emissions by Principal Source Category (Data from EPA CEP study)

Pollutant	Total Emissions (ton/day)	Point Source Contribution (%)	Area Source Contribution (%)	Mobile Source Contribution (%)	Background Concentration as Percent of Modeled Mean Concentration (%)
Acrolein*	2.13		64	36	0
Arsenic	0.09	94	4	2	0
Benzene	25.76	5	28	67	32
Butadiene	3.89	2	32	66	0
Carbon Tetrachloride	0.04	42	58		100
Chloroform	0.34	83	17		94
Chromium	0.07	83	12	5	0
Ethylene Dibromide	0.00				100
Formaldehyde*	15.40	9	33	58	26
Nickel	0.18	77	19	4	0

* The emissions for these pollutants are for direct emissions only. Secondary formation of acrolein was not studied in the CEP study. But the nationwide source contribution for the secondary formed formaldehyde is similar as the Minnesota source contribution for direct formaldehyde emissions.

Overall, point sources are the primary emission sources for metals and chloroform, contributing about 80% or more of the emissions. Point sources also contribute 42% of carbon tetrachloride emissions. However, their contributions to other volatile organic compounds (VOC), such as formaldehyde, benzene, 1,3-butadiene, and acrolein, are either insignificant or negligible. This is an overall result for the entire state but not necessarily for the ambient air concentrations next to a particular facility where the facility may contribute significantly.

In contrast with point sources, mobile sources have insignificant or negligible contributions to emissions of metals, chloroform, and carbon tetrachloride, but dominate the emissions of

benzene, formaldehyde, and 1,3-butadiene. Mobile sources also contribute significantly to acrolein emissions; the contribution is 36%.

Area sources are responsible for emissions of almost all pollutants listed in Table 1. Their contributions are more than 12% for each pollutant (except for arsenic and ethylene dibromide). Area sources also are the primary emission sources for acrolein and carbon tetrachloride.

Formaldehyde is present in the atmosphere due to two processes: direct emissions and secondary formation. The secondary formation, or creation of chemicals by atmospheric chemical reactions, of formaldehyde could account for as much as 88 percent of formaldehyde concentrations in air. Table 1 lists direct emissions of formaldehyde in Minnesota. EPA's Cumulative Exposure Project (CEP) study does not provide information on secondarily-formed formaldehyde for Minnesota. However, it shows that the nationwide source contribution for secondarily-formed formaldehyde is similar to the Minnesota source contribution for direct formaldehyde emissions. That means mobile sources are the major contributors to both direct and secondary formaldehyde emissions.

Estimated emissions of carbon tetrachloride were about 0.04 ton/day in Minnesota in 1990. Although the production of carbon tetrachloride was gradually reduced by the phase-out in the end of 1995, chemical-manufacturing processes, waste-water-treatment processes, waste incineration, landfill waste disposal and petroleum-refining processes emitted the chemical in 1990. However, a significant part of measured ambient concentrations for carbon tetrachloride is attributed to accumulated background concentrations due to its long atmospheric lifetime. There are no emissions estimated for ethylene dibromide estimated by the CEP study.

It should be noted that information is limited for air toxics emissions in Minnesota. The EPA CEP study used 1990 data and an estimation method that was not recommended by the EPA Office of Air Quality Planning and Standards, Emission Factor and Inventories Group. Large uncertainties were associated with the study results. The MPCA staff completed a comprehensive emission inventory for point, area, and mobile sources for calendar year 1996. Further analysis of source contributions is presented in Section 2.2.

2.2 MPCA's Emission Inventory and EPA's National Toxics Inventory

Environmental agencies use emissions inventories to determine pollutant sources, model chemical concentrations, and other regulatory work. Each inventory may use unique source category definitions or include different pollutants. It is important to understand these differences when comparing inventory and modeling data.

After the EPA CEP study (Woodruff et al, 1998; Caldwell et al, 1998; Rosenbaum et al, 1999; SAI, 1999), MPCA staff developed a comprehensive statewide air toxics emission inventory (MNEI) (MPCA, 2000) for point, area, and mobile sources for calendar year 1996. The U.S. EPA, also, compiled a 1996 National Toxics Inventory (NTI) (U.S. EPA,

2000) with emission data submitted from the MPCA for certain point and area sources. The definitions of point and area sources, as well as pollutants estimated, vary from one emission inventory to another. Summarized comparisons are shown in Tables 2 and 3. Final NTI data may differ slightly from that presented in following sections due to ongoing quality assurance.

Table 2. Comparison of definitions and coverage of point sources in three inventories

Inventory	Point	Number of Facilities	Number of Counties
CEP	<ul style="list-style-type: none"> • Facilities reporting to the Toxic Release Inventory (TRI) <ul style="list-style-type: none"> • Processing > 12.5 tons, or • Using > 5 tons of a Hazardous Air Pollutant (HAP) And <ul style="list-style-type: none"> • Point sources from the National Interim Volatile Organic Compound (VOC) or Particulate Matter less than 10 microns (PM₁₀) Inventories <ul style="list-style-type: none"> • Emitting > 100 tons for VOC, Carbon Monoxide (CO), Nitrogen Oxides (NO_x), Sulfur Dioxide (SO₂), or PM 	493	66
96MNEI	<ul style="list-style-type: none"> • Facilities required to submit their annual inventories of criteria pollutants with potential to emit <ul style="list-style-type: none"> • ≥ 100 tons of VOC, CO, NO_x, or PM, • ≥ 50 tons of SO₂, • ≥ 25 tons of PM₁₀ • ≥ 0.5 tons of lead, • ≥ 10 tons of one HAP, or • ≥ 25 tons of any combination of HAPs and <ul style="list-style-type: none"> • Facilities reporting to the Toxic Release Inventory (TRI) <ul style="list-style-type: none"> • Processing > 12.5 tons, or • Using > 5 tons for a MNEI pollutant 	827	82
96NTI	<ul style="list-style-type: none"> • Major sources defined in the CAA with potential to emit <ul style="list-style-type: none"> • ≥ 10 tons of one HAP, or • ≥ 25 tons of any combination of HAPs and <ul style="list-style-type: none"> • Facilities with source-specific emissions from 96MNEI 	210	54

The area source definition is dependent on the point source definition. An area source is defined as any stationary source of targeted pollutants, which does not qualify as a point source. Therefore, one source classified as an area source in one emission inventory may be covered as a point source in another emission inventory.

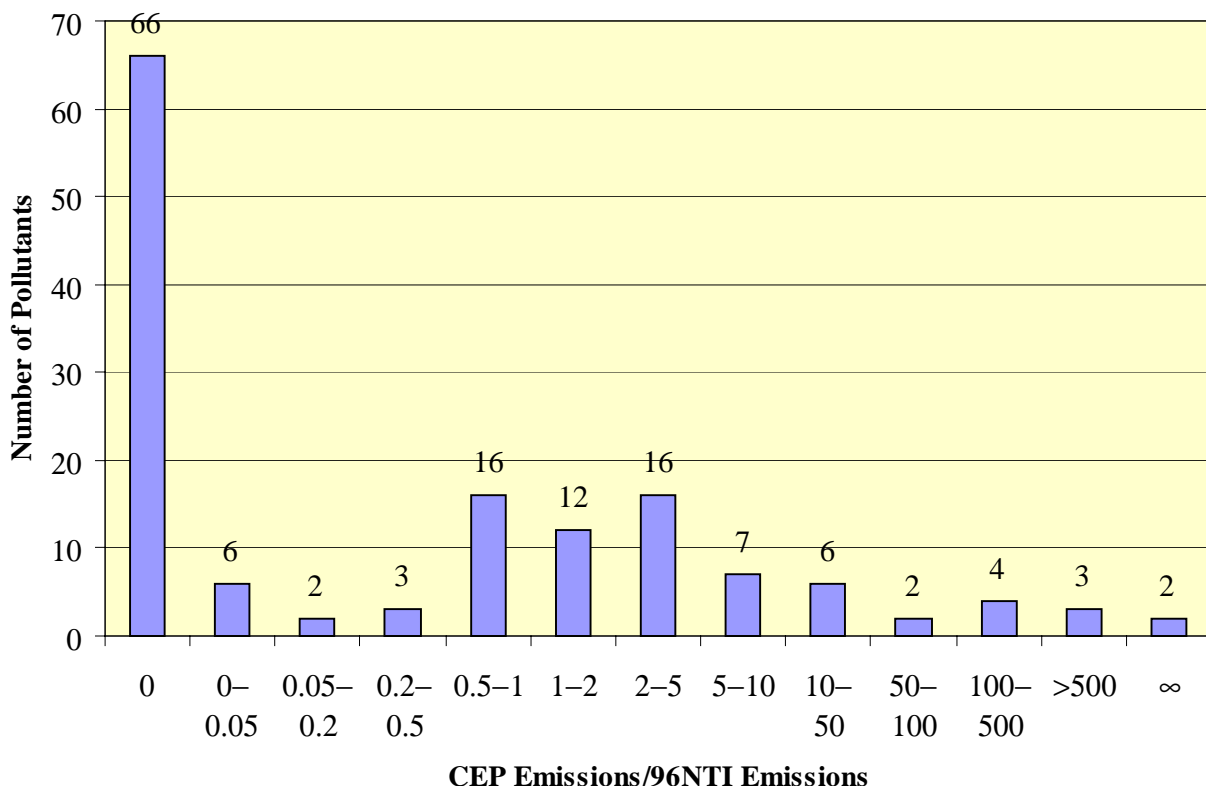
The definition of mobile sources, sometimes referred to as non-stationary sources, is consistent in all three emissions inventories. Mobile sources include on-road vehicles and non-road sources, such as aircraft, locomotives, construction equipment, lawn mowers, and recreational vehicles.

Table 3. Comparison of pollutant coverage in three inventories

<u>Inventory</u>	<u>Year</u>	<u>Targeted Pollutants</u>	<u>Estimated Pollutants</u>
CEP	1990	188 HAPs	79
96MNEI	1996	109 MN Pollutants	86
96NTI	1996	188 HAPs	143

Although both the CEP and the 96NTI focused on the 188 HAPs, only 77 pollutants were common in these two inventories. A comparison of CEP emissions and 96NTI emissions is shown in Figure 1. Emissions for 66 pollutants were estimated in the 96NTI but not in the CEP (CEP emissions/96NTI emissions equals 0). Two pollutants were estimated with emissions in the CEP but not in the 96NTI (CEP emissions/96NTI emissions equals ∞). Estimated emissions for 28 pollutants were within a factor of 2 range for these two emission inventories ($0.5 < \text{CEP emissions}/96\text{NTI emissions} < 2$).

Figure 1. Comparison of emission estimates from the CEP and the 96NTI



A comparison of 96MNEI emissions and 96NTI emissions is shown in Figure 2. There are 52 common pollutants in these two emission inventories. However, emissions for 91 pollutants were estimated in the 96NTI but not in the 96MNEI (96MNEI emissions/96NTI emissions equals 0), 34 pollutants in the 96MNEI but not in the 96NTIP (96MNEI emissions/96NTI emissions equals ∞). Estimated emissions for 37 pollutants were within a factor of 2 range for these two emission inventories ($0.5 < 96MNEI \text{ emissions}/96NTI \text{ emissions} < 2$).

Figure 2. Comparison of emission estimates from the 96MNEI and the 96NTI

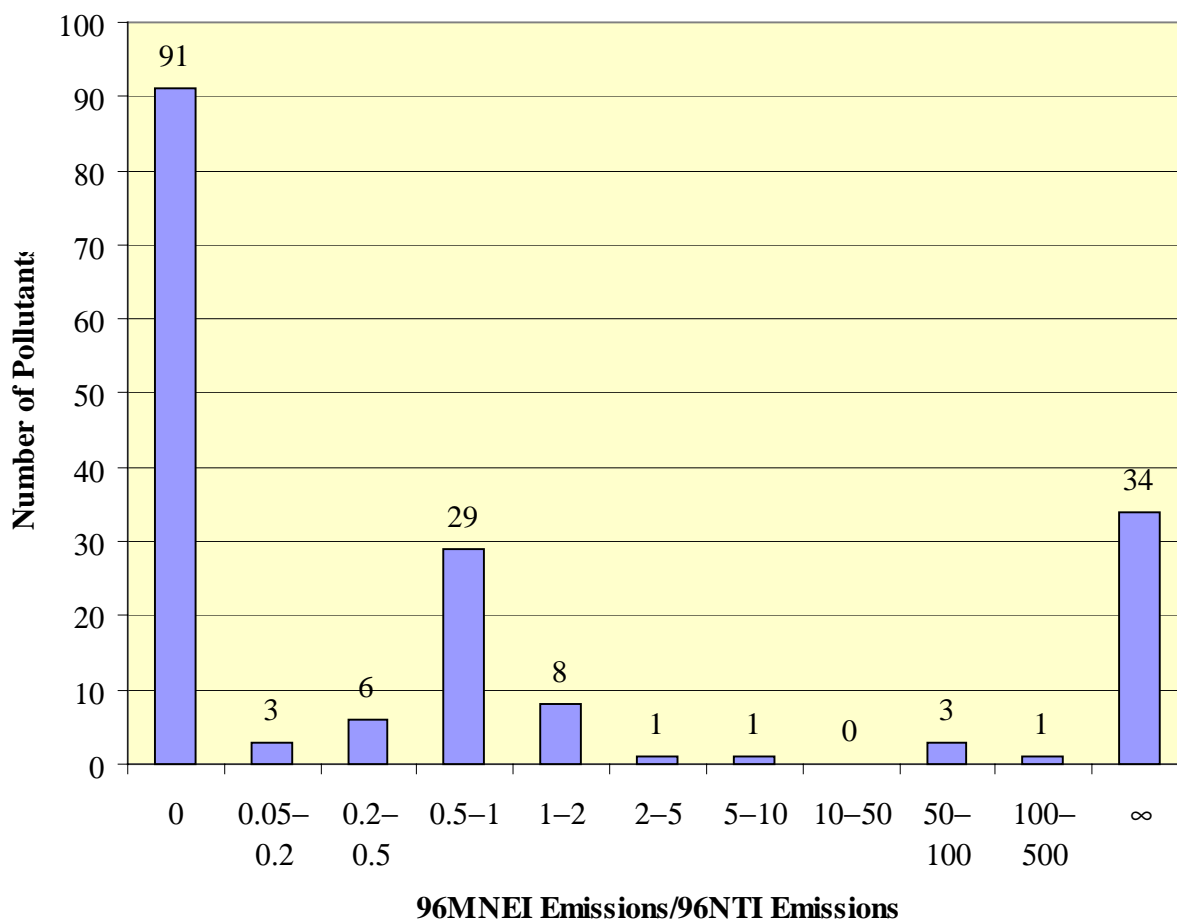


Table 4 shows a comparison of source contributions for the 10 of the 11 pollutants of concern in the MPCA Staff Paper (no data for diesel particles/polycyclic organic matter). Overall, the CEP and 96NTI emissions are within a factor of two for acrolein, benzene, formaldehyde, and chromium. However, CEP emissions are 2.5 to 5.5 times of 96NTI emissions for carbon tetrachloride, chloroform, arsenic, and nickel. On the other hand, CEP estimated about 45% of 96NTI emissions for 1,3-butadiene and zero emissions for ethylene dibromide. In contrast with CEP results, the 96MNEI and 96NTI emissions are

within a factor of two for all pollutants except acrolein for which 96MNEI estimated about 18% of 96NTI values. The comparison of emissions from individual principal source categories shows a larger range than comparison of total emissions. This is mainly due to different source definitions, source coverage, and emission estimation methods.

Table 4. Comparison of source contributions for the 10 pollutants of concern

Pollutant Name	CEP/96NTI (%)				96MNEI/ 96NTI (%)			
	Area	Mobile	Point	Total	Area	Mobile	Point	Total
Acrolein	90.64	105.79	34.47	94.53	0.00	38.43	337.29	18.36
Benzene	81.36	64.24	979.06	72.10	57.08	49.00	129.78	51.34
1,3-Butadiene	19.77	104.43	619.89	44.68	78.45	62.81	40.24	73.94
Carbon Tetrachloride	156.40		1604.51	253.92	85.11		100.00	86.11
Chloroform	196.40		485.69	390.51	99.53		100.00	99.85
Ethylene Dibromide	0.00		0.00	0.00	98.44		106.27	98.44
Formaldehyde	58.18	92.28	144.63	79.24	1.76	85.36	193.71	52.45
Arsenic	383.25	1324.69	553.55	550.80	22.02	3.21	96.27	90.60
Chromium	33.87	125.85	254.24	139.10	73.02	61.14	121.79	94.51
Nickel	139.38	85.47	319.51	238.05	52.45	15.41	102.10	77.47

Every emission inventory has its limitations and uncertainties. MPCA staff developed the 96MNEI with a full understanding of the uncertainties and limitations for this inventory. The U.S. EPA compiled the CEP and 96NTI. MPCA staff could not access every detail of these inventories. Therefore, the ability of MPCA staff to evaluate the data from EPA's CEP and 96NTI is limited, so the MPCA decided to use the 96MNEI in the development of air toxics control strategies. Table 5 shows a summary results of the 96MNEI, including pollutant names, estimated emissions, and contributions from each principal source category.

Table 5. Summary results of the 96MNEI

Pollutant Name	Cas No.	Total Emissions	Contribution to Total (%)		
		(lb)	Area	Point	Mobile
Polycyclic Aromatic Hydrocarbons (PAHs)					
Acenaphthene	83329	14,403	99.96	0.04	0.00
Acenaphthylene	208968	305,294	99.98	0.02	0.00
Anthracene	120127	20,253	99.53	0.38	0.09
Benz(a)anthracene	56553	29,190	98.72	0.52	0.76
Benzo(a)pyrene	50328	10,016	57.50	41.03	1.47
Benzo(b)fluoranthene	205992	8,808	98.08	0.12	1.80
Benzo(ghi)perylene	191242	6,207	92.78	0.01	7.22
Benzo(k)fluoranthene	207089	3,023	95.24	0.00	4.76
Chrysene	218019	18,097	95.47	0.11	4.42
Dibenz(a,h)anthracene	53703	5,783	99.59	0.01	0.40
Fluoranthene	206440	29,052	99.12	0.26	0.61

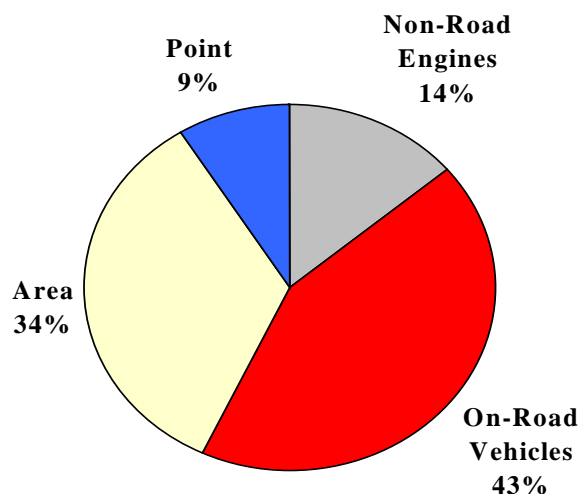
Pollutant Name	Cas No.	Total Emissions	Contribution to Total (%)		
		(lb)	Area	Point	Mobile
Fluorene	86737	34,573	99.95	0.05	0.00
Indeno(1,2,3-cd)pyrene	193395	28,823	99.91	0.00	0.09
Naphthalene	91203	1,042,418	97.16	2.78	0.06
Phenanthrene	85018	112,421	99.90	0.02	0.08
Pyrene	129000	34,720	99.52	0.01	0.46
Non-Metal Compounds (Excluding PAHs)					
Acetaldehyde	75070	2,818,299	0.00	2.20	97.80
Acetone	67641	2,373,610	70.29	2.07	27.64
Acrolein	107028	302,588	0.00	32.48	67.52
Acrylonitrile	107131	8,369	98.97	1.03	0.00
Atrazine	1912249	679,139	100.00	0.00	0.00
Benzaldehyde	100527	251,702	2.72	0.01	97.27
Benzene	71432	13,389,480	27.77	0.99	71.24
1,3-Butadiene	106990	4,694,301	75.67	0.07	24.27
Butyraldehyde	123728	111,582	0.00	0.00	100.00
Carbon tetrachloride	56235	10,304	92.18	7.82	0.00
Chlorobenzene	108907	336,981	99.96	0.04	0.00
Chloroform	67663	63,604	32.80	67.20	0.00
Crotonaldehyde	123739	165,315	0.00	0.00	100.00
1,2-Dichlorobenzene(o)	95501	42,557	100.00	0.00	0.00
1,3-Dichlorobenzene(m)	541731	2,723	100.00	0.00	0.00
1,4-Dichlorobenzene(para)	106467	389,140	100.00	0.00	0.00
Di-n-butyl phthalate	84742	1,038	85.69	14.31	0.00
Diethylhexyl phthalate (DEHP)	117817	2,689	0.00	100.00	0.00
Ethylbenzene	100414	4,273,668	15.44	4.94	79.62
Ethylene dibromide (Dibromoethane)	106934	7,127	100.00	0.00	0.00
Ethylene dichloride (1,2-Dichloroethane)	107062	9,544	99.68	0.32	0.00
Ethylene oxide	75218	831,850	99.99	0.01	0.00
Ethylidene dichloride (1,1-Dichloroethane)	75343	2,017	100.00	0.00	0.00
Formaldehyde	50000	7,439,264	1.53	17.30	81.17
Glycol ethers	0	1,059,986	20.05	79.95	0.00
Hexachlorobenzene	118741	1	14.77	85.23	0.00
Methyl bromide (Bromomethane)	74839	1,079,019	96.34	3.66	0.00
Methyl chloride	74873	119,886	30.13	69.87	0.00
Methyl chloroform (1,1,1-Trichloroethane)	71556	1,951,754	94.00	6.00	0.00
Methylene chloride (Dichloromethane)	75092	1,013,311	62.23	37.77	0.00
Methylene diphenyl diisocyanate (MDI)	101688	1,530	0.00	100.00	0.00
Phenol	108952	236,236	0.61	96.90	2.49
Propionaldehyde	123386	357,488	0.00	1.26	98.74
Propylene dichloride (1,2-Dichloropropane)	78875	397	99.94	0.06	0.00
Styrene	100425	2,933,760	0.09	39.11	60.80
1,1,2,2-Tetrachloroethane	79345	2,322	95.69	4.31	0.00
2,3,7,8-tetrachlorodibenzo-furan (TCDF)	51207319	2	33.14	66.86	0.00
2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)	1746016	0.013	81.05	18.95	0.00
Tetrachloroethylene (Perchloroethylene)	127184	852,254	81.13	18.87	0.00

Pollutant Name	Cas No.	Total Emissions		Contribution to Total (%)		
		(lb)	Area	Point	Mobile	
2,4-Toluene diisocyanate	584849	4	0.00	100.00	0.00	
Toluene	108883	35,530,797	35.20	8.15	56.65	
Total polychlorinated biphenyls (PCBs)	1336363	5	1.60	98.40	0.00	
Total polychlorinated dibenzodioxins (PCDDs)	0	4	92.64	7.36	0.00	
Total polychlorinated dibenzofurans (PCDFs)	0	27	83.78	16.22	0.00	
1,1,2-Trichloroethane	79005	453	60.85	39.15	0.00	
Trichloroethylene	79016	581,361	32.60	67.40	0.00	
Trichlorofluoromethane (CFC-11)	75694	450,954	100.00	0.00	0.00	
Trifluralin	1582098	42,490	100.00	0.00	0.00	
1,2,4-Trimethylbenzene	95636	3,051,818	0.00	3.55	96.45	
1,3,5-Trimethylbenzene	108678	1,027,865	0.00	0.33	99.67	
Trimethylbenzene	2551137	260,369	93.98	6.02	0.00	
Vinyl chloride	75014	30,092	99.09	0.91	0.00	
Vinylidene chloride (1,1-Dichloroethylene)	75354	5,254	99.99	0.01	0.00	
Xylenes (Isomers and mixture)	1330207	21,824,066	30.25	8.66	61.10	
m-Xylenes	108383	5,051,469	3.79	0.01	96.21	
m/p-Xylenes	0	55,126	86.94	0.21	12.86	
o-Xylenes	95476	3,467,951	16.41	5.01	78.57	
p-Xylenes	106423	109,303	100.00	0.00	0.00	
Metal Compounds						
Antimony	7440360	1,415	0.00	100.00	0.00	
Arsenic	7440382	11,168	1.54	98.43	0.04	
Beryllium	7440417	253	18.70	81.30	0.00	
Cadmium	7440439	2,376	10.30	89.70	0.00	
Chromium	7440473	33,133	37.86	58.54	3.60	
Chromium (6)	18540299	3,020	65.48	34.52	0.00	
Cobalt	7440484	36,564	90.78	9.22	0.00	
Copper	7440508	49,803	2.67	36.39	60.94	
Lead	7439921	158,652	0.86	94.62	4.52	
Manganese	7439965	87,974	2.26	96.45	1.29	
Mercury	7439976	3,269	7.29	83.06	9.65	
Nickel	7440020	43,915	22.00	76.05	1.95	

Polycyclic aromatic hydrocarbon (PAH) emissions were dominated by area sources, which contributed more than 92.8% of total emissions for 15 PAHs and 57.5% for benzo(a)pyrene. Emissions of metal compounds were mainly from point sources, which were responsible for nearly 60% or more of total emissions for 9 out of 12 metal compounds. Area and mobile sources were responsible for more than 60% of emissions of chromium (6), cobalt, and copper. For non-metal compounds (excluding PAHs), each principal source category accounted for more than two thirds of total emissions of individual pollutants: area sources for 29 pollutants, point sources for 11 pollutants, and mobile sources for 13 pollutants.

The total mass of emissions can be summed to determine sources of air toxics emitted in Minnesota. The total mass of air toxics emissions emitted in Minnesota in 1996 was estimated to be 56,378 short tons. Figure 3 shows the breakdown of total air toxics emissions in Minnesota in 1996. It is important to note that a large mass of emissions is not necessarily indicative of high human health or ecological risk, as discussed in section 1.0.

Figure 3: 1996 Minnesota Air Toxics Emissions by Principal Source Category



The inventory results represent emissions for calendar year 1996 only. The pollutants in the inventory were limited to a subset of 109 air toxics that have emission information. Area source categories covered in the 96MNEI are not comprehensive. Many other area sources need to be explored in the future.

There were a number of uncertainties associated with the methodology used to compile the 96MNEI. The most significant uncertainties resulted from a lack of source-specific emission information, the use of default activity data for non-road mobile sources, and the use of generic emission factors. These uncertainties caused an underestimation for a whole subcategory of non-road mobile sources and inaccurate results for some pollutants from certain source categories. The examples are an overestimation of 1,3-butadiene emissions from Gasoline Marketing and inappropriate ethylene oxide emissions from Industrial Surface Coating. A detailed discussion of uncertainties can be found at MPCA web site: <http://www.pca.state.mn.us/air/toxics.html#3.5>.

Further work is needed to improve the reliability of the Minnesota air toxics emissions inventory to support regulatory activities in the MPCA, the Great Lakes region, and the EPA, but the 96MNEI inventory represents an important milestone towards the development of a comprehensive and reliable emission inventory in Minnesota.

3.0 Air Toxics - Concentrations and Trends

In 1999, the MPCA compared EPA's Cumulative Exposure Project (CEP) modeling with ambient monitoring from Minnesota. Since that time, the MPCA collected more monitoring information and EPA released its National Air Toxics Assessment modeling, based on more recent data than the CEP. This section looks at the MPCA's updated monitoring and EPA's newer modeling data and compares it to earlier data and conclusions.

3.1 Summary of MPCA Staff Paper Modeling and Monitoring

In November 1999, the MPCA completed the *MPCA Staff Paper on Air Toxics* (MPCA, 1999). The MPCA Staff Paper compared ambient air concentration modeling from the U.S. Environmental Protection Agency's Cumulative Exposure Project (CEP) and ambient air monitoring from MPCA's monitoring networks to inhalation health benchmarks. MPCA listed eleven chemicals as pollutants of concern because (1) the CEP model-predicted concentration exceeded the inhalation health benchmark in at least one census tract or (2) the monitored annual average concentration at one or more monitoring sites exceeded the inhalation health benchmark. These substances are listed in Table 6.

Table 6. Pollutants of Concern from the MPCA Staff Paper

POLLUTANT	Exceeded Inhalation Health Benchmark Based on CEP Modeling	Exceeded Inhalation Health Benchmark Based on Monitoring
Formaldehyde	X	X
Benzene	X	X
Carbon tetrachloride	X	X
Chloroform		X
Ethylene dibromide		X
1,3-butadiene	X	<i>No monitoring data</i>
Acrolein	X	<i>No monitoring data</i>
Arsenic	X	X*
Nickel	X	
Chromium	X	X*
Diesel Particles / Polycyclic Organic Matter (POM)	X**	

*Data for arsenic and chromium are mostly below the minimum quantifiable level, but there is an indication that concentrations may exceed inhalation health benchmarks at some sites.

** The inhalation health benchmark for POM was assumed to be that of benzo(a)pyrene.

The MPCA determined that ambient air concentrations of four of these chemicals: formaldehyde, benzene, carbon tetrachloride, and chloroform warranted action. The remaining pollutants were highlighted for further study.

Further details regarding the information in the MPCA Staff Paper were published in Environmental Health Perspectives in September 2000 (Pratt et al, 2000).

In preparation for this legislative report, MPCA updated the analysis of the air toxics monitoring data. In the MPCA Staff Paper, monitoring data from 1991 to mid-1998 was analyzed. In this update, the monitoring data has been analyzed up to the end of 1999. This update is necessary in order to continue tracking concentration trends for the primary pollutants of concern and to help determine for which pollutants MPCA action is warranted.

3.2 Summary of MPCA Air Toxics Monitoring Efforts

Siting of Monitors

The MPCA operates a set of statewide toxic air pollution monitoring sites that developed over several years to address multiple concerns. Some sites were established to measure concentrations in the vicinity of specific point sources. Other sites were established to collect baseline data on air toxics concentrations in the Minneapolis-St. Paul metropolitan area (metro area) and in Duluth. A third group of sites was established as part of a legislatively mandated statewide air toxics monitoring network (SATMN). Throughout this appendix, references to 'SATMN' sites refer only to this subset of monitoring locations, and not the entire monitoring effort of the MPCA. The objective of the SATMN study was to collect one-year snapshots of concentrations at sites throughout the state. These sites were randomly selected with weighting for geographic coverage and population density. Sites were typically located at rooftop level and away from immediate pollution sources following guidance provided by EPA (EPA, 1994). All rooftops are made of either pitch and gravel or rubber membrane. The only sites located at ground level were Holloway and Sandstone.

The MPCA air toxics monitoring network is one of the most extensive of its kind in the U.S. The siting of individual monitors was done by professionals with more than 20 years of experience in siting air pollution monitors. One of the important considerations in monitor siting is to represent the air quality of the general location of the monitor while avoiding the influence of microenvironmental sources such as rooftop vents and stacks, idling vehicles, paint or solvent use and storage, construction activity, etc. These considerations are described in the EPA monitoring guidance document.

The SATMN sites were selected using a peer review process in which three professors from the University of Minnesota provided advice and extensive comments on versions of the network protocol. After several iterations the network design was finalized. The network was designed to characterize typical ambient air concentrations that residents are

potentially exposed to in a city, town, or township. The intent is not to target specific industries, and monitoring equipment has not been placed at the fence line of any industry.

The potential “pool” of sampling sites included all minor civil divisions (MCDs) in the state. (A minor civil division is defined as any community that is a city or a township.) A weighting score that incorporated emissions and population was calculated for each MCD. The MCDs were separated into six geographic regions to ensure geographic coverage of the state. Within each region, the MCDs were ranked by the population and emissions weighting score. The ranked scores were divided into five tiers. Tier one contained the top 20 percent of the scores, tier 2 contained the next 20 percent, tier 3 the next 20 percent, tier 4 the next 20 percent, and tier 5 contained the lower 20 percent. In general, tiers 1 to 3 contained most of the highly populated areas of each region, while tiers 4 and 5 contained MCDs with small populations.

One sampling location (MCD) was selected at random from each tier for sampling. The order in which the five sites in each region were to be monitored was also chosen at random. Within each MCD selected for sampling, a location was chosen that represented the MCD as well as possible given the need for power and security for the monitors. Under this protocol, each MCD within a given tier and a given region had an equal probability of being selected. This procedure means that the monitoring results can be considered representative of the state since the results from each randomly selected site can be related back to the population of sites from which it was drawn.

Results

For the purposes of this update, the MPCA focused on the SATMN sites for mean ambient air concentration data. This was done so that a similar time frame would be compared between sites (all the SATMN sites have one year of data). In addition, the SATMN sites are located away from immediate pollution sources, so bias from near-by point sources was removed. Finally, the SATMN sites are organized under a single study and allow for comparisons with fewer caveats. This focus on the SATMN sites allowed the MPCA to update some of the conclusions originally found in the MPCA Staff Paper.

A listing and characterization of the SATMN monitoring sites is given in Table 7. Note that some of the designations can be deceptive. For example, the Duluth 7550 site is located on a hilltop away from both the town center and most of the industry and traffic. As a result, many of the chemical concentrations seem to correlate better with a small town than an urban area. It is also important to consider the exact location of the monitor.

Table 7: The Statewide Air Toxics Monitoring Network

Site Name	Site I.D.	Monitoring Year	Region	Tier	Population of MCD*	Site Type**
Alexandria	2010	1996-97	3	3	8,251	SATMN-S
Bemidji	2302	1998-99	Added		11,494	SATMN-S
Duluth 7550	7550	1998-99	1	1	85,746	SATMN-U
Elk River	3050	1997-98	2	2	12,811	SATMN-S
Fergus Falls	2005	1997-98	3	2	12,596	SATMN-S
Granite Falls	4003	1997-98	4	4	3,049	SATMN-S
Harding High	871	1998-99	6	2	271,660	SATMN-U
Hibbing	7014	1997-98	1	3	17,964	SATMN-S
Holloway	4500	1998-99	4	5	120	SATMN-R
International Falls 1241	1241	1996-97	Added		7,811	SATMN-S
Little Falls	3049	1996-97	2	3	7,595	SATMN-S
Minnehaha Academy	958	1997-98	6	1	366,480	SATMN-U
Moorhead	2103	1998-99	3	1	33,618	SATMN-U
Pipestone	4002	1996-97	4	3	4,559	SATMN-S
Plymouth	260	1996-97	6	3	57,391	SATMN-U
Rochester	5008	1997-98	5	1	76,865	SATMN-U
Sandstone	1400	1996-97	1	5	280	SATMN-R
St. Cloud	3052	1998-99	2	1	50,143	SATMN-U
Warroad	2401	1997-98	Added		1,815	SATMN-S
Winona	5210	1998-99	5	2	25,805	SATMN-U
Zumbrota	5356	1996-97	5	5	946	SATMN-R

*1994 population data taken from the Statewide Air Toxics Monitoring Study: Background Information and Project Plan, 1996 (MPCA, 1996).

**Abbreviations: SATMN, Statewide Air Toxics Monitoring Network; -R, rural site; -S, small town site; -U, urban site.

For trend analysis, MPCA has relied primarily on monitoring data in urban areas such as the Twin Cities and Duluth and, in some cases, sites near point sources. These monitoring sites have concentrations dating back to 1991 and were the only locations that allowed for long-term trend analysis. The data were analyzed through 1999. The five sites in table 8 were analyzed for trends.

Table 8: Air Toxics Monitoring Sites Used for Trends Analysis

Site Name	Site I.D.	Monitoring Year	Site Type*
Duluth	7549	1994-active	Urban
Holman Field	816	1991-active	Urban
Koch420	420	1991-active	Industrial
Minneapolis Library	945	1991-active	Urban
St. Paul Park	436	1993-active	Industrial

*Abbreviations: Urban - site located to characterize the urban area;
Industrial - site located near an industrial facility.

Analytical Techniques

Three types of samples are collected for MPCA air toxics monitoring: volatile organic compounds (VOCs), carbonyls, and particulate matter $\leq 10 \mu\text{m}$ in aerodynamic diameter (PM_{10}). All sample types are collected for 24 hours every sixth day. VOCs are collected using stainless steel canisters and samples are analyzed using a gas chromatograph/mass spectrometer as prescribed in the U.S. federal reference method TO-14A (EPA, 1999a). Carbonyls are analyzed according to U.S. federal reference method TO-11A (EPA, 1999b). The PM_{10} samples are analyzed for metals using energy dispersive X-ray fluorescence (XRF).

Lower Detection Limits (LDLs)

MPCA reports lower detection limits (LDLs) that are determined as described below.

VOCs and carbonyls: The LDL is determined by the following procedure. A standard is prepared one to five times the estimated LDL. A minimum of seven samples of this standard are processed through the entire analytical method. The resulting concentration data are input to the following equation:

$$\text{LDL} = t \times (\text{SD}), \text{ where}$$

t = the t-value appropriate for a 99% confidence level for the standard deviation with $n-1$ degrees of freedom, and
SD = the standard deviation of replicate analyses.

Metals (XRF): Using the XRF instrument, an element's peak is detected above background with 99% confidence if the peak counts are greater than three times the square root of the background counts:

$$\text{LDL} = (3 \times (\text{Ib})^{1/2}) / \text{Ip} * 1/(\text{T}^{1/2}) * \text{concentration, where:}$$

Ib = background (cps, or counts per second),
Ip = peak (cps), and
T = time.

Protocol for treating values below detection

Although some measurements are below the level of reliable quantification, the information contained in the reading is valuable and should not be discarded. Likewise, it would represent a loss in information to assign some arbitrary value, such as one-half the detection limit. Therefore, all valid data, including values below detection, zeroes and negative values, are retained in the database used for statistical analysis.

In the case of several metals, a large fraction of the measurements are below the lower detection limit (LDL). In addition, since the reading from a blank filter is subtracted from each measurement, there are some negative values in the data. These negative values could be censored in some way, such as converting them to zero (or one-half the LDL). However this censoring would alter the frequency distribution. The best method for treating such data is a matter of debate in the scientific literature. The MPCA chose to retain all the raw values in the data for the statistical analyses reported here.

A blank subtraction is also done with the carbonyl data. With VOCs, there is presently no blank subtraction; however, some blank subtraction was done early on, resulting in a few negative values. There are also several VOCs and carbonyls that are often below the LDL.

Five of the chemicals from the MPCA Staff Paper (1,3-butadiene, arsenic, chromium, ethylene dibromide and nickel) could only be briefly summarized because the majority of the monitored values from the SATMN data were below the LDL. The quality of the data, therefore, made it unfeasible to do more than a cursory analysis. Table 9 gives the percent of samples for each chemical above the LDL for all the SATMN sites combined from 1996-1999. The sites were also screened to ensure that certain sites did not have significantly higher percentages above the LDL.

Table 9: LDLs for Pollutants from the MPCA Staff Paper

Chemical Name	CAS Number	Percent Above the Lower Detection Limit	Number of Valid Samples
1,3-Butadiene	106-99-0	1%	160
Arsenic	7440-38-2	3%	687
Benzene	71-43-2	99%	1135
Carbon Tetrachloride	56-23-5	99%	1136
Chloroform	67-66-3	37%	1134
Chromium	7440-47-3	18%	873
Ethylene dibromide	106-93-4	1%	1134
Formaldehyde	50-00-0	99%	1193
Nickel	7440-02-2	2%	1137

Statistical Software

All statistical analyses were done using either SPSS version 8.0 or Microsoft Excel 97.

Data Gaps in Monitoring Analysis

During analysis of MPCA's ambient air toxics monitoring data, several gaps in the monitoring data have been determined. These gaps include: concern with the lower detection limits of some compounds, the lack of monitoring data for persistent and bioaccumulative chemicals, and the lack of trend data for non-urban locations.

Lower Detection Limits of Compounds

The MPCA found several low detection limits to be of concern during the analysis of the Minnesota ambient air monitoring data. Of over 40 chemicals analyzed that have available inhalation health benchmarks, five of these chemicals have lower detection limits that are higher than the lowest inhalation health benchmark, as shown in Table 10.

Table 10: LDLs Compared with Inhalation Health Benchmarks

Chemical Name	CAS Number	Lowest Health Benchmark (ug/m3)	Average LDLs (ug/m3) *updated through 1999	LDL Comparison to Health Benchmarks
1,3-butadiene	106-99-0	0.04	0.187	Benchmark<LDL
Arsenic	7440-38-2	0.002	0.005	Benchmark<LDL
Cadmium	7440-43-9	0.01	0.016	Benchmark<LDL
Chromium VI	7440-47-3	0.0008	0.002	Benchmark<LDL
Ethylene dibromide	106-93-4	0.05	0.271	Benchmark<LDL

In the case of 1,3-butadiene, arsenic, and chromium, this causes a particular concern because modeling concentrations from the CEP study indicate that these chemicals may require further analysis. This analysis cannot be done when the majority of the data is below the LDL.

In the case of the metal compounds, part of the problem with the LDLs stems from the energy dispersive X-ray fluorescence (XRF) equipment used for analysis. This screening technique is capable of determining the concentration of 33 elements, however, since it is a screening technique, the LDLs can be relatively high. More refined (and expensive) techniques could be used for analysis of individual metals that might allow analysis down to the level of the inhalation health benchmarks.

Lack of Information on Semi-Volatile Chemicals

A second significant data gap is the limitations on the compounds for which MPCA monitors. Currently, MPCA has ambient air monitoring data for seven carbonyl compounds, 37 volatile organic compounds (VOCs), and 33 metal elements. The MPCA does not have ambient air monitoring data for any semi-volatile organic compounds. These semi-volatile compounds are more likely to persist and bioaccumulate in the environment than VOCs or carbonyl compounds.

From a long-term perspective, these persistent and bioaccumulating toxics (PBTs) may pose a greater risk to human health and the environment than the VOCs. PBTs include compounds such as polycyclic aromatic hydrocarbons (PAHs), dioxins, furans, pesticides, polychlorinated biphenyls (PCBs), polybrominated biphenyls (PBBs), and pollutants of emerging concern (e.g., endocrine disruptors). More information on these chemicals is available in [Appendix F, Persistent Bioaccumulative Toxics](#).

The PBTs are much more complex and expensive to monitor and analyze than the chemicals which MPCA currently monitors. The MPCA has been unable thus far to monitor these chemicals due to budget and staffing considerations.

Lack of Non-Urban Trend Data

The MPCA has up to ten years of trend data for air toxics at select monitoring locations. However, all of this trend data is located in either urban areas or near specific facilities with large emissions. These sites were located in consideration of MPCA's limited monitoring budget. The monitoring took place in areas that were likely to have higher ambient air concentrations.

However, this strategy has left a gap in trend data for rural and small town areas of the state. Without trend data for rural areas of the state, it is difficult to know which compounds may be a concern statewide and which are exclusively urban or industry-specific concerns. Since nearly half of Minnesota's population lives in rural or small-town areas, this lack of trend data makes it difficult to judge their continuing exposure to air toxics.

3.3 Updated Monitoring Data for Pollutants from MPCA Staff Paper

The chemicals identified as pollutants of concern in the MPCA Staff Paper on Air Toxics were examined more closely through evaluation of the first three years of monitoring data from the statewide air toxics monitoring network (SATMN). The chemicals basically fell into three categories:

- Chemicals which could be evaluated in more depth (benzene, carbon tetrachloride, chloroform, and formaldehyde).
- Chemicals which could not be evaluated in depth due to detection limitations (ethylene dibromide, 1,3-butadiene, arsenic, nickel, chromium).
- Chemicals which are not monitored at MPCA (acrolein, polycyclic organic matter (POM)).

Five of the chemicals could only be briefly summarized because the majority of the monitored values were below the lower detection limit (LDL). The quality of the data, therefore, made it unfeasible to do more than a cursory analysis. Only compounds with a significant amount of data above the LDL was analyzed in depth.

MPCA focused on the Statewide Air Toxics Monitoring Network (SATMN) sites, a subset of all sites, for mean ambient air concentration data for the benzene, carbon tetrachloride, chloroform, and formaldehyde updates. This was done so that a similar time frame would be compared between sites (all the SATMN sites have one year of data). In addition, the SATMN sites are located away from immediate pollution sources, so bias from near-by point sources was removed. Finally, the SATMN sites are organized under a single study and allow for comparisons with fewer caveats. The SATMN data analyzed in this update were collected from 1996 to 1999.

Benzene Update

Conclusions from MPCA Staff Paper

Monitoring data and modeling studies showed that benzene concentrations in Minnesota were elevated above the lower bound of the inhalation health benchmark ($1.3 \mu\text{g}/\text{m}^3$) in the Twin Cities metropolitan area and in other smaller population centers in the state (e.g., Duluth, St. Cloud, Rochester, Mankato). Since 1991, it appeared that benzene concentrations in the metropolitan area had decreased slightly. The reason for the decrease was unclear. Given the magnitude of the measured concentrations, especially in the metropolitan area and other smaller population centers, the MPCA Staff Paper concluded that benzene in the air presented potential health problems in Minnesota.

Update on benzene ambient concentrations

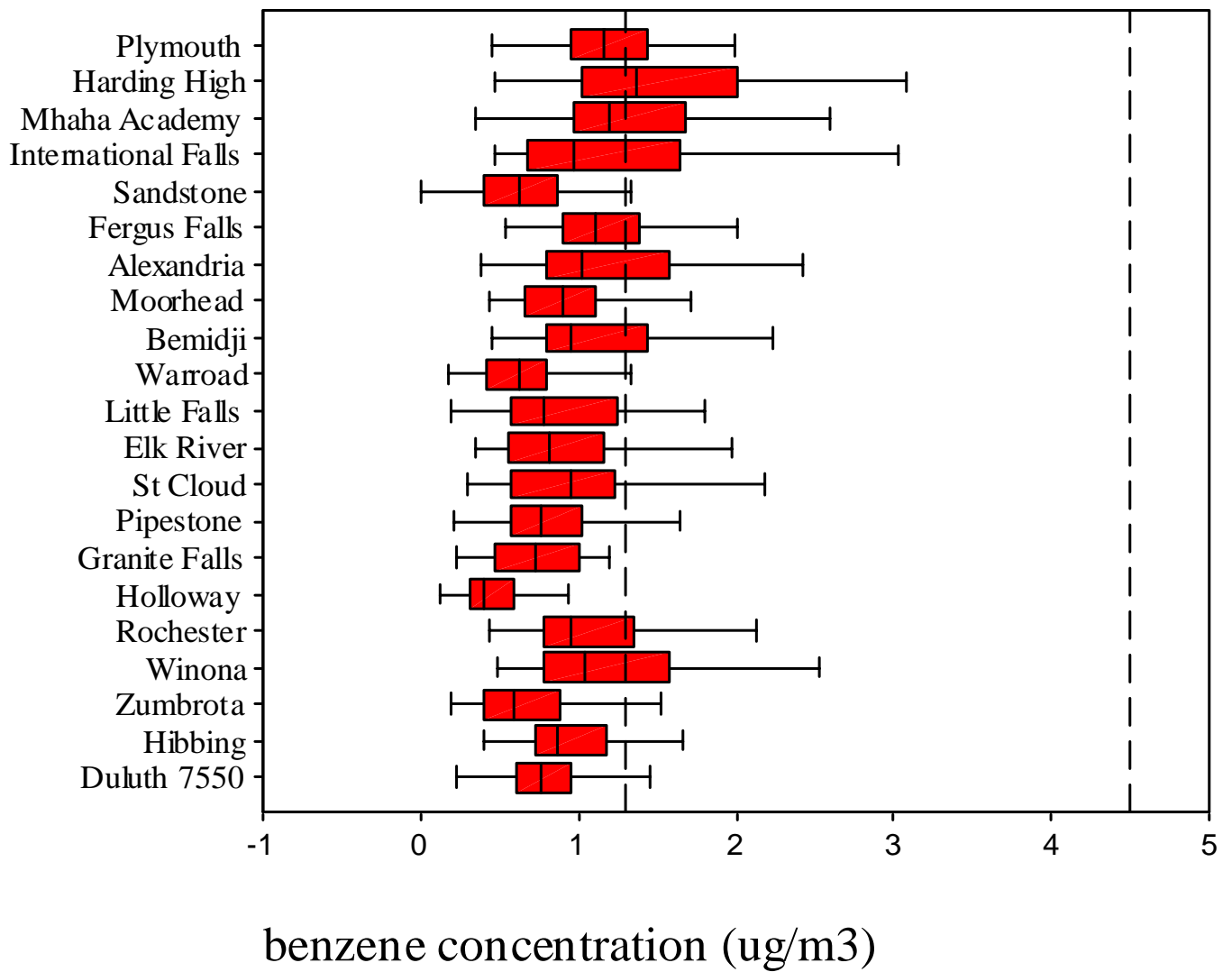
Mean benzene concentrations ranged from $1.70\text{-}0.47 \mu\text{g}/\text{m}^3$ between sites. Median benzene concentrations ranged from $1.38\text{-}0.41 \mu\text{g}/\text{m}^3$. The inhalation health benchmark for benzene is based on the MDH's proposed health risk value (HRV) and which is a range from $1.3\text{-}4.5 \mu\text{g}/\text{m}^3$. The mean values at several urban sites exceeded the lower range of the cancer inhalation health benchmark ($1.3 \mu\text{g}/\text{m}^3$). Only the median value for Harding High (St. Paul, MN) exceeded the lower range of the inhalation health benchmark for cancer. Excluding outliers and extremes, none of the data exceeded the upper range of the inhalation health benchmark. There was an indication that the highest levels were found in urban areas, while lower concentrations were found in small town and rural locations. Table 11 shows monitored benzene concentrations.

Table 11: Benzene Concentrations for Statewide Monitoring Network (1996-1999)

Site Name	Site No.	Year Collected	N	Mean	Median	Standard Deviation
Plymouth	260	1996-97	55	1.31	1.16	0.74
Harding High	871	1998-99	56	1.70	1.38	1.11
Minnehaha Academy	958	1997-98	57	1.44	1.20	0.76
International Falls 1241	1241	1996-97	57	1.37	0.98	1.15
Sandstone	1400	1996-97	52	0.67	0.63	0.34
Fergus Falls	2005	1997-98	48	1.18	1.10	0.45
Alexandria	2010	1996-97	58	1.22	1.02	0.57
Moorhead	2103	1998-99	52	0.98	0.90	0.47
Bemidji	2302	1998-99	56	1.23	0.95	0.77
Warroad	2401	1997-98	47	0.64	0.62	0.32
Little Falls	3049	1996-97	56	0.90	0.78	0.45
Elk River	3050	1997-98	58	0.95	0.82	0.54
St. Cloud	3052	1998-99	53	1.10	0.96	0.78
Pipestone	4002	1996-97	47	0.82	0.76	0.35
Granite Falls	4003	1997-98	45	0.93	0.73	0.99
Holloway	4500	1998-99	55	0.47	0.41	0.22
Rochester	5008	1997-98	59	1.11	0.95	0.46
Winona	5210	1998-99	51	1.36	1.04	1.04
Zumbrota	5356	1996-97	54	0.65	0.59	0.30
Hibbing	7014	1997-98	59	1.01	0.87	0.51
Duluth 7550	7550	1998-99	60	0.86	0.76	0.47
1996-97	--	1996-97	379	1.00	0.86	0.69
1997-98	--	1997-98	373	1.05	0.91	0.64
1998-99	--	1998-99	383	1.10	0.86	0.83
All Sites	--	1996-99	1135	1.05	0.87	0.73

Figure 4 includes SATMN data collected from 1996-1999. The center line within each box represents the median for the site. The box itself encompasses the 25th percentile to the 75th percentile. The bars at each end of the box represent the highest and lowest values that are not considered outliers. The vertical dotted lines are located at the values of the benzene inhalation health benchmark range (1.3-4.5 $\mu\text{g}/\text{m}^3$).

Figure 4: Benzene Concentrations by Site



When the SATMN five-year study is completed, some conclusions regarding geographic distribution of concentrations may be possible. Since this update only looks at three years of data, it is difficult to make any conclusions regarding geographic or urban/rural differences. However, as Table 12 indicates, there was evidence that the highest levels were found in urban areas, while lower concentrations were found in small town and rural locations.

Table 12: Benzene Concentrations by Range

Mean Range (1.70-1.31 ug/m3)			Mean Range (1.23-0.98 ug/m3)			Mean Range (0.93-0.47 ug/m3)		
Median Range (1.38-0.98 ug/m3)			Median Range (1.10-0.95 ug/m3)			Median Range (0.78-0.41 ug/m3)		
Site	Year	Site Type	Site	Year	Site Type	Site	Year	Site Type
Harding High	1998-99	SATMN-U	Fergus Falls	1997-98	SATMN-S	Sandstone	1996-97	SATMN-R
Plymouth	1996-97	SATMN-U	Alexandria	1996-97	SATMN-S	Holloway	1998-99	SATMN-R
Mhaha Academy	1997-98	SATMN-U	Moorhead	1998-99	SATMN-U	Warroad	1997-98	SATMN-S
I Falls	1996-97	SATMN-S	Bemidji	1998-99	SATMN-S	Little Falls	1996-97	SATMN-S
Winona	1998-99	SATMN-U	Elk River	1997-98	SATMN-S	Pipestone	1996-97	SATMN-S
			St Cloud	1998-99	SATMN-U	Granite Falls	1997-98	SATMN-S
			Rochester	1997-98	SATMN-U	Zumbrota	1996-97	SATMN-R
			Hibbing	1997-98	SATMN-S	Duluth	1998-99	SATMN-U

*In most cases, the high range is statistically different from the low range. The middle range is not statistically different from either the high or the low ranges.

Update on benzene trends

For benzene trend analysis, five sites in urban areas such as the Twin Cities and Duluth were analyzed. These monitoring sites have concentrations dating back to 1991 which allowed for long-term trend analysis.

As in the MPCA Staff Paper, there continued to be a statistically significant decrease in benzene concentrations in the metropolitan area and in Duluth. The regression coefficients (R^2 values) ranged from 0.042 at the Koch 420 site to 0.115 in Duluth. These relatively low regression coefficients indicate that the change in time accounts for only a small part of the variation in benzene concentration data. The regression equations show that the benzene concentrations have been decreasing by $0.14 \mu\text{g}/\text{m}^3$ per year at Minneapolis public library, $0.12 \mu\text{g}/\text{m}^3$ per year at Koch 420, $0.28 \mu\text{g}/\text{m}^3$ per year at St. Paul Park, $0.10 \mu\text{g}/\text{m}^3$ per year at Holman Field, and $0.19 \mu\text{g}/\text{m}^3$ per year in Duluth.

Benzene concentrations were found to be decreasing by $0.02 \mu\text{g}/\text{m}^3$ per year in the MPCA Staff Paper. The updated benzene concentration data indicates that benzene concentrations may be decreasing at a higher rate than previously found. It will take several years of monitoring to determine the robustness of the downward trend. Concentrations at some sites (especially the inner city sites) are still above the lower bound of the inhalation health benchmark. In addition, benzene is typically emitted from sources in close proximity to where people are breathing.

Figure 5 is a scatterplot of benzene concentrations is plotted with a smoothed trend line. The horizontal dashed lines are at the bounds of the inhalation health benchmark range for benzene ($1.3-4.5 \mu\text{g}/\text{m}^3$).

Figure 5: Trend in Benzene Measurements at the Minneapolis Library

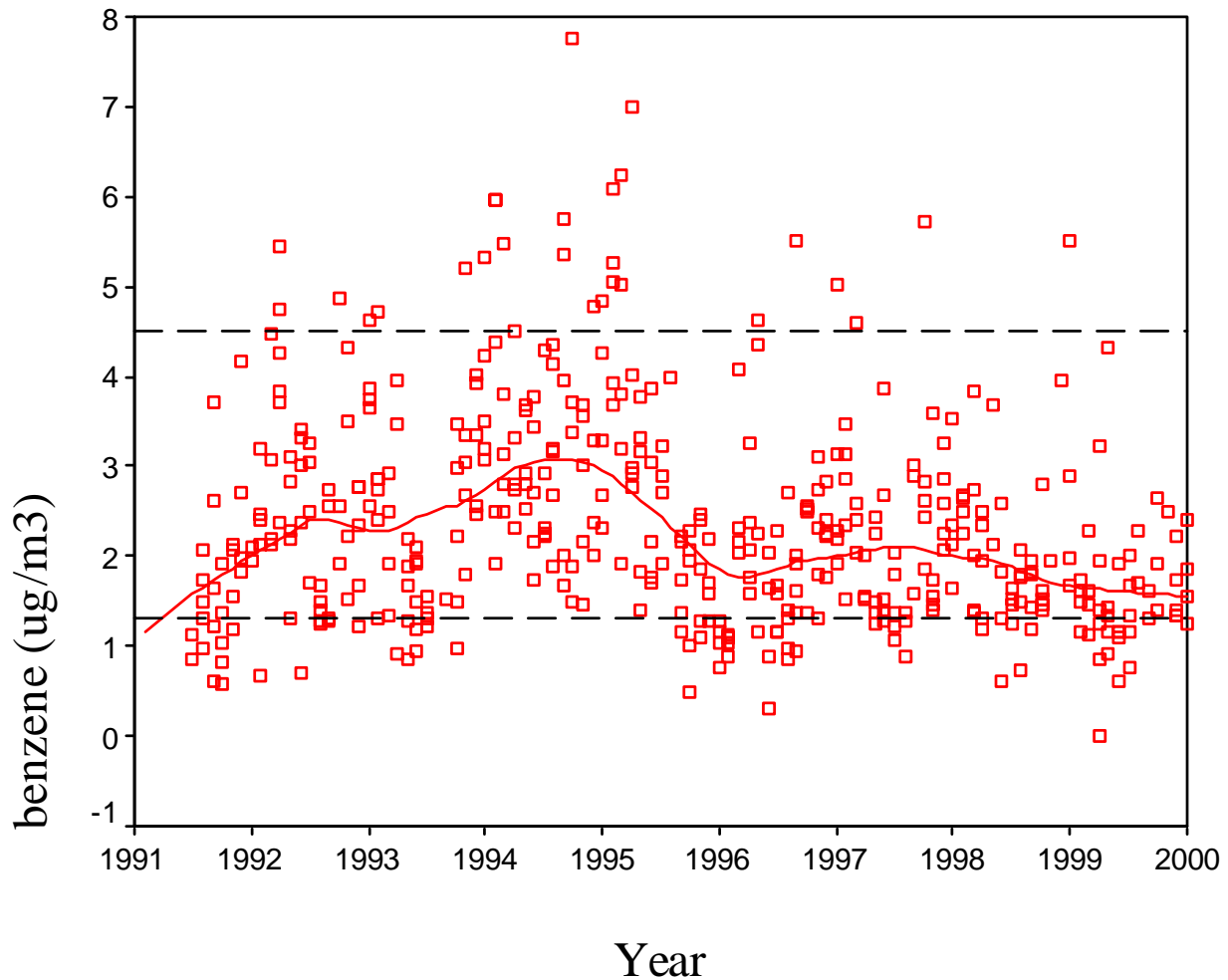
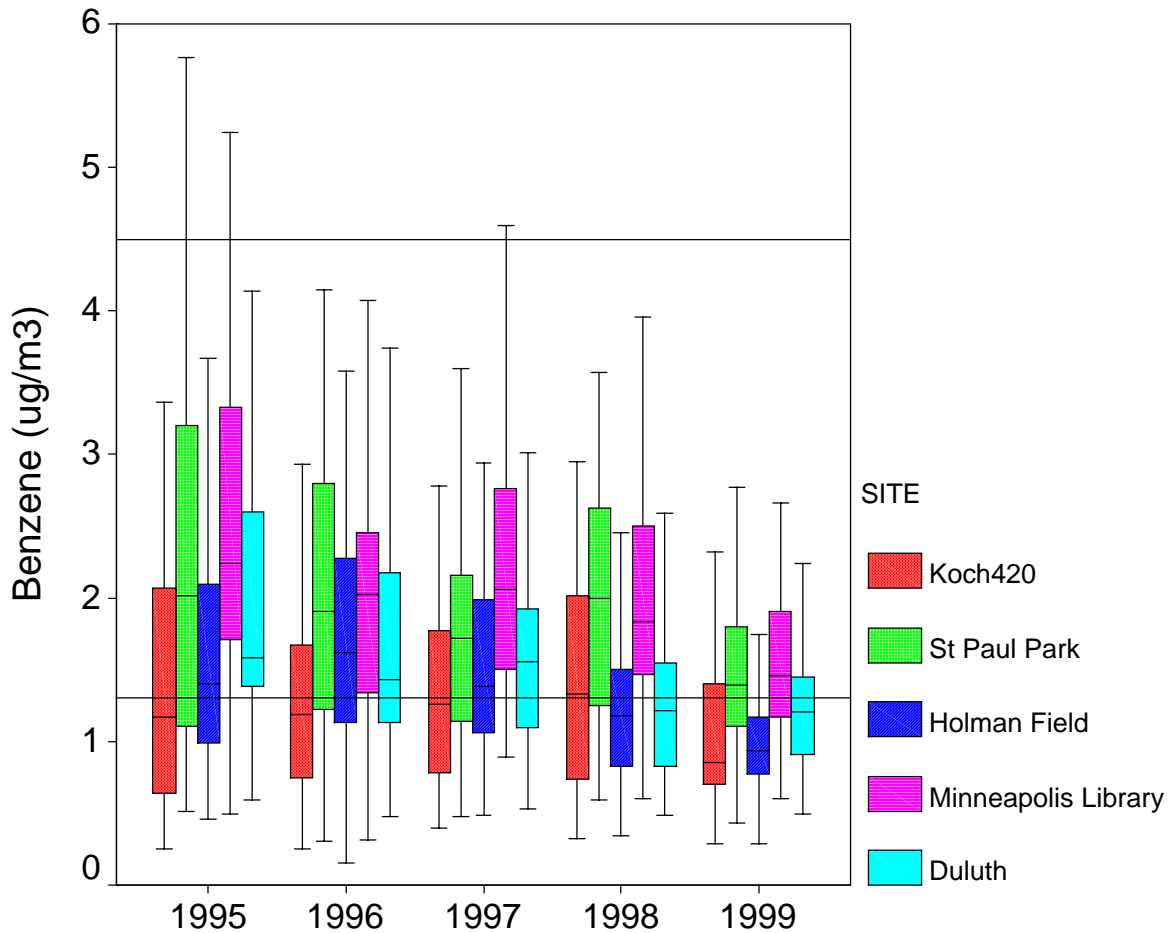


Figure 6 shows the concentrations of benzene at five sites in Minnesota. The center line within each box represents the median for the site. The box itself encompasses the 25th percentile to the 75th percentile. The bars at each end of the box represent the highest and lowest values that are not considered outliers. The vertical dotted lines are located at the inhalation health benchmark range for benzene ($1.3-4.5 \mu\text{g}/\text{m}^3$).

Figure 6. Benzene Concentrations at Several Long Term Monitoring Sites



According to the CEP final report (SAI, 1999), natural background levels of benzene in 1985 were 0.48 ug/m³.

Carbon Tetrachloride Update

Conclusions from MPCA Staff Paper

Monitoring data from the MPCA Staff Paper showed that carbon tetrachloride exceeded the inhalation health benchmark of 0.7 ug/m³ throughout Minnesota. MDH did not find adequate information to develop a health risk value (HRV) for carbon tetrachloride, so this inhalation health benchmark was developed based on cancer potency information from the EPA IRIS database. Carbon tetrachloride has been banned internationally under the Montreal Protocol treaty, which limits

production and emission of substances that destroy the stratospheric ozone layer. Despite the ban and the end of U.S. production in 1996, the monitoring data did not yet show a clear trend toward decreasing concentrations. The high measured concentrations suggested a potentially important public health issue from carbon tetrachloride in the atmosphere. Therefore, carbon tetrachloride was listed as a pollutant of concern in the MPCA Staff Paper.

Update on carbon tetrachloride ambient concentrations

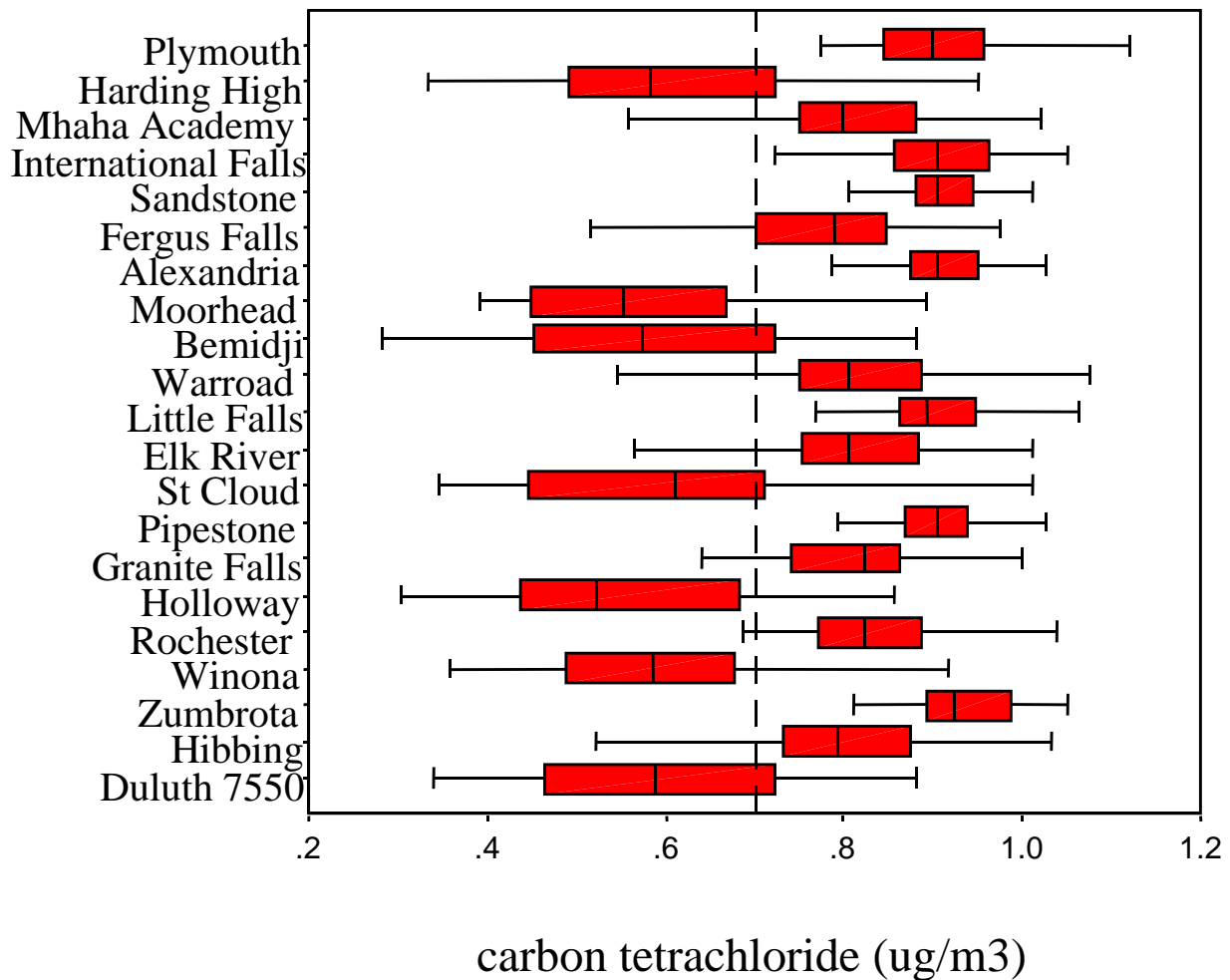
Table 13 lists carbon tetrachloride concentrations at SATMN monitoring sites. Mean carbon tetrachloride concentrations ranged from 0.93-0.54 ug/m³ between sites. Median carbon tetrachloride concentrations ranged from 0.92-0.52 ug/m³ between sites.

Table 13: Carbon Tetrachloride Concentrations for Statewide Monitoring Network (1996-9)

<u>Site Name</u>	<u>Site No.</u>	<u>Year Collected</u>	<u>N</u>	<u>Mean</u>	<u>Median</u>	<u>Standard Deviation</u>
Plymouth	260	1996-97	55	0.91	0.91	0.095
Harding High	871	1998-99	56	0.60	0.58	0.15
Minnehaha Academy	958	1997-98	57	0.80	0.80	0.12
International Falls 1241	1241	1996-97	57	0.91	0.91	0.077
Sandstone	1400	1996-97	51	0.91	0.91	0.061
Fergus Falls	2005	1997-98	48	0.78	0.79	0.13
Alexandria	2010	1996-97	58	0.92	0.91	0.088
Moorhead	2103	1998-99	52	0.57	0.55	0.17
Bemidji	2302	1998-99	56	0.57	0.57	0.17
Warroad	2401	1997-98	47	0.82	0.81	0.11
Little Falls	3049	1996-97	56	0.91	0.89	0.072
Elk River	3050	1997-98	59	0.79	0.81	0.16
St. Cloud	3052	1998-99	53	0.60	0.61	0.15
Pipestone	4002	1996-97	47	0.92	0.91	0.084
Granite Falls	4003	1997-98	46	0.77	0.82	0.19
Holloway	4500	1998-99	55	0.54	0.52	0.19
Rochester	5008	1997-98	59	0.81	0.82	0.12
Winona	5210	1998-99	51	0.60	0.59	0.13
Zumbrota	5356	1996-97	54	0.93	0.92	0.061
Hibbing	7014	1997-98	59	0.79	0.79	0.13
Duluth 7550	7550	1998-99	60	0.59	0.59	0.14
1996-97	--	1996-97	378	0.92	0.91	0.078
1997-98	--	1997-98	375	0.80	0.81	0.14
1998-99	--	1998-99	383	0.58	0.58	0.16
All Sites	--	1996-99	1136	0.76	0.81	0.19

Figure 7 includes SATMN data collected from 1996-1999. The center line within each box represents the median for the site. The box itself encompasses the 25th percentile to the 75th percentile. The bars at each end of the box represent the highest and lowest values that are not considered outliers. The vertical dotted line is located at the carbon tetrachloride inhalation health benchmark ($0.7 \mu\text{g}/\text{m}^3$).

Figure 7: Carbon Tetrachloride Concentrations by Site



When the SATMN five-year study is completed, some conclusions regarding geographic distribution of concentrations may be possible. Since this update only looks at three years of data, it is difficult to make any conclusions regarding geographic or urban/rural differences. However, as the tables below indicate, there does not appear to be geographic differences in carbon tetrachloride concentrations. Apparently the differences seen between sites is due to the monitoring year, not the site location. Carbon tetrachloride appears to be declining in concentration with time. The trends are shown in Table 14.

Table 14: Carbon Tetrachloride Concentrations by Range

Mean Range (0.93-0.91ug/m3)			Mean Range (0.82-0.77ug/m3)			Mean Range (0.60-0.54ug/m3)		
Median Range (0.92-0.89ug/m3)			Median Range (0.82-0.79ug/m3)			Median Range (0.61-0.52ug/m3)		
Site	Year	Site Type	Site	Year	Site Type	Site	Year	Site Type
Plymouth	1996-97	SATMN-U	Mhaha Academy	1997-98	SATMN-U	Harding High	1998-99	SATMN-U
I Falls	1996-97	SATMN-S	Fergus Falls	1997-98	SATMN-S	Moorhead	1998-99	SATMN-U
Sandstone	1996-97	SATMN-R	Warroad	1997-98	SATMN-S	Bemidji	1998-99	SATMN-S
Alexandria	1996-97	SATMN-S	Elk River	1997-98	SATMN-S	St Cloud	1998-99	SATMN-U
Little Falls	1996-97	SATMN-S	Granite Falls	1997-98	SATMN-S	Holloway	1998-99	SATMN-R
Pipestone	1996-97	SATMN-S	Rochester	1997-98	SATMN-U	Winona	1998-99	SATMN-U
Zumbrota	1996-97	SATMN-R	Hibbing	1997-98	SATMN-S	Duluth	1998-99	SATMN-U

*In most cases, the three ranges are statistically different from one another. The three monitoring years are statistically different from one another.

Update on carbon tetrachloride trends

Carbon tetrachloride appears to vary less by geographic location than by time. Although the statewide monitoring network is not designed for trend data, all of the site results indicate that the concentrations of carbon tetrachloride have trended downward each year. Table 15 includes all of the SATMN data through the 1999 sampling year.

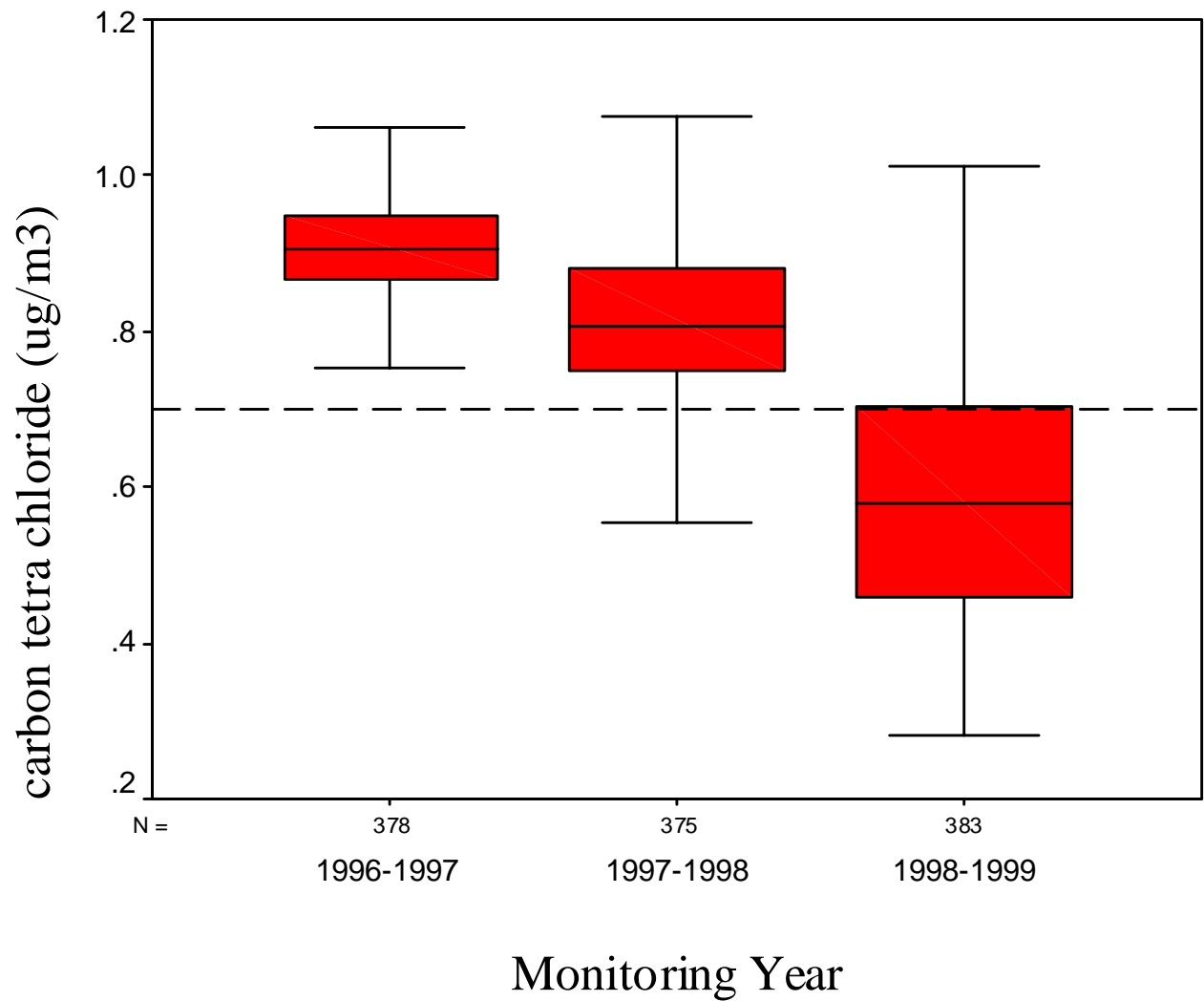
Table 15: Carbon Tetrachloride Concentrations by Monitoring Year

Monitoring Year	Mean Range (ug/m ³)	Median Range (ug/m ³)
1996-97	0.93-0.91	0.92-0.89
1997-98	0.82-0.77	0.82-0.79
1998-99	0.60-0.54	0.61-0.52

The differences between the years are statistically significant.

Figure 8 includes all of the SATMN data collected from 1996-1999. The center line within each box represents the median for the site. The box itself encompasses the 25th percentile to the 75th percentile. The bars at each end of the box represent the highest and lowest values that are not considered outliers. The horizontal dotted line is located at the carbon tetrachloride inhalation health benchmark (0.7 ug/m³).

Figure 8: Carbon Tetrachloride Concentrations by Monitoring Year

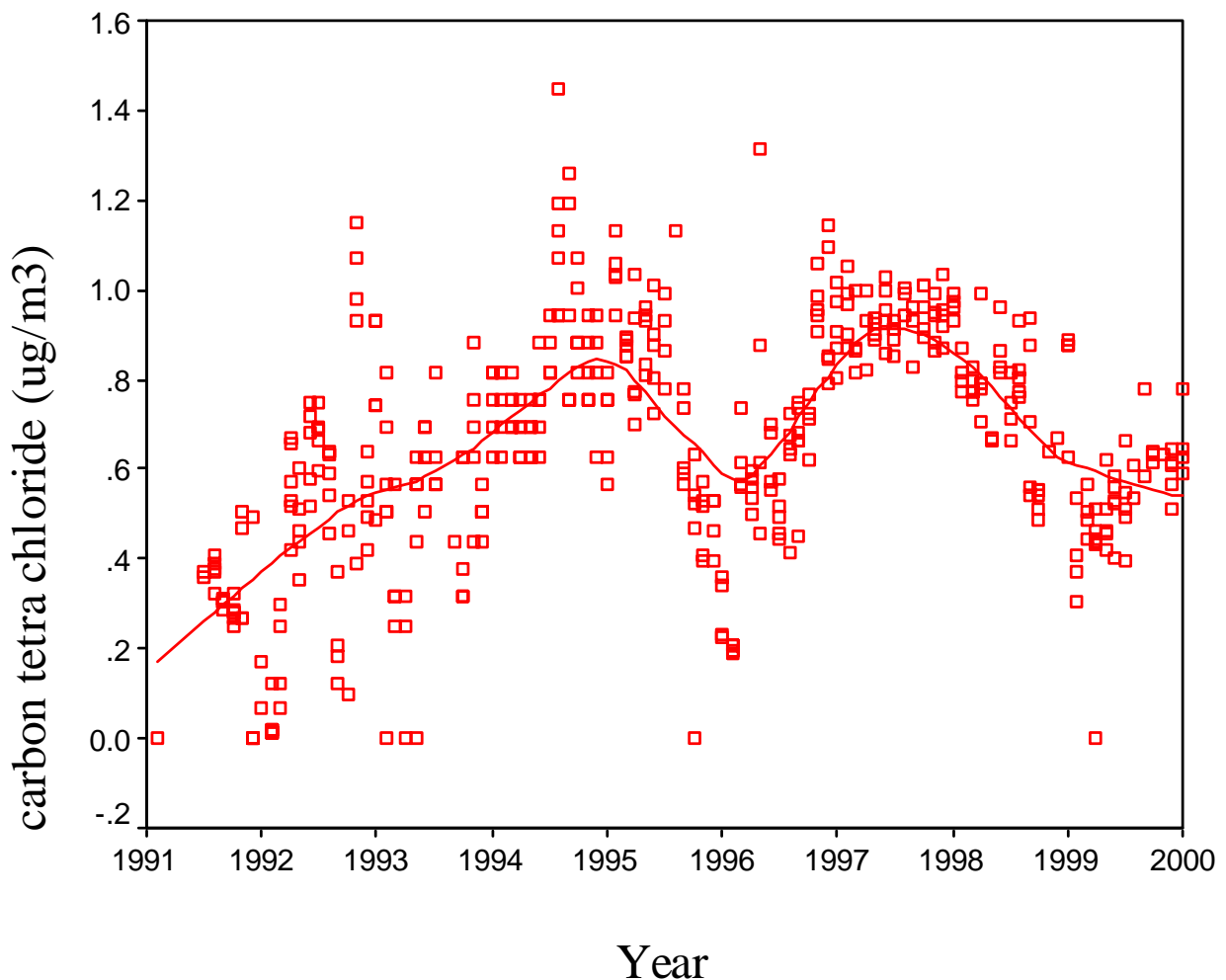


Five sites in urban areas such as the Twin Cities and Duluth were also considered for carbon tetrachloride trend analysis. These monitoring sites have concentrations dating back to 1991 which allowed for long-term trend analysis.

Updated data indicates that concentrations of carbon tetrachloride have been decreasing since 1997. The downward trend of carbon tetrachloride values indicates that average levels in Minnesota are now below the inhalation health benchmark of $0.7 \mu\text{g}/\text{m}^3$. This coincides well with the banning of U.S. production of carbon tetrachloride in 1996 due to the Montreal Protocol.

Figure 9 shows carbon tetrachloride concentrations plotted with a smoothed trend line. The horizontal dashed lines are at the bounds of the inhalation health benchmark range for carbon tetrachloride ($0.7 \mu\text{g}/\text{m}^3$).

Figure 9: Trend in Carbon Tetrachloride Measurements at Minneapolis Library



According to the CEP final report (SAI, 1999), anthropogenic (human-made) background levels of carbon tetrachloride in 1990 were 0.88 ug/m³.

Chloroform Update

Conclusions from MPCA Staff Paper

Ambient air monitoring data showed that chloroform concentrations were below the inhalation health benchmark of 0.4 ug/m³ used in the MPCA Staff Paper at all sites in Minnesota except one. MDH did not find adequate information to develop a cancer-based chronic health risk value (HRV) for chloroform, so this inhalation health benchmark was developed based on cancer potency information from the EPA IRIS database. The mean and median chloroform concentrations at the customs station site in International Falls exceeded the inhalation health benchmark. This site is adjacent to the Boise Cascade paper mill and across the river from the Stone Consolidated paper mill in Fort Francis, Ontario. It appears that emissions from one or both of these facilities caused the elevated chloroform concentrations at the customs station monitoring site. The chloroform concentrations at a second International Falls monitoring site about one mile southwest of the customs station was below the inhalation health benchmark value.

Update on chloroform ambient concentrations and toxicity assessment

Mean chloroform concentrations for the SATMN sites ranged from 0.17-0.03 ug/m³ between sites. Median chloroform concentrations ranged from 0.14-0.000 ug/m³ between sites. Recently MDH developed a policy recommending a inhalation health benchmark concentration of 100 ug/m³ for chloroform based on developmental effects, which MPCA will use for this report. None of the data (excluding outliers and extremes) was above the previous cancer inhalation health benchmark of 0.4 ug/m³ or the revised inhalation health benchmark of 100 ug/m³

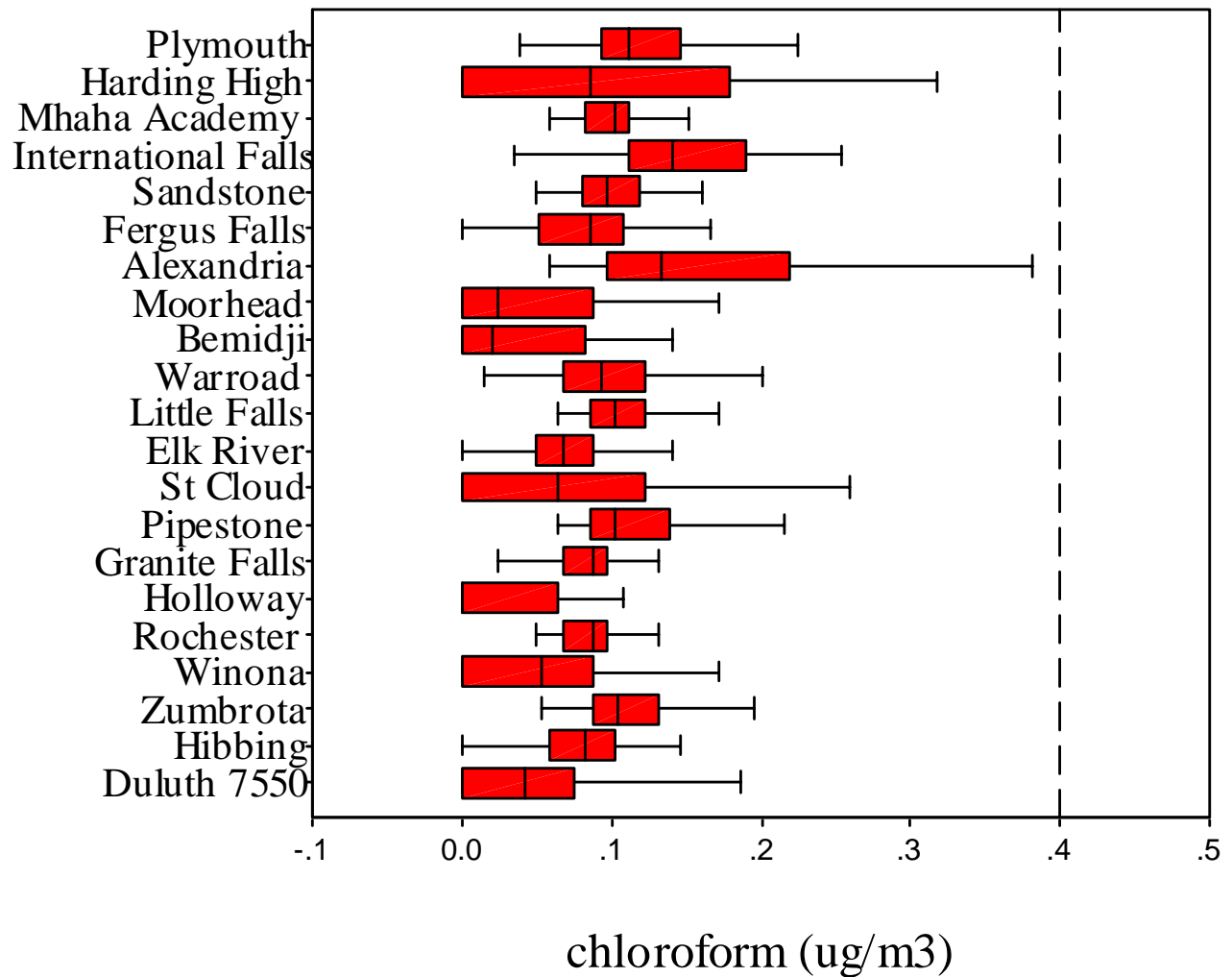
The SATMN sites do not include the International Falls monitoring location that had concentrations exceeding the previous cancer inhalation health benchmark. The mean chloroform concentration in 1998 at the non-SATMN International Falls location was 1.28 ug/m³ and the median chloroform concentration was 0.88 ug/m³. Chloroform was measured July-Nov 1998, so it is not a complete year of data. This high concentration is expected to be from a point source and not indicative of a regional ambient air concern.

Table 16: Chloroform Concentrations for Statewide Monitoring Network (1996-1999)

<u>Site Name</u>	Site No.	Year Collected	N	Mean	Median	Standard Deviation
Plymouth	260	1996-97	55	0.13	0.11	0.070
Harding High	871	1998-99	56	0.099	0.085	0.094
Minnehaha Academy	958	1997-98	57	0.11	0.10	0.051
International Falls 1241	1241	1996-97	57	0.15	0.14	0.068
Sandstone	1400	1996-97	51	0.10	0.098	0.036
Fergus Falls	2005	1997-98	48	0.085	0.085	0.051
Alexandria	2010	1996-97	58	0.17	0.13	0.096
Moorhead	2103	1998-99	52	0.046	0.024	0.051
Bemidji	2302	1998-99	56	0.043	0.020	0.047
Warroad	2401	1997-98	47	0.10	0.093	0.056
Little Falls	3049	1996-97	56	0.11	0.10	0.038
Elk River	3050	1997-98	58	0.073	0.068	0.040
St. Cloud	3052	1998-99	53	0.071	0.063	0.064
Pipestone	4002	1996-97	47	0.13	0.10	0.066
Granite Falls	4003	1997-98	45	0.084	0.088	0.021
Holloway	4500	1998-99	55	0.030	0.000	0.037
Rochester	5008	1997-98	59	0.089	0.088	0.040
Winona	5210	1998-99	51	0.049	0.054	0.049
Zumbrota	5356	1996-97	54	0.11	0.11	0.032
Hibbing	7014	1997-98	59	0.082	0.083	0.040
Duluth 7550	7550	1998-99	60	0.044	0.041	0.047
1996-97	--	1996-97	378	0.13	0.11	0.066
1997-98	--	1997-98	373	0.088	0.088	0.045
1998-99	--	1998-99	383	0.054	0.049	0.062
All Sites	--	1996-99	1134	0.090	0.088	0.066

Figure 10 includes SATMN data collected from 1996-1999. The center line within each box represents the median for the site. The box itself encompasses the 25th percentile to the 75th percentile. The bars at each end of the box represent the highest and lowest values that are not considered outliers. The vertical dotted line is located at the previous chloroform inhalation health benchmark (0.4 µg/m³).

Figure 10. Chloroform Concentrations by Monitoring Site



When the SATMN five-year study is completed, some conclusions regarding geographic distribution of concentrations may be possible. Since this update only looks at three years of data, it is difficult to make any conclusions regarding geographic or urban/rural differences. However, ANOVA statistical analysis did not show a geographical trend or urban/rural trend.

The data indicates that chloroform levels may be decreasing with time since the highest median and mean levels are all from 1996-97 while the lowest are all from the 1998-99 monitoring year.

Table 17: Chloroform Concentration by Range

Mean Range (0.17-0.13 ug/m3)			Mean Range (0.11-0.071 ug/m3)			Mean Range (0.049-0.03 ug/m3)		
Median Range (0.14-0.10 ug/m3)			Median Range (0.11-0.063 ug/m3)			Median Range (0.054-0.000 ug/m3)		
Site	Year	Site Type	Site	Year	Site Type	Site	Year	Site Type
I Falls	1996-97	SATMN-S	Fergus Falls	1997-98	SATMN-S	Holloway	1998-99	SATMN-R
Plymouth	1996-97	SATMN-U	Sandstone	1996-97	SATMN-R	Moorhead	1998-99	SATMN-U
Alexandria	1996-97	SATMN-S	Warroad	1997-98	SATMN-S	Bemidji	1998-99	SATMN-S
Pipestone	1996-97	SATMN-S	Little Falls	1996-97	SATMN-S	Winona	1998-99	SATMN-U
			Granite Falls	1997-98	SATMN-S	Duluth	1998-99	SATMN-U
			Elk River	1997-98	SATMN-S			
			St Cloud	1998-99	SATMN-U			
			Mhaha Academy	1997-98	SATMN-U			
			Harding High	1998-99	SATMN-U			
			Zumbrota	1996-97	SATMN-R			
			Rochester	1997-98	SATMN-U			
			Hibbing	1997-98	SATMN-S			

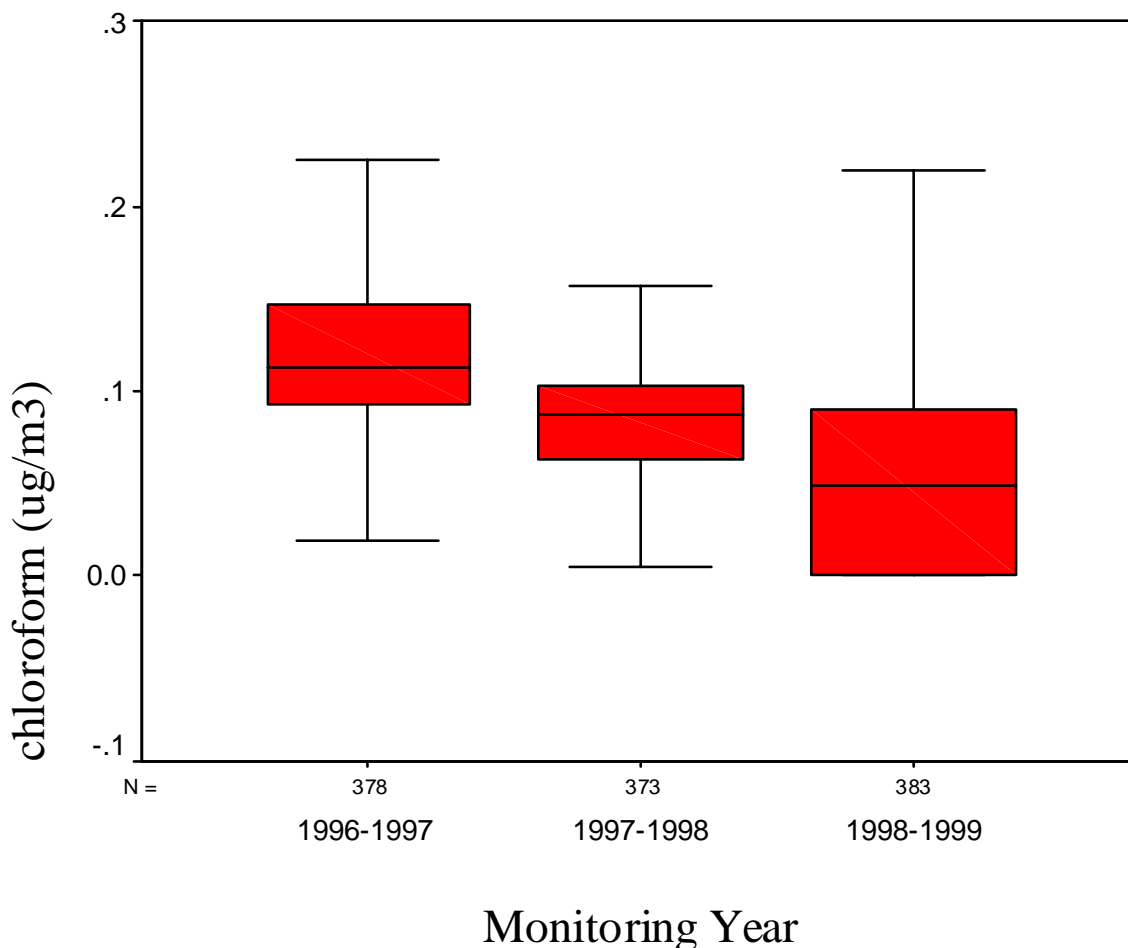
*In most cases, the high range is statistically different from the low range. The middle range is not necessarily statistically different from either the high or the low ranges. The chloroform ranges seem somewhat correlated with the monitoring year.

Update on chloroform trends

The SATMN data indicates that chloroform concentrations are decreasing over the last three monitoring years (1996-1999). However, the data from the SATMN is not definitive since the network was not designed to analyze time trends. The differences between years are statistically significant according to ANOVA analysis. All of the data is below the inhalation health benchmark of 100 µg/m³.

Figure 11 includes all of the SATMN data collected from 1996-1999. The center line within each box represents the median for the site. The box itself encompasses the 25th percentile to the 75th percentile. The bars at each end of the box represent the highest and lowest values that are not considered outliers. The chloroform inhalation health benchmark (100 µg/m³) is not shown.

Figure 11: Chloroform Concentrations by Monitoring Year



According to the CEP final report (SAI, 1999), natural background levels of chloroform in 1990 were 0.083 $\mu\text{g}/\text{m}^3$.

Formaldehyde Update

Conclusions from MPCA Staff Paper

Statewide air monitoring data from 1991-98 showed that the mean ambient air concentrations of formaldehyde at 25 sites in Minnesota were above the inhalation health benchmark of 0.8 $\mu\text{g}/\text{m}^3$. This inhalation health benchmark is based on the MDH proposed health risk value (HRV), which was derived from the EPA IRIS database. The highest values were observed at the sites in and near the Twin Cities metropolitan area.

Formaldehyde concentrations appeared to be stable from 1995-98. The widespread exceedances of inhalation health benchmarks for formaldehyde in ambient air suggested potential concerns about human health risks. Therefore, the MPCA Staff Paper listed formaldehyde as a pollutant of concern for which current information warranted action by MPCA.

Update on formaldehyde ambient concentrations

Mean formaldehyde concentrations ranged from 2.48-0.85 ug/m³ between sites. Median formaldehyde concentrations ranged from 2.05-0.85 ug/m³ between sites. All of the mean and median values at the SATMN sites exceeded the cancer inhalation health benchmark of 0.8 ug/m³. The availability of fairly recent toxicity information relating to humans may result in this benchmark being somewhat increased in the future. However, all mean and median values were below the non-cancer benchmark of 3 ug/m³.

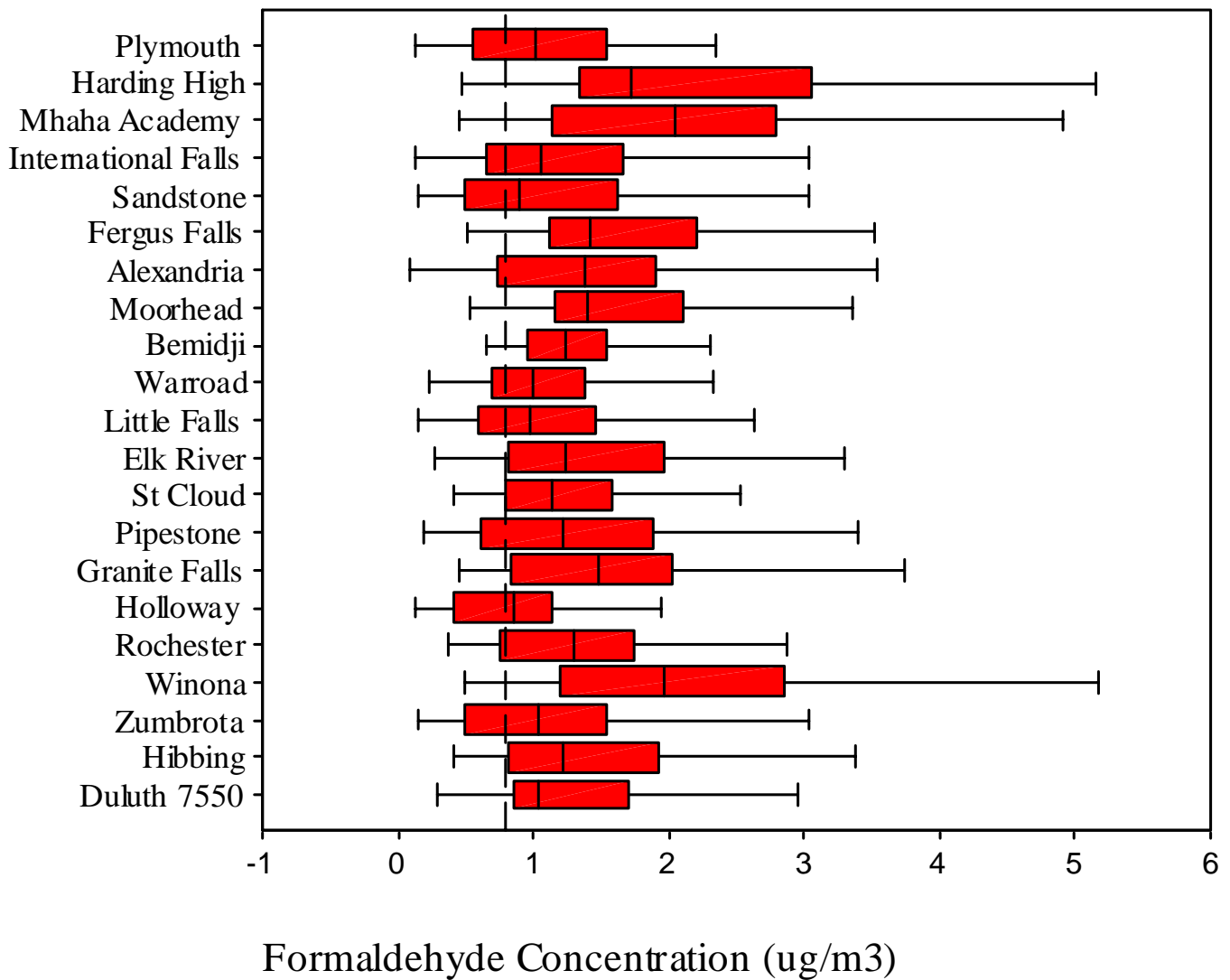
Table 18: Formaldehyde Concentrations for Statewide Monitoring Network (1996-1999)

<u>Site Name</u>	Site No.	Year Collected	N	Mean	Median	Standard Deviation
Plymouth	260	1996-97	50	1.24	1.01	0.97
Harding High	871	1998-99	60	2.23	1.73	1.44
Minnehaha Academy	958	1997-98	56	2.48	2.05	1.96
International Falls 1241	1241	1996-97	57	1.28	1.05	0.85
Sandstone	1400	1996-97	48	1.17	0.89	0.87
Fergus Falls	2005	1997-98	59	1.66	1.42	0.80
Alexandria	2010	1996-97	56	1.42	1.38	0.87
Moorhead	2103	1998-99	58	1.70	1.40	0.90
Bemidji	2302	1998-99	55	1.38	1.25	0.56
Warroad	2401	1997-98	59	1.22	1.00	0.88
Little Falls	3049	1996-97	58	1.11	0.98	0.69
Elk River	3050	1997-98	61	1.43	1.23	0.83
St. Cloud	3052	1998-99	57	1.49	1.13	1.67
Pipestone	4002	1996-97	55	1.26	1.21	0.83
Granite Falls	4003	1997-98	53	1.98	1.47	2.74
Holloway	4500	1998-99	57	0.85	0.85	0.52
Rochester	5008	1997-98	60	1.36	1.30	0.67
Winona	5210	1998-99	59	2.16	1.97	1.22
Zumbrota	5356	1996-97	53	1.16	1.04	0.78
Hibbing	7014	1997-98	61	1.57	1.22	1.03
Duluth 7550	7550	1998-99	61	1.40	1.05	0.96
1996-97	--	1996-97	377	1.24	1.04	0.84
1997-98	--	1997-98	409	1.66	1.34	1.47
1998-99	--	1998-99	407	1.61	1.32	1.20
All Sites	--	1996-99	1193	1.51	1.24	1.25

Figure 12 includes SATMN data collected from 1996-1999. The center line within each box represents the median for the site. The box itself encompasses the 25th percentile to the

75th percentile. The bars at each end of the box represent the highest and lowest values that are not considered outliers. The vertical dotted line is located at the formaldehyde inhalation health benchmark (0.8 $\mu\text{g}/\text{m}^3$).

Figure 12: Formaldehyde Concentrations by Monitoring Site



When the SATMN five year study is completed, some conclusions regarding geographic distribution of concentrations may be possible. Since this update only looks at three years of data, it is difficult to make any conclusions regarding geographic or urban/rural differences. However, as Tables 19 and 20 indicate, urban sites tended to have higher concentrations of formaldehyde than small town or rural sites, although this observation was not entirely consistent.

Table 19: High Range of Formaldehyde Concentrations

Mean Range (2.48-2.16 ug/m3)		
Median Range (2.05-1.73 ug/m3)		
Site	Year	Site Type
Harding High	1998-99	SATMN-U
Mhaha Academy	1997-98	SATMN-U
Winona	1998-99	SATMN-U

Table 20: Low Range of Formaldehyde Concentrations

Mean Range (1.98-0.85 ug/m3)		
Median Range (1.47-0.85 ug/m3)		
Site	Year	Site Type
Plymouth	1996-97	SATMN-U
International Falls 1241	1996-97	SATMN-S
Sandstone	1996-97	SATMN-R
Fergus Falls	1997-98	SATMN-S
Alexandria	1996-97	SATMN-S
Moorhead	1998-99	SATMN-U
Bemidji	1998-99	SATMN-S
Warroad	1997-98	SATMN-S
Little Falls	1996-97	SATMN-S
Elk River	1997-98	SATMN-S
St. Cloud	1998-99	SATMN-U
Pipestone	1996-97	SATMN-S
Granite Falls	1997-98	SATMN-S
Holloway	1998-99	SATMN-R
Rochester	1997-98	SATMN-U
Zumbrota	1996-97	SATMN-R
Hibbing	1997-98	SATMN-S
Duluth 7550	1998-99	SATMN-U

*In most cases, the high range is statistically different from the low range. Both ranges are higher than the inhalation health benchmark.

Update on formaldehyde trends

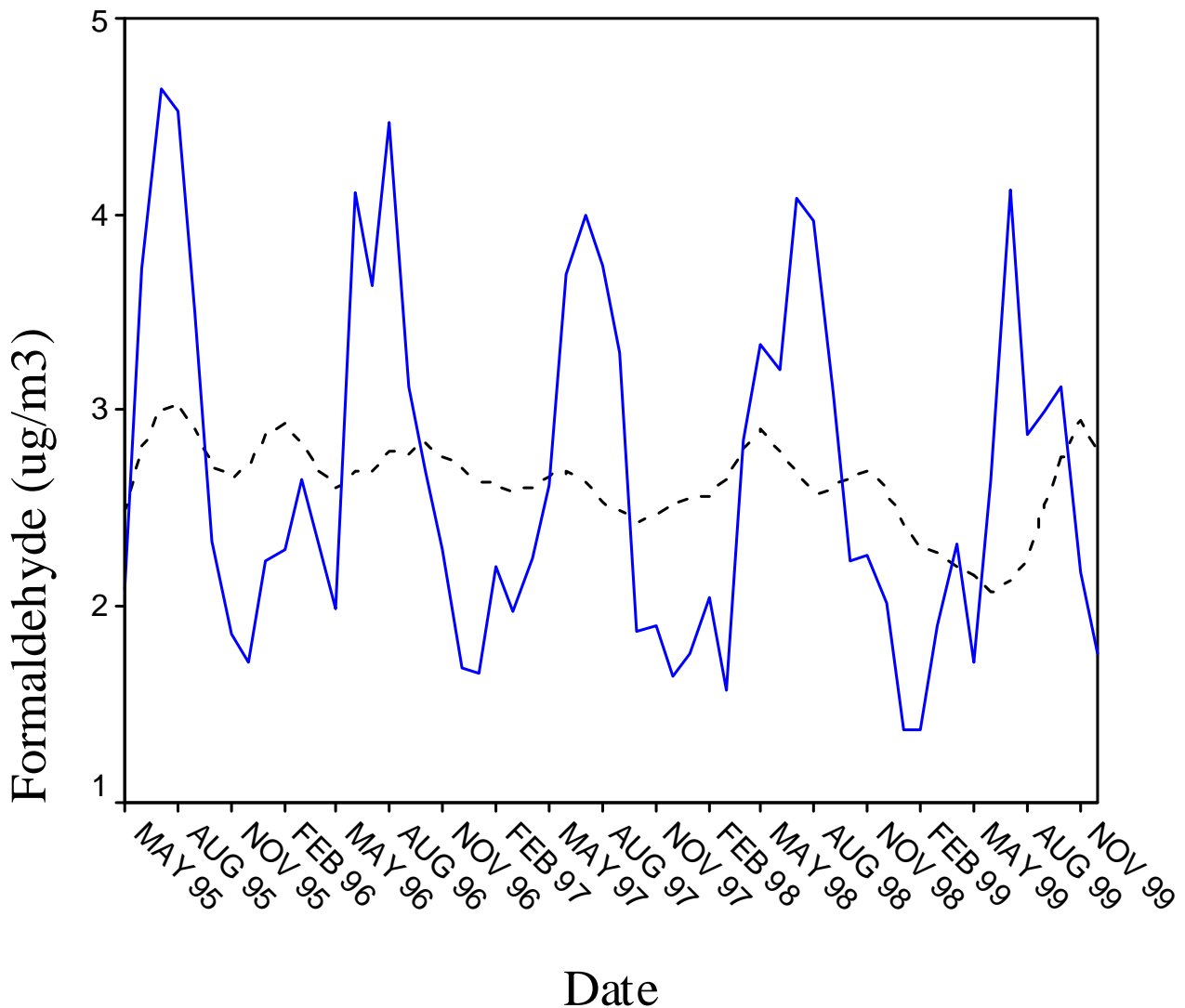
For formaldehyde trend analysis, five sites in urban areas such as the Twin Cities and Duluth were analyzed. These monitoring sites have concentrations dating back to 1991

which allowed for long-term trend analysis. In May 1995, the monitoring technique for carbonyls was changed by adding ozone scrubbing. Ozone present in ambient air will react with and destroy formaldehyde in a sample, so scrubbing the ozone will lead to higher and more accurate measurements. Therefore, only data from May 1995 to the end of 1999 were analyzed for trends.

The formaldehyde data were seasonal, with maximum concentrations occurring in the summer and minimums in the winter. The Minneapolis Public Library site shows this seasonality most clearly, but the other four locations also show similar seasonal variation.

Figure 13 shows the trend in formaldehyde measurements at site 945, the Minneapolis Public Library site. The solid line shows monthly average concentrations. The dotted line is a deseasonalized, smoothed trend line. All values are higher than the inhalation health benchmark for formaldehyde ($0.8 \mu\text{g}/\text{m}^3$).

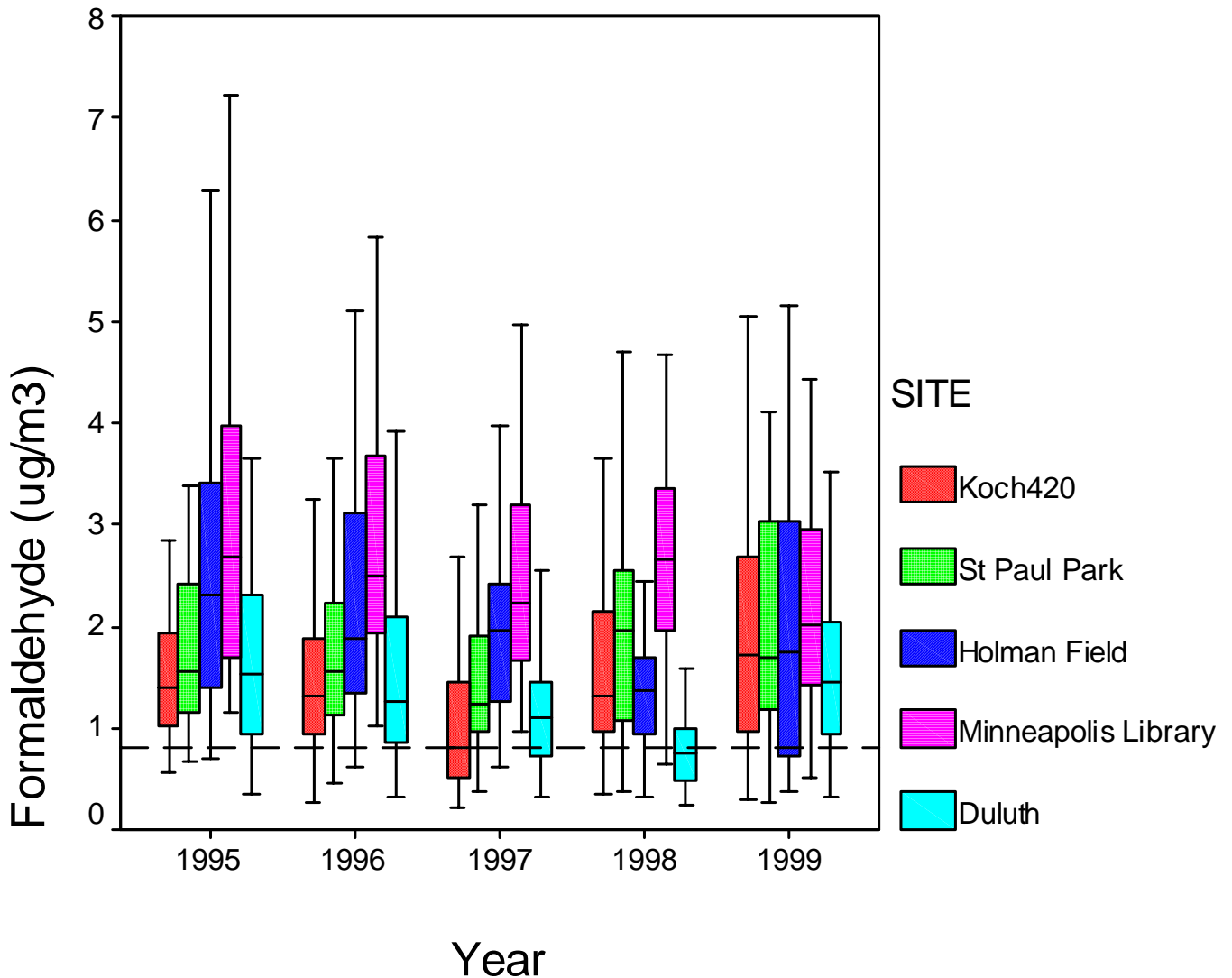
Figure 13: Formaldehyde Trends at the Minneapolis Library Monitoring Site



The Minneapolis Library site was the only site with a small, but statistically significant trend from 1995-1999. The Minneapolis Library site showed a small decrease in formaldehyde concentration of $0.086 \mu\text{g}/\text{m}^3$ per year. The other sites did not show statistically significant changes in formaldehyde concentration for the five-year time-frame. Boxplots of the mean formaldehyde concentrations for the five trend sites are in the table below.

Figure 14 shows the concentrations of formaldehyde at five sites in Minnesota. The center line within each box represents the median for the site. The box itself encompasses the 25th percentile to the 75th percentile. The bars at each end of the box represent the highest and lowest values that are not considered outliers. The vertical dotted line is located at the formaldehyde inhalation health benchmark. ($0.8 \mu\text{g}/\text{m}^3$).

Figure 14: Formaldehyde Concentrations by Monitoring Site



According to the CEP final report (SAI, 1999), natural background levels of formaldehyde in 1981 were 0.25 ug/m³.

Chemicals Below LDL

The following chemicals could not be analyzed in depth due to the low percentage of values above the lower detection limits. Some indications regarding concentrations can be derived from the monitoring data.

Arsenic

Only three percent of the arsenic monitoring data was above the LDL. However, the data indicates that arsenic concentrations across the state may be approaching the cancer inhalation health benchmark value of 0.002 ug/m³ which is based on the MDH proposed health risk value (HRV). It would be helpful to analyze arsenic using a more sensitive technique than screening level x-ray fluorescence.

1,3-Butadiene

1,3-butadiene began to be analyzed in the 1998-99 monitoring year. The adequacy of the monitoring technique is still somewhat uncertain. So far, only one percent of the monitoring data has been above the LDL. No exceedences of the cancer inhalation health benchmark of 0.04 ug/m³ have been indicated by the SATMN data. This inhalation health benchmark is based on MDH's proposed health risk value (HRV). EPA will likely modify the 1,3-butadiene cancer assessment shortly (Koppikar, personal communication with MPCA, January, 10, 2001). This new assessment, if adopted by MDH, would result in a higher inhalation health benchmark.

There are indications that 1,3-butadiene breaks down too rapidly to be monitored at rooftop level monitoring sites. Street-level monitoring has resulted in higher concentrations being measured. If the benchmark is raised to a significantly higher value, there would likely be no need for a more sensitive analysis technique.

Chromium

Eighteen percent of chromium monitoring data was above the LDL. The data indicates the chromium levels may be approaching the chromium VI cancer inhalation health benchmark value of 0.0008 ug/m³. The inhalation health benchmark for chromium VI is based on MDH's proposed health risk value (HRV). However, it is uncertain how applicable the chromium VI benchmark value is to the total chromium analyzed by MPCA. Minnesota-specific chromium speciation information is currently unavailable. It would be helpful to analyze chromium using a more sensitive technique than screening level x-ray fluorescence.

Ethylene Dibromide

Only one percent of the ethylene dibromide data was above the LDL. The SATMN data indicates that a few sites may be approaching the cancer inhalation health benchmark of 0.05 ug/m^3 . This inhalation health benchmark is based on the MDH proposed health risk value (HRV). According to the CEP final report (SAI, 1999), background levels of ethylene dibromide in 1991 were 0.0077 ug/m^3 . A more sensitive monitoring technique would be helpful in further characterizing ethylene dibromide concentrations.

Nickel

Two percent of nickel data was above the LDL. However, the cancer inhalation health benchmark of 0.02 ug/m^3 is well above the LDL of 0.002 ug/m^3 . This inhalation health benchmark is based on the MDH proposed health risk value (HRV) for nickel subsulfide which represents the most hazardous nickel compounds. The makeup of ambient nickel concentrations may be somewhat less hazardous. None of the SATMN data concentrations approach the cancer inhalation health benchmark. Therefore, it does not seem necessary to investigate a more sensitive monitoring approach for nickel at this time.

3.4 EPA's National-Scale Air Toxics Assessment (NATA)

On August 17, 2000, the Environmental Protection Agency (EPA) released the first two steps of a national assessment of the potential health risks associated with exposure to air toxics. The information includes 1996 estimated air toxics emissions and estimated outdoor concentrations of 32 common air toxics identified as posing the greatest potential risks to public health in urban areas. EPA will complete the next two steps of the assessment – estimates of exposure and health risk – and will submit the entire assessment for scientific peer review in early 2001.

When complete, the assessment will look at 34 air pollutants nationwide, in both urban and rural areas. Those pollutants include diesel particulate matter (DPM) and the 33 air toxics that the EPA identified in its Integrated Urban Air Toxics Strategy as posing the greatest potential risks to public health in urban areas. Diesel PM is an indicator of diesel exhaust, a pollutant mixture that EPA has recently proposed as a mobile source air toxic and is addressing in several regulatory actions. EPA plans to update this assessment every three years. The next assessment, due in 2003, will focus on 1999 emissions, concentrations and risks. These assessments will help EPA measure progress in reducing risks from exposure to toxics in the air.

In 1998, EPA released the findings of its Cumulative Exposure Project, which estimated 1990 outdoor levels of 148 air toxics nationwide. The NATA used the same computer model as the CEP, but predicts concentrations of just 34 pollutants. NATA is based on more recent meteorological and emissions data (1996) and will include a step that the Cumulative Exposure Project did not, that is the estimation national inhalation risk through computer modeling of inhalation.

The National-Scale Air Toxics Assessment comprises four steps, the first two of which were released on August 17, 2000:

1. A national inventory of air toxics emissions from sources in the contiguous 48 states, Puerto Rico and the Virgin Islands. The types of emissions sources in the inventory include large sources such as waste incinerators and factories and smaller sources, such as dry cleaners, small manufacturers and wildfires. Also included in the inventory are emissions from on-road and non-road mobile sources, such as cars, trucks and boats. (Completed)
2. Estimates of average concentrations of toxics in the outdoor air. These estimates are developed using a computer model that analyzes a number of factors, including total emissions, the number of emissions sources in a particular area, weather patterns and pollution source characteristics. (Completed)
3. Estimates of population exposures. Exposure estimates are based on estimated outdoor concentrations and on a model that looks at the amount of an air toxic a person is likely to inhale in a year's time. The average concentration of a pollutant that people breathe is known as an exposure concentration. Estimating exposure is a key step in determining potential health risk. (Target date: early 2001)
4. Characterization of potential public health risks. This last phase of the assessment will look at cancer and other health problems potentially associated with breathing air containing toxics. This characterization will quantify, where appropriate, potential cumulative risks to public health caused by breathing air toxics in the outdoor air. It also will discuss the uncertainties and limitations of the assessment, and identify other potential risks to public health from air toxics. (Target date: early 2001)

About the NATA Emissions and Concentration Data

- In order to understand the overall performance and limitations of the concentration estimates, EPA compared them to available monitoring data. This quality assurance check was done for seven pollutants: benzene, perchloroethylene, formaldehyde, acetaldehyde, cadmium, chromium and lead. The results of the model-to-monitor comparison can be found at <http://www.epa.gov/ttn/uatw/nata/nata2/draft5.html>.
- The model-to-monitor comparisons generally showed reasonably good agreement between concentration estimates and monitored values. Due to uncertainties in modeled source locations, EPA cautioned that the model estimates are uncertain on a local scale, and that they are more reliably interpreted as being a value likely to be found somewhere within 30 kilometers of the census tract centroid location.
- In general, as shown in Figure 15, the model estimates tended to be lower than the monitored values. The medians of the model/monitor ratios were 0.92 for benzene, 0.52 for perchloroethylene, 0.65 for formaldehyde, 0.60 for acetaldehyde, 0.176 for

lead, 0.18 for cadmium, and 0.15 for chromium. The performance of the model for metals was worse than for VOCs, in part because metals are emitted mainly from point sources, and the point source locations were often uncertain.

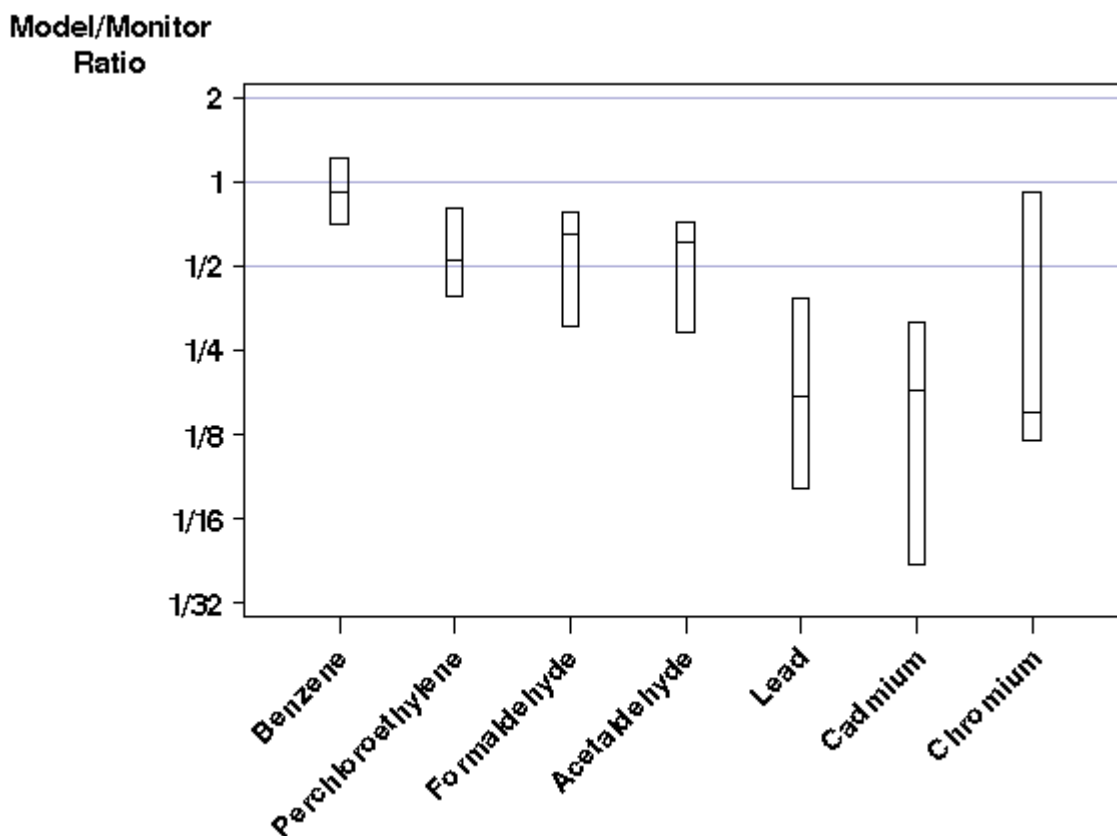


Figure 15. Box plot showing the distribution of model/monitor ratios for seven pollutants. The bottom of each box is the 25th percentile, the top is the 75th percentile, and the horizontal line in the middle is the median.

- The data show that both emissions and estimated concentrations of the 32 air pollutants generally were higher in urban than in rural areas. Urban areas tended to have heavier concentrations of factories, vehicles and other commercial activities that emit toxic air pollutants.
- Some pollutants, such as benzene (which is present in gasoline), were relatively evenly distributed across the country, while others, such as vinyl chloride, were linked to areas of industrial activity.
- No single state had the highest concentration of all 32 air toxics. Because different types of sources contribute to emissions in different areas of the country, the state with the highest average outdoor concentration varied by pollutant.

- No single type of source (major industrial, highway vehicles, non-road vehicles, and smaller sources) contributed the most to the estimated concentrations of all the 32 pollutants. However, results of the concentration analysis showed that, on a national level, smaller sources as a group dominated contributions for about half of the pollutants.
- The National-Scale Air Toxics Assessment web site is available at: <http://www.epa.gov/ttn/uatw/nata/>

A Closer Look at the NATA Results for Minnesota

The MPCA analyzed past EPA data from the Cumulative Exposure Project (CEP) in detail and compared the modeling results with monitored data (Pratt et al., 2000). Those results showed that, in general, the CEP modeling results underestimated concentrations where comparable monitoring data were available. This present analysis compares the newly released NATA modeling data with the CEP modeling results and with monitored data.

Tables 21a and 21b show the summary statistics for the NATA-CEP and NATA-monitor comparisons, respectively. Figures 16-19 show the comparisons graphically. In general, the NATA modeled concentrations compared favorably with the CEP modeled concentrations. For 12 pollutants the NATA predictions were significantly higher than the CEP predictions, while for 9 pollutants the CEP predictions were significantly higher. For 18 pollutants the average ratio between the NATA and CEP predictions was within a factor of two. The correlation coefficients between the two model predictions were generally high and were statistically significant for all but 5 pollutants.

Table 21a shows summary statistics for the fifteen pollutants and sixteen sites that could be compared between NATA and the MPCA monitoring data. In general, the two compared favorably. The monitored results were statistically higher for acetaldehyde, ethylene dibromide and tetrachloroethylene, while the NATA modeled results were higher for carbon tetrachloride. The lack of statistically significant differences (when compared with the NATA-CEP statistical analysis) is in part due to the small sample size. The correlation coefficients between the NATA results and the monitoring data were statistically significant for eight of the fifteen pollutants. In three cases, carbon tetrachloride, 1,3-dichloropropene, and trichloroethylene, the correlation coefficients were negative. For 6 pollutants the average ratio between the NATA and monitor data was within a factor of two.

Figure 16 shows boxplots of the distribution of values for the NATA and CEP modeled concentrations. It is clear that the NATA concentrations of benzene, ethylbenene, toluene, acrolein, 1,3-butadiene, POM, acrylonitrile, and chromium were higher than the CEP-predicted concentrations. On the other hand, the CEP predictions were clearly higher for MTBE, tetrachloroethylene, trichloroethylene, lead, and manganese. Figure 17 shows the differences between the CEP and NATA results graphed as standardized differences. CEP

concentrations were higher for those pollutants with negative values, while for those with positive values the NATA-predicted concentrations were higher.

Figure 18 shows boxplots of the distribution of values for the NATA modeled results and for the comparable monitored data. Monitored concentrations of acetaldehyde, ethylbenzene, formaldehyde, chloroform, propionaldehyde, styrene, tetrachloroethylene, and trichloroethylene were clearly higher than the NATA modeled results. NATA modeled results were clearly higher for 1,3-dichloropropene. Figure 19 shows the differences graphed as standardized differences. Monitored concentrations were higher for those pollutants with negative values, while for those with positive values the NATA-predicted concentrations were higher. It is clear from the graph that the monitored concentrations generally exceeded the modeled values.

Table 21a. A comparison of the NATA and CEP modeling results for Minnesota census tracts. The number of census tracts (the number of observations in each case) is 1230. Mean values that are shaded are significantly higher than the mean value for the opposite modeling study.

	NATA						CEP					
	Mean	SE	Median	Min	Max	95th Percentile	Mean	SE	Median	Min	Max	95th Percentile
Acetaldehyde	0.6092	0.0174	0.3024	0.0112	3.7157	1.4659	0.4746	0.0124	0.3225	0.0043	2.2967	1.2015
Acrolein	0.1765	0.0044	0.1175	0.0029	0.8712	0.3928	0.1116	0.0033	0.0759	0.0010	1.1005	0.2888
Acrylonitrile	0.0005	0.0000	0.0003	0.0000	0.0133	0.0011	0.0005	0.0001	0.0003	0.0000	0.0489	0.0015
Arsenic	0.0001	0.0000	0.0001	0.0000	0.0015	0.0003	0.0006	0.0001	0.0002	0.0000	0.1065	0.0019
Benzene	1.6981	0.0299	1.4606	0.4963	5.1012	3.5066	1.4995	0.0281	1.2093	0.4868	8.0584	3.3992
Beryllium	0.0000	0.0000	0.0000	0.0000	0.0005	0.0000	0.0000	0.0000	0.0000	0.0000	0.0003	0.0000
1,3-Butadiene	0.2928	0.0115	0.2135	0.0019	6.7047	0.8609	0.1066	0.0033	0.0663	0.0007	1.0397	0.3252
Cadmium	0.0004	0.0000	0.0001	0.0000	0.0122	0.0013	0.0001	0.0000	0.0001	0.0000	0.0020	0.0004
Carbon Tetrachloride	0.8828	0.0004	0.8802	0.8800	1.0585	0.8901	0.8836	0.0002	0.8818	0.8810	0.9582	0.8911
Chloroform	0.0873	0.0005	0.0838	0.0830	0.3244	0.0979	0.0880	0.0003	0.0855	0.0830	0.2834	0.0979
Chromium	0.0025	0.0001	0.0012	0.0000	0.0337	0.0078	0.0014	0.0001	0.0007	0.0000	0.0528	0.0050
1,3-Dichloro- propene	0.0617	0.0017	0.0453	0.0001	0.3273	0.1754	0.0247	0.0007	0.0204	0.0000	0.1299	0.0690
Ethyl Benzene	0.4749	0.0118	0.3585	0.0041	1.6800	1.1866	0.3280	0.0093	0.2270	0.0015	2.4353	0.9315
Ethylene Dibromide	0.0077	0.0000	0.0077	0.0077	0.0077	0.0077	0.0077	0.0000	0.0077	0.0077	0.0077	0.0077
Ethylene Dichloride	0.0621	0.0001	0.0617	0.0610	0.0892	0.0642	0.0621	0.0001	0.0614	0.0607	0.1907	0.0647
Ethylene Oxide	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0008	0.0000	0.0007	0.0000	0.0042	0.0022
Formaldehyde	0.9870	0.0218	0.7473	0.2678	6.6824	1.9380	0.9566	0.0203	0.7506	0.2573	6.1505	2.1243

	NATA						CEP					
	Mean	SE	Median	Min	Max	95th Percentile	Mean	SE	Median	Min	Max	95th Percentile
Hexachloro- benzene	0.0001	0.0000	0.0001	0.0001	0.0001	0.0001	0.0001	0.0000	0.0001	0.0001	0.0001	0.0001
Hexane	0.5732	0.0175	0.4353	0.0045	6.5019	1.4499	0.5920	0.0179	0.4458	0.0025	6.7895	1.5421
Lead	0.0037	0.0002	0.0016	0.0000	0.1712	0.0119	0.0049	0.0002	0.0038	0.0000	0.2328	0.0117
Manganese	0.0009	0.0000	0.0007	0.0000	0.0132	0.0025	0.0029	0.0001	0.0024	0.0000	0.0406	0.0080
Mercury	0.0016	0.0000	0.0016	0.0015	0.0022	0.0018	0.0018	0.0000	0.0017	0.0015	0.0038	0.0022
MTBE	0.0199	0.0006	0.0174	0.0005	0.2699	0.0511	0.3792	0.0107	0.2722	0.0008	3.1051	1.0374
PCBs	0.0004	0.0000	0.0004	0.0004	0.0004	0.0004	0.0004	0.0000	0.0004	0.0004	0.0004	0.0004
POM	0.3467	0.0090	0.2978	0.0024	2.0185	0.8927	0.1697	0.0045	0.1233	0.0010	1.1174	0.4573
Propionaldehyde	0.1335	0.0036	0.0652	0.0020	0.6037	0.3147	0.1055	0.0027	0.0666	0.0005	0.4197	0.2617
Propylene Dichloride	0.0000	0.0000	0.0000	0.0000	0.0002	0.0000	0.0000	0.0000	0.0000	0.0000	0.0041	0.0001
Styrene	0.0513	0.0020	0.0330	0.0003	1.0292	0.1315	0.0448	0.0020	0.0214	0.0001	1.2111	0.1398
Tetrachloro- ethylene	0.2253	0.0029	0.1797	0.1400	0.9177	0.4061	0.4987	0.0124	0.3594	0.1423	3.3562	1.2874
Toluene	3.4174	0.0877	2.5515	0.0394	15.1370	8.6263	3.0599	0.1035	1.9865	0.0107	56.488	8.8087
Trichloroethylene	0.1916	0.0063	0.1109	0.0810	4.1884	0.4852	0.4589	0.0176	0.2482	0.0806	7.0837	1.3685
Vinyl Chloride	0.0058	0.0007	0.0011	0.0000	0.3346	0.0196	0.0026	0.0001	0.0020	0.0000	0.0832	0.0068
Xylenes	2.2812	0.0579	1.7459	0.0173	10.5570	5.6953	2.6142	0.0784	1.7805	0.1750	23.349	7.6331

Table 21b. A comparison of the NATA modeling results with monitored values. Monitoring data were collected at 16 sites in 1996 that could be compared with the NATA modeling result, and thus the number of observations in each case is 16. Mean values that are shaded are significantly higher than the opposite mean value.

	NATA						Monitored					
	Mean	SE	Median	Min	Max	95th Percentile	Mean	SE	Median	Min	Max	95th Percentile
ACETALDEHYDE	0.5858	0.1321	0.4925	0.0364	1.5716	1.5716	0.9987	0.1092	1.0714	0.3924	1.7566	1.7566
BENZENE	1.6381	0.2560	1.4625	0.5614	4.2376	4.2376	1.5177	0.1617	1.4831	0.7533	3.0333	3.0333
CARBON TETRACHLORIDE	0.8835	0.0020	0.8804	0.8800	0.9113	0.9113	0.7718	0.0301	0.6979	0.5988	0.9101	0.9101
CHLOROFORM	0.0957	0.0054	0.0860	0.0830	0.1439	0.1439	0.2149	0.0702	0.1420	0.0954	1.2592	1.2592
ETHYL BENZENE	0.4134	0.1068	0.2681	0.0162	1.4498	1.4498	0.4547	0.0523	0.4349	0.1994	0.9317	0.9317
ETHYLENE DIBROMIDE	0.0077	0.0000	0.0077	0.0077	0.0077	0.0077	0.0584	0.0029	0.0542	0.0431	0.0825	0.0825
FORMALDEHYDE	1.0283	0.1469	1.0983	0.3441	2.2300	2.2300	1.2431	0.1937	1.2409	0.3715	2.8528	2.8528
PROPIONALDEHYDE	0.1300	0.0294	0.1072	0.0067	0.3217	0.3217	0.1782	0.0202	0.1946	0.0814	0.2914	0.2914
1,3-DICHLORO- PROPENE	0.0508	0.0107	0.0393	0.0019	0.1559	0.1559	0.0332	0.0058	0.0257	0.0122	0.1084	0.1084
STYRENE	0.0799	0.0235	0.0261	0.0013	0.2787	0.2787	0.1308	0.0089	0.1278	0.0772	0.1878	0.1878
TETRACHLORO- ETHYLENE	0.2058	0.0162	0.1915	0.1409	0.3564	0.3564	0.3612	0.0470	0.3309	0.1872	0.9988	0.9988
TOLUENE	3.3892	0.9176	2.1624	0.1389	14.0550	14.0550	2.6134	0.3569	2.3036	0.8857	5.7761	5.7761
TRICHLOROETHENE	0.2127	0.0807	0.1264	0.0812	1.4000	1.4000	0.6009	0.1558	0.3713	0.1090	2.2723	2.2723
VINYL CHLORIDE	0.0070	0.0037	0.0011	0.0000	0.0591	0.0591	0.0020	0.0007	0.0015	0.0000	0.0115	0.0115
XYLENES	2.3071	0.6357	1.3628	0.0752	9.4026	9.4026	2.1232	0.2768	1.8995	0.8513	4.6070	4.6070

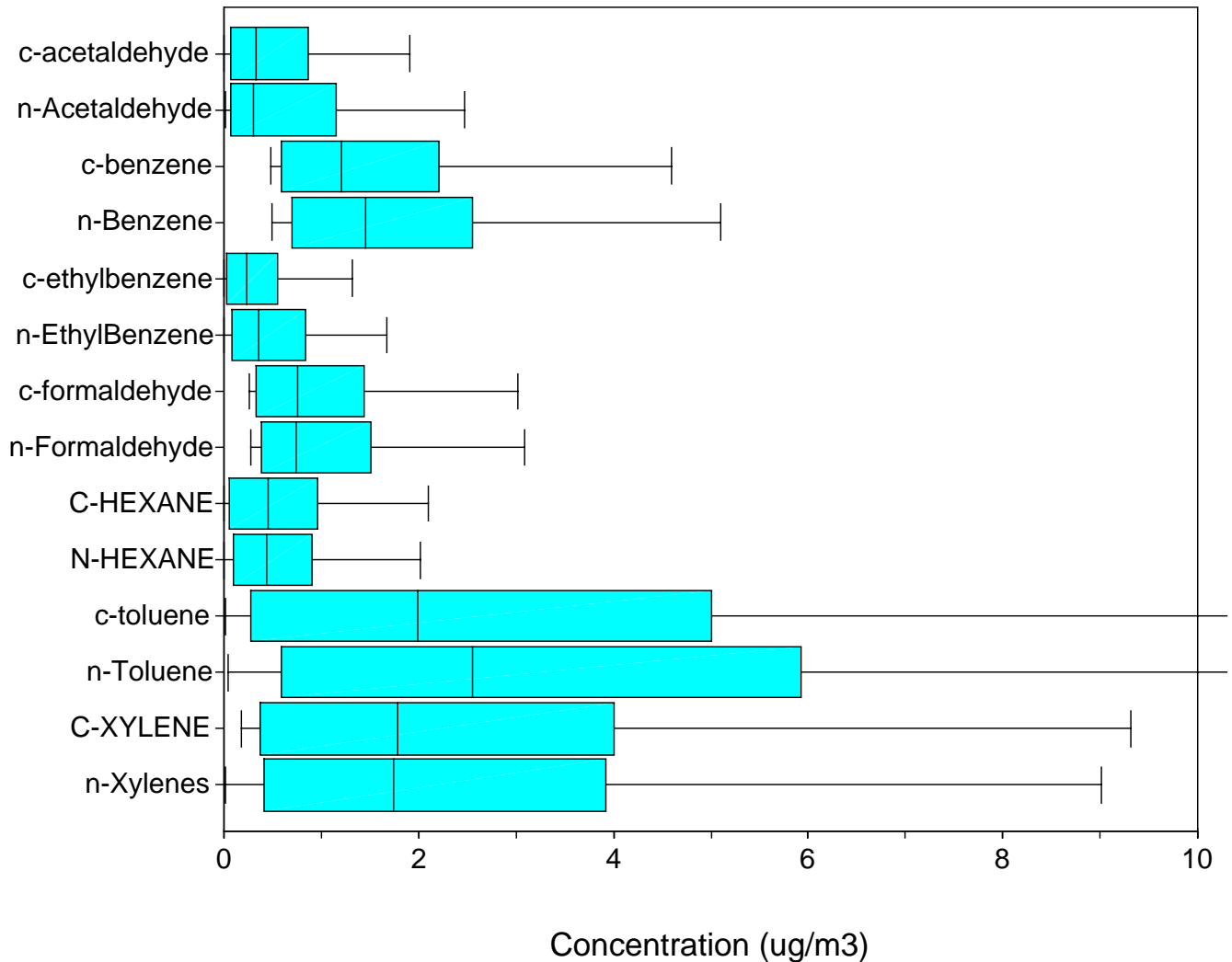


Figure 16a. A comparison of the NATA and CEP modeled concentrations of selected pollutants in Minnesota census tracts. The prefix ‘c’ before the pollutant indicates CEP and ‘n’ indicates NATA. The box incorporates the 25th to the 75th percentile values, the centerline in the box indicates the median value, and the arms encompass all values that are not considered outliers.

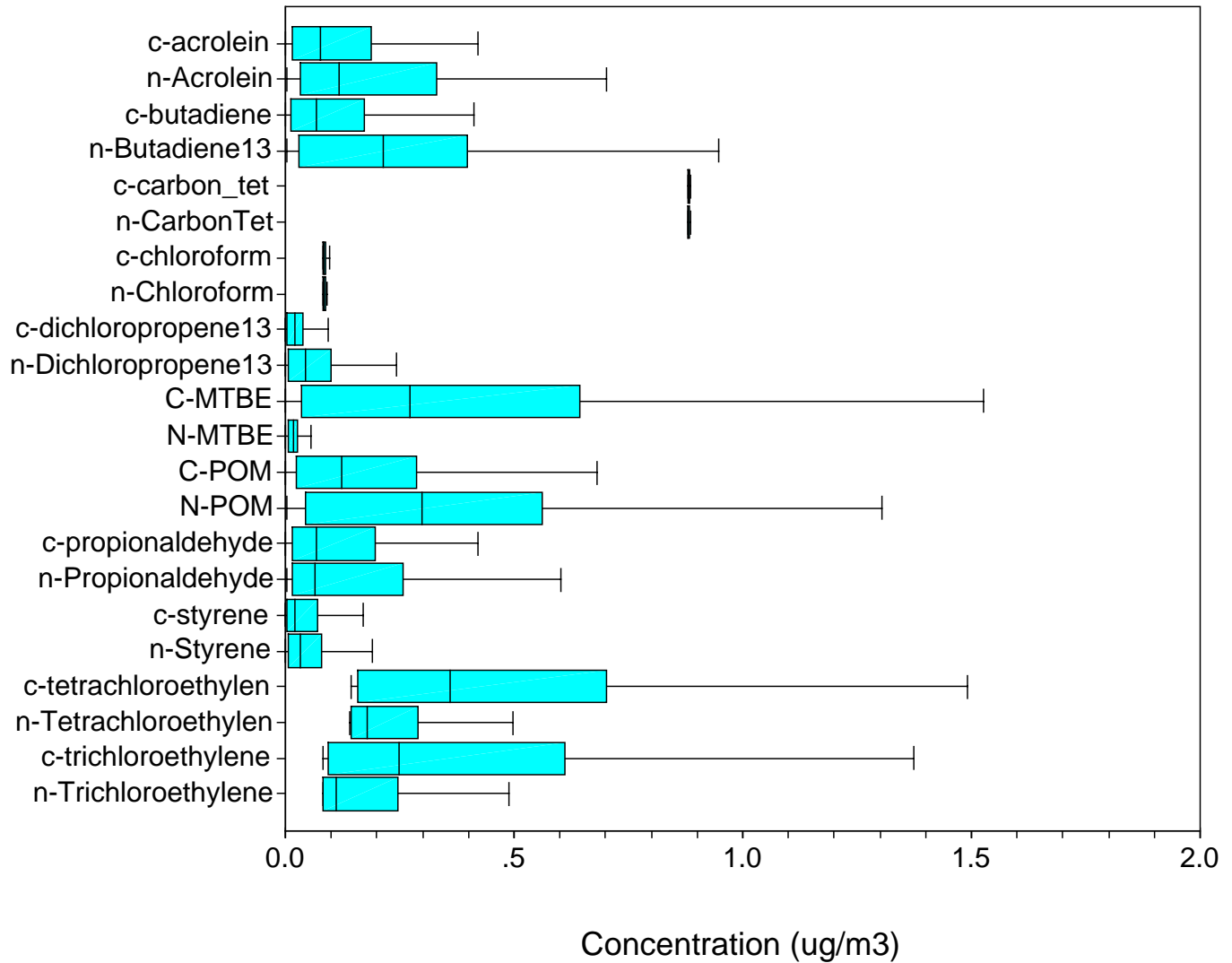


Figure 16b. A comparison of the NATA and CEP modeled concentrations of selected pollutants in Minnesota census tracts. The prefix ‘c’ before the pollutant indicates CEP and ‘n’ indicates NATA. The box incorporates the 25th to the 75th percentile values, the centerline in the box indicates the median value, and the arms encompass all values that are not considered outliers.

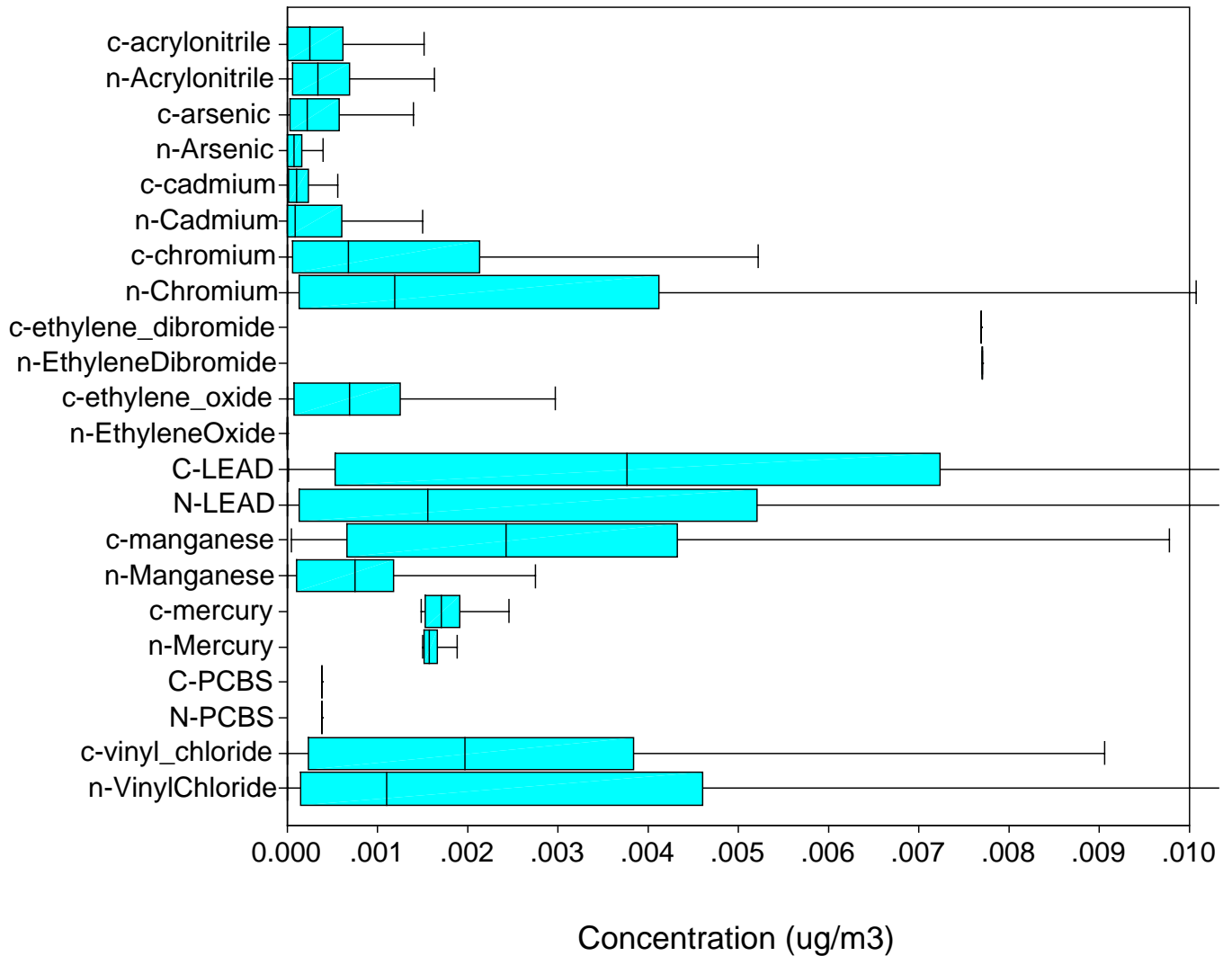


Figure 16c. A comparison of the NATA and CEP modeled concentrations of selected pollutants in Minnesota census tracts. The prefix ‘c’ before the pollutant indicates CEP and ‘n’ indicates NATA. The box incorporates the 25th to the 75th percentile values, the centerline in the box indicates the median value, and the arms encompass all values that are not considered outliers.

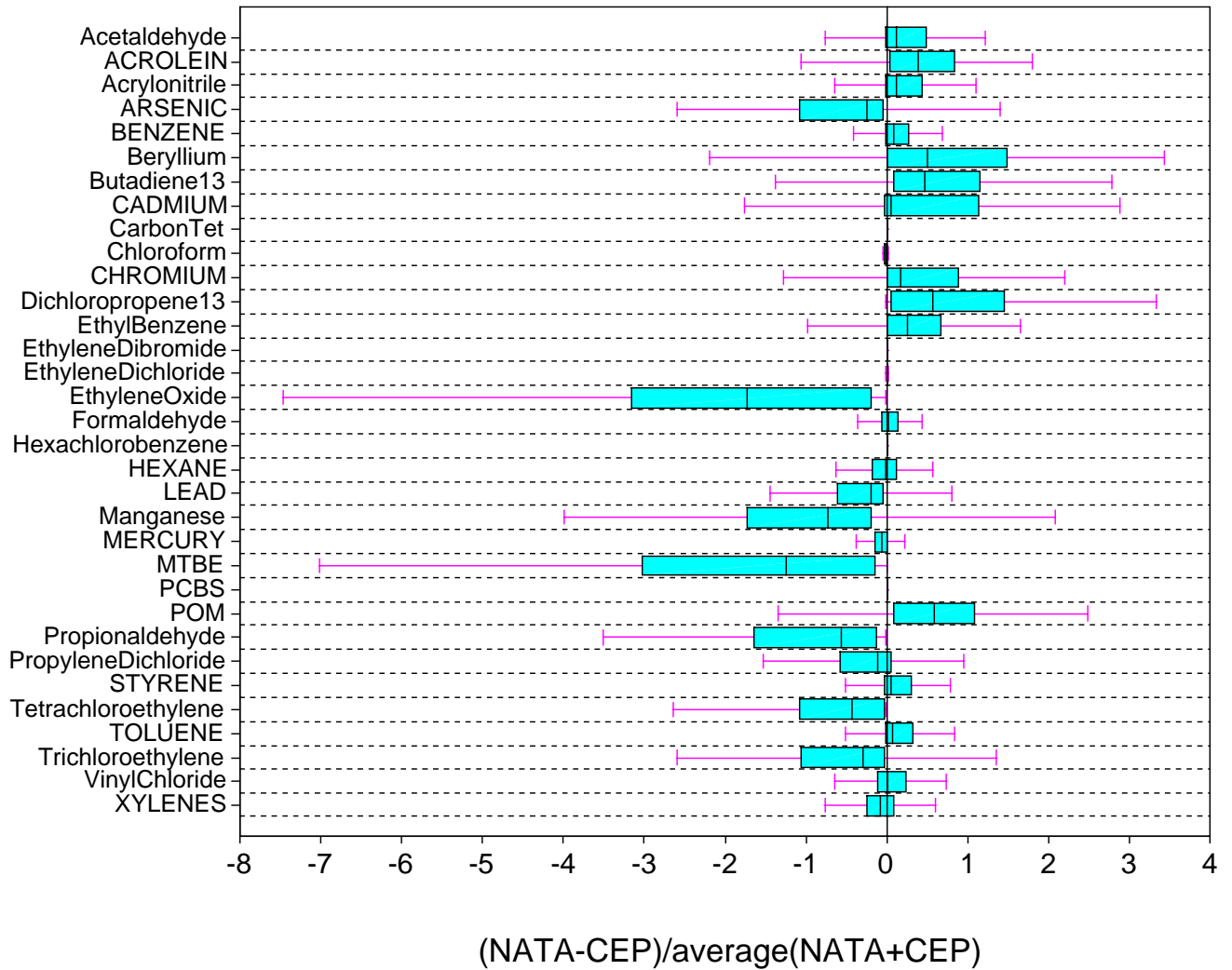


Figure 17. A comparison of the NATA and CEP modeled concentrations in Minnesota census tracts. Each bar encompasses the 25th to the 75th percentile value for the standardized difference (calculated as shown). Negative values indicate that the CEP prediction was higher. Positive values indicate that the NATA values were higher.

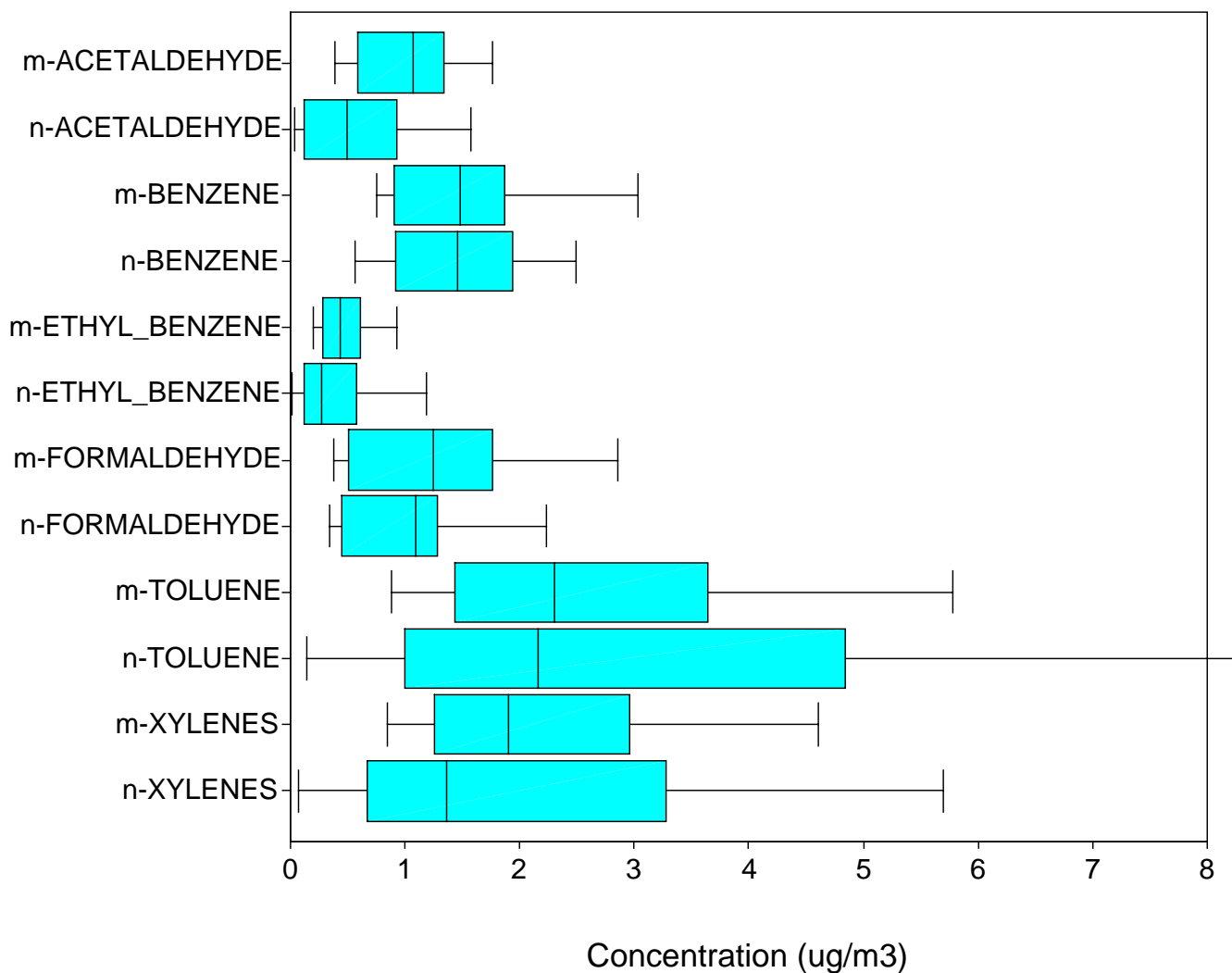


Figure 18a. A comparison of the NATA modeled predictions and monitored concentrations of selected pollutants in Minnesota census tracts. The prefix ‘m’ before the pollutant indicates modeled concentrations and ‘n’ indicates NATA predictions. The box incorporates the 25th to the 75th percentile values, the centerline in the box indicates the median value, and the arms encompass all values that are not considered outliers.

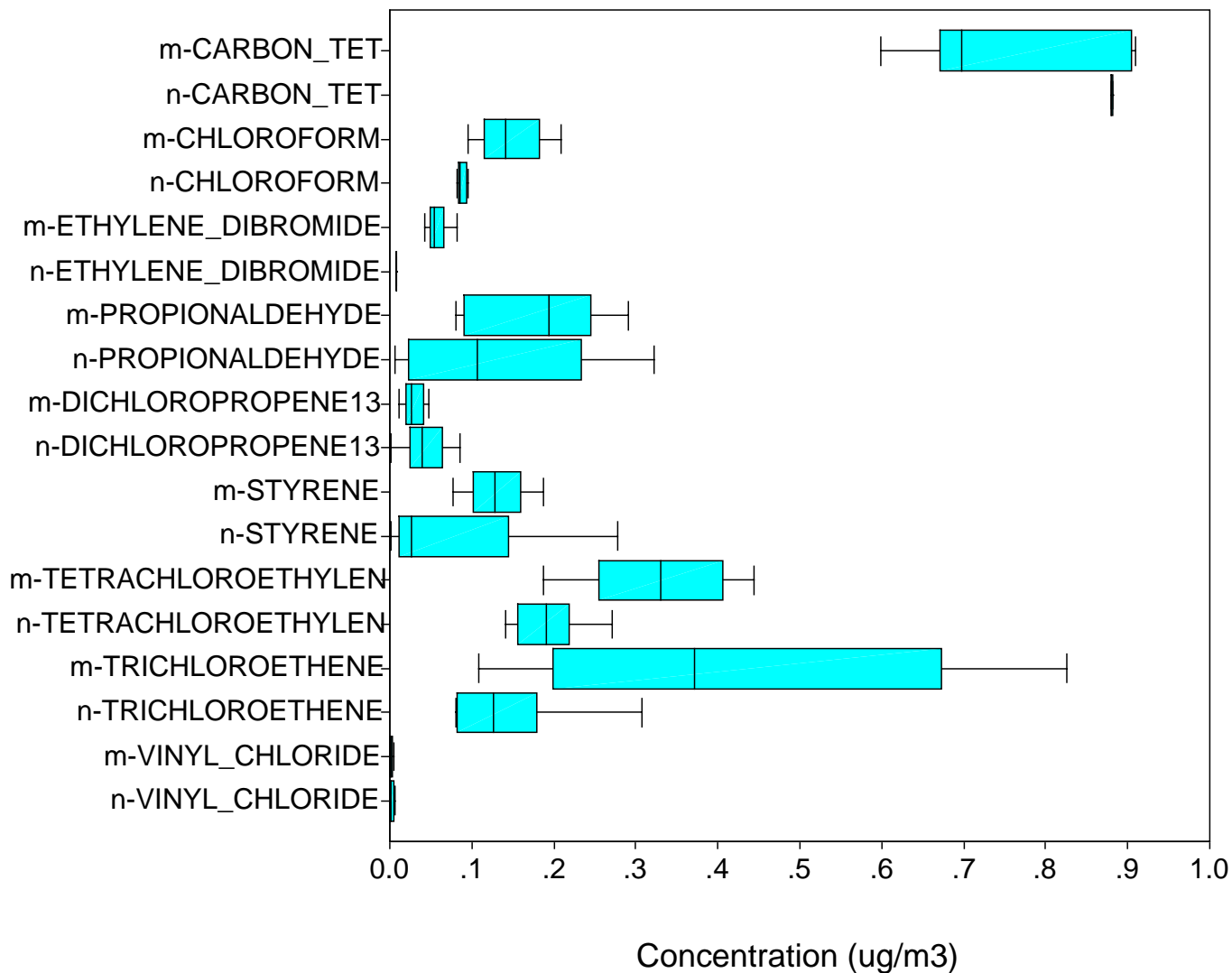


Figure 18b. A comparison of the NATA modeled predictions and monitored concentrations of selected pollutants in Minnesota census tracts. The prefix ‘m’ before the pollutant indicates modeled concentrations and ‘n’ indicates NATA predictions. The box incorporates the 25th to the 75th percentile values, the centerline in the box indicates the median value, and the arms encompass all values that are not considered outliers.

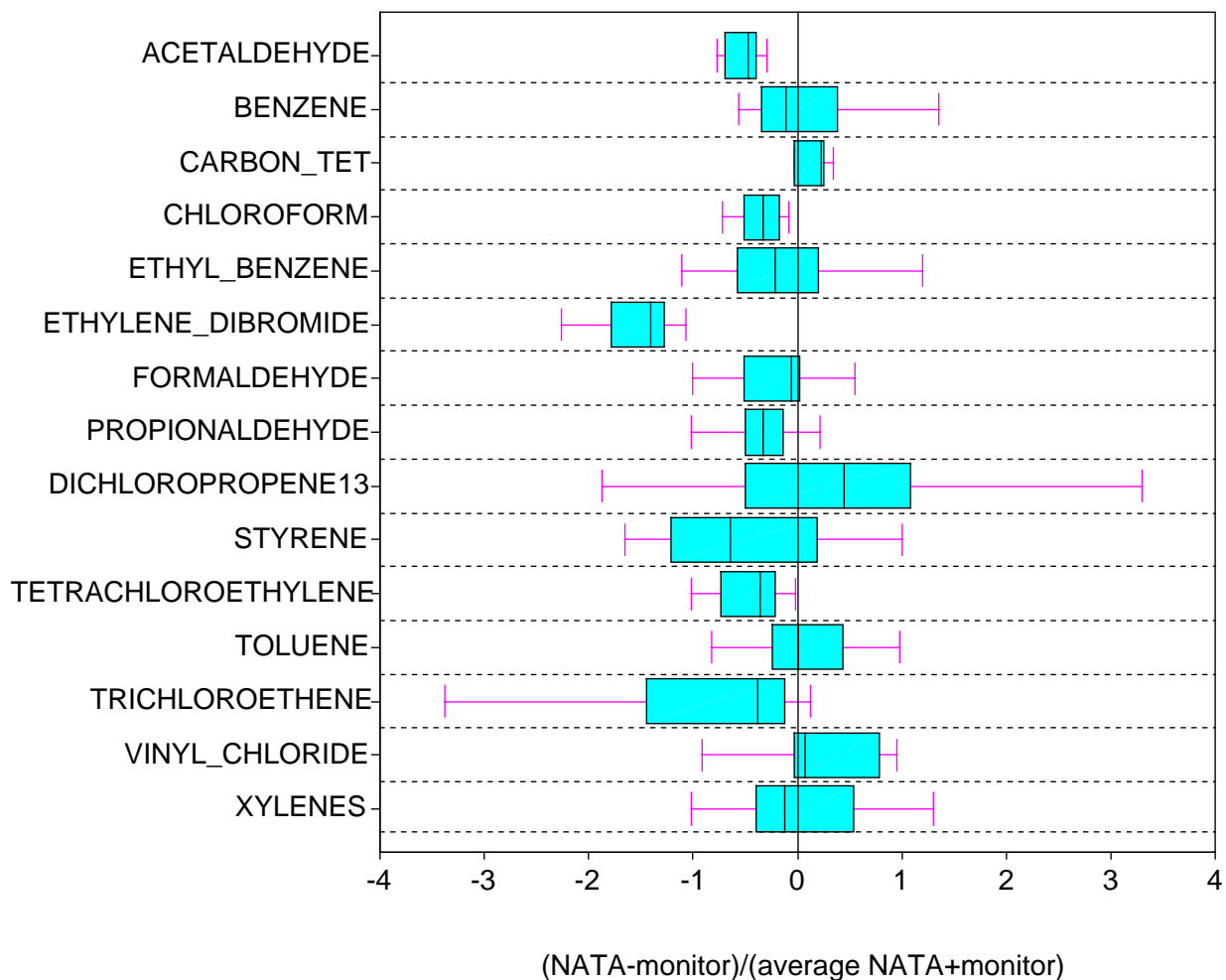


Figure 19. A comparison of the NATA and CEP modeled concentrations in Minnesota census tracts. Each bar encompasses the 25th to the 75th percentile value for the standardized difference (calculated as shown). Negative values indicate that the CEP prediction was higher. Positive values indicate that the NATA values were higher.

4.0 Health Information

Inhalation health benchmarks are chemical concentrations in the air believed to be essentially safe based on available information. They are developed and chosen in different ways by different organizations. These values are derived in a manner different than the National Ambient Air Quality Criteria (NAAQS).

4.1 Inhalation Health Benchmarks: Selection and Development

The MPCA relies on the following hierarchy to identify inhalation health benchmarks for protection from cancer or from other effects:

- (1) MPCA's preferred benchmarks are the Minnesota Department of Health (MDH) proposed Health Risk Values (HRVs). The MDH proposed HRVs for chemicals in air if adequate health risk information was available. For a given chemical an inhalation HRV may be designed to provide protection from cancer, noncancer effects or both.
- (2) For chemicals lacking HRVs, MPCA used available air quality inhalation health benchmark information from other agencies including U.S. EPA (<http://www.epa.gov/iris>) and California EPA (http://www.oehha.ca.gov/air/hot_spots/index.html). and the most current version of the EPA's Health Effects Assessment Summary Tables (HEAST) (EPA, 1997).
- (3) For some chemicals, no inhalation health benchmark information was available from MDH or these other agencies. In this case, for screening purposes MPCA may assume an inhalation health benchmark developed for a different chemical approximates the inhalation health benchmark for the chemical of interest.
- (4) In some cases, additional specific guidance from MDH or other additional information may supplement this hierarchy for identifying inhalation health benchmark values.

MDH Proposed Inhalation Health Risk Values (HRVs) for Long-term Exposures

The Minnesota Department of Health (MDH) proposed inhalation health risk values (HRVs) for Minnesota. These are concentrations of individual chemicals or chemical mixtures in air that MDH scientists are confident pose no appreciable risk to human health. As of February 2001, the HRVs have not yet been adopted into rule.

For exposures to many toxic air pollutants, the amount of harm depends on how long people breathe the polluted air. Proposed HRVs were calculated for short-term (acute) and/or long-term (chronic) exposures. Chronic HRVs, used for comparison with annual average ambient outdoor concentrations, are discussed in this appendix.

Sources of Toxicity Information

MDH scientists develop inhalation HRVs by reviewing scientific information about the harmful effects of the air pollutant. They use several types of information including epidemiological studies, animal studies, and in vitro studies, to understand how chemicals may harm people.

Epidemiology studies are used to investigate possible cause and effect relationships between a hypothesized risk factor (such as a chemical) and a human disease. Often these studies are done using information from worker exposures in occupational settings. These

studies are challenging and involve an extensive resource commitment, but when done well can provide the best evidence of disease causation. Difficulties in epidemiology studies for cancer include: long times between exposure and the cancer outcome, lack of good exposure information, and confounding factors (e.g., smoking). By itself smoking is strongly related to several diseases, so this can overwhelm the effects of much less important potential causes of the same diseases. In order to determine that a particular risk factor, such as benzene, causes a particular disease, such as leukemia, a number of conditions must be met:

- Strength of Association – The larger the relative risk (i.e., the ratio of the amount of disease in the more chemically exposed population to the amount of disease in the less exposed population) the greater likelihood that the chemical is causally related to the disease.
- Consistent Association - Higher exposures should result in more disease.
- Logical timing – The chemical exposure must occur before the disease.
- Specific Association – The greater the extent to which the chemical exposure is a major factor in predicting the disease the better. When there are other key factors in disease causation, it is more difficult to show the link. For example, because smoking causes a large portion of the lung cancer deaths, it is difficult to detect the relatively small additional risk that toxic air pollutants, including diesel exhaust particles, may contribute.
- Biological Plausibility– A scientific explanation for how a chemical may cause the disease strengthens the link

Because human information is very limited for most toxic air pollutants, scientists often conduct studies on laboratory animals. Animal studies are performed under controlled laboratory conditions so that a variety of health effects can be studied by exposing animals to pollutants at varied concentrations and for varied time periods. When this information is extrapolated to humans, it is important to be aware of possible differences between the human and animal response.

Short term tests on isolated tissues, cells, single celled organisms, or cellular components study whether a chemical may cause cancer by testing whether it will chemically alter DNA, (i.e., is it mutagenic).

Health Risk Value Derivation – Minnesota Department of Health

MDH is developing proposed HRVs for breathing toxic air pollutants using two general approaches depending on whether the chemical is believed to have a threshold for causing cancer or other types of adverse (noncancer) effects. In the future, as scientists better understand the biological mechanisms by which these chemicals cause harm, and specifically whether each chemical has a threshold for the various effects, this information may be used in developing inhalation health benchmarks.

MDH uses a conservative approach to develop the HRVs (i.e., by design MDH chooses to err in the direction of protecting public health). This approach is consistent with the traditional EPA risk assessment guidelines. Information describing the specific methodology and rationale for developing the HRVs can be obtained from MDH. A brief description of the HRV development method is provided below.

To develop proposed HRVs for chemicals that may cause cancer, MDH assumes even the smallest exposure has some potential to cause cancer. This approach assumes that as a person's exposure increases, the chance of getting cancer also increases.

Because it is assumed for regulatory purposes that exposure to a single molecule of a carcinogen could cause cancer, HRVs for carcinogens are derived based on what MDH considers a negligible target cancer risk level. Proposed HRVs for carcinogens are calculated lifetime exposure concentrations of a given chemical that may result in a 1 in 100,000 or less chance of getting cancer from this exposure. This 1 in 100,000 estimate is an upper estimate of the cancer risk (typically a 95 percent upper bound) and the true cancer risk is likely to be lower and may be zero. One way to interpret this risk is that there could be one person or less, within a population of 100,000 people breathing this specific toxic air pollutant exposure for 70 years, who may develop cancer because of this exposure. Scientists believe there is at least a 95% chance there will be one or fewer additional cases of cancer from this air pollutant level of exposure.

The benzene HRV was developed in a similar manner with a few exceptions. The benzene HRV is a range and it was developed using the maximum likelihood estimate approach. Epidemiology studies of people exposed to benzene show a clear link between exposure and cancer (i.e., chiefly acute myelogenous leukemia in humans) at concentrations in the range of 30,000 ug/m³ benzene and above. The maximum likelihood estimate approach provides a more likely cancer risk estimate than the 95% upper bound estimate approach typically used. The HRV for long term exposure to benzene is given as a range from 1.3 ug/m³ to 4.5 ug/m³. The range reflects some of the uncertainty in the assessment. Exposure to benzene concentrations in the HRV range, for a 70-year period, may cause up to a 1 in 100,000 chance of getting cancer. This "incremental" risk is in addition to the approximately 1 in 2 overall chance of cancer from all causes.

In contrast to carcinogenic effects, the MDH approach to developing non-cancer benchmarks assumes that no adverse health effects will be observed from very small exposures to chemicals that do not cause cancer. A well-designed toxicity test will generally show that, for these chemicals, there is a level of exposure called the threshold, below which exposure is not harmful. This threshold is the amount of chemical that an animal can take into the body, metabolize, and pass out of the body without harm.

MDH develops proposed HRVs for protection from adverse effects other than cancer (noncancer effects) by first identifying an exposure concentration near or below the lowest threshold for any known health effects. The HRV is set at a lower concentration than the

near threshold level by applying uncertainty factors. For example, MDH may use uncertainty factors to account for:

- the variation in sensitivity among the members of the human population;
- the uncertainty in extrapolating animal data to estimate human health effects;
- the uncertainty in extrapolating from data obtained in a study that is of less than lifetime exposure;
- the uncertainty in using the lowest observable adverse effect level data rather than the no observed adverse effect level data; and
- an incomplete data base, generally with regards to developmental or reproductive toxicity.

(excerpted from Minnesota Department of Health, Health Risk Values rule, Briefing Paper #8, October 1996).

The near threshold level is divided by a combination of these uncertainty factors to calculate the HRV. Overall most uncertainty factors range from 30 to 1000 and result in proposed HRV concentrations that are typically 30 to 1000-times lower than the lowest measured threshold for health effects. Figure 20 illustrates the distribution of uncertainty factors used in developing the proposed chronic HRVs for non-cancer effects.

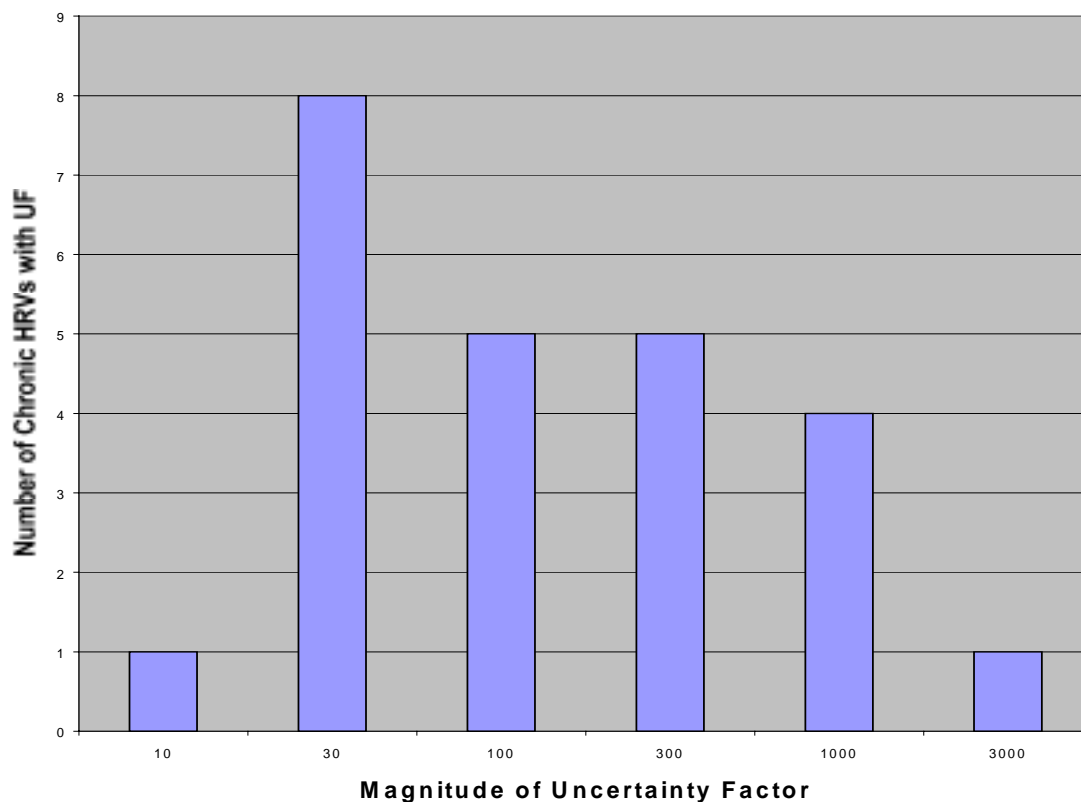


Figure 20. Distribution of Chronic HRV Uncertainty Factors for Non-cancer Effects

Inhalation Health Benchmarks Derivation - Other Agencies

For toxic air pollutants lacking MDH-derived proposed HRVs, MPCA used similar values available from other organizations (listed below). These organizations provide reference air concentrations (in ug/m³) for protection against non-cancer effects and unit risk values (in m³/ug) for cancer effects. In a manner analogous to the HRV development process, inhalation health benchmark concentrations for cancer effects are calculated by dividing the 1 in 100,000 MDH target excess cancer risk value by the unit risk values.

- U.S. Environmental Protection Agency Integrated Risk Information System (IRIS) RfCs and unit risk values (<http://www.epa.gov/ngispgm3/iris/index.html>)
- California Cancer Potency Factors (unit risk values) and Reference Exposure Levels (RELs) (http://www.oehha.ca.gov/air/hot_spots/index.html)
- U.S. Environmental Protection Agency Health Effects Assessment Summary Tables (HEAST) RfCs and unit risk values (EPA, 1997)

Surrogate Inhalation Health Benchmarks

For many toxic air pollutants there are no available proposed HRVs or scientifically derived regulatory inhalation health benchmarks specific to the substance in question. For these chemicals or mixtures it is not reasonable to assume that all exposure concentrations would be safe. Therefore, for these pollutants, and especially when available information suggests that environmental levels may be of concern, surrogate inhalation health benchmarks can be useful as very rough approximations of acceptable exposure concentrations. Surrogate inhalation health benchmarks are assigned by assuming pollutants with similar chemical structures have similar toxic properties and therefore should be assigned similar inhalation health benchmarks. An example of a surrogate inhalation health benchmark was the use of a benzo[a]pyrene inhalation health benchmark for particulate organic matter in the MPCA Staff Paper and a recent publication (MPCA, 1999, Pratt et al., 2000).

Like proposed HRVs and other inhalation health benchmarks, surrogate inhalation health benchmarks should not be interpreted as accurate measures of concentrations which will result in actual health effects. Rather, they may be useful as health protective (precautionary) air concentrations when little other information is available. For chemicals lacking other inhalation health benchmarks, surrogate inhalation health benchmarks are used because without them it may falsely be concluded that pollutants lacking inhalation health benchmarks pose no risks at any concentrations.

Additional Sources of Benchmark Values

A final source of inhalation health benchmark information, that may supersede the standard hierarchy, includes specific recommendations from MDH. MDH may recommend alternate inhalation health benchmarks for MPCA to use based on updated toxicity information not yet available through EPA. An example is a recent policy memo relating

to an increase in the chloroform inhalation health benchmark value. Benchmark information may also be used from other credible sources.

Inhalation Health Benchmarks for Use in Assessing the NATA Concentrations for this Report

Table 22 provides a summary of the inhalation health benchmarks used to assess the pollutants of concern for this analysis.

Table 22. Inhalation Health Benchmarks for Monitored and NATA Chemicals

NATA Chemicals / Urban Hazardous Air Pollutants	CAS Number	MPCA Health Benchmark (ug/m3)	Source	EPA Cancer Wt. Evid.	Non-cancer Uncertainty Factor	Basis	Comment
acetaldehyde	75-07-0	5	HRV	B2	1000	Cancer	
* acrolein	107-02-8	0.02	IRIS		1000	Noncancer	
acrylonitrile	107-13-1	0.1	HRV	B1	1000	Cancer	
* arsenic compounds	7440-38-2	0.002	HRV	A	NA	Cancer	See note (4)
* benzene	71-43-2	1.3	HRV	A	NA	Cancer	Lower bound HRV range; See note (4)
beryllium compounds	7440-41-7	0.004	HRV	B1	10	Cancer	
* butadiene(1,3-)	106-99-0	0.04	HRV	B2	NA	Cancer	See note (1)
cadmium compounds	7440-43-9	0.006	HRV	B1	NA	Cancer	
* carbon tetrachloride	56-23-5	0.7	IRIS	B2	NA	Cancer	
* chloroform	67-66-3	100	MDH	B2	1000	Noncancer	MDH policy (1/11/01).
* chromium VI	18540-29-9	0.0008	HRV	A		Cancer	See note 4.
coke oven emissions	8007-45-2	0.02	HRV	A		Cancer	
dichloropropene(1,3-)	542-75-6	3	HRV	B2	30	Cancer	
dioxin	1746-01-6	0.0000003	CAL EPA	A		Cancer	NATA emissions estimates not yet available. SAB draft for wt of evid.
* ethylene dibromide	106-93-4	0.05	HRV	B2	NA	Cancer	
ethylene dichloride	107-06-2	0.4	IRIS	B2	NA	Cancer	
ethylene oxide	75-21-8	0.1	CAL EPA	B1	NA	Cancer	
* formaldehyde	50-00-0	0.8	HRV	B1	10	Cancer	See note (2).
hexachlorobenzene	118-74-1	0.02	IRIS	B2		Cancer	
hydrazine	302-01-2	0.002	HRV	B2	NA	Cancer	
lead compounds	7439-92-1	0.8	CAL EPA	B2		Cancer	
manganese compounds	7439-96-5	0.2	HRV		100	Noncancer	
mercury compounds	7439-97-6	0.3	IRIS		30	Noncancer	Elemental mercury
methylene chloride	75-09-2	20	HRV	B2	100	Cancer	

	NATA Chemicals / Urban Hazardous Air Pollutants	CAS Number	MPCA Health Benchmark (ug/m3)	Source	EPA Cancer Wt. Evid.	Non-cancer Uncertainty Factor	Basis	Comment
*	nickel compounds	7440-02-0	0.04	CAL EPA	A	30	Cancer	For nickel subsulfide Not modeled in NATA
	polychlorinated biphenyls (PCBs)	1336-36-3	0.10	IRIS	B2		Cancer	
*	polycyclic organic matter (POM)	NA	NA		Varies		Cancer	
	propylene dichloride	78-87-5	4	IRIS	B2	300	Noncancer	
	quinoline	91-22-5	NA		C			
	tetrachloroethane (1,1,2,2-)	79-34-5	0.2	IRIS	C		Cancer	
	tetrachloroethylene (perchloroethylene)	127-18-4	1.7	CAL EPA	B2-C	NA	Cancer	
	trichloroethylene	79-01-6	5	CAL EPA	B2-C	100	Cancer	
	vinyl chloride	75-01-4	1	HRV	A	30	Cancer	
	Diesel Particulates	NA	5	MDH No Value	B1	30	Noncancer	MDH Policy 2/5/01 recommends against using any quantitative toxicity value for cancer at this time. Also see Appendix E.
	Benzo[a]pyrene	50-32-8	0.011	EPA - NCEA	B2		Cancer	See note (3) Not modeled for NATA

Notes:

Cancer benchmarks for a given target risk level, e.g., 1 in 100,000 (or 10⁻⁵) are derived by dividing the target risk by the unit risk estimate (URE): 10⁻⁵/URE.

* Highlighted in the Staff Paper (MPCA, 1999)

HRV is the Minnesota Department of Health, Draft Health Risk Values, October, 2000.

IRIS is the U.S. Environmental Protection Agency Integrated Risk Information System, December, 2000.

CAL EPA is the California Environmental Protection Agency Office of Environmental Health Hazard Assessment. Cancer unit risks, April 1999; chronic reference exposure levels (RELs), May 2000.

(1) EPA's draft reassessment shows a 25 fold decrease in the cancer potency estimate (for a benchmark of 1 ug/m³) and a 2 fold decrease in the RfC (to 4 ug/m³) (Koppikar, 2001). As of 1/10/01 consensus review was incomplete

(2) Formaldehyde HRV may not be protective for hypersensitive individuals (MDH draft HRV rule, October, 2000). Note the IRIS unit risk may decrease in the future.

(3) NCEA - EPA's National Center for Environmental Assessment provided a draft inhalation unit risk value for benzo[a]pyrene. This is very close to the CAL EPA value of 9.1 x 10⁻³ ug/m³.

(4) Arsenic, benzene and chromium VI unit risks were derived using a maximum likelihood estimator (MLE) method rather than 95% upper bound

Koppikar, 2001. Dr. Aparna Koppikar, EPA's contact for the 1,3-Butadiene toxicity reassessment, provided this information to MPCA on January, 10, 2001.

NA - Not available

4.2 Inhalation Health Benchmarks: Use and Uncertainty

Comparing ambient air concentrations with inhalation health benchmarks can provide a first estimate of toxic air pollutants that may be of concern. Several issues must be understood when comparing inhalation health benchmarks to ambient concentrations and when interpreting risk descriptions.

Actuarial (Most Likely) Risks vs. Inhalation Health Benchmark Risk Estimates

It is very important that people using inhalation health benchmark (or upper bound cancer risk) information understand what it means and what it doesn't mean. In daily life people hear about other risk estimates, for example, of the chance of being killed in a car accident. Records are kept of the actual number of auto-accident deaths that occur. These familiar actuarial risk estimates are close to being accurate, i.e., to the true likelihood or chance of being killed in a car accident. They can be developed with a reasonable amount of precision and certainty. It is easy to count these deaths and the cause of death is clear (auto accidents). Many common measures of public health risks, such as injury risks and certain disease rates, are of this nature.

Regulatory scientists develop inhalation health benchmarks based on a very different kind of risk information. Directly measured statistics describing the number of people who actually get cancer after being exposed to the relatively low levels of chemicals present in the ambient environment do not exist. For example, because cancers develop a long time following the exposure, and can occur for many different reasons, it is difficult to identify health effects specifically from low air pollutant exposure levels. Current epidemiological methods are not sensitive enough to detect an increased cancer risk of 1 in 100,000. In Minnesota, an individual's current lifetime risk of cancer from all factors (including smoking, diet, alcohol, etc.) is roughly 1 in 2. A study that could detect a 1 in 100,000 additional risk would have to have the statistical power to detect roughly a 0.005% increase. Estimates of appropriate inhalation health benchmarks and cancer risks must be made based on findings from animal studies or human epidemiology studies involving exposures to much higher levels than the ambient concentrations in outdoor air.

The inhalation health benchmarks developed for cancer effects have been used to develop upper bound excess cancer risk estimates. For example, lifetime exposures to air concentrations at the inhalation health benchmark concentrations, by definition, would result in excess cancer risks of up to 1 in 100,000. Similarly, lifetime exposures to air concentrations two times the HRV would be associated with up to a 2 in 100,000 excess cancer risk.

These risk estimates probably don't predict how many people would really get cancer from the given chemical exposure. These upper bound excess cancer risk estimates are intentionally designed to be higher than the real chance of getting cancer from a chemical exposure situation.

Variable Quality of Inhalation Health Benchmarks

Health-benchmarks are developed to be air concentrations likely to be without appreciable risk of harmful effects on humans. However, depending on the chemical, the level that could cause harm may be slightly higher than, or far above, the inhalation health benchmark. Lower inhalation health benchmarks may occur either because the chemical is relatively more dangerous or because it is a chemical for which little information is available. The use of inhalation health benchmarks for accurate prioritization is therefore problematic.

The inhalation health benchmarks are of varying quality with regard to how far they are below actual human effect levels. Although useful for setting protective levels below which appreciable harm is not expected, the inhalation health benchmarks are not designed to accurately predict risk and different inhalation health benchmarks involve different amounts of uncertainty. For chemicals with an extensive database of human toxicity information covering a broad range of concentrations, there is less uncertainty in setting the inhalation health benchmarks. For other chemicals for which information is limited, larger uncertainty factors or farther extrapolations are involved in developing the inhalation health benchmarks. In addition, inhalation health benchmarks are developed based on the available knowledge of adverse effects. For subtle and difficult to measure effects that have not been identified in toxicity or epidemiology studies, the inhalation health benchmark may not be protective of these unknown effects.

Inhalation health benchmarks are not available for many airborne chemicals for which there is essentially no toxicity information. Inhalation health benchmarks are available for some of the high-use chemicals, which are often better understood. The HRV development process by design is limited to the use of available peer reviewed toxicological and epidemiological information. Due to the intensive data requirements for HRV development, MDH has been unable to develop proposed HRVs for many chemicals present in ambient air. In some cases, the proposed HRV may be available for protection of some but not all of potential health effects. An example is diesel exhaust particulate matter. MDH identified adequate toxicity data to propose a HRV for protection from chronic noncancer effects, but inadequate information was available to develop a proposed HRV based on potential cancer effects. For most chemicals there may be proposed HRVs for some but not all exposure time periods (i.e., acute, subchronic or chronic).

By policy, MDH has not developed HRVs for the criteria air pollutants (ozone, lead, nitrogen oxides, sulfur dioxide, carbon monoxide, and particulate matter). Similarly, EPA has not included toxicity information for these air pollutants in the IRIS database. Unlike many air toxic chemicals for which there is little toxicity information, extensive human health effect information is available in the literature and in EPA Criteria Documents for the criteria pollutants, which are well known to have caused measurable and serious effects at ambient concentrations. These effects lead to the passage of the Clean Air Act and the enforceable National Ambient Air Quality Standards (NAAQS).

MPCA staff suspects that if the HRV development protocol was used to develop HRVs for criteria pollutants such as fine particulate matter, the HRVs would be lower than the current NAAQS.

Uncertainty in Exposures

People's exposures to chemicals in the air fluctuate daily and during their lifetimes. In contrast, chronic inhalation health benchmarks are developed to reflect average lifetime exposure concentrations. Currently, most ambient air assessments based on measured air concentrations rely on monitors located at a few central locations. These may not accurately reflect an individual's actual exposures. For example, some sources of exposures to air toxics, such as motor vehicle exhaust or the air in a gas station while filling a gas tank, are likely to be at higher concentrations than the ambient monitors. Personal exposures to some pollutants may therefore be higher than ambient monitored concentrations.

Uncertainty about Human Carcinogens

For many chemicals, there is uncertainty as to whether they can cause cancer in humans. National and international organizations publish their scientific judgements regarding the strength of evidence linking the chemical (in any amount) to human cancer. This weight of evidence approach, shown below, is how scientists report their level of uncertainty (or certainty). When interpreting cancer risk estimates, the evidence that these chemicals cause cancer should be considered.

For example, the EPA's cancer "weight of evidence" scheme is shown here:

- A - Human Carcinogen
- B1 - Probable Human Carcinogen - limited evidence in humans
- B2 - Probable Human Carcinogen - sufficient evidence in animals
- C - Possible Human Carcinogen - limited evidence in animals
- D - Not Classifiable as to Human Carcinogenicity - inadequate evidence
- E - Evidence of Noncarcinogenicity

The American Conference of Governmental Industrial Hygienists (ACGIH), the U.S. National Toxicology Program (NTP), and the International Agency for Research on Cancer (IARC) are other organizations that assign cancer weight of evidence ratings to chemical mixtures. They use similar rating schemes and typically report similar judgements.

Mixtures

In most cases, inhalation health benchmarks are developed for individual chemicals. Inhalation health benchmarks are available for exposures to a few specific mixtures (such as diesel exhaust). However, people are exposed to many chemical mixtures in daily life. Scientists would like better methods to estimate the health effects of exposures to

combinations of chemicals. The overall harm caused by chemical mixtures may be more or less harmful than the sum of the effects caused by each one alone.

Variability in Human Populations

Various groups of people, such as children, the elderly, asthmatics, the immunocompromised, and others may be more sensitive than the general population to the effects of a given chemical. Lacking any specific information about these differences, some uncertainty factors have been incorporated to protect sensitive sub-populations from adverse non-cancer effects of chemicals.

HRVs are developed using public health protection practices that advocate the protection of the most sensitive portion of the population. However, HRVs may not be protective of every individual. As has been demonstrated in occupational settings which provide information to identify the hazards of chemical exposures, certain people are sensitized (develop an allergic immune response) by exposures to high concentrations of certain chemicals. Well known examples include the heightened immune responses which infrequently occur to a fraction of the people after repeated exposures to toluene diisocyanate or latex. MDH is unable to derive HRVs that would be protective of all sensitized individuals.

Inhalation Health Benchmarks Differ for Cancer vs. Non-Cancer Effects

Regulatory scientists use very different approaches to develop inhalation health benchmarks for protection from cancer vs. non-cancer effects. Some chemicals have been shown to cause both cancer and non-cancer effects. In most of these cases, inhalation health benchmarks derived for cancer protection are lower concentrations than those derived for protection from other effects. The inhalation health benchmark that is more protective, based either on cancer or noncancer effects, is used.

Exposure to Air Toxics

With respect to a number of volatile organic compounds found in outdoor ambient air, such as benzene, personal exposures (i.e., the actual chemical concentrations a person breathes in daily life) are typically influenced to a large extent by what he or she does during the day, such as driving in their car, pumping gas, smoking or living around environmental tobacco smoke, working in some occupations, and using consumer products in the home.

Recent efforts to identify priority toxic air pollutants have assessed risks of breathing a number of chemicals in air. Breathing the air is only one way that people are exposed to chemicals. Health effects may also result from chemical exposures due to eating food, drinking beverages, and contacting products and other chemicals on the skin. The environmental and human health impacts of air toxics that persist and accumulate in the environment and the food chain, such as mercury, dioxin, and certain pesticides, have not been included in these assessments, but are discussed in [Appendix F](#).

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