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RISK ASSESSMENT REPORT

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1.0 EXECUTIVE SUMMARY

Background

Exposure to air toxics in urban areas may be of significant concern because people and sources of emissions are concentrated in the same geographic area. Studies show that levels of some air toxics tend to be similar across the country, while others can vary significantly from one location to another. Understanding which air toxics in an urban area contribute the most to potential health risks can provide valuable information in developing strategies to reduce these risks. The Detroit Air Toxics Initiative (DATI) project was initiated to gather this information for the Detroit area, and use it to help reduce exposure to air toxics to people living in this area.

The DATI project consists of two components: one to assess the health risks from exposure to air toxics in the Detroit area, and the other to fund projects to help reduce identified risks. A stakeholder group consisting of representatives from community and environmental based groups, federal, state and local government, industry and academia is providing assistance with the project. This report provides the background, methodology, and results for the risk assessment phase of the project.

Methods

The DATI risk assessment used air toxics monitoring data collected in the Detroit area to characterize exposures from toxic chemicals present in the ambient air. The data came from an intensive one year air toxics monitoring study that ran from April 2001 through April 2002. Monitoring data on over 200 compounds was evaluated, including metals, volatile organic compounds, semi-volatile organic compounds, and carbonyl compounds. Monitoring occurred at seven Detroit area sites, including six located in Wayne County and one in Oakland County. In addition, data from two additional sites were analyzed to provide comparisons of Detroit to other areas in Michigan. These included a site in Washtenaw County (Ypsilanti) that provides data from a smaller urban area located in a predominantly upwind direction from Detroit, and a site located in rural central Michigan near Houghton Lake.

Only exposures via inhalation were evaluated as part of the risk assessment. Other routes of exposure such as consumption of contaminated food or water, dermal exposures, and other exposures that may occur from deposition of air toxics to land and water were not included in this assessment.

Cancer, chronic non-cancer, and acute effects were evaluated as part of the DATI project. In general, risk assessment guidance developed by the U.S. Environmental Protection Agency, or methodologies for implementation of Michigan Department of Environmental Quality air toxic programs, were used to evaluate health risks from exposure to the monitored levels of air toxics.

Results

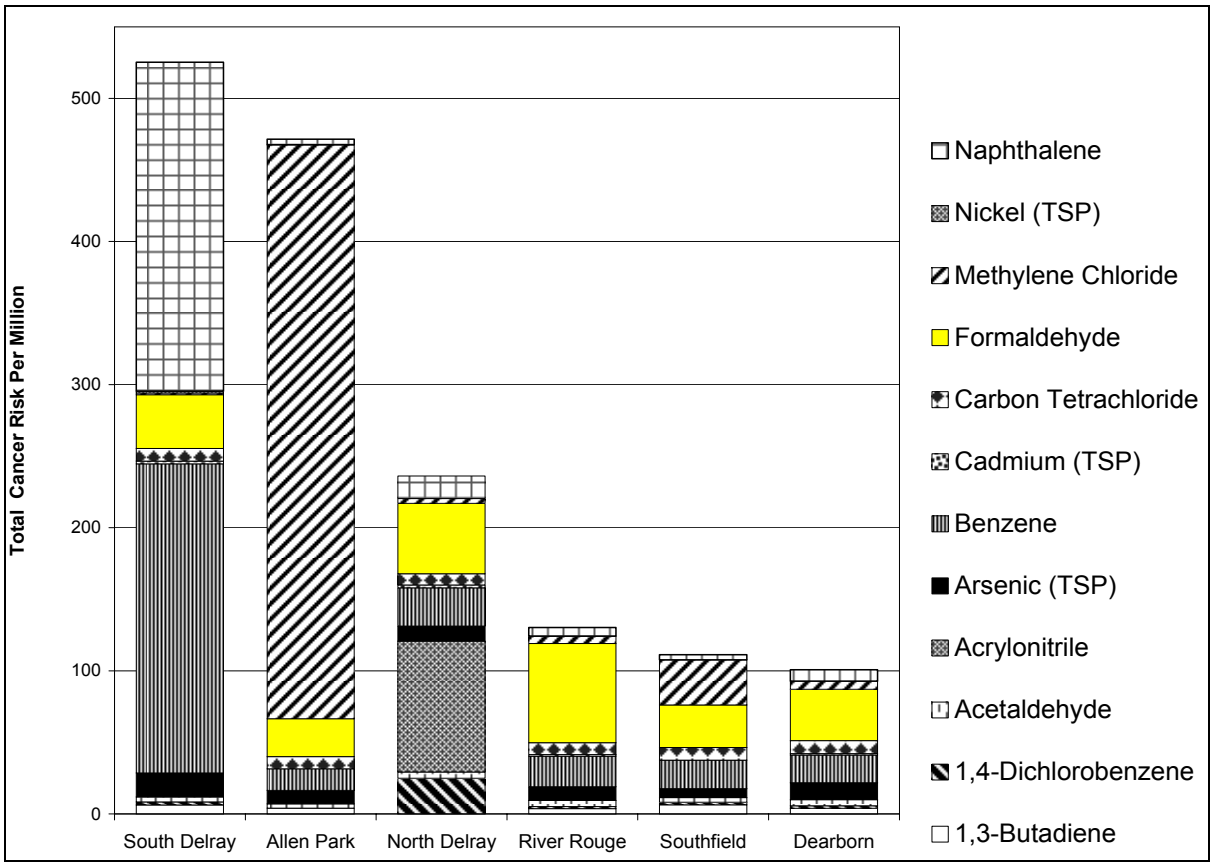
Cancer Risk: The ambient air levels of 12 chemicals were associated with increased cancer risks of 1×10^{-6} (1 in one million) or higher at one or more monitoring site. These 12 chemicals and their highest estimated risk at any one site are shown in **Table ES-1**:

Table ES-1: Highest Cancer Risk at any Site for Chemicals Associated with Risks Greater than 1 X 10^{-6}

INCREASED LIFETIME CANCER RISK	CHEMICALS
100 to 400 in one million	methylene chloride naphthalene benzene
10 to 100 in one million	acrylonitrile formaldehyde 1,4-dichlorobenzene arsenic
1 to 10 in one million	carbon tetrachloride 1,3-butadiene acetaldehyde cadmium nickel

Additive risks for the 12 carcinogens identified as having an individual risk > 1 X 10^{-6} varied across the sites. The additive cancer risk estimates for each site are shown in **Figure ES-1.**

Figure ES-1: Additive Cancer Risk by Site*



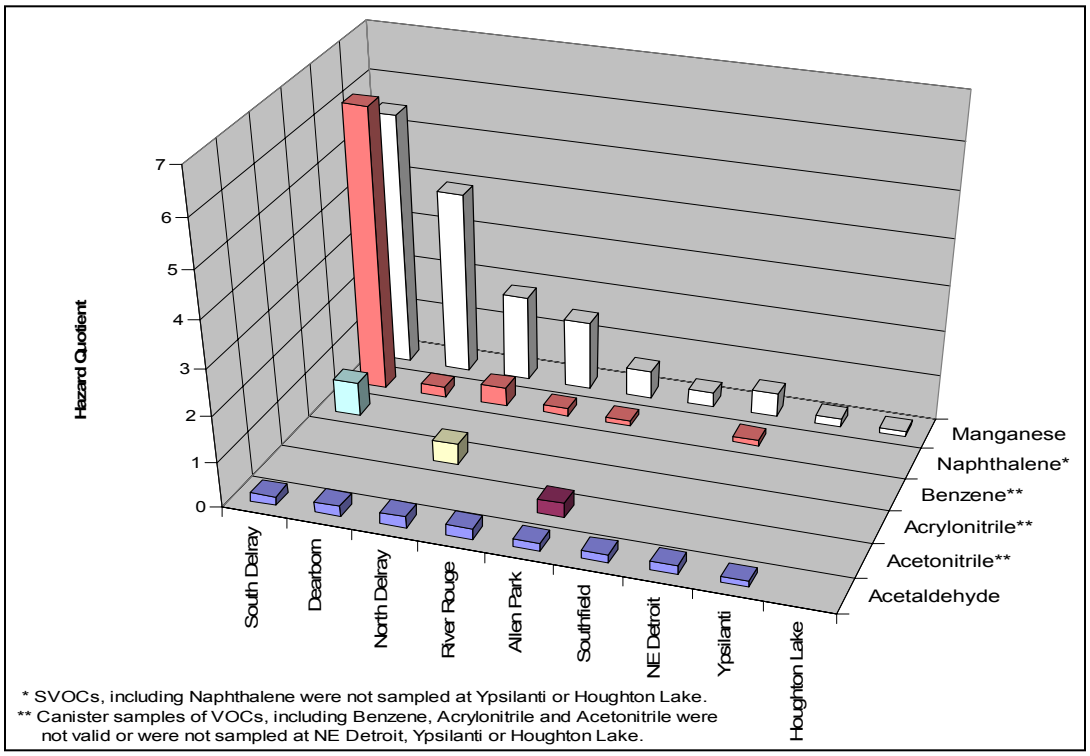
*The high-end estimated cancer risk is the incremental lifetime (70 years) risk by inhalation to the individual. The estimates of cancer risks should not be viewed as actual cancer cases resulting from air pollution but as upper bound estimates so the agency can prioritize its efforts to reduce exposures. It should also be noted that these risk estimates are based on 2001-2002 monitoring data, and the unusually high levels of methylene chloride at Allen Park and naphthalene and benzene at South Delray during that period have not persisted in subsequent monitoring.

Within the Detroit area, the additive risks varied between sites, with about a five fold difference between the highest site and the lowest site. Additive cancer risks are not shown for the sites located in Northeast Detroit, Ypsilanti, and Houghton Lake since a less complete set of air toxics were measured at these sites.

Elevated cancer risks from diesel particulate matter (DPM) were also found as part of this study, however, these risks were not included in the above estimates and comparisons due to the greater uncertainty associated with determining DPM ambient concentrations, estimating risks, and the limited number of sites with surrogate monitoring data. Based on source apportionment modeling, and using monitored levels of elemental carbon (EC), $PM_{2.5}$, and other compounds, DPM concentrations were estimated to be approximately 1-2 $\mu g/m^3$ DPM for the Allen Park and Southfield sites. Although these estimated values are relatively uncertain, they serve to provide a general sense of the contribution DPM may add to the cancer risk from air toxics in the Detroit area. This concentration range resulted in an estimated increased cancer risk in the range of approximately 300 to 600 X 10^{-6} associated with the estimated levels at the Detroit sites. These estimates are consistent with USEPA's roughly estimated lifetime cancer risk of 10 to 1000 X 10^{-6} associated with diesel emissions in the U.S. These estimated ranges of cancer risk are considered to have significant uncertainty. However, they suggest that diesel emissions may be a significant risk driver in the context of the total cancer risks estimated in this report for the other Detroit-area air toxics.

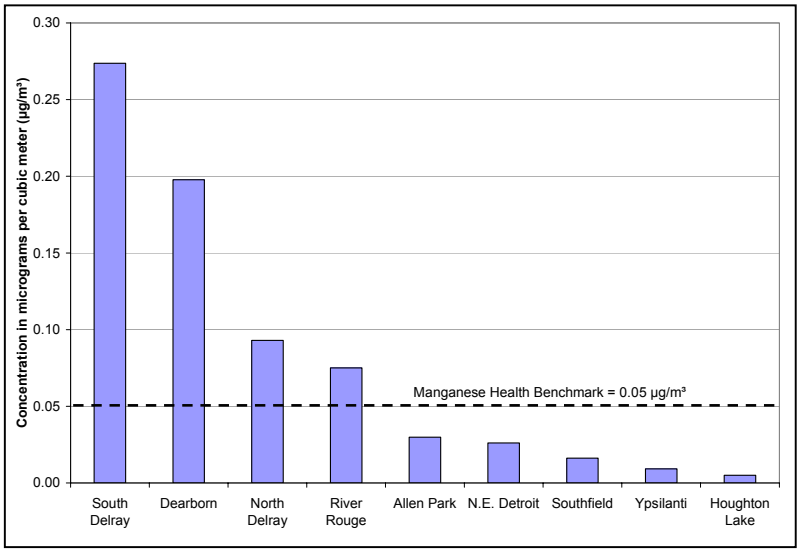
Non-Cancer Effects: With regard to non-cancer effects, monitored levels of six compounds were found to be present at levels greater than one tenth of the chronic health protective benchmark value, indicating a hazard quotient (HQ) > 0.1. These six compounds included acetaldehyde, acetonitrile, acrylonitrile, benzene, manganese, and naphthalene. Only two of these compounds, manganese and naphthalene exceeded their health protective benchmark values (HQ > 1) at certain sites. **Figure ES-2** shows this information.

Figure ES-2: HQs by Chemical and Site



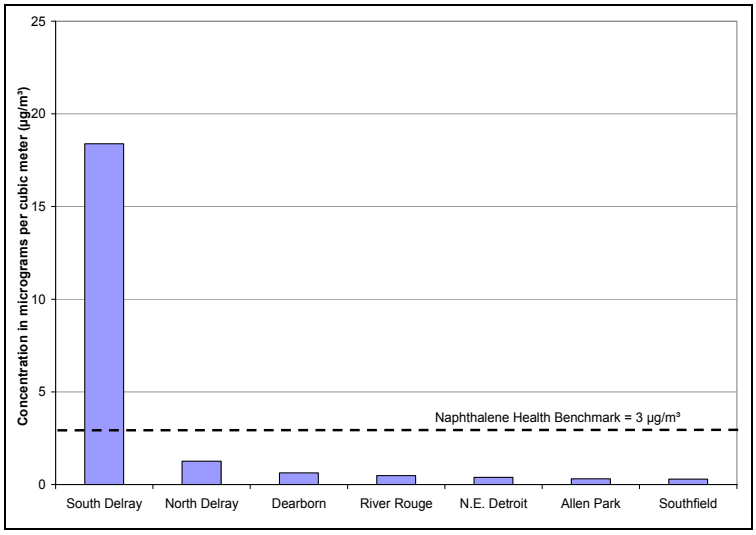
As shown in **Figure ES-3**, annual average manganese concentrations at four Detroit sites (South Delray, Dearborn, North Delray, and River Rouge) were 1.5 to 5.5 times higher than the chronic health benchmark value of 0.05 µg/m^3. At high enough levels, exposure to manganese can cause neurological effects such as impairment of fine motor skills and coordination.

Figure ES-3: Annual Average Manganese Air Concentrations



While the monitored levels of manganese were greater than the chronic health benchmark value at these four sites, this value is set significantly below actual exposure concentrations known to cause adverse effects in humans. Setting a health benchmark value in this manner is done to help ensure that the most sensitive members of the population (such as the sick, young, and elderly) will be protected for a lifetime of exposure to manganese, when data on effects are only available for healthy adult workers exposed for just a portion of their lifetime. This reduction in the margin of safety between the actual effect levels of manganese and the chronic health benchmark value is a concern. This concern is elevated further when considering the suggestive evidence in experimental animals for manganese accumulation in the brain, even at relatively low, environmentally relevant exposures. For sites where historical data are available, annual averages consistently above the benchmark are also a concern.

Figure ES-4: Annual Average Naphthalene Air Concentrations



The annual average concentration of naphthalene exceeded the chronic health protective benchmark value at only one site. That site was South Delray, where the monitored level was about six times higher than the health protective benchmark value. **Figure ES-4** shows the naphthalene levels at each site in comparison to the health protective benchmark value.

Exposure to naphthalene by inhalation at high enough concentrations causes respiratory effects. As with manganese, the health protective benchmark for naphthalene is set below the level at which adverse health effects are observed in order to provide a margin of safety. The highest naphthalene levels observed in the Detroit area are a cause for concern because they indicate a

decrease in this margin of safety, although they are still below the levels at which effects have been observed in scientific studies.

Acute Non-Cancer Effects: With regard to acute non-cancer health effects, only methylene chloride had any monitored levels higher than the acute health reference level. A total of three 24-hour monitored levels of methylene chloride exceeded the acute health reference level by a factor of 6 or less at Allen Park during the period of April 2001 to April 2002. However, further monitoring later in 2002 and in 2003 at the same location showed levels well below the health reference level. Benzene approached but did not exceed the health reference level at South Delray. A lack of continuous daily monitoring at most sites makes it difficult to draw definitive conclusions with regards to acute health effects. A lack of health benchmarks for the majority of monitored chemicals also adds to this uncertainty.

Other Pollutants: Acrolein, which was not sampled and analyzed as part of the intensive one-year monitoring study conducted by the MDEQ, was identified as a compound that could pose potentially significant inhalation risks. This finding for acrolein was based upon preliminary monitoring data at one site in Detroit from an ongoing USEPA study[1], as well as modeled impacts from another USEPA study.

Comparison of Detroit to Other Areas: The Detroit-area monitoring data were compared to the findings of other air toxics monitoring initiatives for other cities in the U.S. The comparison indicates that:

a) the pollutants creating the greatest risks vary across the cities studied;
b) the high levels of methylene chloride, benzene, and manganese noted above are generally relatively high in comparison to the other cities;
c) some studies reported higher levels of the compounds of greatest concern in Detroit, some were comparable, and others were lower; and,
d) there was a lot of variability in the levels of some of the compounds of concern across the Detroit-area sites, while other cities had more consistency across sites.

Uncertainty: Areas of uncertainty exist in all of the primary steps of the risk assessment – monitoring data collection, exposure assessment, toxicity assessment, and risk characterization. Substantial uncertainties are present, despite the application of state-of-the-art monitoring methods, inclusion of the air toxics of highest interest, a well-designed and extensive monitoring protocol, and the use of the best available health protective benchmarks. Although not fully quantified, several areas of uncertainty have been described with regard to the conservatism or lack of conservatism imparted upon the risk assessment.

Conclusion: The risk assessment for the DATI started with monitoring data for over 200 chemicals. From this list of chemicals, 13 chemicals were identified as the highest concern. In addition to these 13 air toxics, this assessment also indicates that diesel exhaust may be an important pollutant to focus on for mitigation of air toxics health risks. Additional data from other monitoring initiatives suggest that acrolein is also an important air toxic to consider.

[1] Acrolein information based on actual monitored data in Detroit as part of EPA's Detroit Exposure Aerosol Research Study (DEARS) is discussed in **Section 11.5**.

2.0 INTRODUCTION AND OVERVIEW

2.1 Purpose of this Report

The purpose of the DATI report is to provide the background, methodology, and results for the risk assessment from exposure to toxic air pollutants ("air toxics") in the Detroit area that was done as part of the DATI project. The report also provides comparisons of the monitored and modeled levels of air toxics in Detroit with other areas of the country, to help put in perspective the relative magnitude of air toxics in Detroit compared to these areas. **Appendix A** contains the acronyms and their definitions utilized in this report.

2.2 Background on the Detroit Air Toxics Initiative

The DATI is a project initiated by the Michigan Department of Environmental Quality (MDEQ), Air Quality Division (AQD), and funded through the U.S. Environmental Protection Agency's (USEPA's) Fiscal Year 2003 Community Assistance and Risk Reduction Initiative. This initiative includes two components: one to assess the health risks from exposure to air toxics in the Detroit area, and the other to fund projects to help reduce identified risks. This document details the findings of the risk assessment phase of the initiative. In addition to this technical document that provides the details of the risk assessment methodologies and results, a Technical Summary and Public Summary of the risk assessment results have been prepared.

The risk assessment performed for the DATI has focused primarily on air toxics monitoring data from the Detroit Pilot Project to characterize exposures from toxic chemicals present in the ambient air. The Detroit Pilot Project was an intensive one-year air toxics monitoring study in the Detroit area that ran from April 2001 through April 2002. More than 200 compounds were analyzed including metals, volatile organic compounds (VOCs), semi-volatile organic compounds (SVOCs), and carbonyl compounds. Monitoring occurred at seven Detroit area sites, including six located in Wayne County and one in Oakland County. In addition, an upwind site was located in Washtenaw County. Although not formally part of the pilot project, air toxics measurements were also collected at a site located in rural central Michigan near Houghton Lake.

In addition to risks from exposure to air toxics in outdoor ambient air, increased health risks may result from many other sources such as exposures to toxic chemicals in the indoor air environment, diet, smoking, etc. The risk assessment performed for the DATI does not address any of these other sources of risks.

The risk reduction phase of the DATI is ongoing and will focus on identifying, prioritizing, and funding risk reduction activities. The results from the risk assessment phase of DATI, as provided in this document, will be used to help in the identification and prioritization of risk reduction activities that could be funded through the risk reduction phase of the initiative.

A stakeholder group consisting of representatives from community and environmental based groups, federal, state and local government, industry, and academia is also an important part of the DATI. The purpose of the stakeholder group has been to provide feedback on the design and development of the technical risk assessment document and the Public summary, as well as help to facilitate communication regarding the project to the local community. The stakeholder group will also be involved in identifying, prioritizing, and providing recommendations on funding potential risk reduction activities. A complete list of stakeholder members is provided in **Appendix B**.

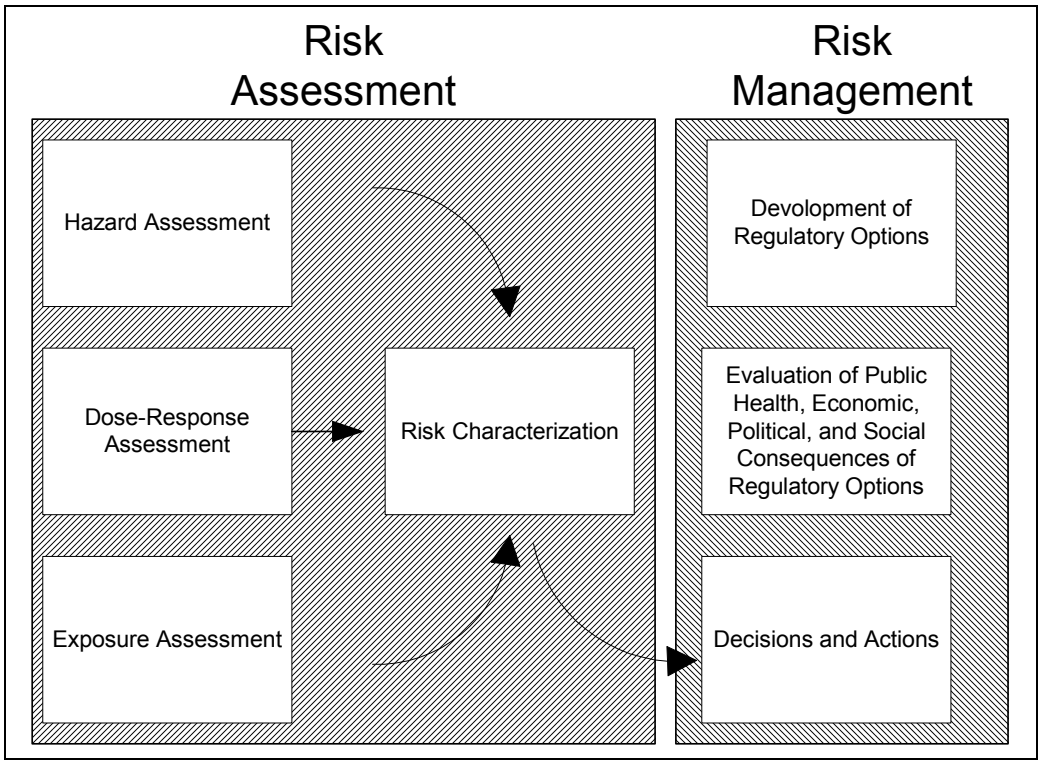
2.3. Overview of the Risk Assessment Process

The assessment of risk from exposure to chemicals in the environment has evolved over time. Several reports have been prepared by various scientific groups that have formed the basis for the current risk assessment process. Some of these include the following:

- "Risk Assessment in the Federal Government: Managing the Process" (Natural Resources Commission [NRC], 1983)
- "Science and Judgment in Risk Assessment" (NRC, 1994)
- "Risk Assessment and Risk Management in Regulatory Decision Making" (Commission on Risk Assessment and Risk Management, 1996)

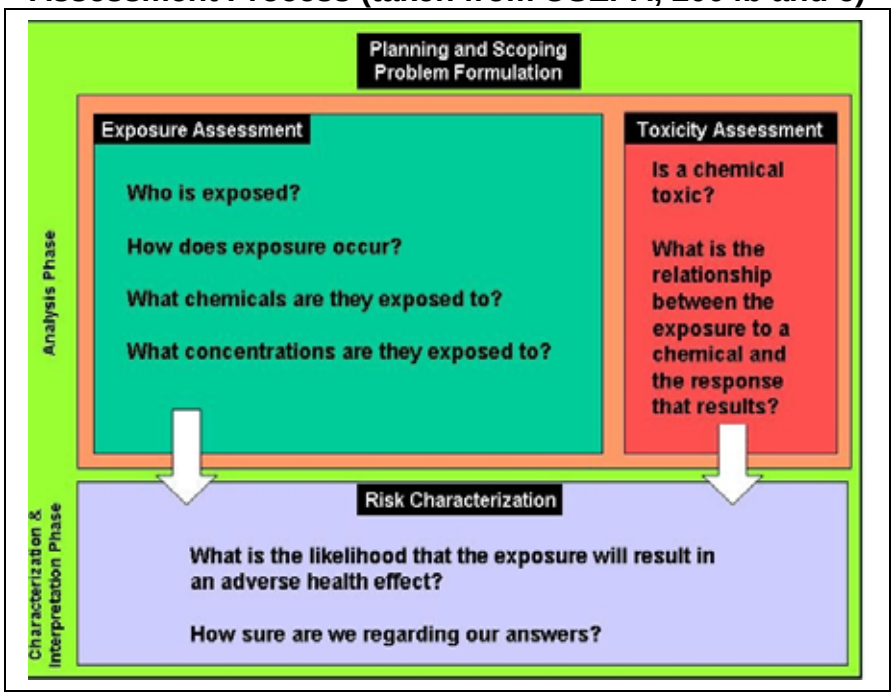
The definitions for "risk assessment" and "risk management" that were provided in the NRC (1983) report are still commonly used today. This NRC report defined risk assessment as "the characterization of the potential adverse health effects of human exposure to environmental hazards." The risk assessment process was recognized to contain four steps: hazard identification, dose-response assessment, exposure assessment, and risk characterization. Risk management was defined as "the process of evaluating alternative regulatory actions and selecting among them." Risk management integrates "the results of risk assessment with engineering data and with social, economic, and political concerns to reach a decision." **Figure 1** provides a diagram that illustrates the risk assessment process and its relationship with risk management.

Figure 1: Risk Assessment/Risk Management Paradigm (Adapted from NRC, 1983)



The USEPA has developed a number of risk assessment guidelines and documents that incorporate the principles of the earlier reports on risk assessment (NRC, 1983 and 1994). Two recent documents, "Air Toxics Risk Assessment Reference Library Volume 1 and Volume 2" (USEPA, 2004b and 2004c) are especially pertinent to the assessment of risks from air toxics.[2] **Figure 2** presents a schematic of the risk assessment process outlined in these documents. The risk assessment for the DATI presented in this report generally follows this process.

Figure 2: The General Air Toxics Health Risk Assessment Process (taken from USEPA, 2004b and c)



2.4 Planning, Scoping, and Problem Formulation

USEPA guidance includes "planning, scoping and problem formulation" as a significant and relatively new part of the air toxics risk assessment process, which should always be done for any systematic investigation (USEPA, 2004b). Included in this phase are a clear articulation of the assessment questions, a statement of the quantity and quality of data needed, determining what analyses will be needed and the assessors involved, and product and documentation requirements.

According to the USEPA guidance, "problem formulation" is a component that results in the production of a study-specific conceptual model and an analysis plan. The conceptual model identifies the physical boundaries of the study area, the local emission sources, the air toxics included in the risk assessment, a basic description of the exposed population, the routes of exposure, and the health outcomes to be evaluated. The analysis plan describes in detail how the elements of the conceptual model are going to be studied.

The initial availability of USEPA's Air Toxics Risk Assessment guidance (late 2003, in stages) came after the Detroit Pilot Project monitoring was completed and the risk assessment of the data was underway. Most of the USEPA's elements of planning, scoping, and problem formulation were already incorporated in the process before this guidance was available, although not as a discrete phase with detailed documentation as recommended by this USEPA guidance. The present risk assessment report provides a description of the risk assessment goals, key questions, process, data analysis, and risk characterization fulfilling the critical elements of the USEPA guidance.

[2] The Air Toxics Risk Assessment Reference Library is available at http://www.epa.gov/ttn/fera/risk_atra_main.html.

2.5 Key Questions

USEPA guidance in the Air Toxics Risk Assessment Reference Library recommends that risk assessments should address explicit key questions. The key questions to answer in the present risk assessment are:

1. What substances are the most significant compounds of potential concern (COPCs) (cancer, chronic and acute noncancer) of the air toxics monitored?

2. What is the magnitude of cancer risk posed by air toxics and how does it vary by site location?

3. What is the magnitude of chronic noncancer hazard posed by air toxics and how does it vary by site location?

4. What is the magnitude of acute noncancer hazard posed by air toxics and how does it vary by site location?

5. Do the levels of the key COPCs vary between sites?

6. Of the levels of total chromium, how much is hexavalent chromium (the carcinogenic form)?

7. Do the monitoring data and risk assessment help to scope the potential relative health significance of diesel emissions in the Detroit area?

8. How do the monitored levels of COPCs in Detroit compare to levels in other parts of the United States (U.S.)?

9. How do the health risks from air toxics in Detroit compare to other parts of the U.S.?

10. Are there specific sources or activities suggested to be a cause of specific COPCs?

11. Are there other data which suggest the DATI risk assessment did not identify all COPCs?

12. What are some of the important limitations or uncertainties of the DATI findings?

13. Does the risk assessment provide useful information to guide community-level actions which may significantly mitigate air toxics health risks?

14. Do the monitoring data and risk assessment provide a sufficient baseline to support future assessments?

15. Are there other issues not specifically addressed by key questions.

Additionally, it should be noted that there are other key issues pertaining to the pilot project (e.g., inter-laboratory comparisons; assessing the precision of laboratory analytical methods; seasonality of air toxics levels; sampling frequency issues; proximity and coverage of monitor placement) that are being addressed separately from the present risk assessment report (Heindorf, 2005).

2.6 Organization of Report

The remainder of this report (**Sections 3-14)** describes the Detroit Pilot Project monitoring study that forms the primary basis for the exposure assessment, the risk assessment methodology and results, and comparisons of monitored and modeled levels of air toxics with other areas in the country. A brief description of each remaining section is provided below:

Sections 3-6 describe the Detroit Pilot Project in detail, providing information on the study area (**Section 3**), the compounds monitored (**Section 4**), laboratory analytical methods (**Section 5**), and monitoring data results (**Section 6**).

Section 7 describes the exposure assessment phase of the risk assessment which is primarily based upon the monitoring data from the Detroit Pilot Project.

Section 8 describes the toxicity assessment which includes identifying the hazard or toxic effects of a chemical and characterizing the relationship between dose and response.

Section 9 characterizes the risk from exposure to air toxics based on the information from **Sections 7 and 8**.

Section 10 provides information on mercury and diesel. The potential health risks from these two pollutants could not be assessed in the same manner as the other air toxics in this report due to various data limitations and/or the unique properties of the compounds. However, these pollutants are of significant interest and concern to citizens in Michigan, so each was evaluated within the constraints of the available data in order to provide a better understanding of the relative magnitude of concern.

Section 11 provides the uncertainty analysis for the DATI risk assessment. This analysis includes uncertainties associated with the Detroit Pilot Project monitoring study (**Sections 3–6**), the exposure assessment (**Section 7**), the toxicity assessment (**Section 8**), and the risk characterization (**Section 9**).

Section 12 discusses the difficulties and issues associated with comparing risk assessment studies, and what may contribute to observed differences in results. This section also provides a comparison of health risks from air toxics in Wayne County (which largely comprises the Detroit area) with other counties in the U.S., based on USEPA's 1996 National Air Toxics Assessment (NATA).[3]

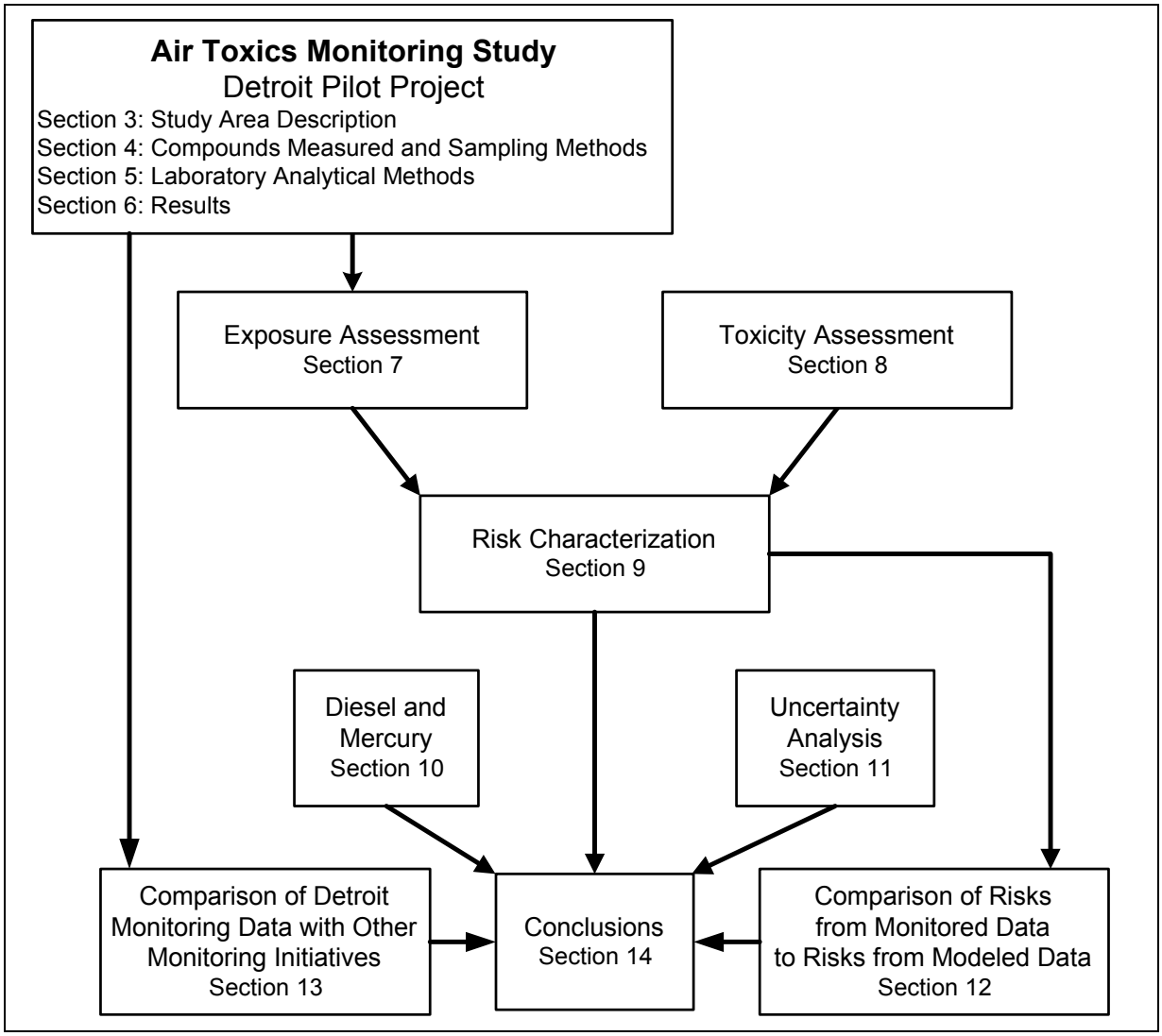
Section 13 provides a comparison between the monitored levels of air toxics in the Detroit area with those in other parts of the U.S.

Section 14 provides the conclusions for the report.

[3] Information on the USEPA 1996 NATA is available at http://www.epa.gov/ttn/atw/nata/nsata1.html.

Figure 3 provides a diagram which illustrates the organization of the report for **Sections 3–14**.

Figure 3: Organization of Report



3.0 STUDY AREA DESCRIPTION

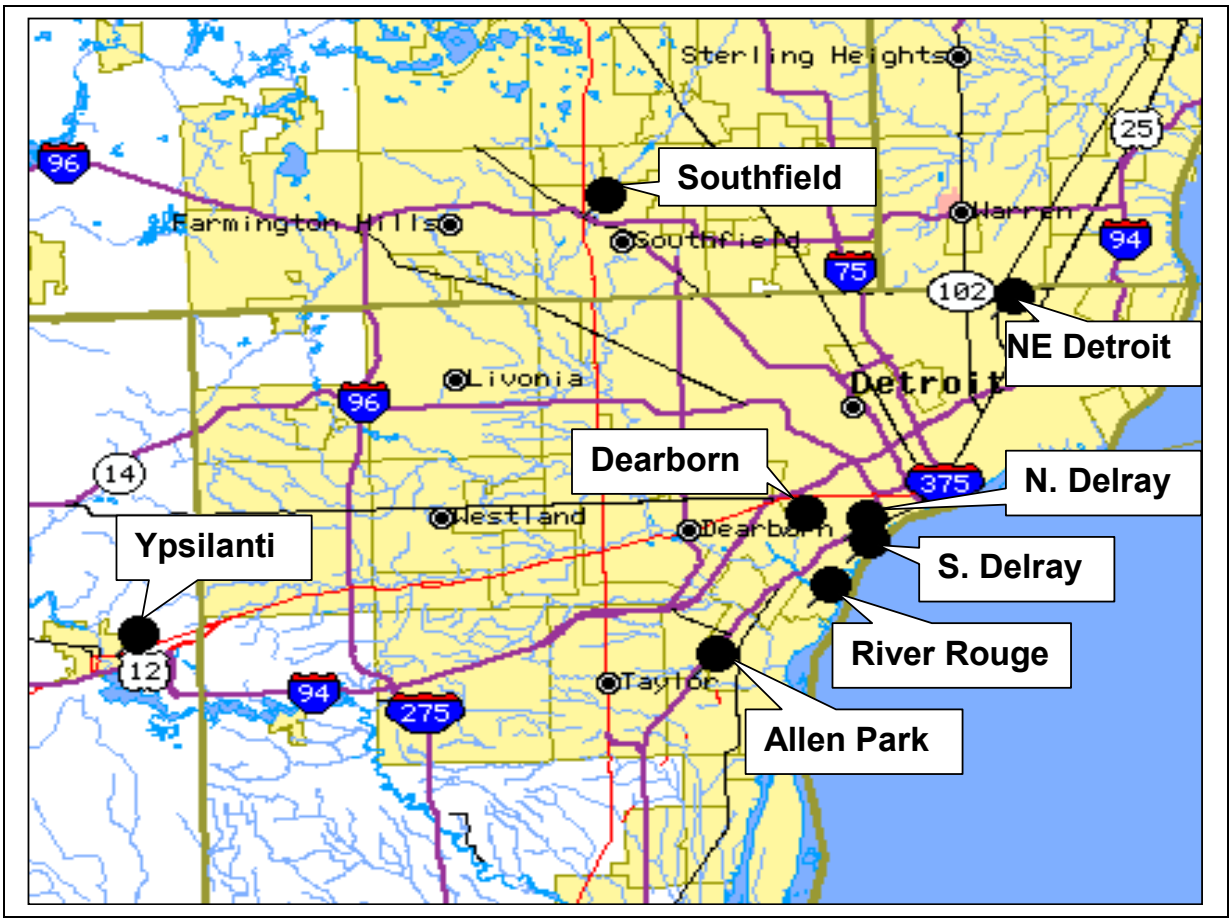
The ambient monitoring data from the Detroit Pilot Project has been used in this assessment to characterize the risks from air toxics in the Detroit area. The Detroit Pilot Project was part of a national effort initiated by USEPA to generate data about the spatial and temporal variability of air toxics in urban and rural areas, as well as small cities. Monitoring for air toxics occurred from April 2001 through April 2002 during the Detroit Pilot Project, and samples were collected at eight different sites. This risk assessment also includes analysis of data from a monitoring site located in Houghton Lake, Michigan to provide a comparison to risks at a rural "background" site. **Table 1** lists and provides further detail on each of the monitoring sites for this assessment.

Table 1: Detroit Pilot Project Monitoring Stations

SITE NAME	AIRSID	ADDRESS	LAT/ LONG	DESCRIPTION/ MONITORING OBJECTIVE
Sites Inside of Wayne County:				
Allen Park	261630001	14700 Goddard, Allen Park, 48101	42.2283; -83.2092	Mobile source/ Population-oriented
River Rouge	261630005	315 Genesee, River Rouge, 48218	42.2672; -83.1322	Industrial source/ Population-oriented
N. Delray	261630015	6921 W. Fort, Detroit, 48209	42.3042; -83.1072	Population-oriented
N.E. Detroit	261630019	11600 E. Seven Mile, Detroit, 48205	42.4308; -83.0083	Population-oriented
Dearborn	261630033	2842 Wyoming, Dearborn, 48120	42.3075; -83.1500	Industrial source/ Population-oriented
S. Delray	261630027	7701 W. Jefferson, Detroit, 48209	42.2922; -83.1069	Industrial Source
Sites Outside of Wayne County:				
Southfield	261250010	696/Lodge/Telegraph, Southfield, 48034	42.4903; -83.2800	Mobile Source
Ypsilanti	261610008	555 Towner Ave, Ypsilanti, 48198	42.2400; -83.5997	Urban Area/ Population-oriented
Houghton Lake	261130001	1769 S. Jeffs Road, Merritt, 49667	44.3106; -84.8919	Rural Area

Due to the very short time frame to implement the pilot program, most of the monitoring stations in the Detroit Pilot Project were selected from the existing network infrastructure in Southeast Michigan. This network infrastructure included sites monitoring for criteria pollutants, air toxics, or both. The pre-existing air toxics monitoring network is called the Michigan Toxics Air Monitoring Program (MITAMP). One new site was created at the intersection of Southfield and Telegraph in Oakland County. A map showing the locations is shown in **Figure 4**.

Figure 4: Detroit Pilot Project Air Monitoring Sites

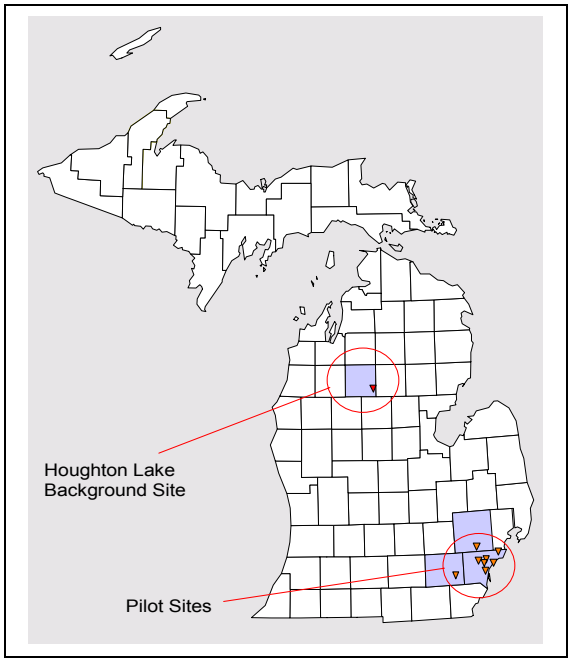


Though not a part of the Detroit Pilot Project, Houghton Lake (shown in **Figure 5**) provided data for background comparisons.

Most of the sites used in the pilot project are located in areas where people live, work, and play. In the Detroit area, residential communities are interspersed with many industrial sources so it is difficult to categorize the nature of each monitoring location with a single description. All of the monitoring stations and the instruments placed at them conform to the siting requirements set forth by USEPA in 40 Code of Federal Regulations (CFR) Part 58.

Following is a brief description of each monitoring site which includes their Aerometric Information System identification number (AIRS ID) and rationale for their inclusion as part of the Detroit Pilot Project.

Figure 5: Houghton Lake Background Site



Allen Park (AIRS ID 261630001): The Allen Park site is located near a residential neighborhood 300 feet south of Interstate 75 (I-75). Historically, this site has been used to detect impacts from mobile sources. Of all the population-oriented sites in the Detroit Metropolitan Statistical Area (MSA), the Allen Park monitoring station had the highest levels of PM_{10} (particulate matter with an aerodynamic diameter of less than [<] 10 microns [µm]), and monitoring data is not meeting the annual National Ambient Air Quality Standard (NAAQS) for fine particulate or $PM_{2.5}$ (PM with an aerodynamic diameter of < 2.5 µm).

River Rouge (AIRS ID 261630005): The River Rouge site, located at 315 Genesee in Detroit, has been part of the MITAMP network since 12/21/93. It is located in a residential neighborhood in Wayne County that is also impacted by I-75, industrial sources, and is within five miles of the N. Delray site. Emissions from a steel plant, which occupies a few miles along the river front, impact the site. Also near the site are drywall manufacturing companies, a waste water treatment plant (WWTP), a sewage incinerator, an asphalt plant, an oil refinery, coke batteries, coke oven by-product production facilities, various types of power generation plants, coal- and oil-fired combustion sources, paint shops, and assembly plants.

North Delray (AIRS ID 261630015): The N. Delray site, located at 6921 W. Fort Street in Detroit near Southwest High School, has been part of the MITAMP network since 1990, and serves as the long-term trend location for air toxics. It is located in a residential neighborhood in Wayne County that is impacted by industrial sources and is near I-75. The major sources near the site include two steel mills, a used oil reclamation plant, and various manufacturing facilities. The recent empowerment zone status achieved by the area is bringing in new industries and businesses. One of the largest and most recent additions is Arvin Meritor, a manufacturer of sunroofs. The Detroit WWTP is also close by. The N. Delray monitoring station is not meeting the annual $PM_{2.5}$ NAAQS.

Dearborn (AIRS ID 261630033): The Dearborn site, located within the city at 2842 Wyoming, was planned as a new addition to the MITAMP network, prior to the beginning of the Detroit Pilot Project. The site is located in a residential neighborhood with industrial impacts. An auto and steel manufacturing plant is located in close proximity to the monitoring station. The site lies between I-75 and I-94. Previous violations of the PM_{10} standard have occurred at this monitoring site and the station is not meeting the annual $PM_{2.5}$ NAAQS.

South Delray (AIRS ID 261630027): The S. Delray Site, also in Wayne County, is a source-oriented monitoring site located in the center of a highly industrialized area. The primary influences are from a nearby coke oven battery, steel mill, and a coal tar processing facility. The station is about 2.25 miles away from the Dearborn site and 0.75 miles away from

N. Delray. This is a middle scale monitoring station sited to capture maximum concentrations. It was included in the Detroit Pilot Project to provide information about the degree of heterogeneity of toxic air contaminants across a small scale.

Northeast Detroit (AIRS ID 261630019): The N.E. Detroit site at Osborn School represents a location downwind from the Detroit urban center city area, and is located in a residential neighborhood five miles west of I-94. The N.E. Detroit monitoring station does not meet the 8-hour ozone NAAQS.

Southfield (AIRS ID 261250010): The Southfield site, located in Oakland County, is near the intersection of three major highways and is further removed from the inner central city area. Due to concerns about the risks from mobile source emissions highlighted by the results of various studies, such as USEPA's Cumulative Exposure Project and NATA, it was considered desirable to add a second mobile source-oriented site to the Detroit Pilot Project. Although the Allen Park site is close to I-75, traffic pattern analysis indicated that other locations presented an even greater potential concentration of air toxics resulting from mobile source emissions.

Ypsilanti (AIRS ID 261610008): The Ypsilanti site is located in Washtenaw County at 555 Towner Avenue, Ypsilanti. This site is part of the MITAMP monitoring network, and was selected for this network because the asthma incidence rate in this zip code is substantially higher than elsewhere in Washtenaw County and higher than the overall incidence rates in Michigan and the U.S. (Washtenaw County Public Health, 1997). The Ypsilanti site served as an upwind site for the Detroit Pilot Project, and was intended to provide comparative measurements within an urban area adjacent to the Detroit MSA.

Houghton Lake Station (AIRS ID 261130001): The Houghton Lake station is located in Missaukee County in north central Michigan's Lower Peninsula. Background levels of air toxics have been collected at the station since 1998. Primary industries in the area include year-round tourism (boating, fishing, hunting, and snowmobiling), as well as Christmas tree farming. The county is sparsely populated, but attracts many tourists as it is a prime recreational area containing many lakes, rivers, and streams. The station is located at a deer research facility five miles west of U.S. Route 27 and 1.5 miles north of M-55. Though not located close to the site, oil and natural gas production occurs in counties to the south and north, as Michigan is the nation's 4th largest oil and gas producing state.

The relative distances between the stations are shown in **Table 2**. N. Delray, S. Delray, River Rouge, and Dearborn are all within five miles of each other, yet they show distinctly different patterns in the profiles of some toxic air compounds. **Table 2** also shows the total population residing within one mile of each monitoring station.

Table 2: Distances* Between Detroit Pilot Project Sites (as driven)

SITE 1 NAME & ADDRESS	SITE 2 NAME & ADDRESS	DISTANCE IN MILES	POPULATION WITHIN 1 MILE RADIUS OF SITE 1**
Southfield at 696/Lodge/Telegraph Rd.	Houghton Lake, 1769 S Jeffs Rd	178.8	9,366
	Ypsilanti, 555 Towner Ave.	31.7	
	Allen Park, 14700 Goddard	22.6	
	River Rouge, 315 Genesee	21.7	
	N. Delray, 6401 W. Fort St.	21.0	
	N.E. Detroit, 11600 E. Seven Mile	17.3	
	S. Delray, 7701 W. Jefferson	18.8	
	Dearborn, 2842 Wyoming St.	16.5	
Houghton Lake, 1769 S. Jeffs Rd.	Ypsilanti, 555 Towner Ave.	192.2	88
	Allen Park, 14700 Goddard	212.4	
	River Rouge, 315 Genesee	207.2	
	N. Delray, 6401 W. Fort St.	201.5	
	N.E. Detroit, 11600 E. Seven Mile	194.8	
	S. Delray, 7701 W. Jefferson	202.7	
	Dearborn, 2842 Wyoming St.	202.6	
Ypsilanti 555 Towner Ave.	Allen Park, 14700 Goddard	25.6	13,008
	River Rouge, 315 Genesee	30.3	
	N. Delray, 6401 W. Fort St.	31.8	
	N.E. Detroit, 11600 E. Seven Mile	42.0	
	S. Delray, 7701 W. Jefferson	31.8	
	Dearborn, 2842 Wyoming St.	29.9	
Allen Park, 14700 Goddard	River Rouge, 315 Genesee	6.8	14,822
	N. Delray, 6401 W. Fort St.	8.6	
	N.E. Detroit, 11600 E. Seven Mile	21.8	
	S. Delray, 7701 W. Jefferson	7.8	
	Dearborn, 2842 Wyoming St.	7.6	
River Rouge, 315 Genesee	N. Delray, 6401 W. Fort St.	3.6	13,275
	N.E. Detroit, 11600 E. Seven Mile	17.1	
	S. Delray, 7701 W. Jefferson	2.4	
	Dearborn, 2842 Wyoming St.	4.3	
N. Delray 6401 W. Fort St.	N.E. Detroit, 11600 E. Seven Mile	13.5	16,298
	S. Delray, 7701 W. Jefferson	1.4	
	Dearborn, 2842 Wyoming St.	3.2	
N.E. Detroit, 11600 E. Seven Mile	S. Delray, 7701 W. Jefferson	14.0	19.224
	Dearborn, 2842 Wyoming St.	13.9	
S. Delray, 7701 W. Jefferson	Dearborn, 2842 Wyoming St.	2.8	3,422
Dearborn, 2842 Wyoming St.			11,517

* Distances determined by "Yahoo Get Local Maps!"

** U.S. Census, 2000 (Population numbers include estimates for census tracts that fall partially within the 1 mile radius.)

The potentially significant point, area, and mobile air emission sources within <u>four miles</u> of each monitor are listed in **Table 3**.

Table 3: Significant Emission Sources Within Four Miles of Monitoring Sites

SITE (AIRS ID)	POINT AND AREA SOURCES	MOBILE SOURCES
Houghton Lake (261130001)	Fire places/wood stoves, Christmas tree farming, oil and gas production	U.S.-127, boating, snowmobiling
Southfield (261250010)	Paint Manufacturing, metal heat treating, machine shop, auto paint shop, asphalt, ready-mixed concrete	I-696, Telegraph, and Lodge
Ypsilanti (261610008)	Equipment manufacturing, WWTP, commercial printing, plastic products, power generation plants	I-94
Allen Park (261630001)	Bulk petroleum stations, refuse services, quarry, metal fabrication, chemical manufacturing/processing, power generation plants, plastic resin manufacturing	I-75
River Rouge (261630005)	Steel plant, drywall manufacturing, WWTP, sewerage incinerator, asphalt plant, oil refinery, coke batteries, coke-by-product production facility, power generation plants, coal and oil fired combustion, paint shops, assembly plants (heavy industrial)	I-75
N. Delray (261630015)	2 steel mills, used oil reclamation plant, asphalt plant, oil refinery, coke batteries, coke-by-product production facility, WWTP, sunroof manufacturer, power generation plants (heavy industrial)	I-75
N.E. Detroit (261630019)	Automotive manufacturing and stamping, chemical preparations, power generation plants, foundry, metal coating, refuse systems	I-94
S. Delray (261630027)	Coke battery , asphalt plant, oil refinery, coke-by-product production facility, steel mill, power generation plants, (heavy industrial)	I-75
Dearborn (261630033)	Auto & steel manufacturing, power generation plants, asphalt plant, oil refinery, coke batteries, coke-by-product production facility, (heavy industrial)	Between I-75 & I-94

4.0 COMPOUNDS MEASURED AND AMBIENT AIR SAMPLING METHODS

Over 200 compounds were measured as part of the Detroit Pilot Project. These included various VOCs, SVOCs, carbonyls, and trace metals as total suspended particulate (TSP). Because the Detroit Pilot Project was part of a national initiative, there were certain compounds that USEPA required be monitored by all participating pilot cities.

All of the pilot cities were required to measure a "core" list of 18 compounds that were identified by USEPA's Science Advisory Board and Air Toxics Steering Committee. The 18 core compounds included the following:

- benzene
- 1,3-butadiene
- carbon tetrachloride
- chloroform
- 1,2-dichloropropane
- methylene chloride
- tetrachloroethylene
- trichloroethylene
- vinyl chloride
- acetaldehyde
- formaldehyde
- beryllium and compounds
- cadmium and compounds
- chromium and compounds
- lead and compounds
- manganese and compounds
- nickel and compounds
- arsenic and compounds (only monitored by areas with infrastructure in place)

In developing the list of core compounds, USEPA first focused on the list of 188 hazardous air pollutants (HAPs) identified in Section 112(b) of the federal Clean Air Act (CAA). Recognizing that it would not be possible to measure all HAPs at every monitoring location, USEPA prioritized the list to focus on a subset of these pollutants which are thought to have the greatest impact on the public and the environment in urban areas. This subset consists of the 33 HAPs identified in USEPA's Integrated Urban Air Toxics Strategy commonly referred to as the "Urban HAP List."[4] USEPA recognized that a toxics monitoring network should address the full Urban HAP list, however, this list had to be further reduced because of practicality and resource limitations or lack of standard sampling and/or analytical methodology for a particular compound.

Mercury and dioxins, two compounds that have been of significant interest and concern in Michigan, were not included in USEPA's core list of compounds. Analysis of mercury requires special sampling and analytical equipment to measure all three forms of mercury (particulate [Hg(p)], elemental [Hg^0], and reactive gaseous mercury [RGM]) to adequately characterize atmospheric levels. In prioritizing the core list of compounds, USEPA also considered the fact that mercury is of low concern for direct inhalation from the air pathway. Consumption of mercury contaminated fish is the main exposure pathway for humans and wildlife, with atmospheric deposition considered the primary source of mercury resulting in this contamination. Although mercury was not on USEPA's core list of compounds, limited monitoring of this compound was included as part of the Detroit Pilot Project. This is discussed in more detail in **Section 10.1**. Dioxins were not included in the core list of compounds or the Detroit Pilot Project due to the very high costs associated with sampling and analyzing these compounds. USEPA also considered that some dioxin monitoring was occurring at the national level through its National Dioxin Air Monitoring Network (NDAMN) and for the near term (< five years) this data might be sufficient.

In addition to the core compounds and mercury, the MDEQ included measurements of hexavalent chromium (Cr^{+6}) and various polynuclear aromatic hydrocarbon (PAH) compounds as part of the Detroit Pilot Project. Hexavalent chromium is the most toxic oxidation state of

[4] The complete FR notice (64 Federal Register [FR] 38705) is available at http://www.epa.gov/ttn/atw/urban/fr19jy99.pdf.

chromium compounds. A 1992 study of ambient air in Windsor, Ontario found that Cr^{+6} levels were approximately 20 to 25% of the measured total chromium level (Bell and Hipfner, 1997). Data from California (South Coast Air Quality Management District [SCAQMD], 2000) indicates that Cr^{+6} is a very small percentage of total chromium (3% or less), unlike the value of 30% to 50% commonly used in various risk assessments, such as USEPA's NATA. MDEQ wanted to determine the Cr^{+6}/total chromium ratio in the Detroit area. PAHs were deemed to be important because of the various sources in the Detroit area.

Another special feature of the Detroit Pilot Project was the operation of an automated gas chromatograph (GC) equipped with a thermal desorption unit at the N.E. Detroit site to determine levels of ozone precursor compounds downwind from Detroit. Although the GC is normally in operation from June through September to coincide with the ozone monitoring season, it was operated for one full year to support the Detroit Pilot Project. Hourly measurements of VOCs were collected using the GC from April 2001 through January 2002 and March 2002. The collection of hourly VOC data was considered useful for helping assess the potential acute (short-term) health effects of these compounds. Since the primary purpose of the GC is to measure ozone precursors, the specific VOCs measured with this equipment differed from that of the standard VOCs that are measured at all other sites using method TO-15 (See **Section 5.0** on Laboratory Methods).

The sampling and analytical methodology for a particular compound often allows the measurement of a large number of other compounds of similar chemical nature at no extra cost. For example, the first nine core compounds listed previously all are considered VOCs, and are sampled and analyzed by the same methodology. In addition to these nine compounds, this methodology includes the measurement of about 40 other compounds. **Appendix C** contains a complete listing of all compounds measured as part of the Detroit Pilot Project, including those compounds measured with the automated GC at the N.E. Detroit site (a Photochemical Assessment Monitoring Station or PAMS).

The sampling frequency for a particular compound class varied among the sites due to budget limitations. In addition, not all parameters were measured at all sites. Hexavalent chromium was measured at four sites on a once every 12 day sampling schedule. VOCs, carbonyl, trace metals as TSP, and semi-volatile PAHs were measured at eight urban locations for one year. **Table 4** summarizes the parameters measured in the pilot project by site as well as the sampling frequencies for each.

Table 4: Sampling Frequencies by Parameter- Detroit Pilot Project

Site Name and AIRS ID	VOC Canisters	Carbonyls	TSP Metals	Cr +6	PAHs	Wind Speed	Wind Direction	Temp-erature	Barom. Pressure
Allen Park 261630001	1 in 12	1 in 6	1 in 6	1 in 12*	1 in 12	hourly	hourly	hourly	hourly
Dearborn 261630033	1 in 1**	1 in 1**	1 in 6	1 in 12	1 in 6	hourly	hourly	hourly	hourly
Houghton Lake 261130001	1 in 12***	1 in 6	1 in 12			hourly	hourly	hourly	hourly
N. Delray 261630015	1 in 12	1 in 12	1 in 6****		1 in 12	hourly	hourly	hourly	hourly
NE Detroit 261630019		1 in 6	1 in 6		1 in 12	hourly	hourly	hourly	hourly
River Rouge 261630005	1 in 6	1 in 12	1 in 6	1 in 12	1 in 12*				
S. Delray 261630027	1 in 6	1 in 6	1 in 6		1 in 6				
Southfield 261250010	1 in 6	1 in 6	1 in 6	1 in 12	1 in 6	hourly	hourly	hourly	
Ypsilanti 261610008	1 in 12***	1 in 12	1 in 12			hourly	hourly	hourly	hourly

* Co-located 1 in 12
** Co-located 1 in 3
*** VOC monitoring data at Houghton Lake and Ypsilanti were invalidated because of sampling errors.
**** Co-located 1 in 6

Data collected 4/19/2001 through 4/30/2002

As shown in **Table 4**, the sampling frequency varied by site. Daily VOC and carbonyl samples were collected by the primary monitor at Dearborn to assess the day to day variability in levels of air toxics. This data also supports analysis activities to determine the optimal sampling frequency. The co-located monitor at Dearborn operated on a once every three day sampling frequency to assess sources of variability. Sites deemed likely to experience elevated levels of VOCs, carbonyls, or PAHs sampled on a once every six day schedule, while the others conformed to the once every 12 day frequency. This was done to assess spatial and temporal variability at the sites likely to measure elevated concentrations and as a cost containment measure. Hexavalent chromium was measured on a once every 12 day schedule because this was exploratory field work to validate the method and compare Cr^{+6}/total chromium ratios found in Michigan to California values. Trace metals were collected at a 1 in 6 schedule at most sites, but the MITAMP sites retained the 1 in 12 day frequency. Hourly meteorological measurements were made at sites with overhead clearance that would support deployment of the meteorological tower.

The Detroit Pilot Project also included some special sampling that was focused on quality control issues dealing with a national air toxics monitoring network. Precision and accuracy of the measurements from such a network are important aspects to be considered when analyzing and comparing data. **Table 4** also shows the locations of sites receiving precision monitors. Unlike procedures normally used in the operation of an air toxics monitoring network, multiple stainless steel (SUMMA® brand) canisters (used to measure VOCs) or 2,4-diphenylhydrazine-treated (DNPH) cartridges (used to measure carbonyls) were not collected using a single sampler. Instead, for the purposes of the Detroit Pilot Project, two distinct samplers were deployed to various locations to provide an indication of inter-sampler precision. This was done because the data would be used to determine spatial variability across a monitoring network in an urban area. If the inter-sampler precision could not be assessed, it would be impossible to extrapolate the true variability caused by spatial differences. Multiple VOC or carbonyl samples from a single sampler, to assess intra-sampler variability through laboratory precision, could not be collected due to budgetary constraints. However, precision data of this nature is available through the historical MDEQ MITAMP database.

5.0 LABORATORY ANALYTICAL METHODS

Because laboratory tests are designed to exploit the different chemical and physical properties of each analyte, most methods provide a suite of measurements at the same cost that would be required for a single compound. Therefore, many additional measurements were included in the Detroit Pilot Project database beyond the core compounds identified by USEPA. The core compounds and the corresponding method used to measure them are shown in **Table 5**:

Table 5: Core Compounds, Classification, and Methods Used for Measurement

COMPOUND	CLASSIFICATION	METHOD
Benzene	VOC	TO-15
1,3-butadiene	VOC	TO-15
carbon tetrachloride	VOC	TO-15
Chloroform	VOC	TO-15
1,2-dichloropropane (propylene dichloride)	VOC	TO-15
Methylene chloride (dichloromethane)	VOC	TO-15
tetrachloroethylene (perchloroethylene, PCE)	VOC	TO-15
trichloroethylene, TCE	VOC	TO-15
vinyl chloride	VOC	TO-15
Acetaldehyde	Carbonyl	TO-11A
Formaldehyde	Carbonyl	TO-11A
Beryllium and compounds	Trace metals	IO-3.5 & 6020 (ICP/MS)
Cadmium and compounds	Trace metals	IO-3.5 & 6020 (ICP/MS)
chromium (total chromium)	Trace metals	IO-3.5 & 6020 (ICP/MS)
lead and compounds	Trace metals	IO-3.5 & 6020 (ICP/MS)
manganese and compounds	Trace metals	IO-3.5 & 6020 (ICP/MS)
nickel and compounds	Trace metals	IO-3.5 & 6020 (ICP/MS)
arsenic (monitored by areas with infrastructure currently in place)	Trace metals	IO-3.5 & 6020 (ICP/MS)

ICP/MS = inductively coupled plasma spectroscopy/mass spectrometry

VOCs were collected by pumping ambient air into evacuated 6 liter stainless steel SUMMA® canisters using a variety of samplers (a multi-port sampler built by MDEQ, Anderson AVOCs and Nu-tech samplers). The canisters were sent to MDEQ and/or the Eastern Research Group (ERG) laboratories as co-located or split pairs of samples. This experimental design allowed inter-laboratory precision, inter-sampler precision, and precision of the analytical method to be assessed. The laboratories both used USEPA method TO-15 to analyze the contents of the SUMMA® canisters within 30 days after sample collection. In this method, the contents of the canisters are humidified, sent through a preconcentration device, separated via a GC, and quantified using a mass spectrometer. Each laboratory cleaned its own canisters down to its own detection limit. Both laboratories used a maximum sample hold time of 30 days after collection.

Carbonyls were collected by pumping ambient air through potassium iodide treated scrubbers to remove ozone, which will scavenge carbonyl compounds. The air sample then passed through a cartridge filled with a silica gel packing material that was coated with 2,4-dinitrophenyl hydrazine. The samples were collected, cartridge tips were capped, and they were placed in foil lined envelopes. The samples for MDEQ were placed in glass jars containing charcoal and sent to the laboratory. The samples for ERG were placed in coolers, along with the other more perishable samples and sent via over night mail to the laboratory. Like the VOC experimental design, the cartridges were sent to the MDEQ and/or ERG as co-located or split pairs of samples.

Carbonyls were determined by the laboratories using USEPA method TO-11A where the 2,4-dinitrophenylhydrazine derivatives were extracted off of the silica cartridges using acetonitrile, concentrated, and analyzed using High Pressure Liquid Chromatography coupled with an ultraviolet detector at 360 nanometers (nm). All cartridges were extracted by the laboratories within 14 days of exposure.

Trace metal samples were collected as TSP matter by using high volume samplers fitted with glass fiber filters. Contrary to the other pilot cities, glass fiber filters were used to maintain consistency with MDEQ's historical database. One lot of glass fiber filters was used throughout the study. Separate high volume sampling devices were placed at the N. Delray site to determine inter-sampler precision. This site was selected because there was insufficient space at Dearborn and this site is within five miles from Dearborn. In addition, data from N. Delray has historically been used to determine precision for TSP. The co-located samples were collected at every sampling event exceeding the 10% precision requirement established by the National Air Toxics Trend Site (NATTS) requirements. The filters were placed into envelopes and mailed to MDEQ laboratory for analysis. The filters were digested with acid, concentrated, and analyzed by the MDEQ laboratory using either inductively coupled argon plasma spectroscopy or inductively coupled plasma spectroscopy/mass spectrometry (ICP/MS), depending on the element in question and the anticipated concentration range. The methods used by the laboratory were USEPA 6010B (ICP) and 6020 (ICP/MS).

Hexavalent chromium or Cr^{+6}, was collected by passing ambient air through ashless cellulose filters that were impregnated with sodium bicarbonate. They were collected, placed in sampling envelopes, placed on ice, and sent to ERG for analysis. A co-located sampler was placed at River Rouge to assess inter-sampler precision. The filters were analyzed by ERG using a modification of California's Air Resource Board (ARB) method MLD 039. The filters were extracted using deionized water via sonication, then analyzed using an ion chromatograph equipped with a post column derivatization module to form a diphenylcarbohydrazide complex. The derivatives were detected at 540 nm. The samples were analyzed by the laboratory within 30 days of collection.

PAHs/SVOCs were collected using USEPA method TO-13A with Anderson model PS-1 polyurethane foam samplers. The samplers were equipped with glass fiber filters to collect the particulate material and glass thimbles were filled with XAD-2® resin to capture volatile compounds. The glass thimbles and filters were placed into their protective housings after the completion of sampling, placed on ice and mailed to ERG. According to USEPA laboratory method 3542, both the filter and resin were extracted and concentrated. The extracts were analyzed by ERG using GC mass spectrometry (Method 8270).

6.0 MONITORING DATA RESULTS

6.1 Summary Statistics

As part of the national initiative, the data from the Detroit Pilot Project were analyzed and compared to data generated by the other pilot cities through a series of data analysis grants funded by USEPA. The results of the various data analysis projects are summarized in a series of reports and by presentations. In general, these findings are discussed on a national level, with selected details shown for each pilot city (Battelle Memorial Institute, 2003a, 2003b, 2003c; Battelle Memorial Institute & Sonoma Technology Inc. [STI], 2001; STI, 2004a, 2004b, 2004c, 2004d, 2004e, 2004f, 2004g, 2004h).

In a separate document, MDEQ is preparing a detailed analysis of only the Detroit-specific data (Heindorf, 2005). It provides site specific details, urban area summaries, discussion of ambient levels, seasonal trends, and site-to-site comparisons for the Detroit Pilot Project. The detection frequency, annual averages, 24-hour (hr) maximum values, and seasonal trends were determined on a pilot-wide basis and by site. In addition, the impact of various sources of variability on the data base, such as laboratory choice, individual monitor, laboratory method, etc., were assessed. Other specialized analyses such as the impact of sampling frequency on the calculation of annual averages, impact of weather patterns on the measured concentration, and sources of variability were addressed.

Some much generalized conclusions may be drawn from MDEQ's analysis of the Detroit Pilot Project data (ibid). The spatial profiles of carbonyls, VOCs, trace metals as TSP, and PAHs vary by compound. The locations where maximum concentrations and or maximum site averages were determined are not limited to one or two stations. Rather, the toxic compounds show much variability across the area and are highly dependent upon the parameter and station (see **Section 6.4** for more information).

Appendix D contains a summary of the monitoring data results from the Detroit Pilot Project including annual average concentrations, 24-hr maximum values, frequency of detection, etc. In calculating an annual average concentration, the method detection limit (MDL) divided by 2 (MDL/2) was substituted for any value reported as non-detect (ND). This issue is discussed in further detail in the following section.

6.2 Method Detection Limits, Non-Detects, and Data Treatment

In interpreting air monitoring data for risk assessment purposes, some important issues to consider include the MDL, ND values, and how this data is treated in the risk assessment process. The definition and use of MDLs are specified by the USEPA (USEPA, 1987):

> "The MDL is defined as the minimum concentration of a substance that can be measured and reported with 99% confidence that the analyte concentration is greater than zero and is determined from analysis of a sample in a given matrix containing the analyte."

In practice, the MDL is thought of as the lowest amount of a chemical that can reliably be observed (with 99% confidence) above the normal, random noise of an analytical instrument or method. MDLs are chemical-specific and instrument-specific and are determined by statistical treatment of multiple analyses of a standard sample. MDLs are determined by a laboratory at least once a year or whenever a substantial change, such as a repair to the instrumentation occurs. Depending on many factors, the MDL for a given chemical may vary from laboratory to

laboratory. Typically, levels of chemicals below the MDL are not quantified and are often referred to as < the MDL or ND.

In the Pilot City measurement guidelines, USEPA recommended that all data be reported from the laboratory, even measurements made that are below the MDL (USEPA, 2001b). This was suggested in order to gather additional "uncensored" data to help improve the annual average determinations and shed light on the quality of data at and below the MDL. For the Detroit Pilot Project, the MDEQ laboratory did not report data below the MDL, however, the ERG laboratory did.

In using air monitoring data as part of a risk assessment, values reported as < the MDL or ND can be problematic. Various procedures have been used in risk assessments to treat NDs, ranging from the assumption that the chemical is absent (i.e., the true concentration is zero) to the assumption that the chemical was present in a sample at a level infinitesimally beneath the MDL (i.e. very close to the MDL, and essentially equal to the MDL). A common method that is often used is to substitute ½ the MDL for ND values. That was the approach followed in the present risk assessment. It may be noted that USEPA (2004b) suggests that statisticians may reevaluate the data if the percentage of NDs of a compound at a monitoring location is between 15 and 90% and contributes significantly to risk. That additional step was not included in this assessment.

In the Air Toxics Risk Assessment Library (USEPA, 2004b and c), USEPA describes another type of detection limit available: the sample quantitation limit or SQL. According to USEPA, a quantitation limit is the lowest level at which a chemical can be accurately and reproducibly quantitated. Because SQLs take into account sample characteristics, sample preparation (e.g., dilution factors), and analytical adjustments, USEPA considers them to be the most reliable quantitation limit for evaluating ND situations, and recommends their use for risk assessment purposes. However, USEPA also states that if SQLs are not available, the MDL may be used as the quantitation limit, with the understanding that in some cases this will underestimate the SQL. In using monitoring data for risk assessments, USEPA also specifically recommends use of ½ the SQL for ND samples.

As previously discussed and recommended by the USEPA in the Pilot City measurement guidelines, ERG and MDEQ laboratories only reported MDLs, and thus SQLs were not available to use for risk assessment purposes. For the DATI risk assessment, therefore, a value of ½ the MDL was substituted for all ND values for determining annual average concentrations of a chemical.

6.3 Total Chromium and Hexavalent Chromium

One of the key risk assessment objectives of the Detroit Pilot Project and the present risk assessment was to evaluate the levels of total chromium and Cr^{+6} in the study area. This is important because Cr^{+6} is a known human inhalation carcinogen, which is also relatively high in potency. Other forms of chromium that may be present in emissions and ambient air samples are not recognized as carcinogens. If ambient air data or estimates are only available for total chromium, risk assessments may then require an assumption for the percentage of Cr^{+6} in the total chromium, and that can be a very crucial assumption in the overall risk assessment. For example, for the 1996 NATA, USEPA assumed that 34% of all atmospheric chromium was hexavalent, and the result was that Cr^{+6} was estimated to be one of the largest cancer risk drivers overall.

For the four sites where speciated chromium was measured, detectable levels occurred in 100% of the samples of total chromium, but only in 50% to 62% of the samples for Cr^{+6}. The

annual average concentration of total chromium and Cr^{+6} at each of the four sites was determined, with the value of MDL/2 substituted for the ND values. This information is shown in **Figures 6** and **7**.

Figure 6: Average TSP Chromium Concentrations

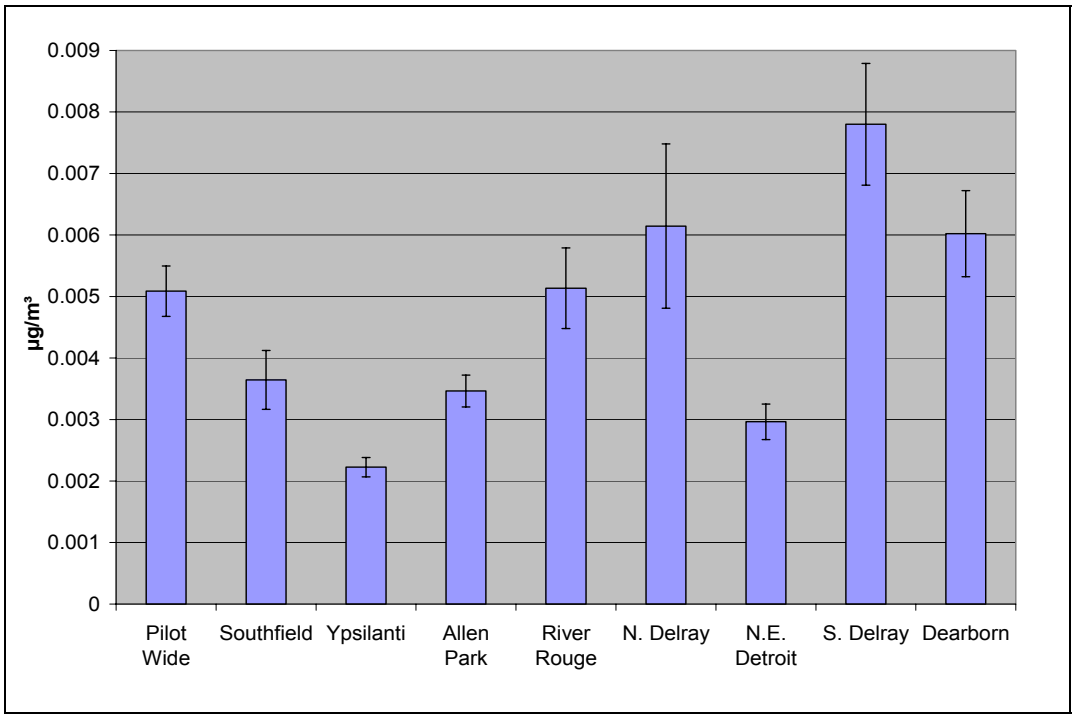
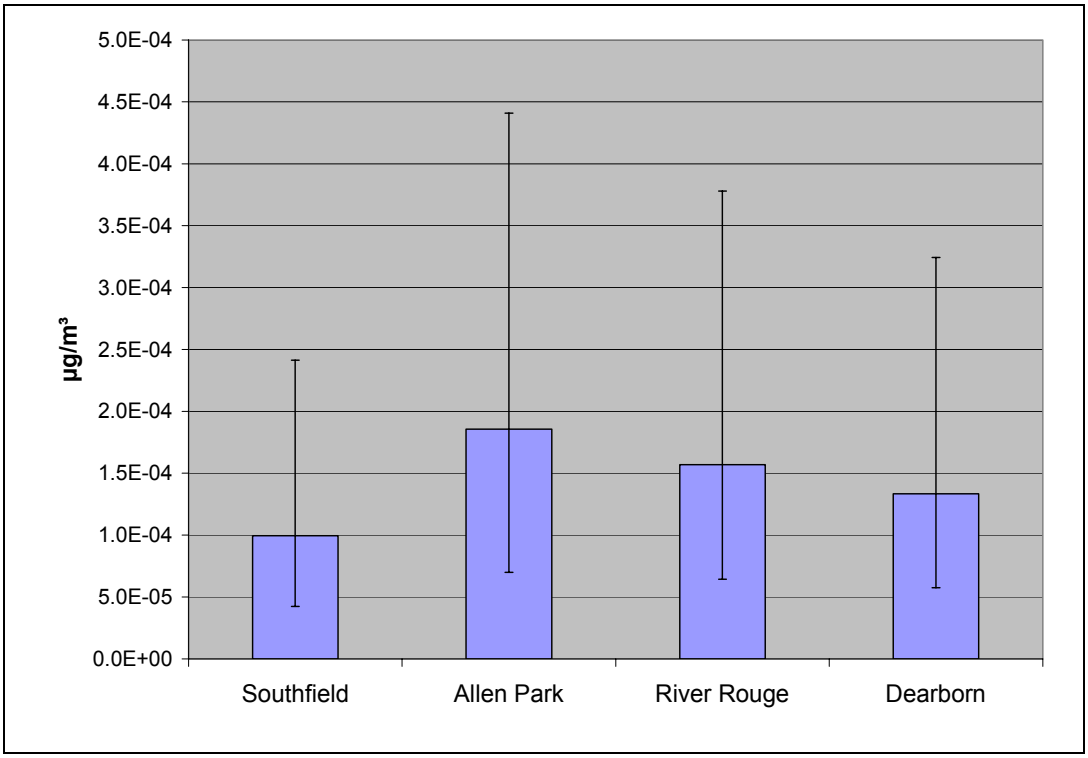


Figure 7: Average Hexavalent Chromium Concentrations



In the absence of Cr^{+6} monitoring data at the remaining sites, a surrogate approach was desired. The chosen approach was to determine the percentage of Cr^{+6} in total chromium at the sites for which these data are available. This percentage was then applied to the total chromium concentrations at the other sites to estimate the potential Cr^{+6} concentrations for those sites.

The ratio of Cr^{+6} to total chromium was determined by two methods. In the first case, all samples were included in the analysis, including those with reported ND values. In this case the MDL/2 was substituted for the ND value. In the second case, the ND Cr^{+6} data were excluded. For both cases, the percentage of Cr^{+6} in total chromium was determined for each sampling event, and these percentages were averaged for each site.

The first method, which included the ND values, resulted in the following percentages of Cr^{+6} in total chromium: 1.2% (Southfield); 1.3% (Allen Park); 0.77% (River Rouge); and 0.60% (Dearborn), with an overall average of 0.76%.

The second method, which excluded the ND values, resulted in slightly higher percentages. **Table 6** shows the average percentages of Cr^{+6} in total chromium for this method, and other related summary statistics. This approach was considered to be the most appropriate for the risk assessment of hexavalent chrome at non-monitored sites, because it is based only on measured data without any assumptions about the potential levels of Cr^{+6} that may have been present below the detection limit.

Table 6: Percentage of Hexavalent Chromium in Total Chromium
(Excluding NDs)

ARITHMETIC MEAN (average)	SOUTHFIELD	ALLEN PARK	RIVER ROUGE	DEARBORN
	2.41%	**2.05%**	**1.37%**	**0.96%**
Number of samples	16	18	17	16
90% Confidence limit on the mean	0.80	0.47	0.32	0.26
Standard Deviation	1.94	1.22	0.81	0.63

Figures 6 and **7**, and the above analysis, suggest that the levels of Cr^{+6}, total chromium, and their ratios, are site dependent. Linear regressions of total chromium versus Cr^{+6} did not yield good correlations. This may be due to site-specific influences from sources emitting varying percentages of total chromium, and the impact of meteorological conditions. It may be noted that the two sites (Allen Park and Southfield) with the higher Cr^{+6} ratios are mobile source-oriented sites, while the other two sites are relatively more residential with impacts from industrial sources.

6.4 Site-to-Site Comparisons of Measured Levels

As previously noted, a separate draft MDEQ report (Heindorf, 2005) provides a fairly detailed assessment of the site-to-site comparisons of the measured levels from the Detroit Pilot Project. That report includes an assessment of all substances monitored, which is important and insightful in a number of ways. However, the present report attempts to focus attention upon the specific air toxics which are estimated to pose the most substantial levels of risk. In **Section 9** of this report, a subset of 13 “COPCs” was identified, including 12 “cancer risk drivers” and a key noncarcinogen (manganese). These COPCs were established on the basis

of meeting certain risk or hazard-based criteria, which are described in **Sections 8** and **9**. The present discussion of site-to-site comparisons of monitored levels addresses only those 13 COPCs listed below (not in any particular order of priority):

- 1,3-butadiene
- 1,4-dichlorobenzene
- acetaldehyde
- acrylonitrile
- arsenic
- benzene
- cadmium
- carbon tetrachloride
- formaldehyde
- manganese
- methylene chloride
- naphthalene
- nickel

The annual average levels of these COPCs at each site were calculated and compared by analysis of variance testing. To determine which sites had means with statistically significant differences, Duncan's Multiple Range Test was performed. For this section, all of the following discussion of the site-to-site comparisons of levels refers to the annual average levels, not 24-hr measurements or any other averaging time.

Overall, most of the air toxics that were frequently detected in the Detroit area showed a high degree of spatial variability and the magnitude of the spatial differences varied by compound. As described in **Section 11.3**, most of the COPCs at sites with levels of potential concern had a high (90% or higher) or moderate (50 to 90%) detection rate; 1,4-dichlorobenzene was the only COPC with a relatively low detection rate (16 to 35%).

An attempt was made to assess the relative homogeneity and heterogeneity among the monitoring sites, including all VOC substances (not just the COPCs) based on the annual averaged levels. An analysis of variance and Duncan's Multiple Range test were run on the combined dataset to determine which compounds had similar means at all stations (homogeneity). Across all seven of the Detroit Pilot Project sites, each of the seven VOC COPCs (except 1,4-dichlorobenzene) had a distribution which was heterogeneous due to significant differences between two or more sites. 1,4-Dichlorobenzene levels were homogeneous across these sites.

Generally, the family of carbonyl compounds were very heterogeneous across sites, except for a relatively high consistency for Southfield and Allen Park (the only mobile source-oriented sites). The formaldehyde levels at N. Delray and River Rouge were significantly higher than the other sites. Acetaldehyde levels were relatively homogeneous (within a 4-fold range across all sites), although the level at the highest site (N. Delray) was significantly higher than the levels at the lowest four sites in the Detroit area including Ypsilanti.

Assessment of the VOCs also found significant site-to-site differences. The methylene chloride level at Allen Park was significantly elevated in comparison to all other sites. The benzene level at S. Delray was significantly higher than at the other sites. Removing the S. Delray benzene data from the dataset resulted in a finding that N. Delray was significantly higher than other sites. For carbon tetrachloride, the range of levels across the Detroit-area sites appeared to be fairly consistent, but there were statistically significant differences between the three highest sites and the three lowest sites in the Detroit area. The acrylonitrile level at N. Delray was significantly higher than at all other sites. 1,3-Butadiene levels at five sites meeting the criterion for a COPC had a range of only 2-fold, yet certain groupings of sites did show significant differences compared to other groupings.

For the trace metals (as TSP), manganese had the greatest amount of site-to-site variability in annual average levels. The sites with the highest manganese levels, S. Delray and Dearborn, were not significantly different from each other but were significantly higher than the other sites.

The highest annual average arsenic level was measured at S. Delray, which was significantly higher than all other sites. The highest annual average cadmium levels were found at N. Delray and S. Delray, which did not differ from each other but which were significantly higher than all other sites. The site with the highest nickel level, S. Delray, was significantly higher than the level at four other Detroit-area sites. The highest annual average level of naphthalene (and several other PAHs) was found at the S. Delray site, which was significantly different from all other sites.

In order to evaluate the relative spatial variability across all sites and all substances, site-to-site pair-wise comparisons were also compiled to determine for each site the percentage of the total number of combinations which had significant differences. This exercise determined that, among the COPCs, a relatively high level of site-to-site spatial variability (heterogeneity) existed for formaldehyde (with a spatial difference in 61% of the comparisons) and acetaldehyde (47%) among the carbonyls; and for benzene (61%) among the VOCs. A relatively homogeneous pattern was found for nickel (25%) and methylene chloride. A low frequency of detection at some sites precluded this type of comparison for some COPCs, such as chloroform and acrylonitrile. This method served to help describe tendencies across sites, but the approach provides results which may be prone to misunderstandings since COPC-specific findings may be hidden (e.g., methylene chloride levels were relatively homogenous across all sites, but were unusually high at Allen Park).

7.0 EXPOSURE ASSESSMENT

The exposure assessment is the step of a risk assessment that involves evaluating how human contact with the contaminated media occurs. According to the USEPA Technology Transfer Network (TTN) website for Fate, Exposure and Risk Analysis (FERA),[5]

> "Exposure is defined as the contact between a target organism and a pollutant at the outer boundary of the organism. Exposure may be quantified as the amount of the pollutant available at the boundary of the receptor organism per specified time period. From an exposure modeling standpoint, the principal goal is to estimate exposure as a function of both the relevant human factors and the measured or estimated pollutant concentrations in the contact or exposure media."

For an assessment of inhalation exposure, which is the scope of the present assessment, the metric representing the inhalation exposure is called the exposure concentration. (Other risk assessment efforts may be broader, evaluating what people are exposed to from various media and what activities result in the exposure.) When exposure information is combined with information about the toxicity of the chemicals in question, estimates are derived for the likelihood that the exposure may result in human health impacts. This is called the *risk characterization*.[6]

Human inhalation exposure via ambient air can be evaluated by ambient air monitoring or by constructing emission inventories and applying atmospheric dispersion modeling. The USEPA's Air Toxics Risk Assessment Reference Manual (Volume 1, Section 10.3) has an excellent discussion of the relative strengths and weaknesses of both approaches (USEPA, 2004b). With either approach, it is appropriate to acknowledge or address the differences which may exist between the ambient air estimates or measurements and actual human inhalation exposures. For example, USEPA's NATA (1996) provided ambient air impact estimates, and adjusted those according to human activities and locations which can affect exposure to ambient air toxics. This assessment found that the estimated human exposure inhalation levels tended to be approximately 80% to 100% of the modeled ambient air estimates. The USEPA's TTN website describes the Air Pollutants Exposure Model, which simulates the movement of individuals through time and space and their inhalation exposure to the given pollutant in various micro-environments (e.g., outdoors, indoors residence, in-vehicle).[7] Monitoring studies may also attempt to characterize "personal exposure" using monitors in receptor's microenvironments; ambient air monitoring may be used to test the degree of agreement with modeled estimates of ambient air levels.

For the present assessment, potential human exposure was determined on the basis of ambient air monitoring data. As stated in **Section 2**, the key questions posed were considered to be amenable to assessment utilizing the relative wealth of air toxics data measured in 2001-2002 in the greater Detroit area, the urban background site (Ypsilanti) and the relatively unimpacted site in the lower peninsula (Houghton Lake). The inhalation pathway of exposure was the only exposure pathway evaluated. The list of core compounds developed by the USEPA was utilized in the monitoring study and in the present risk assessment.

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[5] USEPA TTN FERA website is located at http://www.epa.gov/ttn/fera/.

[6] A more detailed description by the USEPA of the exposure assessment process is found, among other places, in the Air Toxics Risk Assessment Reference Library, Volume 1.

[7] General information on the USEPA TTN Air Pollutants Exposure Model is available at http://www.epa.gov/ttnmain1/fera/human_apex.html.

The associated health risks that may result from inhalation exposure addressed in this assessment are cancer, chronic noncancer, and acute effects. Repeated or extended exposure to relatively low concentrations of air toxics over long periods of time (chronic exposures) may result in chronic cancer or noncancer effects. Ideally, chronic exposure would be determined by continuous daily monitoring. For the Detroit Pilot Project, the monitoring frequency for VOCs and carbonyls was daily at the Dearborn site, while the frequency was 1 in 6 days or 1 in 12 days for all of the other substances and sites (see **Table 4**). The intent of the present initiative was to base the risk assessment on the estimated long-term average exposure levels. It was assumed that this would be best represented by the arithmetic average concentrations for the one-year monitoring period. However, as noted by USEPA (2004b, Appendix I), this approach may underestimate or overestimate the true annual average. USEPA adds that use of the 95% Upper Confidence Limit (UCL) on the mean instead of or in addition to a measure of central tendency (such as a mean or median) may be used as a public health protective estimate of the true annual average. This approach is likely to overestimate the true long-term average but virtually obviates the risk of underestimating the true exposure. The use of the 95% UCL rather than the average level was utilized in two recent air toxics risk assessment studies of Chattanooga (USEPA, 2001a) and West Louisville (Metro Louisville Air Pollution Control District [MLAPCD], 2003). The latter study additionally utilized the median level as another estimate of chronic exposure.

Exposure to relatively high concentrations of air toxics over relatively short periods of time (acute exposure) may result in near-term health effects or long-term effects. Acute risk was evaluated by examining the highest hourly concentrations (PAMS data) and the highest 24-hr concentrations (all other substances).

The exposure assessment for this report is specific to the study area (described in **Section 3**) and the analytic results of the Detroit Pilot Project for the substances monitored at the sites (see **Section 6**). The data represent an individual's potential ambient air exposures and associated risk levels at the monitor location. The exposure assessment further assumes that the duration of personal exposures to ambient air levels monitored would be short-term (for acute noncancer risk assessment) or long-term (for chronic noncancer and cancer risk assessment). For cancer risk assessment, the unit risk factors utilized to characterize the potential cancer risk are based upon a lifetime (70 years) of exposure at the annual averaged monitored concentrations. The exposure assessment approach for this monitoring-based assessment does not target "high-end" exposure estimates commonly included in modeling-based assessments, such as the maximum exposed individual or the maximum individual risk.[8]

The nature of the ambient urban monitoring approach does not enable risk assessors to define the size or characteristics of the study populations. Monitoring results are valid for the point of measurement, but the size and shape of areas represented by the data are not known and cannot be reliably estimated. Therefore, this assessment evaluates the potential risks to individuals but does not estimate of the sizes of populations associated with risk estimates.

USEPA (2004b, Appendix I) discusses several issues pertaining to data treatment and the handling of ND data. Some of those issues have been discussed in **Section 6** and earlier in the present section. An additional important issue is the criterion chosen for data inclusion (percent detect criterion) and data exclusion (percent ND criterion). USEPA (ibid.) recommends that summary statistics should be developed (for risk assessment purposes) if 15% or more of the samples for a substance and site are quantifiable (with adjustment for NDs, which is a separate issue discussed in **Section 6**). **Appendix D** provides the summary statistics for all of the

[8] See USEPA (2004b) Air Toxics Risk Assessment Reference Library, Volume 1, 13.2.

monitoring data, regardless of the frequency of detection. However, for the exposure assessment and other steps of the risk assessment process in this report, only the substances and sites with at least 15% detection frequencies are considered to be adequately representative of exposure levels for use in quantitative risk assessment (after adjustment for NDs). This is intended to be consistent with the recommendations of USEPA (ibid.). As a further exercise of interest (although not originally intended to be included in the present report), the MDLs for each substance that were excluded from the risk assessment due to a high frequency of NDs, may be compared to the health benchmark levels. This may allow a useful distinction between air toxics which are not COPCs (i.e., the MDLs are sufficiently below the health benchmark levels) and those which may potentially pose significant risks (i.e., their MDLs are higher than their health benchmark levels).

Finally, it should be noted that the exposure assessment and associated risk assessment results that are based upon a single year's worth of monitoring data represent a "shapshot" in time, and may not reflect past or future risks from exposure to air toxics.

8.0 TOXICITY ASSESSMENT

Toxicity assessment consists of hazard identification, evaluating dose-response relationships, and establishing health protective benchmark concentrations. This section briefly describes the methodology applied for determining the potential human health effects that may be associated with inhalation exposure to the monitored substances. Toxicological literature reviews were completed and standard risk assessment methods applied to determine the potential health impacts (hazard identification) and the experimental exposure conditions associated with those effects (dose-response relationships). The focus was on the inhalation exposure pathway. When sufficient toxicological data involving inhalation exposure were not available or adequate, oral exposure toxicological data were utilized if available and sufficient for risk assessment purposes (Note: Only inhalation data was utilized in the acute risk evaluation). This involved appropriate route-to-route conversion of exposures, unless there was evidence indicating that doing so would be inappropriate. **Appendix E** provides a cancer narrative that shows the cancer risk drivers, their health effects, quantitative risk, and weight of evidence available.

Cancer and noncancer (acute and chronic) effects were addressed. USEPA guidance was utilized if available, either to identify the hazards and dose-response relationships of substances, or as general guidance to apply in the toxicological literature review. This assessment consisted of identifying if the substances met the available USEPA and MDEQ-AQD criteria for inclusion in the quantitative cancer risk assessment, and the identification of the noncancer effects that could occur, particularly for the most sensitive endpoints. Quantitative cancer risk assessment was considered appropriate if the substance's toxicological data were consistent with an USEPA classification of Group A, B, or C (under the USEPA 1986 Guidance), and if the available dose-response data were adequate for quantitative cancer risk assessment modeling.

8.1 Cancer

USEPA and MDEQ have developed criteria for determining if exposure to substances may pose cancer risks (positive evidence of human or laboratory animal carcinogenicity), and whether those data provide dose-response information that supports quantitative cancer risk assessment. **Appendix F** indicates substances included in the Detroit Pilot Project that met those criteria. For those substances, inhalation unit risk (IUR) values were obtained from the USEPA or were derived by MDEQ-AQD toxicology staff utilizing the USEPA guidance (1986) or, in some cases, the Draft Final Guidelines for Carcinogen Risk Assessment (USEPA, 2003b). Unit risk values are used to quantify cancer risk from inhalation exposure to air toxics. A unit risk value represents the quantitative estimate of risk for a concentration of 1 microgram per cubic meter ($\mu g/m^3$) in the air.

The USEPA's Integrated Risk Information System (IRIS)[9] was the primary source of USEPA derived unit risk values. IRIS is an electronic database maintained by USEPA and containing information on human health effects that may result from exposure to various chemicals in the environment. Secondary sources that have been utilized in some cases in lieu of IRIS values include the USEPA's Health Effects Assessment Summary Tables (HEAST) and Health Assessment Documents. HEAST provides health benchmark values and cancer potency values that have been developed by USEPA program offices, but which have not received agency-wide consensus and have not been incorporated into the IRIS database.

[9] USEPA IRIS website is located at http://www.epa.gov/iris.

The unit risk values were used to derive Initial Risk Screening Levels (IRSLs), which are levels associated with an upper-bound cancer risk estimate of 1 in one million (1×10^{-6}), as specified in the AQD air toxics rules. The IRSLs were used as benchmarks to characterize the potential cancer risk from exposure to the annual average concentration of each individual carcinogenic chemical found at each monitoring site.

The characterization of potential cancer risk associated with ambient air monitoring data should include certain caveats. First, as previously discussed in **Section 7.0,** the exposure assumption employed is a continuous exposure to the monitored concentration for a lifetime (assumed to be 70 years). Also, it should be noted that many substances have been identified as potential human carcinogens on the basis of animal studies, because laboratory animals are presumed to be reasonable surrogates for humans and their susceptibility to cancer. Experimental studies typically employ higher exposure concentrations than the environmental exposures of interest, in order to detect an increase in tumor induction with reasonably sized animal exposure groups. The results from these studies are presumed to be suitable for modeling the estimated dose-response at much lower exposure levels. And, unless there is information to support a conclusion that there is a threshold for carcinogenic effects, it is assumed that there is no threshold. Finally, the 95% UCL of the modeled dose-response relationship at extrapolated low exposures is normally the basis for determining the unit risk value (inhalation exposure) or slope factor (oral exposure). As stated by USEPA (2004b, 13.2), "Because IURs are typically upper-bound estimates, actual risks may be lower than predicted, and the true value of the risk is unknown and may be as low as zero."

In addition to considering the risk from individual chemicals, it is relevant to consider the total (cumulative) cancer risk estimates including all of the carcinogenic chemicals found at a site. Total cancer risk estimates were derived by summing the individual cancer risk estimates at each site. USEPA (2000b) found that the method of simply adding upper-bound risk estimates results in a risk estimate for the mixture which is not excessively high, although over-conservatism may be a concern as the number of component chemicals increases. This process of simple response addition assumes that at low exposure levels involving the same or different target organs, there is an absence of toxicological interaction (e.g., synergism or antagonism). USEPA (2004b, 13.2 and 22.2) also recommends this screening-level approach, and the USEPA's NATA (1996) utilized this response addition approach for characterizing total cancer risk.

8.1.1 Presentation of Cancer Risk Levels in Lieu of Criteria for Acceptable Target Levels

The decision to utilize a cancer risk level criterion of 1×10^{-6} for data presentation and discussion in the risk assessment is a practical step to promote a focus upon the more substantial risk levels. It should be noted that there is no USEPA or MDEQ ambient air quality standard for an acceptable level of carcinogens in ambient air, for individual substances or cumulatively for multiple co-located carcinogens. This is an important issue to address in risk characterization and risk communication. Despite this situation, it is possible to provide a general sense of the significance of the cancer risk levels estimated in the present ambient air assessment, by comparison to criteria for acceptable cancer risk in other environmental programs. For example, the AQD permitting program for new and modified air emission sources utilizes acceptable risk levels of 1×10^{-6} for each substance emitted from the proposed emission unit or units, and one in 100,000 (1×10^{-5}) for each substance accounting for facility-wide emissions. Other MDEQ programs utilize an acceptable target cancer risk level (per carcinogen) of 1×10^{-5} for permitting releases to surface water, groundwater and soil, and for cleanup of contaminated sites.

Some USEPA programs for air quality assessment also utilize target cancer risk levels. The 1989 benzene National Emissions Standards for Hazardous Air Pollutants (NESHAP) presented a structure for applying an ample margin of safety to setting air emission standards for carcinogens (USEPA, 1989). The two-step structure included an analytical first step to determine an "acceptable risk" after considering all health information, including risk estimation uncertainty. In the case of benzene, this included a presumptive limit on maximum individual lifetime cancer risk of approximately one in 10,000 (1×10^{-4}). In the second step, the standard is set at a level that provides an ample margin of safety in consideration of all health information, including the number of persons at risk levels higher than approximately 1×10^{-6}, as well as other factors such as costs and economic impacts, technological feasibility, and factors relevant to the particular decision (USEPA, 1999). USEPA's methodology for evaluating residual risks posed by air emission sources regulated under the Maximum Achievable Control Technology (MACT) emission standards program [CAA's Section 112(f)] has established objectives which are founded upon and are consistent with the policy established in the 1989 benzene NESHAP framework. The residual risk assessments conducted will characterize incremental cancer risks posed, and may also consider and present the incremental risks in the context of the available information on background concentrations and risks. Also, the residual risk levels will be determined additively for multiple carcinogenic HAPs in refined analyses, and additivity may also be assumed in screening analyses. The specific cancer risk criteria utilized by USEPA in the CAA's Section 112(f) residual risk assessments for facilities in a source category under review, are that for emission impacts: "Upper-end individual cancer risk values less than or equal to ($\leq$) 10^{-6} are generally considered acceptable," "Upper-end individual cancer risk of roughly 1 in 10,000 is ordinarily considered the upper end of the range of acceptability (USEPA, 1999). Similarly, under Section 112(c)(9)(B) of the CAA Amendments of 1990, one of the criteria under which USEPA may delete source categories from regulation is a showing that the emissions and impacts of all sources in the category do not pose a lifetime cancer risk greater than 1×10^{-6} to the most exposed individuals.

USEPA's Cumulative Exposure Project (evaluating 1990 emissions) utilized a 1×10^{-6} cancer risk level per chemical in establishing benchmark concentrations representing "potential regulatory threshold concentrations of concern" (Caldwell et al., 1998), or, "a level of potential regulatory and public health concern" (Woodruff et al, 1998). The USEPA's subsequent NATA (1996) emissions also utilized benchmark concentrations associated with a risk level of 1×10^{-6}. However, USEPA emphasized that there were no risk levels representing an acceptable or unacceptable regulatory threshold. They advised that the results of the assessment should be used to target further measurement and assessment activities, rather than as absolute measures to determine whether risks are acceptable.[10]

8.2 Chronic Noncancer

The noncancer hazard assessment approach involved first determining a health protective benchmark value for each substance. The health protective benchmark values used in this assessment are the Initial Threshold Screening Levels (ITSLs) developed according to the procedure given in AQD Rule 336.1232. **Appendix F** contains a list of the chemical information with benchmarks used in this assessment.

AQD Rule 336.1232 provides a hierarchy of methods to follow when deriving an ITSL, and is dependent on the available data for a given substance. The top methodology in the hierarchy specifies that if a Reference Concentration (RfC) is available, then the ITSL is set at this value.

[10] Information was gathered from the USEPA TTN NATA Website's "Frequently Asked Questions" at http://www.epa.gov/ttn/atw/nata/natsafaq.html (1/23/2002).

The USEPA has defined the RfC as follows:

> "The RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups such as children, asthmatics and the elderly) that is likely to be without an appreciable risk of deleterious effects during a lifetime. It can be derived from various types of human or animal data, with uncertainty factors (UFs) generally applied to reflect limitations of the data used. "[11]

If an RfC is not available, the ITSL may be determined from a Reference Dose (RfD) or occupational exposure limit (OEL) such as a Threshold Limit Value (TLV) developed by the American Conference of Governmental Industrial Hygienists (ACGIH). The OELs may be established on the basis of critical health effects, other objectionable effects, or practical considerations. Also, they may reflect no-observed-adverse-effect levels (NOAELs) or lowest-observed-adverse-effect levels (LOAELs), to relatively healthy adult workers, for discontinuous time periods such as work shifts or portions of work shifts. Therefore, the basis for the OELs were reviewed to determine if they were appropriate for the derivation of ITSLs, with the application of an UF of 100 (generally) as per the AQD air toxics rules.

In the absence of an RfC, RfD, or suitable OEL, the AQD air toxics rules describe the potential derivation of ITSLs from lesser toxicological data. These include subchronic studies and acute lethality data, with specified algorithms and additional conversion factors and UFs. If an ITSL cannot be derived from such data (i.e., in the absence of such data), a default value of 0.1 $\mu g/m^3$ is assigned as the ITSL. It is recognized that ITSLs based on such limited (or lack of) toxicological information may not be appropriate for quantitative risk assessment of ambient air concentrations. This issue was addressed in **Section 9.2**, so that such situations arising in the initial screening of the monitored data were extracted and did not result in unsupportable hazard estimates in this report.

Each ITSL is associated with a specific averaging time as specified in AQD Rule 336.1232(2). All ITSLs based on an annual averaging time as specified in this rule, and all RfC or RfD based ITSLs were compared to the annual average concentration of the compound measured at each site. Under the AQD New Source Review process, ITSLs based on RfCs or RfDs are typically implemented with 24-hr averaging times. It should be noted that USEPA RfDs are defined as dose levels per day, while the USEPA RfCs are not associated with any particular averaging times. In practice, USEPA has implicitly associated an annual averaging time to RfCs, in the NATA initiative for example. In the current assessment, there were no changes made to the benchmark values, or to the monitored levels, to convert averaging times. The ITSLs based on RfCs and RfDs were treated as annually averaged values for comparison to the monitored levels in this section of the report.

After the appropriate health benchmark value (ITSL) was identified, a hazard quotient (HQ) and hazard index (HI) approach was used to determine potential for concern due to exposure at the monitored levels. Unlike linear dose-response assessments for cancer, noncancer hazards generally are not expressed as a probability of an individual suffering an adverse effect. Instead, the "hazard" for noncancer effects typically is quantified by comparing the exposure to the reference level, resulting in a ratio known as the HQ (i.e., the exposure divided by the appropriate chronic or acute health benchmark value).

[11] Information available on the USEPA website at http://www.epa.gov/ttn/atw/nata/gloss.html#unit.

For a given substance, exposures at or below the health benchmark level (i.e., HQ ≤ to 1) indicate that adverse noncancer effects are not likely to occur, and thus can be considered to have negligible hazard. HQs are a simple statement of whether (and by how much) an exposure concentration exceeds the health protective benchmark (ITSL or USEPA RfC). Moreover, the level of concern does not increase linearly or to the same extent as HQs increase above 1 for different chemicals since ITSLs (or RfCs) do not generally have equal accuracy or precision and are generally not based on the same severity of effect (USEPA, 2004b, 13.2.2). As exposures increase above the reference level (i.e., HQs increase above 1), the potential for adverse effects increases, but it is not known by how much. The HQ should not be interpreted as a probability of adverse effects.[12]

Exposure to air toxics generally occurs as a complex mixture. For the assessment of chronic noncancer effects, the potential for interactive effects should be accounted for. USEPA guidance for the risk assessment of complex mixtures recommends that potential additivity should be accounted for when evaluating a complex mixture without adequate toxicity data on the specific mixture of interest, or for a similar mixture. This was accomplished in a tiered fashion. First, only HQs of 0.1 or greater were included in order to place a focus on those substances which may have a significant impact in the additivity assessment and to exclude those which would only have a minimal impact.

A total hazardous index or "Total HI" was determined for each monitoring site by simply summing all of the HQs regardless of the critical effect. This step serves as a quick screen to determine what monitoring sites warrant further assessment (USEPA, 2004b, 22.3.2). However, it should be recognized that it is not biologically plausible to assume additivity for substances with different toxic effects. If Total HIs exceed a value of 1, the next tier in the approach is to evaluate potential additivity with a "dose addition" for substances with the same target organ or critical effect (USEPA, 2000b). For this step, the substances are segregated into separate "bins" specific to the various critical effect. Some substances fall into two or more bins, depending on the hazard and dose-response evaluation (see the critical and secondary effect in **Appendix G**). The resulting HIs are called "Target Organ Specific Hazard Indices," or TOSHIs. The most sensitive and relevant (critical) effects, and other potential effects for the air toxics included in this TOSHI assessment are provided in **Appendix G**. For TOSHIs with a value of 1 or less, a lack of adverse effect may be presumed. For TOSHIs exceeding a value of 1, harmful effects should not be presumed, but safety also cannot be presumed without further evaluation. The greater the TOSHI value exceeding 1, the greater the concern for potential additive effects, although there is no "bright line" for that concern. The risk assessment in that situation proceeds with a more extensive assessment of the HQs which contribute the most to the TOSHI, and are the primary COPCs for the TOSHIs, in an effort to characterize the potential for health impacts. The results of that assessment are described in **Section 9.2**. It should be noted that some TOSHI values may include HQs based upon RfCs or RfDs which incorporate an UF for database completeness, which may inappropriately inflate a TOSHI value. Also, some HQs may be based on very limited data. For these and other reasons, TOSHI values greater than (>) 1 warrant scrutiny and further assessment in order to develop appropriate risk characterizations.

[12] Additional information is available on the USEPA website at: http://www.epa.gov/ttn/atw/toxsource/noncarcinogens.html).

USEPA's NATA[13] provides a useful description of the significance of the HI as an indicator of risk:

> "As with the hazard quotient, aggregate exposures below a HI of 1.0 will likely not result in adverse noncancer health effects over a lifetime of exposure. However, an HI > 1.0 does not necessarily suggest a likelihood of adverse effects. Furthermore, the HI cannot be translated to a probability that adverse effects will occur, and is not likely to be proportional to risk. A respiratory HI > 1.0 can be best described as indicating that a potential may exist for adverse irritation to the respiratory system."

8.3 Acute Noncancer

Acute health effects are effects that may occur upon exposure to pollutants of sufficient magnitude over short durations (typically 24 hours or less). The selection of an appropriate health benchmark for use in the assessment of potential acute health effects should consider the following:

- the purpose of the benchmark value
- the target population it is designed to protect
- the level or degree of protectiveness
- the exposure time associated with the health benchmark value

This assessment considers the most appropriate acute health benchmarks to be those designed to prevent adverse effects from occurring in the general population. Acute health benchmarks designed for use in emergency situations that may result in serious or life threatening effects were not considered appropriate for this assessment.

A number of different organizations have developed acute health benchmark values. These values have been established for different purposes and are designed to be protective for different target populations (See **Table 7**).

Table 7: Sources for Acute Health Benchmark Values

ACUTE VALUE	BASIS	TARGET POPULATION	SOURCE AGENCY
CA REL	NOAEL	General population, including sensitive individuals	California EPA (Cal EPA)
MRL	NOAEL	General population, including sensitive subgroups	Agency for Toxic Substances and Disease Registry (ATSDR)
Acute Exposure Guidance Level (AEGL)	AEGL-1 = mild AEGL-2 = serious AEGL-3 = life-threatening	General population, including susceptible or sensitive individuals, but not hyper-susceptible or hypersensitive individuals	National Advisory Committee for Acute Exposure Guideline Levels for Hazardous Substances
Emergency Response Planning Guidelines (ERPG)	ERPG-1 = mild ERPG-2 = serious ERPG-3 = life-threatening	"Nearly all individuals," but are derived as emergency response guidelines as opposed to exposure guidelines and do NOT contain the safety factors found in other guidelines	American Industrial Hygiene Association's Emergency Response Planning Committee

[13] USEPA's NATA information is available at http://www.epa.gov/ttn/atw/nata/gloss.html#unit.

Table 7: Sources for Acute Health Benchmark Values

ACUTE VALUE	BASIS	TARGET POPULATION	SOURCE AGENCY
Temporary Emergency Exposure Limits (TEEL)	TEEL-0 = NOAEL TEEL-1 = mild TEEL-2 = serious TEEL-3 = life-threatening	Since TEELs are "approximations to ERPGs," presumably the Target Population is the same as for ERPGs	Subcommittee on Consequence Assessment of the Department of Energy

Key:

CA REL = California Reference Exposure Levels
NOAEL = No Observable Adverse Effect Levels
MRL = Minimal Risk Levels
AEGL = Acute Exposure Guideline Levels
ERPG = Emergency Response Planning Guidelines
TEEL = Temporary Emergency Exposure Limits

The Emergency Response Planning Guidelines (ERPG) are derived as emergency response guidelines and do not contain safety factors found in non-emergency exposure guidelines. Temporary Emergency Exposure Limits or TEELs are used for hazard assessments when ERPGs do not exist for a chemical, and are only considered approximations of ERPGs. TEELs have very little built-in safety for sensitive subgroups and are designed as a 15-minute emergency exposure value only. The exposure time for ERPGs and TEELs do not match the averaging times of the monitoring data available for this assessment. Considering the above, it was not considered appropriate to use ERPGs or TEELs for this assessment.

The California Reference Exposure Levels (CA REL) and the Agency for Toxics Substances and Disease Registry (ATSDR) MRL (minimal risk level) acute health benchmark values are based on non-emergency exposure guidelines. Acute Exposure Guideline Levels or AEGLs are based on a combination of emergency and non-emergency exposure guidelines. Only AEGL-1 values for mild effects were utilized in this assessment. These three acute health benchmark sources were considered appropriate for use in assessing acute inhalation risk from air toxics in this study. The rationale for their hierarchy of use is discussed next.

8.3.1 CA REL Values

The California EPA (Cal EPA) has developed CA RELs defined as “an exposure that is not likely to cause adverse effects in a human population, including sensitive subgroups, exposed to that concentration for one hour [unless otherwise noted] on an intermittent basis” (Cal EPA, Office of Environmental Health Hazard Assessment [OEHHA], 1998). The 51 chemicals for which RELs have been developed are a portion of the 450 chemicals for which emissions must be quantified under the California Hot Spots program. This program came about after a Congressional Research Service of the Library of Congress concluded that 75% of the U.S. population lives in proximity to at least one facility that manufactures and potentially releases into the surrounding air significant levels of substances either proven or thought to be potentially hazardous to public health. These releases may create localized concentrations of air toxics "hot spots" where emissions from specific sources may expose individuals and population groups to elevated risks for adverse health effects, and/or contribute to the cumulative health risks from other emission sources in the area.

CA RELs are designed to protect the general public, including sensitive subgroups. These values are intended to define an exposure that is not likely to cause adverse health effects and is based on routine, predictable releases from facilities. The CA REL for each chemical is based on the most appropriate and sensitive adverse health endpoint as documented in the literature. Heavy emphasis is placed on the utilization of available human data, with two-thirds of the acute CA RELs based on observed human health outcomes. Adjustments are made to adopted or derived NOAELs from selected studies to account for the appropriate exposure

scenario – in most cases, a 1-hr averaging time. Those values with longer averaging times are typically derived from studies with reproductive and/or developmental endpoints. In addition, UFs for intra- and inter-species variation are applied. Because UFs are applied in the derivation of these values, exposures that exceed the CA RELs do not necessarily indicate that adverse health effects will occur. In some cases, traditional 10-fold default values for uncertainty have been reduced where improvements in the database or scientific methodology allow.

The objectives of the DATI correlate very well with the California Hot Spots program. Both programs are designed to evaluate ambient emissions of air pollutants from sources in metropolitan areas. The CA RELs are the most appropriately applied benchmark for the DATI because these values account for exposures to sensitive individuals from routine predictable releases from facilities; the LOAEL or NOAEL may be adjusted by UFs; the averaging times are usually for one hour; and heavy emphasis is placed on human data. The RELs derived from this data appear to be of high quality and are generally the most appropriate health benchmark to use for assessing the 1-hr monitoring data obtained from the automated GC located at the N.E. Detroit site.

8.3.2 Acute MRL Values

MRL values are developed by the ATSDR, as an initial response to requirements in the Comprehensive Environmental Response, Compensation, and Liability Act as amended by the Superfund Amendments and Reauthorization Act. The ATSDR uses a methodology similar to that of USEPA's RfD and RfC methodology for deriving substance-specific health guidance levels for non-neoplastic endpoints.

An MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse non-cancer health effects over a specified duration of exposure. These substance-specific estimates, which are intended to serve as screening levels, are used by ATSDR, health assessors, and other responders to identify contaminants and potential health effects that may be of concern at hazardous waste sites. An acute MRL could appropriately be applied to monitoring data with greater than or equal to (≥) 24-hr averaging times since this value corresponds to 1 to 14 day exposures. The MRLs were considered the most appropriate health benchmark for use in assessing acute inhalation risk from monitored concentrations of chemicals with a 24-hr averaging time.

8.3.3 AEGL-1 Values

AEGLs are based, in part, on life-threatening concentrations of chemicals. What makes them unique, however, is a distinct health-based value for a range of exposure periods and a variety of effect levels. Values for chemical exposures have been established for 5 minutes, 10 minutes, 30 minutes, 60 minutes, 4 hours or 8 hours, for reversible/mild effects (AEGL-1), serious/irreversible effects (AEGL-2), and life-threatening effects (AEGL-3). Only the AEGL-1 values were considered appropriate for use as a screening tool for assessing acute effects from inhalation exposure to air toxics.

AEGL-1 values are designed to measure effects that are reversible and mildly symptomatic, or in other words, based on a LOAEL. These values are developed by the National Advisory Committee for Acute Exposure Guideline Levels for Hazardous Substances. The committee has members from government, industry, academic, and private organizations who develop and peer review these guideline values. The primary intent of the AEGLs is to assist organizations with emergency planning, response, and prevention. But since the AEGL-1 is based on a LOAEL, it is appropriate to use as an additional health benchmark for DATI where CA RELs or

MRLs do not exist. The strength of this value is the range of exposure periods from 10 minutes to 8 hours for a LOAEL. AEGL-1 values for a 1-hr exposure time were considered appropriate to use for assessing the 1-hr monitoring data from the automated GC located at the N.E. Detroit site, if an acute CA REL was not available.

8.4 Acute Health Effects Analysis Methodology

The daily concentrations for each pollutant at all eight sites, and the 1-hr monitoring data from the N.E. Detroit site, were examined to determine whether any measured concentrations of chemicals exceeded known acute health benchmark values. The initial screening process involved comparing the maximum daily and 1-hr value for each chemical at any site with CA RELs, MRLs and AEGL-1s. Although the CA RELs are most appropriate for 1-hr data and the MRLs are most appropriate for 24-hr data (due to similar averaging times), averaging time was not considered in the initial screening process. If any chemicals were found to exceed acute health benchmark values under this screening process, the data were further examined to consider exposure duration agreement between monitored values and acute health benchmark values.

In addition to considering the potential for acute health effects from exposure to individual chemicals, it is important to look at cumulative impacts, since people are exposed to a mixture of chemicals in ambient air. The first step in this process is to determine a HQ for each chemical. The HQ is a ratio of the measured environmental concentration of a chemical divided by the appropriate acute health benchmark value. An HQ $\geq$ 0.1 was chosen as a cut-off value in determining which chemicals might contribute to increased risk at each site. The selection of the criterion of HQ of 0.1 is a pragmatic step intended to set aside the data, which would have very little impact on risk assessment and characterization, and to focus on the data that may potentially be of significance in those evaluation steps.

Chemicals with HQs $\geq$ 0.1 (monitored concentrations that are 10% or more of the known acute benchmark) at each site would then be added together to yield a HI value for the site. Sites with an HI > 1 would then go into the next level of scrutiny, which considers similarity of toxic endpoints among the chemicals contributing to the HI as well as the timeframes during which spikes occurred. It is important to note that only chemicals for which there were known acute health benchmarks were considered in this analysis. Thus, only 27 of the 224 chemicals monitored were evaluated.

9.0 RISK CHARACTERIZATION

The risk characterization process integrates information from all preceding steps in the risk assessment (hazard identification, exposure assessment, toxicity assessment), interprets and describes the data, and provides a qualitative and/or quantitative estimate of risk. Key assumptions and uncertainties identified in each step of the assessment are discussed so that the nature and magnitude of the risks described can be placed in context with the study's inherent limitations. The results of the risk assessment can then be used to direct risk management initiatives or inform policy, depending on the stated purposes and aims of the study as identified in the planning and scoping step.

The USEPA (1995) *Policy for Risk Characterization* specifies that a risk characterization "be prepared in a manner that is clear, transparent, reasonable and consistent with other risk characterizations of similar scope prepared across programs in the Agency." USEPA also states that other additional relevant information, such as related studies, may be included to broaden the risk characterization discussion (USEPA, 2004b).

9.1 Cancer

9.1.1 Cancer Risk Estimates for Individual Substances at All Sites

Table 8 presents the cases where the annual average (AVG) monitored levels are associated with potential inhalation cancer risks of 1 X 10^{-6} or greater, listed in descending order. **Appendix H** contains the cancer risk values less than 1 X 10^{-6} in descending order, down to a risk level of one in 10 million (1 X 10^{-7}). The decision to utilize a cancer risk level criterion of 1 X 10^{-6} for data presentation and discussion in the risk assessment is a practical step to promote a focus upon the more substantial risk levels. It should be noted that there is no USEPA or MDEQ ambient air quality standard for an acceptable level of cancer risk in ambient air for individual substances or cumulatively for multiple co-located carcinogens. This issue is discussed in greater detail in **Section 8.1.1**. **Appendix E** provides chemical-specific information on tumor types, unit risk estimates (UREs), and weight of evidence.

Table 8: Chemicals with Estimated Cancer Risk of 1 X 10^{-6} or Greater

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	AVG W/ND=MDL/2 (µg/m³)	AQD IRSL (µg/m³)	1 X 10^{-6} RISK (AVG/IRSL)
Allen Park	Methylene Chloride	33	33	803	2	401.35
S. Delray	Naphthalene	58	58	18.4	0.08	229.88
S. Delray	Benzene	60	60	21.6	0.1	215.90
N. Delray	Acrylonitrile	26	12	0.914	0.01	91.37
River Rouge	Formaldehyde	31	30	5.55	0.08	69.39
N. Delray	Formaldehyde	29	29	3.94	0.08	49.24
S. Delray	Formaldehyde	51	51	3.00	0.08	37.46
Dearborn	Formaldehyde	324	319	2.87	0.08	35.81
N.E. Detroit	Formaldehyde	61	61	2.81	0.08	35.14
Southfield	Methylene Chloride	59	59	62.9	2	31.47
Southfield	Formaldehyde	61	60	2.38	0.08	29.70
Ypsilanti	Formaldehyde	29	29	2.28	0.08	28.55
N. Delray	Benzene	26	26	2.67	0.1	26.66
Allen Park	Formaldehyde	61	60	2.12	0.08	26.48
Houghton Lake	Formaldehyde	30	30	2.02	0.08	25.28

Table 8: Chemicals with Estimated Cancer Risk of 1 X 10^{-6} or Greater

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	AVG W/ND=MDL/2 (µg/m³)	AQD IRSL (µg/m³)	1 X 10^{-6} RISK (AVG/IRSL)
N. Delray	1,4-Dichlorobenzene	26	9	3.51	0.14	25.04
River Rouge	Benzene	60	60	2.14	0.1	21.37
Southfield	Benzene	59	59	1.99	0.1	19.85
Dearborn	Benzene	342	340	1.93	0.1	19.25
S. Delray	Arsenic (TSP)	65	65	0.00336	0.0002	16.80
N. Delray	Naphthalene	29	29	1.26	0.08	15.74
Allen Park	Benzene	33	33	1.50	0.1	14.98
Dearborn	Arsenic (TSP)	70	70	0.00241	0.0002	12.05
N. Delray	Arsenic (TSP)	65	65	0.00213	0.0002	10.64
River Rouge	Arsenic (TSP)	65	65	0.00191	0.0002	9.56
Allen Park	Arsenic (TSP)	71	71	0.00189	0.0002	9.47
Dearborn	Carbon Tetrachloride	342	339	0.633	0.07	9.05
Southfield	Carbon Tetrachloride	59	59	0.622	0.07	8.88
S. Delray	Carbon Tetrachloride	60	59	0.618	0.07	8.83
Allen Park	Carbon Tetrachloride	33	33	0.597	0.07	8.53
N.E. Detroit	Arsenic (TSP)	68	68	0.00169	0.0002	8.45
River Rouge	Carbon Tetrachloride	60	60	0.579	0.07	8.27
Dearborn	Naphthalene	49	49	0.6311	0.08	7.89
N. Delray	Carbon Tetrachloride	26	25	0.546	0.07	7.80
Southfield	1,3-Butadiene	59	49	0.191	0.03	6.37
S. Delray	1,3-Butadiene	60	48	0.190	0.03	6.33
Southfield	Arsenic (TSP)	59	59	0.00126	0.0002	6.28
River Rouge	Naphthalene	30	30	0.486	0.08	6.07
Dearborn	Methylene Chloride	342	326	11.6	2	5.81
River Rouge	Methylene Chloride	60	51	10.1	2	5.07
N.E. Detroit	Naphthalene	26	26	0.391	0.08	4.88
N. Delray	Acetaldehyde	29	29	2.17	0.5	4.34
River Rouge	Acetaldehyde	31	30	2.11	0.5	4.23
Dearborn	Acetaldehyde	324	318	2.09	0.5	4.19
Allen Park	1,3-Butadiene	33	22	0.12	0.03	4.00
Allen Park	Naphthalene	29	29	0.316	0.08	3.95
Dearborn	1,3-Butadiene	342	191	0.118	0.03	3.94
Ypsilanti	Arsenic (TSP)	18	18	0.000778	0.0002	3.89
River Rouge	1,3-Butadiene	60	20	0.110	0.03	3.68
Southfield	Naphthalene	59	59	0.292	0.08	3.65
N.E. Detroit	Acetaldehyde	61	61	1.78	0.5	3.57
S. Delray	Acetaldehyde	51	51	1.72	0.5	3.45
N. Delray	Methylene Chloride	26	19	6.66	2	3.33
Southfield	Acetaldehyde	61	61	1.59	0.5	3.17
Allen Park	Acetaldehyde	61	59	1.54	0.5	3.08
Ypsilanti	Acetaldehyde	29	29	1.09	0.5	2.17
S. Delray	1,4-Dichlorobenzene	60	10	0.297	0.14	2.12
N. Delray	Cadmium (TSP)	65	65	0.00122	0.0006	2.03
S. Delray	Cadmium (TSP)	65	65	0.00113	0.0006	1.89
Southfield	1,4-Dichlorobenzene	59	11	0.264	0.14	1.88
Dearborn	1,4-Dichlorobenzene	342	54	0.249	0.14	1.78
Houghton Lake	Acetaldehyde	30	29	0.859	0.5	1.72
River Rouge	1,4-Dichlorobenzene	60	10	0.220	0.14	1.57
N.E. Detroit	1,4-Dichlorobenzene	26	26	0.195	0.14	1.39
S. Delray	Methylene Chloride	60	58	2.53	2	1.27

Table 8: Chemicals with Estimated Cancer Risk of 1 X 10^{-6} or Greater

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	AVG W/ND=MDL/2 (µg/m³)	AQD IRSL (µg/m³)	1 X 10^{-6} RISK (AVG/IRSL)
S. Delray	Nickel (TSP)	65	65	0.00491	0.0042	1.17
River Rouge	Cadmium (TSP)	65	65	0.000621	0.0006	1.03
Dearborn	Cadmium (TSP)	70	70	0.000606	0.0006	1.01

9.1.2 Cancer Risk Estimates for Each Cancer Risk Driver Grouped by Chemical

Tables 9 – 20 provide a summary of the chemicals with estimated cancer risks > 1 X 10^{-6} grouped by chemical name.

Table 9: Cancer Risk Estimates for 1,3-Butadiene

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	AVG W/ND=MDL/2 (µg/m³)	AQD IRSL (µg/m³)	1 X 10^{-6} RISK (AVG/IRSL)
Southfield	1,3-Butadiene	59	49	0.191	0.03	6.37
S. Delray	1,3-Butadiene	60	48	0.190	0.03	6.33
Allen Park	1,3-Butadiene	33	22	0.12	0.03	4.00
Dearborn	1,3-Butadiene	342	191	0.118	0.03	3.94
River Rouge	1,3-Butadiene	60	20	0.110	0.03	3.68

Table 10: Cancer Risk Estimates for 1,4-Dichlorobenzene

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	AVG W/ND=MDL/2 (µg/m³)	AQD IRSL (µg/m³)	1 X 10^{-6} RISK (AVG/IRSL)
N. Delray	1,4-Dichlorobenzene*	29	9	3.51	0.14	25.04
S. Delray	1,4-Dichlorobenzene	60	10	0.297	0.14	2.12
Southfield	1,4-Dichlorobenzene	59	11	0.264	0.14	1.88
Dearborn	1,4-Dichlorobenzene	342	54	0.249	0.14	1.78
River Rouge	1,4-Dichlorobenzene	60	10	0.220	0.14	1.57

*** VOC method only**

Table 11: Cancer Risk Estimates for Acetaldehyde

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	AVG W/ND=MDL/2 (µg/m³)	AQD IRSL (µg/m³)	1 X 10^{-6} RISK (AVG/IRSL)
N. Delray	Acetaldehyde	29	29	2.17	0.5	4.34
River Rouge	Acetaldehyde	31	30	2.11	0.5	4.23
Dearborn	Acetaldehyde	324	318	2.09	0.5	4.19
N.E. Detroit	Acetaldehyde	61	61	1.78	0.5	3.57
S. Delray	Acetaldehyde	51	51	1.72	0.5	3.45
Southfield	Acetaldehyde	61	61	1.59	0.5	3.17
Allen Park	Acetaldehyde	61	59	1.54	0.5	3.08
Ypsilanti	Acetaldehyde	29	29	1.09	0.5	2.17
Houghton Lake	Acetaldehyde	30	29	0.859	0.5	1.72

Table 12: Cancer Risk Estimates for Acrylonitrile

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	AVG W/ND=MDL/2 (µg/m³)	AQD IRSL (µg/m³)	1 X 10^{-6} RISK (AVG/IRSL)
N. Delray	Acrylonitrile	26	12	0.914	0.01	91.37

Table 13: Cancer Risk Estimates for Arsenic

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	AVG W/ND=MDL/2 (µg/m³)	AQD IRSL (µg/m³)	1 X 10^{-6} RISK (AVG/IRSL)
S. Delray	Arsenic (TSP)	65	65	0.00336	0.0002	16.8
Dearborn	Arsenic (TSP)	70	70	0.00241	0.0002	12.05
N. Delray	Arsenic (TSP)	65	65	0.00213	0.0002	10.64
River Rouge	Arsenic (TSP)	65	65	0.00191	0.0002	9.56
Allen Park	Arsenic (TSP)	71	71	0.00189	0.0002	9.47
N.E. Detroit	Arsenic (TSP)	68	68	0.00169	0.0002	8.45
Southfield	Arsenic (TSP)	59	59	0.00126	0.0002	6.28
Ypsilanti	Arsenic (TSP)	18	18	0.000778	0.0002	3.89

Table 14: Cancer Risk Estimates for Benzene

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	AVG W/ND=MDL/2 (µg/m³)	AQD IRSL (µg/m³)	1 X 10^{-6} RISK (AVG/IRSL)
S. Delray	Benzene	60	60	21.59	0.1	216
N. Delray	Benzene	26	26	2.67	0.1	26.7
River Rouge	Benzene	60	60	2.14	0.1	21.4
Southfield	Benzene	59	59	1.99	0.1	19.9
Dearborn	Benzene	342	340	1.93	0.1	19.25
Allen Park	Benzene	33	33	1.50	0.1	14.98

Table 15: Cancer Risk Estimates for Cadmium

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	AVG W/ND=MDL/2 (µg/m³)	AQD IRSL (µg/m³)	1 X 10^{-6} RISK (AVG/IRSL)
N. Delray	Cadmium (TSP)	65	65	0.00122	0.0006	2.03
S. Delray	Cadmium (TSP)	65	65	0.00113	0.0006	1.89
River Rouge	Cadmium (TSP)	65	65	0.000621	0.0006	1.03
Dearborn	Cadmium (TSP)	70	70	0.000606	0.0006	1.01

Table 16: Cancer Risk Estimates for Carbon Tetrachloride

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	AVG W/ND=MDL/2 (µg/m³)	AQD IRSL (µg/m³)	1 X 10^{-6} RISK (AVG/IRSL)
Dearborn	Carbon Tetrachloride	342	339	0.633	0.07	9.05
Southfield	Carbon Tetrachloride	59	59	0.622	0.07	8.88
S. Delray	Carbon Tetrachloride	60	59	0.618	0.07	8.83
Allen Park	Carbon Tetrachloride	33	33	0.597	0.07	8.53
River Rouge	Carbon Tetrachloride	60	60	0.579	0.07	8.27
N. Delray	Carbon Tetrachloride	26	25	0.546	0.07	7.80

Table 17: Cancer Risk Estimates for Formaldehyde

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	AVG W/ND=MDL/2 (µg/m³)	AQD IRSL (µg/m³)	1 X 10^{-6} RISK (AVG/IRSL)
River Rouge	Formaldehyde	31	30	5.55	0.08	69.39
N. Delray	Formaldehyde	29	29	3.94	0.08	49.24
S. Delray	Formaldehyde	51	51	3.00	0.08	37.46
Dearborn	Formaldehyde	324	319	2.87	0.08	35.81
N.E. Detroit	Formaldehyde	61	61	2.81	0.08	35.14
Southfield	Formaldehyde	61	60	2.38	0.08	29.70
Ypsilanti	Formaldehyde	29	29	2.28	0.08	28.55
Allen Park	Formaldehyde	61	60	2.12	0.08	26.48
Houghton Lake	Formaldehyde	30	30	2.02	0.08	25.28

Table 18: Cancer Risk Estimates for Methylene Chloride

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	AVG W/ND=MDL/2 (µg/m³)	AQD IRSL (µg/m³)	1 X 10^{-6} RISK (AVG/IRSL)
Allen Park	Methylene Chloride	33	33	802	2	401.35
Southfield	Methylene Chloride	59	59	62.9	2	31.47
Dearborn	Methylene Chloride	342	326	11.6	2	5.81
River Rouge	Methylene Chloride	60	51	10.1	2	5.07
N. Delray	Methylene Chloride	26	19	6.66	2	3.33
S. Delray	Methylene Chloride	60	58	2.53	2	1.27

Table 19: Cancer Risk Estimates for Naphthalene

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	AVG W/ND=MDL/2 (µg/m³)	AQD IRSL (µg/m³)	1 X 10^{-6} RISK (AVG/IRSL)
S. Delray	Naphthalene	58	58	18.4	0.08	229.88
N. Delray	Naphthalene	29	29	1.26	0.08	15.74
Dearborn	Naphthalene	49	49	0.631	0.08	7.89
River Rouge	Naphthalene	30	30	0.486	0.08	6.07
N.E. Detroit	Naphthalene	26	26	0.391	0.08	4.88
Allen Park	Naphthalene	29	29	0.316	0.08	3.95
Southfield	Naphthalene	59	59	0.292	0.08	3.65

Table 20: Cancer Risk Estimates for Nickel

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	AVG W/ND=MDL/2 (µg/m³)	AQD IRSL (µg/m³)	1 X 10^{-6} RISK (AVG/IRSL)
S. Delray	Nickel (TSP)	65	65	0.00491	0.0042	1.17

9.1.3 Cancer Risk Estimates for Each Cancer Risk Driver Grouped by Site

Tables 21 – 29 provide a summary of the chemicals with estimated cancer risks > 1 X 10^{-6}, grouped by site. (NOTE: canister VOC monitoring was not done at the N.E. Detroit site but PAMS monitoring was done; therefore, some cancer risk drivers [non-PAMS VOCs] which were monitored at the other sites are not included for this site.)

Table 21: Cancer Risk Estimates for Allen Park Site

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	AVG W/ND=MDL/2 (µg/m³)	AQD IRSL (µg/m³)	1 X 10^{-6} RISK (AVG/IRSL)
Allen Park	Methylene Chloride	33	33	803	2	401
	Formaldehyde	61	60	2.12	0.08	26.5
	Benzene	33	33	1.50	0.1	15.00
	Arsenic (TSP)	71	71	0.00189	0.0002	9.47
	Carbon Tetrachloride	33	33	0.597	0.07	8.53
	1,3-Butadiene	33	22	0.12	0.03	4
	Naphthalene	29	29	0.316	0.08	3.95
	Acetaldehyde	61	59	1.54	0.5	3.08
					Total	**472**

Table 22: Cancer Risk Estimates for Dearborn Site

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	AVG W/ND=MDL/2 (µg/m³)	AQD IRSL (µg/m³)	1 X 10^{-6} RISK (AVG/IRSL)
Dearborn	Formaldehyde	324	319	2.87	0.08	35.8
	Benzene	342	340	1.92	0.1	19.3
	Arsenic (TSP)	70	70	0.00241	0.0002	12.1
	Carbon Tetrachloride	342	339	0.633	0.07	9.05
	Naphthalene	49	49	0.631	0.08	7.89
	Methylene Chloride	342	326	11.6	2	5.81
	Acetaldehyde	324	318	2.09	0.5	4.19
	1,3-Butadiene	342	191	0.118	0.03	3.94
	1,4-Dichlorobenzene	342	54	0.249	0.14	1.78
	Cadmium (TSP)	70	70	0.000606	0.0006	1.01
					Total	**101**

Table 23: Cancer Risk Estimates for S. Delray Site

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	AVG W/ND=MDL/2 (µg/m³)	AQD IRSL (µg/m³)	1 X 10^{-6} RISK (AVG/IRSL)
S. Delray	Naphthalene	58	58	18.39	0.3	230
	Benzene	60	60	21.59	0.1	216
	Formaldehyde	51	51	3.00	0.08	37.5
	Arsenic (TSP)	65	65	0.00336	0.0002	16.8
	Carbon Tetrachloride	60	59	0.618	0.07	8.83
	1,3-Butadiene	60	48	0.190	0.03	6.33
	Acetaldehyde	51	51	1.72	0.5	3.45
	1,4-Dichlorobenzene	60	11	0.297	0.14	2.12
	Cadmium (TSP)	65	65	0.00113	0.0006	1.89
	Methylene Chloride	60	58	2.53	2	1.27
	Nickel (TSP)	65	65	0.00491	0.0042	1.17
					Total	**525**

Table 24: Cancer Risk Estimates for N.E. Detroit Site

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	AVG W/ND=MDL/2 (µg/m³)	AQD IRSL (µg/m³)	1 X 10^{-6} RISK (AVG/IRSL)
N.E. Detroit	Formaldehyde	61	61	2.81	0.08	35.14
	Arsenic (TSP)	68	68	0.00169	0.0002	8.45
	Naphthalene	26	26	0.391	0.08	4.88
	Acetaldehyde	61	61	1.78	0.5	3.57
					Total	**52**

Table 25: Cancer Risk Estimates for Houghton Lake Site

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	AVG W/ND=MDL/2 (µg/m³)	AQD IRSL (µg/m³)	1 X 10^{-6} RISK (AVG/IRSL)
Houghton Lake	Formaldehyde	30	30	2.02	0.08	25.28
	Acetaldehyde	30	29	0.859	0.5	1.72
					Total	**27**

Table 26: Cancer Risk Estimates for Southfield Site

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	AVG W/ND=MDL/2 (µg/m³)	AQD IRSL (µg/m³)	1 X 10^{-6} RISK (AVG/IRSL)
Southfield	Methylene Chloride	59	59	62.9	2	31.5
	Formaldehyde	61	60	2.38	0.08	29.7
	Benzene	59	59	1.99	0.1	19.9
	Carbon Tetrachloride	59	59	0.622	0.07	8.89
	1,3-Butadiene	59	49	0.191	0.03	6.37
	Arsenic (TSP)	59	59	0.00126	0.0002	6.28
	Naphthalene	59	59	0.292	0.08	3.65
	Acetaldehyde	61	61	1.59	0.5	3.17
	1,4-Dichlorobenzene	59	11	0.264	0.14	1.88
					Total	**111**

Table 27: Cancer Risk Estimates for River Rouge Site

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	AVG W/ND=MDL/2 (µg/m³)	AQD IRSL (µg/m³)	1 X 10^{-6} RISK (AVG/IRSL)
River Rouge	Formaldehyde	31	30	5.55	0.08	69.39
	Benzene	60	60	2.14	0.1	21.37
	Arsenic (TSP)	65	65	0.00191	0.0002	9.56
	Carbon Tetrachloride	60	60	0.579	0.07	8.27
	Naphthalene	30	30	0.486	0.08	6.07
	Methylene Chloride	60	51	10.1	2	5.07
	Acetaldehyde	31	30	2.11	0.5	4.23
	1,3-Butadiene	60	20	0.110	0.03	3.68
	1,4-Dichlorobenzene	60	10	0.220	0.14	1.58
	Cadmium (TSP)	65	65	0.000621	0.0006	1.03
					Total	**130**

Table 28: Cancer Risk Estimates for N. Delray Site

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	AVG W/ND=MDL/2 (µg/m³)	AQD IRSL (µg/m³)	1 X 10^{-6} RISK (AVG/IRSL)
N. Delray	Acrylonitrile	26	12	0.914	0.01	91.4
	Formaldehyde	29	29	3.94	0.08	49.2
	Benzene	26	26	2.67	0.1	26.7
	1,4-Dichlorobenzene	26	9	3.51	0.14	25.0
	Naphthalene	29	29	1.26	0.08	15.74
	Arsenic (TSP)	65	65	0.00213	0.0002	10.7
	Carbon Tetrachloride	26	25	0.546	0.07	7.80
	Acetaldehyde	29	29	2.17	0.5	4.34
	Methylene Chloride	26	19	6.66	2	3.33
	Cadmium (TSP)	65	65	0.00122	0.0006	2.03
					Total	**236**

Table 29: Cancer Risk Estimates for Ypsilanti Site*

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	AVG W/ND=MDL/2 (µg/m³)	AQD IRSL (µg/m³)	1 X 10^{-6} RISK (AVG/IRSL)
Ypsilanti	Formaldehyde	29	29	2.28	0.08	28.55
	Arsenic (TSP)	18	18	0.000778	0.0002	3.89
	Acetaldehyde	29	29	1.09	0.5	2.17
					Total	**35**

***Does not include VOCs.

9.1.4 Cumulative Cancer Risk for Each Site

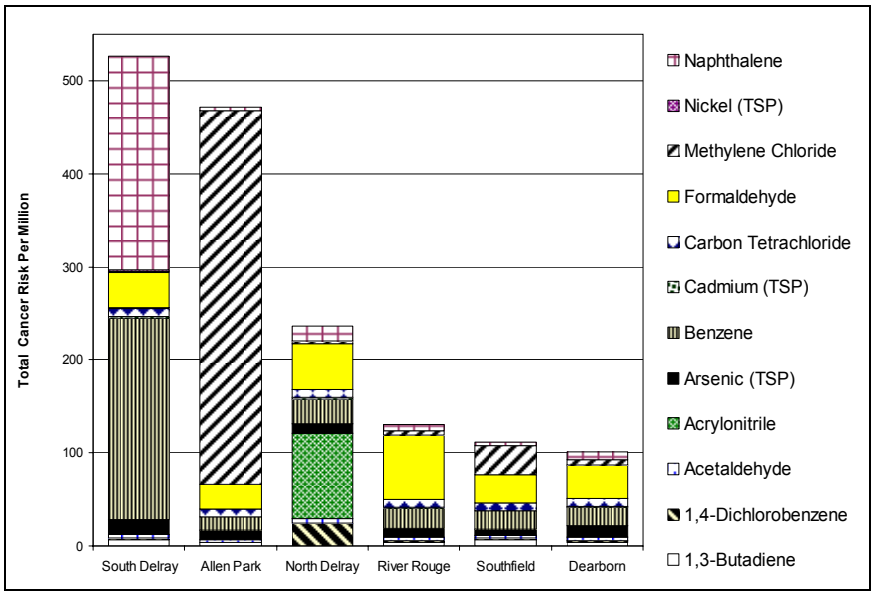
Besides an interest in the substances posing the highest estimated lifetime cancer risks, it is also important to consider the total (cumulative) risk estimates associated with exposure to the carcinogenic components of the mixtures. Total cancer risk estimates are derived by summing the individual cancer risk estimates at each site. This method of "response addition" is appropriate, under the assumption that the components are toxicologically dissimilar and act independently, and that decreasing the exposure levels of components reduces the probability of an effect, but not its severity. Under these appropriate default assumptions, simultaneous exposure to several chemicals could accumulate many small risks and may be unacceptable in combination even though the individual risks may be acceptably small. The conventional approach for the addition of individual cancer risk estimates is to simply add them together, noting that they have each been derived as the plausible upper bound on the risk which may add some conservatism to the total.

The resulting total cancer risk estimates are shown in the tables in **Section 9.1.3**, and in the following **Figure 8**. **Figure 8** includes the 12 carcinogenic substances which were found to pose an incremental lifetime upper-bound risk estimate of 1 X 10^{-6} or higher, at one or more sites. These substances are referred to as "cancer risk drivers" in this report, although it should be noted that some of these substances are obviously much more significant than others in terms of the risk estimates. The N.E. Detroit, Ypsilanti, and Houghton Lake sites are not included in **Figure 8** because the air toxics monitored were a more limited set compared to the other sites (VOC data were not obtained); see **Section 9.1.3** for the total cancer risk estimate for those sites.

Figure 8 indicates that there were considerable differences in the total cancer risk estimates across sites. It also shows that several of the cancer risk drivers posed a fairly consistent magnitude of risk at most or all sites. Other carcinogens were at high levels only at individual sites, which had a major impact on the total cancer risk estimates for those sites (methylene chloride at Allen Park, naphthalene and benzene at S. Delray, and acrylonitrile at N. Delray).

As noted above, the site-by-site comparison of total cancer risk in **Figure 8** includes only six sites, because the remaining three monitoring sites (N.E. Detroit, Ypsilanti, and Houghton Lake) lacked data for the VOCs, seven of which were found to be cancer risk drivers based on the sites where they were measured.

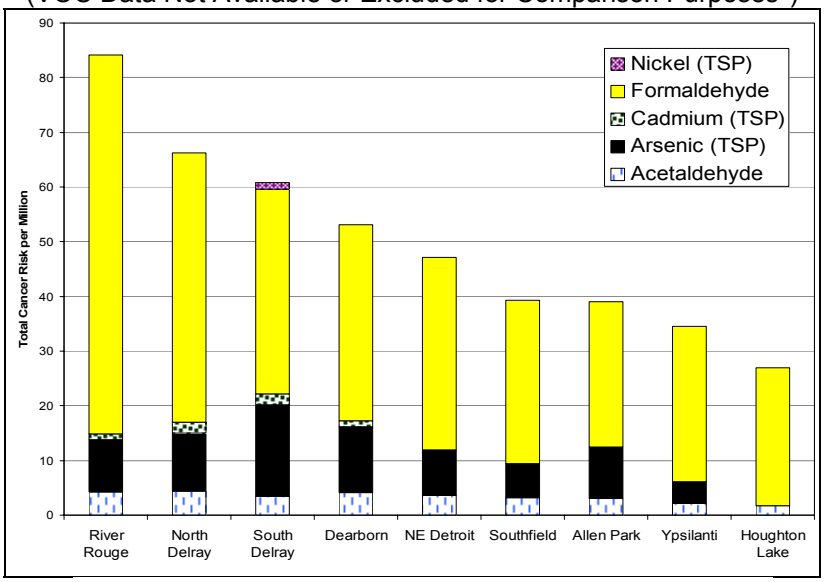
Figure 8: Lifetime Cancer Risk Estimates for Risk Drivers at Detroit Area Monitoring Sites*



*The high-end estimated cancer risk is the incremental lifetime (70 years) risk by inhalation to the individual. The estimates of cancer risks should not be viewed as actual cancer cases resulting from air pollution but as upper bound estimates so the agency can prioritize its efforts to reduce exposures. It should also be noted that these risk estimates are based on 2001-2002 monitoring data, and the unusually high levels of methylene chloride at Allen Park and naphthalene and benzene at S. Delray during that period have not persisted in subsequent monitoring.

Figure 9: Lifetime Cancer Risk Estimates for Carbonyl and Metal Substances Only

(VOC Data Not Available or Excluded for Comparison Purposes*)



* The high-end estimated cancer risk is the incremental lifetime (70 year) risk by inhalation to the individual. The estimates of cancer risk should not be viewed as actual cancer cases resulting from air pollution but as upper bound estimates so the agency can prioritize its efforts to reduce exposures.

Figure 9 provides a site-by-site comparison across all nine sites for the substances that were measured at these sites. This includes the cancer risk drivers which were metals (nickel, cadmium, and arsenic) or carbonyls (formaldehyde and acetaldehyde). It should be noted that the vertical scales for **Figures 8** and **9** are quite different.

Several carcinogenic compounds had significant issues warranting further discussion in the following subsections: hexavalent chromium, nickel, formaldehyde, naphthalene, and PAHs.

9.1.5 Hexavalent Chromium

As discussed previously in **Section 6**, Cr^{+6} is a known human carcinogen by inhalation, and it is relatively high in potency. Total chromium was measured at each site, while Cr^{+6} was measured at four sites. As previously discussed in **Section 6**, the site-specific averages of the percentages of Cr^{+6} in total chromium (excluding all ND data) are shown in **Table 30**.

Table 30: Percentage of Hexavalent Chromium in Total Chromium

MONITORING SITE				
Average of each daily measured fraction, as %	**Southfield**	**Allen Park**	**River Rouge**	**Dearborn**
	2.4%	2.0%	1.4%	1.0%

The measured levels of Cr^{+6} were utilized in the risk assessment for those four sites. For all other sites, an appropriate surrogate was desired. The decision was made to use a fraction of 0.024 (2.4%) to adjust the average measured total chromium levels for each site lacking direct measurement of Cr^{+6}, to derive reasonable estimates for Cr^{+6}. When all ND data were excluded, the value of 2.4% was the highest of the four sites' percentages (**Table 30**). This was considered to be an appropriate conservative surrogate because there appears to be a significant difference across sites. It should nevertheless be noted that there may be unmonitored locations with even higher ratios due to local emission sources. That concern is supported by the USEPA NATA (1996) report that a high-end percentage for utility boiler emissions is 34%, and electroplating processes may have up to 70% Cr^{+6}. At the four sites where Cr^{+6} was measured, the levels were associated with risk levels less than the 1×10^{-6}. For the other sites, assuming that the Cr^{+6} level was 2.4% of the measured total chromium level (**Appendix D**), only S. Delray (2.25×10^{-6}) and N. Delray (1.66×10^{-6}) had estimated levels associated with risks exceeding 1×10^{-6}. Although these estimates exceed the 1×10^{-6} risk level used in this report as a criterion for risk drivers, they were not included in that category because the levels were only estimated via an assumed fraction of total chromium.

9.1.6 Formaldehyde Cancer Risk Assessment

USEPA has listed formaldehyde as a probable human carcinogen for inhalation exposure based on limited human data and sufficient animal data. The assessment of cancer risk for formaldehyde in the present study resulted in a finding that it is a significant cancer risk driver at all of the monitored sites. Formaldehyde ranked in the top three highest cancer risks at all sites. The levels of risk ranged from 27 to 69×10^{-6} at the Detroit and Ypsilanti sites, and was 25×10^{-6} at Houghton Lake. The formaldehyde risk levels across sites were fairly uniform, compared to some of the other cancer risk drivers.

However, USEPA has recently reconsidered their inhalation URE for formaldehyde. The following is quoted from the USEPA TTN Air Toxics Website:

> "A new USEPA IRIS assessment is underway in light of a CIIT [Chemical Industry Institute of Toxicology] analysis that supports a URE on the order of 5.5E-9 per $\mu g/m^3$. This value is substantially lower than the current IRIS URE of 1.3E-5 per $\mu g/m^3$ (CIIT, 1999)."[14]

Only the newer, lower unit risk value is listed in the USEPA TTN list of dose-response assessments. In the USEPA's risk assessment for a potential de-listing of the plywood and

[14] The USEPA TTN Air Toxics Website, "Adjustments and Special Cases for Chronic Inhalation Risk Assessment" is available at http://www.epa.gov/ttnatw01/toxsource/adjustments.html.

composite wood products (PCWP), only the newer, lower unit risk was utilized. Reportedly, this is also the case with at least one other USEPA proposed delisting (combustion turbines). Nevertheless, the USEPA IRIS database continues to list the higher URE. The MDEQ IRSL also continues to be based on the established higher URE. Therefore, for the present risk assessment, the established higher URE was used. However, it should be noted that the finding of formaldehyde as a significant cancer risk driver at all of the monitoring sites would be an insignificant contributor to total cancer risk under the newer, 2,363 times lower URE.

9.1.7 Nickel Cancer Risk Assessment

Nickel posed a cancer risk of > 1 X 10^{-6} at only one site (S. Delray; 1.2 X 10^{-6}). The unit risk factor for nickel refinery dust (classified by USEPA as a known human carcinogen), as used by MDEQ in the IRSL and listed by USEPA in IRIS, was applied to the total nickel monitored. This is a somewhat conservative step, since not all nickel is expected to be in carcinogenic forms. The USEPA NATA (1996) cited limited information and conservatively assumed that 65% of emitted nickel was in the carcinogenic insoluble crystalline form. This was also assumed by USEPA in their risk assessment for the proposed delisting of sources in the PCWP source category (Jenkins et al., 2004). In the USEPA Utility Study, the baseline risk assessment had a conservative, high-end assumption that all of the nickel emitted was as carcinogenic as nickel subsulfide.

9.1.8 Naphthalene Cancer Risk Assessment

Naphthalene cancer risk estimates exceeded 1 X 10^{-6} at six Detroit-area sites, ranging from 3.6 to 230 X 10^{-6}. The highest was at the S. Delray site. The URE utilized was 1.2 X 10^{-5} per µg/m^3 as derived by the AQD based on male rat nasal tumors reported in a 2000 National Toxicology Program (NTP) bioassay. USEPA does not currently have a URE in IRIS, or at their TTN website for air toxics health protection benchmarks.

A recent USEPA external review draft IRIS summary [6/23/04] proposes a 95% upper bound URE that is approximately 10 times larger (1 X 10^{-4} per µg/m^3) than the AQD derived value (USEPA, 2004d). If adopted this URE would increase cancer risk estimates by about 10-fold over current estimates. The weight-of-evidence characterization is, "…naphthalene is likely to be carcinogenic to humans by the inhalation route of exposure…." The status of this external review draft is, "Do not cite or quote." In August 2004, USEPA released an External Peer Review draft report, without any "Do not cite or quote" qualifier (USEPA, 2004e). This draft is currently undergoing review. For the NATA 1999 assessment, USEPA is currently utilizing a URE of 3.4 X 10^{-5} per µg/m^3, based upon the California OEHHA adopted value, pending the peer review process for the proposed USEPA value.[15]

9.1.9 PAHs Cancer Risk Assessment

The group of substances known as polycyclic aromatic hydrocarbons or PAHs includes seven which are regulated as carcinogens. Data sufficient for quantitative cancer risk assessment are available for only benzo(a)pyrene [B(a)P]. For the other six, relative potency factors relate their estimated potency to that of B(a)P based on the available toxicological data. This approach was provided by USEPA (1993). The AQD's IRSL of 0.0005 µg/m^3 for B(a)P is normally applied to the combined emissions and impacts of these seven carcinogenic PAHs using the relative potency approach.

[15] Personal communication with Roy Smith, USEPA-Risk and Exposure Assessment Group, 9/15/04.

The Detroit Pilot Project included analysis for PAHs at all seven Detroit-area sites on a 1 in 6 day or 1 in 12 day schedule, but did not include PAHs at Ypsilanti or Houghton Lake. The only site which had at least 15% detection frequency for any of the seven carcinogenic PAHs was S. Delray (chrysene - 53%; benzo(k)fluoroanthene - 26%; benzo(b)fluoroanthene - 26%; benzo(a)anthracene - 52%). Substituting MDL/2 for NDs, and using the relative potency factor approach, the average levels of these four PAHs were collectively associated with an incremental cancer risk estimate of 3.0×10^{-6}. At S. Delray, B(a)P was detectable in only 6.9% of the samples, which therefore did not satisfy the 15% detection criterion utilized for quantitative cancer risk assessment. It was detected in 4 out of 58 samples (6.9%). If the NDs for B(a)P were assumed to be MDL/2, or zero, then the average level at S. Delray was associated with an incremental cancer risk estimate of 21 or 2.8×10^{-6}, respectively. These results indicate that the PAHs at S. Delray were not major drivers of total cancer risk, but they did exceed the 1×10^{-6} benchmark for "cancer risk drivers" utilized in this report. However, since the B(a)P results did not qualify for quantitative cancer risk estimation due to excessive NDs, and the other carcinogenic PAHs are normally treated only as a group with B(a)P, these results are presented here rather than in the tables of cancer risk estimates in this section. It is also noted that in subsequent monitoring efforts, a more sensitive MDL will be available to help address this analytical limitation.

9.2 Chronic Noncancer

The noncancer hazard assessment approach was consistent with USEPA guidance and MDEQ rules for deriving screening levels as part of the AQD air toxics rules. The following **Table 31** indicates the substances and monitoring locations where the chronic (annual average) levels were $\geq$ 10% of the chronic noncancer health protective benchmark levels. In other words, the HQs (monitored levels divided by their benchmark levels) were 0.1 or greater. Also in the following tables and in **Section 10** of this report, this information is presented and discussed for each of the monitor locations.

The criterion of HQ of 0.1 or greater was selected for use here and for the assessment of potential additivity later in this subsection. This is a pragmatic step intended to set aside the data that would have very little impact on hazard assessment and characterization, and to focus on the data that may potentially be of significance in those evaluation steps.

The HQs were calculated as the annually averaged monitored levels divided by the health protective benchmark value. The annually averaged monitored levels were derived by first discounting those substances and locations where < 15% of the measurements were at or above the MDL. The criterion of 15% detection, and the treatment of ND data by substituting a value of ½ the analytical benchmark, is consistent with USEPA guidance (USEPA, 2004b, Appendix I) and has been discussed previously in **Section 6.2**. The use of MDLs, rather than some other analytical benchmark, is consistent with past practices. However, this has been disfavored in recent USEPA guidance (ibid.).

The health protective benchmark levels in HQ derivation are the AQD ITSLs with annual or 24-hr averaging times. This includes the ITSLs derived from AQD standard procedures and assigned annual averaging times as prescribed by Rule 336.1232(2) of the AQD's air toxic rules, and the ITSLs based on RfCs and RfDs (derived by USEPA or AQD), which are assigned 24-hr averaging times per this rule. It should be noted that USEPA RfDs are defined as dose levels per day, while the USEPA RfCs are not associated with any particular averaging times. In practice, USEPA has implicitly associated an annual averaging time to RfCs for the NATA initiative. In the current assessment, there were no changes made to the benchmark values or to the monitored levels to convert averaging times. Therefore, in this section of the report, the ITSLs based on RfCs and RfDs were treated as annually averaged values for comparison to the

monitored levels. Some of the AQD ITSLs are based on OELs, divided by 100. OELs which are time-weighted averages (TWAs) result in ITSLs with 8-hour averaging times. These were also compared to annually averaged monitored levels without adjustment for averaging times.

In the initial screening step, HQs were derived utilizing the AQD ITSLs without discrimination as to the approach utilized to derive the ITSLs. As discussed in **Section 8.2**, ITSLs may be derived on the basis of a wide variety of values or data (RfCs, RfDs, OELs, subchronic studies, acute lethality), or by default the ITSL may be set at 0.1 $\mu g/m^3$ in the absence of such data or values. Once the HQs are derived and the focus is placed upon those with HQs of 0.1 or greater, it is appropriate to consider the basis of the ITSL for these HQs. Some ITSLs are based upon very limited datasets, or even a complete lack of useful data resulting in ITSLs set at the default value. This evaluation step determined that there were three substances with HQs of 0.1 or greater, with ITSLs which are not supportive of risk assessment or HQ derivation in the present initiative. Those substances and the basis for their ITSLs are as follows: dibenzofuran (default value); phenanthrene (default value); and 2-methylnaphthalene (lethal concentration 50% for inhalation dosing or LC_{50}). There is also a lack of chronic inhalation noncancer benchmarks for these substances in the USEPA TTN air toxics website. Therefore, it would be inappropriate to include those substances and their ITSLs in quantitative hazard assessment in the current initiative. In this report, they have been eliminated from any further consideration of the potential hazards of single-substances or potential additivity. The inability to assess the potential health effects of these compounds due to inadequate toxicological or epidemiological studies underscores the need for the development of this data.

9.2.1 Chronic Noncancer Hazard Quotients for All Sites

For HQs with a value of 1 or less, a lack of adverse effect may be presumed (with the caveat that potential additivity will be considered later). For HQs exceeding a value of 1, harmful effects should not be presumed, but safety also cannot be presumed without further evaluation. The greater the HQ value exceeding 1, the greater the concern for potential additive effects, although there is no “bright line” for that concern. HQ values > 1 warrant scrutiny and further assessment in order to develop appropriate risk characterizations. The hazard assessment in that situation proceeds with a more extensive evaluation of the basis for the HQs which exceed 1, in an effort to characterize the potential for health impacts. The HQs of 0.1 or greater are presented in three different table formats: **Table 31** shows all chemicals with HQ of 0.1 or greater (in descending order), **Tables 32 - 37** present these HQs sorted by chemical, and **Tables 39 - 47** sorted by site.

Table 31 indicates that two chemicals, naphthalene and manganese, had HQs that exceeded a value of 1, ranging from 1.5 to a highest value of 6.13. The highest HQ was for naphthalene at the S. Delray site. The four other HQs which exceeded 1 were for manganese at four sites. **Figure 10** compares the monitoring sites and the HQs of 0.1 or greater for each site. The significance of the HQs greater than 1 for manganese and naphthalene are discussed in **Sections 9.2.2.1** and **9.2.2.2**, respectively.

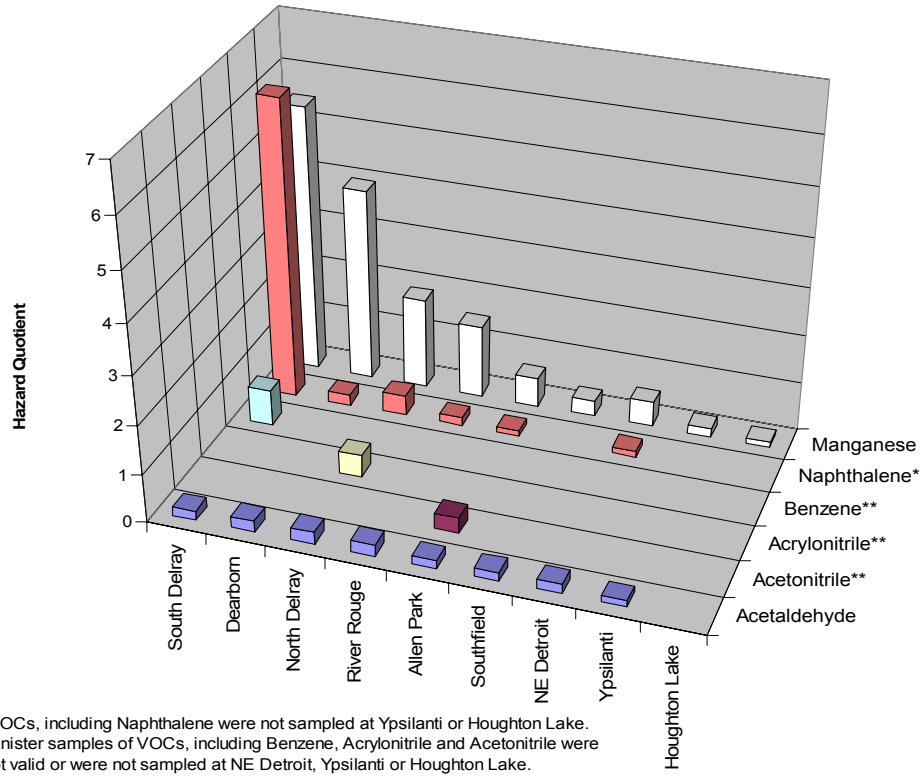
Table 31: Chemicals with HQs of 0.1 or Greater at Any Site
(In Descending Order)

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	MAX1 ($\mu g/m^3$)	AVG ND=MDL/2 ($\mu g/m^3$)	AQD ITSL ($\mu g/m^3$)	HQ
S. Delray	Naphthalene	58	58	93.25	18.4	3	6.13
S. Delray	Manganese (TSP)	65	65	1.94	0.274	0.05	5.47
Dearborn	Manganese (TSP)	70	70	1.19	0.198	0.05	3.95
N. Delray	Manganese (TSP)	65	65	0.188	0.0930	0.05	1.86

Table 31: Chemicals with HQs of 0.1 or Greater at Any Site
(In Descending Order)

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	MAX1 (µg/m³)	AVG ND=MDL/2 (µg/m³)	AQD ITSL (µg/m³)	HQ
River Rouge	Manganese (TSP)	65	65	0.269	0.0750	0.05	1.50
S. Delray	Benzene	60	60	159	21.6	30	0.72
Allen Park	Manganese (TSP)	71	71	0.107	0.0299	0.05	0.60
N.E. Detroit	Manganese (TSP)	68	68	0.0806	0.0261	0.05	0.52
N. Delray	Acrylonitrile	26	12	2.97	0.914	2	0.46
N. Delray	Naphthalene	29	29	13.6	1.26	3	0.42
Southfield	Manganese (TSP)	59	59	0.0488	0.0162	0.05	0.32
Allen Park	Acetonitrile	33	17	238	19.2	60	0.32
N. Delray	Acetaldehyde	29	29	9.26	2.17	9	0.24
River Rouge	Acetaldehyde	31	30	9.29	2.11	9	0.23
Dearborn	Acetaldehyde	324	318	12.1	2.09	9	0.23
Dearborn	Naphthalene	49	49	10.5	0.631	3	0.21
N.E. Detroit	Acetaldehyde	61	61	3.40	1.78	9	0.20
S. Delray	Acetaldehyde	51	51	4.01	1.72	9	0.19
Ypsilanti	Manganese (TSP)	18	18	0.0229	0.00915	0.05	0.18
Southfield	Acetaldehyde	61	61	2.94	1.59	9	0.18
Allen Park	Acetaldehyde	61	59	3.57	1.54	9	0.17
River Rouge	Naphthalene	30	30	2.41	0.486	3	0.16
N.E. Detroit	Naphthalene	26	26	1.24	0.391	3	0.13
Ypsilanti	Acetaldehyde	29	29	1.82	1.09	9	0.12
Allen Park	Naphthalene	29	29	0.796	0.316	3	0.11
Houghton Lake	Manganese	30	30	0.03	0.005	0.05	0.11

Figure 10: HQs by Chemical and Site



* SVOCs, including Naphthalene were not sampled at Ypsilanti or Houghton Lake.
** Canister samples of VOCs, including Benzene, Acrylonitrile and Acetonitrile were not valid or were not sampled at NE Detroit, Ypsilanti or Houghton Lake.

9.2.2 Chronic Noncancer Risk for Each Chemical

Tables 32 - 37 provide those chemicals with HQs > 0.1, sorted by chemical. These tables indicate that acetaldehyde, manganese (TSP), and naphthalene were more frequently found to have HQs of 0.1 or greater, and that manganese most frequently (four sites) had HQs of 1 or greater.

Table 32: Acetaldehyde HQs exceeding 0.1

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	MAX1 (µg/m³)	AVG ND=MDL/2 (µg/m³)	AQD ITSL (µg/m³)	ITSL AVG TIME	HQ
N. Delray	Acetaldehyde	29	29	9.26	2.17	9	24 hr	0.24
River Rouge	Acetaldehyde	31	30	9.29	2.11	9	24 hr	0.23
Dearborn	Acetaldehyde	324	318	12.1	2.09	9	24 hr	0.23
N.E. Detroit	Acetaldehyde	61	61	3.40	1.78	9	24 hr	0.20
S. Delray	Acetaldehyde	51	51	4.01	1.72	9	24 hr	0.19
Southfield	Acetaldehyde	61	61	2.94	1.59	9	24 hr	0.18
Allen Park	Acetaldehyde	61	59	3.57	1.54	9	24 hr	0.17
Ypsilanti	Acetaldehyde	29	29	1.82	1.09	9	24 hr	0.12

Table 33: Acetonitrile HQs exceeding 0.1

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	MAX1 (µg/m³)	AVG ND=MDL/2 (µg/m³)	AQD ITSL (µg/m³)	ITSL AVG TIME	HQ
Allen Park	Acetonitrile	33	17	238	19.2	60	24 hr	0.32

Table 34: Acrylonitrile HQs exceeding 0.1

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	MAX1 (µg/m³)	AVG ND=MDL/2 (µg/m³)	AQD ITSL (µg/m³)	ITSL AVG TIME	HQ
N. Delray	Acrylonitrile	26	12	2.97	0.914	2	24 hr	0.46

Table 35: Benzene HQs exceeding 0.1

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	MAX1 (µg/m³)	AVG ND=MDL/2 (µg/m³)	AQD ITSL (µg/m³)	ITSL AVG TIME	HQ
S. Delray	Benzene	60	60	159	21.6	30	24 hr	0.72

Table 36: Manganese (TSP) HQs exceeding 0.1

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	MAX1 (µg/m³)	AVG ND=MDL/2 (µg/m³)	AQD ITSL (µg/m³)	ITSL AVG TIME	HQ
S. Delray	Manganese (TSP)	65	65	1.94	0.274	0.05	24 hr	5.47
Dearborn	Manganese (TSP)	70	70	1.19	0.198	0.05	24 hr	3.95
N. Delray	Manganese (TSP)	65	65	0.188	0.0930	0.05	24 hr	1.86
River Rouge	Manganese (TSP)	65	65	0.269	0.0750	0.05	24 hr	1.50
Allen Park	Manganese (TSP)	71	71	0.107	0.0299	0.05	24 hr	0.60

Table 36: Manganese (TSP) HQs exceeding 0.1

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	MAX1 (µg/m³)	AVG ND=MDL/2 (µg/m³)	AQD ITSL (µg/m³)	ITSL AVG TIME	HQ
N.E. Detroit	Manganese (TSP)	68	68	0.0806	0.0261	0.05	24 hr	0.52
Southfield	Manganese (TSP)	59	59	0.0488	0.0162	0.05	24 hr	0.32
Ypsilanti	Manganese (TSP)	18	18	0.0229	0.00915	0.05	24 hr	0.18
Houghton Lake	Manganese(TSP)	30	30	0.03	0.005	0.05	24 hr	0.11

Table 37: Naphthalene HQs exceeding 0.1

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	MAX1 (µg/m³)	AVG ND=MDL/2 (µg/m³)	AQD ITSL (µg/m³)	ITSL AVG TIME	HQ
S. Delray	Naphthalene	58	58	93.3	18.4	3	24 hr	6.13
N. Delray	Naphthalene	29	29	13.6	1.26	3	24 hr	0.42
Dearborn	Naphthalene	49	49	10.5	0.631	3	24 hr	0.21
River Rouge	Naphthalene	30	30	2.41	0.486	3	24 hr	0.16
N.E. Detroit	Naphthalene	26	26	1.24	0.391	3	24 hr	0.13
Allen Park	Naphthalene	29	29	0.796	0.316	3	24 hr	0.11

9.2.2.1 Hazard Characterization For Manganese

Manganese was identified as a compound of concern for chronic, non-cancer hazard at S. Delray, Dearborn, N. Delray and River Rouge. Manganese is an essential dietary nutrient as well as a potent neurotoxin at high concentrations. There is a good deal of human data on chronic occupational exposure via inhalation to high levels of manganese. The critical effect is neurological impairment. In particular, "manganism," a syndrome that presents with symptoms similar to that of idiopathic Parkinson's disease, has been studied extensively. This syndrome can be clinically differentiated from Parkinson's disease and is primarily the result of inhalation of large amounts of manganese-laden dust or fumes in the workplace (Finley, 2004; IRIS, 2004).[16]

Other reported effects of manganese toxicity at sufficiently high exposure levels and duration include male sexual dysfunction, respiratory effects and even aggressive behavior (Finley, 2004; Hudnell and Mergler, 1999) (also see footnote 16). There have also been reports on the interactive neurotoxic effects of manganese exposure and heavy alcohol consumption (Sassine et al., 2002). Studies on developmental effects in animals and humans from maternal inhalation exposure to manganese are limited. The requirement for dietary manganese during pregnancy, however, is well documented.

Although exposure to the general population is largely through the diet, inhalation is a crucial route of human exposure since inhaled manganese that enters systemic circulation is transported via the blood directly to the brain, bypassing the liver and the opportunity for first-pass hepatic clearance (see footnote 16). The inhalation toxicity of manganese is generally a function of solubility, particle size and dosimetry and subsequent pharmacokinetics. Bioavailability of manganese is dependent upon the route of exposure, particle size, and species of manganese to which the person is exposed. Particle size distribution in particular is an important consideration for occupational exposure studies because the proportion of total

[16] Additional information is available on the USEPA's IRIS website at http://www.epa.gov/iris.

dust or fume that is respirable can vary widely across different occupational settings (Clewell et al., 2003).

USEPA (IRIS, 2004) assumes that particles ≤ 10 µm are generally "bioavailable" while inhaled particles that deposit in the pulmonary region (fine fraction < 2.5 µm) are 100% bioavailable. This is in contrast to approximately 3 to 10% absorption via the GI tract (IRIS, 2004). The RfC is set considerably lower than the RfD, reflecting the differential toxicity of manganese by route. Some inhalation studies in experimental animals have reported that other neurotoxic metals such as aluminum and cadmium can be directly transported to the brain olfactory bulb via nasal olfactory pathways (Perl and Good, 1987; Evans and Hastings, 1992). Dorman et al. (2002) found that transport of manganese phosphate along olfactory neurons was a relevant pathway in the rat brain. A finding of altered olfactory perception in humans exposed to manganese (Mergler et al., 1994) lends support to the relevance of this pathway in humans. A recent Health Effects Institute (HEI) report concluded that manganese has the potential to accumulate in the brain due to its slow elimination by diffusion from this tissue (Yokel and Crossgrove, 2004).

The most appropriate chronic health benchmark value for manganese is the USEPA (IRIS, 2004) RfC of 0.05 µg/m^3. This value is based on human data from occupational exposure to chronic low levels of manganese dioxide, manganese oxides and salts. Critical endpoints were impairment of neurobehavioral function, especially speed and coordination of motor function. Reported LOAELS from these studies were 150 and 970 µg/m^3 with mean exposure durations ranging from 5.3 to 16.7 years (IRIS, 2004). The LOAEL chosen as the basis of the RfC was derived from an occupational-lifetime integrated respirable dust concentration of manganese dioxide. Roels et al. (1992) did not present data on particle size or purity in the published report, but a median cut point of 5 µm was noted for the respirable dust fraction. By definition, a median value is a 50th percentile value which defines half of the distribution as particles below 5 µm and half above 5 µm. Given these factors, the critical neurological effects observed at the LOAEL do not necessarily reflect effects only from exposure to particles < 5 µm. In addition, given the lack of available data on the relative toxicity of different forms and compounds of manganese, the RfC does not distinguish between the various forms to which an individual might be exposed.

The adjusted LOAEL (human equivalent concentration or HEC) was reported as 50 µg/m^3. A total UF of 1,000 was applied in deriving the RfC – 10 for intraspecies variation (human to sensitive human), 10 for use of a LOAEL rather than a NOAEL, and 10 for database limitations. USEPA described database limitations as reflecting less-than-chronic periods of exposure, lack of developmental data, and unquantified differences in the toxicity of different forms of manganese. USEPA also stated that certain subpopulations, such as children, pregnant women, elderly persons, iron or calcium-deficient individuals, and individuals with liver impairment may have an increased potential for excessive manganese body burdens due to either increased absorption and/or altered clearance mechanisms, which may be of particular importance for those exposed to manganese by multiple routes (IRIS, 2004).

Manganese (TSP) was collected for the Detroit Pilot Project. TSP data was collected so that historical trends could be assessed since data collection in Michigan has been predominantly for TSP metals. For some sites, speciated $PM_{2.5}$ and PM_{10} data has also been collected over the years as part of MDEQ's annual air quality monitoring efforts. For Dearborn (one of the highest reported sites for manganese during the Detroit Pilot Project) recent data show that, on

average, approximately half of the ambient manganese concentration is in the PM_{10} fraction and exceeds the RfC (see **Figures 11** and **12**).[17]

Figure 11: Maximum Manganese Trends at Dearborn

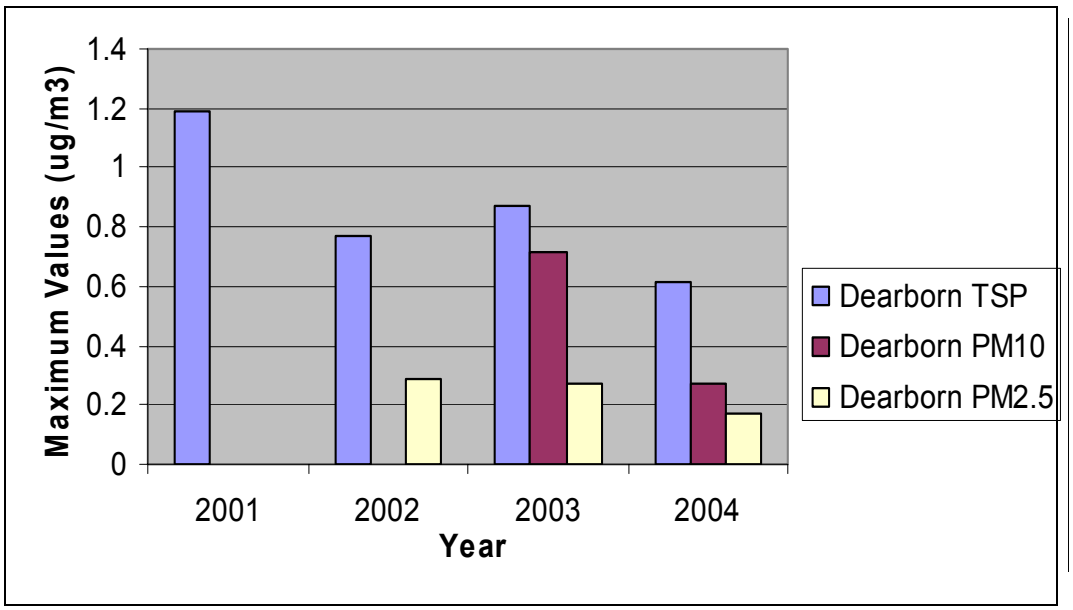
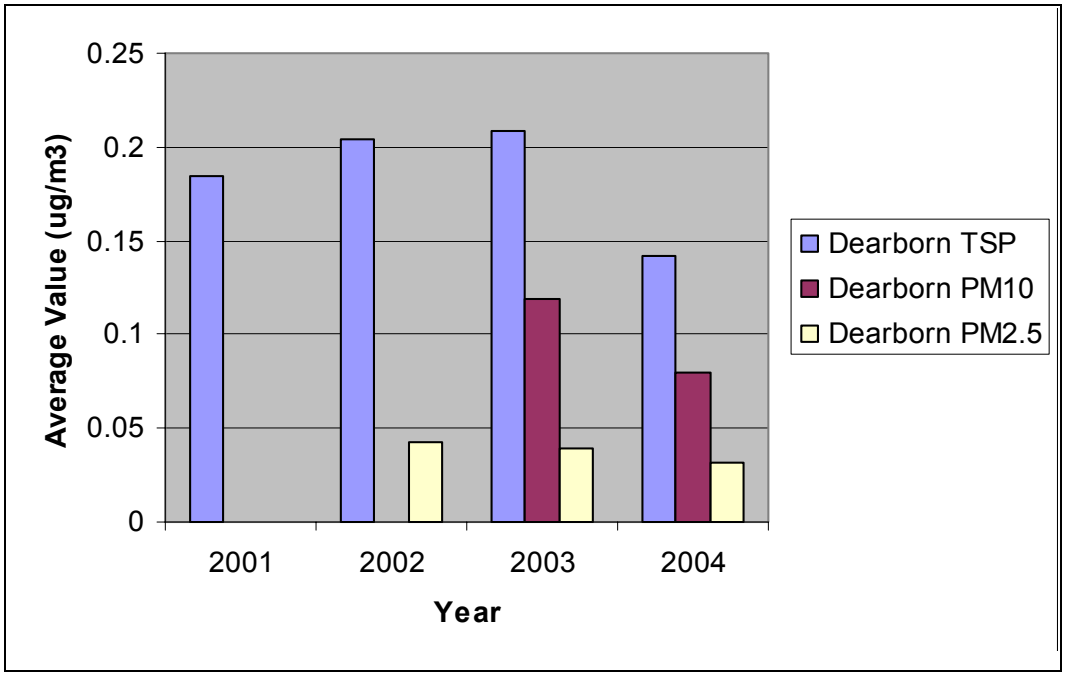


Figure 12: Average Manganese Trends at Dearborn



For these four sites, the HQ for manganese was > 1 (see **Table 38**). This raises some concern about the potential for increased hazard of adverse health effects at these sites. The HQ approach to hazard assessment is essentially a screening tool wherein an HQ < 1 is a value

[17] Note: The four highest measured concentrations of manganese TSP, PM_{10}, and $PM_{2.5}$ at Dearborn for these years all exceed the RfC of 0.05 $\mu g/m^3$ (Range = 0.076 to 1.19 $\mu g/m^3$).

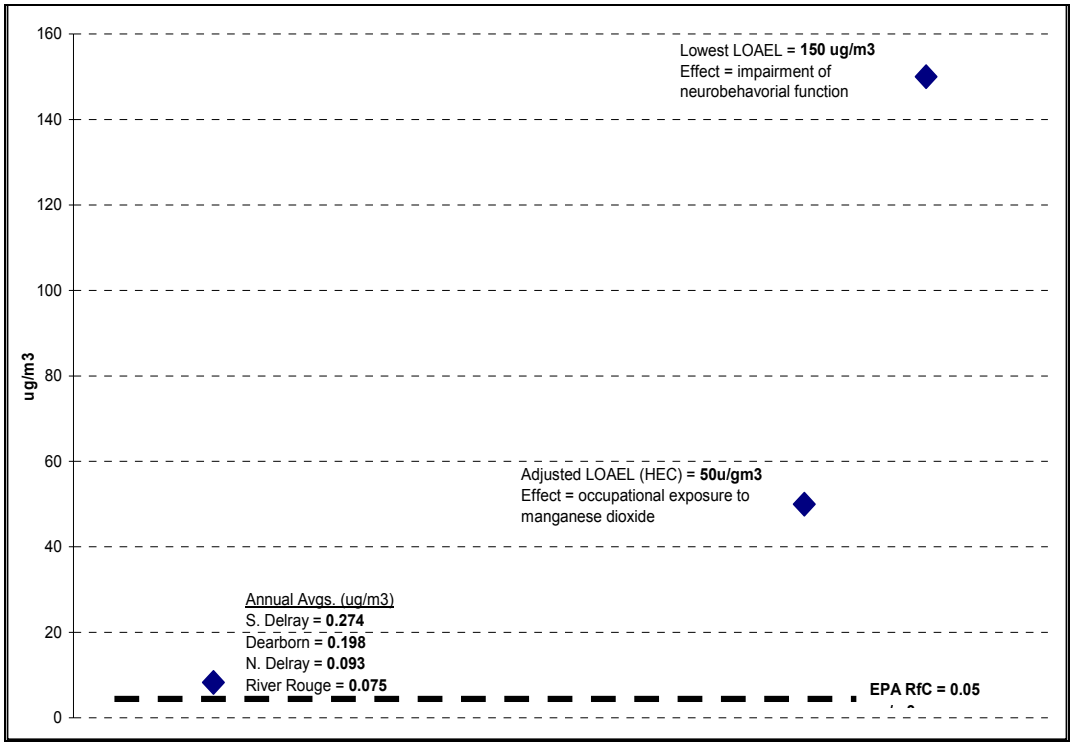
representing an exposure without significant risk while an HQ > 1 triggers a more thorough review of the hazard, dose-response, and health benchmark information, as related to the estimated exposure concentration, to determine the nature and magnitude of potential hazard. In other words, the magnitude of the HQ is not directly correlated with the magnitude of an effect level. An HQ > 1 is simply an indication that there is an increased *potential* for health risk with increasing exposure. The HQ approach and its inherent assumptions and uncertainties are detailed in **Section 8.2**.

Table 38: Sites with HQ > 1 for Manganese

SITE	CHRONIC, NON-CANCER HQ FOR MANGANESE
S. Delray	5.47
Dearborn	3.95
N. Delray	1.86
River Rouge	1.5

The levels of manganese at Dearborn and S. Delray are about an order of magnitude higher than at other sites both within the Detroit Pilot Project and nationally (see **Section 13**). However, these levels are about 180 times less than the adjusted LOAEL used to derive the RfC and approximately 550-fold lower than the lowest actual exposure level at which effects were noted in the RfC key study (**Figure 13** and **Appendix J** provide a graphical display of this information). The total UF of 1,000 would seem to include a considerable margin of safety. However, the factor of 10 for database uncertainty, given the fact that the RfC is based on human data, indicates that crucial information regarding human toxicity from inhalation exposure is lacking and, therefore, effects at or above the RfC are not well understood.

Figure 13: Detroit Manganese Concentrations Compared to Benchmark and RfC LOAEL



The uncertainty with regard to neurodevelopmental effects is encompassed in the factor of 10 for database deficiencies. Although the critical effect on which the RfC is based is neurological impairment, there is no data on neurodevelopmental toxicity of manganese in humans specific to the inhalation route of exposure. One recent epidemiological study in humans reported an association between neurodevelopmental effects and manganese levels *in utero*. Takser et al. (2003) conducted a prospective epidemiological study in 247 healthy pregnant women and their infants to determine the long-term effect of *in utero* manganese levels on child's psychomotor development. Exposure was characterized as "environmental" and therefore samples collected reflect a total body burden dose metric rather than an oral or inhalation exposure level. The authors concluded that fetal development is a vulnerable period for environmental exposure to manganese. Studies in experimental animals generally support the notion that exposure to excess manganese *in utero* is associated with impaired neurodevelopment but the exposures in these studies are largely to excess dietary manganese and exposure levels are generally higher than those reported in human studies.

Based on the known toxicokinetics of manganese by inhalation, it would follow that an inhaled maternal dose would be more readily bioavailable for transfer to fetus than a dietary dose. However, the percentage of an inhaled dose that is transferred from mother to fetus is unknown. Manganese intake by GI absorption is regulated under tight homeostatic control although levels are known to increase during pregnancy (Takser et al., 2004). Once in maternal circulation, manganese readily crosses the placenta (Finely, 2004). USEPA states in its IRIS justification document that "…some evidence suggests that the neonate's inability to maintain manganese homeostasis is due to a limitation in the elimination of manganese rather than in its gastrointestinal absorption (Bell et al., 1989), which would suggest a potentially greater vulnerability of young individuals to excessive manganese exposure regardless of the route…" (IRIS, 2004). Therefore, there is insufficient data to quantify or qualify the hazard for developmental neurotoxicity of manganese though what little data is available is suggestive. Like other persistent bioaccumulative toxins (PBT) metals (e.g. lead, mercury), total body burden of manganese may play a role in its toxicity.

In conclusion, the available information is not adequate to definitively evaluate potential health effects due to exposure to manganese at sites with levels exceeding the RfC. The monitored levels are below levels known to cause adverse effects in scientific studies, although data base gaps and deficiencies raise uncertainties regarding these findings. Use of monitored TSP concentrations for comparison to the health benchmark may result in an overestimation of the health hazard from inhalation exposure since not all fractions will be readily bioavailable. However, when the exposure scenario is unknown, USEPA assumes that it is at least equivalent to the exposure scenario from the critical study for risk assessment purposes. This assumption may underestimate or overestimate the health risk. More recent data at Dearborn demonstrates that the respirable fraction accounts for approximately half of the TSP and that the PM_{10} fraction alone exceeds the RfC. Evidence from inhalation studies in experimental animals and at least one study of manganese body burden in humans does suggest that *accumulation* of manganese in the brain can occur with chronic, low-level environmental exposure. It is unclear what effects such exposure might have in healthy adults, but the potential for bioaccumulation increases the uncertainty with regard to toxicity from exposures at or above the RfC. Also, there is considerable uncertainty with regard to the potential of inhaled manganese as a neurodevelopmental toxicant. Therefore, the reduction in the margin of safety between the monitored levels and known effects is a concern. Considering the above findings, and given the seriousness of the potential neurotoxic effects of manganese, prudent public health policy would suggest focusing on the reduction of ambient manganese levels as a priority.

9.2.2.2 Discussion of Naphthalene HQs

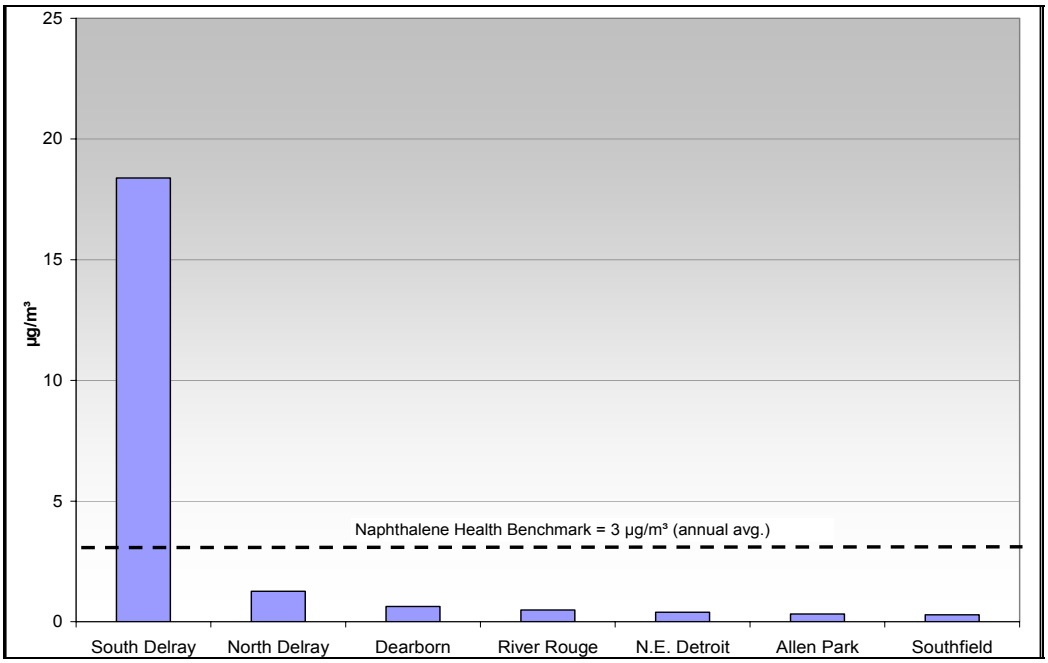
The naphthalene HQs (shown in **Table 37**) are based upon the annual average ITSL of 3 µg/m^3, which is the same value as the USEPA RfC. In its IRIS database, USEPA described the key study used to derive the RfC as well as the critical effects, dose-response, and the UFs. The key study for naphthalene was a NTP (1992) bioassay, in which groups of male and female mice were exposed (whole body) to naphthalene vapors at concentrations of 0, 52, or 157 milligrams per cubic meter (mg/m^3) for 6 hrs/day, 5 days/week for two years. Statistically significant increases in incidences of non-cancer lesions were found in the lung and nose of male and female mice at both 52 and 157 mg/m^3. Observed effects included chronic inflammation of the lung, chronic nasal inflammation, hyperplasia of the respiratory epithelium in the nose, and metaplasia of the olfactory epithelium. The lung inflammation in the exposed mice was described as a chronic inflammatory response with the formation of granulomas. The LOAEL was identified as 52 mg/m^3 (52,000 µg/m^3). A chronic inhalation NOAEL based on respiratory effects was not identified from this study, since the lowest dose tested produced adverse effects.

In 1998, when USEPA derived the RfC, the mouse LOAEL of 52 mg/m^3 was time-adjusted for 6hr/24hr and 5days/7days to obtain a $LOAEL_{ADJ}$[18] of 9.3 mg/m^3 (9,300 µg/m^3) for nasal effects (hyperplasia in respiratory epithelium and metaplasia in olfactory epithelium). USEPA considered that the nasal effects from inhalation exposure to naphthalene were extra-respiratory effects of a category 3 gas, as defined in the USEPA guidance for deriving RfCs (USEPA, 1994). This was based on naphthalene's low water solubility and low reactivity in the extrathoracic and tracheobronchial regions. USEPA hypothesized that naphthalene-related effects on the nasal epithelium are expected to result following absorption of naphthalene and metabolism to reactive oxygenated metabolites, rather than being a result of direct contact. This hypothesis is supported by data on naphthalene metabolism indicating that toxic effects on the respiratory tract are due to a naphthalene metabolite that may be formed either in the liver or in the respiratory tract. For example, necrosis of bronchial epithelial (Clara) cells in mice (O'Brien et al., 1985 and 1989; Tong et al., 1981) and necrosis of olfactory epithelium in mice, rats, and hamsters (Plopper et al., 1992) occurs following intraperitoneal injection of naphthalene.

In the derivation of the RfC, after adjusting for intermittent exposure, the $LOAEL_{ADJ}$ was converted to a HEC by multiplying the adjusted concentration by the ratio of mouse:human blood/gas partition coefficients. However, because the blood/gas coefficients for naphthalene were not available, the default ratio of 1 was used. USEPA took the $LOAEL_{ADJ}$ of 9.3 mg/m^3 and divided it by an UF of 3000 which was comprised of 10 to extrapolate from mice to humans; 10 to protect sensitive humans; 10 to extrapolate from a LOAEL to a NOAEL; and 3 for database deficiencies, including the lack of a two-generation reproductive toxicity study and chronic inhalation data for other animal species. The annual average ambient air concentrations used to calculate the HQs are shown in **Figure 14**.

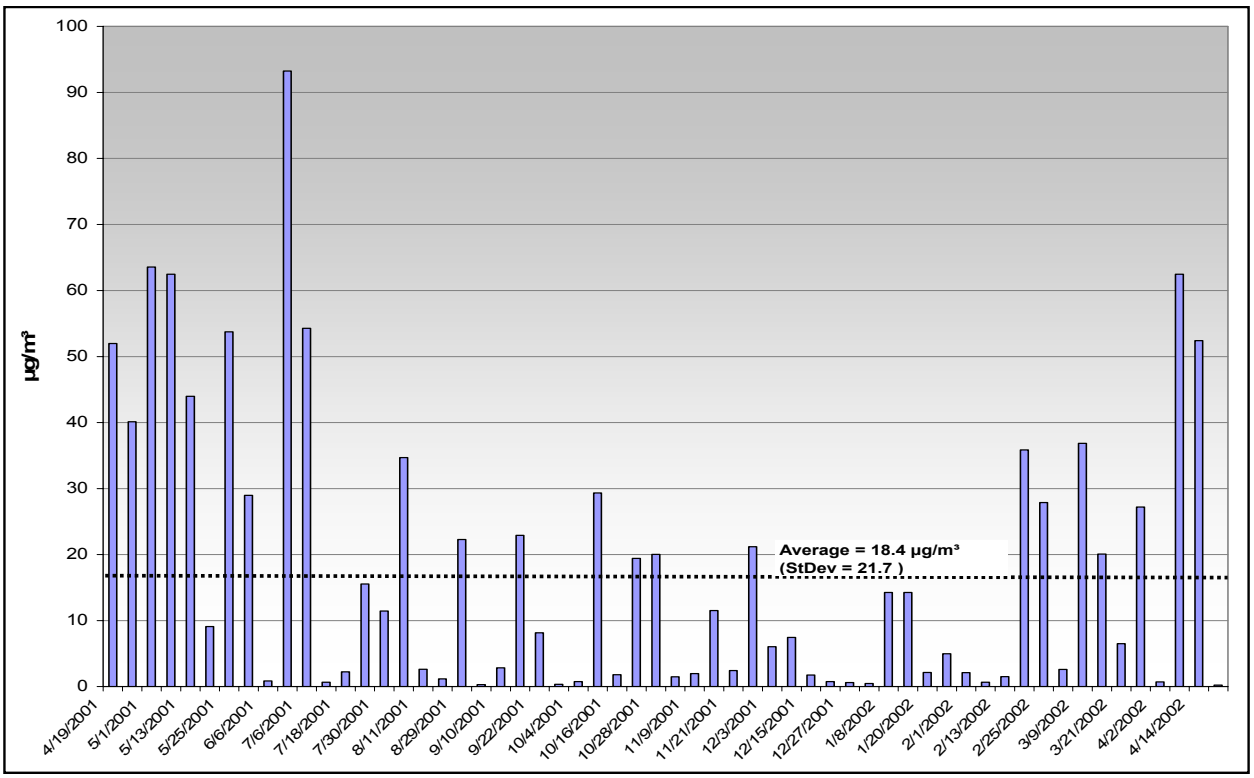
[18] LOAEL adjusted to continuous exposure duration from an intermittent regimen by hour/day and days/7days.

Figure 14: Annual Average Ambient Air Concentrations of Naphthalene 2001-2002



Because the annual average air concentration of naphthalene at the S. Delray monitor exceeds the ITSL and is so much larger than the average air concentrations measured at the other DATI monitoring sites, the air concentrations at S. Delray were examined in greater detail. The daily (24-hr) concentrations measured at S. Delray ranged from 0.2 to 93 µg/m³ (see **Figure 15**). There were 34 of 58 measurements that exceeded the ITSL of 3 µg/m³. On the days that the air concentration exceeded 3 µg/m³, 13 (38%) exceed the ITSL by 10 times or more.

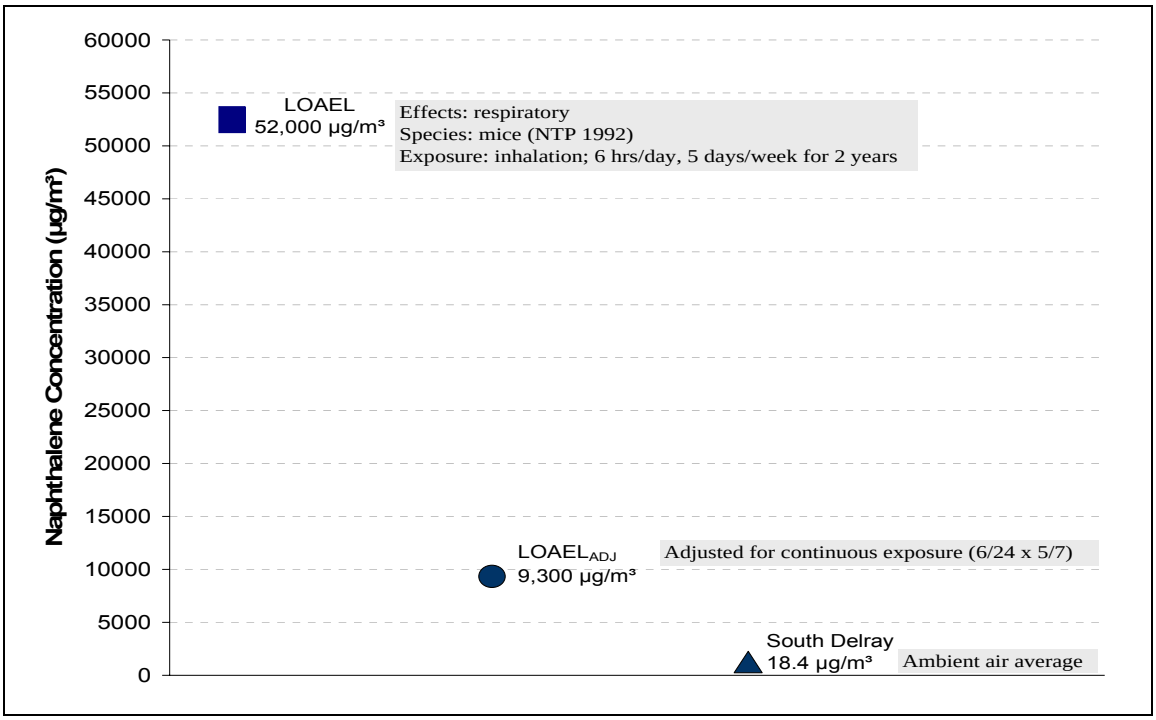
Figure 15: Daily Air Concentrations of Naphthalene Measured at S. Delray



The data in **Figure15** shows that the monitored levels at S. Delray vary significantly from day to day and appear to show seasonal differences. This type of a pattern of exposure is significantly different than that used in the mouse bioassay (NTP, 1992) that was used to establish the inhalation RfC. This variation in exposure pattern adds additional uncertainty in interpreting the significance of an annual average concentration that exceeds the RfC by 6-fold.

The average ambient air concentration at S. Delray was also compared to the effects levels observed in the NTP (1992) mouse bioassay (see **Figure 16**). This concentration (18.4 µg/m^3) was about 500 times below the adjusted exposure level where significant adverse effects were observed in the mouse. While the large difference between the monitored levels at S. Delray and effect levels in animals suggests a lowering of concern for naphthalene, it must be tempered with the significant uncertainty that exists in the data. These uncertainties include the lack of adequate data in humans, lack of data regarding potential sensitive subpopulations, and lack of data that characterizes the no adverse effect level. (**Appendix J** provides a graphical display of this information.) In addition, potential interactive effects due to co-exposure to the criteria pollutants such as ozone and PM which also impact the respiratory system add additional uncertainty.

Figure 16: Comparison of Average Ambient Air Concentration of Naphthalene at S. Delray to the Animal Effect Levels



In conclusion, while the available information suggests that naphthalene exposure at the S. Delray site does not present any immediate public health problem, the reduction in the margin of safety between monitored levels and the known effect levels is a concern. Efforts should be focused on the identification of sources of naphthalene impacting the S. Delray site, and pursuing reductions that address the health concerns.

9.2.3 Chronic Noncancer Hazard for Each Site

Tables 39 - 47 present the chronic noncancer HQs for all HQs of 0.1 or greater at each site. These tables indicate that several sites had multiple substances with HQs of 0.1 or greater. The

Houghton Lake "background" site had only one, but VOC data were not available. The Ypsilanti urban comparison site had two substances which exceeded a HQ of 0.1 by a small amount, but VOC data were not available. All of the Detroit area sites had multiple HQs exceeding 0.1, with variability in magnitude.

Table 39: Allen Park Site Chemicals with HQs of 0.1 or Greater

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	MAX1 (µg/m³)	AVG ND=MDL/2 (µg/m³)	AQD ITSL (µg/m³)	HQ
Allen Park	Manganese (TSP)	71	71	0.107	0.0299	0.05	0.60
	Acetonitrile	33	17	238	19.2	60	0.32
	Acetaldehyde	61	59	3.57	1.54	9	0.17
	Naphthalene	29	29	0.796	0.316	3	0.11

Table 40: Dearborn Site Chemicals with HQs of 0.1 or Greater

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	MAX1 (µg/m³)	AVG ND=MDL/2 (µg/m³)	AQD ITSL (µg/m³)	HQ
Dearborn	Manganese (TSP)	70	70	1.19	0.198	0.05	3.95
	Acetaldehyde	324	318	12.1	2.09	9	0.23
	Naphthalene	49	49	10.5	0.631	3	0.21

Table 41: S. Delray Site Chemicals with HQs of 0.1 or Greater

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	MAX1 (µg/m³)	AVG ND=MDL/2 (µg/m³)	AQD ITSL (µg/m³)	HQ
S. Delray	Naphthalene	58	58	93.3	18.39	3	6.13
	Manganese (TSP)	65	65	1.94	0.274	0.05	5.47
	Benzene	60	60	159	21.6	30	0.72
	Acetaldehyde	51	51	4.01	1.72	9	0.19

Table 42: N.E. Detroit Site Chemicals with HQs of 0.1 or Greater

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	MAX1 (µg/m³)	AVG ND=MDL/2 (µg/m³)	AQD ITSL (µg/m³)	HQ
N.E. Detroit	Manganese (TSP)	68	68	0.0806	0.0261	0.05	0.52
	Acetaldehyde	61	61	3.40	1.78	9	0.20
	Naphthalene	26	26	1.24	0.391	3	0.13

Table 43: Houghton Lake Site Chemicals with HQs of 0.1 or Greater

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	MAX1 (µg/m³)	AVG ND=MDL/2 (µg/m³)	AQD ITSL (µg/m³)	HQ
Houghton Lake	Manganese (TSP)	30	30	0.03	0.005	0.05	0.11

Table 44: Southfield Site Chemicals with HQs of 0.1 or Greater

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	MAX1 (µg/m³)	AVG ND=MDL/2 (µg/m³)	AQD ITSL (µg/m³)	HQ
Southfield	Manganese (TSP)	59	59	0.0488	0.0162	0.05	0.32
	Acetaldehyde	61	61	2.94	1.59	9	0.18

Table 45: River Rouge Site Chemicals with HQs of 0.1 or Greater

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	MAX1 (µg/m³)	AVG ND=MDL/2 (µg/m³)	AQD ITSL (µg/m³)	HQ
River Rouge	Manganese (TSP)	65	65	0.269	0.0750	0.05	1.50
	Acetaldehyde	31	30	9.29	2.11	9	0.23
	Naphthalene	30	30	2.41	0.486	3	0.16

Table 46: N. Delray Site Chemicals with HQs of 0.1 or Greater

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	MAX1 (µg/m³)	AVG ND=MDL/2 (µg/m³)	AQD ITSL (µg/m³)	HQ
N. Delray	Manganese (TSP)	65	65	0.188	0.0930	0.05	1.86
	Acrylonitrile	26	12	2.97	0.914	2	0.46
	Naphthalene	29	29	13.6	1.26	3	0.42
	Acetaldehyde	29	29	9.26	2.17	9	0.24

Table 47: Ypsilanti Site Chemicals with HQs of 0.1 or Greater

SITE NAME	CHEMICAL NAME	NUM OBS	OBS >MDL	MAX1 (µg/m³)	AVG ND=MDL/2 (µg/m³)	AQD ITSL (µg/m³)	HQ
Ypsilanti	Manganese (TSP)	18	18	0.0229	0.00915	0.05	0.18
	Acetaldehyde	29	29	1.82	1.09	9	0.12

9.2.4 Chronic Noncancer Target Organ Specific Hazard Index (TOSHI)

In the initial step to evaluate concerns for potential effects of complex mixtures, a Total HI, regardless of the toxicological endpoints of concern, was first considered for each site. This resulted in Total HIs exceeding a value of 1 for most of the Detroit sites, but not at the N.E. Detroit site (which lacked VOCs data) or at the Southfield site, nor at the Houghton Lake (lacking VOCs) and Ypsilanti (lacking VOCs) comparison sites. The appropriate next step after this screening step is to further evaluate HIs for potential dose additivity on the basis of the specific toxicological endpoints, as follows in the next subsection.

For TOSHIs with a value of 1 or less, a lack of adverse effect may be presumed. For TOSHIs exceeding a value of 1, harmful effects should not be presumed, but safety also cannot be presumed without further evaluation. The greater the TOSHI value exceeding 1, the greater the concern for potential additive effects, although there is no “bright line” for that concern. TOSHI values > 1 warranted scrutiny and further assessment in order to develop appropriate risk characterizations. The hazard assessment in that situation proceeded with an assessment of the HIs for neurological effects and respiratory effects, focusing on the HQs which were the primary COPCs for those TOSHIs, in an effort to characterize the potential for health impacts.

9.2.4.1 Neurological Effects

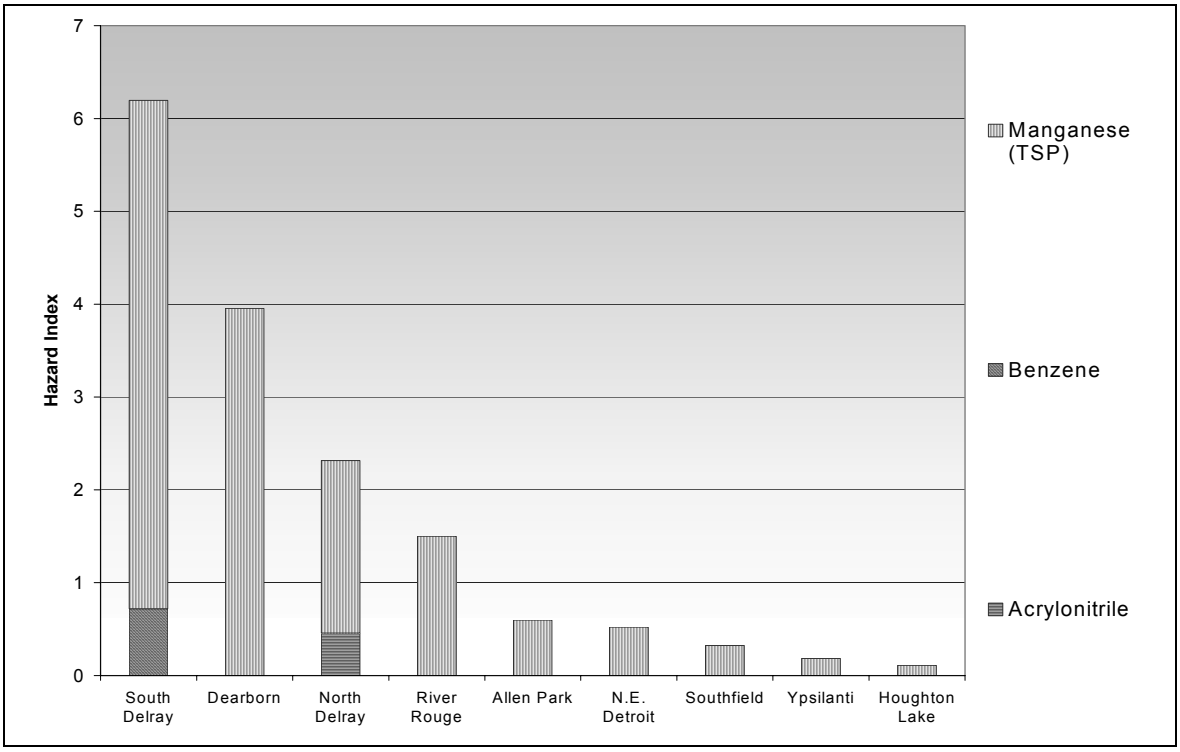
Three chemicals with HQs of 0.1 or greater have the potential to cause neurological effects. Those three chemicals are manganese, benzene, and acrylonitrile. The evaluation of potential additive neurological effects of these chemicals may begin with the consideration of the TOSHIs for each site as shown **Table 48** and **Figure 17**. Four sites had neurological TOSHI values of 1

or greater: S. Delray (6.19), Dearborn (3.95), N. Delray (2.32), and River Rouge (1.5). For each of those sites, the HQ for manganese was the predominant contributor (HQs ranged from 1.5 to 6.19). Benzene also contributed significantly at the S. Delray site (HQ of 0.72), and acrylonitrile also contributed at the N. Delray site (HQ of 0.46). The significance of the manganese HQs greater than 1 and concern with potential neurological effects has already been discussed in **Section 9.2.2.1**.

Table 48: TOSHI – Neurological Effects

CHEMICAL	ALLEN PARK	DEARBORN	HOUGHTON LAKE	S. DELRAY	N.E. DETROIT	SOUTH FIELD	RIVER ROUGE	N. DELRAY	YPSILANTI
Acrylonitrile								0.46	
Benzene				0.72					
Manganese	0.6	3.95	0.11	5.47	0.52	0.32	1.5	1.86	0.18
Total	**0.6**	**3.95**	**0.11**	**6.19**	**0.52**	**0.32**	**1.5**	**2.32**	**0.18**

Figure 17: TOSHI - Neurological Effects



9.2.4.2 Respiratory Effects

Four chemicals with HQs of 0.1 or greater at one or more sites have the potential to cause or contribute to respiratory effects. Those chemicals include naphthalene, acrylonitrile, acetonitrile, and acetaldehyde. The initial consideration of potential additive respiratory effects of these chemicals begins with a viewing of the TOSHIs for each site in the following **Figure 18** and **Table 49**. This indicates that a TOSHI of 1 was exceeded at the S. Delray site (6.32) and the N. Delray site (1.12). At the S. Delray site, the TOSHI of 6.32 is composed of HQs of 6.13 (naphthalene) and 0.19 (acetaldehyde). At the N. Delray site, the TOSHI of 1.12 was composed of HQs of 0.46 (acrylonitrile), 0.42 (naphthalene), and 0.24 (acetaldehyde). The annual average

air concentration of naphthalene forms the overwhelming majority (97%) of the respiratory TOSHI at the S. Delray monitoring site. At the N. Delray site, naphthalene comprises approximately 38% of the respiratory TOSHI. Since naphthalene plays such a large role in the respiratory TOSHI for S. Delray, the remainder of this section will focus on describing the health effects, ambient air concentrations and the potential for naphthalene to contribute to adverse respiratory health outcomes.

Figure 18: TOSHI - Respiratory Effects

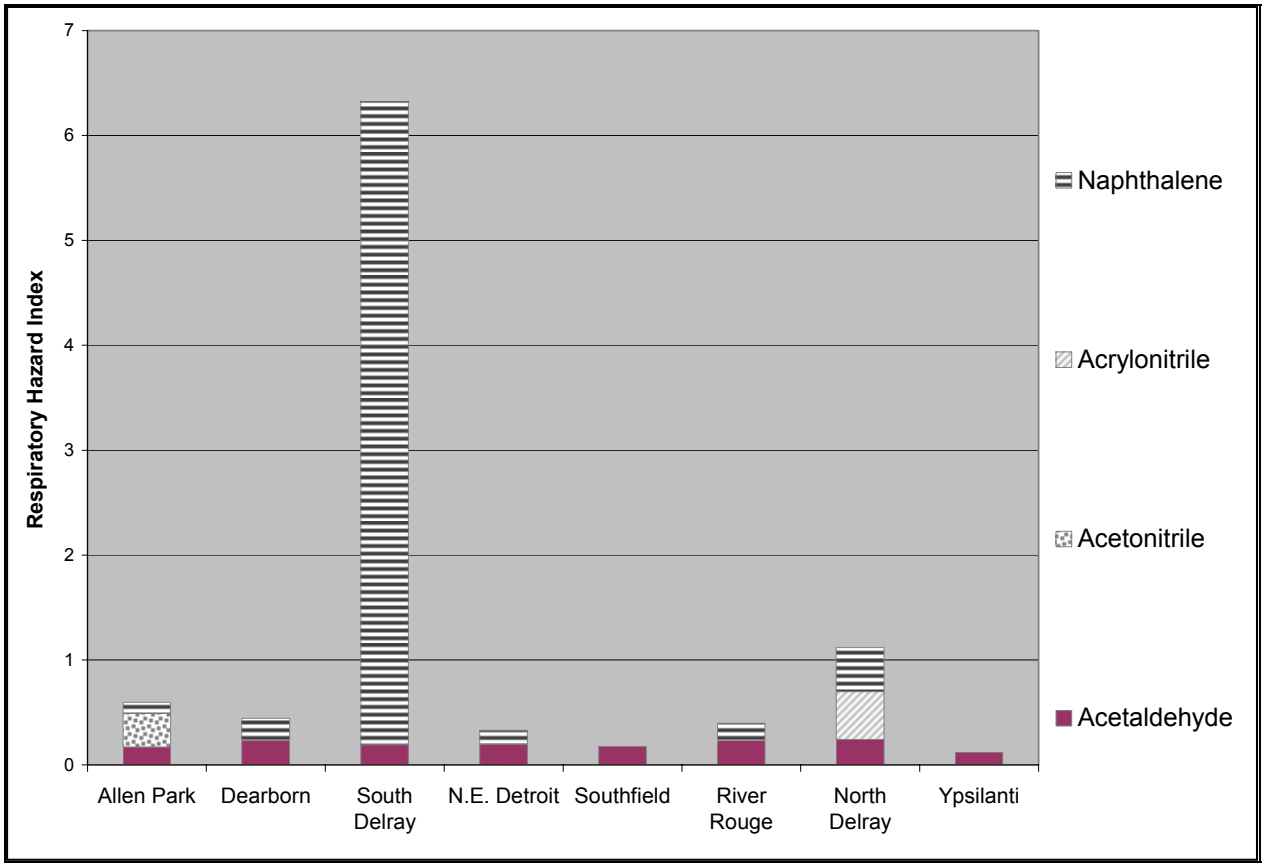


Table 49: TOSHI – Respiratory Effects

Chemical Name	Allen Park	Dearborn	S. Delray	N. E. Detroit	South field	River Rouge	N. Delray	Ypsilanti
Acetaldehyde	0.17	0.23	0.19	0.2	0.18	0.23	0.24	0.12
Acetonitrile	0.32							
Acrylonitrile							0.46	
Naphthalene	0.11	0.21	6.13	0.13		0.16	0.42	
TOSHI	**0.6**	**0.44**	**6.32**	**0.33**	**0.18**	**0.39**	**1.12**	**0.12**

Sources of uncertainty are inherent in the risk assessment process. The extrapolation of toxicity data from laboratory animal studies to human exposure scenarios is an inexact science that introduces much uncertainty into the process. However, these extrapolations are relied upon to determine exposure levels that are adequately protective of public health. To account for these data limitations, UFs are used to help ensure that even the most sensitive humans would not be adversely affected when they are exposed to concentrations at the health benchmark. The application of multiple UFs to derive a health benchmark for public health protection can result

in health benchmarks that are far below the exposures that have been shown to be toxic to certain groups of experimental animals or humans (see **Section 11** for a more complete discussion about uncertainty in the risk assessment process). With this in mind, the discussion that follows will describe the potential respiratory effects of naphthalene exposure, as well as to characterize the potential health effects from naphthalene measured in the ambient air in the Delray neighborhood.

As previously mentioned (in **Section 9.2.2.2**), the naphthalene HQs are based upon the annual average ITSL of 3 $\mu g/m^3$, which is the same value as the USEPA RfC. In the derivation of the RfC for naphthalene, USEPA used an UF of 3 for database deficiencies based on the lack of a two-generation reproductive toxicity study as well as the lack of a chronic inhalation study in another animal species. There are two reasons why, in this case, a database deficiency UF is not warranted in an analysis of the significance of the potential respiratory effects for the TOSHI assessment. First, as just mentioned, one of USEPA's reasons for using this factor included a lack of a chronic study in a second mammalian species. Subsequent to the time in 1998 when the RfC was developed, NTP (2000) performed another naphthalene bioassay, however, in this later study NTP used rats instead of mice which were used in the 1992 bioassay. This newer study made use of the same two lower dose levels used in the mouse study, but added a third higher dose level of 314 mg/m^3. The same non-cancer effects in the respiratory tract were observed in both studies, with a LOAEL of 52 mg/m^3. Because a study was done in a second species of animals (rats), part of USEPA's reasoning for using an UF of 3 is lessened.

The second reason the UF for database deficiencies was used by the USEPA was because of a lack of a two-generation reproductive study. In the present context, an UF to address a concern for potential reproductive effects was deemed not appropriate for the TOSHI assessment of respiratory effects. The potential concern for the lack of reproductive data is adequately addressed in looking at the individual HQs for naphthalene as discussed in **Section 9.2.2.2**.

Additional support for removing this database deficiency UF for the respiratory TOSHI is found in USEPA's guidance for risk assessment of chemical mixtures (USEPA, 2000b). USEPA provides a procedure for deriving a HI for a toxic endpoint of concern, in which they describe the difference in derivation between an RfD and a target organ toxicity dose (TTD). In this case USEPA uses the example of a renal TTD. The same guidance could be applied to respiratory effects via the inhalation route. Specifically, USEPA states the following:

> "The evaluation of quality of the candidate toxicity studies and the choice of UFs should parallel those steps in the RfD process. One difference in the UFs concerns the factor for completeness of the database used for RfD development. For example, if no two-generation study existed for a chemical, there could be an additional UF used to obtain the RfD because the RfD must protect against all toxic effects. When developing a renal TTD, however, no additional factor would be used because the data would only include renal effects (Mumtaz et al., 1997)."

Since the rationale USEPA appropriately uses to justify an UF of 3 in the RfC is not applicable to the analysis of a TOSHI for respiratory effects, the database deficiency UF of 3 was omitted when evaluating naphthalene's respiratory effects. For the respiratory TOSHI, the naphthalene benchmark's total UF was decreased from 3000 to 1000. The HQs for naphthalene at the S. Delray and N. Delray sites then become 2.0 and 0.14, respectively. Accordingly, the TOSHIs for respiratory effects at the S. Delray and N. Delray sites are 2.2 and 0.84, respectively.

Recently, the ATSDR (2003) evaluated the non-cancer health effects of naphthalene and derived a MRL which was similar to the USEPA's RfC. The MRL for naphthalene is 0.0007 parts per million (ppm) which is converted to 3.67 $\mu g/m^3$ (rounded to 4 $\mu g/m^3$). The MRL is

based on the NTP study in rats (NTP, 2000). The ATSDR applied USEPA's methodology for deriving an RfC (USEPA, 1994) for a category 1 gas. Therefore, using the USEPA's RfC methodology for a category 1 gas and the $LOAEL_{ADJ}$ of 9.4 mg/m³ in the rat, the ATSDR calculated the $LOAEL_{HEC}$ as 0.2 ppm.[19] The calculation that ATSDR used to determine a dose equivalency between animals and humans takes into account the rate of respiration (i.e., volume of air breathed per minute, or minute volume) and the surface area of the particular region affected in the respiratory tract. The ratio of the minute volume to surface area in the animal compared to that of the human is called the Regional Gas Dose Ratio (RGDR). As previously noted, USEPA made the determination that naphthalene's effects on the nose were not related to the deposition of dose on the nasal tissue, but were dependent on the amount of naphthalene absorbed into the blood. This is consistent with USEPA's RfC methodology for category 3 gases. Since there was no information about the partitioning of naphthalene in the human or animal blood, USEPA's default approach is to assume that animal and human doses are equivalent. ATSDR's use of the RGDR dosimetric adjustment method used for calculating dose equivalency of category 1 gases can be supported by the finding that the location of the critical effects observed in the rat and mouse chronic inhalation studies occurred in the nasal passages. Hyperplasia, metaplasia, and chronic inflammation of the nasal passages all support a mechanism of action consistent with direct contact irritation, as would be expected with category 1 gases. However, the determination of naphthalene as a category 1 gas is complicated by findings of an oral dosing study (Murata et al., 1993) which showed statistically significant increases in lung pathology, including alveolar proteinosis and increased incidence of lung tumors.

The UFs used by ATSDR in the development of the MRL also differed from the USEPA in that they used a total UF of 300, whereas USEPA used 3000. ATSDR's UFs were 10 for the conversion of the LOAEL to a NOAEL (same as USEPA), 10 for the protection of sensitive individuals (same as USEPA), and 3 for extrapolation of animal data to humans. This last UF for animal to human extrapolation differs from USEPA's assessment because the $LOAEL_{HEC}$ was derived from a dosimetric adjustment, specifically a RGDR. According to USEPA's RfC methodology, when using this method for dosimetric adjustment to derive the HEC, the UF of 10 for animal to human extrapolation is decreased to 3. The ATSDR's calculation of the MRL differs from USEPA's RfC calculation in another important manner. ATSDR did not use an UF of 3 for database deficiencies. As mentioned previously, the UF of 3 was deemed inappropriate for a respiratory specific risk assessment of naphthalene. Nonetheless, the similarity of magnitude of both health benchmarks (the RfC and the MRL) provide assurance that respiratory health effects from chronic exposure to naphthalene below 3 µg/m³ are unlikely.

It should be noted that the assessment of potential interactive respiratory effects within the scope of this study included only the air toxics that were monitored specifically for the DATI. It does not include potential interactive effects with criteria pollutants (e.g., $PM_{2.5}$, ozone) which can elicit acute and chronic respiratory effects, the ambient concentrations of which have exceeded NAAQS for health protection in the Detroit area. The other respiratory system toxicants in the DATI study contribute HQs of 0.46 or less toward the respiratory TOSHI (see **Table 49**). The limited number of HQs and their limited magnitude at these sites indicate that the focus of this TOSHI assessment on naphthalene is adequately inclusive.

As mentioned in **Section 9.2.2.2**, considering the magnitude of the UF applied to the LOAEL of 9.3 mg/m³ (9300 µg/m³) to derive the RfC, it is unlikely that overt respiratory effects would be observed near the S. Delray monitor. However, the naphthalene concentration along with other environmental factors (e.g., indoor air, health status of the individual, exposure to ozone, $PM_{2.5}$),

[19] LOAEL adjusted for dosimetric differences across species to a HEC.

could contribute to respiratory health concerns. In conclusion, the available information suggests that naphthalene exposure at the S. Delray site does not present any immediate danger, although the margin of safety is reduced and potential interactive effects with criteria pollutants is a possibility.

9.3 Acute Noncancer

The potential for increased risk of acute health effects from short-term exposure to chemicals in ambient air is possible if concentrations are sufficiently high. For example, many chemicals are respiratory and mucous membrane irritants and even a one-time exposure of short duration (e.g., 0 to 24 hrs) is sufficient to cause immediate adverse effects. Evaluation of maximum 1-hr or 24-hr averaged concentrations of chemicals allows for consideration of short-term "spikes" in concentration that may adversely impact health.

Based on the initial screening process for acute health effects described in **Section 8.4**, no maximum 1-hr or 24-hr averaged values exceeded any known acute CA REL or AEGL-1 values. Two chemicals required additional analysis based on comparison to ATSDR MRLs: benzene and methylene chloride.

The maximum daily concentration of benzene during the 2001-2002 monitoring period approached but did not exceed the acute MRL value. The CA REL value and the maximum 1-hr averaged concentration for benzene are also presented for comparative purposes (see **Table 50**).

The maximum daily concentration of methylene chloride exceeded the acute MRL value. Methylene chloride was not monitored at the N.E. Detroit site, therefore, there was no 1-hr averaged value. The CA REL value is presented for comparative purposes in **Table 50**.

Table 50: Comparison of Maximum Ambient Concentration and Health Benchmark Value

CHEMICAL	MAXIMUM VALUE ($\mu g/m^3$)	ACUTE MRL ($\mu g/m^3$) (1-14 DAY AVG)	ACUTE MRL BASIS	IS THERE AN ACUTE CA REL?	CA REL BASIS
Benzene	**159 (24-hr avg)**	**160**	Hematologic & immunotoxic effects	Yes, 1300 $\mu g/m^3$ (6-hr avg time)	Reproductive
Benzene (hourly data were only obtained at the N.E. Detroit site)	25 (1-hr avg)	160	Hematologic & immunotoxic effects	Yes, 1300 $\mu g/m^3$ (6-hr avg time)	Reproductive
Methylene Chloride	**11,580 (24-hr avg)**	**2,086**	Neurological	Yes, 14,000 $\mu g/m^3$ (1-hr avg time)	Central Nervous System

One major limitation of the acute hazard analysis is that there are no acute health benchmark values for the vast majority of the chemicals monitored in this study (only about 20% had health benchmarks). Therefore, the acute hazard posed by air toxics in Detroit is difficult to characterize. Another important limitation lies within the sampling strategy. Continuous data from the automated GC located at the N.E. Detroit site was used to determine the maximum 1-hr average concentration for VOCs measured under the PAMS program. Daily 24-hr sampling was done only for VOCs and carbonyls at the Dearborn site. For all other sites and chemicals monitored, the 24-hr maximum concentration was determined from data collected either every 6th or 12th day. Ideally, 24-hr continuous monitoring is desirable for detection of

short-term peaks in concentrations of air pollutants. However, cost and other technical considerations are prohibitive. Inferring the 24-hr maximum from data collected every 6th or 12th day obviously limits our ability to fully characterize acute hazard, but is the best approximation that can be made within the constraints of the study design. Two chemicals identified as chronic, non-cancer COPCs but lacking acute health benchmark values (naphthalene and manganese) were evaluated separately using a surrogate approach to assess the potential for acute hazard (see **Section 9.3.4**). **Appendix I** contains a complete table of all maximum 1-hr and 24-hr ambient concentrations monitored and known acute health benchmarks.

9.3.1 Benzene

Benzene is a known human carcinogen by all routes of exposure. It is also a skin and mucous membrane irritant. Short-term exposure to high concentrations of benzene produces central nervous system (CNS) effects which can range from mild to life-threatening depending on the concentration and duration of exposure. Symptoms of acute exposure include dizziness, weakness, nausea, headache, euphoria and respiratory distress. Extreme exposures may also cause blurred vision, tremors, paralysis and unconsciousness. Secondary effects to acute exposure in experimental animals have been observed in liver, kidney, spleen, bladder, thymus, brain, and the spinal cord (HSDB, 2004; Patty's 5th edition, 2001). In addition, individuals with certain blood disorders, anemias, and heart conditions may be more susceptible to the acute toxicity of benzene. Females may be more sensitive than males due to a higher body fat content and thus a longer retention time in the body (Cal EPA/OEHHA, 1999a).

The ATSDR acute inhalation MRL of 160 µg/m^3 for benzene was derived from a study in which 7 to 8 male C57BL/6J mice per group were exposed to 0, 10.2, 31, 100, or 301 ppm (0, 32.5, 98.9, 319, or 960 mg/m^3) of benzene in whole body inhalation chambers for 6 hr/day for six consecutive days. Control mice were exposed to filtered, conditioned air only. Endpoints evaluated were hematologic and immunotoxic effects. The LOAEL for this study was 10.2 ppm (32.5 mg/m^3) based on adverse immunologic effects (depressed peripheral lymphocytes and depressed femoral B-cell blastogenesis). This LOAEL was then duration adjusted to 2.5 ppm (8 mg/m^3). A total UF of 300 was applied: 10 for use of a LOAEL rather than a NOAEL, 3 for interspecies variability and 10 for intraspecies variability (ATSDR, 1997).

The Cal EPA's Acute CA REL for benzene of 1,300 µg/m^3 has a 6-hr averaging time because it is based on a reproductive study in pregnant female rats. Coate et al (1984) exposed 40 rats per group via inhalation to benzene concentrations of 0, 1, 10, 40 and 100 ppm (0, 3.2, 32, 128, and 319 mg/m^3) for 6 hr/day for five days. Mean fetal weights from females in the highest exposure group (100 ppm or 319 mg/m^3) were significantly decreased as compared to controls. This value was deemed to be the LOAEL for this study. The NOAEL was 40 ppm (128 mg/m^3) based on no observation of teratogenic, maternal, or fetal toxicity at this concentration. A total UF of 100 was applied: 10 for interspecies variability and 10 for intraspecies variability (Cal EPA, 1999).

The highest 24-hr concentration of benzene in this study was 159 µg/m^3 at S. Delray in February of 2002. This value is close to the Acute MRL of 160 µg/m^3. The next highest benzene level was 147 µg/m^3 in October of 2001, also at S. Delray. The third highest concentration of benzene was 127 µg/m^3 at S. Delray in June of 2001. The annual average at S. Delray is 21.6 µg/m^3. This information is summarized in **Table 51**. The highest 24-hr averaged concentration of benzene at a site other than S. Delray is 11.4 µg/m^3 at N. Delray. Maximum values and averages of benzene for all other sites is provided in **Table 52**. **Figure 19** is a graphical representation of benzene fluctuation at the S. Delray site during the study period. Note that the acute health benchmark value is 160 µg/m^3.

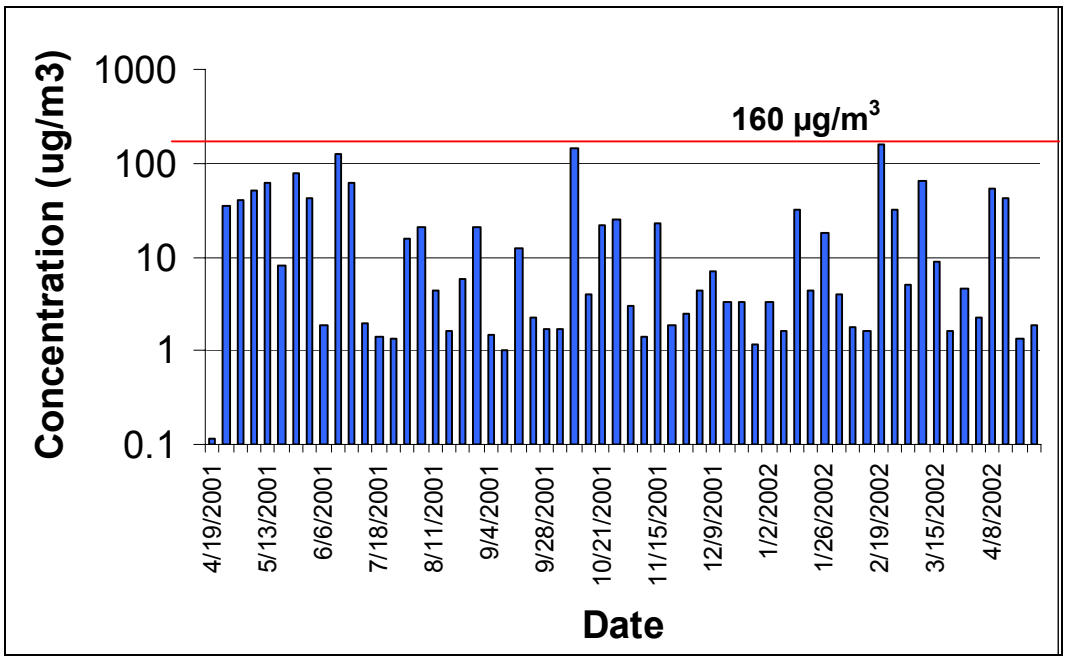
Table 51: Highest 24-Hr Averaged Benzene Concentration and Annual Average Concentration at S. Delray

SITE	# OF SAMPLES COLLECTED	DATE	BENZENE CONCENTRATION ($\mu g/m^3$)	ANNUAL AVERAGE CONCENTRATION 4/01-4/02
S. Delray	60	2/19/02	159	21.6
	60	10/10/01	147	21.6
	60	6/18/01	127	21.6

Table 52: Highest 24-Hr Averaged Benzene Concentration and Annual Average Concentration at All Other Sites

SITE	# OF SAMPLES COLLECTED	DATE	MAX BENZENE CONCENTRATION ($\mu g/m^3$)	ANNUAL AVERAGE CONCENTRATION 4/01-4/02
N. Delray	26	4/14/02	11.4	2.67
Dearborn	342	6/13/01	7.97	1.92
Southfield	59	7/30/01	6.57	1.98
River Rouge	60	5/19/01	6.56	2.14
Allen Park	33	8/5/01	4.38	1.50

Figure 19: Benzene Fluctuations at S. Delray*



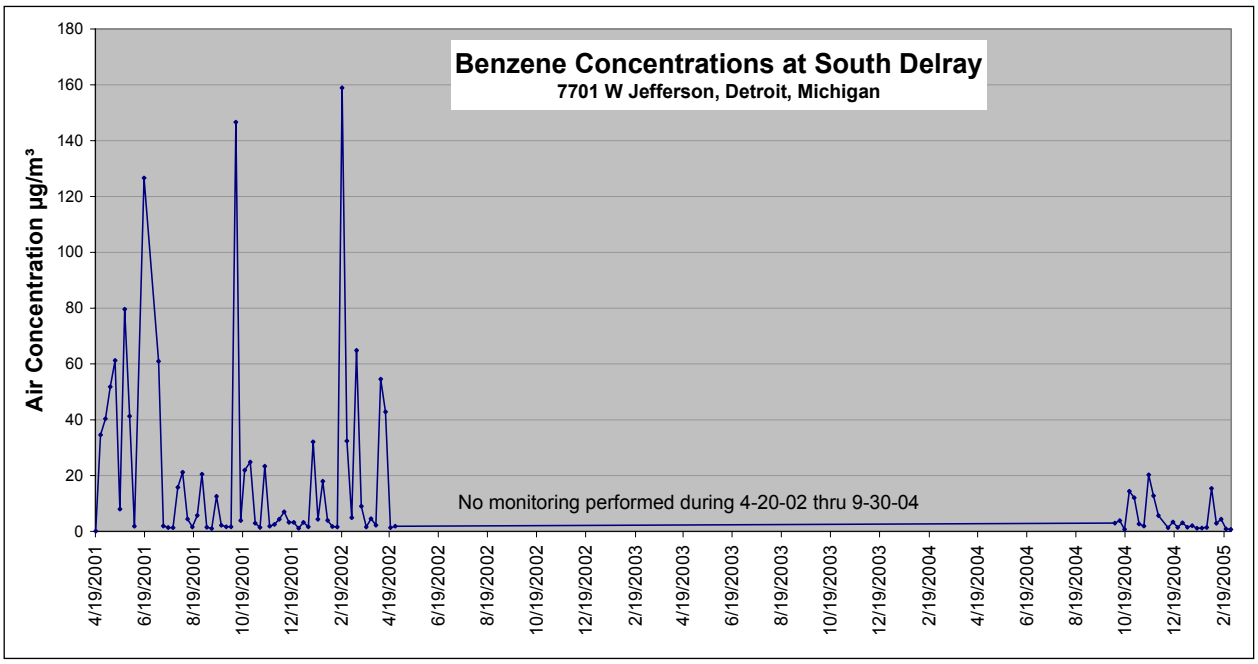
*Note: Data is presented on a log scale for readability.

The maximum 24-hr concentration of benzene at S. Delray is roughly equivalent to the most appropriate acute health benchmark value (MRL = 160 $\mu g/m^3$). Ambient exposures to concentrations below the MRL are generally considered safe while concentrations above the MRL must be evaluated on a case-by-case basis. The level of concern will be affected by a number of factors including the number, frequency and magnitude of the exposure concentration that exceeds the acute health benchmark value. Considering the highest 24-hr concentration of benzene approached but did not exceed the acute health benchmark value, the levels of benzene at S. Delray do not represent an acute inhalation hazard since the acute MRL includes a considerable margin of safety (UF = 300). However, the 24-hr average concentration

of benzene at S. Delray was determined from data collected every 6th day and, therefore, higher spikes of benzene may have been missed which could lead to an underestimation of the acute inhalation hazard.

Another point to consider is that although the acute MRL is the most appropriate benchmark in terms of averaging time, the CA REL value is based on a more sensitive endpoint – namely, reproductive toxicity. If one were to consider the CA REL as the acute health benchmark value instead of the acute MRL, an adjustment would need to be made for duration. Adjusting the 6-hr averaged CA REL to a 24-hr averaged value would yield a 24-hr averaged CA REL of 325 µg/m^3. Utilizing that value as a benchmark, benzene levels at S. Delray fall well below the level of concern for acute health effects. Given the absence of continuous data at S. Delray, concentrations of benzene exceeding the acute benchmark cannot be discounted. Monitoring for benzene at S. Delray began again in October of 2004 on a once every six day collection cycle. To date, monitored levels are quite low (see **Figure 20**). A full season's worth of data will be needed to determine whether benzene levels are currently below a level of concern for acute health effects.

Figure 20: S. Delray Benzene Monitoring Data



9.3.2 Methylene Chloride

Methylene chloride is rapidly absorbed in the lungs and quickly enters the systemic circulation, crossing the blood-brain barrier and the placenta. The acute toxicity of methylene chloride in experimental animals is relatively low although CNS and liver effects are typically observed at high concentrations. It is also moderately irritating to the skin and mucous membranes. The main effects in humans are CNS depression and carboxyhemoglobin formation, both of which are reversible. Impairments to neurological, hepatic, and renal function have also been noted in humans. Methylene chloride is considered a probable human carcinogen and there is one uncorroborated study suggestive of teratogenicity (HSDB, 2004; Patty's 5th Edition). Persons with heart disease, smokers, pregnant women and their fetuses may be more susceptible to the effects of methylene chloride exposure due to greater affinity (e.g., fetuses) or lower tolerance (e.g., smokers with elevated carboxyhemoglobin levels) for carbon monoxide (major metabolic by-product of methylene chloride metabolism).

The ATSDR acute inhalation MRL of 2,086 µg/m^3 for methylene chloride is based on human exposure data and neurological effects. Winneke et al. (1974) exposed 6 to 20 volunteers to 0, 300, 500, or 800 ppm (0, 1043, 1738, or 2781 mg/m^3) methylene chloride vapors for three to four hours in a randomized blind clinical chamber experiment. Control subjects (0 ppm) were exposed to filtered air only. Subjects were tested for neurological effects using standard neurobehavioral tests (visual, auditory, and psychomotor tasks) at 45 minute intervals during the three to four hour exposure period. A statistically significant decrease in critical flicker fusion frequency (a measure of visual performance) was noted at all concentrations as compared to controls. However, a dose-response relationship was noted only at the highest concentration tested (800 ppm or 2781 mg/m^3). Decreases in auditory vigilance were noted at 500 ppm (1738 mg/m^3) and impairment on psychomotor performance tasks was evident at 800 ppm (2781 mg/m^3). The statistical significance of these results is not given. Based on the most sensitive endpoint in this study (visual performance), 300 ppm (1043 mg/m^3) was chosen as the LOAEL for methylene chloride. The LOAEL was then duration adjusted to 60 ppm (209 mg/m^3). A total UF of 100 was used to derive the MRL: 10 for LOAEL to NOAEL extrapolation and 10 for intraspecies variability (ATSDR, 2000).

Cal EPA's Acute CA REL of 14,000 µg/m^3 for methylene chloride is based on CNS effects in human volunteers. This CA REL is a value believed to be protective against mild adverse effects. Twelve healthy adult subjects were exposed via inhalation to 195 ppm (678 mg/m^3) of methylene chloride for 90 minutes (Putz et al., 1976). Subjects exhibited impaired performance on standard neurobehavioral tests, such as dual-task and auditory vigilance performance, after 90 minutes of exposure. Performance continued to decrease with subsequent exposure (duration not given). Blood carboxyhemoglobin levels rose from 1.35% (pre-exposure baseline) to 5.1% (post-exposure baseline). No subjective symptoms such as headache, nausea, or irritation were reported. Therefore, 195 ppm (678 mg/m^3) was deemed a LOAEL for this study and was duration adjusted to 240 ppm (834 mg/m^3). A total UF of 60 was applied: 6 for LOAEL to NOAEL extrapolation and 10 for intraspecies variability.

The highest 24-hr concentration of methylene chloride in this study was 11,580 µg/m^3 at Allen Park in July of 2001. The second and third highest 24-hr averaged concentrations at this site also exceeded the MRL: 4475 µg/m^3 in June of 2001 and 4204 µg/m^3 in March of 2002. The fourth highest concentration at Allen Park is below the MRL at 1446 µg/m^3. The annual average concentration of methylene chloride at Allen Park for the study period is 803 µg/m^3. This information is summarized in **Table 53**. The highest 24-hr concentration of methylene chloride at a site other than Allen Park is 1,152 µg/m^3 at the Southfield monitor. Maximum values and annual averages for all other sites is provided in **Table 54**. **Figure 21** is a graphical representation of the methylene chloride fluctuation at Allen Park for the study duration. Note that the acute health benchmark value is 2,086 µg/m^3.

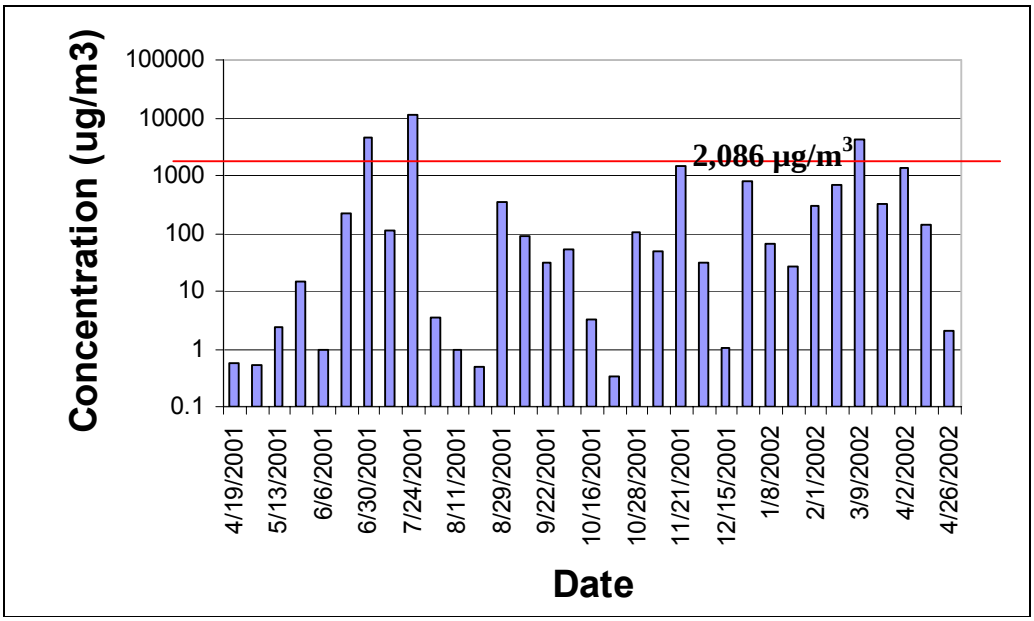
Table 53: Highest 24-Hr Averaged Methylene Chloride Concentration and Annual Average Concentration at Allen Park

SITE	# OF SAMPLES COLLECTED	DATE	METHYLENE CHLORIDE CONCENTRATION (µg/m^3)	ANNUAL AVERAGE CONCENTRATION 4/01-4/02
Allen Park	33	7/24/01	11580	803
	33	6/30/01	4475	803
	33	3/9/02	4204	803
	33	11/21/01	1446	803

Table 54: Highest 24-Hr Averaged Methylene Chloride Concentration and Annual Average Concentration at All Other Sites

SITE	# OF SAMPLES COLLECTED	DATE	MAX METHYLENE CHLORIDE CONCENTRATION (µg/m^3)	YEARLY AVERAGE CONCENTRATION 4/01-4/02
Southfield	59	8/23/01	1152	62.9
Dearborn	342	3/3/02	513	11.6
River Rouge	60	1/20/02	432	10.1
N. Delray	26	9/22/01	96	6.66
S. Delray	60	11/9/01	58.6	2.53

Figure 21: Methylene Chloride Fluctuations at Allen Park



Note: Data is presented on a log scale for readability.

For methylene chloride, the acute MRL is the most appropriate health benchmark for comparison to a monitored concentration with a 24-hr averaging time. The data indicate daily spikes in methylene chloride at Allen Park that exceed the appropriate health benchmark which is based on impairment of neurobehavioral function. The MRL represents a level below which is believed to be protective of adverse health effects to the general population. Concentrations above the MRL do not necessarily imply that adverse effects *will* occur, but that the potential for adverse effects increases with increasing exposure. Due to the use of UFs in developing the MRL, the highest concentration of methylene chloride measured in this study is 90-fold lower than the *actual* lowest concentration observed to produce adverse effects in healthy humans (LOAEL=1043 mg/m^3 or 1,043,000 µg/m^3). The highest concentration of methylene chloride at Allen Park is also 18-fold lower than the duration adjusted LOAEL of 209 mg/m^3 from the same study (209,000 µg/m^3). However, these studies used a very limited number of healthy human subjects that may not adequately account for the potential vulnerability and sensitivity of certain subgroups. This is why UFs are utilized in developing MRLs. In addition, VOCs at Allen Park were monitored on a once every 12 day schedule and therefore, higher spikes of methylene chloride may have been missed leading to an underestimation of the acute risk.

The spikes in methylene chloride at Allen Park are indeed unusual and warrant further investigation. Continued monitoring of methylene chloride at Allen Park beyond the pilot project revealed a maximum 24-hr average of 15 µg/m^3 (4.36 parts per billion-Volume [ppbV]) between May and September of 2002 and a maximum 24-hr average of 0.66 µg/m^3 (0.19 ppbV) between August and November 2003. These levels are approximately 100 to 3,000-fold below the MRL of 2,086 µg/m^3. Investigation of potential industrial sources of methylene chloride at Allen Park is ongoing and continued monitoring is planned to gather a full year's worth of data. Based on current available data, it seems that the unusual spikes of methylene chloride that occurred during the 2001-2002 monitoring period have not been repeated. At this time, increased risk of acute health effects from inhalation of methylene chloride at Allen Park is unlikely. Analysis of additional monitoring data will be required to more fully characterize any potential risk. Monitoring data for VOCs at Allen Park through the Detroit Exposure Aerosol Research Study (DEARS) (7/12/04 to 8/29/04) will also be a valuable source of information to aid in the risk characterization of methylene chloride at Allen Park. DEARS is a three-year study of indoor, outdoor, and personal monitoring for PM and air toxics by the USEPA's Office of Research and Development (ORD).

9.3.3 Cumulative Acute Hazard

When considering the maximum 1-hr concentrations for each chemical by month, benzene and mixed xylenes were the COPCs identified (HQ $\geq$ 0.1). However, there were no HI values > 1 for any one month and, therefore, no further analysis was warranted. One-hour values were considered by month since there was only one site for this data.

When considering the maximum 24-hr concentrations for each chemical by site, four COPCs were identified (HQ $\geq$ 0.1): arsenic, benzene, formaldehyde and methylene chloride. When HQs were added, only two sites had HI values > 1. Those sites are noted in **Table 55** along with the COPCs and their associated values.

Table 55: Acute COPCs with HQ > 0.1 at Allen Park and S. Delray

SITE	CHEMICAL	HIGHEST 24 HR AVERAGED CONCENTRATION (µg/m^3)	ACUTE BENCHMARK (µg/m^3)	HQ
Allen Park	Arsenic	0.015	0.19 CA REL (4-hr avg)	0.1
	Formaldehyde	4.84	49 MRL	0.1
	Methylene Chloride	11,580	2,086 MRL	5.6
S. Delray	Benzene	159	160 MRL	1.0
	Formaldehyde	6.13	49 MRL	0.1

Based on the available data, the target organ for the critical acute effects on which the health benchmark value is based is immunological for benzene, respiratory for formaldehyde, neurological for methylene chloride, and reproductive/developmental for arsenic. Because each of these chemicals affects a different target organ, adding the HQs together to determine a cumulative HI is not appropriate. Therefore, cumulative impact from the combined exposure of COPCs does not pose a concern for increased hazard of acute health effects. This, however, does not take into consideration the potential for combined impacts between monitored air toxics and criteria pollutants.

9.3.4 Pollutants Given Special Consideration For Acute Risk – Naphthalene And Manganese

As mentioned in **Section 8.3**, the vast majority of pollutants monitored did not have a preferred acute health benchmark (CA REL, ATSDR MRL, or AEGL-1 value). Therefore the potential for

hazard of acute health effects is unknown for many chemicals for a variety of reasons including: a lack of general toxicity information, absence of a non-emergency screening level, no acute health effects identified, etc. Two pollutants without preferred acute health benchmark values (manganese and naphthalene) will be given special consideration outside of the standard methodology described in **Section 8.4**. These pollutants were identified as COPC with an HQ > 1 in the chronic, non-cancer hazard characterization section of this report (see **Section 9.2**). The HQ approach to hazard assessment is a trigger for a more in-depth evaluation of those chemicals that surpass the HQ=1 screening level. Because naphthalene and manganese surpassed this chronic hazard screening level, they were evaluated for potential acute effects due to spikes of these pollutants at specific sites.

Lacking acute health benchmark values for manganese and naphthalene, other health benchmark values were considered for the acute assessment. OELs (ACGIH TLVs and National Institute of Occupational Safety and Health [NIOSH] RELs) have been established for manganese and naphthalene, and were used for this purpose. To account for the uncertainty of using a value designed to protect healthy adult workers in an occupational setting, as a benchmark value for the general population, 1% of the TLV or REL was used as a surrogate acute heath benchmark value. There are limitations in using occupational screening levels to assess ambient exposures to communities and the ACGIH (2004) cautions against such use:

> "The values presented in this book are intended for use only as guidelines or recommendations to assist in the evaluation and control of potential workplace health hazards and for no other use (e.g., neither for evaluating or controlling community air pollution; nor for estimating the toxics potential of continuous, uninterrupted exposures or other extended work periods; nor for proving or disproving an existing disease or physical condition in an individual). Further, these values are not fine lines between the safe and dangerous conditions and should not be used by anyone who is not trained in the discipline of industrial hygiene."

ACGIH's documentation of the TLV basis for manganese and naphthalene was reviewed to evaluate the strength of the database supporting these values. Considering the available data, it was felt that use of 1% of the TLV would likely be a conservative, health protective estimate of a safe concentration for a 24-hr acute exposure. Given the absence of appropriate acute benchmarks for these chemicals, the use of this surrogate measure was considered a reasonable approach to use as a screening methodology for evaluating the potential for acute health effects.

The maximum 24-hr concentration for naphthalene was 93.3 $\mu g/m^3$ at S. Delray on 6/18/2001. The TLV for naphthalene is 10 ppm (52,000 $\mu g/m^3$) (ACGIH, 2004). The basis for the TLV is irritation, ocular, and blood effects. There is also an short-term exposure limit of 15 ppm (79,000 $\mu g/m^3$). The short-term exposure limit is a 15 minute TLV not to be exceeded in any 8-hr workday. It is intended to supplement rather than supplant the TLV.

The NIOSH REL for naphthalene is also set at 10 ppm (NIOSH, 2003) and lists target organs as eyes, skin, blood, liver, kidneys, and CNS. The surrogate acute health hazard benchmark using 1% of the TLV gives a screening level of 520 $\mu g/m^3$, with an 8-hr averaging time. The maximum 24-hr average of 93 $\mu g/m^3$ is approximately 5 fold less than the 8-hr averaged surrogate benchmark. Based on this analysis, there is no indication of increased hazard of acute health effects (irritation) from naphthalene at S. Delray or at any other site.

Similarly, a surrogate acute health benchmark value for manganese can be developed utilizing 1% of the TLV. The maximum 24-hr concentration measured for manganese at any site was 1.9 $\mu g/m^3$ at Delray on May 1, 2001. This concentration is approximately at 1% of the ACGIH

TLV of 200 µg/m^3 (8-hr average) for manganese and inorganic compounds as manganese. The basis for the TLV is CNS, lung and reproductive effects.[20]

The NIOSH REL for manganese compounds and fume as manganese is 100 µg/m^3 (up to 10-hr average). Target organs are listed as the respiratory system, CNS, blood and kidneys. The maximum 24-hr averaged concentration at Delray is approximately two times greater than 1% of the REL. There is also an short-term exposure limit of 3,000 µg/m^3 and an OSHA ceiling limit of 5,000 µg/m^3, neither of which would be exceeded using the surrogate approach. There were two other days at Delray with concentrations at 1% of the REL but not exceeding 1% of the TLV.

The second highest 24-hr concentration for manganese was 1.19 µg/m^3 measured at Dearborn site on 12/27/01. This concentration does not exceed 1% of the ACGIH TLV but is approximately 1% of the NIOSH REL. There were no other days at Dearborn on which either surrogate benchmark was met or exceeded. The highest 24-hr concentration of manganese at any other site was 0.27 µg/m^3 at Rouge on 2/19/02. This concentration is five times lower than the lowest surrogate acute benchmark.

This assessment of the maximum measured manganese levels finds that they were close to 1% of the OEL-TWA levels. This does not suggest that acute health effects may be anticipated, but it also does not clearly indicate an ample margin of safety. It must be reiterated that manganese was monitored as TSP and not specifically as respirable particles. Recent average manganese trend data at Dearborn (2003-2004) suggest that at least half of average TSP manganese at this site is in the PM_{10} or smaller range. Due to the differences in the recommended occupational limits set by ACGIH and NIOSH, development of a preferred acute health benchmark, if adequate data are available, should be pursued to help resolve this issue.

9.4 Evaluation of Chemicals without Health Benchmark Values or With Low Detection Frequency

Sections 9.1 and **9.2** have provided a characterization of the chronic health risks (cancer and non-cancer) for those chemicals that had a health benchmark value, and for which at least 15% of the monitored levels were above the detection limit. About 36% (66 out of 186) of the chemicals that were monitored met these criteria. The inability to assess the potential health risks of the remaining 64% of the chemicals adds to the uncertainty of any conclusions. To address this additional uncertainty, and to prioritize the development of health benchmark values, the data for these chemicals were further evaluated according to the following two steps:

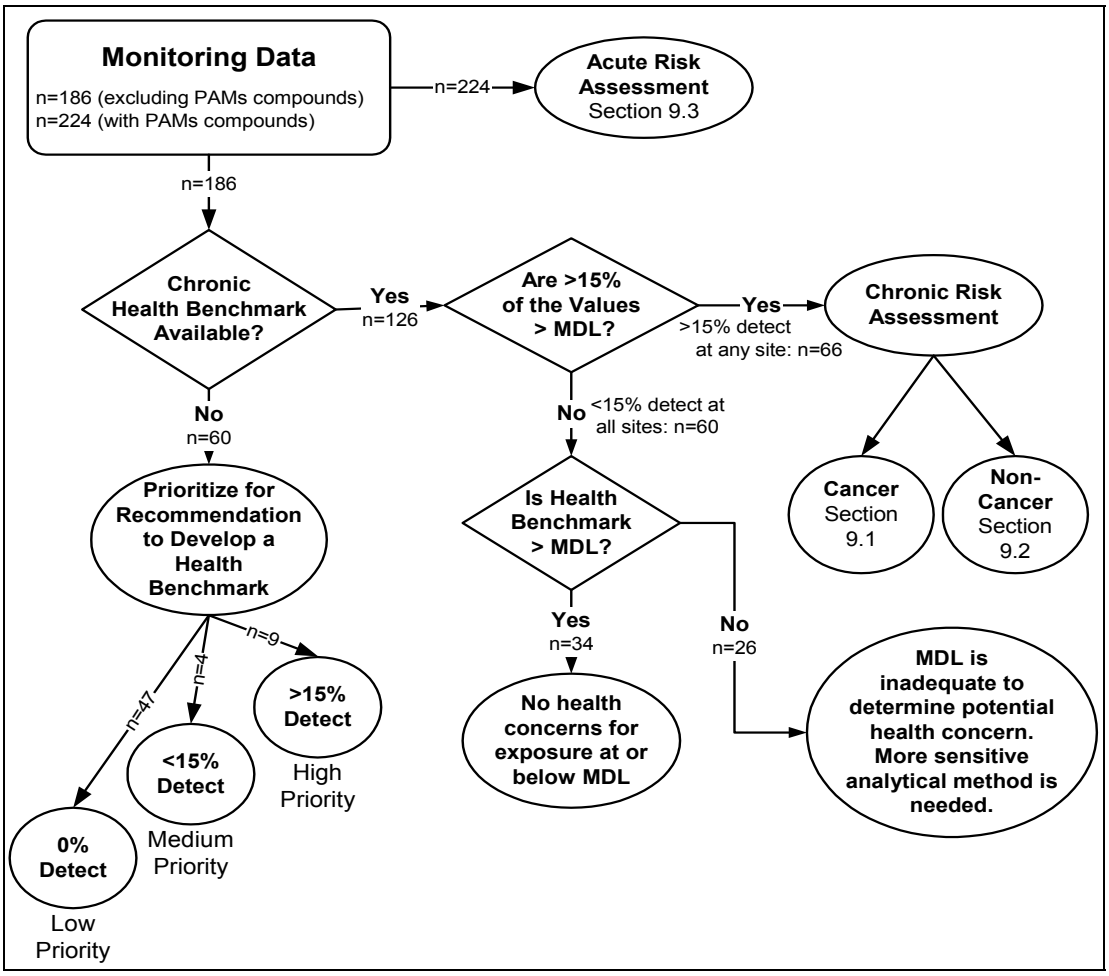
1. Comparison of the MDL to the health benchmark value for those chemicals that had a health benchmark value. For carcinogenic chemicals the health benchmark value used represents the concentration associated with an increased cancer risk of 1×10^{-6}.

2. If no health benchmark value was available, prioritization of these chemicals for development of a health benchmark value based on the frequency of detection.

The following **Figure 22** provides a schematic diagram of the above evaluation, and a breakdown of the number of chemicals in each category.

[20] Note: The TLV for manganese was withdrawn from the notice of intended changes in 2004 – the adopted TLV of 200 µg/m^3 was retained.

Figure 22: Schematic Diagram for the Evaluation of a Chemical Without a Health Benchmark Value and Low Detection Frequency



Section 9.3 provided a characterization of acute health hazards. As previously mentioned in that section, only about 20% of the chemicals had acute health benchmark values. The lack of an acute health bench mark value for the remaining 80% of the chemicals adds uncertainty to any conclusions in this area. For the evaluation of acute health impacts, a single value above the detection limit was considered adequate to use in this assessment. Therefore, low detection frequency with regards to acute health impacts is not evaluated further here.

Chemicals with Chronic Health Benchmark Values and Low Detection Frequency

The results of the above analysis indicate that 60 chemicals (32%) had chronic health benchmark values, but were detected < 15% of the time at all sites. Because of the high number of ND samples for such chemicals, an average concentration cannot be reliably determined for use in the risk assessment. However, the finding that the health benchmark value is greater than the MDL indicates that these chemicals are not likely to present a health concern. Of the 60 chemicals with health benchmark values and low detection frequency, 34 had health benchmark values that were greater than the MDL. Therefore, these chemicals are not expected to present a health concern, even though the standard hazard assessment could not be done due to the low detection frequency. **Table 56** lists these chemicals.

Table 56: Chemicals Detected at a Low Frequency and Not Expected to Present a Health Concern

CHEMICALS WITH HEALTH BENCHMARK VALUE GREATER THAN THE MDL		
1,1-Dichloroethane	Benzyl alcohol	Halocarbon 114
1,2,4-Trichlorobenzene	Bromochloromethane	Hexachlorocyclopentadiene
1,2-Dichlorobenzene	Bromoform	Hexachloroethane
1,2-Dichloropropane	Bromomethane	Isophorone
2,4,5-Trichlorophenol	Butyl benzyl phthalate	Methyl isobutyl ketone
2,4,6-Trichlorophenol	Chlorobenzene	Methyl methacrylate
2,4-Dichlorophenol	cis-1,2-Dichloroethene	o-Toluidine
2,4-Dimethylphenol	Dichlorotetrafluoroethane	Pentachloronitrobenzene
2-Picoline	Dimethyl phthalate	tert-Amyl methyl ether
Acetophenone	Dinoseb	trans-1,2-Dichloroethene
Aniline	Diphenylamine	
Benzo(ghi)perylene	Ethyl tert-butyl ether	

There were 26 chemicals with health benchmark values lower than the MDL. These chemicals are listed in **Table 57**. All of these chemicals are considered carcinogenic compounds. If a cancer risk level higher than 1 X 10^{-6} was selected for the health benchmark value, the number of compounds would likely be different. For these chemicals, no conclusions can be made regarding the potential health concern for ambient levels. A more sensitive analytical method is needed to further characterize potential risks.

Table 57: Chemicals Detected at a Low Frequency and Needing a More Sensitive Analytical Method to Evaluate.

CHEMICALS WITH HEALTH BENCHMARK VALUE LESS THAN THE MDL*		
1,1,2,2-Tetrachloroethane	3,3-Dichlorobenzidine	Ethyl acrylate
1,1,2-Trichloroethane	Azobenzene	Hexachloro-1,3-butadiene
1,1-Dichloroethane	Benzidine	Hexachlorobenzene
1,2-Dibromoethane	Benzo(a)pyrene	Indeno(1,2,3-cd)pyrene
1,2-Dichloroethane	bis (2-Chloroethyl)ether	Nitrobenzene
2,4-Dinitrotoluene	Bromodichloromethane	N-Nitrosodipropylamine
2,6-Dinitrotoluene	Chloromethyl benzene	Pentachlorophenol
2-Chloro-1,3-butadiene	Dibenzo(ah)anthracene	Vinyl chloride
2-Naphthylamine	Dibromochloromethane	

*All chemicals listed are carcinogenic compounds. The health benchmark value used represents the concentration associated with an increased cancer risk of 1 X 10^{-6}.

Chemicals with No Health Benchmark Values

There were 60 chemicals (32%) that had no chronic health benchmark value. Three categories were identified for prioritizing these chemicals for development of a health benchmark value based on the frequency of detection. These categories and the number of chemicals in each are as follows:

- Low Priority (47 chemicals) – No sites had any monitored levels above the MDL.
- Medium Priority (4 chemicals) - Less than 15% of the monitored values were above the MDL at all sites.
- High Priority (9 chemicals) - Greater than 15% of the monitored values were above the MDL at least one site.

Table 58 lists these chemicals and the priority for developing a health benchmark value.

Table 58: Prioritized List of Chemicals for Developing a Health Benchmark Value

HIGH PRIORITY		
2,2,4-Trimethylpentane	Acetylene	n-Octane
2,5-Dimethylbenzaldehyde	Butyr/Isobutyraldehyde	m-Tolualdehyde
3&4-Methylphenol	Carbazole	p-Tolualdehyde
MEDIUM PRIORITY		
1,3-Dichlorobenzene	o-Tolualdehyde	trans-1,3-Dichlorpropene
5-Nitro-o-toluidine		
LOW PRIORITY		
1,2,4,5-Tetrachlorobenzene	4-Aminobiphenyl	Hexachloropropene
1,3-Dinitrobenzene	4-Bromophenyl phenyl ether	Isodrin
1,4-Naphthoquinone	4-Chloro-3-methylphenol	Isosafrole
1-Naphtylamine	4-Chloroaniline	Methyl methanesulfonate
2,3,4,6-Tetrachlorphenol	4-Chlorophenyl-phenyl ether	N-Nitrosodibutylamine
2,4-Dinitrophenol	4-Dimethylaminoazobenzene	N-Nitrosodiethylamine
2,6-Dichlorophenol	4-Nitroaniline	N-Nitrosodimethylamine
2-Acetylaminofluorene	4-Nitrophenol	N-Nitrosomethylethylamine
2-Chloronaphthalene	7,12-Dimethylbenz(a)anthracene	N-Nitrosopiperidine
2-Chlorophenol	bis(2-Chloroethoxy)methane	N-Nitrosopyrrolidine
2-Nitroaniline	bis(2-Chloroisopropyl)ether	Pentachlorobenzene
2-Nitrophenol	Chlorobenzilate	Pentachloroethane
3,3'-Dimethylbenzidine	cis-1,3-Dichloropropene	Phenacetin
3-Methylcholanthrene	Diallate	Pronamide
3-Nitroaniline	Di-n-octyl phthalate	Safrole
4,6-Dinitro-2-methylphenol	Ethyl methanesulfonate	

10.0 OTHER POLLUTANTS

Information regarding mercury and diesel exhaust is presented in this section. Monitoring data for these pollutants were limited in some way that precluded the standard risk assessment and risk characterization done for the other air toxics and presented in **Sections 7 to 9**. Additionally, the PBT properties of mercury, requires consideration of exposure pathways beyond direct inhalation to adequately characterize the risk from the release of these compounds into the atmosphere. Considering the significant interest and concern in Michigan for these compounds, an evaluation of potential health risks is provided, within the constraints of the available data, in order to provide a better understanding of the relative magnitude of concern. Mercury is discussed in **Section 10.1** and diesel exhaust in **Section 10.2**.

10.1 Mercury Monitoring at Southfield and S. Delray

In addition to the 224 chemicals monitored during the Detroit Pilot Project, mercury levels were monitored at two sites during 2001-2002. Mercury was added at the request of the stakeholders group as a pollutant of special concern. Mercury belongs to a subset of pollutants known as PBTs. PBTs may not be present in high enough concentration in ambient air to pose a risk from direct inhalation, but may pose a health risk through indirect exposure subsequent to deposition.

Ambient air concentration of mercury is comprised primarily (over 95%) of elemental mercury or Hg^0. Elemental mercury is dominant due to its volatility and low water solubility and can remain in the atmosphere for up to one year before depositing in water and soil. The remaining few percent of ambient mercury is comprised of RGM (reactive gaseous mercury, sometimes referred to as “oxidized” mercury), and mercury bound to particulate matter or Hg(p) . Once RGM and Hg(p) are emitted into the atmosphere, they deposit much more quickly due to their high water solubility and reactivity. Due to their differential fate in the atmosphere, Hg^0 is typically the predominant form of mercury detected in ambient air (nanograms per cubic meter [ng/m^3] range) while RGM and Hg(p) (picograms per cubic meter [pg/m^3] range) do not add significantly to total gaseous mercury concentrations in ambient air.

The form of mercury that has the greatest potential to impact public health is methylmercury. Methylmercury is formed in the environment through the methylation of inorganic forms of mercury by microorganisms. This is the form of mercury that bioaccumulate in fish. The mercury cycle is enormously complex but despite intense scrutiny and research, there is still no reliable way of determining what percentage of deposited inorganic mercury will end up as methylmercury in the food chain. Air monitoring of Hg^0, then, provides one piece of information to aid in our understanding of the complex process of mercury cycling, deposition, and entry into the food chain as methylmercury. To adequately assess total deposition of mercury, all forms need to be measured including mercury in precipitation. The MDEQ in partnership with the University of Michigan Air Quality Laboratory is conducting a study, funded by the Michigan Great Lakes Protection Fund and USEPA, to determine mercury speciation and concentrations in precipitation to better assess deposition levels within the state.

For the Detroit Pilot Project Hg^0 was monitored to assess whether any spatial variation exists that would indicate possible source impacts. The Tekran 2537A mercury vapor analyzer provides a continuous measure of Hg^0 in air. It detects Hg^0 present at ng/m^3 (parts per trillion [ppt]) concentrations. The Southfield site was chosen due to its proximity to mobile sources (it is completely surrounded by highways) and the S. Delray site was chosen for its proximity to potential point sources of mercury.

Monitoring at the Southfield site proceeded from 12/14/01 to 2/27/02 (winter session) and 6/19/02 to 7/25/02 (summer session). Monitoring at S. Delray proceeded from 12/22/01 to 2/27/02 (winter session) and 6/20/02 to 7/25/02 (summer session). The Tekran instruments were run under identical conditions with weekly calibrations and other quality assurance checks performed at each site. There was significant loss of data during the winter session (percentage of Completeness: Southfield = 62%, S. Delray = 30%). Data capture was much better during the summer session (percentage of Completeness: Southfield = 84%, S. Delray = 70%) (Robinson, 2004).

In general, the daily averages at S. Delray were higher than at the Southfield site (**Figures 23** and **24**).[21] Using the average of the daily averages to come up with an overall site average resulted in an estimate of 1.9 ng/m^3 for Southfield and 2.4 ng/m^3 for S. Delray (see **Figure 25** and footnote 21). These levels are consistent with reported global background concentration of Hg^0 which ranges from 1.6 to 2.0 ng/m^3 (Malcolm et al., 2003). Annual averages for Detroit observed during the University of Michigan Air Quality Laboratory study are around 3.0 ng/m^3 (Taylor Morgan, 2004).

Figure 23: 24-Hr Averages of Mercury Levels Using 75% Complete Data

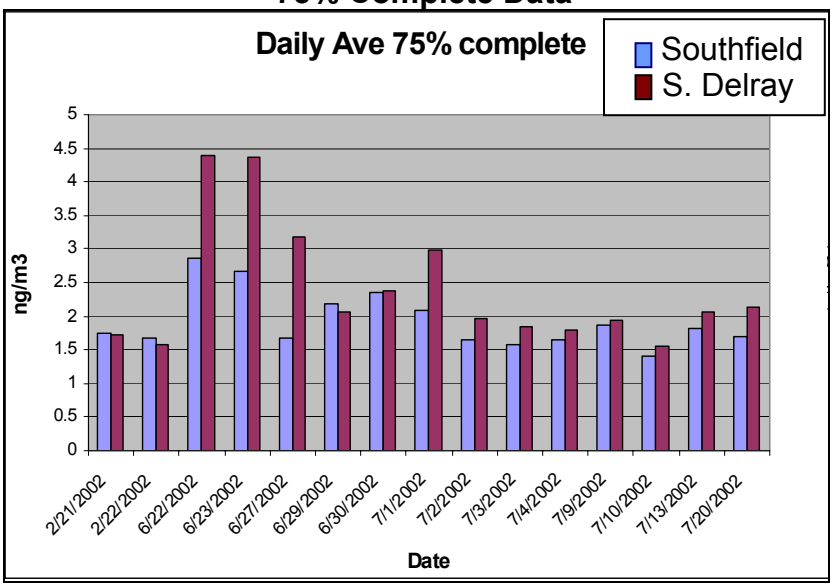
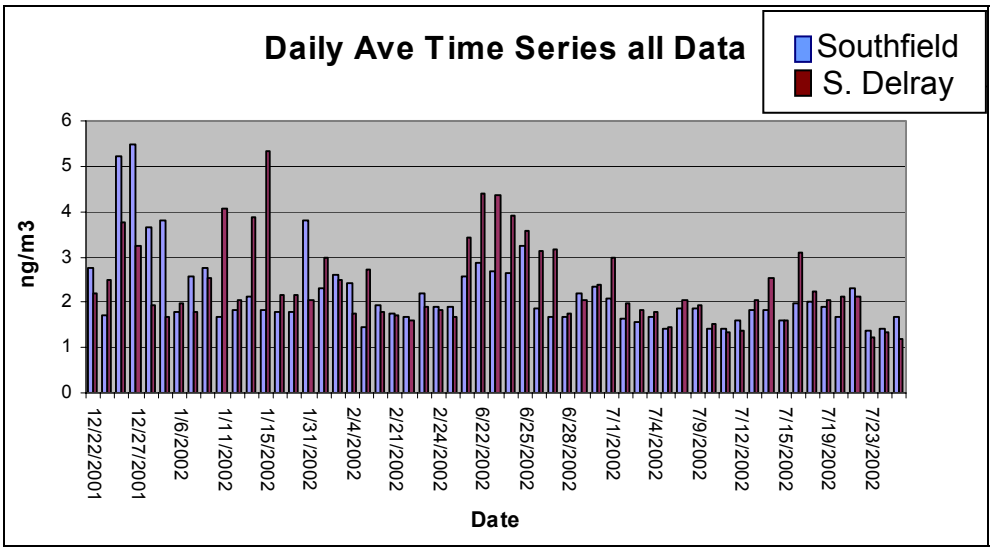


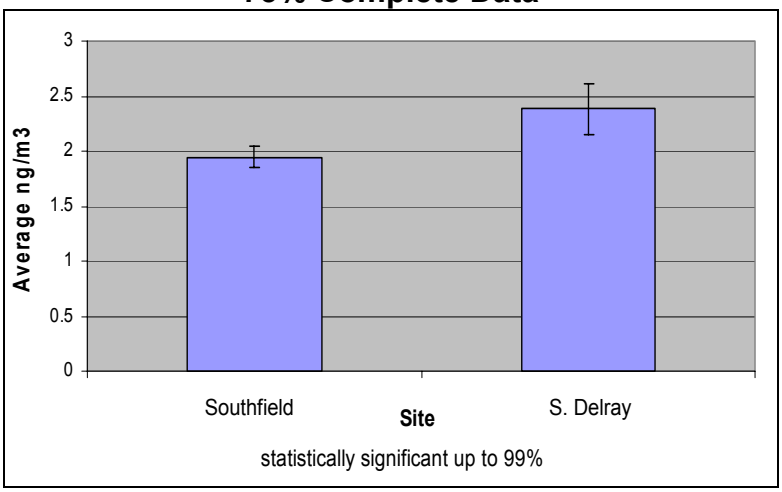
Figure 24: 24-Hr Averages of Mercury Levels Using All Data



The 24-hr averages at both sites exhibited comparable trends except for a few spikes at S. Delray in January and June of 2002 and a few spikes at Southfield in December of 2001. The spikes at S. Delray may be attributable to the influence of local sources but are well below the level of concern for health effects from direct inhalation exposure to

[21] Daily 24 hour average graphs are provided from the 75% completeness dataset (A) and total dataset (B). The 75% completeness dataset omits data that was not continuously collected for at least 45 minutes during a 1-hr sampling period (i.e. the winter session). The overall site average was calculated from the 75% completeness dataset.

Figure 25: Mercury Overall Site Averages Using 75% Complete Data



Hg^0. The spikes at Southfield seem to coincide with those timeframes during which the Tekran experienced instrumentation problems between 12/22/01 and 12/29/01 and therefore may not be valid.

Acute health benchmarks for inhalation of Hg^0 include emergency response guidelines such as ERPGs and TEELs, but only the CA REL was deemed an appropriate reference value for purposes of this report (see **Section 9.3** for discussion on relevance of acute health benchmarks). The acute CA REL for inhalation exposure to Hg^0 is 1.8 µg/m^3 (1,800 ng/m^3) with a 1-hr averaging time (Cal EPA/OEHHA, 1999b). The basis for this guideline is severe reproductive and developmental effects. The highest rolling 1-hr average for the study period is 71 ng/m^3 recorded at S. Delray on 6/20/02. This average is approximately 25-fold below the acute CA REL with the same averaging time. The highest rolling 1-hr average for the study period at Southfield is 5 ng/m^3 on 2/23/02. This average is approximately 360-fold below the acute CA REL. Therefore, Hg^0 levels at S. Delray and Southfield do not pose an acute health hazard from direct inhalation exposure.

The most appropriate health benchmark value for chronic inhalation of Hg^0 is USEPA's RfC at 0.3 µg/m^3 (300 ng/m^3). The RfC represents a daily concentration to which an individual could be exposed over a lifetime without appreciable deleterious effects. The RfC is based on several human occupational studies in which the critical effects were neurological such as hand tremor and memory disturbance. The highest 24-hr average recorded during the monitoring period was 40 ng/m^3 at S. Delray on 2/26/02. This average is 7.5-fold less than the RfC value which includes an UF of 30. The highest 24-hr average at Southfield was 3.9 ng/m^3 on 6/24/02. This average is approximately 75-fold less than the RfC. The longer term average concentrations of 2.4 ng/m^3 at S. Delray and 1.9 ng/m^3 at Southfield are about 125 to 160 times lower than the RfC. Therefore, levels of Hg^0 at S. Delray and Southfield do not pose a chronic health hazard from direct inhalation exposure.

10.2 Diesel

One of the key questions listed in **Section 2.5** addresses the potential risk of exposure to diesel emissions in the following:

> *#7. Do the monitoring data and risk assessment help to scope the potential relative health significance of diesel emissions in the Detroit area?*

In this section, the relevant monitoring data from the Detroit area are presented along with a description of current possible methods and tools by which diesel particulate matter or DPM concentrations may be quantified. In using these methods, the significance and validity/relevance of the estimated diesel particulate concentrations are discussed. It is important to note that the purpose of this section is not necessarily to derive high-confidence risk estimates, but to *"scope the potential relative health significance of diesel emissions in the Detroit area"* (emphasis added).

Introduction

Diesel exhaust is a complex mixture of hundreds of gases and fine particles that is formed when an engine burns diesel fuel. It contains many substances that are considered known or potential human carcinogens such as benzene, arsenic, nickel, formaldehyde, and PAHs. Exposure to diesel exhaust may cause irritation to the eyes and respiratory tract, as well as inflammation in the lungs which may aggravate chronic respiratory symptoms and increase the frequency or severity of asthma attacks. Exposure to diesel exhaust has also been shown to be associated with an increased incidence of lung cancer in a number of human epidemiology studies.

Because diesel exhaust is a complex mixture of substances, it cannot be measured directly in a simple analytical method. DPM mass, which excludes diesel gaseous emissions, has historically been used as a surrogate measure for whole diesel exhaust. In the Health Assessment Document for Diesel Exhaust, USEPA (2002c) states that "Although uncertainty exists as to whether DPM mass (expressed as $\mu g/m^3$ of DPM) is the most appropriate dosimeter for health effect purposes, it is considered to be a reasonable choice until more definitive information is available about the mechanisms or mode(s) of toxicity action of diesel exhaust." DPM may be estimated by several methods discussed below. This estimate of DPM can then be used in a health risk assessment.

Monitored Data

Although the ambient concentration levels determined at the Allen Park and Southfield sites may or may not be typical of Detroit, the data from these sites were used to provide potential risk estimates. The Allen Park site is both a mobile-source-oriented site and population-oriented site while the Southfield site is characterized solely as a mobile-oriented site (See **Section 3**).

The monitoring data which may be useful for estimating DPM were collected from January 2001 to December 2003 at the Allen Park site and from March 2001 to April 2002 at the Southfield site. These data consist of 24-hr measurements:

Allen Park

1) $PM_{2.5}$ mass (Federal Reference Method) – collected daily.
2) $PM_{2.5}$ speciation – Elemental Carbon (EC), Organic Carbon (OC), Trace Metals, Ions – collected once every three days using MET1 samplers.

Southfield

1) $PM_{2.5}$ speciation – EC, OC, Trace Metals, Ions – collected once every six days using MET1 samplers.

Methods for Estimating DPM

The monitoring data are potentially useful for estimating DPM concentration by using methods such as 1) receptor modeling (a tool for source apportionment), 2) EC surrogate calculations, and 3) dispersion modeling.

Receptor models infer the types of sources and relative contributions of sources that are impacting a receptor site based on measurements for specific pollutants made at that receptor site. Briefly, examples of receptor modeling tools include the Chemical Mass Balance model (CMB model), Positive Matrix Factorization model (PMF model), and UNMIX model. The CMB model is a single-sample receptor model. The CMB receptor model apportions sources by using measurements from the receptor sites and source profiles (fingerprints), which are chemical signatures of specific source emissions. In other words, the CMB model requires the

input of known source profile information to produce results. The PMF model and the UNMIX model, on the other hand, do not require preset emission profiles. These two models are multivariate receptor models that provide source apportionment information from the data input of multiple samples. Thus, these models are used when the sources are not known. The two models, single-sample receptor and multivariate receptor, are different, thus the strengths are different. For example, PMF usually provides more accurate profile information since it estimates source contributions as well as source compositions (profiles). These source profiles, however, are still subject to uncertainty from variability among each modeler's interpretation of the profiles. Another aspect of the PMF model is that it has a feature allowing each data point to be adjusted according to the level of uncertainty in each sampling data point. Thus, high-confidence data would hold more weight on the influence of the model results while low-confidence data would carry less weight. Whichever source apportionment tool is used, ultimately, source profiles and source contributions are determined; mass concentrations and light extinction values are apportioned into factors attributable to the most significant sources.

When a receptor modeling analysis is unavailable, a method of surrogate DPM calculation is useful. Currently, there is no unique marker for estimating the concentration of DPM in the air, but research has shown that EC constitutes a large portion of and strongly correlates with the levels of diesel emissions. To support this statement, USEPA cited a 1985 study conducted by Fowler in the Health Assessment Document for Diesel Exhaust (2002c). This study "evaluated several components of diesel exhaust and concluded that EC is the most reliable overall measure of ambient diesel exhaust exposure" (USEPA, 2002c). It is important to note, however, that while diesel exhaust contributes a great deal to EC levels, there are many other contributing sources such as the burning of coal, oil, or wood; charbroiling; cigarette smoke; and road dust. Nevertheless, USEPA (2002c) went on to say that "[b]ecause of the large portion of EC in DPM, and the fact that diesel exhaust is one of the major contributors to EC in many ambient environments, DPM concentrations can be bounded using EC measurement". Thus, use of EC as a surrogate measure of DPM has become generally accepted as a method that is potentially useful in estimating DPM when a more sophisticated modeling tool for source apportioning is unavailable.

The following examples describe the use of EC surrogate calculations. In a study conducted by Gray (1986), about 67% of EC was attributed to originating from diesel engine exhaust, and about 64% of DPM was determined to be EC in the Los Angeles area. This information was used by the SCAQMD in the Multiple Air Toxics Exposure Study-II (MATES-II) in 2000 to estimate DPM concentrations. The EC concentration measured in the ambient air of the South Coast Air Basin was multiplied by 67% and divided by 64%. The derivation of the method is described mathematically below:

Given that:

DPM = Total Diesel Particulate Matter;
DEC = Elemental Carbon Portion of Diesel;
EC = Elemental Carbon of Atmosphere, **using Gray's (1986) results:**

If

0.64DPM = DEC **and**
DEC = 0.67EC, **then by property of substitution,**
0.64DPM = 0.67EC. **This can be reduced to**
DPM = 0.67EC/0.64 or DPM = EC x 1.04.

An alternative method was provided by USEPA (2002c). Data were gathered from a number of studies, and equations were developed for lower-bound and upper-bound estimates of DPM

based on the analyzed profiles of diesel engines. It was determined that the amount of EC in DPM is typically 52-75% and that 46-68% of ambient EC can be attributed to diesel exhaust.

Thus, the equations for lower-bound and upper-bound estimates of DPM are:

Lower-bound estimate of DPM = 0.46EC/0.75 or DPM = EC x 0.62 and
Upper-bound estimate of DPM = 0.68EC/0.52 or DPM = EC x 1.31.
(USEPA, 2002c)

USEPA also provided an equation for an average of the two ranges:

1) amount of EC in DPM: 52% - 75% and
2) ambient EC attributed to diesel exhaust: 46% - 68%, results in the following equation:
Average estimate of DPM = 0.57EC/0.635 or DPM = EC x 0.89.
(USEPA, 2002c)

Although not stated, it appears USEPA calculated the average estimate based on an arithmetic mean of the lower and upper ends of the two ranges.

Dispersion models predict PM concentrations at receptor sites based on emission factors for the sources and the ability to model the advection, mixing, deposition, and chemical transformation of compounds from the source to the receptor site. The Lagrangian model is an example of a dispersion model that has been used to estimate source contributions to DPM. Predictions of PM_{10}, EC, and OC levels have been found to be reasonable using this model (Cass & Gray, 1995; USEPA, 2002c; SCAQMD, 2000; Allen et al., 2001; Battelle and Sonoma, 2002; Battelle and Sonoma, 2004).

DPM Concentration Estimates

DPM concentrations were estimated for the Allen Park and Southfield sites using two of the methods described above: 1) receptor modeling (performed by STI) and 2) EC surrogate calculations (performed by MDEQ-AQD).

As part of the national air toxics Pilot Project Program, STI analyzed the ambient concentration measurements from MET1 samplers collected from the Allen Park and Southfield sites using a source apportionment tool, PMF (Hafner & Brown, 2003b). Default parameters were used on this preliminary run. After the monitoring data was entered into the PMF model, diesel source apportionment was performed using the following characteristics known to be associated with diesel: a greater-than-one EC to OC ratio, and the levels of manganese, SVOCs, and PAHs. Results indicated that about 5% (Allen Park) to 9% (Southfield) of the total $PM_{2.5}$ mass was attributable to a diesel source factor. The average mass of ambient diesel concentrations was 1.15 $\mu g/m^3$ and 2.04 $\mu g/m^3$ in Allen Park and Southfield, respectively. These values are comparable to the lower range of the results from the study conducted in Los Angeles (Gray, 1986), where the DPM concentration "ranged from 10 $\mu g/m^3$ at the most polluted locations to 1.7 to 3.3 $\mu g/m^3$ in less polluted areas" (Cass & Gray, 1995).

To estimate DPM concentrations using EC surrogate calculations, two different estimates of proportions are first necessary: 1) the proportion of DPM load that is EC and 2) the proportion of EC in the ambient air that is associated with diesel. These proportions, however, cannot be estimated without replicating the methods of Gray (1986). Since proportion estimates specific to the Detroit area are not available, a default option of using the USEPA (2002c) equations for lower-bound and upper-bound estimates was used. The monitoring data gathered from the Allen Park site and Southfield site are presented in **Table 59** and **Table 61**, respectively. The USEPA (2002c) equations for DPM and the resulting estimates are tabulated in **Tables 60** and **Table 62** for Allen Park and Southfield, respectively.

Table 59: EC Data from Allen Park

YEAR	# OF SAMPLES	EC CONCENTRATION ARITHMETIC AVERAGE & STANDARD DEVIATION	LOWEST MEASURED VALUE	HIGHEST MEASURED VALUE
2001	N = 95	0.69±0.33 µg/m^3	0.00019 µg/m^3	1.71 µg/m^3
2002	N = 112	0.86±0.47 µg/m^3	0.264 µg/m^3	3.78 µg/m^3
2003	N = 117	0.78±0.39 µg/m^3	0.0255 µg/m^3	2.04 µg/m^3

Table 60: DPM Calculations – Allen Park

YEAR	LOWER-BOUND DPM = (EC x 0.46)/0.75	UPPER-BOUND DPM = (EC x 0.68)/0.52	AVERAGE DPM = EC x 0.89
2001	0.42 µg/m^3	0.90 µg/m^3	0.61 µg/m^3
2002	0.52 µg/m^3	1.1 µg/m^3	0.76 µg/m^3
2003	0.48 µg/m^3	1.0 µg/m^3	0.69 µg/m^3

Table 61: EC Data from Southfield

YEAR	# OF SAMPLES	EC CONCENTRATION ARITHMETIC AVERAGE & STANDARD DEVIATION	LOWEST MEASURED VALUE	HIGHEST MEASURED VALUE
2001	N = 43	0.76±0.32 µg/m^3	0.323 µg/m^3	1.62 µg/m^3
2002	N = 17	0.75±0.33 µg/m^3	0.296 µg/m^3	1.48 µg/m^3

Table 62: DPM Calculations – Southfield

YEAR	LOWER-BOUND DPM = (EC x 0.46)/0.75	UPPER-BOUND DPM = (EC x 0.68)/0.52	AVERAGE DPM = EC x 0.89
2001	0.47 µg/m^3	0.99 µg/m^3	0.68 µg/m^3
2002	0.46 µg/m^3	0.98 µg/m^3	0.66 µg/m^3

The PMF model result of 1.15 µg/m^3 for Allen Park is very comparable to the upper-bound estimates for Allen Park in **Table 60**. The PMF model result of 2.04 µg/m^3 for Southfield is roughly 2-fold higher than the calculated upper-bound estimate for Southfield in **Table 62**. All of these estimates for the Detroit sites are lower than the estimated levels for the eight monitoring sites in California's MATES-II study, which had a range of 2.408 µg/m^3 to 4.742 µg/m^3, and an arithmetic average of 3.523 µg/m^3 (Gray, 1986; Cass and Gray, 1995; ARB, 1998a; Hafner & Brown, 2003a; Hafner & Brown, 2003b).

While the DPM estimates for the Southfield and Allen Park sites are not without some uncertainty, they represent the best estimates based upon the available data. Better data is expected in the near future. USEPA's ORD plans to run source apportionment models as a part of the DEARS ongoing project. Source apportionment modeling tools will be run on monitored data from the Interagency Monitoring of Protected Visual Environments (IMPROVE) network of samplers, which also provides speciated OC data. With greater knowledge of the composition of OC in addition to measurements of EC, the source apportionment results can be more specific and the contribution of diesel can be better quantified. These results, however, will not be available until 2006 at the earliest (Heindorf, 2004).

Risk Characterization

The monitoring data collected were ambient concentration measurements; no personal exposure measurements were made. Thus, for the exploratory purpose of this section, the available ambient monitoring data are assumed to be reasonable surrogate data for personal exposure.

The source apportionment results from the PMF model were the only data available for further analysis and characterization of risk. While there are other modeling methods, such as those from the CMB receptor model, they were not available for analysis.

Because the EC surrogate calculations presented above are based on the USEPA (2002c) nationwide diesel estimates, the resulting DPM estimates can only be extrapolated to Detroit with significant uncertainty. In contrast, the estimates from the PMF model-run analysis were obtained by using species concentration measurements specifically from sites in the Detroit area. Therefore, the results from the PMF source apportionment were considered more appropriate for use in characterizing the potential health risks from exposure to diesel emissions in Detroit.

Non-cancer Assessment

There is not an abundance of information regarding acute toxicity from diesel exhaust. No acute health benchmark has been established for diesel exhaust at present. Some acute inhalation studies were presented by the Air Resource Board (ARB, 1998b) and are discussed briefly below.

A study conducted by Pepelko and Peirano (1983) reported the effects from inhalation exposure to diluted diesel exhaust for two hours or six hours. Mice exposed to 6 mg/m^3 for two hours showed no adverse effects. Six hours of exposure to 6 mg/m^3 of diesel exhaust, however, resulted in greater infectivity of Streptococcus pyogenes leading to higher mortality incidences. The average percent of deaths in the six-hour exposure group was 31.7%, while control mice had an average value of 6.7%. From these study results, ARB concluded that exposure to diesel exhaust "greatly enhances the infectivity of one of the bacterial challenges" (ARB, 1998b). In a study conducted by Ulfvarson et al. (1995), rabbits were exposed to filtered or unfiltered diesel exhaust for 24 hours by inhalation. Both groups of rabbits exposed, either to filtered or unfiltered diesel exhaust, showed increased airway resistance. In addition, lung compliance was increased or decreased for unfiltered and filtered diesel exhaust, respectively.

USEPA has established a DPM RfC value of 5 $\mu g/m^3$. This value is identical to the chronic REL established by California's OEHHA. The RfC was based on a chronic animal (rat) inhalation exposure study where the critical effects were pulmonary inflammatory and histopathological changes (IRIS, 2005). The HQ value is determined as the estimated DPM concentration divided by the RfC value of 5 $\mu g/m^3$.

Using the estimated DPM concentrations of 1.15 $\mu g/m^3$ and 2.04 $\mu g/m^3$ for the Allen Park and Southfield sites respectively, the HQs are 1.15 $\mu g/m^3 \div 5\ \mu g/m^3 = 0.2$ and 2.04 $\mu g/m^3 \div 5\ \mu g/m^3 = 0.4$. Since these HQs are < 1, this finding suggests that chronic non-cancer effects from exposure to DPM alone would not be expected to occur. DPM may, however, interact with other air toxics and criteria pollutants collectively to affect the respiratory tract.

> "Diesel exhaust exposure could be additive or synergistic to concurrent exposures to other air pollutants. For example, there is evidence that DPM that has been altered by being in the presence of ambient ozone significantly increases the rat lung inflammatory effect compared to DPM that was not subjected to ozone.... Other concerns include the possible impacts for children and adults on the exacerbation of existing allergens resulting from diesel exhaust exposure" (USEPA, 2002c).

Therefore, HQ values of 0.2 and 0.4 could be significant in this overall contribution, depending upon the concentrations of other air toxics present that may impact the respiratory system.

Cancer Assessment

The Cal EPA has established a cancer unit risk value of 3 X 10^{-4} $(\mu g/m^3)^{-1}$ for DPM. This value was derived from analyses of retrospective case-control and cohort studies conducted by Garshick et al. (1987 and 1988, respectively) which studied the association between railroad workers exposed to diesel exhaust and the development of lung cancer. The resulting risk values ranged from 1.3 X 10^{-4} $(\mu g/m^3)^{-1}$ to 24 X 10^{-4} $(\mu g/m^3)^{-1}$. Cal EPA concluded that the unit risk factor would be closer to the lower-end of the range after conducting analyses of these studies as well as a meta-analysis of multiple occupational studies (ARB, 1998b). The cancer unit risk value of 3 X 10^{-4} $(\mu g/m^3)^{-1}$ represents the increased probability that a person will develop cancer when exposed daily to 1 µg of DPM per m^3 in air over a 70-year lifetime.

USEPA has stated that "diesel exhaust is *likely to be carcinogenic* to humans by inhalation exposure at any exposure condition" (IRIS, 2005) after reviewing:

- epidemiologic studies where a positive correlation between occupational exposure and increased risk of cancer was found;
- animal studies where chronic inhalation exposure to DPM concentrations increased the development of lung cancer;
- non-inhalation animal studies (via intratracheal instillation, lung implantation, skin painting, or subcutaneous injection) also showed evidence of carcinogenicity; and
- the known constituents of DPM that have already been established as mutagenic or carcinogenic compounds (e.g., PAHs and derivatives, benzene and acetaldehydes).

After evaluating the carcinogenic effects of diesel exhaust and DPM exposures, USEPA did not establish a cancer unit risk value for diesel exposure. This is due to a lack of confidence in quantitatively assessing the supporting data. Some of the critical issues regarding attempts to quantify the cancer risk from the aforementioned studies include (not all inclusive):

1) retrospective estimation of exposure
2) inclusion of potentially exposed groups
3) accounting for background concentrations
4) complete data collection of lung cancer incidence.

Another issue that may be of concern is that the changes in fuel composition and new engine technology over time may render the above studies inapplicable for health risk assessment of current diesel exhaust exposure. In discussing this issue, however, both USEPA (IRIS, 2005) and HEI (2003) disagreed that this was a compelling point. USEPA (2002c) also addressed the question of the relevancy of occupational exposures to ambient concentrations. They found that the lower end occupational exposures and the higher end of environmental exposures were similar enough to decrease the uncertainty associated with extrapolating possible risk to the environmental setting.

USEPA has evaluated the causal association between DPM exposure and lung cancer development by using the set of criteria put forth by Hill (1965). After completing a thorough analysis, USEPA stated that "[i]n conclusion, the epidemiologic studies of exposure to diesel exhaust and occurrence of lung cancer furnish evidence that is consistent with a causal association" (USEPA, 2002c). A panel of researchers, formed by HEI, looked at the basis for

Cal EPA's unit risk factor (the 1987 and 1988 Garshick et al. studies) and recommended that the value not be used for quantitative risk assessment; however, they do not disregard the biological significance of the epidemiological study results that establish an association. HEI, whose analyses produced negative results, realizes that "the results are generally consistent with findings of a weak association between lung cancer and exposure to diesel exhaust" and that "the possibility that strong biases in these data have masked a true association cannot be ruled out" (HEI, 1999).

While USEPA has concluded that the available data are too uncertain at this time for a quantitative dose-response analysis and development of a cancer unit risk for DPM, they have attempted to provide some perspective on the possible magnitude of cancer risk. In the Health Assessment Document for Diesel Exhaust (USEPA, 2002c), they state:

> "Although the available data are considered inadequate to confidently estimate a cancer unit risk, this does not mean that there is no information about the possible cancer risk of diesel exhaust. To examine the significance of the potential cancer hazard from environmental exposure to diesel exhaust, all relevant epidemiologic and exposure data as well as simple risk assessment tools can be used. Such an approach does not produce confident estimates of cancer unit risk. Rather, these exploratory approaches provide a perspective on the possible magnitude of cancer risk and thus insight about the potential significance of the hazard."

USEPA (2002c) presents and discusses the estimation and derivation of a possible unit risk factor:

> "A discussion of possible risk is presented in the form of a perspective on the possible magnitude of risk from environmental exposure. The perspective discussion notes the small exposure margins and possible overlap between some occupational and environmental exposure levels. This lessens the uncertainty of extrapolating the occupational hazard and observed risk into the environmental setting. Furthermore, based on a more quantitative approach involving the observed lung cancer from occupational exposures and the magnitude of occupational and environmental exposure differences, an exploratory risk analysis shows that environmental cancer risks possibly range from 10^{-5} to nearly 10^{-3}. Further research is needed to more accurately assess and characterize environmental cancer risks of diesel exhaust."

Notwithstanding USEPA's judgment that the carcinogenic risk of DPM cannot be reliably quantified, it is instructive for the present purposes of the DATI risk assessment report to utilize the Cal EPA unit risk factor to estimate the potential risk from DPM exposure. An estimate of the potential cancer risk can, therefore, be calculated for the Detroit area utilizing the Cal EPA risk unit factor of 3×10^{-4} $(\mu g/m^3)^{-1}$. Using the estimated DPM concentrations for Allen Park and Southfield, the incremental cancer risk from inhalation exposure to DPM in the Detroit area is as follows:

Allen Park: 3×10^{-4} $(\mu g/m^3)^{-1} \times 1.15$ $\mu g/m^3 = 345 \times 10^{-6} \approx 300 \times 10^{-6}$
Southfield: 3×10^{-4} $(\mu g/m^3)^{-1} \times 2.04$ $\mu g/m^3 = 612 \times 10^{-6} \approx 600 \times 10^{-6}$

The estimated cancer risks of 300 and 600 $\times 10^{-6}$ for the Allen Park and Southfield sites, respectively, are consistent with the USEPA (2002c) range of 10^{-5} to 10^{-3} (10 to 1000 $\times 10^{-6}$) estimated by USEPA for environmental exposures to diesel exhaust. It is important to note, again, that these estimated DPM levels for the Detroit area, as well as the Cal EPA unit risk factor, have a substantial level of uncertainty (CARB, 2000; USEPA, 2002c; SCAQMD, 2000; IRIS, 2005).

Other Available Study Results

A few studies are available that have determined estimates of DPM in other parts of the U. S. by using source apportionment tools (e.g., PMF, UNMIX, CMB, etc.). Five sites that were monitored and source apportioned are: Denver, Colorado in 1997; Los Angeles, California in 1982; Phoenix Super Site, 1994-1995; San Joaquin Valley, California in 1995; Western Washington State in 1996-1997; and Seattle, Washington in 1996-1999. The results of these apportionment studies indicate a wide range of values (**Table 63**) for the diesel portion of various parameters.

Table 63: Diesel Source Apportionment Results from Different Cities

STUDY	DIESEL PORTION	TRACER
Denver, Colorado* 1997	49-68% of EC 7-9% of OC 10% of $PM_{2.5}$	EC, OC
Los Angeles, California* 1982	94% of EC 14-30% of OC 12-35% of $PM_{2.5}$	EC, OC
Phoenix Super Site, Arizona** 1994 - 1995	15% of $PM_{2.5}$	EC, OC
San Joaquin Valley, California* 1995	97% of EC 4-12% of $PM_{2.5}$	EC, OC
Western Washington State* 1996 - 1997	8-12% of $PM_{2.5}$	EC, OC
Seattle, Washington* 1996 - 1999	36% of EC 19% of OC 15% of $PM_{2.5}$	EC, manganese (TOR fractions)

*Holdren & Main, 2001
**Maricopa Association of Government, 1999

The percent values presented here are estimates of diesel contribution to ambient EC, OC, and $PM_{2.5}$ concentrations. **Table 63** indicates that each city and each site has a unique composition of diesel exhaust in the ambient air. Had the diesel-specific values from each city been similar, general values for diesel estimates may have been established, allowing for greater confidence in estimating of the amount of DPM in the air in Detroit.

Besides estimated percentages of diesel fractions (shown in **Table 63**), DPM concentrations derived via source apportionment have been reported for the Phoenix Super Site study presented in **Table 63**. Source apportionment of data monitored during November 1994 through March 1995 in Phoenix resulted in an average DPM concentration of 2.4 $\mu g/m^3$ with a maximum level of 5.3 $\mu g/m^3$.

Another study that has reported DPM concentrations in detail is California's MATES-II. The EC monitoring data from eight sites in California are presented in **Table 64**. These results are from data gathered during April 1998 through March 1999. The estimated DPM concentrations are shown in the far right column of the table in bold. The values were obtained by entering the EC concentrations into the equation derived from Gray's study results: DPM = (EC x 0.67)/0.64 (USEPA, 2002c; SCAQMD, 2000; Holdren & Main, 2001).

Table 64: EC Data from MATES-II Study and DPM Calculations

SITE	# OF SAMPLES	EC CONCENTRATION	DPM ESTIMATE DPM = (EC*0.67)/0.64
Anaheim	58	2.30 μg/m^3	2.408 μg/m^3
Burbank	53	3.18 μg/m^3	3.329 μg/m^3
Los Angeles	59	3.53 μg/m^3	3.695 μg/m^3
Fontana	59	3.10 μg/m^3	3.245 μg/m^3
Huntington Park	46	4.53 μg/m^3	4.742 μg/m^3
Long Beach	58	2.54 μg/m^3	2.659 μg/m^3
Pico Rivera	38	4.35 μg/m^3	4.554 μg/m^3
Rubidoux	62	3.39 μg/m^3	3.549 μg/m^3
Average	**–**	**3.365** μg/m^3	**3.523** μg/m^3

Summary

The purpose of this assessment of diesel emissions information is to address key question #7 (from **Section 2.5**):

> *Do the monitoring data and risk assessment help to scope the potential relative health significance of diesel emissions in the Detroit area?* (emphasis added).

Analysis of monitoring data gathered between 2001 and 2003 can be used to provide estimates of DPM concentrations for the Detroit area. Two methods were employed to determine concentration estimates: 1) STI ran a preliminary analysis using the PMF model, and, 2) MDEQ-AQD calculated rough estimates using measured EC levels and nationally-based conversion factors developed by USEPA. The DPM concentrations obtained via the PMF model resulted in estimates of 1.15 μg/m^3 and 2.04 μg/m^3 DPM for the Allen Park and Southfield sites, respectively. Although these values are uncertain, they serve to provide a general sense of how much contribution DPM may add to the risk of carcinogenic and non-carcinogenic adverse health effects. Based on USEPA's RfC value of 5 μg/m^3, the HQ was calculated to be less than one (0.2 for Allen Park and 0.4 for Southfield). Increased cancer risk was calculated assuming California's cancer unit risk factor of 3 X 10^{-4} (μg/m^3)$^{-1}$. This resulted in estimated increased risk of cancer in the range of approximately 300 to 600 X 10^{-6}, based on the estimated DPM levels at the Allen Park and Southfield sites. In the Health Assessment Document for Diesel Exhaust, USEPA (2002c) provided an estimated range of 10^{-5} to 10^{-3} (10 to 1000 X 10^{-6}) for environmental cancer risk from exposure to diesel exhaust. The estimated cancer risks (300 to 600 X 10^{-6}) presented in this section for the Detroit sites fall within the range provided by USEPA. This estimated range of cancer risk is considered to have significant uncertainty, however, it suggests that diesel exhaust may be a significant risk driver in the context of the cancer risk estimates for other Detroit-area air toxics evaluated in the present report.

11.0 UNCERTAINTY ASSESSMENT

11.1 Overview

Risk assessment is based on analysis and interpretation of information on toxicity and exposure, which is integrated into a risk characterization that is useful for risk managers and the public. However, highly accurate information is often not available for many aspects of a risk assessment. Risk assessments are often not as definitive in all important areas as would be desirable, and risk assessors often need to apply professional judgment or standard default assumptions based on policy. In a transparent risk assessment process, it is important to identify and describe the areas of uncertainty. The USEPA's Air Toxics Risk Assessment Reference Library (USEPA, 2004b and c) provides guidance and a useful construct for describing the areas of uncertainty in risk assessment for air toxics, which is utilized in this section.

It is important to distinguish between the terms *uncertainty* and *variability*. Uncertainty occurs because of a lack of knowledge about specific factors, parameters, or models. It can be reduced by collecting more and better data. In contrast, variability refers to true heterogeneity or diversity, for example, differences among people in their activity patterns which can affect their level of exposure to a pollutant, or differences in how they respond to the same level of pollutant exposure. Variability can be better characterized with more data, but it cannot be reduced or eliminated by practical means. If available data on variability are limited, as is often the case with risk assessments, variability can be treated as a type of uncertainty.

Uncertainties affect the confidence of any risk estimates developed for individuals exposed to the substances in question. The presence of uncertainty in risk assessment does not imply that the results of the risk assessment are wrong, but rather that the risks cannot be estimated beyond a certain degree of confidence.

Risk assessments often do not include a detailed, quantitative characterization of the uncertainty in each element of a risk assessment or in the final risk estimates. This is not always possible due to limited data, and it may not be necessary if all reasonable assumptions in the risk assessment lead to essentially the same risk characterization and recommendation. Nevertheless, some level of uncertainty assessment should be included in risk assessments, with qualitative, semi-quantitative, or quantitative approaches. The USEPA (2004f), states that,

> "USEPA has been examining its practice of uncertainty analysis over the years. Generally speaking, USEPA has applied a qualitative approach to characterizing uncertainty (and variability for that matter). Quantitative characterization has been used more unevenly. Also, USEPA typically uses deterministic approaches to characterize risk – although, increasingly often, USEPA applies probabilistic techniques for characterization of risk, usually within exposure assessments."

For the present DATI assessment, deterministic approaches were applied, and quantitative characterization of uncertainty was not performed. Most of the areas of uncertainty are described qualitatively in this section, accompanied where feasible with a description of the level of conservatism or non-conservatism.

The USEPA Air Toxics Risk Assessment Reference Library (USEPA, 2004 b and c) states that the sources of uncertainty in risk assessments are often divided into four categories as follows:

1. Scenario uncertainty: Information to fully define exposure or risk is missing or incomplete. In general, scenario uncertainty occurs when information to fully define

exposure and/or risk is missing or incomplete. This may include descriptive errors regarding the magnitude and extent of chemical exposure and toxicity, temporal and spatial aggregation errors, incomplete analysis (e.g., missing exposure pathways), and potential mis-specification of the exposed population or exposure scenario.

2. Model uncertainty: Algorithms or assumptions used in models may not adequately represent reality. In general, model uncertainty is associated with all models used in all phases of a risk assessment, including (1) animal models used as surrogates for evaluating human carcinogenicity, (2) dose-response models, (3) computer models used to predict the fate and transport of chemicals in the environment, and (4) models used to estimate exposures for populations of concern.

3. Parameter uncertainty: Values for model parameters cannot be estimated precisely. In general, parameter uncertainty refers to the limitations in the modelers' ability to estimate precise values for certain parameters (variables) in the chosen models. It is a generic term that in common usage can refer to either variability or uncertainty, and generally indicates a situation where a given variable may take a range of values, rather than a single point estimate. Parameter uncertainty is generally addressed in risk assessment through gathering additional data, sensitivity analysis, or probabilistic modeling.

4. Decision-rule uncertainty: Policy and other choices made during the risk assessment may influence risk estimates. In general, decision-rule uncertainty is associated with policy and other choices made during the risk assessment. For example, the number of COPC evaluated at a given tier of assessment may be reduced through use of a toxicity-weighted or risk-based screening analysis. This type of judgment introduces uncertainties about the contribution of the omitted air toxics to overall exposure or risk. As another example, risk managers may decide to select as chemicals for risk reduction efforts only those chemicals that, individually, pose a risk above some specified level. For any given risk assessment, some or all of these practices may be questioned, either on technical grounds (e.g., a risk number has been generated, but it is highly uncertain) or for policy reasons.

Although USEPA's distinction between the above four types of uncertainty is informative, an alternative construct for presenting the areas of uncertainty was utilized for better clarity in this report. In the following, the areas of uncertainty are discussed according to their appearance in each step in the risk assessment process (i.e., monitoring data collection; exposure assessment; toxicity assessment; and risk characterization).

11.2 Monitoring Data Collection

Basing a risk assessment on air toxics monitoring data carries with it several areas of uncertainty, which contrast with the set of uncertainties associated with risk assessments based on emission inventories and dispersion modeling. The uncertainty of each analytical measurement is dependent upon many factors, which include: the concentration of the analyte and proximity to limit of detection, the performance of the instrumentation on a given day, the skill of the analyst, the most recent instrument maintenance, the cleanliness of the sample media, sample handling and transportation issues, etc. The uncertainty of each measurement will vary by day, sample and analyst, precluding definitive estimates of uncertainty for each parameter in each sample for each day.

During the Detroit Pilot Project, MDEQ implemented a variety of split samples in an attempt to better understand sources of variability and uncertainty in laboratory measurements of air

toxics. The variability was influenced by the source (interlaboratory, intersite, intersampler, intramethod) and was very dependent upon the analyte. To better understand interlaboratory variability, samples have been exchanged by states within Region 5 from 2002 to 2004. Also, the California ARB and USEPA Region 4 have shared samples on a national basis, from 2001 to the present. The results are helping to estimate the relative performance of each laboratory for each analyte over time.

Beginning in 2004, USEPA implemented a National Performance Audit Program, for all laboratories involved in the NATTS program. The data from this program will allow an assessment of the accuracy of analyses for the USEPA's 18 core air toxics compounds. MDEQ is in the process of compiling the results. This database will enable the estimation of the uncertainty of the air toxics measurements. The findings are available as quarterly reports.[22]

Variability of ambient monitoring data is impacted by the equipment used to collect the samples. The presence of a manifold, the make and model of the sampler, the system leaks, the accuracy of the flow rate, adherence to calibration and maintenance schedules, exterior temperature and humidity are some of the factors that may introduce additional variability into the reported concentrations. The lot numbers of filters or cartridges may also play a role, if that specific lot arrives contaminated from the supplier.

Perhaps the greatest contribution to the levels of uncertainty associated with laboratory results is the proximity of the concentration of the analyte to the detection limit. As the concentration of the analyte decreases, the level of uncertainty will increase as the MDL is approached. Several other factors will also impact the uncertainty associated with measurements generated by different laboratories. The accuracy and precision of the results will vary by laboratory. The uncertainty associated with each measurement is influenced by factors that include: instrumentation and procedures, skill of the analysts, sample handling practices, maintenance schedules, quality control practices, humidity levels, etc.

The number of samples collected or sampling frequency impacts the variance around the annual average. Obviously, as the sampling frequency varies by site, the number of samples that are collected per year at that site will also vary. When these results are averaged by site, the datasets with a larger number of samples usually will show a smaller variance around the mean because the sample subset becomes more representative of the universe of samples. Conversely, using a smaller number of samples to calculate an annual mean may actually be more biased toward either tail of the distribution, resulting in the calculation of a larger variance around the mean.

Despite concerns that some monitoring data may not be as accurate or precise as desired, the data utilized in this report were collected using state-of-the-art methods and quality control measures. The data are considered to be useful and appropriate for inhalation risk assessment purposes with proper caveats regarding uncertainties with the data. The overall confidence in the data should be at least as high as it is for other contemporary monitoring efforts with relatively fewer monitoring sites and less comprehensive scopes. Air toxics risk assessments based upon emissions estimates and the modeling of dispersion and fate also entail multiple areas of uncertainty, although a comparison of the relative magnitude of uncertainty between the two approaches was beyond the scope of this study.

Another source of uncertainty is in the assessment of DPM in Detroit. Although the level of DPM was not measured directly as part of this study, surrogate measurements were taken,

[22] E.g., MDEQ NATTS Quarterly Report, October 1, 2004 to December 31, 2004. January 19, 2005. 23pp.

which adds uncertainty in the assessment. **Section 10** includes a discussion of the potential levels of DPM that may be present. There is substantial uncertainty in doing so, as described in **Section 10**. However, for the purposes of this assessment it is an appropriate issue to pursue in order to estimate the potential levels of DPM which may be present in an urban location like Detroit.

11.3 Exposure Assessment

Only the inhalation pathway of exposure was evaluated. As discussed in **Section 2.0**, the goal of the risk assessment was to assess the health risks from inhalation exposure to air toxics in the Detroit area. It is recognized that some air toxics are persistent in the environment and may deposit to the ground or to water bodies, potentially leading to non-inhalation (multipathway) routes of exposure. Accounting for potential multipathway exposure generally involves many estimates and assumptions, with inherent uncertainties and variability. Multipathway exposure and risk assessment was beyond the scope of this initiative's goals.

Monitoring was not intentionally conducted at particular "industrial fenceline" locations or sites estimated by emissions inventory and modeling of impacts to represent maximally impacted locations from individual or collective air emission sources. Therefore, there is no assurance that the monitored data represent worst-case impacts, and more impacted "hot spots" may exist. This area of uncertainty indicates a lack of conservatism in the exposure and risk assessment; higher impacted locations and higher risk estimates may be present in the Detroit area.

All monitoring was conducted in areas that generally are populated (although relatively sparsely populated, in the case of the background monitoring site at Houghton Lake). Notwithstanding the potential for inaccuracies in the measurements of air toxics (as discussed in **Section 11.2**), ambient air monitoring data provide a more direct approach to exposure assessment than assessments based on emission estimates, dispersion modeling, and the estimation of potential ambient air levels.

Chronic exposure and risk assessments for most sites utilized discontinuous monitoring of the daily levels to estimate annual average levels. The chronic exposure parameter was the central tendency estimate, obtained as the arithmetic average of the daily monitoring data. The intention was to base the exposure and risk estimates on the estimated long-term average exposure levels. It is recognized that this method may underestimate or overestimate the true annual average. As discussed in **Section 7**, USEPA (2004b, App. I) suggested that the 95% UCL of the mean may be used as a health protective (conservative, upper-bound) estimate of the true annual average. For the present assessment, a comparison was made of the difference between the arithmetic annual average values and the 95% UCL on the arithmetic average (assuming normal distributions). This was done for the 12 cancer risk drivers (which includes naphthalene) and the primary noncancer compounds of interest (manganese and naphthalene). A summary of the findings is presented in **Table 65**. This characterizes the difference between the annual average levels utilized in the risk assessment and the high-end potential annual average levels:

Table 65: Percent Increase In The Annual Average Concentration If Based On The 95% UCL On The Mean Rather Than The Mean.[1]

CHEMICAL	ACROSS ALL SITES, RANGE OF THE % INCREASE BETWEEN THE ANNUAL AVERAGE AND THE 95% UCL ON THE ANNUAL AVERAGE
1,3-butadiene	9% (Dearborn) to 29% (River Rouge)
1,4-dichlorobenzene	4% (Dearborn) to 178% (N. Delray)

Table 65: Percent Increase In The Annual Average Concentration If Based On The 95% UCL On The Mean Rather Than The Mean.[1]

CHEMICAL	ACROSS ALL SITES, RANGE OF THE % INCREASE BETWEEN THE ANNUAL AVERAGE AND THE 95% UCL ON THE ANNUAL AVERAGE
Acetaldehyde	10% (Dearborn) to 26% (River Rouge)
Acrylonitrile	3% (S. Delray) to 29% (N. Delray)
Arsenic	15% (N. Delray) to 30% (S. Delray)
Benzene	6% (Dearborn) to 41% (S. Delray)
Cadmium	12% (Allen Park) to 71% (N. Delray)
Carbon tetrachloride	2% (Dearborn) to 9% (N. Delray)
Formaldehyde	9% (Dearborn) to 31% (Houghton Lake)
Manganese	11% (N. Delray) to 27% (S. Delray)
Methylene chloride	34% (Dearborn) to 140% (River Rouge)
Naphthalene	11% (Southfield) to 74% (N. Delray)
Nickel	12% (Allen Park) to 86% (Southfield)

[1] The air toxics listed in this table include the 12 cancer risk drivers (with a cancer risk estimate of 1×10^{-6} or higher at one or more sites) and the chronic noncancer compounds of greatest interest (naphthalene and manganese; HQ of 1 or greater at one or more sites).

Table 65 indicates that for most of the substances there is a fairly substantial increase in the estimated annual level, if estimated by the 95% UCL on the mean rather than the mean (which was utilized in this report). It should be recognized that the noted increases in concentrations for specific substances and sites is not an indication of the magnitude of increase in the total cancer risk for those sites, since the relative contribution of each carcinogen to the total cancer risk per site varied greatly. **Table 66** provides the total cancer risk by site based on the annual average level (as presented in **Section 9.1.3**), in comparison to the total cancer risk by site based on the 95% UCL on the mean, and the percent increase. This comparison indicates that the use of the 95% UCL results in relatively modest increases in the estimated total cancer risk for some sites, ranging up to fairly substantial increases for other sites (**Table 66**).

Table 66: The Site-Specific Total Cancer Risk Estimates (lifetime incremental risk per million) Using the 95% UCL on the Mean Rather than the Mean Levels

SITE	TOTAL CANCER RISK (PER MILLION) BASED ON THE MEAN LEVELS	TOTAL CANCER RISK (PER MILLION) BASED ON THE 95% UCL ON THE MEAN LEVELS	PERCENT INCREASE BETWEEN THE MEAN LEVEL AND THE 95% UCL
Allen Park	473	860	82
Dearborn	101	115	15
S. Delray	525	697	33
N.E. Detroit	53	59	10
Houghton Lake	27	35	30
Southfield	111	146	32
River Rouge	130	166	27
N. Delray	236	348	48
Ypsilanti	35	43	25

The data utilized for assessing acute exposures and risks carries a substantial amount of variability and uncertainty. For most sites, monitoring was conducted only every 6th or 12th day. This limitation of the data may fail to capture the highest daily levels. The exception to this was

the Dearborn site where daily monitoring occurred for VOCs and carbonyls, providing greater confidence in capturing the highest daily levels. Also, the smallest time increment of the data for acute exposure and risk assessment was one hour for one site (N.E. Detroit, for VOCs which are PAMs) and 24 hours for all other sites. Therefore, the data do not enable the assessment of peak levels over periods of < 1 hour (one site) or < 24 hours (all other sites). Under those limitations, the acute exposure and risk assessment focused upon the highest measured 24-hr or 1-hr level (as available).

This study utilized ambient air data as a surrogate for actual exposures. There was no accounting for the variability of human activity, or for the reasonableness of actual human presence at the monitoring sites. This approach is characteristic of a "screening level" type of approach. Actual exposures would include exposures while people are indoors in residences, workplaces, in vehicles, etc. As noted in **Section 7.0**, the USEPA's NATA (1996) study adjusted the modeled ambient air levels of air toxics to account for human activities and locations which can affect exposure to ambient air toxics. In that study, the estimated human exposure levels tended to be approximately 80% to 100% of the modeled ambient air estimates. The results of a recent study conducted for the HEI (2004), "… support an assumption used in many epidemiologic studies of acute health effects of $PM_{2.5}$: that central site monitoring data reasonably estimate the contribution of outdoor PM to personal and indoor exposure over space and time within a given region for a homogeneous subpopulation of adults". Of course, the above only addresses the potential exposure to ambient air levels, and does not account for exposure to air toxics that are generated in microenvironments, such as in homes and in vehicles.

The exposure and risk assessment was primarily based upon the Detroit Pilot Project monitoring, from April 2001 through April 2002. These data formed the basis for estimated exposure levels over that year, and they were assumed to be representative of ambient air levels and inhalation exposure over a lifetime. Other than follow-up activities to investigate some of the particularly high levels of certain air toxics at certain sites, there was no attempt to collect further longitudinal information on air toxics levels for the risk assessment. Air toxics levels may vary over time, and the monitoring data utilized may or may not adequately represent exposure levels occurring before or after the year of the Pilot Project. This is a common limitation of such studies, and this is a noteworthy uncertainty in the risk assessment. It is not feasible to characterize this uncertainty as to the level of conservatism that it may or may not impart to the risk assessment.

To enable the assessment of chronic exposure based on datasets containing ND levels, data which were analytically nondetectable were treated as ½ of the MDL. This is a standard USEPA practice. In general, more confidence is imparted in the exposure and risk estimates for datasets with relatively high percentages of detectable levels, in comparison to datasets with relatively lower detectability (yet still above the 15% criterion). **Table 67** shows the percentage of samples with detectable levels, for those sites and air toxics which met the criteria for being COPC in this study. This table indicates that most of these datasets had very high detection rates (90% or higher). A few of the datasets had moderate (generally 50-90%) detection (1,3-butadiene; acrylonitrile), while 1,4-dichlorobenzene had relatively lower detection rates (17-35%). Of the datasets with relatively low or moderate detection rates, 1,3-butadiene was a relatively small risk driver (< 10×10^{-6} incremental cancer risk) while acrylonitrile (91×10^{-6}) and 1,4-dichlorobenzene (1 to 25×10^{-6}) were relatively significant contributors to the total cancer risk estimates.

Table 67: COPC (With Cancer Risk of 1 X 10^{-6} or Higher, or HQ of 1 or Higher) at Specific Sites and Percent Detection

CHEMICAL NAME	SITE NAME	% DETECT	HQ AVG/ITSL	RISK PER MILLION (AVG/IRSL)
1,3-Butadiene	Allen Park	67		4.00
1,3-Butadiene	Dearborn	56		3.94
1,3-Butadiene	S. Delray	80		6.33
1,3-Butadiene	Southfield	83		6.37
1,3-Butadiene	River Rouge	33		3.68
1,4-Dichlorobenzene	Dearborn	16		1.78
1,4-Dichlorobenzene	S. Delray	18		2.12
1,4-Dichlorobenzene	Southfield	19		1.88
1,4-Dichlorobenzene	River Rouge	17		1.57
1,4-Dichlorobenzene	N. Delray	35		25.04
Acetaldehyde	Allen Park	97		3.08
Acetaldehyde	Dearborn	98		4.19
Acetaldehyde	S. Delray	100		3.45
Acetaldehyde	E 7 Mile	100		3.57
Acetaldehyde	Houghton Lake	97		1.72
Acetaldehyde	Southfield	100		3.17
Acetaldehyde	River Rouge	97		4.23
Acetaldehyde	N. Delray	100		4.34
Acetaldehyde	Ypsilanti	100		2.17
Acrylonitrile	N. Delray	46		91.37
Arsenic (TSP)	Allen Park	100		9.47
Arsenic (TSP)	Dearborn	100		12.05
Arsenic (TSP)	S. Delray	100		16.80
Arsenic (TSP)	E 7 Mile	100		8.45
Arsenic (TSP)	Southfield	100		6.28
Arsenic (TSP)	River Rouge	100		9.56
Arsenic (TSP)	N. Delray	100		10.64
Arsenic (TSP)	Ypsilanti	100		3.89
Benzene	Allen Park	100		14.98
Benzene	Dearborn	99		19.25
Benzene	S. Delray	100		215.90
Benzene	Southfield	100		19.85
Benzene	River Rouge	100		21.37
Benzene	N. Delray	100		26.66
Cadmium (TSP)	Dearborn	100		1.01
Cadmium (TSP)	S. Delray	100		1.89
Cadmium (TSP)	River Rouge	100		1.03
Cadmium (TSP)	N. Delray	100		2.03
Carbon Tetrachloride	Allen Park	100		8.53
Carbon Tetrachloride	Dearborn	99		9.05
Carbon Tetrachloride	S. Delray	98		8.83
Carbon Tetrachloride	Southfield	100		8.88
Carbon Tetrachloride	River Rouge	100		8.27
Carbon Tetrachloride	N. Delray	96		7.80

Table 67: COPC (With Cancer Risk of 1 X 10^{-6} or Higher, or HQ of 1 or Higher) at Specific Sites and Percent Detection

CHEMICAL NAME	SITE NAME	% DETECT	HQ AVG/ITSL	RISK PER MILLION (AVG/IRSL)
Carbon Tetrachloride	Ypsilanti	97		7.93
Formaldehyde	Allen Park	98		26.48
Formaldehyde	Dearborn	98		35.81
Formaldehyde	S. Delray	100		37.46
Formaldehyde	E 7 Mile	100		35.14
Formaldehyde	Houghton Lake	100		25.28
Formaldehyde	Southfield	98		29.70
Formaldehyde	River Rouge	97		69.39
Formaldehyde	N. Delray	100		49.24
Formaldehyde	Ypsilanti	100		28.55
Manganese (TSP)	Dearborn	100	3.95	
Manganese (TSP)	S. Delray	100	5.47	
Manganese (TSP)	River Rouge	100	1.50	
Manganese (TSP)	N. Delray	100	1.86	
Methylene Chloride	Allen Park	100		401.35
Methylene Chloride	Dearborn	95		5.81
Methylene Chloride	S. Delray	97		1.27
Methylene Chloride	Southfield	100		31.47
Methylene Chloride	River Rouge	85		5.07
Methylene Chloride	N. Delray	73		3.33
Naphthalene	Allen Park	100		3.95
Naphthalene	Dearborn	100		7.89
Naphthalene	S. Delray	100	6.13	229.88
Naphthalene	E 7 Mile	100		4.88
Naphthalene	Southfield	100		3.65
Naphthalene	River Rouge	100		6.07
Naphthalene	N. Delray	100		15.74
Nickel (TSP)	S. Delray	100		1.17

USEPA guidance was followed by including in the summary statistics and the risk characterization only those site- and chemical-specific datasets which had at least 15% detectable levels (USEPA, 2004b, App. I). This approach introduces uncertainty into the estimated annual average levels, but is not believed to be particularly conservative or nonconservative. **Section 9.1.9**, in particular, discusses an unfortunately high rate of ND data for the group of seven carcinogenic PAHs. As discussed in that section, the available Detroit Pilot Project data did not suggest that the PAHs were major drivers of cancer risk relative to the other air toxics, although they may have collectively posed a cancer risk of more than 1 X 10^{-6} at S. Delray and possibly other sites. More recent monitoring is ongoing, with lower detection limits, providing data with all samples above the detection limits. The preliminary results thus far are quite limited, but they suggest that the carcinogenic PAHs may pose a risk of < 2 X 10^{-6}, predominantly due to B(a)P. Longer-term monitoring results (e.g., a full year) are needed before more conclusive characterizations can be made.

11.4 Toxicity Assessment

In the present DATI assessment, the health protective benchmark values were derived on the basis of the best toxicity data, which often involve animal studies or human occupational studies. In the absence of information indicating otherwise, the biologically relevant results of well-conducted animal studies are assumed to be applicable to humans. Relevant occupational exposure studies with health effects information are also assumed to be applicable to the human population, with recognition that the general population may include sensitive subgroups not represented in occupational scenarios. Health protective benchmark values vary on the basis of the key underlying studies, and hence they vary with regard to the amount of uncertainty in those benchmarks. Values based on human toxicity or epidemiology studies generally are regarded to have less uncertainty than those that are based on animal data. **Appendix E** presents the USEPA weight of evidence categories and narratives for the 12 substances identified as cancer risk drivers in the present study. The two primary air toxics of interest in the chronic noncancer risk assessment of this report have health benchmarks derived from human studies (manganese) or laboratory animal studies with findings extrapolated to humans (naphthalene), as further described elsewhere in this report.

There is uncertainty associated with the quantitative determination and application of health protective benchmark levels for use in the risk assessment. The health protective benchmarks used for chronic noncancer and cancer risk assessment were the MDEQ AQD's screening levels. These are generally very consistent with the best available information from USEPA (RfCs, RfDs, cancer UREs or slope factors). When those preferred USEPA benchmark sources were not available, AQD toxicologists performed literature searches and derived screening levels based on USEPA guidance or procedures in the AQD administrative rules, as applicable. With regard to the limitations and uncertainties of the USEPA values, which also apply to the AQD-derived values, the following excerpts from USEPA's IRIS website are particularly relevant:[23]

> "The primary qualitative and quantitative health hazard information in IRIS, the oral reference doses (RfDs), inhalation reference concentrations (RfCs), and carcinogenicity assessments, can serve as guides in evaluating potential health hazards and selecting a response to alleviate a potential risk to human health. . . The RfD and RfC can be used to estimate a level of environmental exposure at or below which no adverse effect is expected to occur. The RfD and RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious effects during a lifetime. RfDs and RfCs are based on an assumption of lifetime exposure and may not be appropriately applied to less-than lifetime exposure situations. The oral slope factor is an upper-bound estimate of the human cancer risk per milligram (mg) of agent/kg body weight/day. In general IRIS values cannot be validly used to accurately predict the incidence of human disease or the type of effects that chemical exposures have on humans. This is due to the numerous uncertainties involved in risk assessment, including those associated with extrapolations from animal data to humans and from experimental doses to lower environmental exposures. The organs affected and the type of adverse effect resulting from chemical exposure may differ between study animals and humans. In addition, many factors besides exposure to a chemical influence the occurrence and extent of human disease".

Noncancer benchmarks incorporate UFs to help ensure protectiveness in view of substantial areas of uncertainty in the key studies, when attempting to derive values that are protective of

[23] IRIS information is located at http://www.epa.gov/iris/limits.htm.

all people, including sensitive subgroups. In the development of RfCs, USEPA applies standard UFs for extrapolations of: 1) data on effects of average healthy humans to sensitive humans; 2) laboratory animal data to humans; 3) studies of subchronic to chronic duration; 4) a LOAEL to a NOAEL; and, 5) from an incomplete to complete database. The individual UFs are generally an order of magnitude, although they may be less depending on the available data on adjusting animal doses to the equivalent human doses, the severity of the LOAEL, or other considerations. The appropriate UFs are multiplied together to derive the composite UF. Each individual UF is generally conservative from the standpoint of the behavior of the average chemical (USEPA, 1994). As specified in the MDEQ-AQD administrative rules, AQD screening levels may be derived from even lesser databases than accepted by USEPA for RfC development, with the application of other adjustment factors. The application of the UFs is intended to err on the side of conservatism in recognition of the data limitations, and the resulting health benchmark is regarded as a protective level. However, it would not be appropriate to speculate in broad terms on the degree of conservatism that may or may not be present in health protective benchmarks, although chemical-specific assessments can address that question (see manganese and naphthalene evaluations in this report).

The noncancer hazard indices were calculated by compiling only the HQs of 0.1 or greater for each TOSHI. This was found to be a useful rule, because there were several substances exceeding this criterion, enabling a focusing of the risk assessment. Also, the HQs lower than 0.1 would not affect the aggregate TOSHI to an extent that the risk characterization or potential hazard reduction options would significantly change.

Cancer risk assessments derive unit risk factors (inhalation) which are derived via computer modeling from the observed dose-response data extrapolated to the target risk level of 1×10^{-6}. This generally involves an assumption of no threshold for carcinogenic effects, with linear extrapolation to low dose-responses. This assumption may under- or overestimate low-dose extrapolated risks for individual substances, if their true dose-responses are actually supralinear or sublinear (or, below a threshold), respectively.

The cancer unit risk factors derived from animal studies are generally based upon the statistical 95% UCL rather than the maximum likelihood estimate. USEPA qualifies the unit risk factors, acknowledging the conservatism of this step and the assumption of no threshold, by noting that the risk is unlikely to be higher than estimated, but is likely to be lower, and may be as low as zero at the extrapolated low doses for the target risk. Some carcinogens (e.g., benzene) have unit risk factors based on human studies and maximum likelihood estimates, which provide better certainty about the potency at exposures in the observed exposure range. Uncertainty still exists in the extrapolation of the findings to the low risk range (e.g., 1×10^{-6}).

As discussed in **Section 10**, the toxicity assessment for diesel emissions is problematic. Although the USEPA considers DPM to be carcinogenic to humans, they have not supported the use of a specific unit risk factor to quantify the cancer risk. Nevertheless, this issue is explored in **Section 10** utilizing the unit risk factor provided by the Cal EPA, in order to compare the potential cancer risk of DPM to the risk estimates in this report. This approach is heavily qualified, yet it is a useful approach for screening and scoping purposes in this report.

11.5 Risk Characterization

In the present DATI assessment, the scope of the air toxics monitoring was founded upon a list of “core compounds” which was determined by USEPA as being particularly important for urban air toxics risk assessment. Also included were monitoring data for other air toxics which were available via the same monitoring and analytical methods. Nevertheless, this approach may have excluded some air toxics which may contribute significantly to air toxics risk assessments

in some situations. For example, acrolein was not included due to a lack of acceptable monitoring/analysis methods. This substance has been a compound of concern in urban air based, in part, on USEPA's findings of the 1996 NATA initiative. Additionally, acrolein shares the short-term critical effect of irritancy with multiple air toxics as well as criteria pollutants. Therefore, the risk assessment scenario has uncertainties / variability with regard to omitted substances such as acrolein. The related issues of complex mixtures (model uncertainty) and the list of target compounds (decision rule uncertainty) are discussed elsewhere in this section. It may also be noted that the USEPA's DEARS is currently measuring acrolein at the Allen Park site with a relatively new passive method. USEPA and ERG are in the process of developing a canister-based method for acrolein sampling and analysis.

Many of the monitored substances do not have health protective benchmark levels for comparison and for risk characterization. While many of the substances did have particular health benchmarks which enabled the assessment of absolute and relative risk for certain endpoints (e.g., carcinogenicity, chronic or acute noncancer risk), many were missing one or more of the needed health benchmarks. This was most apparent in the assessment of acute noncancer risk, where health protective benchmarks were available for only about 20% of the chemicals monitored. The absence of the full spectrum of established high-confidence health benchmarks is a very substantial uncertainty in this and other risk assessments. The inability to characterize the risk associated with inhalation exposure to monitored substances obviously precludes the risk assessment for those specific substances and for the assessment of potential interactive effects of the complex mixtures. In general, this limitation results in the potential underestimation of risk. There may also be cases where limited available data are used to establish health benchmarks which are (unintentionally) overly conservative. In those cases, the health benchmarks may be set at higher values if a more complete dataset were available. In the present assessment, chronic noncancer health risks were initially screened by determining the HQ based on the AQD screening levels. Substances without HQs of at least 0.1 were screened out from further assessment including the additivity assessment. Also, two substances were eliminated from further consideration because their screening levels were based on extremely limited toxicity data which did not support quantitative risk assessment for this initiative.

The nature of the ambient air monitoring approach, rather than emissions modeling, does not enable the characterization of the population size or populated area that are represented by the risk estimates. Monitoring results are valid for the point of measurement and some surrounding area, but the size and shape of areas represented by the data are not known and cannot be reliably estimated. Elsewhere in this report, the differences in spatial variability in the monitoring data for the Detroit area (relative homogeneity or heterogeneity) are discussed. That information can help suggest if the chemical-specific data across monitoring sites may or may not represent the levels over broad areas. Emission inventories and dispersion modeling (e.g., USEPA's NATA studies) also provide information that is relevant for this issue.

The health protective benchmarks are generally developed and applied to individual substances, or in some cases to groups of substances that are structurally and toxicologically similar (e.g., carcinogenic PAHs). In contrast, the ambient air contains mixtures of air toxics, some of which may demonstrate interactive effects (additivity, synergism, or antagonism). Since there is a lack of toxicity information for the complex mixtures in the ambient air in Detroit, the standard risk assessment approach is to evaluate each substance individually, and also to assume that there is a potential joint toxic action of additivity for the substances which appear to have the same mode of action. This approach constitutes an area of uncertainty. However, it is believed to be the most biologically plausible approach to account for potential interactive effects, without engendering over- or under-estimation of risks. The appropriateness of the

additivity model may be greatest when dealing with exposures to substances which approach or exceed their effect thresholds individually. The approach may be conservative when the levels of individual substances are well below their effect thresholds. Another uncertainty is the potential interaction that the air toxics may have with criteria pollutants, particularly ozone and $PM_{2.5}$, which are known to be elevated in the Detroit area. There is a potential for interactive effects on the respiratory system for those criteria pollutants and some air toxics. Some of the air toxics evaluated in the present study are components of PM, therefore those substances are included in the risk assessment. Otherwise, the criteria pollutants are not accounted for in the present study. According to the USEPA (2004b, Chapter 4, 4.2.3; p. 4-6), "In general, the criteria pollutants ozone, nitrogen dioxide, sulfur dioxide, (and) carbon monoxide are not usually considered in air toxics risk assessments". This approach is an area of uncertainty which is nonconservative, particularly for the assessment of potential effects on the respiratory system.

As discussed in **Section 6.3** and **9.1.5**, total chromium was monitored at all sites, while Cr^{+6} was monitored at only four Detroit-area sites. The risk assessment required an assumption of the fraction of Cr^{+6} in total chromium, for the sites lacking the Cr^{+6} measurements. The chosen approach was to use the site-specific data (excluding NDs) and determine the ratio of hexavalent to total chromium for each monitoring day and site. Then, for each site, the annual average was estimated as the arithmetic average of the daily ratios. The highest site-specific average ratio was 0.024 (2.4%) at the Southfield site. Then, the assumed Cr^{+6} level for exposure and risk assessment at non-monitored sites was derived as that percentage (2.4%) of total chromium which was hexavalent. An alternative approach would be to develop a cumulative distribution frequency of all the site- and day-specific ratios, and utilize a specific percentile of that distribution. That exercise found that the chosen approach (2.4% assumption) was approximately the 80th percentile of the distribution. For comparison, the 95th percentile of the distribution was 3.7%. Either of these approaches may or may not be conservative, depending on the potential for local emission sources of Cr^{+6} which may influence this fraction at the sites lacking measurements.

Another issue which should be addressed here is the precision (number of significant figures) with which the results of the risk assessment should be reported. USEPA (2004f) noted that, "In a final risk description, all cancer risks and non-cancer hazard indices should be presented with one significant figure only". Although that practice is appropriate according to the principles of mathematics regarding the supportable number of significant figures, the present report does not consistently follow that recommendation. The reasons are that the retention of more significant figures in the results enables other risk assessors to check the accuracy of the risk calculations, and that the audience for the findings (risk managers and the public) typically do not object to the retention of multiple significant figures, albeit not mathematically justified. In future risk communications to public audiences, fewer significant figures (1 or 2) will generally be presented.

The presentation of cancer risks per substance utilized a rule that "cancer risk drivers" for compiling total cancer risk had a criterion of 1×10^{-6} or greater at any site. This was found to be an appropriate rule for focusing upon the most significant findings, because there were 12 substances (a manageable number) which were "risk drivers" and the individual and total cancer risks were well above this risk level.

12.0 COMPARISON OF AIR TOXICS HEALTH RISKS IN DETROIT TO OTHER LOCATIONS

12.1 Approaches to Comparing Ambient Air Toxics Risks from Different Studies and Locations

Although **Section 13** of this report includes a comparison of the Detroit Pilot Project's monitoring results to the results in other cities, a comparison of *risks* among the Pilot Project cities has not been conducted by USEPA or other parties and was not within the scope of the present initiative. When considering the possible ways to compare risk estimates outside the realm of the DATI report, consistency in methodology is a critical factor. Consistency is often lacking when attempting to make such comparisons. This is a concern because data utilized for risk assessment may be "apples and oranges" with regard to the time period, the geographic area of study, the substances included, the emission inventory completeness and accuracy, or the monitoring methods, locations, and analytical detection limits. It is recognized that some of these inconsistencies, such as different time periods or geographic areas, may actually be desirable characteristics for evaluating the differences in risks associated with those factors. Additionally, risk assessments may have significant differences in their methodologies, which confound attempts to compare risk estimates across studies. Examples include the treatment of ND data, the scope and basis for emission estimates, the exposure assumptions, and the health protective benchmarks and criteria.

The characterization of the health risks of ambient air toxics could potentially be based upon emission estimates and modeled cumulative ambient air impacts, or by monitoring ambient air as was done in the present report. Each approach has a number of strengths and limitations, with regard to the ability to support relevant and useful exposure and risk estimates. For example, a monitoring approach can provide information on the cumulative levels of air toxics at specific locations and time periods, but it does not define area-wide exposure levels and cannot support population risk estimates, and it may be hindered by technical infeasibility (e.g., acrolein) or prohibitive costs (e.g., dioxins). On the other hand, an approach based on emission inventories and modeling may avoid some of the limitations of monitoring studies, but it can itself be limited by the omission of significant sources, incomplete or inaccurate emission inventories, failure to accurately account for atmospheric transformation processes, and potentially a lack of characterization of levels at a local scale. The USEPA (2004b) Air Toxics Risk Assessment Reference Library (Section 10-3) provides further details and comparisons.

Some of the limitations of monitoring studies can be addressed to some extent by comparing the findings with estimated ambient air levels derived from emission inventories and modeling of ambient air impacts. However, it should not be anticipated that ambient air monitoring data and modeling estimates would be in close agreement, for several reasons. For example, there may be differences in the geographic scale: such as monitoring data may reflect relatively localized emission and meteorological conditions, while modeling estimates such as NATA may portray these conditions over larger areas. Temporal differences may also occur: monitoring results are dependent on meteorological conditions and emissions over a relatively short time scale (e.g., 24 hours), while emission inventories and dispersion modeling may characterize impacts over longer time periods.

To illustrate the different air toxics risk assessment methodologies, and the resulting limitations in attempting to compare the findings, two studies will be briefly described. USEPA (1990a) integrated the findings of several air toxics monitoring studies (including also some modeling of impacts) which were conducted nationwide or in specific geographic areas. The study included

up to 90 substances. They found that 12 substances accounted for over 90% of the total inhalation cancer risk. The major contributors, and their contribution to the total cancer risk, were:

- products of incomplete combustion (using B(a)P as the surrogate; 35%);
- 1,3-butadiene (12%);
- Cr^{+6} (9%);
- benzene (8%);
- formaldehyde (5%); and
- chloroform (5%).

The national average lifetime total cancer risk estimate was 490 to 770 X 10^{-6}. For urban areas generally, the lifetime total cancer risk estimate was in the range of 10 to 1000 X 10^{-6}. The authors noted that,

> "The numerical estimates presented in this report should be viewed only as rough indications of the potential for cancer risk caused by a limited group of pollutants found in the ambient air. Many of the risks cited in this report are almost certainly inaccurate in an absolute sense. The best use of the risk estimates is in describing the broad nature of cancer risk posed by these toxic air pollutants and by making relative comparisons of risks between pollutants and sources."

A second noteworthy air toxics risk study is USEPA Region 5 (1990b), which involved an air toxics emissions inventory and modeled dispersion and deposition for the Southeast Michigan / Windsor-Sarnia area. The study included 72 air toxics, including 27 carcinogens. The highest cancer risk drivers, and their percent contribution to the total lifetime inhalation cancer risk estimate for the Canadian grid cell, were as follows:

- formaldehyde (33.3%);
- coke oven emissions (23.1%);
- 1,3-butadiene (16.4%);
- carbon tetrachloride (12.8%);
- Cr^{+6} (3.8%);
- polycyclic organic matter (3.3%);
- dioxin (3.2%); and
- all others (4.1%).

The lifetime total inhalation cancer risk estimate was 90 X 10^{-6}, with a maximum for the study area at 120 X 10^{-6}. The authors noted that,

> "The risk estimates presented in this report should be regarded as only rough approximations of total cancer cases and individual lifetime risks, and are best used in a relative sense. Estimates for individual pollutants are highly uncertain and should be used with particular caution."

These two examples help demonstrate the varying types of air toxics risk studies which exist, some of the fundamental differences between their design and their findings, and the precautionary notes which accompany them.

For the present report, the approach that is considered to be the best available for comparing air toxics health risks in Detroit to other locations, with a consistency in methodology and scope, is

the USEPA's (1996) NATA. The USEPA has developed a National Emissions Inventory, which they are utilizing to develop the NATA for 1999 emissions. The complete NATA results for 1999 (including modeled ambient air impacts, exposure levels and risk estimates) are not yet available. The USEPA's (1996) NATA results are available, and they are utilized in this section in order to provide a "level playing field" comparison of risks between locations. The USEPA's NATA could be very useful for this purpose, because it involved the evaluation of all counties in the contiguous 48 states, Puerto Rico and the Virgin Islands, with a consistent methodology. This is intended to lend a broader perspective to the significance of air toxics health risks in Wayne County (including Detroit), in Michigan, and nationwide. It should be emphasized that the NATA risk comparisons are appropriate at the countywide scale (i.e., countywide averages), but not at the level of census tracts. Local areas (e.g., census tracts) will have varying estimates of ambient air levels and associated health risk estimates. The NATA (ibid) study was not designed to provide accuracy at the level of census tracts. Also, this report does not attempt to compare the monitoring-based risk estimates to the modeling-based NATA findings, to validate one versus the other. Although beyond the scope of the present study, a potential future comparison between the DATI and the 1999 NATA results may be an insightful endeavor, to identify significant "gaps" and limitations inherent in each approach.

12.2 NATA (1996) Comparison of Modeled Cancer Risk Estimates for Wayne County and Other Counties Nationally

For NATA, USEPA (1996) included 32 of the 33 "Urban Air Toxics HAPs" considered by USEPA to pose the greatest concern for health impacts in urban areas. Only dioxin was excluded, due to lack of consensus on the emissions inventory. Additionally, USEPA included DPM, but quantitative cancer risk assessment was not considered feasible. These substances were selected based on a number of factors, included toxicity-weighted emissions, monitoring data, past air quality modeling analyses, and a review of the existing literature. Of the 12 substances which posed a cancer risk of at least 1 X 10^{-6} in the DATI risk assessment, 10 were included in the 33 substances evaluated in NATA (USEPA, 1996). The excluded substances were 1,4-dichlorobenzene and naphthalene. In DATI, 1,4-dichlorobenzene posed a risk of 25 X 10^{-6} at the N. Delray site and 1-2 X 10^{-6} at seven other sites. Naphthalene posed a risk of approximately 4 to 230 X 10^{-6} at the seven Detroit-area sites in DATI.

For the 1996 NATA, USEPA compiled a list of 13 substances and their estimated nationwide "background" concentrations (see **Table 68**). The term "background" in this context refers to outdoor concentrations attributable to long-range transport, resuspension of historical emissions, and nonanthropogenic sources. These background concentrations are based on monitored values identified previously by USEPA in the Cumulative Exposure Project. USEPA determined the background levels and cancer UREs for those 13 substances. Combined, they posed a lifetime cancer risk estimate of 25 X 10^{-6}. This means that the levels of air toxics in the ambient air throughout the contiguous U.S. are associated with an estimated lifetime cancer risk level of at least 25 X 10^{-6}.

Table 68: NATA 1996 Background Concentration Estimates

SUBSTANCE	BACKGROUND CONCENTRATION (µg/m^3)
Benzene	0.48
Carbon tetrachloride	0.88
Chloroform	0.083
Ethylene dibromide	0.0077
Ethylene dichloride	0.061
Formaldehyde	0.25

Table 68: NATA 1996 Background Concentration Estimates

SUBSTANCE	BACKGROUND CONCENTRATION (µg/m^3)
Hexachlorobenzene	0.000093
Mercury compounds	0.0015
Methylene chloride	0.15
Polychlorinated biphenyls	0.00038
Perchloroethylene (Tetrachloroethylene)	0.14
Trichloroethylene	0.081

The comparison of cancer risks posed by ambient air levels in all counties in the NATA (1996) study is summarized in **Table 69** and **70**. As noted above, all counties evaluated in NATA were estimated to have a risk level of at least 25 X 10^{-6} (due to the background air toxics estimated in the study), while the risk ranged as high as 240 X 10^{-6} (Stoddard County, Missouri). In Michigan, Wayne County was the highest at 91 X 10^{-6}. Twenty counties in the U.S. had county-average risk levels greater than 100 X 10^{-6}. For more than 20 million people nationwide, the air toxics levels exceeded a risk level of 100 X 10^{-6} (USEPA, 1996). The results of the 1996 NATA (ibid) assessment indicate that the cancer risk levels in Michigan urbanized counties are fairly typical of the national scene. Twenty six counties in the U.S. were estimated to have countywide cancer risk estimates higher than Wayne County.

Table 69: Estimated Lifetime Cancer Risks from Inhalation Exposure to Air Toxics for Michigan and Nationwide Averages (USEPA NATA [1996] Study)

MICHIGAN COUNTIES SUBSET:	ESTIMATED AVERAGE CANCER RISK (LIFETIME) IN ONE MILLION	PREDOMINANT URBAN AREA
Wayne County	91	Detroit
Macomb County	83	Northeast Detroit
Oakland County	74	Northwest Detroit
Kent County	57	Grand Rapids
Washtenaw County	54	Ann Arbor
Muskegon County	53	Muskegon
Genesee County	48	Flint
Kalamazoo County	44	Kalamazoo
Ingham County	41	Lansing
Saginaw County	39	Saginaw
Midland County	32	Midland
MICHIGAN OVERALL:		
Michigan (all counties)	59	
Michigan (urban counties)	66	
Michigan (rural counties)	32	
NATIONAL:		
U.S. (all)	55	
U.S. (all urban counties)	61	
U.S. (all rural counties)	31	

Table 70: Rank Order of the 27 Highest County Average Lifetime Cancer Risk Estimates (USEPA NATA [1996] Study)

DESCENDING RANK BY RISK LEVEL	COUNTY	ESTIMATED AVG CANCER RISK (LIFETIME) IN ONE MILLION	STATE	PREDOMINANT URBAN AREA
1.	Stoddard County	240	Missouri	-
2.	New York County	222	New York	NYC
3.	Adams County	197	Indiana	-
4.	Vermilion Parish	161	Louisiana	-
5.	Bronx County	151	New York	NYC
6.	Kings County	147	New York	Brooklyn
7.	Queens County	141	New York	Brooklyn
8.	Berkeley County	140	West Virginia	-
9.	Philadelphia County	134	Pennsylvania	Philadelphia
10.	Cabell County	130	West Virginia	-
11.	Terrebonne Parish	129	Louisiana	-
12.	Hudson County	128	New Jersey	Newark
13.	Hennepin County	111	Minnesota	Minneapolis
14.	Ramsey County	110	Minnesota	Minneapolis
15.	Otoe County	109	Nebraska	
16.	Marion County	108	Indiana	Indianapolis
17.	Etowah County	107	Alabama	-
18.	San Francisco County	105	California	San Francisco
19.	Orange County	105	California	Santa Ana, Irvine
20.	Camden County	105	New Jersey	-
21.	Washington County	100	Arkansas	
22.	Los Angeles County	98	California	Los Angeles
23.	Baltimore city	95	Maryland	Baltimore
24.	St. Louis city	94	Missouri	St. Louis
25.	Milwaukee County	93	Wisconsin	Milwaukee
26.	Cobb County	91.1	Georgia	
27.	**Wayne County**	**90.7**	**Michigan**	**Detroit**

12.3 NATA (1996) Comparison of Modeled Noncancer Hazard Estimates for Wayne County and Other Counties Nationally

The 1996 NATA chronic noncancer hazard results were presented by USEPA in the form of spreadsheets showing Total HIs for each county, respiratory irritancy TOSHIs for 8 chemicals for each county, and chemical-specific HQs for each census tract. Spreadsheets were not provided for countywide average HQs for each chemical. Maps were provided showing countywide HQs for each chemical, but the presentation shows color-coded ranges of HQ values for each county, not specific HQ values for each county.

In the following, some of the most relevant noncancer hazard findings of NATA (USEPA, 1996) are briefly presented. This is followed by some specific information regarding acrolein (which posed the greatest noncancer hazard estimates in NATA, but which was not included in the Pilot Project or DATI), manganese and naphthalene (the DATI noncancer hazard COPCs), and DPM (which is of special interest in this DATI report).

The USEPA found that only acrolein posed a national noncancer hazard (defined as compounds with HQ > 1 for more than 25 million persons). This was based on acrolein's ability to cause respiratory irritation. Likewise, the aggregate respiratory HI (TOSHI) for 8 respiratory

irritants was dominated by acrolein. The respiratory TOSHI exceeded 1.0 for nearly the entire U.S. population, and exceeded 10 for more than 20 million people. Regional or urban scale hazard drivers (defined as compounds with HQ > 1 for more than 10,000 individuals) were identified as acetaldehyde, arsenic, 1,3-butadiene, formaldehyde, and manganese. It was noted that cadmium, chromium, and lead would also have been included if exposure adjustments had been made to reflect average model-to-monitor comparisons.

Presented in **Table 71** are the results of aggregated chronic HIs for the U.S. and Michigan based on examination of 27 HAPs. These estimates should be considered with caution since they combine HQs with multiple toxic endpoints that cannot be assumed to be additive.

Table 71: U.S. and Michigan Aggregated Chronic Hazard Indexes
(based on examination of 27 HAPs)

ESTIMATED CHRONIC HAZARD INDEX									
STATE	COUNTY	PERCENTILE DISTRIBUTION OF RISK ACROSS CENSUS TRACTS							
		5th	10th	25th	Median	Average	75th	90th	95th
National	All	3.86E-01	9.31E-01	1.92E+00	3.74E+00	5.36E+00	6.50E+00	1.09E+01	1.54E+01
National	All Urban Counties	7.35E-01	1.41E+00	2.72E+00	4.62E+00	6.11E+00	7.39E+00	1.19E+01	1.73E+01
National	All Rural Counties	1.14E-01	3.43E-01	9.36E-01	1.58E+00	1.98E+00	2.35E+00	3.38E+00	4.24E+00
Michigan	State Total	8.86E-01	1.16E+00	1.91E+00	3.59E+00	4.21E+00	6.64E+00	7.60E+00	8.07E+00
Michigan	State Urban Counties	1.51E+00	1.88E+00	2.72E+00	5.35E+00	4.87E+00	6.97E+00	7.81E+00	8.22E+00
Michigan	State Rural Counties	6.56E-01	7.46E-01	9.77E-01	1.43E+00	1.61E+00	1.99E+00	2.54E+00	2.86E+00
Michigan	Wayne County	5.64E+00	6.31E+00	6.81E+00	7.36E+00	7.40E+00	7.91E+00	8.49E+00	9.25E+00

Table 72 provides excerpts from the NATA TOSHI for eight chemicals which are respiratory irritants: acetaldehyde, acrolein, acrylonitrile, arsenic, 1,3-dichloropropene, ethylene dibromide, formaldehyde, and trichloroethylene (USEPA, 1996). These HQs are appropriately combined since they share the same critical effect. The TOSHI for Wayne County ranked as the 251st highest among counties in the contiguous U.S.

Table 72: NATA (1996) Respiratory Irritancy TOSHI Values for U.S. Counties

RELATIVE RANK OF COUNTY	STATE (COUNTY)	RESPIRATORY HI
1	California (Los Angeles)	167
2	Texas (Harris)	138
3	New York (New York)	136
251	Michigan (Wayne)	30

Acrolein: Estimated chronic HQs for acrolein based on NATA (ibid) ranged from 0.0 to 160 across all census tracts in the contiguous U.S. The highest census tract in Wayne County ranked 258th out of 61,641 census tracts nationwide with an HQ estimate of 29.7. The highest HQs estimated for acrolein were in census tracts in Los Angeles County, California (HQ=160), Harris County, Texas (HQ=136) and New York County, New York (HQ=131). Acrolein was the major non-cancer hazard driver accounting for nearly all of the respiratory irritancy TOSHI value. The NATA (ibid) included a map indicating that Wayne County had an acrolein HQ in the 3-10 range; other counties in Michigan had acrolein HQs in the ranges of 0.3-1; 1-3; or, 3-10. County-average exposure concentrations were provided, but as previously noted, county-

specific HQ values were not provided in spreadsheets. **Table 73** utilizes the USEPA RfC of 0.02 µg/m^3 to derive HQs for acrolein from the reported average exposure concentrations.

Table 73: Acrolein HQs Based on NATA (1996)

AREA	AVERAGE EXPOSURE CONC. (µg/m^3)	HQ
National – all counties	1.03E-01	5
National – all urban counties	1.18E-01	6
National – all rural counties	3.81E-02	2
Michigan – all counties	8.07E-02	4
Michigan – all urban counties	9.33E-02	5
Michigan – all rural counties	3.09E-02	2
Wayne County	1.42E-01	7

However, it should be noted that the NATA (ibid) estimated acrolein levels are substantially lower than preliminary Detroit ambient air monitoring results (awaiting final validation). From July 15 - August 31, 2004, the USEPA DEARS project measured acrolein levels averaging approximately 2 µg/m^3 at Allen Park and other ambient air locations in Detroit (Williams, 2005). Based on the USEPA RfC of 0.02 µg/m^3, these preliminary monitoring data are associated with a HQ of 100. This suggests that acrolein may be a significant noncancer COPC which was not included in the DATI risk assessment due to technical infeasibility during the 2001-2002 Pilot Project.

Manganese: Estimated chronic HQs for manganese compounds based on NATA (USEPA, 1996) ranged from 0.0 to 11.4 across all census tracts in NATA. The highest HQ in any census tract in Wayne County, Michigan ranked 825th out of 61,641 census tracts nationwide with an HQ estimate of 0.282. This HQ is considerably lower than the individual HQs estimated in DATI for S. Delray, Dearborn, N. Delray and River Rouge. According to NATA estimates, several Michigan census tracts ranked higher than the highest census tract in Wayne County for manganese, with HQs ranging from 0.285 to 2.0. Those census tracts were within Menominee, Calhoun, Saginaw, Macomb, Muskegon, Dickinson, and Kent Counties.

At the county level, the USEPA NATA (ibid) included a map indicating that Wayne County had a manganese HQ in the 0.03-0.1 range; other counties in Michigan had manganese HQs in the ranges of 0-0.03; 0.03-0.1; or, 0.1-0.3. In Michigan, only Saginaw and Menominee Counties were shown to have countywide average manganese HQs in the range of 0.1-0.3. County-average exposure concentrations were provided, but as previously noted, county-specific HQ values were not provided in spreadsheets. **Table 74** utilizes the USEPA RfC of 0.05 µg/m^3 to derive HQs for manganese from the reported average exposure concentrations.

Table 74: Manganese HQs Based on NATA (1996)

AREA	AVERAGE EXPOSURE CONC. (µg/m^3)	HQ
National – all counties	2.24E-03	0.05
National – all urban counties	2.41E-03	0.05
National – all rural counties	1.46E-03	0.03
Michigan – all counties	1.76E-03	0.04
Michigan – all urban counties	1.94E-03	0.04
Michigan – all rural counties	1.07E-03	0.02
Wayne County	2.24E-03	0.04

DPM: In NATA (ibid), annual exposure concentrations were estimated but no risk estimates were calculated for either cancer or non-cancer endpoints. However, USEPA (ibid) addressed the potential hazard from diesel qualitatively, stating that there is, "…significant potential for

non-cancer health effects as well [as cancer effects], based on the contribution of DPM to ambient levels of fine particles. Exposure to fine particles contributes to harmful respiratory and cardiovascular effects and to premature mortality." **Table 75** utilizes the USEPA RfC of 5 µg/m^3 to derive HQs for DPM from the reported average exposure concentrations.

Table 75: DPM HQs Based on NATA (1996)

AREA	AVERAGE EXPOSURE CONC. (µg/m^3)	HQ
National – all counties	1.44	0.3
National – all urban counties	1.64	0.3
National – all rural counties	0.55	0.1
Michigan – all counties	1.12	0.2
Michigan – all urban counties	1.26	0.2
Michigan – all rural counties	0.54	0.1
Wayne County	1.62	0.3

Naphthalene: Naphthalene was a noncancer COPC in the DATI assessment, but it cannot be compared to other areas via NATA because it was not one of the 33 substances included in the NATA (USEPA, 1996).

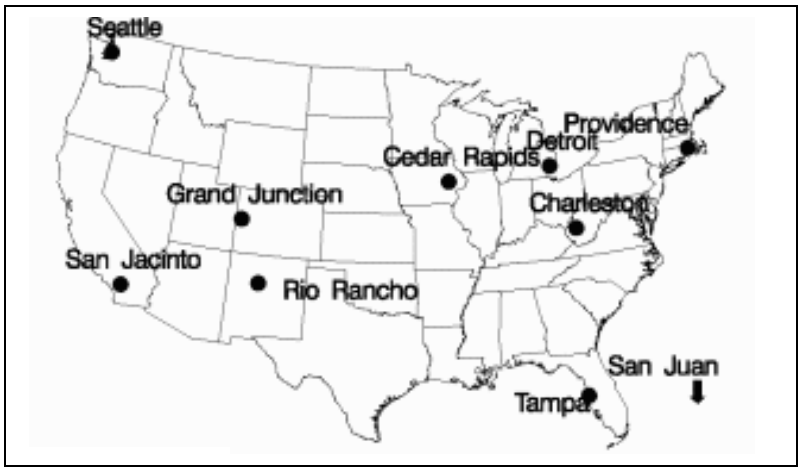
13.0 COMPARISON TO THE FINDINGS OF OTHER MONITORING INITIATIVES

13.1 Comparisons of COPCs & Cancer Risk Driver Compounds Monitored During the Detroit Pilot Project with Levels Detected in Other Pilot Program Cities

Introduction

In an effort to design a national air toxics monitoring strategy, USEPA provided funding for several pilot projects that included ten U.S. cities and one U.S. Territory. Pilot projects were conducted in four urban areas and six small city/rural locations during 2001 through 2002. The exact start up and shut down dates varied by city and were influenced by equipment purchases, contracts with the national laboratory, site set up and a host of other factors. The locations are shown in **Figure 26**.

Figure 26: Map of Ten Cities in the Monitoring Pilot Project



Efforts were made among the states, local agencies, USEPA, and laboratories conducting the analysis to standardize sample collection and analytical procedures as much as possible.

The resulting data was analyzed, under an USEPA contract, by Battelle. A series of reports and graphs were generated to aid in the development of a national air toxics monitoring network. Much of the data discussed in this report were obtained from the Battelle data set. These numbers may be slightly different than those calculated by the AQD.

Scope Of Analysis

The Detroit Pilot Project involves the development of a risk characterization based on the Detroit area's monitored air toxics concentrations and other related information. The DATI risk assessment report is the "first of its kind" in Michigan.

Preliminary analysis has indicated that there are 12 carcinogens that pose a risk of at least 1×10^{-6} at one or more of the Detroit monitoring sites. Also, manganese and naphthalene were identified as non-carcinogens of interest for comparison to levels at other sites. These compounds are identified in **Table 76**:

Table 76: Cancer Risk Drivers & COPCs

CARCINOGENS			NON-CARCINOGENS
1,3-Butadiene	Arsenic (TSP)	Formaldehyde	Manganese (TSP) Naphthalene
1,4-Dichlorobenzene	Benzene	Methylene chloride	
Acetaldehyde	Cadmium (TSP)	Naphthalene	
Acrylonitrile	Carbon tetrachloride	Nickel (TSP)	

A brief summary of how Detroit compares with other pilot cities in the national program is presented in **Table 77**. The MDEQ is developing a full technical report that will provide detailed tables and figures of all pilot cities and select pilot sites as well as a more detailed comparative analysis (Heindorf, 2005).

Table 77: Summary Of 12 Cancer Risk Drivers & Manganese - Comparisons Between Monitored Concentrations From The Detroit Pilot Project and Levels Detected In Other Pilot Cities*

CHEMICALS	ANNUAL AVERAGE CONCENTRATION DISTRIBUTION (µg/m^3)									
	Cedar Rapids, IOWA	Charleston, South Carolina	Detroit, Michigan	Grand Junction, Colorado	Providence, Rhode Island	Rio Rancho, New Mexico	San Jacinto, California	San Juan, Puerto Rico	Seattle, Washington	Tampa Bay, Florida
1,3-Butadiene	0.11000	N/A	0.13000	0.20000	0.12000	**0.29000**	0.12000	0.22000	0.11000	0.13000
1,4-Dichlorobenzene	N/A	N/A	0.42000	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Acetaldehyde	**2.86000**	N/A	1.82000	2.48000	1.85000	2.31000	2.12000	2.11000	1.25000	1.79000
Acrylonitrile	0.26000	N/A	0.28000	0.27000	0.03000	N/A	0.16000	0.23000	N/A	**0.38000**
Arsenic (TSP)	N/A	N/A	0.00203	0.00148	N/A	0.00074	0.00200	N/A	0.00106	**0.00215**
Benzene	1.07000	N/A	**3.48000**	2.34000	0.90000	1.22000	0.85000	1.56000	1.33000	0.97000
Cadmium (TSP)	N/A	N/A	0.00075	0.00046	0.00029	0.00019	**0.07000**	N/A	0.00013	0.00028
Carbon tetrachloride	0.56000	N/A	0.62000	0.51000	0.61000	0.63000	**0.69000**	0.52000	0.63000	0.62000
Formaldehyde	2.34000	N/A	2.87000	**6.57000**	3.52000	2.84000	2.05000	3.22000	1.32000	4.72000
Manganese (TSP)	N/A	N/A	**0.10000**	0.04000	0.00851	0.03000	0.02000	N/A	0.00664	0.00518
Methylene Chloride	0.78000	N/A	**49.17000**	0.60000	0.38000	1.17000	0.59000	7.29000	1.75000	0.46000
Naphthalene	N/A	N/A	4.23000	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Nickel (TSP)	N/A	N/A	0.00313	0.00273	**0.00572**	0.00167	0.01000	N/A	0.00142	0.00427

***Values in bold represent the highest annual average of all pilot cities for which data were available.**

For three of the compounds (benzene, methylene chloride, and manganese) Detroit had the highest annual average of all pilot cities for which data was available. In the case of benzene and methylene chloride, Detroit's annual average is substantially skewed by unusually high spikes at S. Delray and Allen Park, respectively. Although Detroit's annual average for acrylonitrile is similar to other pilot city averages, acrylonitrile will be highlighted here since it was identified as a COPC *within* the Detroit Pilot Project.

There are several important differences between the methods of collection and detection from pilot city to pilot city and even from site to site within a pilot city. For some compounds (e.g., acrylonitrile) the percent of samples that were NDs was very high. The convention of substituting the MDL/2 value for an actual monitored concentration skews the annual average. In addition, different laboratories may have different MDLs for each compound which also impedes direct city-to-city and site-to-site comparisons. The frequency of sampling for each compound may also differ between pilot cities and pilot sites which further impacts the comparability of the annual averages. These differences make it quite difficult to make meaningful comparisons between cities and sites although some general trends are discernible and will be discussed below.

Detroit's annual average of 0.28 $\mu g/m^3$ for acrylonitrile fell within the range of other pilot city averages (between 0.23 and 0.29 $\mu g/m^3$). Tampa Bay had the highest average for this compound (0.38 $\mu g/m^3$) and since the detection level was greater than 97%, it is likely that this number is valid. The Detroit average is skewed by the higher concentrations of acrylonitrile detected at one site, N. Delray. In addition, the ND level for Detroit is quite high (about 98%). Comparing Detroit Pilot Project sites with Tampa Bay pilot sites indicates that acrylonitrile levels in Tampa Bay are somewhat uniform from site to site while levels in Detroit show greater variation between sites. This variability in acrylonitrile between Detroit sites is somewhat spurious, however, since N. Delray was the only site at which acrylonitrile was consistently detected (in about 50% of the samples). It is difficult to draw any conclusions for acrylonitrile based on the shortcomings of the data but Detroit seems to be on par with other pilot cities in the national program. The mean site average for N. Delray within Detroit, however, is about twice that of all mean site averages within Tampa (See **Table 78**).

Detroit's annual average of 3.48 $\mu g/m^3$ for benzene was the highest of all the pilot cities. Again, this average is skewed by three unusually high spikes at one site, S. Delray. Benzene's detection level at all of the pilot cities was over 95% which makes comparison between cities more reliable. Sometimes it is useful to remove outliers from the dataset when making global comparisons between two means. The range of means of Detroit Pilot Project sites including the highest (S. Delray) and lowest (Allen Park) site is 1.50 to 21.94 $\mu g/m^3$. The range of means within the second highest pilot city for benzene, Grand Junction, is much more homogeneous (2.14 to 2.77 $\mu g/m^3$) although it should be noted that the Grand Junction pilot project involved only two sites compared to Detroit's eight. If the high and low sites within Detroit are excluded, the range of Detroit sites means is between 1.89 and 2.64 $\mu g/m^3$. If S. Delray and Allen Park are removed from the analysis, Detroit's adjusted annual average is 1.9 $\mu g/m^3$. This adjusted average falls within the range of other pilot city averages (0.85 to 2.34 $\mu g/m^3$) and is likely more representative of benzene levels at Detroit monitoring sites overall. This in no way, however, negates the importance of the site-specific findings at S. Delray which were discussed in previous sections. Although mobile sources generally account for the majority of ambient air concentrations of benzene, the S. Delray site is not considered a high traffic area. There are two major industrial point sources of benzene near S. Delray and one of those has since ceased production. The general conclusion is that benzene levels in the Detroit area are comparable to other pilot cities although there seems to be a local source impact at S. Delray which warrants

further investigation. The S. Delray site mean is approximately one order of magnitude higher than other pilot site means within and outside Detroit (See **Table 79**).

Detroit's annual average of 49.17 µg/m^3 for methylene chloride was by far the highest of all the pilot cities, once again due to some abnormally high concentration spikes at one site, Allen Park. In contrast, methylene chloride levels at sites within Providence Rhode Island, Seattle Washington, and Tampa Bay Florida were quite uniform. The percent ND for all pilot cities was low (0-20%) which makes for more meaningful comparisons between cities. However, to compare Detroit's overall average to other pilot cities, it is useful to consider Detroit's mean with and without "outliers." The range of site means within Detroit including Allen Park (highest) and S. Delray (lowest) is 2.57 to 802.31 µg/m^3. The range of site means excluding outliers is 3.08 to 62.92 µg/m^3. Excluding Allen Park and S. Delray from the analysis gives an adjusted mean of 16.20 µg/m^3 for Detroit. The range of means for all other pilot cities in the national program is 0.38 to 7.29 µg/m^3. Even without Allen Park, Detroit's average is still considerably higher than the other pilot cities due largely to higher means at Southfield, Dearborn, and River Rouge (see **Table 80**). Subsequent monitoring at Allen Park has not detected similar spikes of methylene chloride as were seen during the 2001-2002 study period. The source(s) of these unusual spikes at Allen Park during the study has not been identified. Clearly though, there are still site-specific impacts with regard to methylene chloride at Southfield, Dearborn, and River Rouge, all of which consistently showed higher levels as compared to other Detroit sites and other sites within selected pilot cities (See **Table 80**).

Detroit's annual average for manganese was the highest of all pilot cities at 0.10 µg/m^3. Higher levels at S. Delray and Dearborn account for this higher average. The national average is 0.04 µg/m^3. Detroit showed greater variability of manganese from site to site as compared to other cities with higher levels centered about central Detroit and declining further from the S. Delray and Dearborn areas. Detections levels of manganese were good for all pilot cities (see **Table 81**). If S. Delray is excluded. the adjusted mean for Detroit is 0.08 µg/m^3 which is still above the national average and remains the highest pilot city mean (range = 0.005 to 0.04 µg/m^3). Site means at S. Delray and Dearborn are one to two orders of magnitude higher than other pilot cities' site means. High background due to historical emissions of manganese in Detroit may account for a portion of the higher levels observed.

Table 78: Summary of Acrylonitrile Pilot Data (Concentration Distribution) in Selected Pilot Cities (Detroit, 1 small and 1 urban) by Site*

POLLUTANT PILOT CITY	SITE	TOTAL OBS[1]	PERCENT ND	CONCENTRATION DISTRIBUTION (µg/m^3)						
				Mean	Std Dev	Min	Q1	Median	Q3	Max
Acrylonitrile Grand Junction, Colorado	Mesa Co Health Dept	72	100.00	0.23	0.00	0.23	0.23	0.23	0.23	0.23
	Mesa Co Traffic Services	148	91.22	0.29	0.39	0.15	0.23	0.23	0.23	3.44
Acrylonitrile Tampa Bay, Florida	Azalea Park	60	0.00	0.44	0.23	0.03	0.27	0.44	0.56	1.08
	Dunedin	177	1.13	0.39	0.27	0.02	0.20	0.35	0.50	1.78
	East Lake	57	3.51	0.32	0.23	0.02	0.17	0.26	0.43	1.19
	Gandy	53	0.00	0.43	0.30	0.02	0.20	0.39	0.56	1.63
	Lewis	60	6.67	0.36	0.28	0.02	0.17	0.30	0.51	1.74
	Simmons Park	60	3.33	0.35	0.23	0.04	0.18	0.28	0.47	1.08
Acrylonitrile Detroit, Michigan	Southfield	59	100.00	0.23	0.00	0.23	0.23	0.23	0.23	0.23
	Allen Park	33	100.00	0.23	0.00	0.23	0.23	0.23	0.23	0.23
	Dearborn	528	99.62	0.27	0.08	0.13	0.23	0.23	0.23	0.42
	River Rouge	58	98.28	0.32	0.10	0.16	0.23	0.23	0.42	0.42
	N. Delray	25	52.00	0.93	0.68	0.42	0.42	0.42	1.16	2.95
	S. Delray	59	100.00	0.23	0.03	0.23	0.23	0.23	0.23	0.42

*ND data were replaced with MDL/2, invalid data not included in summary. Key: Q1 = First Quarter, Q3 = Third Quarter *Produced by Battelle*

Table 79: Summary of Benzene Pilot Data (Concentration Distribution) in Selected Pilot Cities (Detroit, 1 small and 1 urban) by Site

POLLUTANT PILOT CITY	SITE	TOTAL OBS[1]	PERCENT ND	CONCENTRATION DISTRIBUTION (µg/m^3)						
				Mean	Std Dev	Min	Q1	Median	Q3	Max
Benzene Grand Junction, Colorado	Mesa Co Health Dept	72	0.00	2.77	1.43	0.70	1.81	2.44	3.52	8.69
	Mesa Co Traffic Services	148	0.00	2.14	1.34	0.69	1.07	1.75	2.83	6.76
Benzene Seattle, Washington	Beacon Hill	139	0.00	1.25	0.67	0.33	0.79	1.11	1.50	5.44
	Georgetown	67	1.49	1.80	1.58	0.02	0.73	1.35	1.93	7.10
	Lake Forest Park	61	0.00	1.65	1.09	0.45	0.96	1.35	2.07	5.28
	Lake Sammamish	57	0.00	1.17	0.50	0.36	0.73	1.21	1.56	2.16
	Maple Leaf Reservoir	62	0.00	1.13	0.45	0.37	0.78	1.09	1.47	2.14
	Sea Tac	63	0.00	1.04	0.52	0.32	0.67	0.90	1.36	3.03

Table 79: Summary of Benzene Pilot Data (Concentration Distribution) in Selected Pilot Cities (Detroit, 1 small and 1 urban) by Site

POLLUTANT PILOT CITY	SITE	TOTAL OBS[1]	PERCENT ND	CONCENTRATION DISTRIBUTION (µg/m^3)						
				Mean	Std Dev	Min	Q1	Median	Q3	Max
Benzene Detroit, Michigan	Southfield	59	0.00	1.98	0.92	0.77	1.30	1.72	2.37	6.56
	Allen Park	33	0.00	1.50	0.71	0.73	1.02	1.37	1.79	4.38
	Dearborn	528	0.19	1.89	1.04	0.11	1.18	1.66	2.28	7.96
	River Rouge	58	0.00	2.16	1.29	0.61	1.28	1.61	2.71	6.55
	N. Delray	25	4.00	2.64	2.53	0.11	1.05	1.87	2.62	11.40
	S. Delray	59	0.00	21.94	34.77	1.01	1.83	4.44	32.09	158.87

1 ND data were replaced with MDL/2, invalid data not included in summary.

Produced by Battelle

Table 80: Summary of Methylene Chloride Pilot Data (Concentration Distribution) in Selected Pilot Cities (Detroit, 1 small and 1 urban) by Site

POLLUTANT PILOT CITY	SITE	TOTAL OBS[1]	PERCENT ND	CONCENTRATION DISTRIBUTION (µg/m^3)						
				Mean	Std Dev	Min	Q1	Median	Q3	Max
Methylene chloride Barceloneta, San Juan, Puerto Rico	Barceloneta	57	0.00	12.83	11.89	1.46	5.46	8.51	17.04	66.56
	San Juan	47	17.02	0.57	0.43	0.10	0.30	0.53	0.71	1.98
Methylene chloride Seattle, Washington	Beacon Hill	69	0.00	1.52	0.60	0.52	1.12	1.40	1.81	3.40
	Georgetown	66	0.00	1.85	0.28	1.38	1.64	1.83	2.03	2.57
	Lake Forest Park	61	0.00	1.78	0.58	0.65	1.39	1.78	2.25	2.71
	Lake Sammamish	56	0.00	1.72	0.21	1.32	1.54	1.74	1.88	2.16
	Maple Leaf Reservoir	62	0.00	1.98	0.49	1.10	1.67	1.85	2.20	3.15
	Sea Tac	63	0.00	1.67	0.52	0.98	1.24	1.59	2.00	2.82
Methylene chloride Detroit, Michigan	Southfield	59	0.00	62.92	197.93	0.14	0.31	0.62	2.35	1151.69
	Allen Park	33	0.00	802.31	2207.07	0.33	2.44	51.23	329.23	11575.02
	Dearborn	528	5.11	12.25	48.72	0.07	0.67	1.95	5.78	691.91
	River Rouge	58	15.52	10.00	57.14	0.07	0.40	0.79	1.10	432.30
	N. Delray	25	28.00	3.08	4.53	0.40	0.40	1.35	3.16	18.56
	S. Delray	59	1.69	2.57	7.66	0.10	0.56	0.83	2.15	58.59

1 ND data were replaced with MDL/2, invalid data not included in summary.

Produced by Battelle

Table 81: Summary of Manganese TSP Pilot Data (Concentration Distribution)in Selected Pilot Cities (Detroit, 1 small and 2 urban) by Site

POLLUTANT PILOT CITY	SITE	TOTAL OBS [1]	% ND	CONCENTRATION DISTRIBUTION (µg/m^3)						
				Mean	Std Dev	Min	Q1	Median	Q3	Max
Manganese TSP Grand Junction, Colorado	Mesa Co Health Dept	59	0.00	0.02000	0.03000	0.00425	0.01000	0.02000	0.02000	0.25000
	Mesa Co Traffic Services	88	0.00	0.05000	0.02000	0.00926	0.02000	0.05000	0.07000	0.11000
Manganese TSP Providence Rhode Island	East Providence	62	1.61	0.00766	0.00513	0.00004	0.00446	0.00642	0.01000	0.03000
	Urban League	63	0.00	0.00934	0.00455	0.00013	0.00627	0.00879	0.01000	0.03000
Manganese TSP Seattle, Washington	Lake Forest Park	72	0.00	0.00504	0.00250	0.00070	0.00338	0.00471	0.00656	0.01000
	Lake Sammamish	63	0.00	0.00691	0.00466	0.00106	0.00297	0.00547	0.00910	0.02000
	Maple Leaf Reservoir	62	0.00	0.00551	0.00278	0.00135	0.00350	0.00483	0.00639	0.01000
	Sea Tac	133	0.00	0.00791	0.00653	0.00114	0.00355	0.00553	0.01000	0.04000
Manganese TSP Detroit, Michigan	Southfield	63	0.00	0.02000	0.00997	0.00107	0.01000	0.01000	0.02000	0.05000
	Allen Park	77	0.00	0.03000	0.02000	0.00809	0.02000	0.03000	0.04000	0.11000
	Dearborn	74	0.00	0.20000	0.18000	0.02000	0.09000	0.15000	0.26000	1.19000
	N.E. Detroit	71	0.00	0.03000	0.02000	0.00272	0.02000	0.02000	0.03000	0.08000
	River Rouge	70	0.00	0.08000	0.05000	0.00931	0.04000	0.06000	0.09000	0.27000
	N. Delray	168	0.00	0.09000	0.05000	0.00820	0.05000	0.08000	0.12000	0.28000
	S. Delray	72	0.00	0.27000	0.29000	0.03000	0.09000	0.20000	0.34000	1.94000
	Ypsilanti	18	0.00	0.00915	0.00467	0.00294	0.00653	0.00762	0.01000	0.02000

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In general, Detroit sites showed greater variability between sites than other pilot cities. This is an important distinction to make when considering comparisons between pilot cities. It seems that in Detroit there are site-specific instances of high average concentrations for some COPCs. In contrast, other pilot cities' means were largely the result of homogeneous concentrations at all sites. *In other words, the overall risk from the combined COPCs for Detroit is not evenly distributed*. The overall risk from combined COPCs in pilot cities where levels were more homogeneous, is more representative of the general risk for that population. **Table 82** compares Detroit means with other pilot city means both including (overall mean) and excluding (adjusted mean) the highest and lowest site. For example, when N. Delray and S. Delray are excluded, Detroit's adjusted means for acrylonitrile and benzene fall within the range of other pilot city means. For methylene chloride and manganese, however, Detroit's mean remains above the range of other pilot cities even after excluding Allen Park and S. Delray, respectively. Although the adjusted means for Detroit do not in any way negate the high levels at specific sites, they do allow for more meaningful comparisons between Detroit and other pilot cities in the national study.

Table 82: Comparison of Detroit Means With Other Pilot City Means
(Including Overall Mean and Excluding Adjusted Mean)

COPCs	MEAN AT HIGHEST DETROIT SITE (µg/m^3)	DETROIT OVERALL MEAN (µg/m^3)	DETROIT ADJUSTED MEAN* (µg/m^3)	RANGE OF ALL PILOT CITY MEANS (µg/m^3) (excluding Detroit)	RANGE OF MEANS OF DETROIT PILOT SITES (µg/m^3) (excluding outliers)
Acrylonitrile	0.93 (N. Delray)	0.28	0.26	0.03 – 0.38	0.23 - 0.42
Benzene	21.94 (S. Delray)	3.48	1.95	0.85 – 2.34	1.89 – 2.64
Methylene Chloride	802.31 (Allen Park)	49.17	16.20	0.38 – 7.29	3.08 – 62.92
Manganese	0.27 (S. Delray)	0.10	0.08	0.006 – 0.04	0.009 – 0.20

*Excluding high and low values.

13.2 Comparing The Detroit Pilot Project to Other Urban Air Toxics Initiatives

There have been very few large-scale air toxics monitoring studies in major urban areas from which comparisons can be made to the Detroit Pilot Project. How Detroit compares with other major cities within USEPAs national pilot program was presented in the previous section. This section will focus on comparing monitoring data from the Detroit Pilot Project with monitoring data from similar initiatives in other urban areas. The caveats for comparing data between sites with different sampling methodologies and data analysis strategies apply here as well. Since many published studies report only summary data, such as annual averages and minimum or maximum values, comparisons are limited to this level of analysis. Those chemicals identified as Detroit COPCs for acute and chronic non-cancer effects as well as cancer effects will be the focus of these comparisons. Detroit COPCs are identified in **Table 83**.

Table 83: Detroit COPCs & Cancer Risk Drivers

ACUTE, NON-CANCER (exceeds known acute benchmark)	CHRONIC, NON-CANCER (HQ > 0.1)	CANCER RISK (> 1 X 10^{-6})	
Methylene Chloride	Acetaldehyde Acetonitrile Acrylonitrile Benzene Manganese (TSP) Naphthalene	1,3-Butadiene 1,4-Dichlorobenzene Acetaldehyde Acrylonitrile Arsenic (TSP) Benzene	Cadmium (TSP) Carbon Tetrachloride Formaldehyde Methylene Chloride Naphthalene Nickel (TSP)

THE BALTIMORE CASE STUDY

In April of 2000, USEPA published the Baltimore Community Environmental Partnership Air Committee Technical Report (USEPA, 2000c). This report is a summary of an air screening assessment for five neighborhoods in the southern Baltimore area performed by the Air Committee of the Baltimore Community Environmental Partnership between 1992 and 1996. The study area is industrialized with a large concentration of industrial, commercial, and waste treatment and disposal facilities. The air study relied heavily on modeling results from the emissions inventory but monitoring data was also available from the statewide monitoring network. Monitoring data from one site within the study area was reported for four chemicals identified as COPCs, three of which were also identified as COPCs in Detroit: 1,3-butadiene, benzene, and carbon tetrachloride. Only the most recent monitoring data available at the time (1996 annual averages) was reported. The appendix of the Baltimore report also included a table of annual averages reported from other monitoring initiatives. No quantitative risk estimates were provided. Annual averages from the Baltimore monitoring network and from other available monitoring data as reported in the Baltimore study are presented in **Table 84** along with Detroit annual averages for these same compounds.

Table 84: Comparison of Annual Averages Between the Baltimore Study and Detroit Pilot Project

LOCATION	ANNUAL AVERAGE CONCENTRATIONS (µg/m^3)		
	1,3-Butadiene	Benzene	Carbon Tetrachloride
Los Angeles, California	1.04	7.3	0.50
Fresno, California	0.43	4.5	0.49
Fremont, California	0.34	4.1	0.48
Louisiana	N/A	3.8	1.0
Baltimore	0.22	3.4	0.94
NY Air Toxics	N/A	2.67	1.1
Texas Air Toxics	0.91	1.9	0.57
Detroit	**0.13**	**3.48**	**0.62**

THE CHATTANOOGA AIR TOXICS STUDY

An air toxics monitoring study was conducted from November 1998 to October 1999 by USEPA's Region 4 and the Chattanooga-Hamilton County Air Pollution Control Bureau.[24] The risk assessment focused on direct inhalation exposure to airborne toxicants. Chemical and site-specific risk estimates were derived for a high-end exposure scenario for adults and children using the 95th UCL on the mean. Risk estimates were derived quite differently from DATI risk estimates, therefore, caution is advised in making direct risk comparisons.

[24] Contact information for the Chattanooga air toxics study is available on USEPA's website at http://www.epa.gov/region04/sesd/reports/1999-0775.html.

Several of the six monitoring sites are concentrated in an area that is heavily industrial, 99% African American, 65% below poverty level, and in close proximity to a Superfund site. No acute COPCs were identified at any of the sites. Two of the three chemicals identified as chronic, non-cancer COPCs were also COPCs in Detroit: formaldehyde and manganese. It should be noted, however, that Chattanooga's COPCs were identified as those chemicals with an HQ > 1, a less stringent screening criterion than was used for Detroit which used an HQ > 0.1 as an initial screening level. Chemicals identified as having a > 1 x 10^{-6} cancer risk in Chattanooga that were also cancer risk drivers in Detroit are:

- 1,4-Dichlorobenzene
- Arsenic
- Benzene
- Carbon tetrachloride
- Formaldehyde
- Nickel

The range of annual averages for shared COPCs and cancer risk drivers and for two additional Detroit cancer risk drivers are presented in **Table 85**.

Table 85: Comparison of Monitored Pollutants - Range of Annual Averages for Chattanooga and Detroit

POLLUTANTS	RANGE OF ANNUAL AVERAGES (µg/m^3)	
	Chattanooga	Detroit
Chronic, Non-Cancer COPCs		
Formaldehyde	1.75 - 5.24	2.02 – 5.55
Manganese	0.0113 –0.0367	0.00915 – 0.274
Cancer Risk Drivers		
1,4-Dichlorobenzene	0.933 – 1.09	0.24 – 3.51
Arsenic	0.00116 – 0.00172	0.000778 – 0.00241
Benzene	1.51 – 3.52	1.50 – 21.59
Carbon tetrachloride	0.692 – 0.872	0.052 – 0.633
Formaldehyde	1.75-5.24	2.02 – 5.55
Nickel	0.00316 – 0.00910	0.00084 – 0.00491
***Other COPCs**		
Methylene Chloride	0.872 – 1.91	2.57 – 802
Naphthalene	0.786 – 1.06	0.316 – 18.4

*Identified as both chronic, non-cancer and cancer risks for Detroit only

THE WEST LOUISVILLE AIR TOXICS STUDY

The West Louisville Air Toxics Study was a large scale monitoring initiative conducted between April 2000 and April 2001 at twelve community sites in the West Louisville, Kentucky area (USEPA, 2000b). Residential sites included schools, churches, and one private residence, therefore, monitors were placed where people frequented rather than exclusively at high impact sites. Participants in this monitoring initiative were the MLAPCD, USEPA, the Commonwealth of Kentucky, and a stakeholders group, the West Jefferson County Community Task Force. A risk analysis of the data was contracted to and performed by Sciences International, Inc. VOCs were the focus of this study although SVOCs, metals, and PBT analytes were also measured at some sites. In total, over 200 chemicals were monitored.

Table 86 is a summary data table comparing shared COPCs between West Louisville and Detroit. It is important to note that for some chemicals (1,3-butadiene, 1,4-dichlorobenzene, acrylonitrile) the percent detected at West Louisville was quite poor (below 50% detection at more than half of the monitoring sites). Formaldehyde, arsenic, manganese, and nickel detection levels at West Louisville were quite high (approximately 100% at all sites) and thus there is higher confidence in the data.

Table 86: Comparison of Monitored Pollutants Between West Louisville Air Toxics Study and the Detroit Pilot Project

POLLUTANTS	MAXIMUM DETECTED CONCENTRATION AT ANY DETROIT SITE (µg/m^3)	MAXIMUM DETECTED CONCENTRATION AT ANY WEST LOUISVILLE SITE (µg/m^3)	RANGE OF MEAN CONCENTRATIONS AT DETROIT SITES (µg/m^3)	RANGE OF MEAN CONCENTRATIONS AT WEST LOUISVILLE SITES (µg/m^3)
Formaldehyde	25.8	12	2.022 – 5.551	0.707 – 2.48
Naphthalene	93.3	2.75	0.2917 – 18.39	0.0283 – 1.41
1,3-butadiene	0.74	57	0.04425* - 0.1911	0.360 – 5.83
1,4-dichlorobenzene	83.3	8.38	0.24 – 3.506	0.355 – 0.990
Acrylonitrile	2.97	1.9	0.2279* - 0.9137	0.218 – 2.2
Benzene	159	13.23	1.5 – 21.59	0.741 – 2.44
Carbon Tetrachloride	1.07	1.72	0.52 – 0.6333	0.578 – 0.738
Methylene Chloride	11,579	82.43	0.57 – 802.7	0.450 – 9.21
Arsenic	0.025	0.011	0.0007776 – 0.003359	0.00107 – 0.00195
Cadmium	0.029	0.0056	0.0001663 – 0.001218	0.000355 – 0.000994
Manganese	1.94	0.056	0.009151 – 0.2737	0.00571 – 0.0203
Nickel	0.097	0.056	0.0008411 – 0.004909	0.00315 – 0.00936

NOTE: data for acetaldehyde and acetonitrile were not available from West Louisville Air Toxics Study
*represents the MDL/2 since all observations were below the MDL.

When considering cancer risk from all COPC, estimates from West Louisville ranged from 76 X 10^{-6} to 690 X 10^{-6} assuming a high end exposure (95% UCL on the mean). Median exposure cancer risks for all COPCs were reported between 38 to 180 X 10^{-6}.

THE CALIFORNIA MATES II PROJECT

The MATES-II was initiated in 1997 to monitor and evaluate urban air toxics in the South Coast Air Basin which included sites in Los Angeles, San Bernardino, Riverside, and Orange Counties. The MATES-II consisted of several elements including two separate monitoring initiatives and an evaluation of emissions inventory and modeling. For comparison to the Detroit Pilot Project only the results from the larger monitoring initiative will be discussed. Monitoring

consisted of a network of 12 fixed sites for which approximately 30 of the more common air toxics were measured between April 1998 through March 1999.

As shown in **Table 87**, the cancer risks from ambient exposure to monitored air toxics ranged from 1,120 to 1,740 X 10^{-6} (average risk is 1,400 X 10^{-6}). These risk estimates were driven largely by mobile sources when diesel particulate was added to the analysis (monitoring of EC was used as a surrogate for diesel particulates).

Table 87: Ambient Concentrations at 10 Fixed Sites Analyzed by Two Laboratories for CA MATES-II (April 1998-March 1999) Compared to Detroit Pilot Project Means (April 2001-April 2002)

POLLUTANTS	RANGE OF MEANS BY AQMD (µg/m^3)	RANGE OF MEANS BY ARB (µg/m^3)	RANGE OF MEANS DATI (µg/m^3)
1,3-butadiene	0.508-1.41	0.375-1.35	0.04425* - 0.1911
Methylene chloride	1.74-2.95	2.05-4.65	2.57 – 802.7
Benzene	2.62-5.74	2.11-5.81	1.50 – 21.59
Carbon Tetrachloride	0.629-0.692**	0.629-0.692	0.52 – 0.6333
Formaldehyde	3.33-6.47		2.022 – 5.551
Acetaldehyde	1.98-4.23		0.859-2.172
Chromium	0.0010-0.0048**	0.0044-0.0132	0.002226-0.0035
Hexavalent Chromium	0.00015-0.00030	0.00010-0.00022**	0.0000363-0.0000444
Manganese	0.0151-0.0741	0.0207-0.0656	0.009151 – 0.2737
Mercury	---	0.0015-0.0025**	0.0019-0.0024
Nickel	0.0084-0.0161	0.0041-0.009	0.0008411 – 0.004909

***represents the MDL/2 since all observations were below the MDL.**
****The number of samples below the MDL for these compounds was nearly 100%.**
AQMD = Air Quality Management District
ARB = Air Resource Board

Comparisons of Detroit Pilot Project means with California MATES-II site means reveal higher overall averages in Detroit for methylene chloride, benzene and manganese. Detroit levels of 1,3-butadiene, chromium, Cr^{+6}, and nickel are considerably lower than those measured in the MATES-II study while other COPC are comparable.

USEPA NATIONAL AIR TOXICS MONITORING STRATEGY

In pursuit of a national air toxics monitoring strategy, two initiatives were developed by USEPA: 1) creation and analysis of a national air toxics database and 2) analysis of data from the national pilot monitoring program. Battelle Memorial Institute and STI were contracted by the Lake Michigan Air Director's Consortium (LADCO), an USEPA partner, to perform these tasks. The national pilot monitoring program was discussed in the previous section. The purpose of the database project was to "mine" all existing data for information regarding spatial pattern, temporal profile, and general characteristics of air toxics compounds. Much of the initial work from this project focused on assessing the overall quality and completeness of data to address design and implementation needs for a national monitoring network. Characterization of air toxics concentrations at the national and local scale will be addressed in future phases.

Data for the historical database were collected from a host of programs with differing objectives, monitoring methods, reporting units, and requirements. There were differences between pilot city screening, monitoring and analysis methodologies as well. Therefore, any conclusions drawn from this data alone should be considered preliminary until supported by a well-designed

national air toxics monitoring network. However, a few general observations were made and are reported below.[25]

Analysis of background levels from all data sources (pilot, historical, STN-urban, IMPROVE-rural, and National Oceanic and Atmospheric Administration - Climate Monitoring and Diagnostics Laboratory [NOAA-CMDL]) indicate that national background concentrations of formaldehyde, benzene, carbon tetrachloride, and total chromium ($PM_{2.5}$) exceed the 1×10^{-6} cancer risk benchmark (McCarthy et. al, 2004).

Analysis of $PM_{2.5}$ metals utilizing all available data (pilot, historical, STN-urban, IMPROVE-rural) indicate that over 90% of rural sites had lower concentrations than the lowest 20% of urban sites. Concentrations at urban sites can vary quite dramatically. For instance, typical urban concentrations of nickel fall into the 0.002 to 0.007 $\mu g/m^3$ range. The average ambient concentration at one Dallas, TX site was reported at 0.5 $\mu g/m^3$ (1990 – 2002) due to an electroplating facility located next door to the monitoring site. Detroit sites' annual averages for nickel fell within the national urban range.

Of all the $PM_{2.5}$ metals, manganese concentrations nationally are the most similar between urban and rural sites while chromium concentrations show the largest differences. The similarity in manganese concentrations between urban and rural sites nationally seems to suggest that the overall contribution of local sources to manganese levels in urban areas may be small. However, regional differences in manganese do exist as do local "hotspots". For example, monitoring data from sites in Birmingham, Alabama, Marietta, Ohio and several rural sites in Georgia reported elevated levels of manganese in 2001 and/or 2002 that were greater than or similar to elevated levels in Detroit. **Tables 88** and **89** show those sites reporting monitored concentrations of manganese that exceeded the chronic health benchmark for manganese in 2001 and 2002.[26]

State Name Acronyms used in **Table 88** and **Table 89** include:

CA = California	MI = Michigan	SC = South Carolina
CO = Colorado	MN = Minnesota	TN = Tennessee
GA = Georgia	NM = New Mexico	WA = Washington
IL = Illinois	OH = Ohio	WV = West Virginia
IN = Indiana	OR = Oregon	WI = Wisconsin

[25] For more details go to http://ladco.org/toxics.html.
[26] Retrieved from the USEPA AQS database http://www.epa.gov/air/data/index.html on 11/04.

Table 88: Manganese (TSP) 2002 – Nationally Reported Annual Means Meeting or Exceeding the USEPA RfC of 0.05 µg/m^3 (Source: USEPA Air Data, AQS Database http://www.epa.gov/air/data/index.html)

MEAN (µg/m^3)	SITE ADDRESS	CITY	COUNTY	ST	LOCATION TYPE	URBANIZED AREA
0.337	Sparta - Lewis Middle School		Hancock Co	GA	Rural	
0.285	Talbotton - Big Lazer Wild Life Management		Talbot Co	GA	Rural	
0.243	Lincolnton		Lincoln Co	GA	Rural	
0.20434	**2842 Wyoming**	**Dearborn**	**Wayne Co**	**MI**	**Suburban**	**Detroit, MI**
0.191	State Rt. 676; Washington Career Center	Marietta	Washington Co	OH	Suburban	Parkersburg, WV-OH
0.1827	3535 E. 114th St.	Chicago	Cook Co	IL	Suburban	Chicago, IL-Northwestern IN
0.16211	**7701 W. Jefferson**	**Detroit**	**Wayne Co**	**MI**	**Urban And Center City**	**Detroit, MI**
0.144	1840 Otsego Ave., Coshocton	Coshocton	Coshocton Co	OH	Urban And Center City	
0.1073*	**6921 West Fort**	**Detroit**	**Wayne Co**	**MI**	**Urban And Center City**	**Detroit, MI**
0.1031	E. 56th St., Ferro Corp., Cleveland	Cleveland	Cuyahoga Co	OH	Suburban	Cleveland, OH
0.09763*	**6921 West Fort**	**Detroit**	**Wayne Co**	**MI**	**Urban And Center City**	**Detroit, MI**
0.09054	15th & Madison	Granite City	Madison Co	IL	Urban And Center City	St. Louis, MO-IL
0.0785	803 N. Loren Ave., Azusa	Azusa	Los Angeles Co	CA	Suburban	Los Angeles, CA
0.0735	Grant Rd., Ferro Corp., Cleveland	Cleveland	Cuyahoga Co	OH	Suburban	Cleveland, OH
0.0733	2901 Yankee Rd., Middletown 45044	Middletown	Butler Co	OH	Suburban	Middletown, OH
0.06819	2522 Burnside St Knoxville 37921	Knoxville	Knox Co	TN	Urban And Center City	Knoxville, TN
0.06415	**315 Genesee**	**River Rouge**	**Wayne Co**	**MI**	**Suburban**	**Detroit, MI**
0.0639	5888 Mission Blvd., Rubidoux	Rubidoux	Riverside Co	CA	Suburban	Riverside-San Bernardino, CA
0.05859	2522 Burnside St Knoxville 37921	Knoxville	Knox Co	TN	Urban And Center City	Knoxville, TN
0.05597	City Hall Roof	Virginia	St. Louis Co	MN	Urban And Center City	
0.049	201 Prosperity SW		Bernalillo Co	NM	Rural	Albuquerque, NM
0.04879	1919 Vermont Avenue	Knoxville	Knox Co	TN	Urban And Center City	Knoxville, TN
0.0473	735 W Harrison St	Chicago	Cook Co	IL	Urban And Center City	Chicago, IL-Northwestern IN
0.045	924 Fourth Ave.	Grand Junction	Mesa Co	CO	Urban And Center City	Grand Junction, CO

***Co-located samples were analyzed to assess interlaboratory and intersampler precision.**

Table 89: Manganese (TSP) 2001 – Nationally Reported Annual Means Meeting or Exceeding the USEPA RfC 0.05 µg/m^3 (Source: USEPA Air Data, AQS Database http://www.epa.gov/air/data/index.html)

MEAN (µg/m^3)	SITE ADDRESS	CITY	COUNTY	ST	LOCATION TYPE	URBANIZED AREA
0.31907	**7701 W. Jefferson**	**Detroit**	**Wayne Co**	**MI**	**Urban And Center City**	**Detroit, MI**
0.3167	State Rt. 676; Washington Career Center	Marietta	Washington Co	OH	Suburban	Parkersburg, WV-OH
0.18485	2842 Wyoming	Dearborn	Wayne Co	MI	Suburban	Detroit, MI
0.1779	3535 E. 114th St.	Chicago	Cook Co	IL	Suburban	Chicago, IL-Northwestern IN
0.1344	Fraser U.S. 17) & Bourne	Georgetown	Georgetown Co	SC	Urban And Center City	
0.1136	E. 56th St., Ferro Corp.	Cleveland	Cuyahoga Co	OH	Suburban	Cleveland, OH
0.10391*	**6921 West Fort**	**Detroit**	**Wayne Co**	**MI**	**Urban And Center City**	**Detroit, MI**
0.10354	15th & Madison	Granite City	Madison Co	IL	Urban And Center City	St. Louis, MO-IL
0.0908	2901 Yankee Rd., Middletown, 45044	Middletown	Butler Co	OH	Suburban	Middletown, OH
0.0892	Grant Rd., Ferro Corp.	Cleveland	Cuyahoga Co	OH	Suburban	Cleveland, OH
0.08835*	**6921 West Fort**	**Detroit**	**Wayne Co**	**MI**	**Urban And Center City**	**Detroit, MI**
0.0858	2850 West 3rd	Cleveland	Cuyahoga Co	OH	Urban And Center City	Cleveland, OH
0.0839	803 N. Loren Ave.	Azusa	Los Angeles Co	CA	Suburban	Los Angeles, CA
0.0725	South Side Of West 3rd St.	Cleveland	Cuyahoga Co	OH	Urban And Center City	Cleveland, OH
0.06725	**315 Genesee**	**River Rouge**	**Wayne Co**	**MI**	**Suburban**	**Detroit, MI**
0.0639	401 Cleland Street (At Duke St)	Georgetown	Georgetown Co	SC	Urban And Center City	
0.06284	Forest Heights/1706 NW 24th Avenue	Portland	Multnomah Co	OR	Urban And Center City	Portland-Vancouver, OR-WA
0.0597	735 W Harrison St	Chicago	Cook Co	IL	Urban And Center City	Chicago, IL-Northwestern IN
0.0593	Butts & Merriman	Georgetown	Georgetown Co	SC	Urban And Center City	
0.0577	5888 Mission Blvd.	Rubidoux	Riverside Co	CA	Suburban	Riverside-San Bernardino, CA
0.0525	5111 West 164th Street	Brook Park	Cuyahoga Co	OH	Suburban	Cleveland, OH
0.052	924 Fourth Ave.	Grand Junction	Mesa Co	CO	Urban And Center City	Grand Junction, CO
0.0517	2547 St Tikhon St	Cleveland	Cuyahoga Co	OH	Urban And Center City	Cleveland, OH
0.05043	E3520 Hwys 22/54 Waupaca	Waupaca	Waupaca Co	WI	Suburban	
0.046	924 Fourth Ave.	Grand Junction	Mesa Co	CO	Urban And Center City	Grand Junction, CO

***Co-located samples were analyzed to assess interlaboratory and intersampler precision.**

URBAN AIR TOXICS MONITORING PROGRAM (UATMP) REPORTS FOR 2001 AND 2002

The UATMP was initiated in 1987 by USEPA to characterize the composition and magnitude of urban air pollution through extensive ambient air monitoring. During the years of the Detroit Pilot Project (2001-2002) the monitoring program ran by calendar year. Since the Detroit Pilot Project ran from April 2001-April 2002, both the UATMP 2001 report (USEPA, 2002a) and 2002 (USEPA, 2003a) results will be summarized here.

2001 UATMP: The 2001 effort included 12 months of sampling of over 200 VOC, SVOC, carbonyl, and semi-volatile non-methane organic compounds (SNMOC) at 43 monitoring sites in or near 27 urban areas (USEPA, 2002a). Program-wide prevalent compounds are defined as those compounds detected in at least 75% of all samples and that contributed to at least 90% of the mass concentration within a compound group. For these compounds, accuracy of the data is quite high and therefore meaningful comparisons can be made between monitoring sites. The 13 program-wide prevalent compounds for 2001 are shown in **Table 90**.

Table 90: 2001 UATMP Prevalent Compounds

HYDROCARBONS	HALOGENATED HYDROCARBONS	POLAR	CARBONYLS
Acetylene Benzene Ethylbenzene o-Xylene Propylene Toluene	Chloromethane Dichlorofluoromethane Methylene Chloride	Methyl Ethyl Ketone	Acetaldehyde Acetone Formaldehyde

The mean (geometric) concentrations of four prevalent compounds at Detroit sites are as follows:

Acetaldehyde: N. Delray and River Rouge had geometric means of acetaldehyde approximately 2.5 times greater than at all other sites.

Benzene: The geometric mean of benzene at Allen Park was 2 to 4 times greater than at all other sites.

Formaldehyde: N. Delray and River Rouge had geometric means for formaldehyde that were more than two times greater than all other sites. One site in Missouri had the highest geometric mean, approximately twice that of N. Delray and River Rouge.

Methylene Chloride: The geometric mean at Allen Park was six times greater than most other program means. This mean was three times greater than the second highest mean in Puerto Rico. Allen Park also had the highest mean concentration in 2000.

2002 UATMP: The 2002 UATMP effort included 12 months of sampling of over 200 VOC, SVOC, carbonyl, and SNMOC at 55 monitoring sites in or near 32 urban areas (USEPA, 2003a). A few sites also sampled metals and one site sampled Cr^{+6}. Due to the additional sites participating in this year's program, the criteria for identifying program-wide prevalent compounds were modified as follows: 1) detected in at least 60% of all samples at a site, 2) contributed to at least 90% of the mass concentration within a compound group and, 3) the compound satisfied criteria 1 and 2 for the majority of sites collecting these compounds (75%). Again, these data are of high quality and are useful in making comparisons between sites. **Table 91** shows the 12 program-wide prevalent compounds for the 2002 UATMP.

Table 91: 2002 UATMP Prevalent Compounds

HYDROCARBONS	HALOGENATED HYDROCARBONS	CARBONYLS
1,2,4-trimethylbenzene Acetylene Benzene Ethylbenzene M,p-xylene o-xylene Propylene Toluene	Chloromethane Dichlorodifluoromethane Trichlorofluoromethane	Formaldehyde

Table 91 Summary of mean (geometric) concentration at Detroit sites:

Benzene: S. Delray and N.E. Detroit were the sites with the highest geometric means for benzene. Mean concentrations were 3 to 4 times greater than the majority of other sites.

Formaldehyde: Geometric means at Detroit sites were all within range of other site means. The highest concentrations were at sites located in Missouri, Colorado, and Iowa.

Tables 92 and **93** are summary statistics for the program-wide prevalent compounds in 2001 and 2002 as compared to monitored concentrations in Detroit.

Table 92: Comparison of Summary Statistics for UATMP 2001 and Detroit Pilot Project

UATMP 2001 ($\mu g/m^3$)						DETROIT PILOT PROJECT (2001-2002) ($\mu g/m^3$)		
Compound	**Min**	**Max**	**Average**	**Geometric Mean**	**Median**	**Min**	**Max**	**Averages**
Acetaldehyde	0.0018	35.3	2.37	1.84	1.99	0.022	9.26	0.859 – 2.172
Benzene	0.064	146.0	2.22	1.51	1.40	0.115	159*	1.50 – 21.59
Formaldehyde	0.021	73.1	4.85	3.25	3.32	0.014	25.8	2.022 – 5.551
Methylene Chloride	.056	11,585**	16.1	0.758	0.556	0.069	11,580**	2.57 – 802.7

*The max UATMP 2001 value for benzene is from S. Delray. The Detroit max of 159 $\mu g/m^3$ for benzene occurred at S. Delray in 2002 (see **Table 93**).

**The max for methylene chloride is from Allen Park. Values may differ due to differences in converting from ppbV to $\mu g/m^3$ and rounding.

Table 93: Comparison of Summary Statistics for UATMP 2002 and Detroit Pilot Project

UATMP 2002 ($\mu g/m^3$)						DETROIT PILOT PROJECT 2001-2002 ($\mu g/m^3$)		
Compound	**Min**	**Max**	**Average**	**Geometric Mean**	**Median**	**Min**	**Max**	**Averages**
Benzene	0.204	159	1.82	1.63	1.63	0.115	159	1.50 – 21.59
Formaldehyde	0.033	71.1	3.14	2.35	2.77	0.014	25.8	2.022 – 5.551

Overall, the profile of air toxics in Detroit is very similar to other urban areas in terms of the nature and prevalence of detected compounds. For instance, nationwide background concentrations of formaldehyde, benzene, and carbon tetrachloride exceed the 1×10^{-6} cancer risk screening level. Not surprisingly, these compounds were also found to exceed that threshold in Detroit. In contrast, levels of methylene chloride at Allen Park, benzene and naphthalene at S. Delray, and manganese at Dearborn and S. Delray far exceeded concentrations reported in other urban areas for the 2001-2002 monitoring season. Average manganese concentrations at Dearborn and S Delray, for example, were consistently one order of magnitude higher than average concentrations found in Chattanooga, West Louisville, California's South Coast Air Basin and the ten national pilot program cities. While monitoring data can help to determine which air toxics are likely to contribute to risk in a particular area, they do not necessarily represent the actual concentrations to which an individual might be exposed. Clearly, site-specific levels of methylene chloride, naphthalene, benzene and manganese in Detroit suggest an increased potential for risk of health effects. Further investigation and follow-up is necessary to accurately describe and characterize that potential risk.

In addition to continued monitoring efforts by MDEQ, USEPA is currently conducting a three year study in Detroit to characterize human exposure to air pollution. The DEARS seeks to combine residential indoor and outdoor air monitoring, personal monitoring, analysis of human activity patterns, and source apportionment data to determine the association between ambient concentrations and human exposure concentrations to selected air toxics and PM. The central station USEPA is utilizing for their ambient estimate is located at Allen Park. VOCs, PAHs, criteria pollutants, and speciated $PM_{2.5}$ will be evaluated. Because the DEARS neighborhoods are overlapping with the Detroit Pilot Project monitoring sites, it will be possible to get some idea of how closely measured ambient air concentrations of pollutants predict human exposure concentrations, and consequently, the potential for risk to human health.

13.3 Ambient Air Concentration Trends in Michigan

In addition to comparing ambient air concentrations of air toxics in the Detroit area with levels monitored elsewhere in the country to understand spatial differences, it is useful to compare recently monitored levels to historical data to understand temporal trends. **Figures 27** to **33** show trend data for Michigan sites of monitored levels of two of the cancer risk drivers and one chemical of concern identified in the DATI. Annual average air concentrations were used to assess long term trends in this section.

Figure 27: Benzene Annual Average Air Concentration Trends

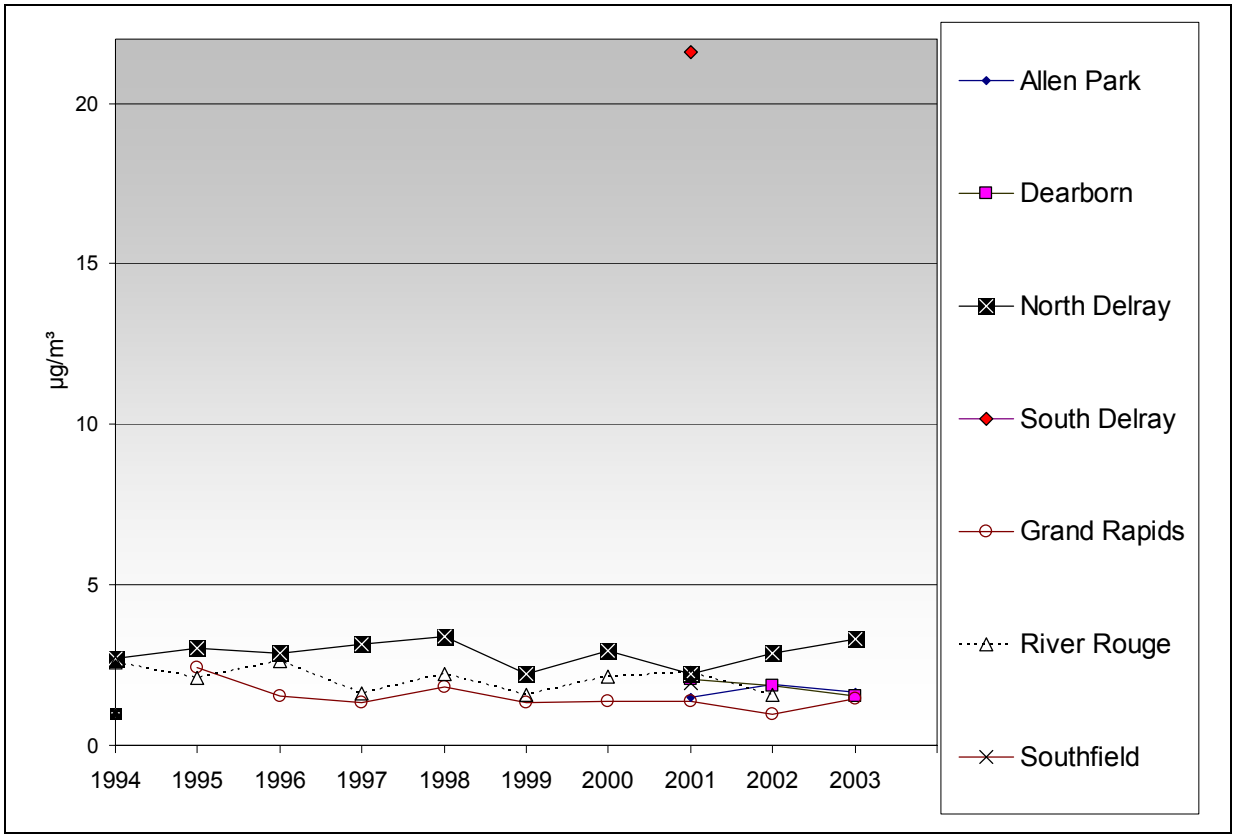


Figure 28: Formaldehyde Annual Average Air Concentration Trends

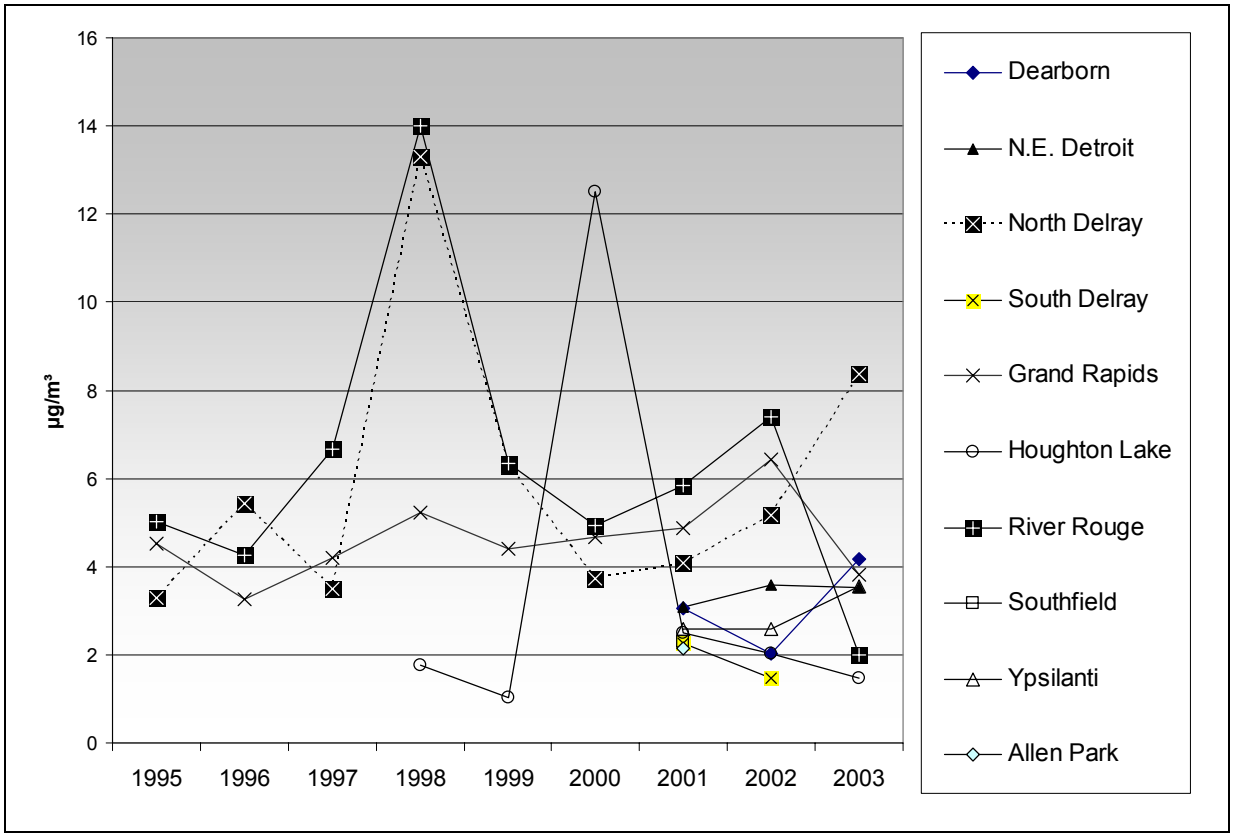
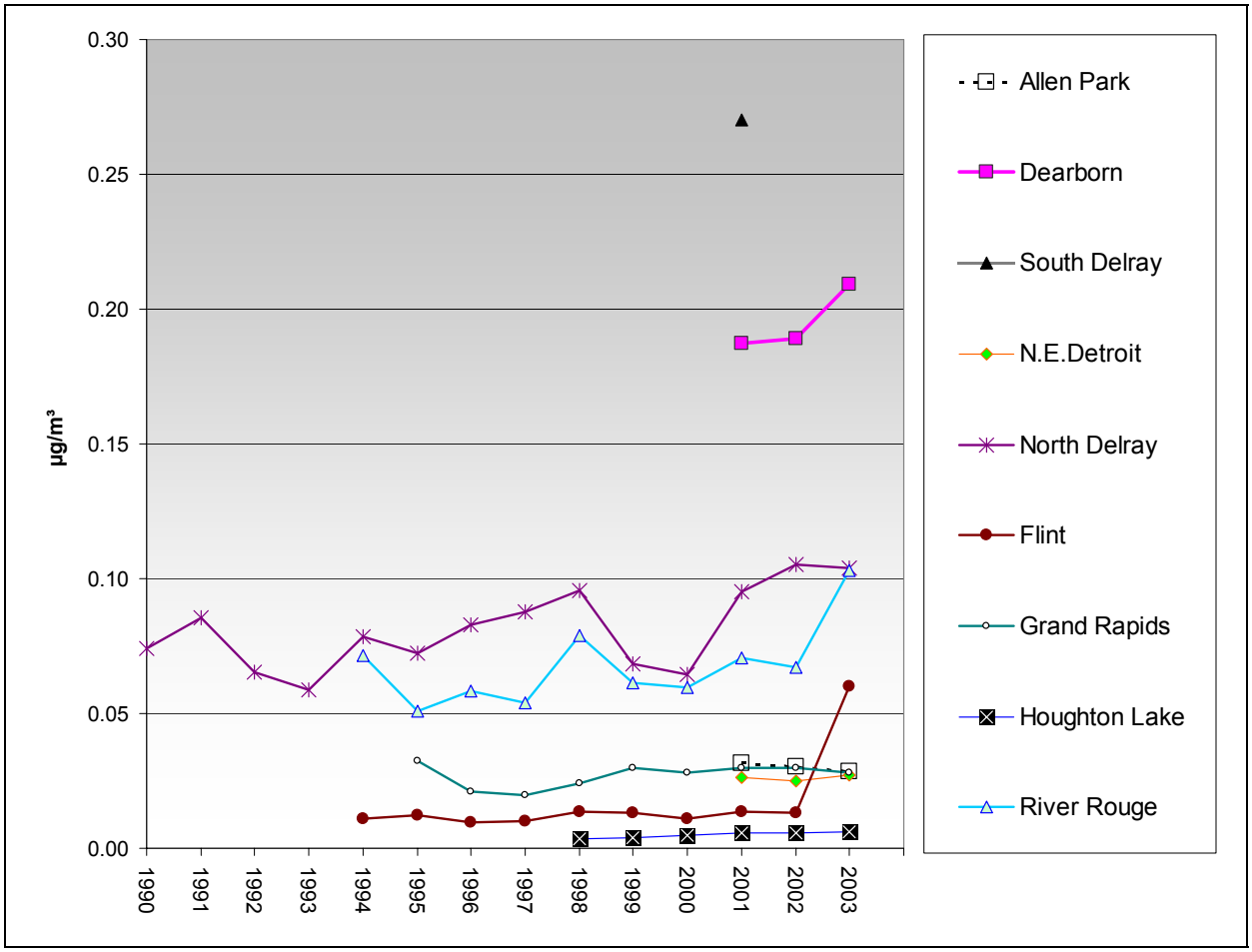


Figure 29: Manganese Annual Average Air Concentration Trends



Manganese Trends in Wayne County (**Figures 30-33**)

Figure 30: Average Manganese TSP Trends (for Wayne County)

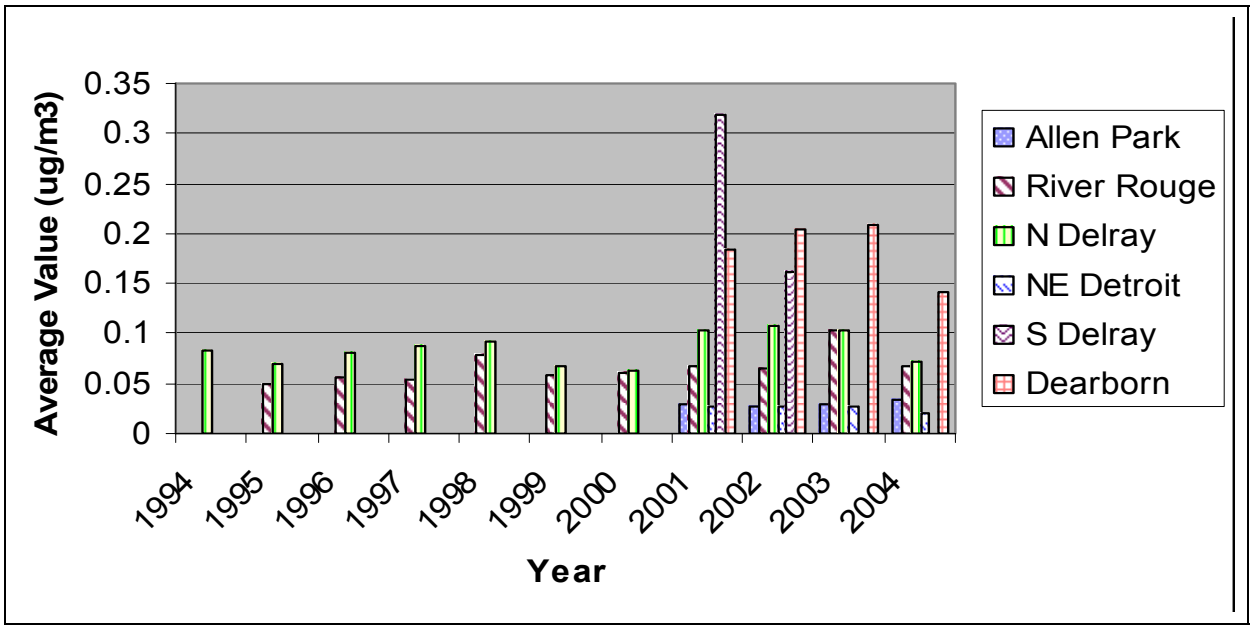
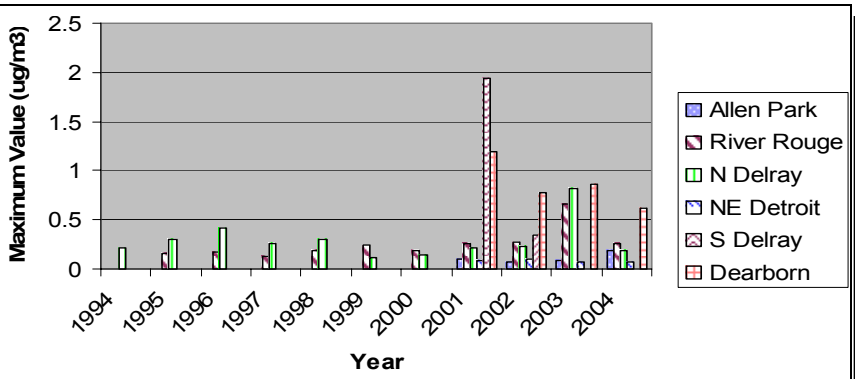


Figure 31: Maximum Manganese TSP Trends (for Wayne County)



Note: The four highest measured concentrations at all sites meet or exceed the RfC of 0.05 ug/m3 (Range = 0.047 to 1.937 ug/m3) except for NE Detroit in 2004.

Figure 32: Average Manganese $PM_{2.5}$ Trends (for Wayne County

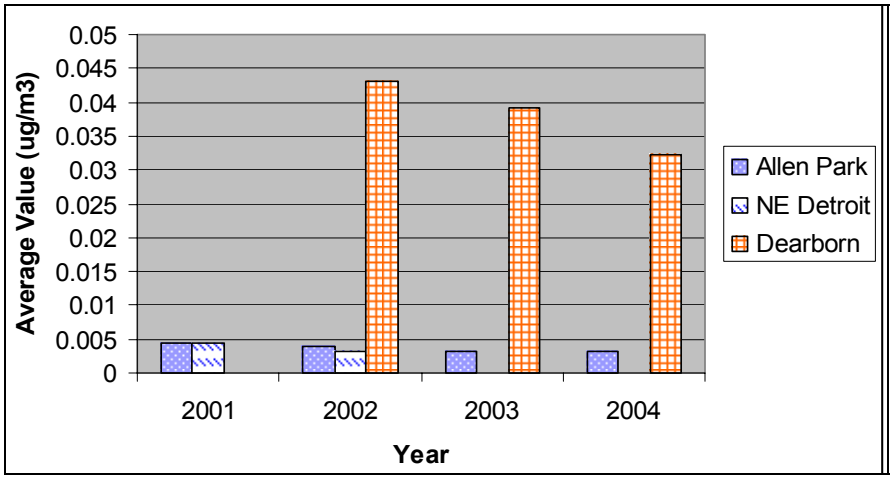
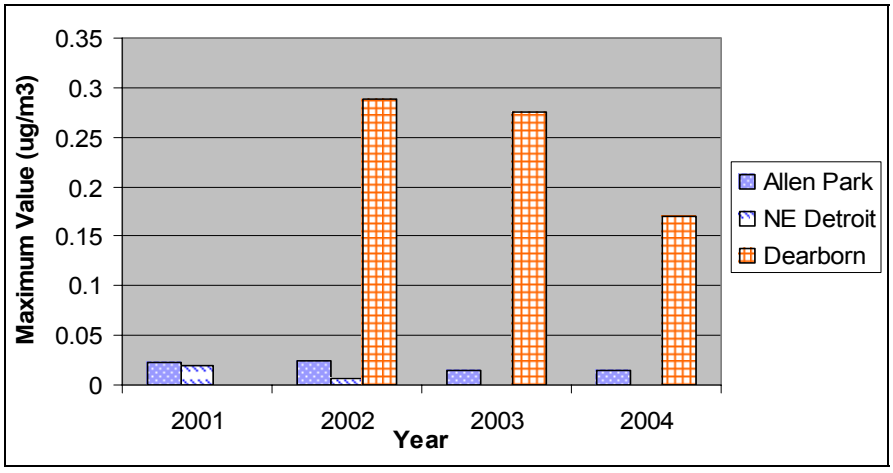


Figure 33: Maximum Manganese $PM_{2.5}$ Trends (for Wayne County)



14.0 CONCLUSIONS

As discussed in **Section 2**, the goals of the DATI were to characterize the levels of air toxics in the Detroit area, to assess the health risks associated with those levels, and to work with a broadly representative stakeholder group to develop reports on the findings and to recommend risk reduction activities. The "Planning, Scoping, and Problem Formulation" step included the listing of specific "key questions" to answer in the risk assessment phase. The conclusions of this report are presented as follows, for each of the key questions:

1. **What substances are the most significant COPCs (cancer, chronic, and acute noncancer) of the air toxics monitored?**

   The ambient air levels of 12 chemicals were associated with increased cancer risks of 1×10^{-6} or higher at one or more monitoring site. These 12 chemicals and their highest estimated risk at any one site are shown in **Table 94**:

Table 94: 12 Chemicals Associated with Increased Cancer Risks

INCREASED LIFETIME CANCER RISK	CHEMICALS
$100 \text{ to } 400 \times 10^{-6}$	methylene chloride* naphthalene* benzene*
$10 \text{ to } 100 \times 10^{-6}$	acrylonitrile formaldehyde 1,4-dichlorobenzene arsenic
$1 \text{ to } 10 \times 10^{-6}$	carbon tetrachloride 1,3-butadiene acetaldehyde cadmium nickel

*Current data show greatly decreased levels of methylene chloride at Allen Park and naphthalene and benzene at S. Delray.

   Two chemicals, manganese and naphthalene, were identified as COPCs with regard to chronic non-cancer effects. Monitored levels of these chemicals exceeded the chronic health protective benchmark value at select sites.

   With regard to acute non-cancer health effects, only methylene chloride had any monitored levels higher than the acute health protective benchmark. However, the vast majority of compounds had no available acute health benchmarks, thereby limiting the risk assessment for acute effects.

2. **What is the magnitude of cancer risk posed by air toxics, and how does it vary by site location?**

   Total additive risks for the 12 carcinogens identified as having an individual risk greater than 1×10^{-6} varied across the sites. The total cancer risk estimates for each site are shown in **Table 95**:

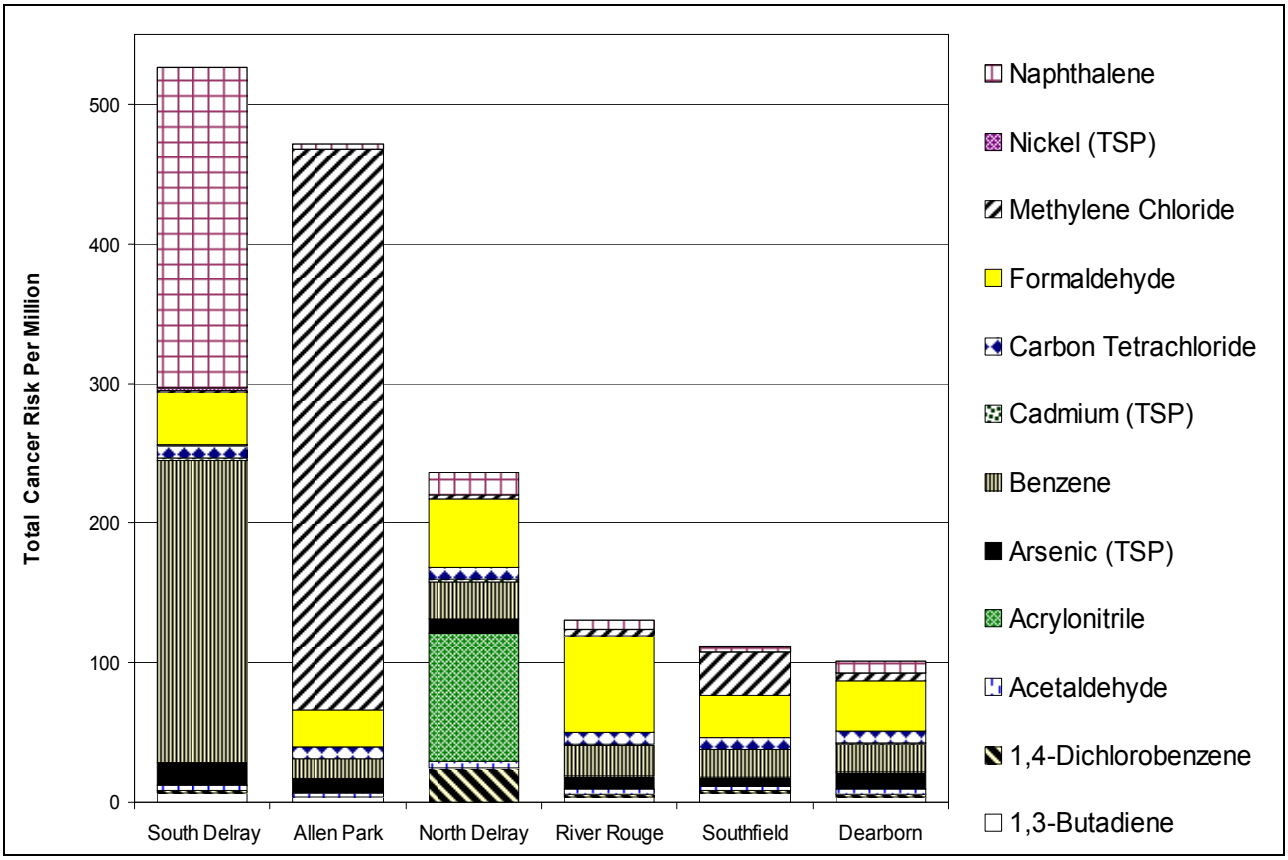
Table 95: Total Cancer Risk Estimates for Each Monitoring Site

MONITORING SITE	TOTAL CANCER RISK (PER MILLION)
S. Delray	525
Allen Park	472
N. Delray	236
River Rouge	130
Southfield	111
Dearborn	101
N.E. Detroit	52*

*The N.E. Detroit site was monitored for a more limited set of air toxics.

The total cancer risk for each site, along with the breakdown by chemicals contributing to that risk is shown below in **Figure 34** which is taken from **Section 9.2**.

Figure 34: Lifetime Cancer Risk Estimates for Risk Drivers at Detroit Area Monitoring Sites*



*The high-end estimated cancer risk is the incremental lifetime (70 years) risk by inhalation to the individual. The estimates of cancer risks should not be viewed as actual cancer cases resulting from air pollution but as upper bound estimates so the agency can prioritize its efforts to reduce exposures. It should also be noted that these risk estimates are based on 2001-2002 monitoring data, and the unusually high levels of methylene chloride at Allen Park and naphthalene and benzene at South Delray during that period have not persisted in subsequent monitoring.

Within the Detroit area, the total risks varied between sites, with about a five fold difference between the highest site and the lowest site. This comparison excludes the N.E. Detroit site since this site monitored for a more limited set of air toxics.

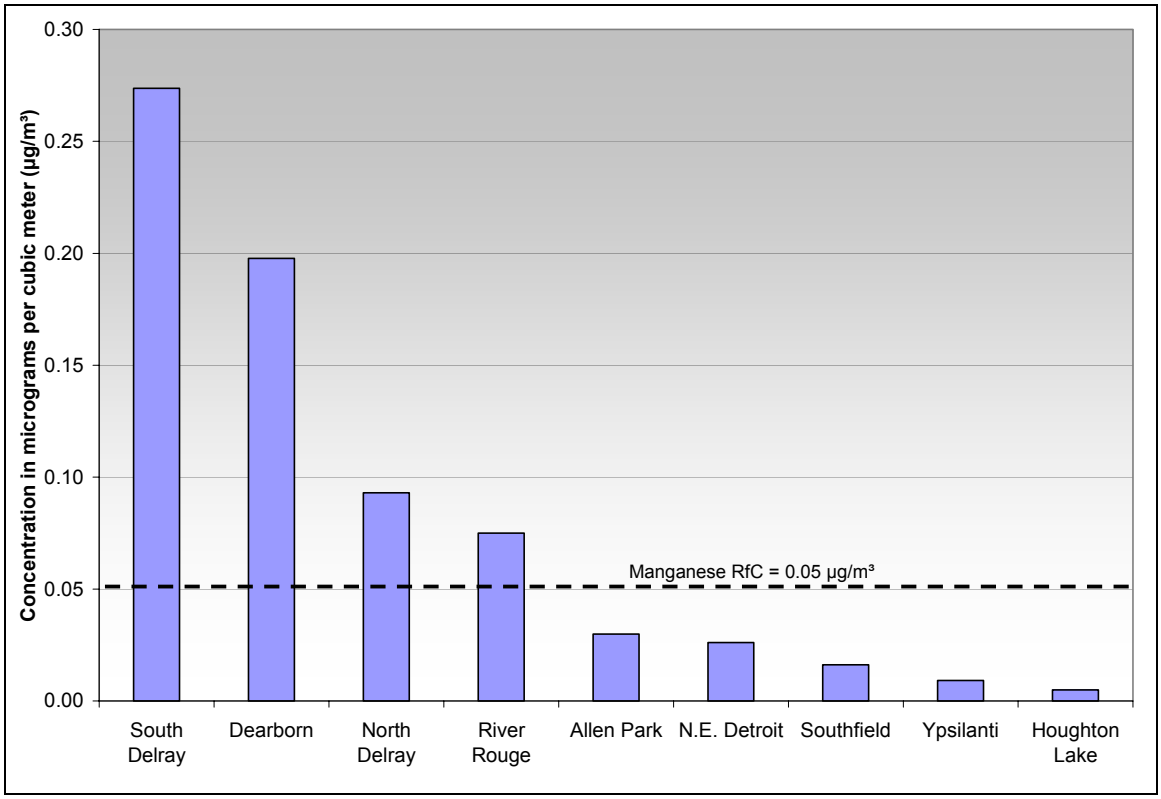
The above estimates and comparisons of cancer risks from air toxics did not include diesel exhaust due to the greater uncertainty associated with risk estimations and the limited number of sites with monitoring data. Diesel exhaust is specifically discussed in **Question 8**.

3. What is the magnitude of chronic noncancer risk posed by air toxics and how does it vary by site location?

Manganese and naphthalene were identified as COPCs for chronic noncancer risks.

As shown in **Figure 35**, annual average manganese concentrations at four Detroit sites (S. Delray, Dearborn, N. Delray, River Rouge) were 1.5 to 5.5 times higher than the chronic health benchmark value (RfC) of 0.05 µg/m^3.

Figure 35: Annual Average Manganese Air Concentrations

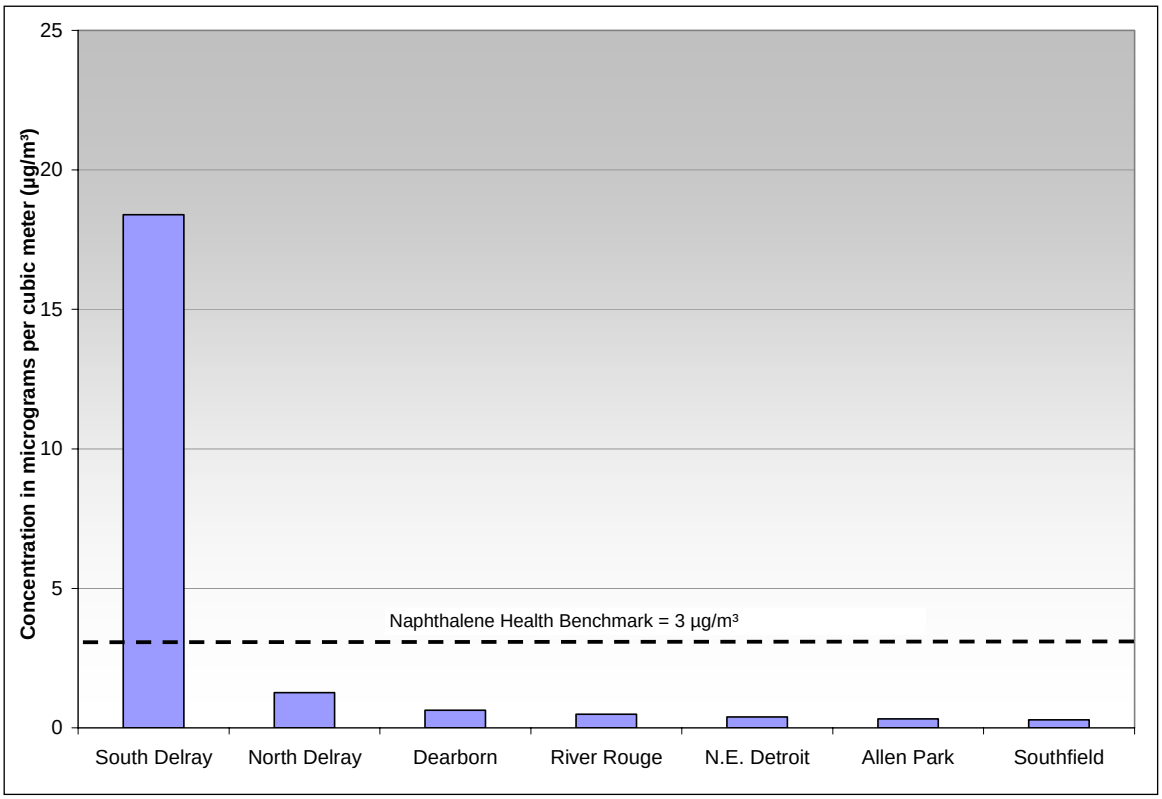


A review of the scientific basis for the RfC and the magnitude of the UFs applied in its derivation was conducted to help characterize the risk of neurotoxicity from chronic manganese exposure above the health benchmark. The monitored levels are below actual exposure concentrations known to cause adverse effects in humans, however, the reduction in the margin of safety built into the RfC by use of UFs is a concern. This concern is elevated further when considering the suggestive evidence in experimental animals for manganese accumulation in the brain, even at relatively low, environmentally relevant

exposures. For sites where historical data are available, annual averages consistently above the benchmark are also a concern. Reduction of ambient manganese levels at impacted sites was identified as a priority for the risk reduction phase of DATI.

The annual average concentration of naphthalene exceeded the chronic health protective benchmark value at only one site. That site was S. Delray, where the monitored level was about six times higher than the health protective benchmark value. **Figure 36** shows the naphthalene levels at each site in comparison to the health protective benchmark value.

Figure 36: Annual Average Naphthalene Air Concentrations



Exposure to naphthalene by inhalation at high enough concentrations causes respiratory effects. As with manganese, the health protective benchmark for naphthalene is set below the level at which adverse health effects are observed in order to provide a margin of safety. The highest naphthalene levels observed in the Detroit area are a cause for concern because they indicate a decrease in this margin of safety, although they are still below the levels at which effects have been observed in scientific studies.

4. What is the magnitude of acute noncancer risk posed by air toxics and how does it vary by site location?

The lack of appropriate acute health benchmarks limits the ability to fully characterize acute risks for Detroit sites. Of the 224 chemicals considered, only about 20% were actually evaluated for acute risk. Other limitations in the assessment include lack of continuous monitoring data at all sites (except Dearborn) and use of two sampling timeframes (1-hr and 24-hr averages). Two chemicals, benzene at S. Delray and methylene chloride at Allen Park, were identified as COPCs. On three separate days, the 24-hr concentration of

methylene chloride at Allen Park exceeded the acute health benchmark value, while on a single day the highest 24-hr benzene concentration at S. Delray was just below the acute health benchmark value. After reviewing the scientific basis for the acute benchmarks and the magnitude of the UFs utilized in their derivation, it was concluded that the hazard for acute effects from benzene at S. Delray was likely to be low and that the hazard for acute effects from methylene chloride at Allen Park warranted further investigation. Review of additional monitoring data at Allen Park, subsequent to the original April 2001 – April 2002 time period, has shown that concentrations of methylene chloride have decreased and are now well below the benchmark value. Therefore, at this time, acute health effects from methylene chloride at the Allen Park site are unlikely.

Naphthalene and manganese were given special consideration for their potential for acute hazard, because they were identified as COPCs for chronic non-cancer hazard. Given the absence of appropriate acute health benchmarks for these chemicals, a surrogate approach using 1% of the TLV was used as a screening level to assess the likelihood of acute hazard. There was no indication that naphthalene poses an increased hazard of acute health effects (irritation) at S. Delray. For manganese, however, the assessment did not dissuade the potential for acute hazard. It was determined that development of an appropriate acute benchmark should be pursued (if adequate data are available) to better assess the potential for acute hazard.

5. Do the levels of the key COPCs vary between sites?

A site-to-site comparison focused upon the annual average levels of the 13 COPCs. Statistical analysis found that overall, most COPCs showed a high degree of spatial variability and the magnitude of the spatial differences varied by compound.

The formaldehyde levels at N. Delray and River Rouge were significantly higher than the other sites. Acetaldehyde levels were relatively homogeneous (within a 4-fold range across all sites), although the level at the highest site (N. Delray) was significantly higher than the levels at the lowest four sites in the Detroit area including Ypsilanti. The methylene chloride level at Allen Park was significantly elevated in comparison to all other sites. The benzene level at S. Delray was significantly higher than at the other sites. Removing the S. Delray benzene data from the dataset resulted in a finding that N. Delray was significantly higher than the other sites. For carbon tetrachloride, the range of levels across the Detroit-area sites appeared to be fairly consistent, but there were statistically significant differences between the three highest sites and the four lowest sites in the Detroit area. The acrylonitrile level at N. Delray was significantly higher than at all other sites. For the trace metals, manganese had the greatest amount of site-to-site variability in annual average levels. The sites with the highest manganese levels, S. Delray and Dearborn, were not significantly different from each other but were significantly higher than the other sites. The highest annual average arsenic level was measured at S. Delray, which was significantly higher than all other sites. The highest annual average cadmium levels were found at N. Delray and S. Delray, which did not differ from each other but which were significantly higher than all other sites. The site with the highest nickel level, S. Delray, was significantly higher than the level at four other Detroit-area sites. The highest annual average level of naphthalene (and several other PAHs) was found at the S. Delray site, which was significantly different from all other sites. These significant differences in concentrations between sites suggest strong influences from local sources.

6. Of the levels of total chromium, how much is Cr^{+6} (the carcinogenic form)?

Hexavalent Chromium (Cr^{+6}) (the carcinogenic form of chromium) and total chromium were monitored at four sites in the Detroit area. Hexavalent chromium was found to constitute only a small fraction (ranging from 1.0% to 2.4%) of the total chromium level. The measured levels of Cr^{+6} posed a lifetime inhalation cancer risk of $< 1 \times 10^{-6}$, therefore it was not found to be a significant component of the total cancer risk due to air toxics in Detroit. However, other Detroit-area sites had somewhat higher levels of total chromium without accompanying data for Cr^{+6}. The measured ratios of Cr^{+6} to total chromium suggest that Cr^{+6} at these other sites may be at levels associated with a lifetime risk of approximately 2×10^{-6} or lower. Also, it is possible that there may be locations with elevated levels of hexavalent chromium due to local emission sources.

7. Do the monitoring data and risk assessment help to scope the potential relative health significance of diesel emissions in the Detroit area?

Since DPM cannot currently be measured directly, measurements of surrogate substances or modeling must be utilized. For the DATI risk assessment, measured EC levels served as a surrogate for estimating DPM. Modeling estimates and monitoring data gathered between 2001 and 2003 were used to provide estimates of DPM concentrations in the Detroit area. Levels of approximately 1-2 $\mu g/m^3$ DPM were estimated for the Allen Park and Southfield sites. Although these estimated values are relatively uncertain, they serve to provide a general sense of the contribution DPM may add to the risk of noncarcinogenic and carcinogenic health effects. Based on these estimates, the HQ was calculated to be less than one (approximately 0.2 for Allen Park and 0.4 for Southfield). These HQs suggest that DPM alone would not cause respiratory tract irritancy, although it could contribute to such effects if present along with other air contaminants. DPM is also recognized as an air contaminant that is likely to be carcinogenic to humans. Increased cancer risk was calculated using the Cal EPA's cancer unit risk factor. This resulted in an estimated increased cancer risk in the range of approximately 300 to 600×10^{-6} associated with the estimated levels at the Detroit sites. These estimates are consistent with USEPA's roughly estimated lifetime cancer risk of 10 to 1000×10^{-6} associated with diesel emissions in the U.S. These estimated ranges of cancer risk are considered to have significant uncertainty. However, they suggest that diesel emissions may be a significant risk driver in the context of the total cancer risks estimated in this report for the other Detroit-area air toxics.

8. How do the monitored levels of COPCs in Detroit compare to levels in other parts of the U.S.?

An effort was made to put the results of the DATI in a national perspective by comparing results with recent monitoring initiatives conducted in other states. Although there are pitfalls in comparing studies utilizing different methodologies to address different key questions, and in comparing results from different geographic regions impacted by various types of industry, such comparisons are useful in identifying COPCs that may be unique to Detroit sites. One general observation was that the Detroit sites tended to be much more heterogeneous than sites in other urban areas. While the prevalence of some global air toxics (e.g. formaldehyde, benzene, carbon tetrachloride) was quite uniform between cities, site-specific concentrations of methylene chloride, manganese and naphthalene in the Detroit area far exceeded concentrations reported from other urban air monitoring initiatives. The magnitude of the differences in concentrations of these chemicals between cities and between sites within Detroit suggests a strong influence from local sources.

9. How do the health risks from air toxics in Detroit compare to other parts of the United States?

The available air toxics risk assessments vary greatly with regard to the approach used to estimate exposure, the substances included, the health risk benchmarks used, and other important methodological factors. These differences hinder attempts to appropriately compare findings across studies. It was concluded that the most appropriate approach was to utilize the USEPA's NATA. The 1999 version of NATA may be available soon, but the present report utilized the most recent NATA (USEPA, 1996) version. That assessment provided hazard and risk estimates from air toxics for every county in the contiguous 48 states, Puerto Rico and the Virgin Islands, utilizing a consistent methodology for estimating emissions, modeling the ambient air impacts and exposures, and estimating health hazards and risk levels.

The cancer risk comparisons provided by NATA (USEPA, 1996) indicate that all counties in the contiguous U.S. had a total air toxics cancer risk estimate of at least 25 X 10^{-6}. Twenty-six counties in the U.S. had air toxics cancer risk estimates that were higher than Wayne County (91 X 10^{-6}), ranging as high as 240 X 10^{-6}. In Michigan, Wayne County was estimated to have the highest risk level (91 X 10^{-6}), followed by Macomb County (83 X 10^{-6}), Oakland County (74 X 10^{-6}), Kent County (57 X 10^{-6}), and Washtenaw County (54 X 10^{-6}). The findings indicate that the cancer risk levels in Michigan urbanized counties were fairly typical of the national scene.

In the evaluation of chronic noncancer hazards, the NATA assessment found that acrolein posed the highest HQs across the U.S. Several other substances (e.g., manganese, acetaldehyde, arsenic, 1,3-butadiene, and formaldehyde) also exceeded a HQ of 1 in some census tracts nationally. Wayne County and its census tracts appear to be fairly comparable to other large urban areas, with regard to the air toxics included in the NATA study.

10. Are there specific sources or activities suggested to be a cause of specific COPCs?

The DATI report presents the results of the monitoring data, the health risks associated with those levels, and site-to-site comparisons. There were some COPCs which had fairly consistent levels across the Detroit-area sites, while some varied greatly. The latter finding suggests that there may be local emission sources of significance. An accompanying report will be developed which will complement the present report by focusing on the air emission sources which may be contributing to the compounds which posed relatively significant, hazard or risk levels.

11. Are there other data which suggest the DATI risk assessment did not identify all COPCs?

Additional data evaluated suggests that acrolein is a COPC that was not included in the monitoring study that forms the basis for the DATI risk assessment. Acrolein was not included in the Detroit Pilot Project due to the lack of an adequate sampling and analytical methodology. However, the USEPA's (1996) NATA found that acrolein posed a national noncancer hazard (defined as compounds with HQ > 1 for more than 25 million persons). This was based on acrolein's ability to cause respiratory irritation. Likewise, the aggregate respiratory HI (TOSHI) for 8 respiratory irritants was dominated by acrolein. The respiratory TOSHI exceeded 1.0 for nearly the entire U.S. population, and exceeded 10 for more than 20 million people. It should be noted that the NATA estimated acrolein levels are

substantially lower than preliminary Detroit ambient air monitoring results (awaiting final validation; USEPA's DEARS). These preliminary monitoring data are associated with a HQ of 100 based on the USEPA RfC.

12. What are some of the important limitations or uncertainties of the DATI findings?

The uncertainties of the risk assessment have been described in detail in Section 10 of this report. In brief, areas of uncertainty exist in all of the primary steps of the risk assessment – monitoring data collection, exposure assessment, toxicity assessment, and risk characterization. Substantial uncertainties are present, despite the application of state-of-the-art monitoring methods, inclusion of the air toxics of highest interest, a well-designed and extensive monitoring protocol, and the use of the best available health protective benchmarks. Although not fully quantitated, several areas of uncertainty have been described with regard to the conservatism or lack of conservatism imparted upon the risk assessment.

Significant limitations of the study include an incomplete set of health benchmark values for many air toxics, and a low detection frequency (<15% of the monitored levels above the MDL) for some substances, which limited the number of chemicals that could be completely assessed for potential health effects. Assessment of potential chronic health effects could only be fully evaluated for about 36% of the chemicals that were measured as part of the monitoring plan for the Detroit Pilot Project, while 32% could not be assessed due to the absence of a health benchmark value and 32% could not be assessed due to a low detection frequency. Of the 32% that could not be assessed due to low detection frequency, 34 of the 60 chemicals were not expected to present a health concern based on a health benchmark value that was greater than the MDL. A more sensitive analytical method was needed to determine the potential health effects for the remaining 26 of these chemicals. Recommendations for priority of development of health benchmark values for the 32% of chemicals lacking such criteria resulted in 9 chemicals identified as high priority, 4 as medium priority, and 47 as low priority based on the frequency of detection. Acute health effects could be assessed for only about 20% of the chemicals due to the lack of an acute health benchmark value for the remainder.

13. Does the risk assessment provide useful information to guide community-level actions which may significantly mitigate air toxics health risks?

The Detroit Pilot Project included monitoring for over 200 chemicals. From this list of chemicals, 13 chemicals were identified as the highest concern (see key question #1), based on the standard risk assessment process described in this report. In addition to these 13 air toxics, this assessment also indicates that diesel exhaust may be an important pollutant to focus on for mitigation of air toxics health risks. Additional data from other monitoring initiatives suggest that acrolein is also an important air toxic to consider.

14. Do the monitoring data and risk assessment provide a sufficient baseline to support future assessments?

The Detroit Pilot Project provided an unprecedented amount of air toxics monitoring data for Detroit and Wayne County. Data for metals and carbonyl compounds were collected for an urban comparison site (Ypsilanti) and a relatively remote site (Houghton Lake). The monitoring was relatively comprehensive, and utilized state-of the-art methods. The DATI risk assessment report is sufficiently extensive and transparent to support future assessments, and also to indicate whether certain follow-up activities may warrant

prioritization. Given the heterogeneity of air toxics concentrations found in the present assessment across Detroit sites, expansion of the monitoring network in future assessments may be warranted.

15. Are there other issues not specifically addressed by the previous key questions?

An assessment of the mercury air monitoring data was conducted at the request of the DATI stakeholders group, because mercury is an environmental contaminant of high concern. The dominant pathway for human exposure is the ingestion of methylmercury in fish after conversion from inorganic forms of mercury and bioaccumulation in the aquatic food chain. Significant risk of adverse health effects from direct inhalation exposure to Hg^0 was not expected. Monitoring for airborne Hg^0 was conducted for one 10-week session during the winter season (2001-2002) and one 5-week session during the summer season (2002) at the Southfield and S. Delray sites. The annual average concentrations at these sites were consistent with reported global background concentrations. The maximum 24-hr and 1-hr average concentrations at both sites were well below the chronic and acute health protective benchmarks, respectively. Therefore, the levels measured do not pose significant health risks from direct inhalation exposure.

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APPENDICIES

Appendix A: Acronyms and Their Definitions

Appendix B: Stakeholder Participants

Appendix C: List of Compounds by Methods and MDLs

Appendix D: Monitoring Data Summary

Appendix E: Cancer Narrative

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Appendix G: TOSHI Assessment

Appendix H: Rank of Cancer Risk

Appendix I: Daily and Hourly Maximum Concentrations Plus Acute Benchmarks

APPENDIX A: Acronyms and Their Definitions

Acronym	Definition
<	less than
≤	less than or equal to
>	more than
≥	more than or equal to
µg	microgram
$\mu g/m^3$	micrograms per cubic meter (1000 µg = 1 mg)
µm	micron
ACGIH	American Conference of Governmental Industrial Hygienists
AEGL	Acute Exposure Guidance Level
AEGL-1	AEGL - reversible/mild effects
AEGL-2	AEGL - serious/irreversible effects
AEGL-3	AEGL - life-threatening effects
AIRS ID	Aerometric Information System Identification
AQD	Air Quality Division
ARB	Air Resource Board (California EPA)
ATSDR	U.S. Agency for Toxics Substances and Disease Registry
AVG	Average
B(a)P	benzo(a)pyrene
CA	California
CA REL	California Reference Exposure Level
CAA	Clean Air Act
Cal EPA	California EPA
CAS	Chemical Abstract Service
CFR	Code of Federal Regulations
CIIT	Chemical Industry Institute of Toxicology
CMB	Chemical Mass Balance (model)
CNS	central nervous system
CO	Colorado
COPCs	Compounds Of Potential Concern
Cr^{+6}	Hexavalent Chromium
DATI	Detroit Air Toxic Initiative
DEARS	Detroit Exposure Aerosol Research Study
DNPH	2,4-diphenylhydrazine-treated
DPM	diesel particulate matter
EC	elemental carbon
ERG	Easter Research Group (laboratory)
ERPG	Emergency Response Planning Guidelines
ERPG-1	ERPG - mild
ERPG-2	ERPG - serious
ERPG-3	ERPG - life-threatening
FERA	Fate, Exposure and Risk Analysis
fg	femtograms
fg/m^3	Femtograms per cubic meter
FR	Federal Register
GA	Georgia
GC	Gas Chromatograph (analytical instrument)
HAP	Hazardous Air Pollutant
HEAST	Health Effects Assessment Summary Tables

APPENDIX A: Acronyms and Their Definitions

Acronym	Definition
HEC	human equivalent concentration
HEI	Health Effects Institute
Hg	Mercury
Hg°	Elemental mercury
Hg(p)	Particulate mercury
HI	hazard index (sum of the hazard quotients for a particular effect grouping)
HQ	hazard quotient (ratio of predicted ambient impact/initial threshold screening level)
hr	hour
IADN	Integrated Atmospheric Deposition Network
ICP/MS	Inductively Coupled Plasma/Mass Spectrometer
IL	Illinois
IMPROVE	Interagency Monitoring of Protected Visual Environments
IN	Indiana
IRIS	Integrated Risk Information System (USEPA database)
IRSL	initial risk screening level
ITSL	initial threshold screening level
IUR	inhalation unit risk
kg	kilogram
LADCO	Lake Michigan Air Director's Consortium
LC_{50}	Lethal Concentration 50% (for inhalation dosing)
LD_{50}	Lethal Dose 50% (for oral dosing)
LOAEL	lowest-observed-adverse-effect level
$LOAEL_{ADJ}$	LOAEL adjusted to continuous exposure duration from an intermittent regimen by hour/day and days/7days
$LOAEL_{HEC}$	LOAEL adjusted for dosimetric differences across species to a HEC
MACT	maximum achievable control technology
MATES II	Multiple Air Toxics Exposure Study II
Max 1	highest concentration monitored during the sampling period
MDL	Method Detection Limit
MDL/2	Method Detection Limit divided by 2
mg	milligrams
mg/m^3	milligrams per cubic meter (1000 mg = 1 gram)
MDEQ	Michigan Department of Environmental Quality
MI	Michigan
MITAMP	Michigan Toxics Air Monitoring Program
MLAPCD	Metro Louisville Air Pollution Control District
MN	Minnesota
MRL	Minimal Risk Level (derived by the ATSDR)
MSA	Metropolitan Statistical Area
N.	North
N. Delray	Detroit's West Fort Street monitoring site (also called "Southwest High School")
N.E. Detroit	Northeast Detroit's East Seven Mile Road monitoring site
NAAQS	National Ambient Air Quality Standard
NATA	National Air Toxics Assessment, USEPA
NATTS	National Air Toxics Trend Site

APPENDIX A: Acronyms and Their Definitions

Acronym	Definition
NCEA	National Center for Environmental Assessment
ND	Non-detect (i.e., lower than the MDL)
NDAMN	National Dioxin Air Monitoring Network
NESHAP	National Emission Standards for Hazardous Air Pollutants
ng/m^3	nanograms per cubic meter
NIH	National Institute of Health (U.S. Dept. of Health & Human Services)
NIOSH	National Institute of Occupational Safety and Health
nm	nanometer
NM	New Mexico
NOAA-CMDL	National Oceanic and Atmospheric Administration - Climate Monitoring and Diagnostics Laboratory
NOAEL	no-observed-adverse-effect level
$NOAEL_{ADJ}$	NOAEL adjusted to continuous exposure duration from an intermittent regimen by hour/day and days/7 days.
$NOAEL_{HEC}$	NOAEL adjusted for dosimetric differences across species to a HEC.
NRC	National Resources Commission
NTP	National Toxicology Program
Num Obs	Number of observations during the sampling period
Obs > MDL	number of observations greater than the method detection limit
OAQPS	Office of Air Quality Planning and Standards (USEPA)
OC	organic carbon
OEHHA	Office of Environmental Health Hazard Assessment
OEL	Occupational Exposure Limit
OH	Ohio
OR	Oregon
ORD	Office of Research and Development (USEPA)
PAH	Polycyclic Aromatic Hydrocarbon
PAMS	Photochemical Assessment Monitoring Stations
PBT	persistent bioaccumulative toxics
PCE	perchloroethylene
PCWP	plywood and composite wood products
PDF	Portable Document Format (Adobe Acrobat)
pg	picogram
pg/m^3	picograms per cubic meter
PM	particulate matter
PM_{10}	particulate matter less than 10 µm in diameter
$PM_{2.5}$	particulate matter less than 2.5 µm in diameter
PMF	Positive Matrix Factorization (model)
ppbV	parts per billion in volume
ppm	parts per million (air concentration)
ppt	parts per trillion
PR	Puerto Rico
REL	Recommended Reference Level
RfC	Reference Concentration
RfD	Reference Dose
RGDR	Regional Gas Dose Ratio
RGM	reactive gaseous mercury
RI	Rhode Island

APPENDIX A: Acronyms and Their Definitions

Acronym	Definition
River Rouge	River Rouge monitoring site location
RTP	Research Triangle Park
S. Delray	Detroit's West Jefferson monitoring site
SC	South Carolina
SCAQMD	South Coast Air Quality Management District
SNMOC	semi-volatile non-methane organic compounds
Southfield	I-696/Lodge Freeway monitoring site location
SQL	sample quantitation limit
St Dev	Standard Deviation
STI	Sonoma Technology Incorporated
SVOC	Semi-volatile organic compound
TCDD	2,3,7,8-tetrachlorodibenzo-*p*-dioxin
TCE	trichloroethylene
TEEL	Temporary Emergency Exposure Limit
TEEL-0	TEEL - NOAEL
TEEL-1	TEEL - mild
TEEL-2	TEEL - serious
TEEL-3	TEEL - life-threatening
TEQ	Toxic equivalent concentration
TLV	Threshold Limit Value (used by the ACGIH)
TN	Tennessee
TOSHI	Target Organ Specific Hazard Index
Total HI	Total Hazardous Index
TSP	Total suspended particulate
TTD	Target Organ Toxicity Dose
TTN	Technology Transfer Network (USEPA internet based information)
TWA	time-weighted average
UATMP	Urban Air Toxics Monitoring Program
UCL	Upper Confidence Limit
UF	uncertainty factor
URE	Unit Risk Estimate
U.S.	United States
USEPA	U.S. Environmental Protection Agency
VOC	volatile organic compound
w/ND	with non-detect
WA	Washington
WV	West Virginia
WWTP	Waste Water Treatment Plant

APPENDIX B: Stakeholder Participants

Community/Environmental Group Representatives

- Katherine Edgren, Community Action Against Asthma, University of Michigan, School of Public Health
- Jeff Gearhart, Ecology Center
- Lisa Goldstein, Southwest Detroit Environmental Vision
- Dana Guyet, People's Community Services
- Kathryn Savoie, Arab Community Center for Economic and Social Services (ACCESS)
- Donele Wilkins, Detroiters Working for Environmental Justice

Government Representatives

- Joyce Hargrove, Detroit Department of Health and Wellness Promotion
- Chuck Hersey (and Elena Berg), Southeast Michigan Council of Governments
- Jaime Julian, USEPA Region 5
- Josephine Powell, Wayne County Department of Environment
- Olga Savic, Chief of Staff for Steve Tobocman, State Representative
- Smita Srivastava, City of Detroit, Department of Environmental Affairs

Industry and Municipal Facility Representatives

- Vimala Anishetty, Ford Motor Company
- Jeff Bruestle, Marathon Ashland Petroleum
- Alan Greenberg (representing the Detroit Regional Chamber), Horizon Environmental
- Karen Kavanaugh, SW Detroit Business Association
- Louise Lieberman, Detroit Water and Sewerage Department (DWSD)
- Mike Rodenberg, Senior Specialist, DTE Energy
- Del Rector (representing DWSD), NTH Consultants, Ltd.
- Jim Volanski, U.S. Steel Corp., Great Lakes Works
- Don Windeler, Severstal North America

Academia

- Dr. Peter Warner, Wayne State University, Department of Occupational and Environmental Science

Michigan Department of Environmental Quality – AQD Representatives

- Ann Chevalier, Laboratory Specialist
- Mike Depa, Toxicologist
- Mary Ann Heindorf, Ph.D., Environmental Quality Specialist
- Anne Kim, Toxicologist
- Jon Lamb, Environmental Quality Analyst
- Mina McLemore, Southeast Michigan District Office
- Margaret Sadoff, Toxicologist
- Robert Sills, Toxicologist Specialist
- Catherine Simon (Chairperson), Toxicologist Manager

APPENDIX C: List of Compounds by Methods and MDLs (µg/m^3)

SVOCs Chemical Name	MDL ERG µg/m^3
1,2,4,5-Tetrachlorobenzene	0.0527
1,2,4-Trichlorobenzene	0.0681
1,2-Dichlorobenzene	0.0548
1,3-Dichlorobenzene	0.06025
1,3-Dintrobenzene	0.038
1,4-Dichlorobenzene	0.0543
1,4-Naphthoquinone	0.029
1-Naphthylamine	0.0271
2,3,4,6-Tetrachlorophenol	0.04835
2,4,5-Trichlorphenol	0.0345
2,4,6-Trichlorophenol	0.04175
2,4-Dichlorophenol	0.07275
2,4-Dimethylphenol	0.0882
2,4-Dinitrophenol	0.05025
2,4-Dinitrotoluene	0.04855
2,6-Dichlorophenol	0.0905
2,6-Dinitrotoluene	0.0469
2-Acetylaminofluorene	0.017
2-Chloronaphthalene	0.0924
2-Chlorophenol	0.04995
2-Methyl Naphthalene	0.0568
2-Methylphenol	0.0472
2-Naphthylamine	0.0511
2-Nitroaniline	0.0615
2-Nitrophenol	0.05035
2-Picoline	0.05575
3&4-Methylphenol	0.042
3,3'-Dichlorobenzidine	0.04475
3,3'-Dimethylbenzidine	0.25
3-Methylcholanthrene	0.032
3-Nitroaniline	0.0435
4,6-Dinitro-2-methylphenol	0.05655
4-Aminobiphenyl	0.048
4-Bromophenyl phenyl ether	0.0565
4-Chloro-3-methylphenol	0.084
4-Chloroaniline	0.08415

SVOCs Chemical Name	MDL ERG µg/m^3
4-Chlorophenyl-phenyl ether	0.03395
4-Dimethylaminoazobenzene	0.022
4-Nitroaniline	0.052
4-Nitrophenol	0.0368
5-Nitro-o-toluidine	0.026
7,12-Dimethylbenz(a)anthracene	0.0952
Acenaphthene	0.0291
Acenaphthylene	0.04435
Acetophenone	0.06935
Aniline	0.081
Anthracene	0.0853
Azobenzene	0.087
Benzidine	0.25
Benzo(a)anthracene	0.04165
Benzo(a)pyrene	0.09085
Benzo(b)fluoranthene	0.0867
Benzo(ghi)perylene	0.07525
Benzo(k)fluoranthene	0.11805
Benzyl alcohol	0.04075
bis (2-Chloroethyl)ether	0.0583
bis(2-Chloroethoxy)methane	0.07015
bis(2-Chloroisopropyl)ether	0.05535
bis(2-Ethylhexyl)phthalate	0.0581
Butyl benzyl phthalate	0.0583
Carbazole	0.062689
Chlorobenzilate	0.016
Chrysene	0.05285
Diallate	0.024
Dibenzo(ah)anthracene	0.07925
Dibenzofuran	0.0464
Diethyl phthalate	0.036
Dimethyl phthalate	0.04575
Di-n-butyl phthalate	0.07005
Di-n-octyl phthalate	0.06595
Dinoseb	0.031
Diphenylamine	0.12695

SVOCs Chemical Name	MDL ERG µg/m^3
Ethyl methanesulfonate	0.147912
Fluoranthene	0.07245
Fluorene	0.05005
Hexachloro-1,3-Butadiene	0.06615
Hexachlorobenzene	0.0706
Hexachlorocyclopentadiene	0.1087
Hexachloroethane	0.02825
Hexachloropropene	0.032
Indeno(123-cd)pyrene	0.0728
Isodrin	0.023
Isophorone	0.11305
Isosafrole	0.029
Methyl methanesulfonate	0.0825
Naphthalene	0.0773
Nitrobenzene	0.1243
N-Nitrosodibutylamine	0.1121
N-Nitrosodiethylamine	0.036
N-Nitrosodimethylamine	0.130736
N-Nitrosodipropylamine	0.1074
N-Nitrosomethylethylamine	0.035
N-Nitrosopiperidine	0.0866
N-Nitrosopyrrolidine	0.037
o-Toluidine	0.038
Pentachlorobenzene	0.0487
Pentachloroethane	0.044
Pentachloronitrobenzene	0.05185
Pentachlorophenol	0.0737
Phenacetin	0.081921
Phenanthrene	0.05145
Phenol	0.1124
Pronamide	0.062873
Pyrene	0.0527
Pyridine	0.059
Safrole	0.029

APPENDIX C: List of Compounds by Methods and MDLs (µg/m³) (Continued)

HEXAVALENT CHROMIUM Chemical Name	MDL ERG µg/m³
Chromium VI (TSP)	0.000013

METALS (TSP) Chemical Name	MDL MDEQ µg/m³
Arsenic (TSP)	0.000016
Beryllium (TSP)	0.00001
Cadmium (TSP)	0.000024
Chromium (TSP)	0.000014
Lead (TSP)	0.00001
Manganese (TSP)	0.00001
Nickel (TSP)	0.00001

CARBONYLS Chemical Name	MDL MDEQ µg/m³	MDL ERG µg/m³
2,5-dimethylbenzaldehyde	0.0176	0.0274
Acetaldehyde	0.0167	0.0252
Acetone	0.0255	0.019
Benzaldehyde	0.0167	0.0174
Butyr/Isobutyraldehyde		0.0147
Crotonaldehyde	0.0225	0.0172
Formaldehyde	0.0098	0.0197
Hexanaldehyde	0.0206	0.0164
Isovaleraldehyde	0.0431	0.0141
m,p-Tolualdehyde	0.0118	
n-Butyraldehyde	0.0206	
o-Tolualdehyde	0.0118	
Propionaldehyde	0.196	0.0166
Tolualdehydes		0.0393
Valeraldehyde	0.0775	0.0106

VOCs Chemical Name	MDL MDEQ µg/m³	MDL ERG
1,1,1-Trichloroethane	0.262	0.327
1,1,2,2-Tetrachloroethane	0.556	0.412
1,1,2-Trichloroethane	0.262	0.327
1,1-Dichloroethane	0.36	0.324
1,1-Dichloroethene	0.182	0.396
1,2,4-Trichlorobenzene	0.601	0.445
1,2,4-Trimethylbenzene	0.305	0.344
1,2-Dibromoethane	0.415	0.615
1,2-Dichlorobenzene	0.397	0.361
1,2-Dichloroethane	0.348	0.243
1,2-Dichloropropane	0.273	0.323
1,3,5-Trimethylbenzene	0.28	0.344
1,3-Butadiene	0.0885	0.155
1,3-Dichlorobenzene	0.319	0.301
1,4-Dichlorobenzene	0.319	0.541
2,2,4-Trimethylpentane	0.0794	
2-Chloro-1,3-Butadiene	0.109	0.362
Acetonitrile	0.873	0.42
Acetylene		0.138
Acrylonitrile	0.847	0.456
Benzene	0.224	0.128
Bromochloromethane		0.635
Bromodichloromethane	0.489	0.402
Bromoform	0.61	0.827
Bromomethane	0.155	0.35
Carbon Tetrachloride	0.239	0.503
Chlorobenzene	0.184	0.276
Chloroethane	0.106	0.211
Chloroform	0.332	0.244
Chloromethane	0.128	0.124
Chloromethyl Benzene	0.393	0.362

VOCs Chemical Name	MDL MDEQ µg/m³	MDL ERG
cis-1,2-Dichloroethene	0.226	0.396
cis-1,3-Dichloropropene	0.245	0.454
Dibromochloromethane	0.503	0.682
Dichlorodiflouromethane	0.237	0.198
Dichlorotetrafluoroethane		0.35
Ethyl Acrylate		0.655
Ethyl Tert-Butyl Ether		0.627
Ethylbenzene	0.36	0.174
Halocarbon 113	0.261	
Halocarbon 114	0.294	
Hexachloro-1,3-Butadiene	0.672	0.64
m/p -Xylene	0.869	0.217
Methyl Ethyl Ketone	5.01	0.442
Methyl Isobutyl Ketone	3.44	0.615
Methyl Methacrylate		0.737
Methyl Tert-Butyl Ether	0.22	0.649
Methylene Chloride	0.799	0.208
n-Hexane	1.76	
n-Octane		0.28
o-xylene	0.187	0.217
Propylene		0.0861
Styrene	0.23	0.298
Tert-Amyl Methyl Ether		0.502
Tetrachloroethene	0.482	0.407
Toluene	0.264	0.226
trans-1,2-Dichloroethene		0.238
trans-1,3-Dichloropropene	0.281	0.499
Trichloroethene	0.204	0.376
Trichloroflouromethane	0.27	0.225
Trichlorotrifluoroethane		0.536
Vinyl Chloride	0.112	0.153

APPENDIX C: List of Compounds by Methods and MDLs (µg/m^3) (Continued)

PAMS COMPOUNDS
1,2,3-Trimethylbenzene
1,2,4-Trimethylbenzene
1,3,5-Trimethylbenzene
1,4-Diethylbenzene
1-Butene
1-Pentene
2,2-Dimethylbutane
2,3,4-Trimethylpentane
2,3-Dimethylbutane
2,3-Dimethylpentane
2,4-Dimethylpentane
2-Methyl-1-Pentene
2-Methyl-2-Pentene
2-Methylheptane
2-Methylhexane
2-Methylpentane
3-Methyl-1,3-Butadiene
3-Methylheptane
3-Methylhexane
3-Methylpentane
Acetylene
Benzene
Butane
Cis-2-Butene
Cis-2-Pentene
Cyclohexane
Cyclopentene
Decane

PAMS COMPOUNDS
Ethane
Ethanol
Ethylbenzene
Heptane
Hexane
Isobutane
Isooctane
Isopentane
Isopropylbenzene
m/p Xylene
m-Diethylbenzene
Methylcyclohexane
Methylcyclopentane
m-Ethyltoluene
Nonane
Octane
o-Ethyltoluene
o-Xylene
Pentane
p-Ethyltoluene
Propane
Propylbenzene
Propylene
Styrene
Toluene
Trans-2-Butene
Trans-2-Pentene
Undecane

APPENDIX D: Monitoring Data Summary by Monitoring Site

ALLEN PARK MONITORING SITE - AIRS ID 261630001

CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
1,1,1-Trichloroethane	0.262	0.327	33	24	72.7%	0.336	0.325	0.273	0.202	0.0588
1,1,2,2-Tetrachloroethane	0.556	0.412	33	0	0.0%	0	0	0	0.206	0
1,1,2-Trichloroethane	0.262	0.327	33	0	0.0%	0	0	0	0.164	0
1,1-Dichloroethane	0.36	0.324	33	0	0.0%	0	0	0	0.162	0
1,1-Dichloroethene	0.182	0.396	33	0	0.0%	0	0	0	0.198	0
1,2,4,5-Tetrachlorobenzene		0.0527	29	0	0.0%	0	0	0	0.0264	0
1,2,4-Trichlorobenzene VOC	0.601	0.445	33	0	0.0%	0	0	0	0.223	0
1,2,4-Trichlorobenzene SVOC		0.0681	29	0	0.0%	0	0	0	0.0341	0
1,2,4-Trimethylbenzene	0.305	0.344	33	32	97.0%	2.47	2.31	1.43	0.838	0.568
1,2-Dibromoethane	0.415	0.615	33	0	0.0%	0	0	0	0.307	0
1,2-Dichlorobenzene VOC	0.397	0.361	33	0	0.0%	0	0	0	0.18	0
1,2-Dichlorobenzene SVOC		0.0548	29	0	0.0%	0	0	0	0.0274	0
1,2-Dichloroethane	0.348	0.243	33	0	0.0%	0	0	0	0.121	0
1,2-Dichloropropane	0.273	0.323	33	0	0.0%	0	0	0	0.162	0
1,3,5-Trimethylbenzene	0.28	0.344	33	26	78.8%	0.983	0.77	0.506	0.293	0.196
1,3-Butadiene	0.0885	0.155	33	22	66.7%	0.364	0.294	0.221	0.12	0.0757
1,3-Dichlorobenzene VOC	0.319	0.301	33	0	0.0%	0	0	0	0.15	0
1,3-Dichlorobenzene SVOC		0.0603	29	0	0.0%	0	0	0	0.0301	0
1,3-Dintrobenzene		0.038	29	0	0.0%	0	0	0	0.019	0
1,4-Dichlorobenzene VOC	0.319	0.541	33	3	9.1%	0.361	0.24	0.06	0.239	0.038
1,4-Dichlorobenzene SVOC		0.0543	29	29	100.0%	0.203	0.162	0.137	0.0574	0.0471
1,4-Naphthoquinone		0.029	29	0	0.0%	0	0	0	0.0145	0
1-Naphthylamine		0.0271	29	0	0.0%	0	0	0	0.0136	0
2,3,4,6-Tetrachlorophenol		0.0484	29	0	0.0%	0	0	0	0.0242	0
2,4,5-Trichlorphenol		0.0345	29	0	0.0%	0	0	0	0.0173	0
2,4,6-Trichlorophenol		0.0418	29	0	0.0%	0	0	0	0.0209	0
2,4-Dichlorophenol		0.0728	29	0	0.0%	0	0	0	0.0364	0
2,4-Dimethylphenol		0.0882	29	0	0.0%	0	0	0	0.0441	0
2,4-Dinitrophenol		0.0503	29	0	0.0%	0	0	0	0.0251	0
2,4-Dinitrotoluene		0.0486	29	0	0.0%	0	0	0	0.0243	0
2,5-dimethylbenzaldehyde	0.0176	0.0274	61	7	11.5%	0.0684	0.0328	0.0291	0.0126	0.00863

ALLEN PARK MONITORING SITE - AIRS ID 261630001										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
2,6-Dichlorophenol		0.0905	29	0	0.0%	0	0	0	0.0453	0
2,6-Dinitrotoluene		0.0469	29	0	0.0%	0	0	0	0.0235	0
2-Acetylaminofluorene		0.017	29	0	0.0%	0	0	0	0.0085	0
2-Chloro-1,3-Butadiene	0.109	0.362	33	0	0.0%	0	0	0	0.181	0
2-Chloronaphthalene		0.0924	29	0	0.0%	0	0	0	0.0462	0
2-Chlorophenol		0.05	29	0	0.0%	0	0	0	0.025	0
2-Methyl Naphthalene		0.0568	29	29	100.0%	0.396	0.35	0.318	0.153	0.0906
2-Methylphenol		0.0472	29	0	0.0%	0	0	0	0.0236	0
2-Naphthylamine		0.0511	29	0	0.0%	0	0	0	0.0256	0
2-Nitroaniline		0.0615	29	0	0.0%	0	0	0	0.0308	0
2-Nitrophenol		0.0504	29	0	0.0%	0	0	0	0.0252	0
2-Picoline		0.0558	29	0	0.0%	0	0	0	0.0279	0
3&4-Methylphenol		0.042	29	1	3.4%	0.0185	0	0	0.0209	0.000467
3,3'-Dichlorobenzidine		0.0448	29	0	0.0%	0	0	0	0.0224	0
3,3'-Dimethylbenzidine		0.25	29	0	0.0%	0	0	0	0.125	0
3-Methylcholanthrene		0.032	29	0	0.0%	0	0	0	0.016	0
3-Nitroaniline		0.0435	29	0	0.0%	0	0	0	0.0218	0
4,6-Dinitro-2-methylphenol		0.0566	29	0	0.0%	0	0	0	0.0283	0
4-Aminobiphenyl		0.048	29	0	0.0%	0	0	0	0.024	0
4-Bromophenyl phenyl ether		0.0565	29	0	0.0%	0	0	0	0.0283	0
4-Chloro-3-methylphenol		0.084	29	0	0.0%	0	0	0	0.042	0
4-Chloroaniline		0.0842	29	0	0.0%	0	0	0	0.0421	0
4-Chlorophenyl-phenyl ether		0.034	29	0	0.0%	0	0	0	0.017	0
4-Dimethylaminoazobenzene		0.022	29	0	0.0%	0	0	0	0.011	0
4-Nitroaniline		0.052	29	0	0.0%	0	0	0	0.026	0
4-Nitrophenol		0.0368	29	0	0.0%	0	0	0	0.0184	0
5-Nitro-o-toluidine		0.026	29	0	0.0%	0	0	0	0.013	0
7,12-Dimethylbenz(a)anthracene		0.0952	29	0	0.0%	0	0	0	0.0476	0
Acenaphthene		0.0291	29	22	75.9%	0.0259	0.0234	0.0196	0.00924	0.00713
Acenaphthylene		0.0444	29	4	13.8%	0.00966	0.00837	0.0055	0.0201	0.00536
Acetaldehyde	0.0167	0.0252	61	59	96.7%	3.57	3.48	3.3	1.54	0.765
Acetone	0.0255	0.019	61	61	100.0%	13.7	7.17	6.79	2.58	1.95

ALLEN PARK MONITORING SITE - AIRS ID 261630001										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
Acetonitrile	0.873	0.42	33	17	51.5%	238	131	106	19.2	48.9
Acetophenone		0.0694	29	0	0.0%	0	0	0	0.0347	0
Acetylene		0.138	33	33	100.0%	2.95	2.71	2.62	1.35	0.692
Acrylonitrile	0.847	0.456	33	0	0.0%	0	0	0	0.228	0
Aniline		0.081	29	0	0.0%	0	0	0	0.0405	0
Anthracene		0.0853	29	0	0.0%	0	0	0	0.0427	0
Arsenic (TSP)	0.000016		71	71	100.0%	0.0148	0.00832	0.00798	0.00189	0.00222
Azobenzene		0.087	29	0	0.0%	0	0	0	0.0435	0
Benzaldehyde	0.0167	0.0174	61	51	83.6%	0.813	0.499	0.412	0.152	0.145
Benzene	0.224	0.128	33	33	100.0%	4.38	2.62	2.24	1.5	0.712
Benzidine		0.25	29	0	0.0%	0	0	0	0.125	0
Benzo(a)anthracene		0.0417	29	0	0.0%	0	0	0	0.0208	0
Benzo(a)pyrene		0.0909	29	0	0.0%	0	0	0	0.0454	0
Benzo(b)fluoranthene		0.0867	29	0	0.0%	0	0	0	0.0434	0
Benzo(ghi)perylene		0.0753	29	0	0.0%	0	0	0	0.0376	0
Benzo(k)fluoranthene		0.118	29	0	0.0%	0	0	0	0.059	0
Benzyl alcohol		0.0408	29	0	0.0%	0	0	0	0.0204	0
Beryllium (TSP)	0.00001		71	71	100.0%	0.000477	0.000477	0.000477	0.0000715	0.0001
bis (2-Chloroethyl)ether		0.0583	29	0	0.0%	0	0	0	0.0292	0
bis(2-Chloroethoxy)methane		0.0702	29	0	0.0%	0	0	0	0.0351	0
bis(2-Chloroisopropyl)ether		0.0554	29	0	0.0%	0	0	0	0.0277	0
bis(2-Ethylhexyl)phthalate		0.0581	29	3	10.3%	0.016	0.0111	0.00578	0.0272	0.00577
Bromochloromethane		0.635	33	0	0.0%	0	0	0	0.318	0
Bromodichloromethane	0.489	0.402	33	0	0.0%	0	0	0	0.201	0
Bromoform	0.61	0.827	33	0	0.0%	0	0	0	0.414	0
Bromomethane	0.155	0.35	33	2	6.1%	0.526	0.103	0	0.183	0.0628
Butyl benzyl phthalate		0.0583	29	0	0.0%	0	0	0	0.0292	0
Butyr/Isobutyraldehyde		0.0147	31	31	100.0%	0.775	0.743	0.718	0.456	0.169
Cadmium (TSP)	0.000024		71	71	100.0%	0.00142	0.00106	0.000826	0.000404	0.000214
Carbazole		0.0627	29	0	0.0%	0	0	0	0.0313	0
Carbon Tetrachloride	0.239	0.503	33	33	100.0%	0.881	0.762	0.717	0.597	0.11
Chlorobenzene	0.184	0.276	33	0	0.0%	0	0	0	0.138	0

ALLEN PARK MONITORING SITE - AIRS ID 261630001										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
Chlorobenzilate		0.016	29	0	0.0%	0	0	0	0.008	0
Chloroethane	0.106	0.211	33	6	18.2%	0.635	0.401	0.385	0.157	0.123
Chloroform	0.332	0.244	33	3	9.1%	0.322	0.224	0.115	0.131	0.0387
Chloromethane	0.128	0.124	33	33	100.0%	1.8	1.8	1.65	1.27	0.222
Chloromethyl Benzene	0.393	0.362	33	0	0.0%	0	0	0	0.181	0
Chromium (TSP)	0.000014		71	71	100.0%	0.00691	0.00547	0.00537	0.0035	0.000887
Chromium VI (TSP)		0.000013	33	19	57.6%	0.000163	0.000144	0.000112	0.0000444	0.0000448
Chrysene		0.0529	29	0	0.0%	0	0	0	0.0264	0
cis-1,2-Dichloroethene	0.226	0.396	33	0	0.0%	0	0	0	0.198	0
cis-1,3-Dichloropropene	0.245	0.454	33	0	0.0%	0	0	0	0.227	0
Crotonaldehyde	0.0225	0.0172	61	30	49.2%	0.745	0.29	0.262	0.0455	0.109
Diallate		0.024	29	0	0.0%	0	0	0	0.012	0
Dibenzo(ah)anthracene		0.0793	29	0	0.0%	0	0	0	0.0396	0
Dibenzofuran		0.0464	29	24	82.8%	0.0342	0.0268	0.0202	0.0125	0.00926
Dibromochloromethane	0.503	0.682	33	0	0.0%	0	0	0	0.341	0
Dichlorodiflouromethane	0.237	0.198	33	33	100.0%	3.71	3.6	3.55	2.93	0.449
Dichlorotetrafluoroethane		0.35	33	3	9.1%	0.129	0.107	0.0699	0.168	0.0225
Diethyl phthalate		0.036	29	1	3.4%	0.0131	0	0	0.0178	0.000912
Dimethyl phthalate		0.0458	29	0	0.0%	0	0	0	0.0229	0
Di-n-butyl phthalate		0.0701	29	2	6.9%	0.0207	0.00832	0	0.0336	0.00554
Di-n-octyl phthalate		0.066	29	0	0.0%	0	0	0	0.033	0
Dinoseb		0.031	29	0	0.0%	0	0	0	0.0155	0
Diphenylamine		0.127	29	0	0.0%	0	0	0	0.0635	0
Ethyl Acrylate		0.655	33	0	0.0%	0	0	0	0.328	0
Ethyl methanesulfonate		0.148	29	0	0.0%	0	0	0	0.074	0
Ethyl Tert-Butyl Ether		0.627	33	0	0.0%	0	0	0	0.314	0
Ethylbenzene	0.36	0.174	33	31	93.9%	31.2	2.95	1.77	1.6	5.35
Fluoranthene		0.0725	29	2	6.9%	0.00509	0.00281	0	0.034	0.00833
Fluorene		0.0501	29	27	93.1%	0.0313	0.0211	0.0209	0.0102	0.00832
Formaldehyde	0.0098	0.0197	61	60	98.4%	4.84	4.73	4.72	2.12	1.15
Hexachloro-1,3-Butadiene VOC	0.672	0.64	33	0	0.0%	0	0	0	0.32	0
Hexachloro-1,3-Butadiene SVOC		0.0662	29	0	0.0%	0	0	0	0.0331	0

ALLEN PARK MONITORING SITE - AIRS ID 261630001										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
Hexachlorobenzene		0.0706	29	0	0.0%	0	0	0	0.0353	0
Hexachlorocyclopentadiene		0.109	29	0	0.0%	0	0	0	0.0544	0
Hexachloroethane		0.0283	29	0	0.0%	0	0	0	0.0141	0
Hexachloropropene		0.032	29	0	0.0%	0	0	0	0.016	0
Hexanaldehyde	0.0206	0.0164	61	46	75.4%	1.03	0.861	0.779	0.177	0.219
Indeno(123-cd)pyrene		0.0728	29	0	0.0%	0	0	0	0.0364	0
Isodrin		0.023	29	0	0.0%	0	0	0	0.0115	0
Isophorone		0.113	29	0	0.0%	0	0	0	0.0565	0
Isosafrole		0.029	29	0	0.0%	0	0	0	0.0145	0
Isovaleraldehyde	0.0431	0.0141	61	20	32.8%	0.359	0.317	0.204	0.0434	0.071
Lead (TSP)	0.00001		71	71	100.0%	1.02	0.0412	0.0227	0.0248	0.12
m,p-Tolualdehyde	0.0118		30	7	23.3%	0.801	0.27	0.197	0.0587	0.155
m/p -Xylene	0.869	0.217	33	33	100.0%	122	9.77	5.17	5.55	21
Manganese (TSP)	0.00001		71	71	100.0%	0.107	0.0643	0.06	0.0299	0.0156
Methyl Ethyl Ketone	5.01	0.442	33	26	78.8%	12.8	6.55	3.69	2.28	2.31
Methyl Isobutyl Ketone	3.44	0.615	33	0	0.0%	0	0	0	0.307	0
Methyl Methacrylate		0.737	33	0	0.0%	0	0	0	0.369	0
Methyl methanesulfonate		0.0825	29	0	0.0%	0	0	0	0.0413	0
Methyl Tert-Butyl Ether	0.22	0.649	33	1	3.0%	0.269	0	0	0.323	0.00967
Methylene Chloride	0.799	0.208	33	33	100.0%	11600	4480	4200	803	2210
Naphthalene		0.0773	29	29	100.0%	0.796	0.737	0.721	0.316	0.202
n-Butyraldehyde	0.0206		30	24	80.0%	0.51	0.413	0.401	0.138	0.126
Nickel (TSP)	0.00001		71	71	100.0%	0.00704	0.00598	0.00527	0.00224	0.00111
Nitrobenzene		0.124	29	0	0.0%	0	0	0	0.0622	0
N-Nitrosodibutylamine		0.112	29	0	0.0%	0	0	0	0.0561	0
N-Nitrosodiethylamine		0.036	29	0	0.0%	0	0	0	0.018	0
N-Nitrosodimethylamine		0.131	29	0	0.0%	0	0	0	0.0654	0
N-Nitrosodipropylamine		0.107	29	0	0.0%	0	0	0	0.0537	0
N-Nitrosomethylethylamine		0.035	29	0	0.0%	0	0	0	0.0175	0
N-Nitrosopiperidine		0.0866	29	0	0.0%	0	0	0	0.0433	0
N-Nitrosopyrrolidine		0.037	29	0	0.0%	0	0	0	0.0185	0
n-Octane		0.28	33	19	57.6%	2.25	1.31	1.13	0.384	0.449

ALLEN PARK MONITORING SITE - AIRS ID 261630001										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
o-Tolualdehyde	0.0118		30	0	0.0%	0	0	0	0.00588	0
o-Toluidine		0.038	29	0	0.0%	0	0	0	0.019	0
o-xylene	0.187	0.217	33	33	100.0%	23.3	3.08	2.24	1.48	3.96
Pentachlorobenzene		0.0487	29	0	0.0%	0	0	0	0.0244	0
Pentachloroethane		0.044	29	0	0.0%	0	0	0	0.022	0
Pentachloronitrobenzene		0.0519	29	0	0.0%	0	0	0	0.0259	0
Pentachlorophenol		0.0737	29	0	0.0%	0	0	0	0.0369	0
Phenacetin		0.0819	29	0	0.0%	0	0	0	0.041	0
Phenanthrene		0.0515	29	27	93.1%	0.0663	0.0567	0.0514	0.0209	0.0165
Phenol		0.112	29	4	13.8%	0.106	0.0936	0.0821	0.06	0.0122
Pronamide		0.0629	29	0	0.0%	0	0	0	0.0314	0
Propionaldehyde	0.196	0.0166	61	51	83.6%	0.76	0.759	0.631	0.255	0.16
Propylene		0.0861	33	33	100.0%	3.15	2.67	2.05	0.958	0.652
Pyrene		0.0527	29	2	6.9%	0.00476	0.00299	0	0.0248	0.0058
Pyridine		0.059	29	0	0.0%	0	0	0	0.0295	0
Safrole		0.029	29	0	0.0%	0	0	0	0.0145	0
Styrene	0.23	0.298	33	18	54.5%	0.545	0.304	0.236	0.167	0.0796
Tert-Amyl Methyl Ether		0.502	33	0	0.0%	0	0	0	0.251	0
Tetrachloroethene	0.482	0.407	33	16	48.5%	2.16	1.62	1.15	0.458	0.456
Tolualdehydes		0.0393	31	29	93.5%	0.472	0.413	0.323	0.163	0.108
Toluene	0.264	0.226	33	33	100.0%	11.5	10.7	6.85	3.57	2.37
trans-1,2-Dichloroethene		0.238	33	1	3.0%	0.399	0	0	0.127	0.0487
trans-1,3-Dichloropropene	0.281	0.499	33	0	0.0%	0	0	0	0.25	0
Trichloroethene	0.204	0.376	33	2	6.1%	0.479	0.249	0	0.199	0.0515
Trichloroflouromethane	0.27	0.225	33	33	100.0%	4.33	3.85	3.15	1.9	0.744
Trichlorotrifluoroethane		0.536	33	33	100.0%	1.24	1.17	1.08	0.833	0.177
Valeraldehyde	0.0775	0.0106	61	38	62.3%	0.255	0.19	0.165	0.0776	0.0457
Vinyl Chloride	0.112	0.153	33	0	0.0%	0	0	0	0.0767	0

DEARBORN MONITORING SITE - AIR ID 261630033										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
1,1,1-Trichloroethane	0.262	0.327	342	216	63.2%	0.913	0.818	0.712	0.199	0.0854
1,1,2,2-Tetrachloroethane	0.556	0.412	342	0	0.0%	0	0	0	0.218	0.0297
1,1,2-Trichloroethane	0.262	0.327	342	0	0.0%	0	0	0	0.158	0.015
1,1-Dichloroethane	0.36	0.324	342	0	0.0%	0	0	0	0.165	0.0113
1,1-Dichloroethene	0.182	0.396	342	0	0.0%	0	0	0	0.179	0.0416
1,2,4,5-Tetrachlorobenzene		0.0527	49	0	0.0%	0	0	0	0.0264	0
1,2,4-Trichlorobenzene VOC	0.601	0.445	342	1	0.3%	0.818	0	0	0.237	0.0449
1,2,4-Trichlorobenzene SVOC		0.0681	49	0	0.0%	0	0	0	0.0341	0
1,2,4-Trimethylbenzene	0.305	0.344	342	328	95.9%	5.06	3.95	3.65	1.02	0.662
1,2-Dibromoethane	0.415	0.615	342	0	0.0%	0	0	0	0.289	0.0409
1,2-Dichlorobenzene VOC	0.397	0.361	342	1	0.3%	0.513	0	0	0.184	0.0215
1,2-Dichlorobenzene SVOC		0.0548	49	0	0.0%	0	0	0	0.0274	0
1,2-Dichloroethane	0.348	0.243	342	0	0.0%	0	0	0	0.13	0.0211
1,2-Dichloropropane	0.273	0.323	342	0	0.0%	0	0	0	0.157	0.0128
1,3,5-Trimethylbenzene	0.28	0.344	342	251	73.4%	1.54	1.33	1.11	0.325	0.21
1,3-Butadiene	0.0885	0.155	342	191	55.8%	0.647	0.615	0.582	0.118	0.0961
1,3-Dichlorobenzene VOC	0.319	0.301	342	1	0.3%	0.473	0	0	0.128	0.025
1,3-Dichlorobenzene SVOC		0.0603	49		0.0%	0	0	0	0.0301	0
1,3-Dintrobenzene		0.038	49	0	0.0%	0	0	0	0.019	0
1,4-Dichlorobenzene VOC	0.319	0.541	342	54	15.5%	0.842	0.661	0.661	0.244	0.082
1,4-Dichlorobenzene SVOC		0.0543	49	47	96.0%	0.349	0.298	0.295	0.0866	0.0762
1,4-Naphthoquinone		0.029	49	0	0.0%	0	0	0	0.0145	0
1-Naphthylamine		0.0271	49	0	0.0%	0	0	0	0.0136	0
2,2,4-Trimethylpentane	0.0794		59	57	96.6%	2.44	1.35	1.26	0.546	0.394
2,3,4,6-Tetrachlorophenol		0.0484	49	0	0.0%	0	0	0	0.0242	0
2,4,5-Trichlorphenol		0.0345	49	0	0.0%	0	0	0	0.0173	0
2,4,6-Trichlorophenol		0.0418	49	0	0.0%	0	0	0	0.0209	0
2,4-Dichlorophenol		0.0728	49	0	0.0%	0	0	0	0.0364	0
2,4-Dimethylphenol		0.0882	49	0	0.0%	0	0	0	0.0441	0
2,4-Dinitrophenol		0.0503	49	0	0.0%	0	0	0	0.0251	0
2,4-Dinitrotoluene		0.0486	49	0	0.0%	0	0	0	0.0243	0
2,5-dimethylbenzaldehyde	0.0176	0.0274	324	102	31.5%	1.53	1.15	0.445	0.0448	0.12

DEARBORN MONITORING SITE - AIR ID 261630033										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
2,6-Dichlorophenol		0.0905	49	0	0.0%	0	0	0	0.0453	0
2,6-Dinitrotoluene		0.0469	49	0	0.0%	0	0	0	0.0235	0
2-Acetylaminofluorene		0.017	49	0	0.0%	0	0	0	0.0085	0
2-Chloro-1,3-Butadiene	0.109	0.362	342	0	0.0%	0	0	0	0.159	0.0487
2-Chloronaphthalene		0.0924	49	0	0.0%	0	0	0	0.0462	0
2-Chlorophenol		0.05	49	0	0.0%	0	0	0	0.025	0
2-Methyl Naphthalene		0.0568	49	49	100.0%	0.518	0.517	0.47	0.224	0.113
2-Methylphenol		0.0472	49	2	4.1%	0.0341	0.00951	0	0.0235	0.00253
2-Naphthylamine		0.0511	49	0	0.0%	0	0	0	0.0256	0
2-Nitroaniline		0.0615	49	0	0.0%	0	0	0	0.0308	0
2-Nitrophenol		0.0504	49	0	0.0%	0	0	0	0.0252	0
2-Picoline		0.0558	49	0	0.0%	0	0	0	0.0279	0
3&4-Methylphenol		0.042	49	5	10.2%	0.0405	0.015	0.0144	0.0208	0.00363
3,3'-Dichlorobenzidine		0.0448	49	0	0.0%	0	0	0	0.0224	0
3,3'-Dimethylbenzidine		0.25	49	0	0.0%	0	0	0	0.125	0
3-Methylcholanthrene		0.032	49	0	0.0%	0	0	0	0.016	0
3-Nitroaniline		0.0435	49	0	0.0%	0	0	0	0.0218	0
4,6-Dinitro-2-methylphenol		0.0566	49	0	0.0%	0	0	0	0.0283	0
4-Aminobiphenyl		0.048	49	0	0.0%	0	0	0	0.024	0
4-Bromophenyl phenyl ether		0.0565	49	0	0.0%	0	0	0	0.0283	0
4-Chloro-3-methylphenol		0.084	49	0	0.0%	0	0	0	0.042	0
4-Chloroaniline		0.0842	49	0	0.0%	0	0	0	0.0421	0
4-Chlorophenyl-phenyl ether		0.034	49	0	0.0%	0	0	0	0.017	0
4-Dimethylaminoazobenzene		0.022	49	0	0.0%	0	0	0	0.011	0
4-Nitroaniline		0.052	49	0	0.0%	0	0	0	0.026	0
4-Nitrophenol		0.0368	49	0	0.0%	0	0	0	0.0184	0
5-Nitro-o-toluidine		0.026	49	0	0.0%	0	0	0	0.013	0
7,12-Dimethylbenz(a)anthracene		0.0952	49	0	0.0%	0	0	0	0.0476	0
Acenaphthene		0.0291	49	47	95.9%	0.163	0.161	0.107	0.033	0.0356
Acenaphthylene		0.0444	49	19	38.8%	0.0159	0.0134	0.0113	0.0165	0.00755
Acetaldehyde	0.0167	0.0252	324	318	98.1%	12.1	9.16	7.94	2.09	1.86
Acetone	0.0255	0.019	324	321	99.1%	13.7	12.4	12.4	3.36	2.93

DEARBORN MONITORING SITE - AIR ID 261630033										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
Acetonitrile	0.873	0.42	342	142	41.5%	172	58.6	36.3	2.93	10.7
Acetophenone		0.0694	49	0	0.0%	0	0	0	0.0347	0
Acetylene		0.138	283	281	99.3%	6.9	6.03	4.98	1.78	0.83
Acrylonitrile	0.847	0.456	342	2	0.6%	0.144	0.128	0	0.26	0.0758
Aniline		0.081	49	0	0.0%	0	0	0	0.0405	0
Anthracene		0.0853	49	7	14.3%	0.0107	0.00604	0.00497	0.0372	0.0135
Arsenic (TSP)	0.000016		70	70	100.0%	0.0101	0.00827	0.00714	0.00241	0.00177
Azobenzene		0.087	49	0	0.0%	0	0	0	0.0435	0
Benzaldehyde	0.0167	0.0174	324	306	94.4%	2.21	1.83	1.56	0.21	0.227
Benzene	0.224	0.128	342	340	99.4%	7.97	6.94	6.23	1.93	1.06
Benzidine		0.25	49	0	0.0%	0	0	0	0.125	0
Benzo(a)anthracene		0.0417	49	0	0.0%	0	0	0	0.0208	0
Benzo(a)pyrene		0.0909	49	0	0.0%	0	0	0	0.0454	0
Benzo(b)fluoranthene		0.0867	49	0	0.0%	0	0	0	0.0434	0
Benzo(ghi)perylene		0.0753	49	0	0.0%	0	0	0	0.0376	0
Benzo(k)fluoranthene		0.118	49	0	0.0%	0	0	0	0.059	0
Benzyl alcohol		0.0408	49	0	0.0%	0	0	0	0.0204	0
Beryllium (TSP)	0.00001		70	70	100.0%	0.00047	0.000304	0.000291	0.000122	7.79E-05
bis (2-Chloroethyl)ether		0.0583	49	0	0.0%	0	0	0	0.0292	0
bis(2-Chloroethoxy)methane		0.0702	49	0	0.0%	0	0	0	0.0351	0
bis(2-Chloroisopropyl)ether		0.0554	49	0	0.0%	0	0	0	0.0277	0
bis(2-Ethylhexyl)phthalate		0.0581	49	9	18.4%	0.0307	0.0157	0.0121	0.0258	0.00755
Bromochloromethane		0.635	283	0	0.0%	0	0	0	0.316	0.0189
Bromodichloromethane	0.489	0.402	342	0	0.0%	0	0	0	0.208	0.02
Bromoform	0.61	0.827	342	0	0.0%	0	0	0	0.394	0.0463
Bromomethane	0.155	0.35	342	6	1.8%	0.356	0.249	0.18	0.159	0.039
Butyl benzyl phthalate		0.0583	49	0	0.0%	0	0	0	0.0292	0
Butyr/Isobutyraldehyde		0.0147	269	267	99.3%	2.36	2.34	2.34	0.607	0.513
Cadmium (TSP)	0.000024		70	70	100.0%	0.00213	0.00179	0.00156	0.000606	0.000369
Carbazole		0.0627	49	0	0.0%	0	0	0	0.0313	0
Carbon Tetrachloride	0.239	0.503	342	339	99.1%	1.07	1.06	1.01	0.633	0.117
Chlorobenzene	0.184	0.276	342	1	0.3%	0.226	0	0	0.13	0.0194

DEARBORN MONITORING SITE - AIR ID 261630033										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
Chlorobenzilate		0.016	49	0	0.0%	0	0	0	0.008	0
Chloroethane	0.106	0.211	342	3	0.9%	1.65	0.81	0.731	0.105	0.101
Chloroform	0.332	0.244	342	39	11.4%	0.318	0.296	0.275	0.135	0.0309
Chloromethane	0.128	0.124	342	341	99.7%	2.94	2.04	2.03	1.24	0.211
Chloromethyl Benzene	0.393	0.362	342	0	0.0%	0	0	0	0.183	0.0116
Chromium (TSP)	0.000014		70	70	100.0%	0.0145	0.0109	0.0108	0.00602	0.00238
Chromium VI (TSP)		0.000013	33	18	54.5%	0.000162	0.000116	0.000111	3.63E-05	4.14E-05
Chrysene		0.0529	49	0	0.0%	0	0	0	0.0264	0
cis-1,2-Dichloroethene	0.226	0.396	342	1	0.3%	1.47	0	0	0.187	0.0772
cis-1,3-Dichloropropene	0.245	0.454	342	0	0.0%	0	0	0	0.208	0.0411
Crotonaldehyde	0.0225	0.0172	324	242	74.7%	0.886	0.819	0.81	0.0723	0.138
Diallate		0.024	49	0	0.0%	0	0	0	0.012	0
Dibenzo(ah)anthracene		0.0793	49	0	0.0%	0	0	0	0.0396	0
Dibenzofuran		0.0464	49	47	95.9%	0.139	0.122	0.112	0.0299	0.0289
Dibromochloromethane	0.503	0.682	342	0	0.0%	0	0	0	0.324	0.0381
Dichlorodiflouromethane	0.237	0.198	342	341	99.7%	5.34	4.67	4.66	3.07	0.463
Dichlorotetrafluoroethane		0.35	283	26	9.2%	0.214	0.145	0.137	0.167	0.0272
Diethyl phthalate		0.036	49	7	14.3%	0.131	0.0578	0.0566	0.0226	0.0182
Dimethyl phthalate		0.0458	49	0	0.0%	0	0	0	0.0229	0
Di-n-butyl phthalate		0.0701	49	42	85.7%	1.22	0.873	0.777	0.239	0.27
Di-n-octyl phthalate		0.066	49	0	0.0%	0	0	0	0.033	0
Dinoseb		0.031	49	0	0.0%	0	0	0	0.0155	0
Diphenylamine		0.127	49	0	0.0%	0	0	0	0.0635	0
Ethyl Acrylate		0.655	283	0	0.0%	0	0	0	0.327	0.0195
Ethyl methanesulfonate		0.148	49	0	0.0%	0	0	0	0.074	0
Ethyl Tert-Butyl Ether		0.627	283	0	0.0%	0	0	0	0.312	0.0186
Ethylbenzene	0.36	0.174	342	318	93.0%	8.22	5.47	2.81	0.777	0.651
Fluoranthene		0.0725	49	21	42.9%	0.0552	0.0383	0.0214	0.0257	0.0147
Fluorene		0.0501	49	46	93.9%	0.125	0.122	0.0742	0.0258	0.0245
Formaldehyde	0.0098	0.0197	324	319	98.5%	25.8	12.9	11.6	2.87	2.36
Halocarbon 113	0.261		59	56	94.9%	1.37	1	0.977	0.637	0.175
Halocarbon 114	0.294		59	0	0.0%	0	0	0	0.147	0

DEARBORN MONITORING SITE - AIR ID 261630033										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
Hexachloro-1,3-Butadiene VOC	0.672	0.64	342	0	0.0%	0	0	0	0.322	0.0185
Hexachloro-1,3-ButadieneSVOC		0.0662	49	0	0.0%	0	0	0	0.0331	0
Hexachlorobenzene		0.0706	49	0	0.0%	0	0	0	0.0353	0
Hexachlorocyclopentadiene		0.109	49	0	0.0%	0	0	0	0.0544	0
Hexachloroethane		0.0283	49	0	0.0%	0	0	0	0.0141	0
Hexachloropropene		0.032	49	0	0.0%	0	0	0	0.016	0
Hexanaldehyde	0.0206	0.0164	324	306	94.4%	4.88	2.96	2.8	0.482	0.708
Indeno(123-cd)pyrene		0.0728	49	0	0.0%	0	0	0	0.0364	0
Isodrin		0.023	49	0	0.0%	0	0	0	0.0115	0
Isophorone		0.113	49	0	0.0%	0	0	0	0.0565	0
Isosafrole		0.029	49	0	0.0%	0	0	0	0.0145	0
Isovaleraldehyde	0.0431	0.0141	324	95	29.3%	1.85	1.33	1.26	0.0978	0.248
Lead (TSP)	0.00001		70	70	100.0%	0.0763	0.0601	0.0592	0.0238	0.0137
m,p-Tolualdehyde	0.0118		55	18	32.7%	0.791	0.762	0.319	0.0818	0.163
m/p -Xylene	0.869	0.217	342	324	94.7%	26.4	15.2	8.5	2.22	1.99
Manganese (TSP)	0.00001		70	70	100.0%	1.19	0.733	0.536	0.198	0.182
Methyl Ethyl Ketone	5.01	0.442	342	208	60.8%	8.61	7.58	7.06	1.9	1.4
Methyl Isobutyl Ketone	3.44	0.615	342	28	8.2%	3.02	2.99	2.68	0.645	0.613
Methyl Methacrylate		0.737	283	0	0.0%	0	0	0	0.367	0.0219
Methyl methanesulfonate		0.0825	49	0	0.0%	0	0	0	0.0413	0
Methyl Tert-Butyl Ether	0.22	0.649	342	53	15.5%	2.11	1.95	1.59	0.334	0.217
Methylene Chloride	0.799	0.208	342	326	95.3%	513	214	213	11.6	37.4
Naphthalene		0.0773	49	49	100.0%	10.5	1.22	1.03	0.631	1.46
n-Butyraldehyde	0.0206		55	45	81.8%	3.76	2.74	1.73	0.374	0.665
n-Hexane	1.76		59	12	20.3%	11.7	5.63	4.08	1.48	1.68
Nickel (TSP)	0.00001		70	70	100.0%	0.00965	0.00802	0.00684	0.00307	0.00166
Nitrobenzene		0.124	49	0	0.0%	0	0	0	0.0622	0
N-Nitrosodibutylamine		0.112	49	0	0.0%	0	0	0	0.0561	0
N-Nitrosodiethylamine		0.036	49	0	0.0%	0	0	0	0.018	0
N-Nitrosodimethylamine		0.131	49	0	0.0%	0	0	0	0.0654	0
N-Nitrosodipropylamine		0.107	49	0	0.0%	0	0	0	0.0537	0
N-Nitrosomethylethylamine		0.035	49	0	0.0%	0	0	0	0.0175	0

DEARBORN MONITORING SITE - AIR ID 261630033										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
N-Nitrosopiperidine		0.0866	49	0	0.0%	0	0	0	0.0433	0
N-Nitrosopyrrolidine		0.037	49	0	0.0%	0	0	0	0.0185	0
n-Octane		0.28	283	151	53.4%	3.51	1.31	1.3	0.272	0.267
o-Tolualdehyde	0.0118		55	2	3.6%	0.0983	0.0688	0	0.00871	0.0149
o-Toluidine		0.038	49	0	0.0%	0	0	0	0.019	0
o-xylene	0.187	0.217	342	334	97.7%	10.9	3.72	3.18	0.895	0.771
Pentachlorobenzene		0.0487	49	0	0.0%	0	0	0	0.0244	0
Pentachloroethane		0.044	49	0	0.0%	0	0	0	0.022	0
Pentachloronitrobenzene		0.0519	49	0	0.0%	0	0	0	0.0259	0
Pentachlorophenol		0.0737	49	0	0.0%	0	0	0	0.0369	0
Phenacetin		0.0819	49	0	0.0%	0	0	0	0.041	0
Phenanthrene		0.0515	49	48	98.0%	0.275	0.274	0.162	0.0532	0.0549
Phenol		0.112	49	20	40.8%	0.284	0.233	0.217	0.0918	0.0575
Pronamide		0.0629	49	0	0.0%	0	0	0	0.0314	0
Propionaldehyde	0.196	0.0166	324	302	93.2%	3.42	2.51	2.32	0.371	0.409
Propylene		0.0861	283	283	100.0%	24.3	19.7	13.1	2.08	2.53
Pyrene		0.0527	49	12	24.5%	0.0266	0.0222	0.0129	0.0223	0.00811
Pyridine		0.059	49	0	0.0%	0	0	0	0.0295	0
Safrole		0.029	49	0	0.0%	0	0	0	0.0145	0
Styrene	0.23	0.298	342	134	39.2%	0.735	0.649	0.59	0.167	0.0831
Tert-Amyl Methyl Ether		0.502	283	0	0.0%	0	0	0	0.25	0.0149
Tetrachloroethene	0.482	0.407	342	173	50.6%	4.54	4.29	3.87	0.453	0.513
Tolualdehydes		0.0393	269	260	96.7%	2.9	1.38	1.2	0.218	0.248
Toluene	0.264	0.226	342	341	99.7%	50.6	32.8	19.8	4.07	3.91
trans-1,2-Dichloroethene		0.238	342	1	0.3%	0.357	0	0	0.128	0.0241
trans-1,3-Dichloropropene	0.281	0.499	342	0	0.0%	0	0	0	0.23	0.0431
Trichloroethene	0.204	0.376	342	22	6.4%	6.82	3.9	1.3	0.211	0.419
Trichloroflouromethane	0.27	0.225	342	339	99.1%	8.65	5.78	4.79	1.76	0.64
Trichlorotrifluoroethane		0.536	283	283	100.0%	1.49	1.46	1.42	0.855	0.181
Valeraldehyde	0.0775	0.0106	324	280	86.4%	1.83	1.33	1.21	0.209	0.255
Vinyl Chloride	0.112	0.153	342	0	0.0%	0	0	0	0.0729	0.00869

SOUTH DELRAY MONITORING SITE - AIR ID 261630027										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
1,1,1-Trichloroethane	0.262	0.327	60	44	73.3%	0.588	0.312	0.309	0.209	0.0693
1,1,2,2-Tetrachloroethane	0.556	0.412	60	0	0.0%	0	0	0	0.207	0.00931
1,1,2-Trichloroethane	0.262	0.327	60	0	0.0%	0	0	0	0.163	0.00423
1,1-Dichloroethane	0.36	0.324	60	0	0.0%	0	0	0	0.162	0.00235
1,1-Dichloroethene	0.182	0.396	60	0	0.0%	0	0	0	0.197	0.0138
1,2,4,5-Tetrachlorobenzene		0.0527	58	0	0.0%	0	0	0	0.0264	0
1,2,4-Trichlorobenzene VOC	0.601	0.445	60	0	0.0%	0	0	0	0.224	0.0101
1,2,4-Trichlorobenzene SVOC		0.0681	58	0	0.0%	0	0	0	0.0341	0
1,2,4-Trimethylbenzene	0.305	0.344	60	59	98.3%	5	3.34	2.92	1.26	0.908
1,2-Dibromoethane	0.415	0.615	60	0	0.0%	0	0	0	0.306	0.0129
1,2-Dichlorobenzene VOC	0.397	0.361	60	0	0.0%	0	0	0	0.181	0.00233
1,2-Dichlorobenzene SVOC		0.0548	58	0	0.0%	0	0	0	0.0274	0
1,2-Dichloroethane	0.348	0.243	60	0	0.0%	0	0	0	0.122	0.00679
1,2-Dichloropropane	0.273	0.323	60	0	0.0%	0	0	0	0.161	0.00328
1,3,5-Trimethylbenzene	0.28	0.344	60	53	88.3%	1.96	1.76	1.63	0.552	0.457
1,3-Butadiene	0.0885	0.155	60	48	80.0%	0.711	0.67	0.547	0.19	0.145
1,3-Dichlorobenzene VOC	0.319	0.301	60	0	0.0%	0	0	0	0.151	0.00116
1,3-Dichlorobenzene SVOC		0.0603	58	0	0.0%	0	0	0	0.0301	0
1,3-Dintrobenzene		0.038	58	0	0.0%	0	0	0	0.019	0
1,4-Dichlorobenzene VOC	0.319	0.541	60	10	16.7%	2.47	0.421	0.361	0.267	0.294
1,4-Dichlorobenzene SVOC		0.0543	58	51	87.9%	0.377	0.329	0.311	0.0904	0.0779
1,4-Naphthoquinone		0.029	58	0	0.0%	0	0	0	0.0145	0
1-Naphthylamine		0.0271	58	0	0.0%	0	0	0	0.0136	0
2,2,4-Trimethylpentane	0.0794		1	1	100.0%	0.561	0	0	0.561	
2,3,4,6-Tetrachlorophenol		0.0484	58	0	0.0%	0	0	0	0.0242	0
2,4,5-Trichlorphenol		0.0345	58	0	0.0%	0	0	0	0.0173	0
2,4,6-Trichlorophenol		0.0418	58	0	0.0%	0	0	0	0.0209	0
2,4-Dichlorophenol		0.0728	58	0	0.0%	0	0	0	0.0364	0
2,4-Dimethylphenol		0.0882	58	0	0.0%	0	0	0	0.0441	0
2,4-Dinitrophenol		0.0503	58	0	0.0%	0	0	0	0.0251	0
2,4-Dinitrotoluene		0.0486	58	0	0.0%	0	0	0	0.0243	0
2,5-dimethylbenzaldehyde	0.0176	0.0274	51	8	15.7%	0.521	0.301	0.29	0.0395	0.0915
2,6-Dichlorophenol		0.0905	58	0	0.0%	0	0	0	0.0453	0

SOUTH DELRAY MONITORING SITE - AIR ID 261630027										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
2,6-Dinitrotoluene		0.0469	58	0	0.0%	0	0	0	0.0235	0
2-Acetylaminofluorene		0.017	58	0	0.0%	0	0	0	0.0085	0
2-Chloro-1,3-Butadiene	0.109	0.362	60	0	0.0%	0	0	0	0.179	0.0164
2-Chloronaphthalene		0.0924	58	0	0.0%	0	0	0	0.0462	0
2-Chlorophenol		0.05	58	0	0.0%	0	0	0	0.025	0
2-Methyl Naphthalene		0.0568	58	57	98.3%	7.21	6.48	4.91	1.62	1.77
2-Methylphenol		0.0472	58	29	50.0%	0.908	0.571	0.498	0.126	0.18
2-Naphthylamine		0.0511	58	0	0.0%	0	0	0	0.0256	0
2-Nitroaniline		0.0615	58	0	0.0%	0	0	0	0.0308	0
2-Nitrophenol		0.0504	58	0	0.0%	0	0	0	0.0252	0
2-Picoline		0.0558	58	6	10.3%	0.325	0.136	0.101	0.0367	0.0423
3&4-Methylphenol		0.042	58	38	65.5%	2.1	1.81	1.57	0.357	0.519
3,3'-Dichlorobenzidine		0.0448	58	0	0.0%	0	0	0	0.0224	0
3,3'-Dimethylbenzidine		0.25	58	0	0.0%	0	0	0	0.125	0
3-Methylcholanthrene		0.032	58	0	0.0%	0	0	0	0.016	0
3-Nitroaniline		0.0435	58	0	0.0%	0	0	0	0.0218	0
4,6-Dinitro-2-methylphenol		0.0566	58	0	0.0%	0	0	0	0.0283	0
4-Aminobiphenyl		0.048	58	0	0.0%	0	0	0	0.024	0
4-Bromophenyl phenyl ether		0.0565	58	0	0.0%	0	0	0	0.0283	0
4-Chloro-3-methylphenol		0.084	58	0	0.0%	0	0	0	0.042	0
4-Chloroaniline		0.0842	58	0	0.0%	0	0	0	0.0421	0
4-Chlorophenyl-phenyl ether		0.034	58	0	0.0%	0	0	0	0.017	0
4-Dimethylaminoazobenzene		0.022	58	0	0.0%	0	0	0	0.011	0
4-Nitroaniline		0.052	58	0	0.0%	0	0	0	0.026	0
4-Nitrophenol		0.0368	58	0	0.0%	0	0	0	0.0184	0
5-Nitro-o-toluidine		0.026	58	1	1.7%	0.178	0	0	0.0158	0.0216
7,12-Dimethylbenz(a)anthracene		0.0952	58	0	0.0%	0	0	0	0.0476	0
Acenaphthene		0.0291	58	57	98.3%	2.64	2.52	2.21	0.625	0.75
Acenaphthylene		0.0444	58	45	77.6%	0.225	0.18	0.167	0.0399	0.0467
Acetaldehyde	0.0167	0.0252	51	51	100.0%	4.01	3.43	2.89	1.72	0.827
Acetone	0.0255	0.019	51	51	100.0%	7.57	6.68	6.67	2.46	1.55
Acetonitrile	0.873	0.42	60	1	1.7%	0.82	0	0	0.224	0.0835

SOUTH DELRAY MONITORING SITE - AIR ID 261630027										
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Acetophenone		0.0694	58	0	0.0%	0	0	0	0.0347	0
Acetylene		0.138	59	57	96.6%	5.89	4.92	4.45	1.92	1.06
Acrylonitrile	0.847	0.456	60	0	0.0%	0	0	0	0.231	0.0252
Aniline		0.081	58	0	0.0%	0	0	0	0.0405	0
Anthracene		0.0853	58	47	81.0%	0.667	0.398	0.378	0.0987	0.133
Arsenic (TSP)	0.000016		65	65	100.0%	0.0247	0.0246	0.00977	0.00336	0.00415
Azobenzene		0.087	58	0	0.0%	0	0	0	0.0435	0
Benzaldehyde	0.0167	0.0174	51	50	98.0%	0.962	0.588	0.571	0.222	0.167
Benzene	0.224	0.128	60	60	100.0%	159	147	127	21.6	34.6
Benzidine		0.25	58	0	0.0%	0	0	0	0.125	0
Benzo(a)anthracene		0.0417	58	30	51.7%	0.0925	0.0637	0.04	0.0216	0.013
Benzo(a)pyrene		0.0909	58	4	6.9%	0.0322	0.0218	0.0213	0.0437	0.00671
Benzo(b)fluoranthene		0.0867	58	15	25.9%	0.0566	0.0313	0.0287	0.037	0.0125
Benzo(ghi)perylene		0.0753	58	0	0.0%	0	0	0	0.0376	0
Benzo(k)fluoranthene		0.118	58	15	25.9%	0.0407	0.0284	0.0158	0.0475	0.0203
Benzyl alcohol		0.0408	58	0	0.0%	0	0	0	0.0204	0
Beryllium (TSP)	0.00001		65	65	100.0%	0.00243	0.0015	0.00115	0.000363	0.000383
bis (2-Chloroethyl)ether		0.0583	58	0	0.0%	0	0	0	0.0292	0
bis(2-Chloroethoxy)methane		0.0702	58	0	0.0%	0	0	0	0.0351	0
bis(2-Chloroisopropyl)ether		0.0554	58	0	0.0%	0	0	0	0.0277	0
bis(2-Ethylhexyl)phthalate		0.0581	58	8	13.8%	0.0268	0.0224	0.0191	0.0268	0.00649
Bromochloromethane		0.635	59	0	0.0%	0	0	0	0.318	0
Bromodichloromethane	0.489	0.402	60	0	0.0%	0	0	0	0.202	0.00562
Bromoform	0.61	0.827	60	0	0.0%	0	0	0	0.412	0.014
Bromomethane	0.155	0.35	60	0	0.0%	0	0	0	0.173	0.0125
Butyl benzyl phthalate		0.0583	58	0	0.0%	0	0	0	0.0292	0
Butyr/Isobutyraldehyde		0.0147	51	51	100.0%	1.75	0.848	0.768	0.416	0.255
Cadmium (TSP)	0.000024		65	65	100.0%	0.00429	0.00347	0.00269	0.00113	0.000773
Carbazole		0.0627	58	28	48.3%	0.328	0.143	0.134	0.0468	0.0485
Carbon Tetrachloride	0.239	0.503	60	59	98.3%	0.903	0.881	0.823	0.618	0.122
Chlorobenzene	0.184	0.276	60	0	0.0%	0	0	0	0.137	0.00594
Chlorobenzilate		0.016	58	0	0.0%	0	0	0	0.008	0
Chloroethane	0.106	0.211	60	0	0.0%	0	0	0	0.105	0.00681

SOUTH DELRAY MONITORING SITE - AIR ID 261630027										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
Chloroform	0.332	0.244	60	10	16.7%	0.284	0.23	0.207	0.131	0.0327
Chloromethane	0.128	0.124	60	59	98.3%	1.94	1.58	1.55	1.22	0.229
Chloromethyl Benzene	0.393	0.362	60	0	0.0%	0	0	0	0.182	0.00201
Chromium (TSP)	0.000014		65	65	100.0%	0.0219	0.0152	0.0136	0.00778	0.00326
Chrysene		0.0529	58	31	53.4%	0.117	0.0693	0.0516	0.0267	0.0164
cis-1,2-Dichloroethene	0.226	0.396	60	0	0.0%	0	0	0	0.197	0.011
cis-1,3-Dichloropropene	0.245	0.454	60	0	0.0%	0	0	0	0.225	0.0135
Crotonaldehyde	0.0225	0.0172	51	42	82.4%	0.626	0.415	0.324	0.0691	0.121
Diallate		0.024	58	0	0.0%	0	0	0	0.012	0
Dibenzo(ah)anthracene		0.0793	58	0	0.0%	0	0	0	0.0396	0
Dibenzofuran		0.0464	58	57	98.3%	2.63	2.19	1.58	0.476	0.604
Dibromochloromethane	0.503	0.682	60	0	0.0%	0	0	0	0.339	0.0116
Dichlorodiflouromethane	0.237	0.198	60	59	98.3%	6.63	5.39	4.43	3.16	0.765
Dichlorotetrafluoroethane		0.35	59	8	13.6%	0.14	0.118	0.113	0.164	0.0287
Diethyl phthalate		0.036	58	2	3.4%	0.0326	0.0181	0	0.0183	0.00192
Dimethyl phthalate		0.0458	58	0	0.0%	0	0	0	0.0229	0
Di-n-butyl phthalate		0.0701	58	47	81.0%	0.49	0.369	0.317	0.102	0.0957
Di-n-octyl phthalate		0.066	58	0	0.0%	0	0	0	0.033	0
Dinoseb		0.031	58	0	0.0%	0	0	0	0.0155	0
Diphenylamine		0.127	58	0	0.0%	0	0	0	0.0635	0
Ethyl Acrylate		0.655	59	0	0.0%	0	0	0	0.328	0
Ethyl methanesulfonate		0.148	58	0	0.0%	0	0	0	0.074	0
Ethyl Tert-Butyl Ether		0.627	59	0	0.0%	0	0	0	0.314	0
Ethylbenzene	0.36	0.174	60	60	100.0%	11.4	6.69	6.42	1.76	1.94
Fluoranthene		0.0725	58	53	91.4%	1.39	0.608	0.577	0.162	0.232
Fluorene		0.0501	58	56	96.6%	2.52	2.02	1.63	0.454	0.589
Formaldehyde	0.0098	0.0197	51	51	100.0%	6.13	5.88	5.77	3	1.52
Halocarbon 113	0.261		1	1	100.0%	0.767	0	0	0.767	
Halocarbon 114	0.294		1	0	0.0%	0	0	0	0.147	
Hexachloro-1,3-Butadiene VOC	0.672	0.64	60	0	0.0%	0	0	0	0.32	0.00207
Hexachloro-1,3-ButadieneSVOC		0.0662	58	0	0.0%	0	0	0	0.0331	0
Hexachlorobenzene		0.0706	58	0	0.0%	0	0	0	0.0353	0

SOUTH DELRAY MONITORING SITE - AIR ID 261630027										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
Hexachlorocyclopentadiene		0.109	58	0	0.0%	0	0	0	0.0544	0
Hexachloroethane		0.0283	58	0	0.0%	0	0	0	0.0141	0
Hexachloropropene		0.032	58	0	0.0%	0	0	0	0.016	0
Hexanaldehyde	0.0206	0.0164	51	51	100.0%	1.03	0.615	0.49	0.179	0.169
Indeno(123-cd)pyrene		0.0728	58	0	0.0%	0	0	0	0.0364	0
Isodrin		0.023	58	0	0.0%	0	0	0	0.0115	0
Isophorone		0.113	58	0	0.0%	0	0	0	0.0565	0
Isosafrole		0.029	58	0	0.0%	0	0	0	0.0145	0
Isovaleraldehyde	0.0431	0.0141	51	11	21.6%	0.179	0.129	0.107	0.0217	0.036
Lead (TSP)	0.00001		65	65	100.0%	0.0789	0.0718	0.0655	0.0293	0.0165
m/p -Xylene	0.869	0.217	60	60	100.0%	20	13.9	13.2	4.07	3.78
Manganese (TSP)	0.00001		65	65	100.0%	1.94	1.01	0.999	0.274	0.301
Methyl Ethyl Ketone	5.01	0.442	60	47	78.3%	7.33	5.06	4.4	1.91	1.38
Methyl Isobutyl Ketone	3.44	0.615	60	1	1.7%	0.859	0	0	0.34	0.195
Methyl Methacrylate		0.737	59	0	0.0%	0	0	0	0.369	0
Methyl methanesulfonate		0.0825	58	0	0.0%	0	0	0	0.0413	0
Methyl Tert-Butyl Ether	0.22	0.649	60	5	8.3%	0.87	0.611	0.577	0.333	0.0984
Methylene Chloride	0.799	0.208	60	58	96.7%	58.6	10.4	8.2	2.53	7.6
Naphthalene		0.0773	58	58	100.0%	93.3	63.5	62.5	18.4	21.7
n-Hexane	1.76		1	0	0.0%	0	0	0	0.881	
Nickel (TSP)	0.00001		65	65	100.0%	0.0225	0.0156	0.0136	0.00491	0.0037
Nitrobenzene		0.124	58	0	0.0%	0	0	0	0.0622	0
N-Nitrosodibutylamine		0.112	58	0	0.0%	0	0	0	0.0561	0
N-Nitrosodiethylamine		0.036	58	0	0.0%	0	0	0	0.018	0
N-Nitrosodimethylamine		0.131	58	0	0.0%	0	0	0	0.0654	0
N-Nitrosodipropylamine		0.107	58	0	0.0%	0	0	0	0.0537	0
N-Nitrosomethylethylamine		0.035	58	0	0.0%	0	0	0	0.0175	0
N-Nitrosopiperidine		0.0866	58	0	0.0%	0	0	0	0.0433	0
N-Nitrosopyrrolidine		0.037	58	0	0.0%	0	0	0	0.0185	0
n-Octane		0.28	59	35	59.3%	1.47	1.08	0.989	0.28	0.255
o-Toluidine		0.038	58	0	0.0%	0	0	0	0.019	0
o-xylene	0.187	0.217	60	60	100.0%	5.74	4.59	4.5	1.48	1.21
Pentachlorobenzene		0.0487	58	0	0.0%	0	0	0	0.0244	0

SOUTH DELRAY MONITORING SITE - AIR ID 261630027

CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
Pentachloroethane		0.044	58	0	0.0%	0	0	0	0.022	0
Pentachloronitrobenzene		0.0519	58	0	0.0%	0	0	0	0.0259	0
Pentachlorophenol		0.0737	58	0	0.0%	0	0	0	0.0369	0
Phenacetin		0.0819	58	0	0.0%	0	0	0	0.041	0
Phenanthrene		0.0515	58	57	98.3%	4.03	2.12	1.94	0.625	0.777
Phenol		0.112	58	43	74.1%	5.05	3.97	3.57	0.942	1.26
Pronamide		0.0629	58	0	0.0%	0	0	0	0.0314	0
Propionaldehyde	0.196	0.0166	51	50	98.0%	0.723	0.708	0.664	0.263	0.166
Propylene		0.0861	59	58	98.3%	6.9	5.93	5.2	1.63	1.29
Pyrene		0.0527	58	52	89.7%	0.806	0.385	0.333	0.103	0.137
Pyridine		0.059	58	19	32.8%	0.807	0.75	0.518	0.0911	0.161
Safrole		0.029	58	0	0.0%	0	0	0	0.0145	0
Styrene	0.23	0.298	60	33	55.0%	1.83	0.965	0.919	0.322	0.307
Tert-Amyl Methyl Ether		0.502	59	0	0.0%	0	0	0	0.251	0
Tetrachloroethene	0.482	0.407	60	22	36.7%	2.34	0.877	0.87	0.293	0.305
Tolualdehydes		0.0393	51	50	98.0%	1.99	0.751	0.623	0.221	0.288
Toluene	0.264	0.226	60	60	100.0%	56.5	42.9	42.2	10.6	11.3
trans-1,2-Dichloroethene		0.238	60	0	0.0%	0	0	0	0.12	0.00665
trans-1,3-Dichloropropene	0.281	0.499	60	0	0.0%	0	0	0	0.248	0.0141
Trichloroethene	0.204	0.376	60	6	10.0%	1.51	1.15	1.08	0.266	0.275
Trichloroflouromethane	0.27	0.225	60	60	100.0%	18	5.24	3.01	2.14	2.17
Trichlorotrifluoroethane		0.536	59	58	98.3%	7.66	1.32	1.15	0.969	0.908
Valeraldehyde	0.0775	0.0106	51	48	94.1%	0.68	0.243	0.238	0.105	0.0989
Vinyl Chloride	0.112	0.153	60	0	0.0%	0	0	0	0.0764	0.00264

NORTHEAST DETROIT MONITORING SITE - AIRS ID 261630019

CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
1,2,4,5-Tetrachlorobenzene		0.0527	26	0	0.0%	0	0	0	0.0264	0
1,2,4-Trichlorobenzene SVOC		0.0681	26	0	0.0%	0	0	0	0.0341	0
1,2-Dichlorobenzene SVOC		0.0548	26	0	0.0%	0	0	0	0.0274	0

NORTHEAST DETROIT MONITORING SITE - AIRS ID 261630019										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
1,3-Dichlorobenzene SVOC		0.0603	26	0	0.0%	0	0	0	0.0301	0
1,3-Dintrobenzene		0.038	26	0	0.0%	0	0	0	0.019	0
1,4-Dichlorobenzene SVOC		0.0543	26	26	100.0%	0.786	0.403	0.402	0.195	0.168
1,4-Naphthoquinone		0.029	26	0	0.0%	0	0	0	0.0145	0
1-Naphthylamine		0.0271	26	0	0.0%	0	0	0	0.0136	0
2,3,4,6-Tetrachlorophenol		0.0484	26	0	0.0%	0	0	0	0.0242	0
2,4,5-Trichlorphenol		0.0345	26	0	0.0%	0	0	0	0.0173	0
2,4,6-Trichlorophenol		0.0418	26	0	0.0%	0	0	0	0.0209	0
2,4-Dichlorophenol		0.0728	26	0	0.0%	0	0	0	0.0364	0
2,4-Dimethylphenol		0.0882	26	0	0.0%	0	0	0	0.0441	0
2,4-Dinitrophenol		0.0503	26	0	0.0%	0	0	0	0.0251	0
2,4-Dinitrotoluene		0.0486	26	0	0.0%	0	0	0	0.0243	0
2,5-dimethylbenzaldehyde	0.0176	0.0274	61	1	1.6%	0.274	0	0	0.0132	0.034
2,6-Dichlorophenol		0.0905	26	0	0.0%	0	0	0	0.0453	0
2,6-Dinitrotoluene		0.0469	26	0	0.0%	0	0	0	0.0235	0
2-Acetylaminofluorene		0.017	26	0	0.0%	0	0	0	0.0085	0
2-Chloronaphthalene		0.0924	26	0	0.0%	0	0	0	0.0462	0
2-Chlorophenol		0.05	26	0	0.0%	0	0	0	0.025	0
2-Methyl Naphthalene		0.0568	26	26	100.0%	0.625	0.446	0.33	0.189	0.127
2-Methylphenol		0.0472	26	0	0.0%	0	0	0	0.0236	0
2-Naphthylamine		0.0511	26	0	0.0%	0	0	0	0.0256	0
2-Nitroaniline		0.0615	26	0	0.0%	0	0	0	0.0308	0
2-Nitrophenol		0.0504	26	0	0.0%	0	0	0	0.0252	0
2-Picoline		0.0558	26	0	0.0%	0	0	0	0.0279	0
3&4-Methylphenol		0.042	26	0	0.0%	0	0	0	0.021	0
3,3'-Dichlorobenzidine		0.0448	26	0	0.0%	0	0	0	0.0224	0
3,3'-Dimethylbenzidine		0.25	26	0	0.0%	0	0	0	0.125	0
3-Methylcholanthrene		0.032	26	0	0.0%	0	0	0	0.016	0
3-Nitroaniline		0.0435	26	0	0.0%	0	0	0	0.0218	0
4,6-Dinitro-2-methylphenol		0.0566	26	0	0.0%	0	0	0	0.0283	0
4-Aminobiphenyl		0.048	26	0	0.0%	0	0	0	0.024	0
4-Bromophenyl phenyl ether		0.0565	26	0	0.0%	0	0	0	0.0283	0
4-Chloro-3-methylphenol		0.084	26	0	0.0%	0	0	0	0.042	0

NORTHEAST DETROIT MONITORING SITE - AIRS ID 261630019										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
4-Chloroaniline		0.0842	26	0	0.0%	0	0	0	0.0421	0
4-Chlorophenyl-phenyl ether		0.034	26	0	0.0%	0	0	0	0.017	0
4-Dimethylaminoazobenzene		0.022	26	0	0.0%	0	0	0	0.011	0
4-Nitroaniline		0.052	26	0	0.0%	0	0	0	0.026	0
4-Nitrophenol		0.0368	26	0	0.0%	0	0	0	0.0184	0
5-Nitro-o-toluidine		0.026	26	0	0.0%	0	0	0	0.013	0
7,12-Dimethylbenz(a)anthracene		0.0952	26	0	0.0%	0	0	0	0.0476	0
Acenaphthene		0.0291	26	24	92.3%	0.0181	0.0112	0.011	0.00547	0.00486
Acenaphthylene		0.0444	26	8	30.8%	0.013	0.0117	0.00753	0.0175	0.00736
Acetaldehyde	0.0167	0.0252	61	61	100.0%	3.4	3.23	3.14	1.78	0.731
Acetone	0.0255	0.019	61	61	100.0%	5.86	5.03	4.99	2.73	1.15
Acetophenone		0.0694	26	0	0.0%	0	0	0	0.0347	0
Aniline		0.081	26	0	0.0%	0	0	0	0.0405	0
Anthracene		0.0853	26	0	0.0%	0	0	0	0.0427	0
Arsenic (TSP)	0.000016		68	68	100.0%	0.00583	0.00546	0.00486	0.00169	0.0014
Azobenzene		0.087	26	0	0.0%	0	0	0	0.0435	0
Benzaldehyde	0.0167	0.0174	61	42	68.9%	0.478	0.421	0.347	0.0923	0.11
Benzidine		0.25	26	0	0.0%	0	0	0	0.125	0
Benzo(a)anthracene		0.0417	26	0	0.0%	0	0	0	0.0208	0
Benzo(a)pyrene		0.0909	26	0	0.0%	0	0	0	0.0454	0
Benzo(b)fluoranthene		0.0867	26	0	0.0%	0	0	0	0.0434	0
Benzo(ghi)perylene		0.0753	26	0	0.0%	0	0	0	0.0376	0
Benzo(k)fluoranthene		0.118	26	0	0.0%	0	0	0	0.059	0
Benzyl alcohol		0.0408	26	0	0.0%	0	0	0	0.0204	0
Beryllium (TSP)	0.00001		68	68	100.0%	0.000487	0.00048	0.000477	7.28E-05	0.000092
bis (2-Chloroethyl)ether		0.0583	26	0	0.0%	0	0	0	0.0292	0
bis(2-Chloroethoxy)methane		0.0702	26	0	0.0%	0	0	0	0.0351	0
bis(2-Chloroisopropyl)ether		0.0554	26	0	0.0%	0	0	0	0.0277	0
bis(2-Ethylhexyl)phthalate		0.0581	26	3	11.5%	1.61	0.0168	0.0134	0.0886	0.309
Butyl benzyl phthalate		0.0583	26	0	0.0%	0	0	0	0.0292	0
Cadmium (TSP)	0.000024		68	68	100.0%	0.00139	0.00137	0.000905	0.00035	0.000242
Carbazole		0.0627	26	0	0.0%	0	0	0	0.0313	0
Chlorobenzilate		0.016	26	0	0.0%	0	0	0	0.008	0

NORTHEAST DETROIT MONITORING SITE - AIRS ID 261630019										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
Chromium (TSP)	0.000014		68	68	100.0%	0.00708	0.00551	0.00461	0.00298	0.00096
Chrysene		0.0529	26	0	0.0%	0	0	0	0.0264	0
Crotonaldehyde	0.0225	0.0172	61	10	16.4%	0.258	0.201	0.143	0.0282	0.049
Diallate		0.024	26	0	0.0%	0	0	0	0.012	0
Dibenzo(ah)anthracene		0.0793	26	0	0.0%	0	0	0	0.0396	0
Dibenzofuran		0.0464	26	23	88.5%	0.0217	0.0193	0.0173	0.00941	0.00718
Diethyl phthalate		0.036	26	6	23.1%	0.131	0.101	0.0923	0.0305	0.0307
Dimethyl phthalate		0.0458	26	0	0.0%	0	0	0	0.0229	0
Di-n-butyl phthalate		0.0701	26	4	15.4%	0.0304	0.017	0.0128	0.0321	0.00784
Di-n-octyl phthalate		0.066	26	0	0.0%	0	0	0	0.033	0
Dinoseb		0.031	26	0	0.0%	0	0	0	0.0155	0
Diphenylamine		0.127	26	0	0.0%	0	0	0	0.0635	0
Ethyl methanesulfonate		0.148	26	0	0.0%	0	0	0	0.074	0
Fluoranthene		0.0725	26	7	26.9%	0.0138	0.00772	0.00592	0.0281	0.0137
Fluorene		0.0501	26	24	92.3%	0.0229	0.0174	0.0155	0.00844	0.00737
Formaldehyde	0.0098	0.0197	61	61	100.0%	5.35	5	4.9	2.81	1.1
Hexachloro-1,3-Butadiene		0.0662	26	0	0.0%	0	0	0	0.0331	0
Hexachlorobenzene		0.0706	26	0	0.0%	0	0	0	0.0353	0
Hexachlorocyclopentadiene		0.109	26	0	0.0%	0	0	0	0.0544	0
Hexachloroethane		0.0283	26	0	0.0%	0	0	0	0.0141	0
Hexachloropropene		0.032	26	0	0.0%	0	0	0	0.016	0
Hexanaldehyde	0.0206	0.0164	61	48	78.7%	1.25	0.721	0.619	0.197	0.227
Indeno(123-cd)pyrene		0.0728	26	0	0.0%	0	0	0	0.0364	0
Isodrin		0.023	26	0	0.0%	0	0	0	0.0115	0
Isophorone		0.113	26	0	0.0%	0	0	0	0.0565	0
Isosafrole		0.029	26	0	0.0%	0	0	0	0.0145	0
Isovaleraldehyde	0.0431	0.0141	61	24	39.3%	0.247	0.219	0.176	0.0582	0.057
Lead (TSP)	0.00001		68	68	100.0%	0.0355	0.0295	0.023	0.0115	0.00584
m,p-Tolualdehyde	0.0118		61	19	31.1%	0.781	0.344	0.3	0.0612	0.126
Manganese (TSP)	0.00001		68	68	100.0%	0.0806	0.0668	0.0636	0.0261	0.0154
Methyl methanesulfonate		0.0825	26	0	0.0%	0	0	0	0.0413	0
Naphthalene		0.0773	26	26	100.0%	1.24	0.883	0.671	0.391	0.253
n-Butyraldehyde	0.0206		61	60	98.4%	0.611	0.599	0.49	0.216	0.134

NORTHEAST DETROIT MONITORING SITE - AIRS ID 261630019										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
Nickel (TSP)	0.00001		68	68	100.0%	0.0111	0.00693	0.00523	0.00239	0.00163
Nitrobenzene		0.124	26	0	0.0%	0	0	0	0.0622	0
N-Nitrosodibutylamine		0.112	26	0	0.0%	0	0	0	0.0561	0
N-Nitrosodiethylamine		0.036	26	0	0.0%	0	0	0	0.018	0
N-Nitrosodimethylamine		0.131	26	0	0.0%	0	0	0	0.0654	0
N-Nitrosodipropylamine		0.107	26	0	0.0%	0	0	0	0.0537	0
N-Nitrosomethylethylamine		0.035	26	0	0.0%	0	0	0	0.0175	0
N-Nitrosopiperidine		0.0866	26	0	0.0%	0	0	0	0.0433	0
N-Nitrosopyrrolidine		0.037	26	0	0.0%	0	0	0	0.0185	0
o-Tolualdehyde	0.0118		61	0	0.0%	0	0	0	0.00569	0.00106
o-Toluidine		0.038	26	0	0.0%	0	0	0	0.019	0
Pentachlorobenzene		0.0487	26	0	0.0%	0	0	0	0.0244	0
Pentachloroethane		0.044	26	0	0.0%	0	0	0	0.022	0
Pentachloronitrobenzene		0.0519	26	0	0.0%	0	0	0	0.0259	0
Pentachlorophenol		0.0737	26	0	0.0%	0	0	0	0.0369	0
Phenacetin		0.0819	26	0	0.0%	0	0	0	0.041	0
Phenanthrene		0.0515	26	25	96.2%	0.059	0.0393	0.0386	0.0179	0.0126
Phenol		0.112	26	6	23.1%	0.126	0.0999	0.0721	0.0615	0.0163
Pronamide		0.0629	26	0	0.0%	0	0	0	0.0314	0
Propionaldehyde	0.196	0.0166	61	49	80.3%	0.789	0.665	0.665	0.288	0.185
Pyrene		0.0527	26	4	15.4%	0.0117	0.00648	0.0057	0.0234	0.00707
Pyridine		0.059	26	0	0.0%	0	0	0	0.0295	0
Safrole		0.029	26	0	0.0%	0	0	0	0.0145	0
Valeraldehyde	0.0775	0.0106	61	21	34.4%	0.451	0.391	0.282	0.0861	0.0859

HOUGHTON LAKE MONITORING SITE - AIRS ID 261130001										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
2,5-dimethylbenzaldehyde	0.0176	0.0274	30	0	0.0%	0	0	0	0.00881	0
Acetaldehyde	0.0167	0.0252	30	29	96.7%	2.55	1.66	1.54	0.859	0.499
Acetone	0.0255	0.019	30	30	100.0%	3.4	2.9	2.83	1.68	0.714

HOUGHTON LAKE MONITORING SITE - AIRS ID 261130001

CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
Arsenic (TSP)	0.000016		30	0	0.0%	0	0	0	0	0
Benzaldehyde	0.0167	0.0174	30	8	26.7%	0.352	0.217	0.174	0.0433	0.0812
Beryllium (TSP)	0.00001		30	0	0.0%	0	0	0	0	0
Cadmium (TSP)	0.000024		30	0	0.0%	0	0	0	0	0
Chromium (TSP)	0.000014		30	0	0.0%	0	0	0	0	0
Crotonaldehyde	0.0225	0.0172	30	2	6.7%	0.373	0.229	0	0.0306	0.0759
Formaldehyde	0.0098	0.0197	30	30	100.0%	7.59	6.99	4.95	2.02	1.73
Hexanaldehyde	0.0206	0.0164	30	12	40.0%	0.41	0.287	0.242	0.057	0.0982
Isovaleraldehyde	0.0431	0.0141	30	5	16.7%	0.106	0.106	0.081	0.0324	0.0256
Lead (TSP)	0.00001		30	18	6000.0%	0.016	0.013	0.009	0.0036	0.00407
Manganese (TSP)	0.00001		30	30	10000.0%	0.03	0.015	0.013	0.00558	0.00598
n-Butyraldehyde	0.0206		30	27	90.0%	0.858	0.59	0.499	0.215	0.194
Nickel (TSP)	0.00001		30	0	0.0%	0	0	0	0	0
o-Tolualdehyde	0.0118		30	0	0.0%	0	0	0	0.00588	0
Propionaldehyde	0.196	0.0166	30	11	36.7%	1.99	0.729	0.33	0.213	0.359
Valeraldehyde	0.0775	0.0106	30	6	20.0%	0.691	0.141	0.106	0.0683	0.12

SOUTHFIELD MONITORING SITE - AIRS ID 261250010

CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
1,1,1-Trichloroethane	0.262	0.327	59	43	72.9%	0.611	0.53	0.351	0.219	0.0833
1,1,2,2-Tetrachloroethane	0.556	0.412	59	0	0.0%	0	0	0	0.206	0
1,1,2-Trichloroethane	0.262	0.327	59	0	0.0%	0	0	0	0.164	0
1,1-Dichloroethane	0.36	0.324	59	0	0.0%	0	0	0	0.162	0
1,1-Dichloroethene	0.182	0.396	59	0	0.0%	0	0	0	0.198	0
1,2,4,5-Tetrachlorobenzene		0.0527	59	0	0.0%	0	0	0	0.0264	0
1,2,4-Trichlorobenzene VOC	0.601	0.445	59	0	0.0%	0	0	0	0.223	0
1,2,4-Trichlorobenzene SVOC		0.0681	59	0	0.0%	0	0	0	0.0341	0
1,2,4-Trimethylbenzene	0.305	0.344	59	58	98.3%	4.48	3.14	2.48	0.99	0.738
1,2-Dibromoethane	0.415	0.615	59	0	0.0%	0	0	0	0.307	0
1,2-Dichlorobenzene VOC	0.397	0.361	59	0	0.0%	0	0	0	0.18	0

SOUTHFIELD MONITORING SITE - AIRS ID 261250010										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
1,2-Dichlorobenzene SVOC		0.0548	59	0	0.0%	0	0	0	0.0274	0
1,2-Dichloroethane	0.348	0.243	59	0	0.0%	0	0	0	0.121	0
1,2-Dichloropropane	0.273	0.323	59	0	0.0%	0	0	0	0.162	0
1,3,5-Trimethylbenzene	0.28	0.344	59	47	79.7%	1.24	1.05	0.732	0.321	0.212
1,3-Butadiene	0.0885	0.155	59	49	83.1%	0.738	0.42	0.376	0.191	0.121
1,3-Dichlorobenzene SVOC	0.319	0.301	59	0	0.0%	0	0	0	0.15	0
1,3-Dichlorobenzene SVOC		0.0603	59	0	0.0%	0	0	0	0.0301	0
1,3-Dintrobenzene		0.038	59	0	0.0%	0	0	0	0.019	0
1,4-Dichlorobenzene VOC	0.319	0.541	59	11	18.6%	0.661	0.361	0.301	0.234	0.075
1,4-Dichlorobenzene SVOC		0.0543	59	57	96.6%	0.232	0.172	0.167	0.0741	0.0489
1,4-Naphthoquinone		0.029	59	0	0.0%	0	0	0	0.0145	0
1-Naphthylamine		0.0271	59	0	0.0%	0	0	0	0.0136	0
2,3,4,6-Tetrachlorophenol		0.0484	59	0	0.0%	0	0	0	0.0242	0
2,4,5-Trichlorphenol		0.0345	59	0	0.0%	0	0	0	0.0173	0
2,4,6-Trichlorophenol		0.0418	59	0	0.0%	0	0	0	0.0209	0
2,4-Dichlorophenol		0.0728	59	0	0.0%	0	0	0	0.0364	0
2,4-Dimethylphenol		0.0882	59	0	0.0%	0	0	0	0.0441	0
2,4-Dinitrophenol		0.0503	59	0	0.0%	0	0	0	0.0251	0
2,4-Dinitrotoluene		0.0486	59	0	0.0%	0	0	0	0.0243	0
2,5-dimethylbenzaldehyde	0.0176	0.0274	61	11	18.0%	0.185	0.0559	0.0509	0.0163	0.0241
2,6-Dichlorophenol		0.0905	59	0	0.0%	0	0	0	0.0453	0
2,6-Dinitrotoluene		0.0469	59	0	0.0%	0	0	0	0.0235	0
2-Acetylaminofluorene		0.017	59	0	0.0%	0	0	0	0.0085	0
2-Chloro-1,3-Butadiene	0.109	0.362	59	0	0.0%	0	0	0	0.181	0
2-Chloronaphthalene		0.0924	59	0	0.0%	0	0	0	0.0462	0
2-Chlorophenol		0.05	59	0	0.0%	0	0	0	0.025	0
2-Methyl Naphthalene		0.0568	59	59	100.0%	0.283	0.271	0.253	0.135	0.0568
2-Methylphenol		0.0472	59	0	0.0%	0	0	0	0.0236	0
2-Naphthylamine		0.0511	59	0	0.0%	0	0	0	0.0256	0
2-Nitroaniline		0.0615	59	0	0.0%	0	0	0	0.0308	0
2-Nitrophenol		0.0504	59	0	0.0%	0	0	0	0.0252	0
2-Picoline		0.0558	59	0	0.0%	0	0	0	0.0279	0
3&4-Methylphenol		0.042	59	5	8.5%	0.0225	0.0149	0.00918	0.0202	0.00324

SOUTHFIELD MONITORING SITE - AIRS ID 261250010										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
3,3'-Dichlorobenzidine		0.0448	59	0	0.0%	0	0	0	0.0224	0
3,3'-Dimethylbenzidine		0.25	59	0	0.0%	0	0	0	0.125	0
3-Methylcholanthrene		0.032	59	0	0.0%	0	0	0	0.016	0
3-Nitroaniline		0.0435	59	0	0.0%	0	0	0	0.0218	0
4,6-Dinitro-2-methylphenol		0.0566	59	0	0.0%	0	0	0	0.0283	0
4-Aminobiphenyl		0.048	59	0	0.0%	0	0	0	0.024	0
4-Bromophenyl phenyl ether		0.0565	59	0	0.0%	0	0	0	0.0283	0
4-Chloro-3-methylphenol		0.084	59	0	0.0%	0	0	0	0.042	0
4-Chloroaniline		0.0842	59	0	0.0%	0	0	0	0.0421	0
4-Chlorophenyl-phenyl ether		0.034	59	0	0.0%	0	0	0	0.017	0
4-Dimethylaminoazobenzene		0.022	59	0	0.0%	0	0	0	0.011	0
4-Nitroaniline		0.052	59	0	0.0%	0	0	0	0.026	0
4-Nitrophenol		0.0368	59	0	0.0%	0	0	0	0.0184	0
5-Nitro-o-toluidine		0.026	59	0	0.0%	0	0	0	0.013	0
7,12-Dimethylbenz(a)anthracene		0.0952	59	0	0.0%	0	0	0	0.0476	0
Acenaphthene		0.0291	59	55	93.2%	0.0283	0.0212	0.0195	0.00712	0.00579
Acenaphthylene		0.0444	59	20	33.9%	0.012	0.0095	0.00769	0.0164	0.00835
Acetaldehyde	0.0167	0.0252	61	61	100.0%	2.94	2.92	2.76	1.59	0.762
Acetone	0.0255	0.019	61	61	100.0%	7.64	6.96	6.63	3.21	1.79
Acetonitrile	0.873	0.42	59	7	11.9%	9.99	9.61	6.7	0.892	2.13
Acetophenone		0.0694	59	1	1.7%	0.0961	0	0	0.0357	0.00799
Acetylene		0.138	59	57	96.6%	6.72	3.41	2.92	1.59	0.961
Acrylonitrile	0.847	0.456	59	0	0.0%	0	0	0	0.228	0
Aniline		0.081	59	0	0.0%	0	0	0	0.0405	0
Anthracene		0.0853	59	2	3.4%	0.00129	0.00104	0	0.0412	0.00757
Arsenic (TSP)	0.000016		59	59	100.0%	0.00597	0.004	0.00312	0.00126	0.00102
Azobenzene		0.087	59	0	0.0%	0	0	0	0.0435	0
Benzaldehyde	0.0167	0.0174	61	56	91.8%	0.529	0.499	0.448	0.179	0.138
Benzene	0.224	0.128	59	59	100.0%	6.57	3.74	3.64	1.99	0.921
Benzidine		0.25	59	0	0.0%	0	0	0	0.125	0
Benzo(a)anthracene		0.0417	59	0	0.0%	0	0	0	0.0208	0
Benzo(a)pyrene		0.0909	59	0	0.0%	0	0	0	0.0454	0

SOUTHFIELD MONITORING SITE - AIRS ID 261250010										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
Benzo(b)fluoranthene		0.0867	59	0	0.0%	0	0	0	0.0434	0
Benzo(ghi)perylene		0.0753	59	0	0.0%	0	0	0	0.0376	0
Benzo(k)fluoranthene		0.118	59	0	0.0%	0	0	0	0.059	0
Benzyl alcohol		0.0408	59	0	0.0%	0	0	0	0.0204	0
Beryllium (TSP)	0.00001		59	59	100.0%	0.000437	0.000427	0.000427	7.77E-05	0.000108
bis (2-Chloroethyl)ether		0.0583	59	0	0.0%	0	0	0	0.0292	0
bis(2-Chloroethoxy)methane		0.0702	59	0	0.0%	0	0	0	0.0351	0
bis(2-Chloroisopropyl)ether		0.0554	59	0	0.0%	0	0	0	0.0277	0
bis(2-Ethylhexyl)phthalate		0.0581	59	6	10.2%	0.439	0.0162	0.0103	0.0343	0.0539
Bromochloromethane		0.635	59	0	0.0%	0	0	0	0.318	0
Bromodichloromethane	0.489	0.402	59	0	0.0%	0	0	0	0.201	0
Bromoform	0.61	0.827	59	0	0.0%	0	0	0	0.414	0
Bromomethane	0.155	0.35	59	0	0.0%	0	0	0	0.175	0
Butyl benzyl phthalate		0.0583	59	0	0.0%	0	0	0	0.0292	0
Butyr/Isobutyraldehyde		0.0147	31	31	100.0%	0.902	0.629	0.592	0.396	0.181
Cadmium (TSP)	0.000024		59	59	100.0%	0.00061	0.000434	0.000429	0.000203	0.000123
Carbazole		0.0627	59	0	0.0%	0	0	0	0.0313	0
Carbon Tetrachloride	0.239	0.503	59	59	100.0%	0.89	0.862	0.764	0.622	0.0962
Chlorobenzene	0.184	0.276	59	0	0.0%	0	0	0	0.138	0
Chlorobenzilate		0.016	59	0	0.0%	0	0	0	0.008	0
Chloroethane	0.106	0.211	59	0	0.0%	0	0	0	0.106	0
Chloroform	0.332	0.244	59	6	10.2%	0.261	0.189	0.171	0.125	0.0234
Chloromethane	0.128	0.124	59	59	100.0%	2.07	1.85	1.67	1.28	0.198
Chloromethyl Benzene	0.393	0.362	59	0	0.0%	0	0	0	0.181	0
Chromium (TSP)	0.000014		59	59	100.0%	0.0121	0.00584	0.00555	0.00362	0.00151
Chromium VI (TSP)		0.000013	32	16	50.0%	0.000234	0.00014	0.000133	4.26E-05	5.63E-05
Chrysene		0.0529	59	0	0.0%	0	0	0	0.0264	0
cis-1,2-Dichloroethene	0.226	0.396	59	0	0.0%	0	0	0	0.198	0
cis-1,3-Dichloropropene	0.245	0.454	59	0	0.0%	0	0	0	0.227	0
Crotonaldehyde	0.0225	0.0172	61	29	47.5%	0.485	0.445	0.29	0.044	0.0916
Diallate		0.024	59	0	0.0%	0	0	0	0.012	0
Dibenzo(ah)anthracene		0.0793	59	0	0.0%	0	0	0	0.0396	0
Dibenzofuran		0.0464	59	56	94.9%	0.0399	0.0287	0.0257	0.0103	0.00787

SOUTHFIELD MONITORING SITE - AIRS ID 261250010										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
Dibromochloromethane	0.503	0.682	59	0	0.0%	0	0	0	0.341	0
Dichlorodiflouromethane	0.237	0.198	59	59	100.0%	4.13	3.75	3.56	2.98	0.362
Dichlorotetrafluoroethane		0.35	59	6	10.2%	0.0839	0.0811	0.0755	0.165	0.0302
Diethyl phthalate		0.036	59	3	5.1%	0.0154	0.00912	0.00652	0.0176	0.0019
Dimethyl phthalate		0.0458	59	0	0.0%	0	0	0	0.0229	0
Di-n-butyl phthalate		0.0701	59	22	37.3%	0.352	0.246	0.212	0.0663	0.0611
Di-n-octyl phthalate		0.066	59	0	0.0%	0	0	0	0.033	0
Dinoseb		0.031	59	0	0.0%	0	0	0	0.0155	0
Diphenylamine		0.127	59	0	0.0%	0	0	0	0.0635	0
Ethyl Acrylate		0.655	59	0	0.0%	0	0	0	0.328	0
Ethyl methanesulfonate		0.148	59	0	0.0%	0	0	0	0.074	0
Ethyl Tert-Butyl Ether		0.627	59	0	0.0%	0	0	0	0.314	0
Ethylbenzene	0.36	0.174	59	57	96.6%	3.56	2.44	1.95	0.769	0.563
Fluoranthene		0.0725	59	16	27.1%	0.0144	0.0124	0.00869	0.0279	0.0138
Fluorene		0.0501	59	54	91.5%	0.0342	0.0258	0.0217	0.0106	0.00773
Formaldehyde	0.0098	0.0197	61	60	98.4%	4.88	4.76	4.74	2.38	1.27
Hexachloro-1,3-Butadiene VOC	0.672	0.64	59	0	0.0%	0	0	0	0.32	0
Hexachloro-1,3-ButadieneSVOC		0.0662	59	0	0.0%	0	0	0	0.0331	0
Hexachlorobenzene		0.0706	59	0	0.0%	0	0	0	0.0353	0
Hexachlorocyclopentadiene		0.109	59	0	0.0%	0	0	0	0.0544	0
Hexachloroethane		0.0283	59	0	0.0%	0	0	0	0.0141	0
Hexachloropropene		0.032	59	0	0.0%	0	0	0	0.016	0
Hexanaldehyde	0.0206	0.0164	61	46	75.4%	0.628	0.532	0.484	0.131	0.147
Indeno(123-cd)pyrene		0.0728	59	0	0.0%	0	0	0	0.0364	0
Isodrin		0.023	59	0	0.0%	0	0	0	0.0115	0
Isophorone		0.113	59	0	0.0%	0	0	0	0.0565	0
Isosafrole		0.029	59	0	0.0%	0	0	0	0.0145	0
Isovaleraldehyde	0.0431	0.0141	61	18	29.5%	0.176	0.159	0.141	0.0352	0.0441
Lead (TSP)	0.00001		59	59	100.0%	0.0167	0.014	0.0138	0.00689	0.00366
m,p-Tolualdehyde	0.0118		30	9	30.0%	0.762	0.295	0.221	0.0644	0.15
m/p -Xylene	0.869	0.217	59	59	100.0%	8.64	7.37	5.46	2.11	1.49

SOUTHFIELD MONITORING SITE - AIRS ID 261250010										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
Manganese (TSP)	0.00001		59	59	100.0%	0.0488	0.0359	0.0323	0.0162	0.00992
Methyl Ethyl Ketone	5.01	0.442	59	48	81.4%	11.4	6.74	5.58	2.53	1.95
Methyl Isobutyl Ketone	3.44	0.615	59	3	5.1%	1.39	0.806	0.451	0.337	0.155
Methyl Methacrylate		0.737	59	0	0.0%	0	0	0	0.369	0
Methyl methanesulfonate		0.0825	59	0	0.0%	0	0	0	0.0413	0
Methyl Tert-Butyl Ether	0.22	0.649	59	4	6.8%	2.06	0.44	0.21	0.351	0.228
Methylene Chloride	0.799	0.208	59	59	100.0%	1150	656	617	62.9	198
Naphthalene		0.0773	59	59	100.0%	0.632	0.603	0.583	0.292	0.13
n-Butyraldehyde	0.0206		30	26	86.7%	0.401	0.351	0.295	0.124	0.0982
Nickel (TSP)	0.00001		59	59	100.0%	0.0969	0.0118	0.00799	0.0037	0.0125
Nitrobenzene		0.124	59	0	0.0%	0	0	0	0.0622	0
N-Nitrosodibutylamine		0.112	59	0	0.0%	0	0	0	0.0561	0
N-Nitrosodiethylamine		0.036	59	0	0.0%	0	0	0	0.018	0
N-Nitrosodimethylamine		0.131	59	0	0.0%	0	0	0	0.0654	0
N-Nitrosodipropylamine		0.107	59	0	0.0%	0	0	0	0.0537	0
N-Nitrosomethylethylamine		0.035	59	0	0.0%	0	0	0	0.0175	0
N-Nitrosopiperidine		0.0866	59	0	0.0%	0	0	0	0.0433	0
N-Nitrosopyrrolidine		0.037	59	0	0.0%	0	0	0	0.0185	0
n-Octane		0.28	59	40	67.8%	2.2	1.5	1.45	0.473	0.461
o-Tolualdehyde	0.0118		30	0	0.0%	0	0	0	0.00588	0
o-Toluidine		0.038	59	0	0.0%	0	0	0	0.019	0
o-xylene	0.187	0.217	59	58	98.3%	3.2	2.63	2.22	0.87	0.558
Pentachlorobenzene		0.0487	59	0	0.0%	0	0	0	0.0244	0
Pentachloroethane		0.044	59	0	0.0%	0	0	0	0.022	0
Pentachloronitrobenzene		0.0519	59	0	0.0%	0	0	0	0.0259	0
Pentachlorophenol		0.0737	59	0	0.0%	0	0	0	0.0369	0
Phenacetin		0.0819	59	0	0.0%	0	0	0	0.041	0
Phenanthrene		0.0515	59	59	100.0%	0.0918	0.0856	0.0655	0.0244	0.0195
Phenol		0.112	59	11	18.6%	0.0962	0.0825	0.0798	0.0578	0.00911
Pronamide		0.0629	59	0	0.0%	0	0	0	0.0314	0
Propionaldehyde	0.196	0.0166	61	51	83.6%	0.719	0.657	0.53	0.249	0.155
Propylene		0.0861	59	59	100.0%	5.95	2.94	2.31	1.31	0.792
Pyrene		0.0527	59	10	16.9%	0.00704	0.00569	0.00431	0.0225	0.00859

SOUTHFIELD MONITORING SITE - AIRS ID 261250010

CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
Pyridine		0.059	59	0	0.0%	0	0	0	0.0295	0
Safrole		0.029	59	0	0.0%	0	0	0	0.0145	0
Styrene	0.23	0.298	59	28	47.5%	1.17	0.661	0.544	0.205	0.167
Tert-Amyl Methyl Ether		0.502	59	0	0.0%	0	0	0	0.251	0
Tetrachloroethene	0.482	0.407	59	19	32.2%	1.42	1.13	0.937	0.309	0.237
Tolualdehydes		0.0393	31	30	96.8%	0.584	0.533	0.463	0.228	0.144
Toluene	0.264	0.226	59	59	100.0%	14.8	13.1	9.82	3.92	2.62
trans-1,2-Dichloroethene		0.238	59	0	0.0%	0	0	0	0.119	0
trans-1,3-Dichloropropene	0.281	0.499	59	0	0.0%	0	0	0	0.25	0
Trichloroethene	0.204	0.376	59	2	3.4%	0.995	0.086	0	0.2	0.106
Trichloroflouromethane	0.27	0.225	59	59	100.0%	3.22	2.96	2.93	1.65	0.404
Trichlorotrifluoroethane		0.536	59	59	100.0%	1.29	1.25	1.2	0.883	0.174
Valeraldehyde	0.0775	0.0106	61	40	65.6%	0.37	0.247	0.213	0.0918	0.0726
Vinyl Chloride	0.112	0.153	59	0	0.0%	0	0	0	0.0767	0

RIVER ROUGE MONITORING SITE - AIRS ID 261630005

CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
1,1,1-Trichloroethane	0.262	0.327	60	27	45.0%	1.14	0.316	0.312	0.194	0.138
1,1,2,2-Tetrachloroethane	0.556	0.412	60	0	0.0%	0	0	0	0.242	0.0363
1,1,2-Trichloroethane	0.262	0.327	60	0	0.0%	0	0	0	0.147	0.0165
1,1-Dichloroethane	0.36	0.324	60	0	0.0%	0	0	0	0.171	0.00918
1,1-Dichloroethene	0.182	0.396	60	0	0.0%	0	0	0	0.145	0.054
1,2,4,5-Tetrachlorobenzene		0.0527	30	0	0.0%	0	0	0	0.0264	0
1,2,4-Trichlorobenzene VOC	0.601	0.445	60	0	0.0%	0	0	0	0.262	0.0393
1,2,4-Trichlorobenzene SVOC		0.0681	30	0	0.0%	0	0	0	0.0341	0
1,2,4-Trimethylbenzene	0.305	0.344	60	55	91.7%	3.45	2.95	2.55	0.909	0.718
1,2-Dibromoethane	0.415	0.615	60	0	0.0%	0	0	0	0.257	0.0504
1,2-Dichlorobenzene VOC	0.397	0.361	60	1	1.7%	0.738	0	0	0.198	0.0714
1,2-Dichlorobenzene SVOC		0.0548	30	1	3.3%	0.0183	0	0	0.0271	0.00165
1,2-Dichloroethane	0.348	0.243	60	0	0.0%	0	0	0	0.148	0.0265

RIVER ROUGE MONITORING SITE - AIRS ID 261630005										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
1,2-Dichloropropane	0.273	0.323	60	0	0.0%	0	0	0	0.149	0.0128
1,3,5-Trimethylbenzene	0.28	0.344	60	29	48.3%	1.13	0.934	0.828	0.282	0.233
1,3-Butadiene	0.0885	0.155	60	20	33.3%	0.597	0.55	0.456	0.11	0.129
1,3-Dichlorobenzene VOC	0.319	0.301	60	1	1.7%	0.714	0	0	0.149	0.077
1,3-Dichlorobenzene SVOC		0.0603	30	0	0.0%	0	0	0	0.0301	0
1,3-Dintrobenzene		0.038	30	0	0.0%	0	0	0	0.019	0
1,4-Dichlorobenzene VOC	0.319	0.541	60	10	16.7%	0.759	0.587	0.403	0.205	0.107
1,4-Dichlorobenzene SVOC		0.0543	30	30	100.0%	0.314	0.178	0.144	0.0779	0.0592
1,4-Naphthoquinone		0.029	30	0	0.0%	0	0	0	0.0145	0
1-Naphthylamine		0.0271	30	0	0.0%	0	0	0	0.0136	0
2,2,4-Trimethylpentane	0.0794		30	30	100.0%	2.1	1.14	0.921	0.579	0.368
2,3,4,6-Tetrachlorophenol		0.0484	30	0	0.0%	0	0	0	0.0242	0
2,4,5-Trichlorphenol		0.0345	30	0	0.0%	0	0	0	0.0173	0
2,4,6-Trichlorophenol		0.0418	30	0	0.0%	0	0	0	0.0209	0
2,4-Dichlorophenol		0.0728	30	0	0.0%	0	0	0	0.0364	0
2,4-Dimethylphenol		0.0882	30	0	0.0%	0	0	0	0.0441	0
2,4-Dinitrophenol		0.0503	30	0	0.0%	0	0	0	0.0251	0
2,4-Dinitrotoluene		0.0486	30	0	0.0%	0	0	0	0.0243	0
2,5-dimethylbenzaldehyde	0.0176	0.0274	31	1	3.2%	0.0011	0	0	0.00856	0.00139
2,6-Dichlorophenol		0.0905	30	0	0.0%	0	0	0	0.0453	0
2,6-Dinitrotoluene		0.0469	30	0	0.0%	0	0	0	0.0235	0
2-Acetylaminofluorene		0.017	30	0	0.0%	0	0	0	0.0085	0
2-Chloro-1,3-Butadiene	0.109	0.362	60	0	0.0%	0	0	0	0.118	0.0639
2-Chloronaphthalene		0.0924	30	0	0.0%	0	0	0	0.0462	0
2-Chlorophenol		0.05	30	0	0.0%	0	0	0	0.025	0
2-Methyl Naphthalene		0.0568	30	30	100.0%	0.545	0.453	0.307	0.172	0.114
2-Methylphenol		0.0472	30	1	3.3%	0.0561	0	0	0.0247	0.00594
2-Naphthylamine		0.0511	30	0	0.0%	0	0	0	0.0256	0
2-Nitroaniline		0.0615	30	0	0.0%	0	0	0	0.0308	0
2-Nitrophenol		0.0504	30	0	0.0%	0	0	0	0.0252	0
2-Picoline		0.0558	30	0	0.0%	0	0	0	0.0279	0
3&4-Methylphenol		0.042	30	1	3.3%	0.0682	0	0	0.0226	0.00862

RIVER ROUGE MONITORING SITE - AIRS ID 261630005										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
3,3'-Dichlorobenzidine		0.0448	30	0	0.0%	0	0	0	0.0224	0
3,3'-Dimethylbenzidine		0.25	30	0	0.0%	0	0	0	0.125	0
3-Methylcholanthrene		0.032	30	0	0.0%	0	0	0	0.016	0
3-Nitroaniline		0.0435	30	0	0.0%	0	0	0	0.0218	0
4,6-Dinitro-2-methylphenol		0.0566	30	0	0.0%	0	0	0	0.0283	0
4-Aminobiphenyl		0.048	30	0	0.0%	0	0	0	0.024	0
4-Bromophenyl phenyl ether		0.0565	30	0	0.0%	0	0	0	0.0283	0
4-Chloro-3-methylphenol		0.084	30	0	0.0%	0	0	0	0.042	0
4-Chloroaniline		0.0842	30	0	0.0%	0	0	0	0.0421	0
4-Chlorophenyl-phenyl ether		0.034	30	0	0.0%	0	0	0	0.017	0
4-Dimethylaminoazobenzene		0.022	30	0	0.0%	0	0	0	0.011	0
4-Nitroaniline		0.052	30	0	0.0%	0	0	0	0.026	0
4-Nitrophenol		0.0368	30	0	0.0%	0	0	0	0.0184	0
5-Nitro-o-toluidine		0.026	30	0	0.0%	0	0	0	0.013	0
7,12-Dimethylbenz(a)anthracene		0.0952	30	0	0.0%	0	0	0	0.0476	0
Acenaphthene		0.0291	30	28	93.3%	0.0974	0.0743	0.0742	0.0198	0.0249
Acenaphthylene		0.0444	30	5	16.7%	0.0239	0.0147	0.00845	0.0204	0.0049
Acetaldehyde	0.0167	0.0252	31	30	96.8%	9.29	3.85	3.62	2.11	1.56
Acetone	0.0255	0.019	31	30	96.8%	13.4	4.87	3.35	2.37	2.23
Acetonitrile	0.873	0.42	60	3	5.0%	11.6	1.92	1.85	0.558	1.48
Acetophenone		0.0694	30	0	0.0%	0	0	0	0.0347	0
Acetylene		0.138	30	30	100.0%	5.13	4.89	4.58	2.22	1.35
Acrylonitrile	0.847	0.456	60	1	1.7%	0.159	0	0	0.324	0.1
Aniline		0.081	30	0	0.0%	0	0	0	0.0405	0
Anthracene		0.0853	30	5	16.7%	0.0102	0.00656	0.00457	0.0364	0.0143
Arsenic (TSP)	0.000016		65	65	100.0%	0.0128	0.00712	0.00468	0.00191	0.00177
Azobenzene		0.087	30	0	0.0%	0	0	0	0.0435	0
Benzaldehyde	0.0167	0.0174	31	28	90.3%	0.749	0.582	0.538	0.174	0.181
Benzene	0.224	0.128	60	60	100.0%	6.56	5.19	4.97	2.14	1.29
Benzidine		0.25	30	0	0.0%	0	0	0	0.125	0
Benzo(a)anthracene		0.0417	30	0	0.0%	0	0	0	0.0208	0
Benzo(a)pyrene		0.0909	30	0	0.0%	0	0	0	0.0454	0

RIVER ROUGE MONITORING SITE - AIRS ID 261630005										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
Benzo(b)fluoranthene		0.0867	30	0	0.0%	0	0	0	0.0434	0
Benzo(ghi)perylene		0.0753	30	0	0.0%	0	0	0	0.0376	0
Benzo(k)fluoranthene		0.118	30	0	0.0%	0	0	0	0.059	0
Benzyl alcohol		0.0408	30	0	0.0%	0	0	0	0.0204	0
Beryllium (TSP)	0.00001		65	65	100.0%	0.0011	0.00099	0.00049	0.000107	0.000194
bis (2-Chloroethyl)ether		0.0583	30	0	0.0%	0	0	0	0.0292	0
bis(2-Chloroethoxy)methane		0.0702	30	0	0.0%	0	0	0	0.0351	0
bis(2-Chloroisopropyl)ether		0.0554	30	0	0.0%	0	0	0	0.0277	0
bis(2-Ethylhexyl)phthalate		0.0581	30	3	10.0%	0.0173	0.00909	0.00677	0.0273	0.00568
Bromochloromethane		0.635	30	0	0.0%	0	0	0	0.318	0
Bromodichloromethane	0.489	0.402	60	0	0.0%	0	0	0	0.223	0.022
Bromoform	0.61	0.827	60	0	0.0%	0	0	0	0.359	0.0547
Bromomethane	0.155	0.35	60	2	3.3%	0.701	0.104	0	0.135	0.0885
Butyl benzyl phthalate		0.0583	30	0	0.0%	0	0	0	0.0292	0
Butyr/Isobutyraldehyde		0.0147	1	1	100.0%	3.74	0	0	3.74	
Cadmium (TSP)	0.000024		65	65	100.0%	0.00529	0.00168	0.00141	0.000621	0.000679
Carbazole		0.0627	30	0	0.0%	0	0	0	0.0313	0
Carbon Tetrachloride	0.239	0.503	60	60	100.0%	0.777	0.755	0.739	0.579	0.0985
Chlorobenzene	0.184	0.276	60	3	5.0%	0.334	0.272	0.23	0.124	0.0435
Chlorobenzilate		0.016	30	0	0.0%	0	0	0	0.008	0
Chloroethane	0.106	0.211	60	0	0.0%	0	0	0	0.0792	0.0266
Chloroform	0.332	0.244	60	2	3.3%	0.111	0.0488	0	0.143	0.0254
Chloromethane	0.128	0.124	60	60	100.0%	1.81	1.81	1.8	1.25	0.199
Chloromethyl Benzene	0.393	0.362	60	0	0.0%	0	0	0	0.189	0.00783
Chromium (TSP)	0.000014		65	65	100.0%	0.0119	0.0105	0.00977	0.00509	0.00212
Chromium VI (TSP)		0.000013	32	20	62.5%	0.000112	0.000111	8.83E-05	3.92E-05	3.57E-05
Chrysene		0.0529	30	0	0.0%	0	0	0	0.0264	0
cis-1,2-Dichloroethene	0.226	0.396	60	0	0.0%	0	0	0	0.156	0.043
cis-1,3-Dichloropropene	0.245	0.454	60	0	0.0%	0	0	0	0.175	0.0526
Crotonaldehyde	0.0225	0.0172	31	9	29.0%	0.373	0.341	0.229	0.0571	0.0959
Diallate		0.024	30	0	0.0%	0	0	0	0.012	0
Dibenzo(ah)anthracene		0.0793	30	0	0.0%	0	0	0	0.0396	0

RIVER ROUGE MONITORING SITE - AIRS ID 261630005										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
Dibenzofuran		0.0464	30	27	90.0%	0.0667	0.0588	0.055	0.0195	0.0183
Dibromochloromethane	0.503	0.682	60	0	0.0%	0	0	0	0.296	0.0451
Dichlorodiflouromethane	0.237	0.198	60	60	100.0%	4.04	3.77	3.75	2.88	0.465
Dichlorotetrafluoroethane		0.35	30	3	10.0%	0.0951	0.072	0.0706	0.165	0.0294
Diethyl phthalate		0.036	30	2	6.7%	0.0814	0.0103	0	0.0199	0.0117
Dimethyl phthalate		0.0458	30	0	0.0%	0	0	0	0.0229	0
Di-n-butyl phthalate		0.0701	30	2	6.7%	0.0446	0.0107	0	0.0345	0.00484
Di-n-octyl phthalate		0.066	30	0	0.0%	0	0	0	0.033	0
Dinoseb		0.031	30	0	0.0%	0	0	0	0.0155	0
Diphenylamine		0.127	30	0	0.0%	0	0	0	0.0635	0
Ethyl Acrylate		0.655	30	0	0.0%	0	0	0	0.328	0
Ethyl methanesulfonate		0.148	30	0	0.0%	0	0	0	0.074	0
Ethyl Tert-Butyl Ether		0.627	30	0	0.0%	0	0	0	0.314	0
Ethylbenzene	0.36	0.174	60	51	85.0%	2.33	2.3	2.15	0.674	0.536
Fluoranthene		0.0725	30	6	20.0%	0.0833	0.0156	0.0111	0.0331	0.0144
Fluorene		0.0501	30	29	96.7%	0.0664	0.0556	0.0486	0.0167	0.0174
Formaldehyde	0.0098	0.0197	31	30	96.8%	15.5	15.3	13.9	5.55	3.98
Halocarbon 113	0.261		30	30	100.0%	1.75	1.27	1.19	0.77	0.266
Halocarbon 114	0.294		30	0	0.0%	0	0	0	0.147	0
Hexachloro-1,3-Butadiene VOC	0.672	0.64	60	1	1.7%	1.12	0	0	0.341	0.102
Hexachloro-1,3-ButadieneSVOC		0.0662	30	0	0.0%	0	0	0	0.0331	0
Hexachlorobenzene		0.0706	30	0	0.0%	0	0	0	0.0353	0
Hexachlorocyclopentadiene		0.109	30	0	0.0%	0	0	0	0.0544	0
Hexachloroethane		0.0283	30	0	0.0%	0	0	0	0.0141	0
Hexachloropropene		0.032	30	0	0.0%	0	0	0	0.016	0
Hexanaldehyde	0.0206	0.0164	31	30	96.8%	0.94	0.84	0.619	0.314	0.227
Indeno(123-cd)pyrene		0.0728	30	0	0.0%	0	0	0	0.0364	0
Isodrin		0.023	30	0	0.0%	0	0	0	0.0115	0
Isophorone		0.113	30	0	0.0%	0	0	0	0.0565	0
Isosafrole		0.029	30	0	0.0%	0	0	0	0.0145	0
Isovaleraldehyde	0.0431	0.0141	31	17	54.8%	0.511	0.5	0.469	0.142	0.17
Lead (TSP)	0.00001		65	65	100.0%	0.0894	0.0547	0.0499	0.0196	0.0141

RIVER ROUGE MONITORING SITE - AIRS ID 261630005										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
m,p-Tolualdehyde	0.0118		30	8	26.7%	0.801	0.261	0.197	0.059	0.154
m/p -Xylene	0.869	0.217	60	52	86.7%	7.41	6.67	6.08	1.9	1.62
Manganese (TSP)	0.00001		65	65	100.0%	0.269	0.26	0.202	0.075	0.0541
Methyl Ethyl Ketone	5.01	0.442	60	29	48.3%	12.7	7.98	6.94	2.93	1.98
Methyl Isobutyl Ketone	3.44	0.615	60	2	3.3%	1.57	1.31	0	1.05	0.705
Methyl Methacrylate		0.737	30	0	0.0%	0	0	0	0.369	0
Methyl methanesulfonate		0.0825	30	0	0.0%	0	0	0	0.0413	0
Methyl Tert-Butyl Ether	0.22	0.649	60	7	11.7%	0.533	0.525	0.457	0.23	0.125
Methylene Chloride	0.799	0.208	60	51	85.0%	433	62.7	30.9	10.1	56.2
Naphthalene		0.0773	30	30	100.0%	2.41	1.97	1.18	0.486	0.533
n-Butyraldehyde	0.0206		30	29	96.7%	0.914	0.826	0.761	0.478	0.207
n-Hexane	1.76		30	7	23.3%	5.68	3.72	3.44	1.41	1.14
Nickel (TSP)	0.00001		65	65	100.0%	0.00932	0.00826	0.007	0.0025	0.00166
Nitrobenzene		0.124	30	0	0.0%	0	0	0	0.0622	0
N-Nitrosodibutylamine		0.112	30	0	0.0%	0	0	0	0.0561	0
N-Nitrosodiethylamine		0.036	30	0	0.0%	0	0	0	0.018	0
N-Nitrosodimethylamine		0.131	30	0	0.0%	0	0	0	0.0654	0
N-Nitrosodipropylamine		0.107	30	0	0.0%	0	0	0	0.0537	0
N-Nitrosomethylethylamine		0.035	30	0	0.0%	0	0	0	0.0175	0
N-Nitrosopiperidine		0.0866	30	0	0.0%	0	0	0	0.0433	0
N-Nitrosopyrrolidine		0.037	30	0	0.0%	0	0	0	0.0185	0
n-Octane		0.28	30	18	60.0%	1.08	0.651	0.608	0.29	0.216
o-Tolualdehyde	0.0118		30	0	0.0%	0	0	0	0.00588	0
o-Toluidine		0.038	30	0	0.0%	0	0	0	0.019	0
o-xylene	0.187	0.217	60	59	98.3%	3.09	2.62	2.61	0.824	0.666
Pentachlorobenzene		0.0487	30	0	0.0%	0	0	0	0.0244	0
Pentachloroethane		0.044	30	0	0.0%	0	0	0	0.022	0
Pentachloronitrobenzene		0.0519	30	0	0.0%	0	0	0	0.0259	0
Pentachlorophenol		0.0737	30	0	0.0%	0	0	0	0.0369	0
Phenacetin		0.0819	30	0	0.0%	0	0	0	0.041	0
Phenanthrene		0.0515	30	28	93.3%	0.102	0.0771	0.0757	0.0312	0.0271
Phenol		0.112	30	4	13.3%	0.271	0.172	0.143	0.071	0.0458

RIVER ROUGE MONITORING SITE - AIRS ID 261630005

CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
Pronamide		0.0629	30	0	0.0%	0	0	0	0.0314	0
Propionaldehyde	0.196	0.0166	31	30	96.8%	2.72	1.17	1.05	0.611	0.464
Propylene		0.0861	30	30	100.0%	6.06	4.61	4.03	1.74	1.42
Pyrene		0.0527	30	3	10.0%	0.00668	0.00592	0.00547	0.0243	0.0062
Pyridine		0.059	30	0	0.0%	0	0	0	0.0295	0
Safrole		0.029	30	0	0.0%	0	0	0	0.0145	0
Styrene	0.23	0.298	60	31	51.7%	4.25	1.22	1.11	0.285	0.563
Tert-Amyl Methyl Ether		0.502	30	0	0.0%	0	0	0	0.251	0
Tetrachloroethene	0.482	0.407	60	19	31.7%	15.9	2.24	2.1	0.655	2.05
Tolualdehydes		0.0393	1	1	100.0%	1.44	0	0	1.44	
Toluene	0.264	0.226	60	60	100.0%	13.3	12.4	11.4	4.31	2.98
trans-1,2-Dichloroethene		0.238	60	0	0.0%	0	0	0	0.145	0.026
trans-1,3-Dichloropropene	0.281	0.499	60	0	0.0%	0	0	0	0.195	0.0549
Trichloroethene	0.204	0.376	60	5	8.3%	1.74	1.57	0.961	0.212	0.295
Trichloroflouromethane	0.27	0.225	60	60	100.0%	3.69	2.59	2.41	1.57	0.415
Trichlorotrifluoroethane		0.536	30	30	100.0%	1.22	1.2	1.17	0.927	0.17
Valeraldehyde	0.0775	0.0106	31	30	96.8%	3.2	1.28	1.26	0.779	0.545
Vinyl Chloride	0.112	0.153	60	0	0.0%	0	0	0	0.0665	0.0103

NORTH DELRAY MONITORING SITE - AIRS ID 261630015

CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL /2 µg/m³
1,1,1-Trichloroethane	0.262	0.327	26	4	15.4%	0.469	0.312	0.3	0.163	0.0823
1,1,2,2-Tetrachloroethane	0.556	0.412	26	0	0.0%	0	0	0	0.278	0
1,1,2-Trichloroethane	0.262	0.327	26	0	0.0%	0	0	0	0.131	0
1,1-Dichloroethane	0.36	0.324	26	0	0.0%	0	0	0	0.18	0
1,1-Dichloroethene	0.182	0.396	26	0	0.0%	0	0	0	0.0912	0
1,2,4,5-Tetrachlorobenzene		0.0527	29	0	0.0%	0	0	0	0.0264	0
1,2,4-Trichlorobenzene VOC	0.601	0.445	26	0	0.0%	0	0	0	0.301	0
1,2,4-Trichlorobenzene SVOC		0.0681	29	0	0.0%	0	0	0	0.0341	0

NORTH DELRAY MONITORING SITE - AIRS ID 261630015										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL /2 µg/m³
1,2,4-Trimethylbenzene	0.305	0.344	26	25	96.2%	4.28	2.85	2.43	1.06	0.952
1,2-Dibromoethane	0.415	0.615	26	0	0.0%	0	0	0	0.208	0
1,2-Dichlorobenzene VOC	0.397	0.361	26	0	0.0%	0	0	0	0.198	0
1,2-Dichlorobenzene SVOC		0.0548	29	0	0.0%	0	0	0	0.0274	0
1,2-Dichloroethane	0.348	0.243	26	0	0.0%	0	0	0	0.174	0
1,2-Dichloropropane	0.273	0.323	26	0	0.0%	0	0	0	0.136	0
1,3,5-Trimethylbenzene	0.28	0.344	26	9	34.6%	1.24	0.794	0.694	0.297	0.273
1,3-Butadiene	0.0885	0.155	26	0	0.0%	0	0	0	0.0443	0
1,3-Dichlorobenzene VOC	0.319	0.301	26	0	0.0%	0	0	0	0.159	0
1,3-Dichlorobenzene SVOC		0.0603	29	0	0.0%	0	0	0	0.0301	0
1,3-Dintrobenzene		0.038	29	0	0.0%	0	0	0	0.019	0
1,4-Dichlorobenzene VOC	0.319	0.541	29	9	31.0%	83.3	1.23	1.03	3.27	15.7
1,4-Dichlorobenzene SVOC		0.0543	29	28	96.6%	0.263	0.25	0.214	0.0932	0.0639
1,4-Naphthoquinone		0.029	29	0	0.0%	0	0	0	0.0145	0
1-Naphthylamine		0.0271	29	0	0.0%	0	0	0	0.0136	0
2,2,4-Trimethylpentane	0.0794		26	24	92.3%	2.49	2.22	1.74	0.692	0.587
2,3,4,6-Tetrachlorophenol		0.0484	29	0	0.0%	0	0	0	0.0242	0
2,4,5-Trichlorphenol		0.0345	29	0	0.0%	0	0	0	0.0173	0
2,4,6-Trichlorophenol		0.0418	29	0	0.0%	0	0	0	0.0209	0
2,4-Dichlorophenol		0.0728	29	0	0.0%	0	0	0	0.0364	0
2,4-Dimethylphenol		0.0882	29	0	0.0%	0	0	0	0.0441	0
2,4-Dinitrophenol		0.0503	29	0	0.0%	0	0	0	0.0251	0
2,4-Dinitrotoluene		0.0486	29	0	0.0%	0	0	0	0.0243	0
2,5-dimethylbenzaldehyde	0.0176	0.0274	29	0	0.0%	0	0	0	0.00898	0.000908
2,6-Dichlorophenol		0.0905	29	0	0.0%	0	0	0	0.0453	0
2,6-Dinitrotoluene		0.0469	29	0	0.0%	0	0	0	0.0235	0
2-Acetylaminofluorene		0.017	29	0	0.0%	0	0	0	0.0085	0
2-Chloro-1,3-Butadiene	0.109	0.362	26	0	0.0%	0	0	0	0.0543	0
2-Chloronaphthalene		0.0924	29	0	0.0%	0	0	0	0.0462	0
2-Chlorophenol		0.05	29	0	0.0%	0	0	0	0.025	0
2-Methyl Naphthalene		0.0568	29	29	100.0%	1.06	0.745	0.398	0.23	0.21

NORTH DELRAY MONITORING SITE - AIRS ID 261630015										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL /2 µg/m³
2-Methylphenol		0.0472	29	2	6.9%	0.0552	0.0114	0	0.0243	0.00637
2-Naphthylamine		0.0511	29	0	0.0%	0	0	0	0.0256	0
2-Nitroaniline		0.0615	29	0	0.0%	0	0	0	0.0308	0
2-Nitrophenol		0.0504	29	0	0.0%	0	0	0	0.0252	0
2-Picoline		0.0558	29	0	0.0%	0	0	0	0.0279	0
3&4-Methylphenol		0.042	29	3	10.3%	0.17	0.0288	0.0133	0.0261	0.0277
3,3'-Dichlorobenzidine		0.0448	29	0	0.0%	0	0	0	0.0224	0
3,3'-Dimethylbenzidine		0.25	29	0	0.0%	0	0	0	0.125	0
3-Methylcholanthrene		0.032	29	0	0.0%	0	0	0	0.016	0
3-Nitroaniline		0.0435	29	0	0.0%	0	0	0	0.0218	0
4,6-Dinitro-2-methylphenol		0.0566	29	0	0.0%	0	0	0	0.0283	0
4-Aminobiphenyl		0.048	29	0	0.0%	0	0	0	0.024	0
4-Bromophenyl phenyl ether		0.0565	29	0	0.0%	0	0	0	0.0283	0
4-Chloro-3-methylphenol		0.084	29	0	0.0%	0	0	0	0.042	0
4-Chloroaniline		0.0842	29	0	0.0%	0	0	0	0.0421	0
4-Chlorophenyl-phenyl ether		0.034	29	0	0.0%	0	0	0	0.017	0
4-Dimethylaminoazobenzene		0.022	29	0	0.0%	0	0	0	0.011	0
4-Nitroaniline		0.052	29	0	0.0%	0	0	0	0.026	0
4-Nitrophenol		0.0368	29	0	0.0%	0	0	0	0.0184	0
5-Nitro-o-toluidine		0.026	29	0	0.0%	0	0	0	0.013	0
7,12-Dimethylbenz(a)anthracene		0.0952	29	0	0.0%	0	0	0	0.0476	0
Acenaphthene		0.0291	29	26	89.7%	0.28	0.153	0.115	0.0373	0.0588
Acenaphthylene		0.0444	29	12	41.4%	0.0403	0.0118	0.00973	0.0172	0.00857
Acetaldehyde	0.0167	0.0252	29	29	100.0%	9.26	4.09	3.55	2.17	1.57
Acetone	0.0255	0.019	29	29	100.0%	17.8	5.91	3.61	2.65	3.1
Acetonitrile	0.873	0.42	26	2	7.7%	1.11	0.956	0	0.483	0.164
Acetophenone		0.0694	29	0	0.0%	0	0	0	0.0347	0
Acrylonitrile	0.847	0.456	26	12	46.2%	2.97	2.08	2.05	0.914	0.679
Aniline		0.081	29	1	3.4%	0.0368	0	0	0.0404	0.000689
Anthracene		0.0853	29	6	20.7%	0.0142	0.00846	0.00741	0.0353	0.0147
Arsenic (TSP)	0.000016		65	65	100.0%	0.00848	0.00524	0.00522	0.00213	0.00131

NORTH DELRAY MONITORING SITE - AIRS ID 261630015										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL /2 µg/m³
Azobenzene		0.087	29	0	0.0%	0	0	0	0.0435	0
Benzaldehyde	0.0167	0.0174	29	23	79.3%	0.77	0.451	0.269	0.117	0.158
Benzene	0.224	0.128	26	26	100.0%	11.4	8.2	5.8	2.67	2.45
Benzidine		0.25	29	0	0.0%	0	0	0	0.125	0
Benzo(a)anthracene		0.0417	29	3	10.3%	0.0105	0.00842	0.00422	0.0195	0.00416
Benzo(a)pyrene		0.0909	29	0	0.0%	0	0	0	0.0454	0
Benzo(b)fluoranthene		0.0867	29	0	0.0%	0	0	0	0.0434	0
Benzo(ghi)perylene		0.0753	29	0	0.0%	0	0	0	0.0376	0
Benzo(k)fluoranthene		0.118	29	0	0.0%	0	0	0	0.059	0
Benzyl alcohol		0.0408	29	0	0.0%	0	0	0	0.0204	0
Beryllium (TSP)	0.00001		65	65	100.0%	0.00524	0.001	0.000527	0.000187	0.000649
bis (2-Chloroethyl)ether		0.0583	29	0	0.0%	0	0	0	0.0292	0
bis(2-Chloroethoxy)methane		0.0702	29	0	0.0%	0	0	0	0.0351	0
bis(2-Chloroisopropyl)ether		0.0554	29	0	0.0%	0	0	0	0.0277	0
bis(2-Ethylhexyl)phthalate		0.0581	29	7	24.1%	0.116	0.07	0.0367	0.0306	0.02
Bromodichloromethane	0.489	0.402	26	0	0.0%	0	0	0	0.245	0
Bromoform	0.61	0.827	26	0	0.0%	0	0	0	0.305	0
Bromomethane	0.155	0.35	26	0	0.0%	0	0	0	0.0777	0
Butyl benzyl phthalate		0.0583	29	0	0.0%	0	0	0	0.0292	0
Butyr/Isobutyraldehyde		0.0147	1	1	100.0%	0.183	0	0	0.183	
Cadmium (TSP)	0.000024		65	65	100.0%	0.029	0.00232	0.00209	0.00122	0.00354
Carbazole		0.0627	29	0	0.0%	0	0	0	0.0313	0
Carbon Tetrachloride	0.239	0.503	26	25	96.2%	0.896	0.704	0.659	0.546	0.134
Chlorobenzene	0.184	0.276	26	0	0.0%	0	0	0	0.0921	0
Chlorobenzilate		0.016	29	0	0.0%	0	0	0	0.008	0
Chloroethane	0.106	0.211	26	0	0.0%	0	0	0	0.0528	0
Chloroform	0.332	0.244	26	2	7.7%	0.405	0.344	0	0.182	0.0573
Chloromethane	0.128	0.124	26	24	92.3%	2.58	2.08	1.91	1.37	0.456
Chloromethyl Benzene	0.393	0.362	26	0	0.0%	0	0	0	0.197	0
Chromium (TSP)	0.000014		65	65	100.0%	0.0111	0.00965	0.00958	0.00574	0.00168
Chrysene		0.0529	29	3	10.3%	0.0116	0.00702	0.00192	0.0244	0.0062

NORTH DELRAY MONITORING SITE - AIRS ID 261630015										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL /2 µg/m³
cis-1,2-Dichloroethene	0.226	0.396	26	0	0.0%	0	0	0	0.113	0
cis-1,3-Dichloropropene	0.245	0.454	26	0	0.0%	0	0	0	0.123	0
Crotonaldehyde	0.0225	0.0172	29	9	31.0%	0.602	0.321	0.201	0.0659	0.127
Diallate		0.024	29	0	0.0%	0	0	0	0.012	0
Dibenzo(ah)anthracene		0.0793	29	0	0.0%	0	0	0	0.0396	0
Dibenzofuran		0.0464	29	29	100.0%	0.3	0.111	0.104	0.0344	0.0583
Dibromochloromethane	0.503	0.682	26	0	0.0%	0	0	0	0.251	0
Dichlorodiflouromethane	0.237	0.198	26	24	92.3%	4.17	3.27	3.24	2.69	0.683
Diethyl phthalate		0.036	29	1	3.4%	0.0464	0	0	0.019	0.00528
Dimethyl phthalate		0.0458	29	0	0.0%	0	0	0	0.0229	0
Di-n-butyl phthalate		0.0701	29	1	3.4%	0.00781	0	0	0.0341	0.00505
Di-n-octyl phthalate		0.066	29	0	0.0%	0	0	0	0.033	0
Dinoseb		0.031	29	0	0.0%	0	0	0	0.0155	0
Diphenylamine		0.127	29	0	0.0%	0	0	0	0.0635	0
Ethyl methanesulfonate		0.148	29	0	0.0%	0	0	0	0.074	0
Ethylbenzene	0.36	0.174	26	16	61.5%	1.78	1.66	1.41	0.59	0.475
Fluoranthene		0.0725	29	12	41.4%	0.0775	0.0519	0.0441	0.0312	0.0156
Fluorene		0.0501	29	29	100.0%	0.271	0.105	0.0954	0.0313	0.0529
Formaldehyde	0.0098	0.0197	29	29	100.0%	14.6	8.81	7.58	3.94	2.76
Halocarbon 113	0.261		26	22	84.6%	5.67	5.39	4.94	2.08	1.65
Halocarbon 114	0.294		26	0	0.0%	0	0	0	0.147	0
Hexachloro-1,3-Butadiene VOC	0.672	0.64	26	0	0.0%	0	0	0	0.336	0
Hexachloro-1,3-ButadieneSVOC		0.0662	29	0	0.0%	0	0	0	0.0331	0
Hexachlorobenzene		0.0706	29	0	0.0%	0	0	0	0.0353	0
Hexachlorocyclopentadiene		0.109	29	0	0.0%	0	0	0	0.0544	0
Hexachloroethane		0.0283	29	0	0.0%	0	0	0	0.0141	0
Hexachloropropene		0.032	29	0	0.0%	0	0	0	0.016	0
Hexanaldehyde	0.0206	0.0164	29	26	89.7%	1.1	0.529	0.479	0.2	0.216
Indeno(123-cd)pyrene		0.0728	29	0	0.0%	0	0	0	0.0364	0
Isodrin		0.023	29	0	0.0%	0	0	0	0.0115	0
Isophorone		0.113	29	0	0.0%	0	0	0	0.0565	0

NORTH DELRAY MONITORING SITE - AIRS ID 261630015										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL /2 µg/m³
Isosafrole		0.029	29	0	0.0%	0	0	0	0.0145	0
Isovaleraldehyde	0.0431	0.0141	29	19	65.5%	0.514	0.321	0.176	0.0984	0.105
Lead (TSP)	0.00001		65	65	100.0%	0.0493	0.0467	0.0442	0.022	0.01
m,p-Tolualdehyde	0.0118		28	10	35.7%	0.718	0.344	0.319	0.0803	0.158
m/p -Xylene	0.869	0.217	26	20	76.9%	5.04	3.89	3.82	1.65	1.22
Manganese (TSP)	0.00001		65	65	100.0%	0.188	0.187	0.175	0.093	0.043
Methyl Ethyl Ketone	5.01	0.442	26	2	7.7%	8.78	5.06	0	2.85	1.31
Methyl Isobutyl Ketone	3.44	0.615	26	0	0.0%	0	0	0	1.72	0
Methyl methanesulfonate		0.0825	29	0	0.0%	0	0	0	0.0413	0
Methyl Tert-Butyl Ether	0.22	0.649	26	2	7.7%	0.625	0.455	0	0.143	0.119
Methylene Chloride	0.799	0.208	26	19	73.1%	96	18.6	14.4	6.66	18.8
Naphthalene		0.0773	29	29	100.0%	13.6	3.55	3.31	1.26	2.55
n-Butyraldehyde	0.0206		28	28	100.0%	0.82	0.678	0.611	0.304	0.174
n-Hexane	1.76		26	11	42.3%	9.85	7.9	7.57	2.83	2.87
Nickel (TSP)	0.00001		65	65	100.0%	0.00927	0.00915	0.00786	0.00339	0.00193
Nitrobenzene		0.124	29	0	0.0%	0	0	0	0.0622	0
N-Nitrosodibutylamine		0.112	29	0	0.0%	0	0	0	0.0561	0
N-Nitrosodiethylamine		0.036	29	0	0.0%	0	0	0	0.018	0
N-Nitrosodimethylamine		0.131	29	0	0.0%	0	0	0	0.0654	0
N-Nitrosodipropylamine		0.107	29	0	0.0%	0	0	0	0.0537	0
N-Nitrosomethylethylamine		0.035	29	0	0.0%	0	0	0	0.0175	0
N-Nitrosopiperidine		0.0866	29	0	0.0%	0	0	0	0.0433	0
N-Nitrosopyrrolidine		0.037	29	0	0.0%	0	0	0	0.0185	0
o-Tolualdehyde	0.0118		28	0	0.0%	0	0	0	0.00546	0.00154
o-Toluidine		0.038	29	0	0.0%	0	0	0	0.019	0
o-xylene	0.187	0.217	26	26	100.0%	2.56	1.88	1.69	0.803	0.588
Pentachlorobenzene		0.0487	29	0	0.0%	0	0	0	0.0244	0
Pentachloroethane		0.044	29	0	0.0%	0	0	0	0.022	0
Pentachloronitrobenzene		0.0519	29	0	0.0%	0	0	0	0.0259	0
Pentachlorophenol		0.0737	29	0	0.0%	0	0	0	0.0369	0
Phenacetin		0.0819	29	0	0.0%	0	0	0	0.041	0

NORTH DELRAY MONITORING SITE - AIRS ID 261630015

CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL /2 µg/m³
Phenanthrene		0.0515	29	29	100.0%	0.333	0.174	0.173	0.0535	0.0708
Phenol		0.112	29	10	34.5%	0.475	0.176	0.155	0.0901	0.0823
Pronamide		0.0629	29	0	0.0%	0	0	0	0.0314	0
Propionaldehyde	0.196	0.0166	29	29	100.0%	3.13	2.3	1.34	0.609	0.649
Pyrene		0.0527	29	10	34.5%	0.05	0.0267	0.0253	0.0227	0.00978
Pyridine		0.059	29	0	0.0%	0	0	0	0.0295	0
Safrole		0.029	29	0	0.0%	0	0	0	0.0145	0
Styrene	0.23	0.298	26	10	38.5%	1.6	0.641	0.385	0.248	0.305
Tetrachloroethene	0.482	0.407	26	2	7.7%	1.3	0.569	0	0.294	0.215
Tolualdehydes		0.0393	1	1	100.0%	0.619	0	0	0.619	
Toluene	0.264	0.226	26	26	100.0%	15.1	14.8	10.4	5.11	3.85
trans-1,2-Dichloroethene		0.238	26	0	0.0%	0	0	0	0.171	0
trans-1,3-Dichloropropene	0.281	0.499	26	1	3.8%	3.14	0	0	0.256	0.589
Trichloroethene	0.204	0.376	26	1	3.8%	0.501	0	0	0.117	0.0782
Trichloroflouromethane	0.27	0.225	26	25	96.2%	10.5	7.82	7	3.98	2.44
Valeraldehyde	0.0775	0.0106	29	28	96.6%	1.19	0.49	0.423	0.247	0.209
Vinyl Chloride	0.112	0.153	26	0	0.0%	0	0	0	0.0562	0

YPSILANTI MONITORING SITE - AIRS ID 261610008

CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
Acetaldehyde	0.0167	0.0252	29	29	100.0%	1.82	1.68	1.68	1.09	0.354
Acetone	0.0255	0.019	29	29	100.0%	3.76	3.1	2.5	1.78	0.683
Arsenic (TSP)	0.000016		18	18	100.0%	0.00213	0.00124	0.00118	0.000778	0.000435
Benzaldehyde	0.0167	0.0174	29	21	72.4%	0.469	0.26	0.191	0.0809	0.0989
Beryllium (TSP)	0.00001		18	18	100.0%	0.00005	0.00005	0.00005	4.99E-05	1E-07
Cadmium (TSP)	0.000024		18	18	100.0%	0.000328	0.000277	0.000243	0.000166	7.02E-05
Chromium (TSP)	0.000014		18	18	100.0%	0.0029	0.0026	0.00245	0.00223	0.000257
Crotonaldehyde	0.0225	0.0172	29	3	10.3%	0.229	0.0573	0.0573	0.022	0.0416

YPSILANTI MONITORING SITE - AIRS ID 261610008										
CHEMICAL NAME	MDL MDEQ µg/m³	MDL ERG µg/m³	Num Obs	Obs > MDL	% Det	Max 1 µg/m³	Max 2 µg/m³	Max 3 µg/m³	AVG w/ND=MDL/2 µg/m³	St Dev w/ND=MDL/2 µg/m³
Formaldehyde	0.0098	0.0197	29	29	100.0%	7.32	5.74	4.86	2.28	1.6
Hexanaldehyde	0.0206	0.0164	29	19	65.5%	0.93	0.52	0.328	0.136	0.197
Isovaleraldehyde	0.0431	0.0141	29	12	41.4%	0.338	0.211	0.201	0.0683	0.0802
Lead (TSP)	0.00001		18	18	100.0%	0.0103	0.00718	0.00715	0.00524	0.00219
Manganese (TSP)	0.00001		18	18	100.0%	0.0229	0.0154	0.0139	0.00915	0.00467
n-Butyraldehyde	0.0206		29	23	79.3%	0.36	0.301	0.266	0.095	0.088
Nickel (TSP)	0.00001		18	18	100.0%	0.00153	0.00144	0.00112	0.000841	0.00031
o-Tolualdehyde	0.0118		29	1	3.4%	0.211	0	0	0.013	0.0382
o-xylene	0.187	0.217	29	26	89.7%	1.43	1.25	1.23	0.529	0.356
Propionaldehyde	0.196	0.0166	29	17	58.6%	0.43	0.39	0.38	0.163	0.0978
Valeraldehyde	0.0775	0.0106	29	8	27.6%	0.398	0.229	0.211	0.0716	0.0799

APPENDIX E: Cancer Narrative

Table E-1: DATI Cancer Risk Drivers, Their Health Effects, Quantitative Risk, and Weight of Evidence Category

CHEMICAL (CAS NO.)*	TUMOR TYPE	EXTRAPOLATION METHOD	AIR UNIT RISKS	ROUTE	WEIGHT OF EVIDENCE (USEPA 1986 CANCER RISK GUIDELINES)
1,3-Butadiene (106-99-0)	Leukemia	Linear extrapolation from LEC01 (0.254 ppm); LEC01 derived from linear relative rate model (RR = 1 + (B)(x)) using life table analysis with leukemia incidence data; an adjustment factor of 2 was applied.	$3x10^{-2}$ per mg/m³	Inhalation	Not Available
1,4-Dichlorobenzene (106-46-7)	Liver adenomas and carcinomas	Linearized multistage: Low-dose linearity utilizing maximum likelihood estimates	0.0000069 per µg/m³	oral	Not Available
Acetaldehyde (75-07-0)	Nasal squamous cell carcinoma or adenocarcinoma	Linearized multistage-variable exposure input form (extra risk)	$2.2x10^{-6}$ per mg/m³	Inhalation	B2: Probable human carcinogen - based on sufficient evidence of carcinogenicity in animals
Acrylonitrile (107-13-1)	Respiratory cancer	Average relative risk	$6.8x10^{-2}$ per mg/m³	Inhalation	B1: Probable human carcinogen - based on limited evidence of carcinogenicity in humans
Arsenic (7440-38-2)	Lung cancer	Absolute-risk linear model	4.3 per mg/m³	Inhalation	A: Human Carcinogen
Benzene (71-43-2)	Leukemia	Low-dose linearity utilizing maximum likelihood estimates	$2.2x10^{-3}$ per mg/m³	Inhalation	A: Human Carcinogen
Cadmium (7440-43-9)	Lung, trachea, bronchus cancer deaths	Two stage; only first affected by exposure; extra risk	1.8 per mg/m³	Inhalation	B1: Probable human carcinogen - based on limited evidence of carcinogenicity in humans
Carbon Tetrachloride (56-23-5)	Hepatocellular carcinomas/hepatomas	Linearized multistage procedure, extra risk	$1.5x10^{-2}$ per mg/m³	Inhalation	B2: Probable human carcinogen - based on sufficient evidence of carcinogenicity in animals
Formaldehyde (50-00-0)	Squamous cell carcinoma	Linearized multistage procedure, additional risk	$1.3x10^{-2}$ per mg/m³	Inhalation	B1: Probable human carcinogen - based on limited evidence of carcinogenicity in humans
Methylene Chloride (75-09-2)	Alveolar and bronchiolar carcinomas and adenomas	Linearized multistage procedure	0.00000047 per µg/m³	Inhalation	Not Available
Naphthalene (91-20-3)	Respiratory epithelial adenoma and olfactory epithelial neuroblastoma of the nose	Linearized multistage procedure	0.000012 per µg/m³	Inhalation	B2: Probable human carcinogen - based on sufficient evidence of carcinogenicity in animals
Nickel (7440-02-0)	Lung cancer	Additive and multiplicative	$2.4x10^{-1}$ per mg/m³	Inhalation	A: Human Carcinogen

CAS NO. = Chemical Abstract Service Number

APPENDIX E: Cancer Narrative (continued)

Table E-2: DATI Cancer Risk Drivers and Their Weight of Evidence Narrative

CHEMICAL (CAS NO.)	WEIGHT OF EVIDENCE NARRATIVE
1,3-Butadiene (106-99-0)	Humans by inhalation supported by the total weight of evidence provided by the following: (1) sufficient evidence from epidemiologic studies of the majority of U.S. workers occupationally exposed to 1,3-butadiene, either to the monomer or to the polymer by inhalation, showing increased lymphohematopoietic cancers and a dose-response relationship for leukemia in polymer workers; (2) sufficient evidence in laboratory animal studies showing that 1,3-butadiene causes tumors at multiple sites in mice and rats by inhalation, and (3) numerous studies consistently demonstrating that 1,3-butadiene is metabolized into genotoxic metabolites by experimental animals and humans. Genotoxic metabolites of 1,3-butadiene: monoepoxide, the diepoxide, and the epoxydiol.
1,4-Dichlorobenzene (106-46-7)	MDEQ-AQD Potency is based on NTP 1987 gavage study with 0, 300, or 600 mg/kg, where male mice had increased incidence of liver adenomas and carcinomas.
Acetaldehyde (75-07-0)	Based on increased incidence of nasal tumors in male and female rats and laryngeal tumors in male and female hamsters after inhalation exposure.
Acrylonitrile (107-13-1)	The observation of a statistically significant increase in incidence of lung cancer in exposed workers and observation of tumors, generally astrocytomas in the brain, in studies in two rat strains exposed by various routes (drinking water, gavage, and inhalation) forms the basis for this classification.
Arsenic (7440-38-2)	Based on sufficient evidence from human data. An increased lung cancer mortality was observed in multiple human populations exposed primarily through inhalation. Also, increased mortality from multiple internal organ cancers (liver, kidney, lung, and bladder) and an increased incidence of skin cancer were observed in populations consuming drinking water high in inorganic arsenic.
Benzene (71-43-2)	Under the proposed revised Carcinogen Risk Assessment Guidelines (USEPA, 1996), benzene is characterized as a known human carcinogen for all routes of exposure based upon convincing human evidence as well as supporting evidence from animal studies. (USEPA, 1979, 1985, 1998; ATSDR, 1997).
Cadmium (7440-43-9)	Limited evidence from occupational epidemiologic studies of cadmium is consistent across investigators and study populations. There is sufficient evidence of carcinogenicity in rats and mice by inhalation and intramuscular and subcutaneous injection. Seven studies in rats and mice wherein cadmium salts (acetate, sulfate, chloride) were administered orally have shown no evidence of carcinogenic response.
Carbon Tetrachloride (56-23-5)	Carcinogenicity in rats, mice, and hamsters
Formaldehyde (50-00-0)	Based on limited evidence in humans, and sufficient evidence in animals. Human data include nine studies with statistically significant associations between site-specific respiratory neoplasms and exposure to formaldehyde. An increased incidence of nasal squamous cell carcinomas observed in long-term inhalation studies in rats and in mice. Supported by in vitro geno-tox data and structural relationships to other carcinogenic aldehydes such as acetaldehyde.
Methylene Chloride (75-09-2)	Potency is based on the 1985 draft report of later finalized NTP 1986 study. Female mice exposed to 0, 2000 or 4000 ppm developed alveolar and bronchiolar carcinomas and adenomas. 5/28/92 - due to USEPA having risk assessment under review, to be completed later in 1992, AQD will wait for USEPA to finish before revising.
Naphthalene (91-20-3)	Based on increased incidence of nasal respiratory epithelium adenomas and olfactory epithelia neuroblastomas in male and female rats and alveolar/bronchiolar adenomas in female mice after inhalation exposure.
Nickel (7440-02-0)	Human data in which exposure to nickel refinery dust caused lung and nasal tumors in sulfide nickel matte refinery workers in several epidemiologic studies in different countries, and on animal data in which carcinomas were produced in rats by inhalation and injection.

APPENDIX F: Chemical Information with Benchmarks

CHEMICAL NAME	CAS NO	MOLECULAR WEIGHT	CARBONYL	VOC	METAL	CR^{+6}	PAH	AQD ITSL	ITSL AVG TIME	AQD IRSL	BASIS ITSL	BASIS IRSL
1,1,1-Trichloroethane	71556	133.4		X				1000	24 hr		USEPA	
1,1,2,2-Tetrachloroethane	79345	167.84		X						0.02		USEPA
1,1,2-Trichloroethane	79005	133.4		X						0.06		USEPA
1,1-Dichloroethane	75343	98.96		X				500	24 hr		USEPA	
1,1-Dichloroethene	75354	96.94		X				200	24 hr	0.02	USEPA	USEPA
1,2,4,5-Tetrachlorobenzene	95943	215.89					X					
1,2,4-Trichlorobenzene	120821	181.44		X				370	1 hr		TLV	
1,2,4-Trichlorobenzene	120821	181.44					X	370	1 hr		TLV	
1,2,4-Trimethylbenzene	95636	120.21		X				1230	8 hr		TLV	
1,2-Dibromoethane	106934	187.88		X						0.005		USEPA
1,2-Dichlorobenzene	95501	147		X				1500	8 hr		TLV	
1,2-Dichlorobenzene	95501	147					X	1500	8 hr		TLV	
1,2-Dichloroethane	107062	98.96		X						0.04		USEPA
1,2-Dichloropropane	78875	112.99		X				4	24 hr		USEPA	
1,3,5-Trimethylbenzene	108678	120.19		X				1230	8 hr		TLV	
1,3-Butadiene	106990	54.1		X				2	24 hr	0.03	USEPA	USEPA
1,3-Dichlorobenzene	541731	147		X								
1,3-Dichlorobenzene	541731	147					X					
1,3-Dintrobenzene	99650	168.11					X					
1,4-Dichlorobenzene	106467	147		X				800	24 hr	0.14	USEPA	AQD
1,4-Dichlorobenzene	106467	147					X	800	24 hr	0.14	USEPA	AQD
1,4-Naphthoquinone	130154	158.16					X					
1-Naphthylamine	134327	143.19					X					
2,2,4-Trimethylpentane	540841	114.26		X								
2,3,4,6-Tetrachlorophenol	58902	231.89					X					
2,4,5-Trichlorphenol	95954	197.45					X	350	24 hr		USEPA	
2,4,6-Trichlorophenol	88062	197.45					X			0.3		USEPA
2,4-Dichlorophenol	120832	163					X	77	annual		AQD	
2,4-Dimethylphenol	105679	122.17					X	70	24 hr		USEPA	
2,4-Dinitrophenol	51285	184.11					X					
2,4-Dinitrotoluene	121142	182.14					X	2	8 hr	0.009	TLV	AQD
2,5-dimethylbenzaldehyde	5779942	134	X									
2,6-Dichlorophenol	87650	163					X					

APPENDIX F: Chemical Information with Benchmarks

CHEMICAL NAME	CAS NO	MOLECULAR WEIGHT	CARBONYL	VOC	METAL	CR^{+6}	PAH	AQD ITSL	ITSL AVG TIME	AQD IRSL	BASIS ITSL	BASIS IRSL
2,6-Dinitrotoluene	606202	182.14					X	15	8 hr	0.005	TLV	USEPA
2-Acetylaminofluorene	53963	223.27					X					
2-Chloro-1,3-Butadiene	126998	88.54		X						0.001		AQD
2-Chloronaphthalene	91587	162.62					X					
2-Chlorophenol	95578	128.56					X					
2-Methyl Naphthalene	91576	142.21					X	10	annual		AQD	
2-Methylphenol	95487	108.139					X	100	8 hr		NIOSH	
2-Naphthylamine	91598	143.19					X			0.00013		AQD
2-Nitroaniline	88744	138.13					X					
2-Nitrophenol	88755	139.1					X					
2-Picoline	109068	93.13					X	24	24 hr		AQD	
3&4-Methylphenol	1319773	108.14					X					
3,3'-Dichlorobenzidine	91941	253.13					X			0.002		AQD
3,3'-Dimethylbenzidine	119937	212.29					X					
3-Methylcholanthrene	56495	268.36					X					
3-Nitroaniline	99092	138.13					X					
4,6-Dinitro-2-methylphenol	534521	198.13					X					
4-Aminobiphenyl	92671	169.23					X					
4-Bromophenyl phenyl ether	101553	249.11					X					
4-Chloro-3-methylphenol	59507	142.58					X					
4-Chloroaniline	106478	127.57					X					
4-Chlorophenyl-phenyl ether	7005723	204.66					X					
4-Dimethylaminoazobenzene	60117	225.29					X					
4-Nitroaniline	100016	138.13					X					
4-Nitrophenol	100027	139.11					X					
5-Nitro-o-toluidine	99558	152.15					X					
7,12-Dimethylbenz(a)anthracene	57976	256.34					X					
Acenaphthene	83329	154.22					X	210	24 hr		USEPA	
Acenaphthylene	208968	152.2					X	35	24 hr		AQD	
Acetaldehyde	75070	44.06	X					9	24 hr	0.5	USEPA	USEPA

APPENDIX F: Chemical Information with Benchmarks

CHEMICAL NAME	CAS NO	MOLECULAR WEIGHT	CARBONYL	VOC	METAL	CR^{+6}	PAH	AQD ITSL	ITSL AVG TIME	AQD IRSL	BASIS ITSL	BASIS IRSL
Acetone	67641	58.05	X					5900	8 hr		NIOSH	
Acetonitrile	75058	41.06		X				60	24 hr		USEPA	
Acetophenone	98862	120.15					X	490	8 hr		TLV	
Acetylene	74862	26.04		X								
Acrylonitrile	107131	53.07		X				2	24 hr	0.01	USEPA	USEPA
Aniline	62533	93.128					X	1	24 hr		USEPA	
Anthracene	120127	178.23					X	1000	24 hr		USEPA	
Arsenic (TSP)	7440382	74.92			X					0.0002		USEPA
Azobenzene	103333	182.22					X			0.03		USEPA
Benzaldehyde	100527	106.13	X							0.4		AQD
Benzene	71432	78.12		X				30	24 hr	0.1	USEPA	USEPA
Benzidine	92875	184.24					X			0.00002		USEPA
Benzo(a)anthracene	56553	228.3					X					
Benzo(a)pyrene	50328	252.32					X			0.0005		USEPA
Benzo(b)fluoranthene	205992	252.31					X					
Benzo(ghi)perylene	191242	276.34					X	12	24 hr		AQD	
Benzo(k)fluoranthene	207089	252.31					X					
Benzyl alcohol	100516	108.14					X	5000	24 hr		AQD	
Beryllium (TSP)	7440417	9.01			X			0.02	24 hr	0.0004	USEPA	USEPA
bis (2-Chloroethyl)ether	111444	143.02					X			0.003		USEPA
bis(2-Chloroethoxy)methane	111911	173.04					X					
bis(2-Chloroisopropyl)ether	108601	171.07					X					
bis(2-Ethylhexyl)phthalate	117817	390.62					X			0.2		AQD
Bromochloromethane	74975	129.39		X				10600	8 hr		TLV	
Bromodichloromethane	75274	163.83		X						0.027		
Bromoform	75252	252.75		X						0.9		USEPA
Bromomethane	74839	94.95		X				5	24 hr		USEPA	
Butyl benzyl phthalate	85687	312.36					X	700	24 hr		USEPA	
Butyr/Isobutyraldehyde		72.12	X									
Cadmium (TSP)	7440439	112.4			X					0.0006		USEPA
Carbazole	86748	167.21					X					
Carbon Tetrachloride	56235	153.81		X						0.07		USEPA

APPENDIX F: Chemical Information with Benchmarks

CHEMICAL NAME	CAS NO	MOLECULAR WEIGHT	CARBONYL	VOC	METAL	CR^{+6}	PAH	AQD ITSL	ITSL AVG TIME	AQD IRSL	BASIS ITSL	BASIS IRSL
Chlorobenzene	108907	112.56		X				70	24 hr		AQD	
Chlorobenzilate	510156	325.19					X					
Chloroethane	75003	64.52		X				10000	24 hr		USEPA	
Chloroform	67663	119.37		X						0.4		AQD
Chloromethane	74873	50.49		X				90	24 hr	1.6	USEPA	AQD
Chloromethyl Benzene	100447	126.59		X						0.02		
Chromium (TSP)	7440473	52			X							
Chromium VI (TSP)	18540300	52				X		0.1	24 hr	0.000083	USEPA	USEPA
Chrysene	218019	228.3					X					
cis-1,2-Dichloroethene	156592	96.94		X				35	24 hr		USEPA	
cis-1,3-Dichloropropene	10061020	110.97		X								
Crotonaldehyde		70.1	X					9	1 hr		TLV	
Diallate	1303164	270.22					X					
Dibenzo(ah)anthracene	53703	278.36					X					
Dibenzofuran	132649	168.19					X	0.1	annual		AQD	
Dibromochloromethane	124481	208.29		X						0.04		AQD
Dichlorodiflouromethane	75718	120.91		X				49500	8 hr		TLV	
Dichlorotetrafluoroethane	76142	170.92		X				69000	8 hr		AQD	
Diethyl phthalate	84662	222.24					X	50	8 hr		TLV	
Dimethyl phthalate	131113	194.186					X	50	8 hr		TLV	
Di-n-butyl phthalate	84742	278.347					X	50	8 hr		TLV	
Di-n-octyl phthalate	117840	390.56					X					
Dinoseb	88857	240.22					X	4	24 hr		USEPA	
Diphenylamine	122394	169.22					X	100	8 hr		TLV	
Ethyl Acrylate	140885	100.13		X						0.07		AQD
Ethyl methanesulfonate	62500	124.15					X					
Ethyl Tert-Butyl Ether	637923	102.2		X				373	24 hr		AQD	
Ethylbenzene	100414	106.18		X				1000	24 hr	3	USEPA	AQD
Fluoranthene	206440	202.26					X	140	24 hr		USEPA	
Fluorene	86737	166.23					X	140	24 hr		USEPA	
Formaldehyde	50000	30.03	X							0.08		USEPA
Halocarbon 113	76131	187.37		X				76700	8 hr		TLV	
Halocarbon 114	76142	170.92		X				69000	8 hr		AQD	

APPENDIX F: Chemical Information with Benchmarks

CHEMICAL NAME	CAS NO	MOLECULAR WEIGHT	CARBONYL	VOC	METAL	CR^{+6}	PAH	AQD ITSL	ITSL AVG TIME	AQD IRSL	BASIS ITSL	BASIS IRSL
Hexachloro-1,3-Butadiene	87683	260.74		X						0.05		USEPA
Hexachloro-1,3-Butadiene	87683	260.74					X			0.05		USEPA
Hexachlorobenzene	118741	284.78					X			0.002		USEPA
Hexachlorocyclopentadiene	77474	272.77					X	0.2	24 hr		USEPA	
Hexachloroethane	67721	236.74					X	3.5	24 hr	0.3	USEPA	USEPA
Hexachloropropene	1888717	248.75					X					
Hexanaldehyde	66251	100.18	X									
Indeno(123-cd)pyrene	193395	276.34					X					
Isodrin	465736	364.91					X					
Isophorone	78591	138.21					X	280	1 hr	3.7	TLV	USEPA
Isosafrole	120581	162.19					X					
Isovaleraldehyde	590863	86.15	X					800	annual		AQD	
Lead (TSP)	7439921	207.19			X			1.5	3 months		NAAQS	
m,p-Tolualdehyde	1334787	120.16	X									
m/p -Xylene	1330207	106.18		X				100	24 hr		USEPA	
Manganese (TSP)	7439965	54.94			X			0.05	24 hr		USEPA	
Methyl Ethyl Ketone	78933	72.12		X				5000	24 hr		USEPA	
Methyl Isobutyl Ketone	108101	100.18		X				3000	24 hr		USEPA	
Methyl Methacrylate	80626	100.13		X				700	24 hr		USEPA	
Methyl methanesulfonate	66273	110.13					X					
Methyl Tert-Butyl Ether	1634044	88.17		X				3000	24 hr		USEPA	
Methylene Chloride	75092	84.93		X						2		USEPA
Naphthalene	91203	128.18					X	3	24 hr	0.3	USEPA	AQD
n-Butyraldehyde	123728	72.12	X					7	24 hr		AQD	
n-Hexane	110543	86.18		X				200	24 hr		USEPA	
Nickel (TSP)	7440020	58.71			X					0.0042		USEPA
Nitrobenzene	98953	123.11					X	0.7	24 hr	0.05	AQD	AQD
N-Nitrosodibutylamine	924163	158.24					X					
N-Nitrosodiethylamine	55185	102.14					X					
N-Nitrosodimethylamine	62759	74.08					X					
N-Nitrosodipropylamine	621647	130.19					X			0.0005		AQD
N-Nitrosomethylethylamine	10595960	88.11					X					
N-Nitrosopiperidine	100754	114.15					X					

APPENDIX F: Chemical Information with Benchmarks

CHEMICAL NAME	CAS NO	MOLECULAR WEIGHT	CARBONYL	VOC	METAL	CR^{+6}	PAH	AQD ITSL	ITSL AVG TIME	AQD IRSL	BASIS ITSL	BASIS IRSL
N-Nitrosopyrrolidine	930552	100.12					X					
n-Octane	111659	114.26		X								
o-Tolualdehyde	529204	120.16	X									
o-Toluidine	95534	107.15					X			0.07		AQD
o-xylene	95476	106.18		X				100	24 hr		USEPA	
Pentachlorobenzene	608935	250.34					X					
Pentachloroethane	76017	202.29					X					
Pentachloronitrobenzene	82688	295.34					X	5	8 hr		TLV	
Pentachlorophenol	87865	266.34					X	100	24 hr	0.03	AQD	AQD
Phenacetin	62442	179.22					X					
Phenanthrene	85018	178.24					X	0.1	annual		AQD	
Phenol	108952	94.11					X	600	1 hr		NIOSH	
Pronamide	23950580	256.13					X					
Propionaldehyde	318989	58.09	X					4	annual		AQD	
Propylene	115071	42.09		X				1500	24 hr		AQD	
Pyrene	129000	202.26					X	100	24 hr		USEPA	
Pyridine	110861	79.1					X	3.5	24 hr		USEPA	
Safrole	94597	162.19					X					
Styrene	100425	104.16		X				1000	24 hr	1.7	USEPA	USEPA
Tert-Amyl Methyl Ether	994058	102.2		X				62	24 hr		AQD	
Tetrachloroethene	127184	165.82		X						1.7		AQD
Tolualdehydes	1334787	120.16	X									
Toluene	108883	92.15		X				400	24 hr		USEPA	
trans-1,2-Dichloroethene	156605	96.94		X				70	24 hr		USEPA	
trans-1,3-Dichloropropene	10061030	110.97		X								
Trichloroethene	79016	131.38		X						0.6		AQD
Trichloroflouromethane	75694	137.36		X				56200	1 hr		TLV	
Trichlorotrifluoroethane	76131	187.376		X				76700	8 hr		TLV	
Valeraldehyde	110623	86.15	X					1760	8 hr		TLV	
Vinyl Chloride	75014	62.5		X				100	24 hr	0.11	USEPA	USEPA

APPENDIX G: TOSHI Assessment

Table G-1: Background Data on Chronic Noncancer Toxicity of Compounds with HQ ≥ 0.1 and ≥ 15% Detection Rates

CHEMICAL	ITSL (AVG. TIME) µg/m³	BASIS	STUDY TYPE	CRITICAL EFFECTS	SECONDARY EFFECTS	OTHER INFORMATION (MODE OF ACTION, ETC.)
2-Methyl Naphthalene	10 (ann.)	AQD (LD_{50})	Animal, 4-hr inhalation	No Deaths	Neurological	Poor toxicological database. Effects expected to be similar to naphthalene
Acetaldehyde	9 (24-hr)	USEPA RfC	Animal, 4-week inhalation	Degeneration of olfactory epithelium		Contact irritation; Category 1 Gas
Acetonitrile	60 (24-hr)	USEPA RfC	Animal, Chronic inhalation	Mortality	Lung	Possible neurological effect in human clinical trial exposure to 67 mg/m³. Category 2 Gas RDGR = 1 (default to Category 3 Gas)
Acrylonitrile	2 (24-hr)	USEPA RfC	Animal, Chronic, inhalation	Degeneration and inflammation of nasal respiratory epithelium; hyperplasia of mucous secreting cells	CNS: gliosis (rat), neuro-behavioral and hemato-pathology (human)	Contact irritation; Category 1 Gas
Benzene	30 (24-hr)	USEPA RfC	Human, occupational, inhalation	Decreased lymphocyte count	Immunotoxicity, Neurotoxicity at higher conc.	
Dibenzofuran	0.1	AQD	(none, default)	Default ITSL	Default ITSL	Default ITSL
Manganese	0.05 (24-hr)	USEPA RfC	Human, occupational , inhalation	Neuro-behavioral		Unknown mode of action
Naphthalene	3 (24-hr)	USEPA RfC	Animal, Chronic inhalation	Nasal effects: hyperplasia and metaplasia in respiratory and olfactory epithelium, respectively	Systemic toxicity causes effects in respiratory tract and cataracts in eyes	Category 3 gas, But possible Category 1 gas
Phenanthrene	0.1	AQD	(none, default ITSL)	Default ITSL	Default ITSL	Default ITSL

APPENDIX G: TOSHI Assessment (continued)

Table G-2: Toxicological Details on the Derivation of Chronic Noncancer Health Benchmarks of Compounds with HQ ≥ 0.1 and ≥ 15% Detection Rates

CHEMICAL NAME	TOXICOLOGICAL DETAILS ON DERIVATION OF HEALTH BENCHMARKS	EFFECT LEVELS, CONVERSION FACTORS AND ASSUMPTIONS
2-Methyl Naphthalene	There were no deaths observed in the highest dose from a rat behavioral testing study following 4 hr inhalation of 527 mg/m^3. Mode of action and toxicity are expected to be similar to naphthalene.	This dose was used as surrogate LC_{50} in the R232(1)(f) equation. Total safety factor = 2,000,000.
Acetaldehyde	Degeneration of olfactory epithelium observed at 728 mg/m³. UF=1000. A factor of 10 was applied to account for sensitive human populations; for both uncertainty in the interspecies extrapolation using dosimetric adjustments; to account for the incompleteness of the data base; and to account for subchronic to chronic extrapolation.	NOAEL: 273 mg/m³ = 150 ppm x 44.5/24.45 = 273 mg/m^3 $NOAEL_{ADJ}$: 48.75 mg/m³ = 273 mg/m³ x 6 hours/day x 5 days/7 days = 48.75 mg/m³. $NOAEL_{HEC}$: 8.7 mg/m³ = was calculated for a gas:respiratory effect in the ExtraThoracic region. MVa = 0.23 cu.m/day, MVh = 20 cu.m/day, Sa(ET) = 11.6 sq. cm, Sh(ET) = 177 sq. cm. RGDR(ET) = (MVa/Sa) / (MVh/Sh) = 0.18. $NOAEL_{HEC}$ = $NOAEL_{ADJ}$ x RGDR = 8.7 mg/m³. LOAEL: 728 mg/m³ (400 ppm) $LOAEL_{ADJ}$: 130 mg/m³ $LOAEL_{HEC}$: 16.9 mg/m³
Acetonitrile	Based on mouse NOAEL of 200 ppm (336 mg/m³) identified from a 13-week inhalation study. Total safety factor = 1000 (UF of 100 and MF of 10, see below for explanation) The $NOAEL_{HEC}$ was calculated for a category 2 gas and extrarespiratory (systemic) effects. Acetonitrile is considered to be a category 2 gas because it has high water solubility, is metabolized to reactive cyanide in the liver but may be detoxified rapidly to thiocyanate, and does not react directly with respiratory tract tissues. The RGDR for a category 2 gas is assumed to be 1. UF = 100. A factor of 3 ($10^{½}$) was used for interspecies extrapolation, a full factor of 10 was used to protect sensitive human subpopulations, and 3 was applied for database deficiencies (e.g., reproductive endpoints, hematology in mice). Because two factors of 3 coalesce to a 10, a total UF of 100 results. No UF was applied to the use of a subchronic study because there was no mortality in the longer term mouse study. Therefore, although this endpoint is of concern based on the subchronic study, increased exposure would not be expected to	NOAEL: 336 mg/m³ = 200 ppm × 41.05/24.45 = 336 mg/m³ $NOAEL_{ADJ}$: 60 mg/m³ = NOAEL (mg/m³) × 6 hours/24 hours × 5/7 days = 60 mg/m³ $NOAEL_{HEC}$: 60 mg/m³ = $NOAEL_{ADJ}$ × 1 = 60 mg/m³

APPENDIX G: TOSHI Assessment (continued)

Table G-2: Toxicological Details on the Derivation of Chronic Noncancer Health Benchmarks of Compounds with HQ ≥ 0.1 and ≥ 15% Detection Rates

CHEMICAL NAME	TOXICOLOGICAL DETAILS ON DERIVATION OF HEALTH BENCHMARKS	EFFECT LEVELS, CONVERSION FACTORS AND ASSUMPTIONS
Acetonitrile (continued)	increase the sensitivity to this endpoint. A partial UF of 3, instead of a full factor of 10, was used for database insufficiencies in the areas of limited data on reproductive endpoints involving exposure of laboratory animals before and during mating through parturition and the absence of hematological measurements in either mouse study. A full factor of 10 was not considered necessary for the following reasons: (1) there is no evidence to suggest that acetonitrile accumulates in the body, (2) the developmental effects observed seem to be marginal, and (3) these effects occur at concentrations that are lethal to dams. MF = 10. A modifying factor of 10 was applied because of the uncertain role that inhalation may have played in the development of the concentration-related increase in the incidence of forestomach lesions in both male and female mice. A potential role of inhalation can be envisioned. Ahmed et al. (1992) administered ^{14}C-acetonitrile to mice via intravenous injection. As early as 5 minutes post injection, label was detected in nasal secretions, esophagus, and stomach contents.	
Acrylonitrile	Mortality was observed at the 400 ppm dose level. Acute toxicity of acrylonitrile resembles cyanide poisoning. The structure of acrylonitrile is CH_2=CHCN. UF = 1000. 10 to protect unusually sensitive individuals and 3 to adjust from a minimally adverse LOAEL to a NOAEL. An UF of 3 for interspecies variability is used because the use of the dosimetric adjustments account for part of this area of uncertainty. An additional factor of 10 is applied due to an incomplete data base, or more specifically, the lack of an inhalation bioassay in a second species, and the lack of reproductive data by the inhalation route with the existence of an oral study showing reproductive effects.	$NOAEL_{ADJ}$=$NOAEL_{HEC}$=60 mg/m³ UF=100, MF=10 to obtain RfC of 60 µg/m³. NOAEL: none LOAEL: 43 mg/m³ = 20 ppm x 53.06/24.45 = 43 mg/m³ $LOAEL_{ADJ}$: 7.7 mg/m³ = 43 mg/m³x 6 hours/24 hours x 5 days/7 days = 7.7 mg/m³ $LOAEL_{HEC}$: 1.9 mg/m³ = The $LOAEL_{HEC}$ was calculated for a gas:respiratory effect in the ExtraThoracic region. MVa = 0.33 cu.m/day, MVh = 20 cu.m/day, Sa(ET) = 11.6 sq. cm., Sh(ET) = 177 sq. cm. RGDR(ET) = (MVa/Sa) / (MVh/Sh) = 0.252. $LOAEL_{HEC}$ = $LOAEL_{ADJ}$ x RGDR = 7.7 mg/m³ x 0.252 = 1.9 mg/m³.

APPENDIX G: TOSHI Assessment (continued)

Table G-2: Toxicological Details on the Derivation of Chronic Noncancer Health Benchmarks of Compounds with HQ ≥ 0.1 and ≥ 15% Detection Rates

CHEMICAL NAME	TOXICOLOGICAL DETAILS ON DERIVATION OF HEALTH BENCHMARKS	EFFECT LEVELS, CONVERSION FACTORS AND ASSUMPTIONS
Benzene	Based on human occupational exposure study, where decreased lymphocyte counts were observed. The lower one-sided confidence limit on the benchmark concentration, $BMCL_{Adj}$ = 8.2 mg/m³. Total UF of 300 was applied to obtain 30 µg/m³. A factor of 3 was used as an effect level extrapolation factor, analogous to the LOAEL-to-NOAEL UF. Second, a factor of 10 was used for intraspecies differences in response (protect potentially sensitive human subpopulations). Third, a UF of 3 for subchronic-to-chronic extrapolation was applied. Finally, a UF of 3 was used to account for database deficiencies, because no two-generation reproductive and developmental toxicity studies for benzene are available. The overall UF of 3 x 10 x 3 x 3 = 300 is used to calculate the RfC.	BMCL (mg/m^3) = 7.2 ppm x MW/24.45 = 23.0 mg/m^3. $BMCL_{ADJ}$ = 23.0 mg/m^3 x 10 m^3/20 m^3 x 5 days/7days = 8.2 mg/m^3.
Dibenzofuran	Based on AQD default ITSL.	
Manganese	The principal health effects associated with chronic manganese (Mn) exposure are neuro-behavioral, which include visual reaction time, mean reaction times, and eye-hand coordination. These effects were observed where the exposure duration was determined to be 8 hours per day for an average of 5.3 years. The RfC was calculated assuming an occupational-lifetime exposure of 40 years. Roels et al., 1987: The LOAEL is based on an 8-hr TWA occupational exposure. The TWA of total airborne manganese dust ranged from 0.07 to 8.61 mg/m³, and the median was 0.97 mg/m³. This is an extrarespiratory effect of a particulate exposure. MVho = 10 m³/day, MVh = 20 m³/day. $LOAEL_{HEC}$ = 0.97 mg/m³ x (MVho/MVh) x 5 days/7 days = 0.34 mg/m³.	The LOAEL is derived from an occupational-lifetime integrated respirable dust (IRD) concentration of manganese dioxide (MnO_2) (based on 8-hr TWA occupational exposure multiplied by individual work histories in years) expressed as mg Mn/m³ x years. The IRD concentrations ranged from 0.040 to 4.433 mg Mn/m³ x years, with a geometric mean of 0.793 mg Mn/m³ x years and a geometric standard deviation of 2.907. The geometric mean concentration (0.793 mg/m³ x years) was divided by the average duration of MnO_2 exposure (5.3 years) to obtain a LOAEL TWA of 0.15 mg/m³. The LOAEL refers to an extrarespiratory effect of particulate exposure and is based on an 8-hr TWA occupational exposure. MVho = 10 m³/day, MVh = 20 m³ /day. $LOAEL_{HEC}$ = 0.15 mg/ m³ x (MVho/MVh) x 5 days/7 days = 0.05 mg/m³.

APPENDIX G: TOSHI Assessment (continued)

Table G-2: Toxicological Details on the Derivation of Chronic Noncancer Health Benchmarks of Compounds with HQ ≥ 0.1 and ≥ 15% Detection Rates

CHEMICAL NAME	TOXICOLOGICAL DETAILS ON DERIVATION OF HEALTH BENCHMARKS	EFFECT LEVELS, CONVERSION FACTORS AND ASSUMPTIONS
Naphthalene	ITSL based on USEPA's RfC. The RfC is based on a chronic mouse LOAEL of 10 ppm reported by NTP (1992). The critical effect is hyperplasia and metaplasia of respiratory and olfactory epithelium. UF = 3000 10 to extrapolate from mice to humans, 10 to protect sensitive humans, 10 to extrapolate from a LOAEL to a NOAEL, and 3 for database deficiencies including the lack of a 2-generation reproductive toxicity study and chronic inhalation data for other animal species. Human experience with acute accidental exposures to naphthalene identifies the development of hemolytic anemia and cataracts as health hazards of concern.	NOAEL: None $LOAEL_{HEC}$: 9.3 mg/m³ Experimental exposure concentrations of 0, 10, and 30 ppm were converted to 0, 52, and 157 mg/m³, respectively; adjusted to a continuous exposure basis in mg/m³ (6/24 hr × 5/7 days) equals mg/m³ × 0.1786: 0, 9.3, and 28 mg/m³. $LOAEL_{HEC}$ =9.3
Phenantrene	Based on AQD default ITSL.	

APPENDIX G: TOSHI Assessment (continued)

Table G-3: Structure and Physical Properties

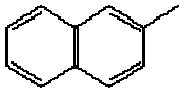
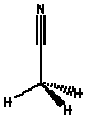
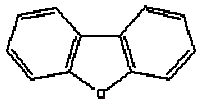
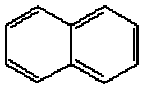
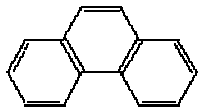
CHEMICAL NAME (CAS NO.)	STRUCTURE	PHYSICAL CHARACTERISTICS	TYPE OF AIR POLLUTANT
2-Methyl Naphthalene (91-57-6)		Crystals	PAH
Acetaldehyde (75-07-0)		Colorless liquid or gas with a penetrating, fruity odor detectable at 0.0068 to 1000 ppm. Lachrymator (causing tears)/Air Sensitive.	Aldehyde
Acetonitrile (75-05-8)		Colorless liquid with an ether-like odor detectable at 40 ppm. Lachrymator, Hygroscopic.	VOC
Acrylonitrile (107-13-1)		Colorless to pale yellow liquid with a mild pyridine-like odor at 2 to 22 ppm. Flammable. Light sensitive. Lachrymator.	VOC

Table G-3: Structure and Physical Properties

CHEMICAL NAME (CAS NO.)	STRUCTURE	PHYSICAL CHARACTERISTICS	TYPE OF AIR POLLUTANT
Benzene (71-43-2)		Colorless liquid with a paint-thinner-like odor detectable at 12 ppm. Flammable	VOC
Dibenzofuran (132-64-9)		Colorless crystals.	PAH
Manganese (7439-96-5)	Mn	Metallic	Metal
Naphthalene (91-20-3)		Colorless to brown solid with an odor of mothballs.	PAH
Phenanthrene (85-01-8)		Colorless solid or monoclinic crystals.	PAH

APPENDIX H: Rank of Cancer Risk

Greater than 15% Detected
Less than 1/million Cancer Risk

SITE NAME	CHEMICAL NAME	RISK (AVG/IRSL)
S. Delray	Beryllium (TSP)	0.91
Southfield	Nickel (TSP)	0.88
N. Delray	Chloromethane	0.86
N. Delray	Nickel (TSP)	0.81
Southfield	Chloromethane	0.80
Allen Park	Chloromethane	0.80
River Rouge	Chloromethane	0.78
Dearborn	Chloromethane	0.78
S. Delray	Chloromethane	0.76
Dearborn	Nickel (TSP)	0.73
Allen Park	Cadmium (TSP)	0.67
N. Delray	1,4-Dichlorobenzene	0.67
S. Delray	1,4-Dichlorobenzene	0.65
Dearborn	1,4-Dichlorobenzene	0.62
River Rouge	Nickel (TSP)	0.59
S. Delray	Ethylbenzene	0.59
N.E. Detroit	Cadmium (TSP)	0.58
N.E. Detroit	Nickel (TSP)	0.57
River Rouge	1,4-Dichlorobenzene	0.56
S. Delray	Benzaldehyde	0.55
Allen Park	Chromium VI (TSP)	0.53
Allen Park	Nickel (TSP)	0.53
Allen Park	Ethylbenzene	0.53
Southfield	1,4-Dichlorobenzene	0.53
Dearborn	Benzaldehyde	0.53
Southfield	Chromium VI (TSP)	0.51
River Rouge	Chromium VI (TSP)	0.47
N. Delray	Beryllium (TSP)	0.47
Southfield	Benzaldehyde	0.45
Dearborn	Chromium VI (TSP)	0.44
River Rouge	Benzaldehyde	0.44
Allen Park	1,4-Dichlorobenzene	0.41
River Rouge	Tetrachloroethene	0.39

SITE NAME	CHEMICAL NAME	RISK (AVG/IRSL)
Allen Park	Benzaldehyde	0.38
Southfield	Cadmium (TSP)	0.34
S. Delray	Chloroform	0.33
Dearborn	Beryllium (TSP)	0.31
N. Delray	Benzaldehyde	0.29
Ypsilanti	Cadmium (TSP)	0.28
Allen Park	Tetrachloroethene	0.27
Dearborn	Tetrachloroethene	0.27
River Rouge	Beryllium (TSP)	0.27
Dearborn	Ethylbenzene	0.26
Southfield	Ethylbenzene	0.26
N.E. Detroit	Benzaldehyde	0.23
River Rouge	Ethylbenzene	0.22
Ypsilanti	Benzaldehyde	0.20
Ypsilanti	Nickel (TSP)	0.20
N. Delray	Ethylbenzene	0.20
Southfield	Beryllium (TSP)	0.19
S. Delray	Styrene	0.19
N.E. Detroit	Beryllium (TSP)	0.18
Southfield	Tetrachloroethene	0.18
Allen Park	Beryllium (TSP)	0.18
S. Delray	Tetrachloroethene	0.17
River Rouge	Styrene	0.17
N. Delray	bis(2-Ethylhexyl)phthalate	0.15
N. Delray	Styrene	0.15
Dearborn	Bis (2-Ethylhexyl)phthalate	0.13
Ypsilanti	Beryllium (TSP)	0.12
Southfield	Styrene	0.12
Houghton Lake	Benzaldehyde	0.11
Dearborn	Styrene	0.10
Allen Park	Styrene	0.10

APPENDIX I: Daily and Hourly Maximum Concentrations Plus Acute Benchmarks (µg/m^3)

Table I-1: 24-Hour Monitoring Data - Maximum Daily Monitored Concentrations and Known Acute Health Benchmark Values

CAS NUMBER	CHEMICAL NAME	MAX VALUE	ACUTE CA REL (AVG TIME)	ACUTE MRL	AEGL-1 (10MIN)	AEGL-1 (30MIN)	AEGL-1 (60MIN)	AEGL-1 (4HR)	AEGL-1 (8HR)	COMMENTS
71556	1,1,1-Trichloroethane	1.14	68000 (1)	10879	1251125	1251125	1251125	1251125	1251125	AEGL-1 is interim
79345	1,1,2,2-Tetrachloroethane	0.00								
79005	1,1,2-Trichloroethane	0.00								
75343	1,1-Dichloroethane	0.00								
75354	1,1-Dichloroethene	0.00								
95943	1,2,4,5-Tetrachlorobenzene	0.00								
120821	1,2,4-Trichlorobenzene	0.82								
95636	1,2,4-Trimethylbenzene	5.06								
106934	1,2-Dibromoethane	0.00								
95501	1,2-Dichlorobenzene	0.74								
107062	1,2-Dichloroethane	0.00								
78875	1,2-Dichloropropane	0.00		231						
108678	1,3,5-Trimethylbenzene	1.96								
106990	1,3-Butadiene	0.74								
541731	1,3-Dichlorobenzene	0.71								
99650	1,3-Dintrobenzene	0.00								
106467	1,4-Dichlorobenzene	83.30		4810						
130154	1,4-Naphthoquinone	0.00								
134327	1-Naphthylamine	0.00								
540841	2,2,4-Trimethylpentane	2.49								
58902	2,3,4,6-Tetrachlorophenol	0.00								
95954	2,4,5-Trichlorphenol	0.00								
88062	2,4,6-Trichlorophenol	0.00								
120832	2,4-Dichlorophenol	0.00								
105679	2,4-Dimethylphenol	0.00								
51285	2,4-Dinitrophenol	0.00								
121142	2,4-Dinitrotoluene	0.00								
5779942	2,5-dimethylbenzaldehyde	1.53								
87650	2,6-Dichlorophenol	0.00								
606202	2,6-Dinitrotoluene	0.00								

Table I-1: 24-Hour Monitoring Data - Maximum Daily Monitored Concentrations and Known Acute Health Benchmark Values

CAS NUMBER	CHEMICAL NAME	MAX VALUE	ACUTE CA REL (AVG TIME)	ACUTE MRL	AEGL-1 (10MIN)	AEGL-1 (30MIN)	AEGL-1 (60MIN)	AEGL-1 (4HR)	AEGL-1 (8HR)	COMMENTS
53963	2-Acetylaminofluorene	0.00								
126998	2-Chloro-1,3-Butadiene	0.00								
91587	2-Chloronaphthalene	0.00								
95578	2-Chlorophenol	0.00								
91576	2-Methyl Naphthalene	7.21								
95487	2-Methylphenol	0.91								
91598	2-Naphthylamine	0.00								
88744	2-Nitroaniline	0.00								
88755	2-Nitrophenol	0.00								
109068	2-Picoline	0.32								
1319773	3&4-Methylphenol	2.10								
91941	3,3'-Dichlorobenzidine	0.00								
119937	3,3'-Dimethylbenzidine	0.00								
56495	3-Methylcholanthrene	0.00								
99092	3-Nitroaniline	0.00								
534521	4,6-Dinitro-2-methylphenol	0.00								
92671	4-Aminobiphenyl	0.00								
101553	4-Bromophenyl phenyl ether	0.00								
59507	4-Chloro-3-methylphenol	0.00								
106478	4-Chloroaniline	0.00								
7005723	4-Chlorophenyl-phenyl ether	0.00								
60117	4-Dimethylaminoazobenzene	0.00								
100016	4-Nitroaniline	0.00								
100027	4-Nitrophenol	0.00								
99558	5-Nitro-o-toluidine	0.18								
57976	7,12-Dimethylbenz(a)anthracene	0.00								
83329	Acenaphthene	2.64								
208968	Acenaphthylene	0.23								
75070	Acetaldehyde	12.11								

Table I-1: 24-Hour Monitoring Data - Maximum Daily Monitored Concentrations and Known Acute Health Benchmark Values

CAS NUMBER	CHEMICAL NAME	MAX VALUE	ACUTE CA REL (AVG TIME)	ACUTE MRL	AEGL-1 (10MIN)	AEGL-1 (30MIN)	AEGL-1 (60MIN)	AEGL-1 (4HR)	AEGL-1 (8HR)	COMMENTS
67641	Acetone	17.83		61677	474438	474438	474438	474438	474438	AEGL-1 is proposed
75058	Acetonitrile	238.37			21800	21800	21800	21800	21800	AEGL-1 proposed
98862	Acetophenone	0.10								
74862	Acetylene	6.90								
107131	Acrylonitrile	2.97		217						
62533	Aniline	0.04			182577	60859	30429	7607	3804	AEGL-1 is final
120127	Anthracene	0.67								
7440382	Arsenic (TSP)	0.02	0.19 (4)							Arsenic & inorganic arsenic compounds
103333	Azobenzene	0.00								
100527	Benzaldehyde	2.21								
71432	Benzene	158.96	1300 (6)	160						
92875	Benzidine	0.00								
56553	Benzo(a)anthracene	0.09								
50328	Benzo(a)pyrene	0.03								
205992	Benzo(b)fluoranthene	0.06								
191242	Benzo(ghi)perylene	0.00								
207089	Benzo(k)fluoranthene	0.04								
100516	Benzyl alcohol	0.00								
7440417	Beryllium (TSP)	0.01								
111444	bis (2-Chloroethyl)ether	0.00								
111911	bis(2-Chloroethoxy)methane	0.00								
108601	bis(2-Chloroisopropyl)ether	0.00								
117817	bis(2-Ethylhexyl)phthalate	1.60								
74975	Bromochloromethane	0.00								
75274	Bromodichloromethane	0.00								
75252	Bromoform	0.00								
74839	Bromomethane	1.05	3900 (1)	194						
85687	Butyl benzyl phthalate	0.00								
	Butyr/Isobutyraldehyde	3.74								
7440439	Cadmium (TSP)	0.03								

Table I-1: 24-Hour Monitoring Data - Maximum Daily Monitored Concentrations and Known Acute Health Benchmark Values

CAS NUMBER	CHEMICAL NAME	MAX VALUE	ACUTE CA REL (AVG TIME)	ACUTE MRL	AEGL-1 (10MIN)	AEGL-1 (30MIN)	AEGL-1 (60MIN)	AEGL-1 (4HR)	AEGL-1 (8HR)	COMMENTS
86748	Carbazole	0.33								
56235	Carbon Tetrachloride	1.07	1900 (7)	189	157464	100777	75583	43460	32753	AEGL-1 is interim
108907	Chlorobenzene	0.33								
510156	Chlorobenzilate	0.00								
75003	Chloroethane	1.65		39877						
67663	Chloroform	13.06	150 (7)	487						AEGL-1 not recommended due to insufficient data
74873	Chloromethane	2.94		1022						
100447	Chloromethyl Benzene	0.00	240 (1)							
7440473	Chromium (TSP)	0.02								
18540300	Chromium VI (TSP)	0.00								MRL=1 for particulates; .005 for aerosol mists
218019	Chrysene	0.12								
156592	cis-1,2-Dichloroethene	1.47			555419	555419	555419	555419	555419	AEGL-1 interim
10061015	cis-1,3-Dichloropropene	0.00								
	Crotonaldehyde	0.89								
1303164	Diallate	0.00								
53703	Dibenzo(ah)anthracene	0.00								
132649	Dibenzofuran	2.63								
124481	Dibromochloromethane	0.00								
75718	Dichlorodiflouromethane	6.63								
76142	Dichlorotetrafluoroethane	0.21								
84662	Diethyl phthalate	0.13								
131113	Dimethyl phthalate	0.00								
84742	Di-n-butyl phthalate	1.22								
117840	Di-n-octyl phthalate	0.00								
88857	Dinoseb	0.00								
122394	Diphenylamine	0.00								
140885	Ethyl Acrylate	0.00								
62500	Ethyl methanesulfonate	0.00								
637923	Ethyl Tert-Butyl Ether	0.00								

Table I-1: 24-Hour Monitoring Data - Maximum Daily Monitored Concentrations and Known Acute Health Benchmark Values

CAS NUMBER	CHEMICAL NAME	MAX VALUE	ACUTE CA REL (AVG TIME)	ACUTE MRL	AEGL-1 (10MIN)	AEGL-1 (30MIN)	AEGL-1 (60MIN)	AEGL-1 (4HR)	AEGL-1 (8HR)	COMMENTS
100414	Ethylbenzene	31.22								
206440	Fluoranthene	1.39								
86737	Fluorene	2.52								
50000	Formaldehyde	25.77	94 (1)	49	1104	1104	1104	1104	1104	AEGL-1 is proposed
76131	Halocarbon 113	5.67								
76142	Halocarbon 114	0.00								
87683	Hexachloro-1,3-Butadiene	1.12								
118741	Hexachlorobenzene	0.00								
77474	Hexachlorocyclopentadiene	0.00								
67721	Hexachloroethane	0.00		58,086						
1888717	Hexachloropropene	0.00								
66251	Hexanaldehyde	4.88								
193395	Indeno(123-cd)pyrene	0.00								
465736	Isodrin	0.00								
78591	Isophorone	0.00								
120581	Isosafrole	0.00								
590863	Isovaleraldehyde	1.85								
7439921	Lead (TSP)	1.02								
1334787	m,p-Tolualdehyde	0.81								
1330207	m/p -Xylene	121.90	22000 (1)	4344	563599	563599	563599	563599	563599	AEGL-1 proposed; values are for total xylenes
7439965	Manganese (TSP)	1.94								
78933	Methyl Ethyl Ketone	12.80	13000 (1)		588957	588957	588957	588957	588957	AEGL-1 proposed
108101	Methyl Isobutyl Ketone	3.02								
80626	Methyl Methacrylate	0.00								
66273	Methyl methanesulfonate	0.00								
1634044	Methyl Tert-Butyl Ether	2.11		7198						
75092	Methylene Chloride	11579.75	14000 (1)	2086						
91203	Naphthalene	93.25								
123728	n-Butyraldehyde	3.76								
110543	n-Hexane	11.69								

Table I-1: 24-Hour Monitoring Data - Maximum Daily Monitored Concentrations and Known Acute Health Benchmark Values

CAS NUMBER	CHEMICAL NAME	MAX VALUE	ACUTE CA REL (AVG TIME)	ACUTE MRL	AEGL-1 (10MIN)	AEGL-1 (30MIN)	AEGL-1 (60MIN)	AEGL-1 (4HR)	AEGL-1 (8HR)	COMMENTS
7440020	Nickel (TSP)	0.10	6 (1)							Nickel and nickel compounds
98953	Nitrobenzene	0.00								
924163	N-Nitrosodibutylamine	0.00								
55185	N-Nitrosodiethylamine	0.00								
62759	N-Nitrosodimethylamine	0.00								
621647	N-Nitrosodipropylamine	0.00								
10595956	N-Nitrosomethylethylamine	0.00								
100754	N-Nitrosopiperidine	0.00								
930552	N-Nitrosopyrrolidine	0.00								
111659	n-Octane	3.50								
529204	o-Tolualdehyde	0.21								
95534	o-Toluidine	0.00								
95476	o-xylene	23.32								Values are for total xylenes
608935	Pentachlorobenzene	0.00								
76017	Pentachloroethane	0.00								
82688	Pentachloronitrobenzene	0.00								
87865	Pentachlorophenol	0.00								
62442	Phenacetin	0.00								
85018	Phenanthrene	4.03								
108952	Phenol	5.05	5800 (1)		123027	123027	123027	123027	123027	AEGL-1 interim
23950584	Pronamide	0.00								
318989	Propionaldehyde	3.42								
115071	Propylene	24.34								
129000	Pyrene	0.81								
110861	Pyridine	0.81								
94597	Safrole	0.00								
100425	Styrene	4.25	21000 (1)		85072	85072	85072	85072	85072	AEGL-1 proposed
994058	Tert-Amyl Methyl Ether	0.00								
127184	Tetrachloroethene	15.91	20000 (1)	1358	237628	237628	237628	237628	237628	AEGL-1 interim
1334787	Tolualdehydes	2.90								

Table I-1: 24-Hour Monitoring Data - Maximum Daily Monitored Concentrations and Known Acute Health Benchmark Values

CAS NUMBER	CHEMICAL NAME	MAX VALUE	ACUTE CA REL (AVG TIME)	ACUTE MRL	AEGL-1 (10MIN)	AEGL-1 (30MIN)	AEGL-1 (60MIN)	AEGL-1 (4HR)	AEGL-1 (8HR)	COMMENTS
108883	Toluene	56.50	37000 (1)	3763	752556	752556	752556	752556	752556	AEGL-1 interim
156605	trans-1,2-Dichloroethene	0.40		793	1110838	1110838	1110838	1110838	1110838	AEGL-1 interim
10061026	trans-1,3-Dichloropropene	3.14								
79016	Trichloroethene	6.81		10716	1393047	1393047	1393047	1393047	1393047	AEGL-1 proposed
75694	Trichloroflouromethane	18.01								
76131	Trichlorotrifluoroethane	7.66								
110623	Valeraldehyde	3.20								
75014	Vinyl Chloride	0.00	180000 (1)	1278	1150307	792434	639059	357873	178937	AEGL-1 is proposed

APPENDIX I: Daily and Hourly Maximum Concentrations Plus Acute Benchmarks (µg/m^3) (continued)

Table I-2: 1-Hour Monitoring Data - Maximum 1-Hour Monitored Concentrations and Known Acute Health Benchmark Values

CAS NUMBER	CHEMICAL NAME	MAX VALUE	ACUTE CA REL (AVG TIME)	ACUTE MRL	AEGL-1 (10MIN)	AEGL-1 (30MIN)	AEGL-1 (60MIN)	AEGL-1 (4HR)	AEGL-1 (8HR)	COMMENTS
526738	1,2,3-Trimethylbenzene	11.57								
95636	1,2,4-Trimethylbenzene	25.38								
108678	1,3,5-Trimethylbenzene	12.69								
105055	1,4-Diethylbenzene	10.94								
106989	1-Butene	16.92								
109671	1-pentene	13.11								
75832	2,2-Dimethylbutane	21.63								
565753	2,3,4-Trimethylpentane	15.01								
79298	2,3-Dimethylbutane	1.45								
565593	2,3-Dimethylpentane	31.96								
108087	2,4-Dimethylpentane	25.69								
625274	2-Methyl-2-Pentene	24.28								
592278	2-Methylheptane	15.12								
591764	2-Methylhexane	13.28								
107835	2-Methylpentane	9.31								
78795	3-Methyl-1,3-Butadiene	1.97								
589811	3-Methylheptane	15.53								
589344	3-Methylhexane	15.69								
96140	3-methylpentane	35.78								
74862	Acetylene	30.96								
71432	Benzene	24.81	1300 (6)	160						
106978	Butane	130.76			22535787	16368098	13047035	13047035	13047035	AEGL-1 proposed
590181	Cis-2-butene	19.69								
627203	Cis-2-pentene	18.40								
110827	Cyclohexane	24.13								
142290	CYCLOPENTENE	10.24								
124185	Decane	17.25								
25340174	Diethylbenzene, mixed									
74840	Ethane	371.18								

Table I-2: 1-Hour Monitoring Data - Maximum 1-Hour Monitored Concentrations and Known Acute Health Benchmark Values

CAS NUMBER	CHEMICAL NAME	MAX VALUE	ACUTE CA REL (AVG TIME)	ACUTE MRL	AEGL-1 (10MIN)	AEGL-1 (30MIN)	AEGL-1 (60MIN)	AEGL-1 (4HR)	AEGL-1 (8HR)	COMMENTS
64175	Ethanol	276.31								
100414	Ethylbenzene	13.13								
25550144	Ethyltoluenes, mixed									
142825	Heptane	15.17								
110543	Hexane	21.07								
75285	Isobutane	35.92								
540841	Isooctane	18.95								
78784	Isopentane	108.77								
98828	Isopropylbenzene	19.21								
1330207	M/P Xylene	44.97	22,000 (1)	13027	1693456	1693456	1693456	1693456	1693456	AEGL-1 proposed; Values are for total xylenes
141935	M-Diethylbenzene	18.23								
108872	Methylcyclohexane	18.25								
96377	Methylcyclopentane	13.88								
620144	M-Ethyltoluene	13.96								
111842	Nonane	15.07								
111659	Octane	18.63								
611143	O-Ethyltoluene	15.41								
95476	O-Xylene	16.47								Values are for total xylenes
109660	Pentane	43.13								
622968	P-Ethyltoluene	19.46								
74986	Propane	108.21			17995910	12417178	9897751	9897751	9897751	AEGL-1 proposed
103651	Propylbenzene	14.46								
115071	Propylene	25.75								
100425	Styrene	10.60	21000 (1)	42.5	85072	85072	85072	85072	85072	AEGL-1 proposed
108883	Toluene	67.25	37000 (1)	3763	752556	752556	752556	752556	752556	AEGL-1 interim
624646	Trans-2-butene	14.04								
646048	Trans-2-pentene	12.96								
1120214	Undecane	14.62								

APPENDIX J: Significance of Manganese and Naphthalene Ambient Concentrations in Relation to the Health Reference Levels

For those pollutants with chronic non-cancer health reference levels, only manganese and naphthalene were found at levels above the health reference levels. The annual average concentration of manganese exceeded the health reference level at four sites: South Delray, North Delray, Dearborn, and River Rouge. The annual average concentration of naphthalene exceeded the health reference level at only the South Delray site. The basis for the health reference levels and significance of the measured ambient levels in comparison to these reference levels is discussed below.

Manganese

Breathing manganese at high enough concentrations causes effects to the nervous system. The health reference level is set far enough below the harmful level to help ensure the protection of all people. The highest manganese levels observed in the Detroit area are a cause for concern because they are higher than the health reference level, although they are still below the levels at which effects have been observed in scientific studies (see **Figure J-1**).

Napthalene

Breathing naphthalene at high enough concentrations causes effects to the respiratory system. As with manganese, the health reference level for naphthalene is set far enough below the harmful level in order to help ensure the protection of all people. The highest levels of naphthalene observed in the Detroit area in 2001-2002 pose a concern because they were higher than the health reference level, although they were still below the levels at which effects have been observed in scientific studies (see **Figure J-2**).

Figure J-1: Manganese Adverse Effect Level & Ambient Concentrations Exceeding the Health Reference Level

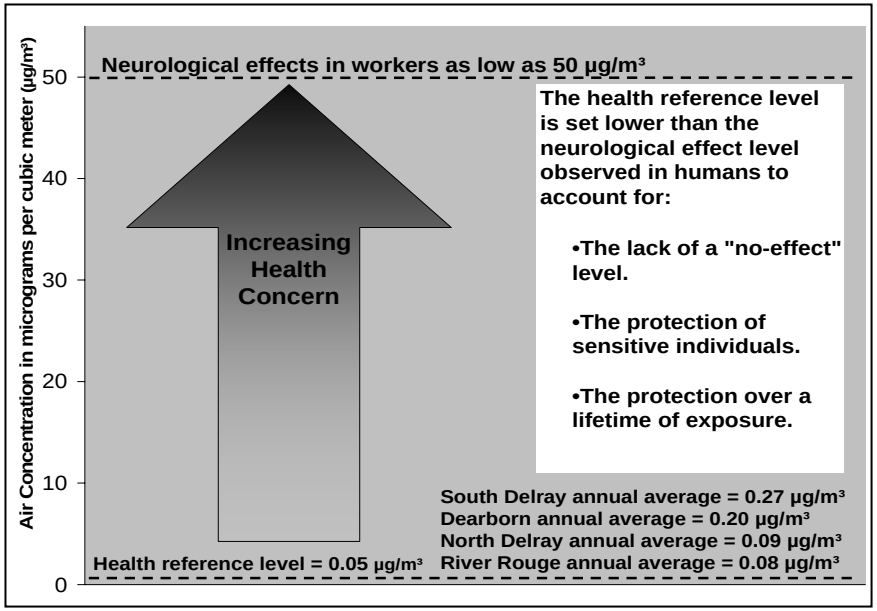
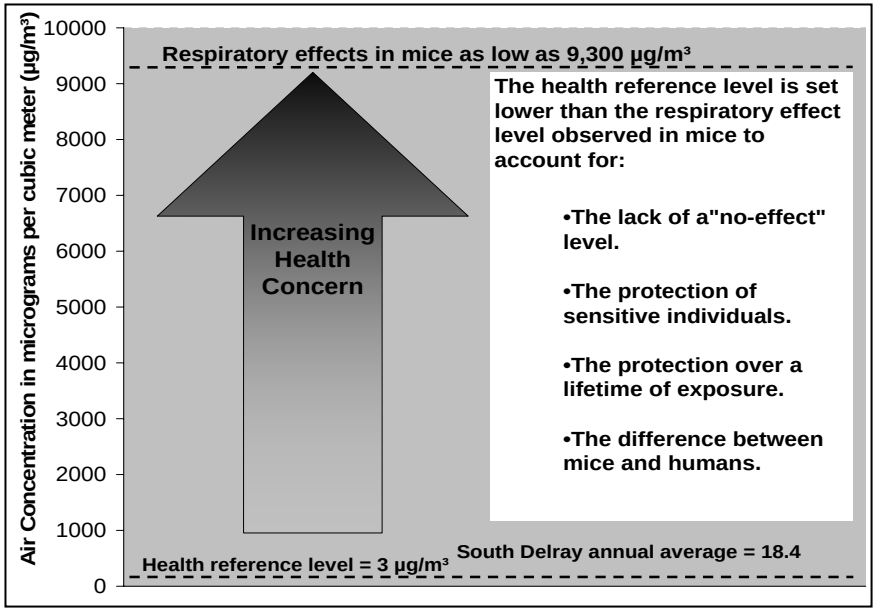



Figure J-2: Naphthalene Adverse Effect Level & Ambient Concentrations Exceeding the Health Reference Level