

Delaware Air Toxics Assessment Study

DATAS

PHASE 1



DELAWARE AIR TOXICS ASSESSMENT STUDY

(PHASE – I)

FINAL REPORT

Prepared for

The Division of Air & Waste Management

Air Quality Management Section

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*Division Of Public Health
Environmental Health Evaluation Branch*



Executive Summary

This DATAS Phase I report represents a major undertaking of the Air Quality Section of Delaware's Department of Natural Resources and Environmental Control (DNREC) with significant technical advice from the Department of Health and Social Services' Division of Public Health (DPH).

The Delaware Air Toxics Assessment Study (DATAS) represents the largest and most comprehensive study of air toxic contaminants and the risks to human health, undertaken in the Mid-Atlantic region. Over a one-year period, 119 air toxic chemicals were studied. When completed in 2006, the study will provide statewide assessments for cancer and chronic non-cancer human health risks from inhalation of ambient concentrations of air toxics.

DATAS was developed as a two-phase project.

Phase I is completed and includes:

- Monitoring program of air toxics
- Expanded emissions inventory of toxic air pollutants
- Prototype study of air dispersion modeling
- Risk assessment of the human health effects of the monitored air toxics concentrations

Phase II will be completed in 2006 and will include:

- Statewide modeling of air toxic pollutants
- Statewide risk assessment of the human health effects associated with modeled concentrations
- Delaware Air Toxics Risk Management Plan

1.0 Background

In early 2002, the Delaware Air Quality Management Section of DNREC embarked on the DATAS to gain a better understanding of ambient concentrations of air toxics throughout Delaware and their potential cancer and chronic non-cancer adverse health effects.

There were two factors that prompted DATAS. First, to better understand Delaware's high cancer incidence, the Delaware Advisory Council on Cancer Incidence and Mortality recommended evaluating environmental factors, including the risk from air toxics. Second, the United States Environmental Protection Agency (EPA) brought attention to air toxics through a nationwide study known as the National Air Toxics Assessment (NATA). The EPA, however, was unable to develop local estimates of air toxic emissions and assess risk at the local level.

This report represents Phase I of DATAS, which includes work completed as of June 30, 2005.

2.0 Monitoring

Monitoring of air toxics was conducted at five locations throughout 2003. The five sites included three in New Castle County – Martin Luther King Blvd. in Wilmington, Delaware City, and Lums Pond State Park near Middletown and one location each in Kent and Sussex Counties — Killens Pond State Park near Felton and Seaford, respectively.

The air toxics of concern were grouped into five compound categories based on sampling requirements. The categories included volatile organic compounds (VOC), carbonyls, metals, polycyclic aromatic hydrocarbons (PAH), and dioxins/furans (D/F). Sampling occurred every sixth day for all chemicals except dioxins/furans, which were sampled as 12-day composites.

A high sample collection rate for air toxics monitoring was achieved. For all five classes of chemicals monitored at each monitoring location, valid sample collection rates ranged from 68% to 95%. Most DATAS chemicals included in the monitoring effort were detected with sufficient frequency to develop annual average concentrations for use in the risk assessment.

The results of the monitoring effort reveal that the highest annual average concentrations were observed at the Martin Luther King (MLK) site. Some notable exceptions include: the highest formaldehyde and bromomethane annual average concentrations observed at the Seaford site; the highest naphthalene at the Lums Pond site; the highest concentration of seven of the seventeen dioxin/furans at the Felton site (the remaining ten were highest at the MLK site); and the highest vinyl chloride at the Delaware City site.

Preliminary comparisons of results for selected compounds show the range of monitored data to be comparable to results reported for other urban sites in the nationwide Urban Air Toxics Monitoring Program (UATMP).

3.0 Emissions inventory

The major source sectors that comprise the inventory include: point sources (facilities including manufacturers and power plants that report to DNREC); area sources (dry cleaners, auto paint shops, etc.); on-road mobile sources (motor vehicles, etc.); and off-road vehicles and equipment (farm vehicles, boats, etc.).

A statewide air toxics inventory was created for 2002 for all sources of DATAS chemicals and projected to 2003 to align with the monitoring data. The inventory represents only directly-emitted DATAS chemicals; the modeling work will characterize any secondary formation of air toxics. The inventory will serve as the primary input to the modeling effort in Phase II.

Mobile sources (on-road and off-road) account for two-thirds of the mass of emissions of reported DATAS chemicals. Mobile sources also account for the majority of VOC and carbonyl

emissions. The majority of metal emissions were reported by point sources, while the majority of PAHs and D/Fs were reported under area sources. Residential wood combustion and the several open burning categories contribute most to the emissions of PAHs and D/Fs.

4.0 Prototype study of modeling

Air toxics modeling, the primary focus of Phase II, will assess air toxic health impacts in communities other than those monitored. As part of the Phase I effort, a modeling prototype study was performed to assess the modeling design. The design is comprised of two modeling scales, local-scale modeling to address the impact from nearby sources, and regional-scale modeling for assessing long-range transport and the secondary production of compounds due to photochemistry.

Local-scale modeling demonstrated the ability to capture spatial and temporal variability of air pollutants which help identify and characterize hot spots of air toxics. Regional-scale simulation provided evidence of the important role of photochemistry in modeling and the importance of long-range transport.

To provide a comprehensive picture of air toxics, the air dispersion modeling effort will generate ambient concentrations at a fine resolution throughout Delaware. Adjustments to the model assumptions and inputs, including reassessment of the emission inventory, may be necessary for Phase II based on comparability with the Phase I monitored results.

5.0 Risk assessment

The Delaware Division of Public Health (DPH) acted as technical advisor by performing an assessment of potential risk posed by the air toxic concentrations observed at the five monitoring locations. Individual chemical risks to human health from exposure to these toxics were calculated. The cumulative risk is determined by totaling the risk values of all the individual chemicals.

Risks for cancer and non-cancer adverse health effects were calculated for three populations—adult exposure, child exposure, and an age-adjusted exposure - a mix of child and adult factors. The methodology used included a conservative margin of safety given the inherent uncertainties with risk assessment. The methodology used EPA published inhalation risk factors when available and oral risk factors when inhalation factors were not available.

The DPH established three categories of risk based on previous risk assessment studies and standard methods and assumptions. These include low risk, increased risk, and high risk, visually defined as green (low), yellow (increased), and red (high).

For the cancer endpoint, low risk is defined as one or less additional cancer case per 100,000 exposed persons. Increased risk is defined as greater than one but less than ten per 100,000 exposed persons, and high risk is defined as ten or more additional cancer cases.

To evaluate non-cancer adverse systemic health effects, hazard quotient (HQ) and hazard index (HI) are used. HQ is the ratio of potential exposure to an individual compound to the level of exposure at which no adverse health effects are expected. A low adverse health effect level is defined by a HQ of one or less, while an increased adverse health effect level is defined as an HQ greater than one but less than ten. A high adverse health effect is defined as a HQ of 10 or greater. When the HQ's are summed, the cumulative adverse health effect level is expressed as HI. Hazard index (HI) of one or less is defined as a low adverse health effect, while a HI of greater than one but less than ten is defined as an increased adverse health effect. A high adverse health effect is defined as a HI of 10 or greater.

5.1 Risk assessment of monitoring results

Exposure to individual chemicals – risk assessment for cancer

Risk assessment results (Table ES-1) show that no single chemical poses a risk greater than 1 additional cancer case per 100,000 exposed people to the population living near the monitoring sites. (Green; low risk)

Exposure to individual chemicals - risk assessment for non-carcinogenic effects

The risk assessment results (Table ES-2) show no single chemical with a hazard quotient (HQ) greater than one (Green; low risk). A hazard quotient of greater than one would result in an increased potential for adverse health effects.

Exposure to all chemicals - cumulative risk assessment for cancer

Cumulative cancer risk from each monitoring station (Table ES-3) did not exceed ten additional cancer cases per 100,000 exposed people. No cumulative risk fell in the high risk (Red) range.

The cumulative risk from chemicals at the Martin Luther King site indicated an increased cancer risk (Yellow) for adult, child, and age-adjusted populations. The potential risks for the three populations range from 1.4 additional cancer cases per 100,000 exposed people to 4.4 additional cancer cases per 100,000 exposed people.

All other monitoring sites have cumulative risk assessments in the increased range (Yellow) for the adult and age-adjusted populations. The cumulative risk ranges from 1.8 additional cancer cases per 100,000 exposed people to 3.5 additional cases per 100,000 exposed people.

All other monitoring sites have cumulative risk assessments in the low range (Green) for the child population. The summed risk is less than one additional cancer case per 100,000 people in the child population.

Exposure to all chemicals - cumulative risk assessment for non-carcinogenic effects

The cumulative adverse health effect levels (Table ES-4) at each monitoring station did not exceed 10. No cumulative adverse health effect level fell in the high HI range.

The Martin Luther King site has cumulative adverse health effect levels for all populations in the increased range (Yellow). The adverse health effects for the three populations at the site range from 1.2 to 2.6.

All other monitoring sites have cumulative adverse health effects for the child population in the increased range (Yellow). The adverse health effects for children range from a HI of 1.3 to a HI of 1.4.

All other monitoring sites have adverse health effects for the adult and age-adjusted populations with a HI of less than one (Green; low risk).

6.0 Air Toxics of Concern

Air toxics of concern or significant contributors to cancer and adverse health effects were those air toxics whose individual risk values or effects were the highest for a given monitoring station. For the purposes of this assessment, an air toxic contributing 10% or more of the cumulative (summed) risk was considered a significant contributor.

Carbon tetrachloride, chromium, and trichloroethylene were significant contributors to cancer at all five monitoring sites. Carbon tetrachloride and manganese were significant contributors to adverse health effects at all five monitoring sites. In addition, for the MLK site, benzene and 1,3-butadiene were significant contributors to cancer, while 1,3-butadiene and 1,2,4-trimethylbenzene were significant contributors to adverse health effects. Lastly, benzene and vinyl chloride were significant contributors to cancer for the Delaware City site.

Existing programs seem to have provided a reasonable level of protection for human exposure to individual compounds. However, this picture may change as the study expands its statewide geographical scope in Phase II and expands the list of compounds during the modeling phase.

7.0 Next Steps

In the fall of 2005, DNREC will begin its outreach and community efforts to build community awareness and involvement based on the risk results for the five monitoring sites.

Phase II will be completed in 2006. This Phase will include air dispersion modeling and the assessment of potential risks to human health throughout Delaware. The air dispersion modeling will enable a better understanding of ambient concentrations of air toxics in each of Delaware's communities and the potential exposure to those air toxics. Related health risks to specific communities will be evaluated and partnerships will be created to help mitigate adverse impacts.

Once Phase II is completed, DNREC will be able to prioritize actions to address the highest areas of risk found in Delaware. As part of its Air Toxics Strategic Plan submitted to the EPA (Region III) in February 2005, DNREC will develop of a statewide Risk Management Plan to address air toxics. In addition, DNREC will establish a stakeholder process to develop (1) action levels based on risk and (2) a process for addressing risks that require action.

Through the DATAS project, DNREC will continue a collaborative effort with the EPA, DPH, and the Delaware Cancer Consortium to evaluate air toxics and help protect the health and well-being of the citizens of Delaware.

Table ES-1: Risk Assessment for cancer¹ from exposure to each individual chemical at the five monitoring sites

Risk Scenarios	Martin Luther King Area Site	Delaware City Area Site	Lums Pond Area Site	Felton Area (Killens Pond) Site	Seaford Area Site
Adult	Less than 1 additional cancer case per 100,000 exposed people	Less than 1 additional cancer case per 100,000 exposed people	Less than 1 additional cancer case per 100,000 exposed people	Less than 1 additional cancer case per 100,000 exposed people	Less than 1 additional cancer case per 100,000 exposed people
Child	Less than 1 additional cancer case per 100,000 exposed people	Less than 1 additional cancer case per 100,000 exposed people	Less than 1 additional cancer case per 100,000 exposed people	Less than 1 additional cancer case per 100,000 exposed people	Less than 1 additional cancer case per 100,000 exposed people
Age-adjusted (combination of adult and child)	Less than 1 additional cancer case per 100,000 exposed people	Less than 1 additional cancer case per 100,000 exposed people	Less than 1 additional cancer case per 100,000 exposed people	Less than 1 additional cancer case per 100,000 exposed people	Less than 1 additional cancer case per 100,000 exposed people

¹ None of the 5 monitoring sites had cancer risk in the High or Increased Risk ranges for any individual chemical.

Legend

High Risk	High Risk: 10 or more additional cancer cases per 100,000 exposed people
Increased Risk	Increased Risk: Greater than 1 but less than 10 additional cancer cases per 100,000 exposed people
Low Risk	Low Risk: 1 or less additional cancer case per 100,000 exposed people

Table ES-2: Risk Assessment for adverse health effect level for non-cancer¹ from exposure to each individual chemical at 5 monitoring sites

Risk Scenarios	Martin Luther King Area Site	Delaware City Area Site	Lums Pond Area Site	Felton Area (Killens Pond) Site	Seaford Site
Adult	Adverse health effect level of less than 1	Adverse health effect level of less than 1	Adverse health effect level of less than 1	Adverse health effect level of less than 1	Adverse health effect level of less than 1
Child	Adverse health effect level of less than 1	Adverse health effect level of less than 1	Adverse health effect level of less than 1	Adverse health effect level of less than 1	Adverse health effect level of less than 1
Age-adjusted (combination of adult and child)	Adverse health effect level of less than 1	Adverse health effect level of less than 1	Adverse health effect level of less than 1	Adverse health effect level of less than 1	Adverse health effect level of less than 1

¹None of the 5 monitoring sites had adverse health effect levels in the High or Increased Risk ranges for any individual chemical.

Legend

High Risk	High Level: Adverse health effect level of ten or greater.
Increased Risk	Increased Level: Adverse health effect level of greater than one but less than ten.
Low Risk	Low Level: Adverse health effect level of one or less.

Table ES-3: Cumulative¹ risk assessment for cancer² cases from exposure to all chemicals at five monitoring sites

Risk Scenarios	Martin Luther King Area Site	Delaware City Area Site	Lums Pond Area Site	Felton Area (Killens Pond) Site	Seaford Area Site
Adult	3.2 additional cancer cases per 100,000 exposed people	2.2 additional cancer cases per 100,000 exposed people	1.8 additional cancer cases per 100,000 exposed people	1.9 additional cancer cases per 100,000 exposed people	1.8 additional cancer cases per 100,000 exposed people
Child	1.4 additional cancer cases per 100,000 exposed people	Less than 1 additional cancer case per 100,000 exposed people	Less than 1 additional cancer case per 100,000 exposed people	Less than 1 additional cancer case per 100,000 exposed people	Less than 1 additional cancer case per 100,000 exposed people
Age-adjusted (combination of adult and child)	4.4 additional cancer cases per 100,000 exposed people	3.5 additional cancer cases per 100,000 exposed people	2.6 additional cancer cases per 100,000 exposed people	2.7 additional cancer cases per 100,000 exposed people	2.5 additional cancer cases per 100,000 exposed people

¹“Cumulative” risk represents the sum of all values of the individual chemicals.

²None of the five monitoring sites had cancer risk in the High Risk range.

Legend

High Risk	High Risk: 10 or more additional cancer cases per 100,000 exposed people
Increased Risk	Increased Risk: Greater than 1 but less than 10 additional cancer cases per 100,000 exposed people
Low Risk	Low Risk: 1 or less additional cancer case per 100,000 exposed people

Table ES-4: Cumulative¹ adverse health effect level for non-cancer² from exposure to all chemicals at 5 monitoring sites

Risk Scenarios	Martin Luther King Area Site	Delaware City Area Site	Lums Pond Area Site	Felton Area (Killens Pond) Site	Seaford Area Site
Adult	Adverse health effect level of 1.2	Adverse health effect level less than 1	Adverse health effect level of less than 1	Adverse health effect level of less than 1	Adverse health effect level of less than 1
Child	Adverse health effect level of 2.6	Adverse health effect level of 1.4	Adverse health effect level of 1.4	Adverse health effect level of 1.3	Adverse health effect level of 1.3
Age-adjusted (combination of adult and child)	Adverse health effect level of 1.6	Adverse health effect level of less than 1	Adverse health effect level of less than 1	Adverse health effect level of less than 1	Adverse health effect level of less than 1

¹“Cumulative” adverse health effect level represents sum of all values of individual chemicals.

²None of the 5 monitoring sites had risk scenarios in the high level range.

Legend

High Risk	High Level: Adverse health effect level of ten or greater.
Increased Risk	Increased Level: Adverse health effect level greater than one but less than ten.
Low Risk	Low Level: Adverse health effect level of one or less.

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List of Acronyms

ALADE	American Lung Association of Delaware
AQM	Air Quality Management
AT	Air Toxics
ATSP	Air Toxics Strategic Plan
CC	Cancer Consortium
CIAC	Community Involvement Advisory Council
CMAQ	Community Multi-scale Air Quality model
CNG	Compressed Natural Gas
CO	Carbon Monoxide
CTF	Cancer Task Force
D/F	Dioxins/Furans
DATAS	Delaware Air Toxics Assessment Study
DC	Delaware City (monitoring station site)
DelDoT	Delaware Department of Transportation
DHSS	Department of Health and Social Services
DNREC	Department of Natural Resources and Environmental Control
DPH	Division of Public Health
dPM	Diesel Particulate Matter
EGAS	Economic Growth Analysis System
EGU	Electric Generating Units

EHEB	Environmental Health Evaluation Branch
EPA	Environmental Protection Agency
HPLC	High Performance Liquid Chromatography
HSCA	Hazardous Substances Cleanup Act
IPP	Inventory Preparation Plan
KP	Killens Pond (monitoring station site)
LP	Lums Pond (monitoring station site)
LPG	Liquid Petroleum Gas
MATES	Multiple Air Toxics Exposure Study
MDL	Minimum Detection Limit
MLK	Martin Luther King (monitoring station site)
NATA	National Air Toxics Assessment
NCAR	National Center for Atmospheric Research
NCEP	National Climate and Environmental Prediction
NERL	National Exposure Research Laboratory
PAH	Polycyclic Aromatic Hydrocarbon
PAMS	Photochemical Assessment Monitoring Station
PIC	Products of Incomplete Combustion
PM	Particulate Matter
PUF	Polyurethane Foam
RBC	Risk Based Concentration
RfC	Reference Concentration

RfD	Reference Dose
RWC	Residential Wood Combustion
SE	Seaford (monitoring station site)
SIP	State Implementation Plan
TEF	Toxic Equivalency Factor
TPY	Tons Per Year
TRI	Toxics Release Inventory
UATMP	Urban Air Toxics Monitoring Program
UCL	Upper Confidence Limit
VMT	Vehicle Miles Traveled
VOC	Volatile Organic Compound
WILMAPCO	Wilmington Area Planning Council

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Section 1

Introduction

Air toxics are the airborne pollutants that have potential to cause adverse impacts to the environment and human health and are emitted into the air either from natural sources (e.g., trees, volcanoes), or manmade sources (e.g., emissions from vehicles, industries and burning of trash). The Delaware Air Quality Management (AQM) Section of the Department of Natural Resources and Environmental Control (DNREC) embarked on the Delaware Air Toxics Assessment Study (DATAS) in early 2002 to gain a better understanding of ambient concentrations of air toxics throughout Delaware, and possible health risks associated with human exposure to those air toxics.

1.1 Historical trends in air toxics monitoring in Delaware

Routine monitoring of air quality in Delaware started in the late 1960s with measurements of specific chemical compounds, termed criteria pollutants. These pollutants included sulfur dioxide (SO₂), nitrogen dioxide (NO₂), carbon monoxide (CO), ozone (O₃), particulate matter (PM), and lead (Pb). Measured concentrations of these criteria pollutants were compared to national ambient air quality standards per 40 CRF, Part 50. In the 1980s, several limited term studies of additional air toxics pollutants were undertaken, concentrating upon fence-line characterization for selected chemical species. However, to-date, there are no national ambient air quality standards for air toxics.

During the mid-1990s, under the Photochemical Assessment Monitoring Station (PAMS) and several other programs, routine sampling and analysis of targeted volatile organic compound (VOC) species began. These programs, however, were limited in either location (monitoring at one or two sites) or duration (monitoring for a few months). The longest period of continued monitoring has taken place in Wilmington, and Figure 1.1 shows trends in selected VOCs in that city. As is evident from these trends, levels of most VOCs have declined over the last decade. However, temporal fluctuations in the concentrations of some chemical species like ethyl benzene, toluene, m,p-xylenes and o-xylene, and possible seasonal variations, highlight the importance of continued measurements.

1.2 Study purpose

DATAS represents the first attempt to simultaneously conduct year-round ambient monitoring of not just VOCs, but also other air toxics including carbonyls, Poly Aromatic Hydrocarbons (PAH), metals and dioxins/furans, throughout Delaware, and use the data to perform human health risk assessment.

Two additional factors contributed to the inception of DATAS. First, to better understand Delaware's high cancer incidence, the Governor's Cancer Task Force (CTF) recommended evaluating environmental factors including the risk from air toxics. Second, the United States

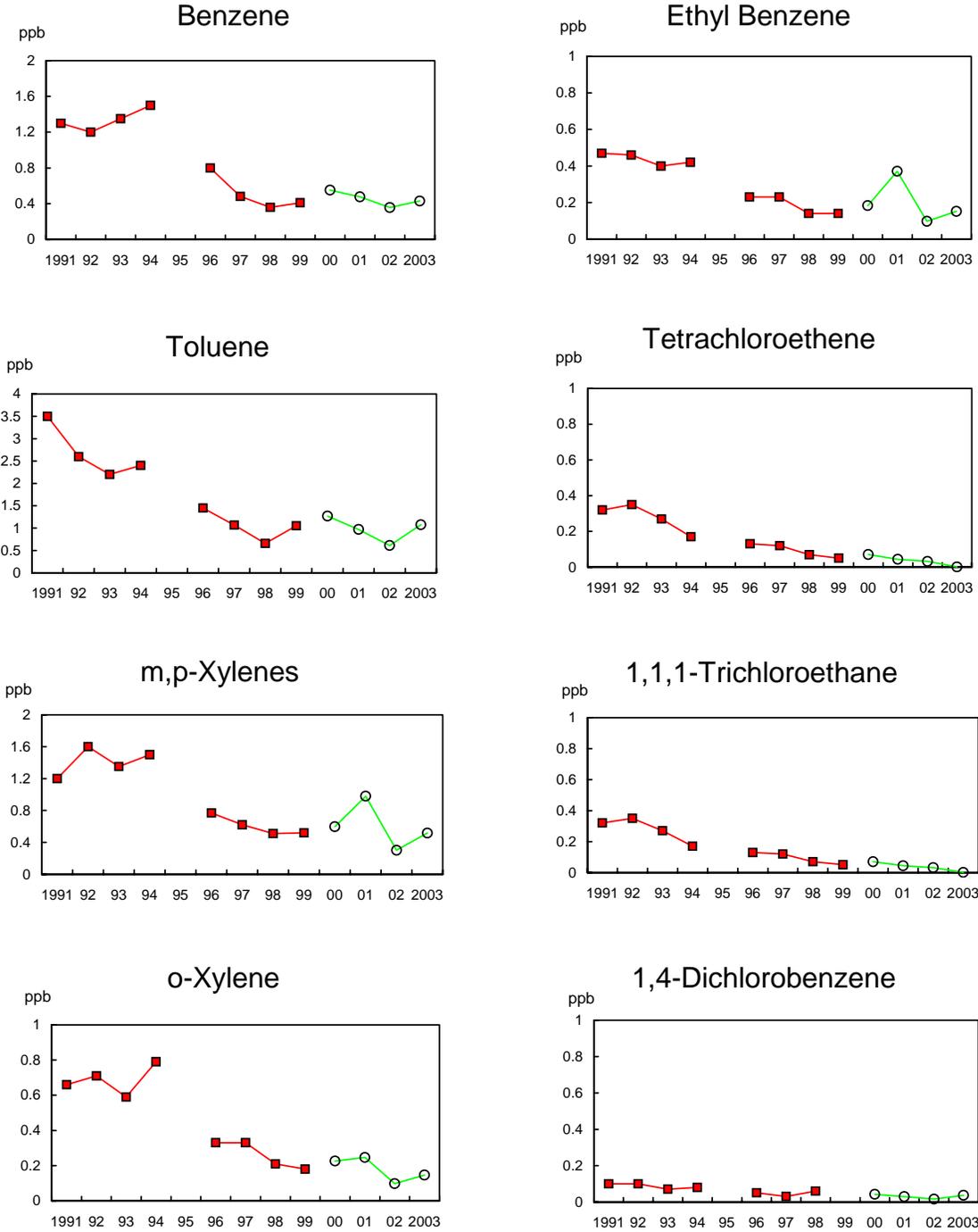


Figure 1.1: Annual averages of selected air toxics in Wilmington, DE [1]. Missing data during 1995 was due to insufficient data points. Since 2000, a new method has been used for analysis [1].

Environmental Protection Agency (EPA) brought attention to air toxics through a nationwide study known as the National Air Toxics Assessment (NATA). The NATA study identified certain air toxics that pose the greatest risk on a national or regional level. However, in many cases, EPA was unable to develop local estimates of air toxics emissions and relied on national default values to generate much of the emissions data that went into NATA analysis. This approach had limited accuracy in identifying risks at county and local levels. AQM designed DATAS to quantify risk from air toxics at the local level with Delaware-specific monitoring and emission inventory data.

The monitoring, modeling, and risk assessment activities that are part of DATAS are consistent with EPA's defined role for the State/Local/Tribal (STL) community. Examples of a few other air toxics projects with some/all of DATAS components include:

- Portland Air Toxics Assessment ,
<http://www.deq.state.or.us/aq/HAP/index.htm>
- Camden Waterfront South Air Toxics Pilot Project,
<http://www.nj.gov/dep/ej/airtoxics.html>
- West Louisville Air Toxics Study,
<http://yosemite.epa.gov/oar/CommunityAssessment.nsf/0ac25a80881d86a685256e510077f406/79cdd986e28be1a985256c41005e447d!OpenDocument>
- Southern Delaware County Air Monitoring Project,
<http://yosemite.epa.gov/oar/CommunityAssessment.nsf/0ac25a80881d86a685256e510077f406/9d2be81197c4b16385256c6e005d8b93!OpenDocument>
- Multiple Air Toxics Exposure Study in the South Coast Air Basin (MATES II),
<http://www.aqmd.gov/matesiidf/matestoc.htm>
- Houston , Texas,
http://www.tceq.state.tx.us/comm_exec/tox/ESLMain.html

In designing DATAS, AQM sought to develop a mature toxics monitoring network, a superior emission inventory, and an air quality dispersion modeling strategy to support the quantitative evaluation, characterization, and tracking of risk-based impacts resulting from ambient levels of air toxics throughout Delaware. Although this required a modeling effort to predict concentrations, the study was defined by an approach to systematically verify modeling simulations through the collection of monitoring data for most of the compounds. Results of the study are expected to satisfy the environmental objectives discussed in the CTF's Four-Year Plan, presented in *Turning Commitment into Action* [2]. The Plan addresses the need to:

- Learn more about the possible health risks of ambient concentrations to toxins,
- Promote the exchange of useful information to the citizens of Delaware,
- Understand where risks can be avoided, and

- Provide information from which sound healthy decisions and good policies can be developed.

The immediate goal of DATAS is to characterize risk associated with ambient concentrations of air toxics as monitored at five locations (Figure 1.2) and modeled over the selected neighborhoods for 2003. The ultimate goal of the study is to create a modeling tool that can be used with future inventories to reassess ambient concentrations and statewide risk in the future.

Products of DATAS will be helpful in (1) providing air quality information that may assist the process of establishing air quality standards based on risk, (2) establishing control strategies for the purpose of meeting the new standards, (3) assisting the permitting process base its decisions on cumulative impacts, and (4) improving future emissions inventory work.

1.3 Air toxics included in DATAS

Air toxics that were included in the DATAS were selected based upon following criteria:

- Selection of toxics identified by EPA as the most prevalent in urban air [3],
- Identification of toxic compounds for which monitoring methods have been developed [4],
- Presentation of the NATA results, and
- Review of the Multiple Air Toxics Exposure Study (MATES) II [5].

The list of selected air toxics for DATAS is detailed in the Appendix (Table A1) of this report. The Appendix also identifies typical sources of each air toxic and the ability to monitor, inventory, model, and/or assess risk from potential exposure to these chemical compounds.

1.4 Project organization

In defining the scope-of-work for this study, AQM was challenged with expanding upon the work performed by EPA under the NATA study to provide enhanced resolution of ambient toxics concentration information to the citizens of Delaware, without compromising the rigor and integrity of existing infrastructure established through many of the EPA-mandated environmental programs.

DATAS objective, therefore, was to support a complete 2003 modeling simulation for selected air toxics such that AQM can confidently predict ambient air toxics concentrations and potential health risk associated with them. Due to the complexity of this requirement, a multi-disciplinary effort was needed to execute this study. Technical expertise from AQM and the Department of Health and Social Services-Division of Public Health (DPH) included:

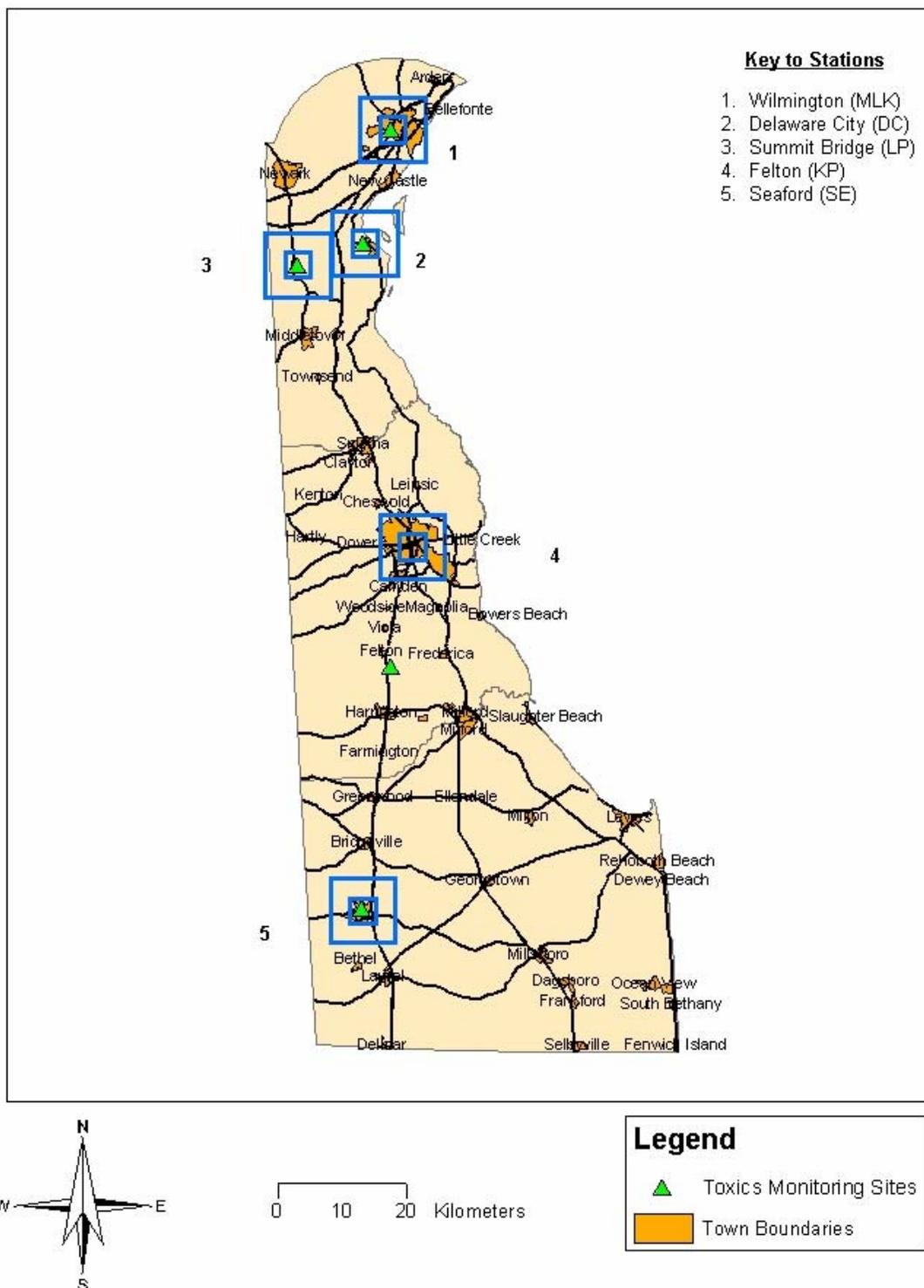


Figure 1.2: Air Toxics Monitoring Locations in Delaware. Blue boxes represent 5 and 10 km modeling grids.

- Air Toxics Monitoring,
- Emissions Inventory Development,
- Human Health Risk Assessment and Management
- Data Modeling, and
- Communication.

Each of these objectives is briefly discussed in the following:

1.4.1 Monitoring

The AQM has strived, throughout this study, to provide detailed outdoor toxics concentration information down to a neighborhood level, i.e., to characterize pollutant concentrations within an extended area that has relatively uniform land use with dimensions in the 0.5 to 4.0-kilometer range. Traditionally, an approach to provide such a characterization would have focused solely on the establishment of monitoring capabilities throughout the state for selected toxics. Recognizing that placement of monitoring stations within every community in Delaware would be impractical and cost-prohibitive, toxics monitoring capabilities for the DATAS chemicals shown in Table A1 (Appendix) at five existing monitoring sites were enhanced. The monitoring effort was combined with an extensive toxics modeling effort for Delaware.

The five monitoring locations (Figure 1.2) indicated by triangles, represent existing monitoring infrastructure, which provide an efficient and cost-effective approach to the establishment of monitoring capabilities. Placement of these monitors satisfies all EPA siting criteria for this type of monitoring [1].

1.4.2 Emissions inventory

A comprehensive air toxics emissions inventory for 2003 was required in order to align the modeling and monitoring efforts. Since it was not possible to develop a full 2003 inventory in the allotted timeframe, the inventory was prepared for 2002, and then projected to 2003, accounting for changes in activity and controls. The 2002 toxics inventory was developed in conjunction with the criteria pollutant, fine particulate precursor, and ozone precursor inventories. In order to serve as a modeling input, special effort was made to develop an inventory with a high degree of spatial and temporal resolution.

1.4.3 Modeling

Air toxics dispersion modeling simulates the fundamental physical and chemical processes of airborne contaminants, thereby enabling the prediction of ambient concentrations of air toxics. This tool, when developed properly, can provide a myriad of information to the public regarding the exposure of ambient air toxics concentrations. Air toxics dispersion modeling provides the best possible estimation of ambient air toxics concentrations provided:

- Compound-specific emissions inventory from both local and regional sources is identified within the modeling domain,

- Necessary meteorological data for the simulation period are collected, and
- Good model performance is assured by comparing predicted concentrations with measured concentrations, and if necessary fine-tuning the model assumptions and emissions inventory data to achieve maximum confidence in the predicted concentrations.

Air toxics modeling for DATAS includes both local- and regional-scale modeling - local-scale modeling to address the impact from nearby sources and regional-scale modeling to account for long-range transport and production of certain carbonyl compounds due to oxidant photochemistry.

To assist in the model development, a prototype modeling simulation of 1999 emissions and 2001 meteorology was performed with the following work objectives:

- Identification of resource requirements to simulate a regional-scale modeling exercise, and
- Development of infrastructure required to perform the regional-scale modeling.

The final design of the 2003 DATAS modeling work is currently being developed based on lessons learned from the prototype work. Results of the prototype modeling simulations are presented in this report. The DATAS local- and regional-scale modeling work will be completed towards the end of 2005 (see Section 9.1.1) and presented at a later date following risk analysis.

1.4.4 Human health risk assessment

Human health risk assessment was performed to characterize possible risk associated with potential exposure to ambient air concentrations of the air toxics. Ambient air concentrations of air toxics, determined from either the monitoring data or prediction through the use of dispersion models, was employed in this risk assessment. The Division of Public Health (DPH) was charged with the task of performing an assessment of risk posed by individual toxics as well as cumulative risk for monitored concentration. Once the modeling work is complete, similar risk assessment will be carried out for the predicted air toxics concentrations.

1.4.5 Communication

Communications for DATAS has been an ongoing effort, and includes internal communications within AQM and external communication between AQM, DPH, and the Cancer Consortium (CC). With publishing of this report, a more comprehensive communication and outreach plan has been developed and is being implemented. This plan includes the initial outreach effort for the five monitoring sites (Phase I), as well as a statewide outreach effort for the modeling results (Phase II). As both carcinogenic and non-carcinogenic risks are addressed in the final study, outreach activities will be focused as a coordinated effort between AQM, DPH, and CC to establish priorities for the state.

1.5 Subject matter experts

Due to Multi-disciplinary nature of DATAS, many technical experts from AQM and DPH participated in the study. Table 1.1 identifies DATAS subject matter experts, by discipline.

A detailed work protocol was developed [7], defining the specific methodologies implemented for each of these disciplines.

Table 1.1: Subject matter experts

Discipline	Agency	Contact Name	Phone Number
Monitoring Air Toxics	AQM (DNREC)	Joe Martini, Najid Hussain	302-323-4542
Monitoring Data Analysis	AQM (DNREC)	Betsy Frey, Sonal Iyer	302-323-4542
Emissions Inventory	AQM (DNREC)	David Fees	302-739-9402
Risk Assessment	DPH	Jerry Llewelyn, George Yocher	302-744-4540
Modeling	AQM (DNREC)	Mohammed Majeed	302-323-4542
Risk Management	AQM (DNREC)	Jim Snead	302-323-4542
Outreach and Communications	DNREC DPH Cancer Consortium	Terri Brixen, Christina Wirtz Heidi Truschel-Light Meg Maley	302-323-4542 302-395-2515 302-744-4907 302-455-1500

1.6 Report organization

DATAS has been organized into two phases. This document presents work and findings for Phase I of the study, which consists of work completed to date, including:

- Quality-assured 2003 monitoring data, including summary statistics,
- Risk assessment of annual averages of monitored concentrations,
- Quality-assured 2003 emission inventory,
- 2001 modeling prototype,
- Process for developing a risk management plan, and

- Communications and outreach efforts to date.

The report also describes work to be completed under Phase II of the study which will include:

- Local and regional scale modeling of select air toxics of concern,
- Risk assessment of those modeled concentrations,
- An in-depth statistical analysis of the monitored concentrations, and
- Process for developing a modified risk management plan (only if needed based on risk assessment from modeled concentrations).

This report is organized as follows:

- Section 1 – Introduction
- Section 2 – Ambient Monitoring
- Section 3 – Emissions Inventory
- Section 4 – Human Health Risk Assessment of Monitored Data
- Section 5 – Results and Discussion
- Section 6 – Modeling
- Section 7 – Communications
- Section 8 – Conclusions
- Section 9 – On-going AQM Air Toxics Activities
- Section 10 – Recommendations for Future Work

The detailed analysis of the 2003 modeling results and risk assessments based on the modeled air toxics concentrations will be presented in Phase II report anticipated for delivery in the mid-2006. Results of an in-depth statistical analysis of the monitoring data will also be included in Phase II report.

Section 2

Ambient Monitoring

2.1 Monitoring stations

The ambient monitoring to support DATAS required characterization of air toxics at locations in each of the three counties throughout Delaware. Because Delaware currently maintains a statewide ambient monitoring network, the integration of DATAS into the existing infrastructure offered an efficient and cost-effective solution to the project.

Delaware's existing monitoring network consists of eleven monitoring stations. To support this project, five of these stations (Figure 1.2) were equipped with toxics monitoring instruments. The five sites, identified in Table 2.1, were selected based upon the following criteria:

- Population within the area surrounding the site,
- Location of the site relative to industrial facilities,
- Location of the site relative to major roadways,
- Ability of existing infrastructure at the site to support additional toxics instrumentation,
- Incorporation of sites already equipped with air toxics instrumentation.

Table 2.1: DATAS monitoring sites

County	Site Name	Site Abbreviation
New Castle	Martin Luther King Boulevard (Wilmington)	MLK
	Route 9 (Delaware City)	DC
	Lums Pond State Park (Summit)	LP
Kent	Killens Pond (Felton)	KP
Sussex	Seaford	SE

A brief history for each site is presented in the following subsections.

2.1.1 MLK Site

Located at the corner of Martin Luther King Jr. Blvd. & Justison St. in the city of Wilmington, this monitoring site was established in January 1999 as a replacement for the site that had been located at the corner of 12th & King Street since 1966. It represents urban central Wilmington and is influenced by emissions from nearby major highways as well as numerous small local sources.

2.1.2 DC Site

This site was originally established in January 1992 when carbon monoxide and sulfur dioxide analyzers were moved to this location from another site in Delaware City (Governor Bacon Health Center). The current site is on Route 9 just northwest of Delaware City and is often influenced by emissions from the Delaware City industrial complex.

2.1.3 LP Site

Monitoring at the current location in Lums Pond began in 1992. The published name of the site was changed from Lums Pond State Park to Summit Bridge during 2000 to help clarify that the measurements represented an area larger than the park itself. The site location was chosen to characterize transport of air pollutants and ozone precursors during the summer months when the wind direction is frequently from the highly urbanized Baltimore/Washington area (southwesterly).

2.1.4 KP Site

This site was established in 1995 as a replacement ozone monitoring station for the Dover monitoring site that was discontinued in 1994. The published name of the site was changed from Killens Pond State Park to Felton in 2000 to help clarify that the measurements represented an area larger than the park itself. It is usually representative of background concentrations although it can also be affected by transported pollution.

2.1.5 SE Site

This is the second ozone monitoring location to be used in the Seaford area. The previous location was near the City of Seaford water tower on Pine St. The current site is on the grounds of the Shipley State Service Center and is used for seasonal ozone monitoring and year-round PM 2.5 monitoring. The station is representative of a smaller urban/suburban area.

2.2 Sampling methods

To characterize the toxic compounds defined for this study (Table A1, Appendix), AQM installed compound-specific (VOC, metal, PAH, D/F, carbonyl) monitors. A separate sampler is therefore required for each compound group.

Sampling equipment includes:

- High volume (HiVol) total particulate samplers for metals,
- Xontech 910 samplers for VOCs,
- Modified R.M. Environmental 925 samplers for carbonyls, and
- HiVol, polyurethane foam (PUF) samplers for PAHs and D/Fs.

All compounds were sampled at all sites, and analyzed in accordance with the following EPA-approved methods:

- Volatile Organic Chemicals (VOCs) – Method TO-15, determination of VOCs in air collected in specially-prepared canisters and analyzed by gas chromatography/mass spectrometry (GC/MS) [8],
- Metals – Method IO-3.5, determination of metals in ambient particulate matter using inductively coupled plasma/mass spectrometry (ICP/MS), TSP [9],
- Polycyclic Aromatic Hydrocarbons (PAHs) – Method TO13a, determination of PAHs in ambient air using gas chromatography/mass spectrometry (GC/MS) [10],
- Carbonyls – Method TO-11a, determination of carbonyls in ambient air using adsorbent cartridge followed by high performance liquid chromatography (HPLC) [active sampling methodology] [11], and
- Dioxins/Furans (D/Fs) – Method TO-9a, determination of polychlorinated dibenzo-p-dioxins and dibenzofurans in ambient air [12].

2.3 Monitoring schedules

The sampling equipment installed at each site required that samples be collected on either a filter or in a container. Following AQM's Standard Operating Procedures, the samples were collected on a pre-determined schedule, for a specified period of time, and then shipped to an off-site laboratory (Table 2.2) for extraction and analysis [13-16].

Monitoring schedules for VOCs, PAHs, metals, and carbonyls consisted of 24-hour samples collected once every sixth day for the calendar year 2003. This one-in-six day schedule was coordinated with the national schedule for particulate, Photochemical Assessment Monitoring Stations, and urban air toxics trend site monitoring. For 2003, there were 61 scheduled sample days for each of VOC, carbonyls, metals, and PAHs. The actual number of samples collected, however, can be less than 61 depending upon equipment failure or various other factors.

For the PAH samples, there was the possibility of evaporative losses between end-of-sampling and sample retrieval during periods of high ambient temperatures. During summer months only, therefore, the PAH samples collected on either a Friday or Saturday were run throughout the weekend, until 12:01 AM Monday. Because of the difference in sampling time and the potential for introducing bias, these samples were not included in generating the annual summary statistics.

Exception to this sample collection schedule was for dioxin/furan sampling. Due to extremely low concentrations of dioxins/furans (D/F) in ambient air, samples were required to be collected continuously for extended periods of time using high-volume sampling methods. For

the DATAS project, therefore, D/Fs were collected according to the protocol used in the National Dioxin Ambient Monitoring Network that requires samples collected continuously over a 12-day period. After 12 days the sampler was turned off, samples removed, new collection gear mounted, and the sampler restarted for another 12-day period. Typical time for the sample retrieval procedure was on the order of an hour. The number of scheduled sample periods for dioxins/furans in 2003 was 26.

2.4 Laboratory support

The EPA provided analytical and in-kind funding support over the course of this program for those compounds identified as part of the regional toxics program, i.e., VOCs and carbonyls, for all five sites identified as part of DATAS (Fig. 1.1). Analytical support for the remaining compound groups (metals, PAHs, and D/F) was obtained from private laboratories while funding support was provided by DATAS funding sources. The list of laboratories that were used for the analysis of samples is presented in Table 2.2.

Table 2.2: List of analytical services providers

Chemical group	Laboratory	EPA/Private
VOC	Maryland Department of the Environment, Baltimore MD	EPA Region III
Carbonyls	Air Management Services Laboratory, Philadelphia, PA	EPA Region III
Metals	Research Triangle Institute, NC	Private
PAHs	Pace Analytical Services, Inc	Private
D/F	Pace Analytical Services, Inc	Private

2.5 Data validation

Initial data validation procedures involved verification of all field and laboratory database entries against hard-copy original documents. Final sample concentrations were compiled and evaluated using descriptive statistical analyses to identify outliers and gross data errors that might not have been identified in the initial validation step. Sample concentrations were also evaluated for deviation from established protocols, along with comments and/or flags from the analyzing laboratory. Unusual data points or outliers thus identified were flagged for further investigation and review.

Final data validation procedures involved further statistical evaluation and analyses including analysis of field and travel blanks, collocated samples, time-series plots and preliminary comparison with emissions information, such as the Toxics Release Inventory [15]

combined with wind-rose data. All sample values identified as invalid were not discarded, but were flagged as invalid and excluded from the final validated data set used for analysis.

2.6 Non-detects and below minimum detection limit (MDL) data

During Phase I of the DATAS project, risk assessment calculations were performed for the 2003 air toxics data set. Risk assessment calculations are based on the annual average concentration of monitored compounds. In order to perform these calculations, several issues identified below required resolution:

- Handling of target compound results that were reported as not detected (ND) or zero by the laboratory,
- Handling of target compound results that were reported by the laboratory, but were below the minimum detection limits (MDL),
- Defining the tolerance level for the number of samples reported as ND without introducing excessive bias to the dataset.

AQM chose the following approaches based on current references [13, 17-20]. It is important to note that there is no single method that is appropriate in all cases.

2.6.1 Sample results reported as not detected

When compounds are not detected in a sample, the laboratory reports the result as either not detected or zero. Such sample results are considered valid as long as they meet the validation criteria described previously (Section 2.5) and specified in the DATAS quality assurance documentation. The true concentration is unknown, but it lies somewhere between 0 and the MDL of the analytical instrument. In the DATAS project there are a number of compounds for which some of the sample results were reported as not detected or 0.

In order to calculate an annual average or mean for a dataset that includes some not detected values, several different approaches may be used. Choice of the best approach is determined mainly by the final use of the data [17-20], which for this part of the DATAS project is risk assessment. For DATAS, therefore, the following approaches were considered:

- Replacement of not detected results with 0,
- Replacement of not detected results with $\frac{1}{2}$ MDL, and
- Replacement of not detected results with the lowest reported concentration value in the dataset.

To evaluate which method was best, summary statistics were generated using these three different replacement methods for all compounds at one site. Criteria used to decide the final replacement method included, a mean which was less than the actual maximum concentration detected, consistency with actual monitored value, and evaluation of potential bias. Since the data are to be used for assessing the risk to human health, it was also necessary to use a conservative approach to data analysis to avoid underestimations.

Replacing not detected sample values with zero can bias the average on the low side and yield low risk estimates. After testing all three replacement methods, AQM therefore decided to replace the not detected values with ½ MDL concentration for data sets with up to 50% of data reported as not detected and no data reported below the MDL.

2.6.2 Sample results reported below MDL

There are also some datasets with not detected values from laboratories that reported concentrations below the MDL concentration. This can occur due to the method used to calculate the MDL (40CFR Part 136, Tables B1-B5, Appendix). The laboratories analyzing samples of VOCs, dioxins/furans, PAHs, and carbonyls reported values below the MDL, while the laboratory analyzing the metals did not report any values below the MDL. In some cases these below MDL concentrations were significantly lower than the MDL.

For these datasets, replacing the not detected values with ½ MDL resulted in a high bias in the annual average. AQM therefore chose to replace the not detected values in datasets from laboratories that reported concentrations below the MDL (VOCs, dioxins/furans, PAHs, and carbonyls) with the lowest reported concentration. This was done for datasets with up to 50% of samples reported as not detected.

2.6.3 Compounds with more than 50% of samples reported as not detected

When more than 50% of samples were reported as not detected, it was necessary to decide if enough actual data exists to generate meaningful summary statistics. Even if not detected values are replaced with another number as described previously, the probability of bias in the final dataset increases with the amount of data replaced.

Following guidance in references [13, 18], for datasets with more than 50% but less than 90% of samples reported as not detected or 0, those reported values were not replaced; rather, a percentile value that is higher than the percent of not detected samples was used in place of the calculated average. The p^{th} percentile is a value so that roughly $p\%$ of the data are smaller and $(100-p)\%$ of the data are larger. For example, at Seaford, chloroethene was reported as not detected in 88% of samples. The 90th percentile concentration was therefore used instead of the average in the final data summary.

For datasets with more than 90% of samples reported as not detected, it was concluded that not enough data exist to calculate an average or percentile value to be used in risk assessment. No further analysis was done on those compounds.

Table 2.3 summarizes the methods used to handle samples reported as not detected based on the amount of data reported as not detected and whether the laboratory reported concentrations below the official MDL.

Table 2.3: Summary of action for not detected and below MDL data

Description	% of Samples Reported as Not Detected (ND)		
	(0-50)%	(50-90)%	>90%
No data reported below MDL (metals)	ND replaced with ½ MDL to calculate the average	Next higher percentile used instead of the average	No analysis
Data reported below MDL (VOCs, carbonyls, dioxins/furans, PAHs)	ND replaced with the lowest reported value to calculate the average	Next higher percentile used instead of the average	No analysis

2.7 Results and Statistical Analysis

Summary results for each DATAS chemical are described in Section 5. Tables with the monitoring data summaries for all compounds at all sites are included in Appendix. These summaries include for each DATAS chemical:

- Average minimum detection limit (MDL),
- Highest sampled concentration (Maximum),
- Lowest sampled concentration (Minimum),
- Lowest quantitated concentration (used to replace not detected or 0 in calculating the mean and UCL for risk assessment),
- Arithmetic mean or percentile,
- Median sample value,
- Percent of samples reported as not detected or 0,
- Upper confidence limit, 95% (calculated using the Chebyshev Inequality Method [18,19]),
- Total number of valid samples collected,
- Standard deviation of sampled values, and
- Coefficient of variation of sampled values.

Section 3

Emission Inventory

A comprehensive air toxics inventory for Delaware was prepared for use as an input to the modeling effort. The inventory attempted to quantify emissions from all sources within the State of Delaware. It is comprised of four major sectors, including point sources, stationary area sources, on-road mobile sources and off-road mobile/non-road sources. The methods used to develop the 2003 air toxics emission inventory are described in this Section. Since the modeled concentrations will be compared to the monitored data to assess model performance, the inventory was prepared to align with the monitoring effort. Information on the air toxics emissions originating from outside Delaware, which will also be used in the modeling effort, will be acquired from EPA's National Emission Inventory database.

3.1 Introduction

Prior to the start of the DATAS project, AQM began developing a statewide comprehensive criteria pollutant inventory as part of Delaware's overall State Implementation Plan (SIP) for air quality. Criteria pollutants include ozone, nitrogen dioxide, carbon monoxide, sulfur dioxide, particulate matter (PM) and lead. In addition to this, the precursors to ozone and particulate formation include volatile organic compounds (VOCs) and ammonia. A comprehensive criteria pollutant and precursor inventory is required to be submitted to the EPA by states every three years, with the most recent year being 2002. AQM decided for reasons of economy and timing that a toxics inventory would be developed for 2002 in conjunction with the criteria pollutant inventory, then projected to 2003. In reality, some source sectors, including on-road mobile sources and most off-road/non-road sources, were recalculated specifically for 2003.

In order to serve as a modeling input, special effort was made to develop an inventory with a high degree of spatial and temporal resolution. Typical SIP inventories include only annual emissions at the county level. The modeling effort is focused on localized effects within five 10-kilometer x 10-kilometer neighborhood areas located throughout the State. Emissions occurring within these local areas were identified. A separate regional-scale model will be employed to develop ambient concentrations outside the local areas. Section 6 contains more details regarding the spatial resolution associated with the modeling effort.

Meteorology plays an important role in determining the distribution of pollutants after they are emitted, as well as the degree of degradation and the degree of secondary formation that may occur. Meteorological conditions change daily as well as show seasonal variations. Therefore, it is important to the modeling effort to allocate emissions over the course of the year, providing monthly, daily, and hourly emission profiles. The development of spatial and temporal resolution within the inventory is described in more detail in the source sector descriptions below.

Despite considerable effort to develop a complete and accurate inventory, there are many limitations associated with the 2003 inventory developed for the DATAS project. Most

emissions data are not based on direct measurements, such as stack tests. Delaware relied on emission factors developed by the EPA, other states (most notably California), and other published works for many of the non-point source categories.

3.1.1 Point sources

The point source sector is represented by individual facilities reporting their emissions and/or activity data to AQM. Facilities report emissions at the process level and include the throughput or activity level associated with each process. The universe of facilities selected for reporting is based on previously reported criteria and toxic pollutant emissions. Delaware's point source inventory is comprised of a variety of facility types, including power plants, the manufacturing sector, large office buildings, landfills, hospitals, universities, and government facilities.

Facilities reporting emissions are required to base emissions on stack test monitoring data when available. While stack test data are highly accurate, most facilities only monitor criteria pollutants. Therefore facilities rely on estimating toxic emissions through the use of emission factors or by applying a mass balance approach.

Emissions from facilities are highly resolved spatially since facilities provide latitude and longitude coordinates when they report. Emissions are quite verifiable since they are reported at the process level and supporting documentation is required to accompany the emissions report. Facilities are required to submit monthly throughput/activity data in order to develop a temporal profile of emissions. Emissions are often directly proportional to throughput.

3.1.2 Stationary area sources

Stationary area sources, often referred to as "area sources", represent a large and diverse set of individual categories. An area source category is either represented by small facilities too numerous to individually inventory, such as dry cleaners or gas stations, or is a common activity, such as the use of paints or cleaning solvents. Area source emissions are estimated through the use of emission factors applied to a particular business sector (e.g., dry cleaners) or an activity (e.g., architectural painting.)

There are many area source categories which contribute air toxic emissions. These categories can be grouped into one of several category types. These include:

- Solvent Use – Many products use solvents to achieve the intended purpose of the product. Paints, cleaners, pesticides, personal care products, inks, and dry cleaners contain or use solvents. A number of chemical solvents are air toxics and are being investigated by the DATAS project,
- Fuel Combustion – The combustion of fuels in commercial and residential furnaces, engines, boilers, and wood stoves create products of incomplete combustion (PICs), many of which are air toxics under DATAS. The fuels themselves contain some air toxic compounds and may be emitted unburned,

- Gasoline Usage – The distribution and use of gasoline in vehicles and other gasoline-powered engines result in emissions whenever the volatile gasoline vapors are allowed to escape,
- Open Burning – Like the fuel combustion categories, open burning creates PICs as well as releases unburned hydrocarbons. Unlike fuel combustion, open burning activities operate at relatively low temperatures, which change the mix and amount of air toxics created. Open burning categories include trash burning, wildfires, prescribed burning, and house and vehicles fires.

Area source spatial and temporal resolution is developed through the use of allocation profiles. For a given source category, a spatial allocation profile is developed based on a surrogate that matches the activity. As an example, architectural painting is concentrated in populated areas. Therefore, the county-wide emissions for architectural coatings are spatially allocated based on the distribution of households throughout the county. Likewise, temporal allocation profiles are developed for each area source category. Again, using the example of architectural coatings, painting activity is concentrated in the warmer months of the year, and the profile is created according to documented usage patterns.

A quality area source inventory relies on accurate, up-to-date emission factors and activity data representative of local conditions for the year of interest. Pollutant-specific emission factors were employed, when available. In the absence of these, chemical speciation profiles were applied to a source category's VOC and PM emissions.

3.1.3 On-road mobile sources

The on-road mobile sector accounts for combustion and evaporative emissions from on-road vehicles including motorcycles, light-duty cars and trucks, heavy-duty trucks, and buses, and further delineates between gasoline- and diesel-powered vehicles. Emissions vary between vehicle types. The age of vehicles also is an important factor in developing on-road mobile emissions. New vehicles meet cleaner standards, while older vehicle were designed under a less clean standard, or their emission control devices have deteriorated over time. Speed is yet another factor that influences overall emissions.

The most important determiner of overall emissions is the collective number of miles driven by vehicles on Delaware's roads. Known as vehicle miles traveled (VMT), the amount of VMT has increased steadily for decades and in some cases has outpaced the beneficial effect of cleaner cars entering the market. Emission factors are developed on a per mile basis, based on vehicle fleet mix (type and age) and typical speeds driven. These per-mile emission factors are multiplied by the VMT to arrive at estimated emissions.

The Delaware Department of Transportation (DelDOT) provides VMT for each roadway segment in Delaware. These segments, also known as links, represent short lengths of roadway which have coordinates associated with them. With thousands of links statewide, the on-road mobile emissions will be represented by a very detailed spatial resolution. Both emission factors and VMT are developed for each month of the year to provide the necessary temporal resolution of the emissions.

3.1.4 Off-road mobile/non-road sources

The off-road/non-road source sector contains a variety of equipment that uses an internal combustion engine that is not stationary and is not considered an on-road vehicle. The equipment can be self-propelled, such as a bulldozer or farm tractor, or equipment that is moved from place to place, such as chainsaws and leaf blowers. As with on-road sources, both engine and evaporative emissions are estimated. Emissions are estimated for equipment powered by gasoline, diesel, compressed natural gas (CNG), or liquefied petroleum gas (LPG, usually propane). Spatial and temporal resolution is handled in the same way as area sources.

Off-road/non-road equipment can be grouped into one of several category types. These include lawn and garden, recreational (land and water based), construction, farm, industrial, commercial, logging, aircraft (landings and takeoffs and support equipment), commercial marine vessels, and locomotives (including maintenance equipment).

3.2 Inventory planning

The first step in achieving a complete and accurate emission inventory was to develop an inventory preparation plan (IPP). The IPP is necessary to identify the detailed scope of work associated with creating the inventory. The IPP served as a working document to the inventory team throughout the completion of the inventory.

The IPP defines each step in the inventory development process. The steps include identifying the objectives of the inventory, the inventory parameters, the source categories to evaluate, the methodologies to employ, activity data needs and the methods for collecting and managing data, the level of documentation needed, and the quality control and quality assurance measures to be employed.

The principal objective of the inventory is to create a useful input to the air dispersion modeling. As a modeling inventory, additional spatial and temporal resolution (as previously discussed) was incorporated into the work plan. Inventory parameters, such as the year of interest (2003), the pollutants to be inventoried (see Appendix A), the geographic coverage (statewide), and the level of spatial and temporal resolution, were established in the IPP. A review of previously inventoried source categories was conducted to determine which categories could be expected to result in emissions of one or more of the DATAS pollutants.

The bulk of the work in developing the IPP was in determining the most appropriate emission estimation method for each source category. This involved determining what emission factors existed for each category and if activity data existed that was compatible with the emission factors. Methodology development also entailed identifying regulatory controls that would result in a reduction in emissions as compared to uncontrolled emissions. The selection of a particular methodology was made based on the quality of the estimated emissions, the quality and availability of method inputs, the importance of the category to the overall inventory, and time constraints.

Data collection methods varied from category to category. For point sources, the collection of data involved emissions reporting directly from facilities. For other source sectors, activity data, emission factors, and other pertinent information were obtained from published documents, through direct requests (by letter, telephone, or e-mail), or through the Internet.

Management of point source data was handled through the use of the *i*-STEPS[®] reporting software and database application. Management of area source and some off-road/non-road source categories was accomplished through the use of a standard spreadsheet template. A spreadsheet was created for each source category using the template. The spreadsheet was organized with multiple tabs, one each for activity data, emission factors, controls, calculations, output format, and quality control documentation.

Documentation for the 2003 inventory includes the IPP documents, the point source database, the area and off-road sources spreadsheets, input files created for several emissions models used for on-road and off-road sources, output files generated from these models, and final reports for each sector.

3.3 Inventory development

In an effort to complete the 2002 and 2003 criteria and toxic pollutant inventories, AQM contracted with E. H. Pechan and Associates (Pechan) to assist in the inventory development. Pechan's involvement included developing emission estimation methodologies, the creation of the area source spreadsheets and on-road and off-road model input files, and the preparation of final sector reports. AQM worked closely with Pechan to develop and approve methodologies, and assist in gathering activity data and other Delaware-specific inputs.

3.3.1 Point sources

AQM surveyed approximately 130 facilities for the 2002 reporting year. The following criteria were used to establish the universe of facilities surveyed:

- Title V permitted facilities (EPA-defined major sources),
- Any facility with emissions of VOCs greater than 5 tons per year (TPY) for any of the years 1999, 2000, or 2001, as previously reported to the AQM inventory program,
- Any facility within one of the following industry sectors: hot-mix asphalt plants, hospitals that use ethylene oxide for sterilization, electric generating units (EGUs), and facilities using anhydrous ammonia as a refrigerant,
- Any facility for which AQM does not have previous inventory data that appears may be a significant source.

These criteria were chosen for reasons of gathering data for criteria pollutants and air toxics. For instance, ammonia is not a DATAS pollutant but rather a precursor to the secondary formation of fine particulate matter.

Current permit/compliance data existed in AQM files for each facility within the chrome plating sector such that these facilities were not surveyed, but nonetheless included in the point

source inventory. Also, seven facilities reporting to the 2002 Toxics Release Inventory (TRI), which did not meet any of the criteria above, were included in the point source inventory based on their reported TRI air emissions of DATAS pollutants.

Since facilities rarely possess emissions data of air toxics from combustion processes, AQM decided to internally estimate emissions from these units for all facilities based on standard unit-specific emission factors applied to the fuel throughput of the unit. This approach allowed for a more complete and consistent method for developing emissions. For a few facilities, TRI or other facility data were used when these data were determined to be more representative of emissions than the standard emission factors. As an example, mercury emissions from several larger EGUs were obtained in this way. For non-combustion processes, facilities were required to report all air toxic emissions.

The reports submitted by facilities were reviewed for completeness and accuracy. AQM contacted facility representatives when problems were identified that could not be resolved internally. The administrative and technical review of the data reported by facilities was extensive. Details of the review process are described in the point source IPP [21].

Spatial allocation of emissions was an important consideration for the 2003 inventory, as previously discussed. For point sources, coordinates were verified and corrected through the use of high-resolution aerial photographs. In addition, coordinates for many stacks within larger facilities were obtained from the use of the aerial photography.

With a completed and quality-assured 2002 toxics inventory, the 2003 inventory was then developed from the 2002 inventory. EPA's Economic Growth Analysis System (EGAS) [22] was used to project 2002 emissions to 2003. Several facilities that reported for 2002 ceased operation either in 2002 or 2003. Emissions for 2003 for these facilities were accounted for accordingly.

Hospitals using ethylene oxide for sterilization were surveyed to obtain their 2003 usage of ethylene oxide. Therefore, 2002 emissions for these facilities were not grown to 2003. Also, 2003 operating schedules for the four chrome-plating operations were obtained to calculate emissions directly for 2003.

3.3.2 Stationary area sources

AQM estimated air toxics emissions for 35 area source categories. Some area source categories do not have emissions of air toxics, such as the fugitive dust categories (e.g., road construction, agricultural production) and bakeries (which emit ethanol, a VOC, but not an air toxic.)

The basic equation that applies to emissions development for most area sources is as follows:

$$\text{Emissions (E)} = \text{Activity Data (Q)} \times \text{Emission Factor (EF)}$$

Emission factors come in a variety of forms. One commonly used is the population-based factor. The use of consumer products that contain VOCs is a category that relies on a population-based emission factor. Another commonly used emission factor is one that is employee based. For source sectors where the emissions occur at small businesses too small and numerous to inventory as point sources, emission factors are related to the number of employees within the industry that engages in the activity that is responsible for the emissions. For combustion sources, emissions are based on fuel-specific emission factors.

An alternative to the use of emission factors, the mass balance approach was used for dry cleaning establishments. Perchloroethylene purchases for 2002 and 2003 were used to quantify emissions, such that emissions were equated to usage minus amounts leaving the site in waste (soaked filter cartridges and condenser sludge.)

Activity data is the other important building block for estimating emissions for area sources. Activity data must relate to the type of emission factor used, and if possible, be obtained from local sources. Delaware population data were obtained from the Delaware Population Consortium. Employment data were obtained from the Delaware Department of Labor. Fuel consumption data were obtained from the U.S. Department of Energy.

If a source category has a regulatory control placed on it from the Federal or State level, the equation expands to the following:

$$E = Q \times EF \times [1 - (CE)(RE)(RP)]$$

CE = control efficiency

RE = rule effectiveness

RP = rule penetration

Control efficiency represents the typical emissions reduction achieved as compared to otherwise uncontrolled emissions. A control may be a piece of equipment, such as condensers used by dry cleaners to capture perchloroethylene, or it may be an operational control, such as the use of only low VOC-content paints.

Rule effectiveness represents how well the particular rule is being met by the regulated community. If a rule is not being followed by the regulated community, then the amount of emissions will be higher than would otherwise be if there was 100% compliance.

Rule penetration represents the percent of sources within a source category that are subject to the rule that requires control. As an example of rule penetration, gas stations that dispense more than 10,000 gallons of gas in a month are required to place vapor recovery nozzles on their gas pumps. Those dispensing less than 10,000 gallons are not required to install controls. Therefore, RP is less than 100%. In the case of the burning of trash or leaves, no person or business is exempt, and thus RP is 100%.

Following extensive quality control checks of the 2002 emissions, attention was turned to developing the 2003 emissions. As with the point sources, growth factors were obtained from

EGAS and were used for most area source categories. Any changes to existing regulations, or addition of new regulations, that took effect for 2003 were evaluated to determine the effect on CE, RE, and/or RP. Adjustments to the grown emissions were made accordingly. For several source categories, 2003 activity existed at the time the 2003 inventory was being projected from the 2002 inventory. These included dry cleaning, Dover Downs Speedway, instructional structure fires, prescribed burning, and wildfires. Emissions for 2003 were calculated directly using the 2003 activity data instead of growing the 2002 data.

3.3.3 On-road mobile sources

Estimating emissions for on-road mobile sources is accomplished using the same basic area source equation, namely, emissions equals the product of an emission factor and associated activity data. Activity data are represented by vehicle miles traveled (VMT). VMT is calculated for AQM by DelDOT using traffic count data obtained from numerous permanent monitoring sites.

Emission factors for toxic pollutants from on-road mobile sources were calculated using EPA's MOBILE6.2 model. Model input files were created that include Delaware-specific fuels data, auto inspection program information, vehicle registration data, and monthly average maximum and minimum temperatures. The MOBILE6.2 model accounts for controls that exist in each vehicle type and model year. The model also accounts for deterioration of these controls. The model generates emission factors for exhaust, evaporative, brake wear, and tire wear. The model was run for each month of the year to generate emission factors specific to each month. These are combined with the monthly VMT derived from the DelDOT data.

Annual VMT estimates by county and road type for 2003 were provided by DelDOT, therefore emissions from 2002 were not grown to 2003. The MOBILE6.2 model was used to generate monthly 2003 emission factors based on input files developed for 2003.

3.3.4 Off-Road mobile sources

Off-road mobile sources are grouped into four categories, including aircraft, locomotives, commercial marine vessels, and a fourth category that includes all other off-road vehicles and equipment. The methodologies for developing aircraft, locomotives, and commercial marine vessels emissions are similar in structure to those applied to the stationary area source categories.

Emissions from all other off-road equipment were calculated through the use of EPA's NONROAD model. To estimate emissions, the NONROAD model multiplies equipment populations and their associated activity by the appropriate emission factors. Appropriate fuel parameters and seasonal temperatures specific to Delaware were input into the model. The NONROAD model was used to estimate VOC and PM emissions. Speciation profiles were then employed to arrive at individual toxic chemical emissions.

Similar to the on-road mobile sources category, the NONROAD model was run for both 2002 and 2003, with input files developed for each run.

Section 4

Human Health Risk Assessment of Monitored Data

4.1 Risk assessment

A risk assessment is an analysis that uses information about toxic substances to estimate a theoretical level of risk for people who might be exposed to these substances. The information comes from scientific studies and environmental data. Risk assessments are conducted for a number of reasons, including: to establish whether an environmental health risk exists or not; to identify the need for additional data collection; to focus on the dangers of a specific pollutant or the risks posed by a specific site; to help develop contingency plans and other responses to pollutant releases. Risk assessments, prepared by EPA and other agencies, are used to determine if levels of toxic substances pose an unacceptable risk as defined by regulatory standards and requirements. The risk assessment helps regulatory officials determine strategies (risk management steps) that will ensure overall protection of human health and the environment.

The risk assessment/risk management paradigm (Figure 4.1) includes Hazard Identification, Exposure Assessment, Toxicity Assessment, Risk Characterization and Risk Management. Hazard Identification involves the determination and sampling of what chemicals may be at the site. Exposure Assessment involves any of the ways for human contact with the contaminants including who are the likely receptors of the exposure, and how often this contact has or will occur. Toxicity Assessment relates to the levels of the contaminants of concern at the site and what effects they are likely to have on human health. Risk Characterization pulls all the above information together as well as the results of the cancer and non-cancer risk modeling estimates. Risk Management provides options and recommendations regarding what to do about these contaminants ranging from no action to removal to using barriers to prevent exposure.

It is important to note that a risk assessment does not measure the actual health effects that hazardous substances have on people. Also people will not necessarily become sick even if they are exposed to compounds at higher dose levels than those estimated by the risk assessment. In other words, during the risk assessment analysis, the most vulnerable people/receptors (e.g., children and the elderly) are carefully considered to make sure all members of the public will be protected. Also, conservative safety margins are built into a risk assessment analysis to ensure protection of the public.

For cancer effects as an endpoint, risks are expressed as probabilities of additional cases of cancer above the expected background level. For non-cancer effects, exposure levels are compared to pre-established levels at which negative (adverse) health effects are not expected. The risk assessment provides an estimate of theoretical risk or hazard,

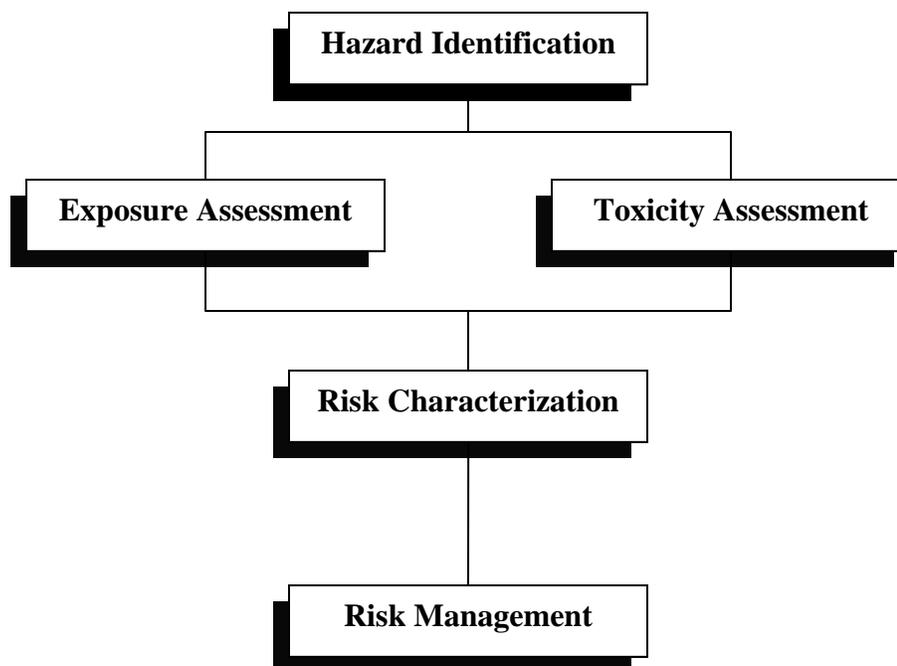


Figure 4.1: Risk Assessment – Risk Management Paradigm

assuming no changes in exposure takes place over the time of the modeling. It focuses on current and potential future exposures. It predicts into the future rather than being retrospective. The quantitative risk estimates are not intended, however, to predict the incidence of disease. For example, a risk assessment may reveal a "one in a hundred thousand chance" of cancer because of exposure to air contaminants but these predictions do not absolutely mean that an actual cancer case will exist. These predictions are based on statistical and biological models that include a number of protective assumptions about exposure and toxicity. By design, they are conservative predictions that generally overestimate health risk (worst case tendency).

Those performing risk assessments seek to determine an acceptable (negligible) level for each potentially dangerous contaminant present. For humans, this is a level at which adverse health effects are unlikely and the probability of cancer is very small [23].

4.2 Risk assessment calculations and modeling

Risk assessments were performed following EPA established calculations [24]. The methods used were initially established for Superfund investigations and have since been applied to many non-Superfund studies. The DATAS risk assessment methodology is described below.

Risk assessment was undertaken to estimate potential health risks posed to Delawareans by contaminants present in air. Calculations were performed to determine the relative risk of experiencing negative (adverse) health effects from exposure to various pollutants for the

population of Delaware. Health risk was estimated for the monitoring data collected at the five monitoring stations located throughout Delaware. Risk of cancer and non-cancer effects from exposure at detected levels were calculated for adults, children and a combination of child and adult exposure.

The risk assessment consisted of a two-component calculation model to estimate the risk of the population to the concentration of air pollutants measured at the five monitoring locations in Delaware. The monitored data was analyzed and quality assurance/quality control was performed by the AQM. The AQM provided the Environmental Health Evaluation Branch (EHEB) with annual mean concentrations, 95% upper confidence limits and other statistical factors that were applied in the model. The two-part calculation began with an estimate of the dose of the chemical, relative to body weight and exposure time. The dose was then applied to the appropriate published factors to determine approximate risk. The methodology is described in more detail below. Calculations and default values are shown in Table 4.1.

4.2.1 Risk of cancer

Risk of developing cancer due to exposure to the compounds included in this study was estimated using inhalation slope factors found on the EPA Region III Risk Based Concentration (RBC) Table, October 2004 edition and are based primarily on data from EPA's Integrated Risk Information System Database (IRIS) [25]. The target level of risk for DATAS is based on Delaware regulations used for hazardous waste sites (Hazardous Substances Cleanup Act, HSCA) and differs from the EPA target level of one additional cancer case per one million people. Cancer risk was considered negligible if equal to or less than one additional cancer case per 100,000 people was expected as per HSCA. Compounds with negligible estimated risks are displayed in green in the risk tables. Cancer risk was considered increased if greater than one but less than 10 additional cancer cases per 100,000 people was predicted. Compounds with increased potential cancer risks are displayed in yellow in the risk tables. Cancer risk was considered high if the number of additional expected cancer cases was greater than 10 per 100,000 people. Chemicals with a high relative cancer risk are displayed in red in the risk tables. This risk in the red also would be the level at which the EPA would take action based on their action level of 1 additional cancer case in 10,000 exposed population.

4.2.2 Risk of non-cancer effects

Risk of developing non-cancer effects was estimated using reference doses from the EPA Region III RBC Table, October 2004 edition and IRIS [26]. Non-cancer effects are possible adverse health conditions other than cancer that develop from chronic exposure. Non-cancer risk was evaluated based on a hazard quotient numerical value. Hazard quotients are used to evaluate the non-cancer health effects of chemicals, which are not carcinogens but can have adverse, systemic health effects. Hazard quotients are the ratio of potential exposure to a particular compound to the level of exposure at which no hazardous/ adverse health effects are expected. Put simply, the calculated exposure dose is divided by the published reference dose with the result being a probability of developing an adverse effect due to exposure to the chemical. As part of this study hazard quotients of 1 or less than 1 are considered negligible or acceptable levels or without adverse health effects (displayed in green). Compounds with a hazard quotient

Table 4.1: Formulas and Default Values for DATAS Risk Assessment

Adult and Child Default Dose Calculation (ADD)

$$\text{Intake (mg/kg/day)} = \text{CA} \times \text{CF} \times \text{IR} \times \text{ET} \times \text{EF} \times \text{ED/BW} \times \text{AT}$$

Age-Adjusted Default Dose Calculation (ADD)

$$\text{Intake (mg/kg/day)} = (\text{CA} \times \text{ET} \times \text{EF/AT}) \times \text{AAF}^*$$

Cancer Estimated Risk Calculation (CR)

$$\text{Estimated Risk} = \text{ADD} \times \text{CSFi}$$

Non-Cancer Estimated Risk Calculation (HQ)

$$\text{Estimated Risk} = \text{ADD/RFDi}$$

Definitions:

- ADD Average Daily Dose
- CSFi Cancer Slope Factor (Inhalation)
- RFDi Reference Dose (Inhalation)
- CA Chemical Concentration
- CR Cancer Risk
- HQ Hazard Quotient
- AAF Age-Adjusted Factor

	Adult	Child	Age-Adjusted
Term	Value	Value	Value
IR = INHALATION RATE (m ³ /day)	20	10	AAF*
ET = EXPOSURE TIME** (hr/day)	1	1	1
CF = CONVERSION FACTOR	1.00E-06	1.00E-06	1.00E-06
EF = EXPOSURE FREQUENCY (day)	350	350	350
ED = EXPOSURE DURATION (years)	30	6	AAF*
BW = BODY WEIGHT (kg)	70	16	AAF*
AT(NC) = AVERAGING TIME - NON-CARCINOGENS (days)	10950	2190	10950
AT(C) = AVERAGING TIME – CARCINOGENS (days)	25550	25550	25550

*Age adjusted factor is partial calculation which combines IR, ED and BW for 30-yr childhood to adult exposure.

**The exposure time factor included in the equation and table is part of the standard inhalation dose assessment formula as prepared by EPA. The purpose of this factor is for risk assessments that consider exposures of less than twenty-four hours per day (i.e. worker’s exposure scenario). As the risk assessment performed as part of DATAS does not estimate dose for an exposure of less than twenty-four hours per day, this factor was represented in the calculation by a value of one. Using a value of one in the equation mathematically removes the factor from the resulting value. The resulting value then represents an estimate of dose from a twenty-four hour exposure. As the calculation is for a twenty-four hour dose it should also be noted that the units for the inhalation rate are m³/day, as opposed to m³/hour which would be used for a less than twenty-four hour dose [27].

of between 1 and 10 are considered to be at an increased level of adverse effects (displayed in yellow). Compounds with a hazard quotient of greater than 10 are considered at a high level (displayed in red).

This classification system is used as a shorthand notation in research and studies involving hazardous materials.

4.3 Health impacts

Cancer risk and non-cancer hazard quotients were estimated for three distinct age receptors (scenarios) as part of this risk assessment. Risk estimates were calculated separately for adult exposure, child exposure and age-adjusted exposure. The adult exposure scenario is used to calculate risk for a member of the study population who has lived in the study area as an adult. The calculation includes default assumptions for physical factors, such as body weight and inhalation rate. The calculation also makes assumptions, based on EPA research and published documents, regarding life span and time spent as a resident of the study area [26].

The child exposure scenario is calculated with the same formula as the adult exposure. The difference in the two is in the factors included in the equation. Factors for body weight and inhalation rate are lower and the residence time is limited to six years. Default assumptions used in the child scenario show a higher intake of pollutants per kilogram of body weight than for the adult or age-adjusted calculations. This increased intake causes the child scenario to be, generally the most susceptible to non-cancer effects of chemical exposure.

The age-adjusted exposure is a combination of childhood and adult exposure. The age-adjusted calculation represents a member of the study population born in the study area who has continued to live in the area into adulthood. The inclusion of the childhood exposure and continued exposure into adulthood makes this scenario, generally the most susceptible population to effects of exposure to carcinogens. The default assumptions for this calculation include a factor that represents a partial calculation that incorporates separate inhalation rates, exposure durations and body weights, for children and adults into one factor. The default assumptions for various parameters for each set of estimates are included in Table 4.1. Default values used as part of this risk estimate are standard values used by EPA Region III [26].

Once the individual estimate of potential negative/adverse health impacts for each carcinogen were developed, the estimates were summed to develop a cumulative cancer risk for each of the five monitoring sites. A cumulative cancer risk was developed for each of the three receptors (adult, child, child/adult combination). A cumulative non-cancer health impact was also assessed for each monitoring site. The direct addition of the potential risk of the individual compounds does not account for different mechanisms of action and target organs for the various compounds. This method does, however, present a conservative picture of the potential exposure to all compounds.

Deviations from the standard calculations and default values were necessary in several circumstances. Risk estimates for compounds on the RBC Table or from IRIS [25] with oral values, but without inhalation values were calculated using the standard formulas. Where oral

values were used in place of inhalation values, the oral value was used as a direct replacement in the final calculation. The replacement of inhalation values with oral values is not a precise estimation of the risk due to the differences in absorption and other intake factors. However, in the absence of inhalation values, this approach is considered a conservative means of providing an estimated risk value.

Chromium was reported in the analytical results as “Total Chromium” rather than being speciated into hexavalent and trivalent chemical forms. The risk assessment was performed with the assumption that 35% of the total measured concentration is hexavalent Chromium. Inhalation factors for trivalent chromium are not available on the October 2004 RBC Table or from IRIS [25]. Oral factors are present for trivalent Chromium, however risk was not calculated for trivalent chromium given the magnitude of potential risk is several orders of magnitude lower than that of hexavalent Chromium.

The 35% is based on published EPA values and presents a conservative estimate of the proportion of hexavalent chromium to trivalent chromium. DNREC has stated that their Toxic Release Inventory indicates that approximately 9% of the total chromium present in the environment would be present as the hexavalent form. The inventory is assumed to incorporate in state locations and most likely presents an accurate portrait of those sources. However, the question must be raised of how the influences of urban and industrial areas outside of the state would affect the 9% assumption. In addition, comparison of the calculations of risk using the 35% and the 9% assumptions show that while potential risk from chromium exposure is obviously reduced using 9%, the effect on the monitoring station totals is minimal. Based on this it seems that 35% is a conservative estimate that can be easily defended as it is an established EPA value, while also being more protective of public health.

Risk estimates for dioxin/furan and polycyclic aromatic hydrocarbon (PAH) compounds monitored in this study were performed using toxic equivalency factors (TEF). Dioxin/furan TEFs used in this risk assessment were published by the World Health Organization in 1998 [28]. TEF's used for PAH risk analysis are those published by the EPA [29]. Dioxin/furan compounds were assessed based on their relative toxicity to 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin. PAH compounds were assessed based on their relative toxicity to benzo(a)pyrene. TEF's for both dioxins/furans and PAHs were applied by multiplying the concentration of the individual compounds by their respective factors and then proceeding with the rest of the risk assessment process.

Risk assessment for exposure to vinyl chloride was performed using the method described in the EPA Region III Vinyl Chloride Memorandum [30]. The procedure differs from the standard calculation for the age-adjusted carcinogen scenario; other calculations are performed using the standard formulas. The age-adjusted formula is altered to include an additional safety factor when considering the exposure of children. The additional factor is included due to the increased risk to children.

Analytical results for xylenes were differentiated into results for “ortho” and “meta” and “para” isomers. The October 2004 RBC Table or IRIS does not differentiate and includes

inhalation values only for total xylenes. During the risk assessment the analytical results for the individual isomers was summed and the risk assessment performed on this total xylene result.

4.3.1 Human health effects and risk

The methods and calculations of risk assessment are based on toxicity research. The following gives a brief overview of this research and how it is used to formulate risk assessment calculations [31, 32].

The Integrated Risk Information System (IRIS, prepared and maintained by the EPA), is an electronic database containing information on human health effects that may result from exposure to various chemicals in the environment. IRIS was initially developed for EPA in response to a growing demand for consistent information on chemical substances for use in risk assessments, decision-making and regulatory activities. The information in IRIS is intended for those without extensive training in toxicology, but with some knowledge of health sciences. The heart of the IRIS system is its collection of computer files covering individual chemicals. These chemical files contain descriptive and quantitative information in the following categories:

- Hazard identification, oral reference doses and inhalation reference concentrations (RfDs and RfCs, respectively) for chronic non-carcinogenic health effects.
- Hazard identification, oral slope factors, and oral and inhalation unit risks for carcinogenic effects.

The information in IRIS is most useful if applied in the larger context of risk assessment. IRIS supports the first two steps of the risk assessment process; namely, the hazard identification and dose-response assessment steps. 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.

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 high 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.

Toxicity Assessment and Hazard Identification are the processes of determining whether exposure to a chemical can cause an increase in the incidence of a particular adverse health effect (e.g., cancer, birth defects) and whether the adverse health effect is likely to occur in humans. The process examines the available scientific data for a given chemical (or group of chemicals) and develops a weight of evidence to characterize the link between the negative effects and the chemical.

Dose-Response Assessment is the process of quantitatively evaluating the toxicity of a given chemical as a function of human exposure to that chemical. The relationship between the dose of the contaminant administered or received and the incidence of adverse health effects in the exposed population forms the basis for the quantitative dose-response relationship. From these relationships, toxicity values (e.g., reference doses and slope factors) are derived that can be used to estimate the incidence or potential for adverse effects in an exposed population.

4.3.2 Human toxicity assessment

The term "toxic" chemical is used frequently in the media and heard often in social settings. Other than suggesting something bad about the chemical, what exactly is meant by saying that a chemical is toxic? Does it mean that it will kill people? Cause cancer? Affect their children? Are there chemicals that can't cause any harm? To a toxicologist, this last question is easiest to answer. Everything is toxic: in sufficient quantities, every chemical has the capacity to cause adverse effects on human health. It is only the degree and type of toxicity that varies from chemical to chemical.

Dose-response studies are used to establish the relationship between the amount of chemical and the seriousness of the effect. The dose (the actual amount of chemical) is gradually increased and the response (the observed effects) at each dose is evaluated. For some chemicals, increasing the dose slightly leads to a large increase in effect (toxicity). These chemicals do not have a large margin of safety. On the other hand, the effects due to another chemical may increase only very slowly as the dose is increased.

Since toxicity refers to a number of possible adverse effects, there are several ways to deal with it. One way is to divide the effects into categories based on two fundamental characteristics of toxicity: how fast the effects occur and what type they are. Effects that happen only after repeated long-term exposure are known as chronic toxicity.

Chronic toxicity also may take many forms. The most well-known chronic effect is cancer. Others are organ damage such as cirrhosis of the liver from long-term alcohol consumption, reproductive effects such as decreased fertility, and effects on the nervous system. An example of a nervous system effect is mental retardation in children who were exposed to high levels of lead over a period of time in early childhood.

In light of the numerous uncertainties associated with toxicity studies, cancer assessments try to err on the side of safety. To accomplish this goal, calculations and conversions are done in such a way that the final toxicity value represents the highest possible risk of cancer from exposure to that chemical.

4.4 EPA's approach for assessing the risks

The EPA often approaches the investigation of a chemical with a particular route of exposure in mind (e.g., an oral exposure for a drinking water contaminant or an inhalation exposure for an air contaminant). In most cases, the toxicological database does not include detailed testing on all possible routes of administration, with their possibly significant

differences in factors such as mechanism-of-action and bioavailability. In general, the EPA's position is that the potential for toxicity manifested via one route of exposure is relevant to considerations of any other route of exposure, unless convincing evidence exists to the contrary. Consideration is given to potential differences in absorption or metabolism resulting from different routes of exposure, and whenever appropriate data (e.g., comparative metabolism studies) are available, the quantitative impacts of these differences on the risk assessment are delineated.

The EPA is concerned about the potential toxic effects in humans associated with all possible exposures to chemicals. The magnitude, frequency, and duration of exposure may vary considerably in different situations. Animal studies are conducted using a variety of exposure durations (e.g., acute, subchronic, and chronic) and schedules (e.g., single, intermittent, or continuous dosing). Information from all these studies is useful in the hazard identification phase of risk assessment. For example, overt neurological problems identified in high-dose acute studies tend to reinforce the observation of subtle neurological changes seen in low-dose chronic studies. Special attention is given to studies involving low-dose, chronic exposures, since such exposures can elicit effects absent in higher dose, shorter exposures, through mechanisms such as accumulation of toxicants in organisms.

4.4.1 Chronic exposure to non-cancer causing chemicals

Chemicals that give rise to toxic endpoints other than cancer and gene mutations are often referred to as "systemic toxicants" because of their effects on the function of various organ systems. Non-cancer effects are adverse health conditions other than cancer that develop from chronic exposure. In addition, chemicals that cause cancer and gene mutations also commonly evoke other toxic effects (i.e., systemic toxicity). Based on our understanding of homeostatic and adaptive mechanisms, systemic toxicity is treated as if there is an identifiable exposure threshold (both for the individual and for populations) below which there are no observable adverse effects. This characteristic distinguishes systemic endpoints from carcinogenic and mutagenic endpoints, which are often treated as non-threshold processes.

The EPA's approach to assessing the risks associated with systemic toxicity (non-cancer) is different from its approach to assessing the risks associated with carcinogenicity, because of the different mechanisms of action thought to be involved in the two cases. In the case of carcinogens, the EPA assumes that a small number of molecular events can evoke changes in a single cell that can lead to uncontrolled cellular proliferation. This mechanism for carcinogenesis is referred to as "non-threshold," since there is theoretically no level of exposure for such a chemical that does not pose a small, but finite, probability of generating a carcinogenic response. In the case of systemic toxicity, however, organic homeostatic, compensating, and adaptive mechanisms exist that must be overcome before a toxic endpoint is manifested. For example, there could be a large number of cells performing the same or similar function whose population must be significantly depleted before the effect is seen.

The threshold concept is important in the regulatory context. The individual threshold hypothesis holds that a range of exposures from zero to some finite value can be tolerated by the organism with essentially no chance of expression of the toxic effect. Further, it is often prudent

to focus on the most sensitive members of the population; therefore, regulatory efforts are generally made to keep exposures below the population threshold, which is defined as the lowest of the thresholds of the individuals within a population.

4.4.2 Chronic exposure to carcinogens

The purpose of the hazard identification and toxicity assessment steps are to determine whether the agent in question poses a carcinogenic hazard in exposed humans. The major types of evidence bearing on this question are these: (1) human studies of the association between cancer incidence and exposure to the agent; and (2) long-term animal studies under controlled laboratory conditions. Also considered is supporting evidence such as short-term tests for genotoxicity, metabolic and pharmacokinetic properties, toxicological effects other than cancer, structure-activity relationships, and physical/chemical properties of the agent.

The chemical's potential for human carcinogenicity is inferred from the available information relevant to the potential carcinogenicity of the chemical and from judgments as to the quality of the available studies. A weight-of-evidence approach is used by the EPA to classify the likelihood the agent in question is a human carcinogen. A three-stage procedure is followed. In the first stage, the evidence is characterized separately for human studies and for animal studies. Secondly, the human and animal evidence are combined into a presumptive overall classification. In the third stage, the provisional classification is adjusted upwards or downwards, based on analysis of the supporting evidence. The result is that each chemical is placed into one of the following five categories:

<u>Group</u>	<u>Category</u>
A	Human carcinogen (known to cause cancer in humans)
B	Probable Human carcinogen B1 limited human evidence B2 indicates sufficient evidence in animals and inadequate or no evidence in humans
C	Possible human carcinogen (some evidence from animals)
D	Not classifiable as to human carcinogenicity (insufficient evidence available)
E	Evidence of non-carcinogenicity for humans

The above classification system is used extensively as a shorthand notation in research and studies involving hazardous materials.

Section 5

Results and Discussion

This section provides summary results of the DATAS project to date, including: (1) the 2003 measured concentrations observed at the five monitoring locations, (2) the 2003 statewide air toxics inventory, and (3) a risk assessment from human exposure to these individual pollutant concentrations and their cumulative effects.

Recognizing that the modeling results will provide a much greater spatial resolution of ambient concentrations of the DATAS chemicals, the initial analysis of the monitoring data focused on the following activities.

- Calculation of descriptive statistics for each of the DATAS chemicals,
- Performance of a site-to-site comparison for select chemicals,
- Preliminary interpretation of seasonal variability in the data, and
- Evaluation of the corresponding risk assessment.

The initial interpretation of the 2003 emission inventory included a breakdown of the emissions for the targeted DATAS chemical groups by source sector, including point, area, on-road mobile, and off-road sources.

Results for the monitoring data interpretation, inventory breakdown, and risk assessment of the monitoring data are presented in subsections 5.1 through 5.3, respectively.

5.1 Monitoring results

In presenting the analysis results for the 2003 monitoring dataset, the DATAS chemicals were organized by pollutant group (VOC, metals, carbonyls, PAH, dioxins/furans) and monitoring location. Data summary tables for each compound at each monitoring site are presented in the Appendix (Tables B1 through B5).

5.1.1 VOC

The annual average VOC concentrations are summarized in Table 5.1, by chemical compound and monitoring site, and plotted in Figure 5.1(a, b, and c). The minimum detection limits (MDL) are also presented for each compound. Additional summary results can be found in Table B1-1 through B1-5 (Appendix).

Comparing these averages, the following observations can be made:

- Average annual concentrations for a majority of VOC compounds fall below the minimum detection limits (MDL),

- The urban MLK site generally has the highest concentrations of many compounds, which is consistent with urban sites in general. Urban areas typically have multiple sources of pollution, including motor vehicles, concentrated in a small area,
- Concentrations of a few compounds (e.g. dichlorodifluoromethane, trichlorofluoromethane, chloromethane, carbon tetrachloride, 1,1,2,2-tetrachloroethane, and hexachloro-1,3-butadiene) show little variation between sites. These concentrations may reflect regional levels of pollution,
- Compounds like toluene, xylenes, 1,3-butadiene, 1,4-dichlorobenzene, and 1-ethyl-4-methylbenzene show very low concentrations at the rural location (KP), while they are higher near an industrial complex (DC) and/or the urban site (MLK). The suburban or small urban sites (LP and SE) show intermediate concentrations,
- Concentrations of chlorobenzene, chloroethene, and to some extent toluene and dichlorodifluoromethane are the highest at DC, which is probably the result of a local source or sources. The nearby industrial complex is a known source of these compounds, although further study is needed to verify the relationship between the sources and the monitored concentrations,
- Concentrations of compounds produced as a result of motor fuel burning, i.e., ethylbenzene, 1,3-butadiene, benzene, toluene, and 1,2,4-trimethylbenzene are the highest at MLK. This is probably due to the site's proximity to major highways, although further study is needed, and
- Bromomethane shows the highest concentration at SE. The reason for this is not readily apparent.

Table 5.1: VOC annual average concentrations ($\mu\text{g}/\text{m}^3$) at five Delaware sites

Compound	MDL	MLK	DC	LP	KP	SE
Dichlorodifluoromethane	0.077	2.702	2.577	2.621	2.583	2.604
Trichlorofluoromethane	0.133	1.493	1.378	1.394	1.387	1.386
Chloromethane	0.083	1.117	1.151	1.118	1.132	1.138
Toluene	0.083	4.066	2.437	0.844	0.609	1.041
1,1,2-Trichloro-1,2,2-trifluoroethene	0.168	0.625	0.610	0.617	0.610	0.610
Benzene	0.035	1.375	1.055	0.590	0.465	0.566
Carbon tetrachloride	0.296	0.541	0.536	0.542	0.560	0.561
M & p- Xylene	0.143	2.250	0.708	0.334	0.206	0.376
Methylene Chloride	0.079	0.644	0.264	0.242	0.227	0.232
1,2,4-Trimethylbenzene	0.076	0.784	0.315	0.172	0.124	0.218
1,1,1-Trichloroethane	0.170	0.172	0.152	0.150	0.146	0.173
Ethylbenzene	0.048	0.660	0.266	0.137	0.098	0.150
Perchloroethylene	0.211	0.534	0.146	0.137	0.108	0.176
o-Xylene	0.067	0.636	0.272	0.131	0.087	0.155
1,2-Dichloro-1,1,2,2,tetrachloroethane	0.511	0.135	0.137	0.128	0.123	0.120
Hexachloro-1,3-Butadiene	0.331	0.107	0.115	0.107	0.107	0.107
Styrene	0.138	0.170	0.106	0.101	0.086	0.107
1,4-Dichlorobenzene	0.066	0.223	0.161	0.092	0.077	0.089
Chloroform	0.128	0.132	0.096	0.087	0.081	0.088
1-Ethyl-4-Methylbenzene	0.099	0.255	0.132	0.081	0.064	0.088
1,2,4-Trichlorobenzene	0.240	0.082	0.102	0.074	0.088	0.077
1,3-Butadiene	0.117	0.301	0.096	0.072	0.045	0.070
1,2-Dichlorobenzene	0.132	0.072	0.096	0.070	0.063	0.064
1,3,5-Trimethylbenzene	0.129	0.237	0.112	0.070	0.056	0.079
1,3-dichlorobenzene	0.066	0.065	0.075	0.065	0.062	0.061
Bromomethane	0.126	0.089	0.054	0.064	0.049	0.237
Trichloroethene	0.123	0.110	0.091	0.058	0.097	0.073
Chlorobenzene	0.127	0.051	0.098	0.050	0.046	0.046
Ethylene Dichloride	0.063	0.045	0.050	0.041	0.042	0.041
Ethyl chloride	0.152	0.047	0.037	0.035	0.035	0.032
Vinyl chloride	0.067	0.055	0.227	0.020	0.050	0.050
Cis-1,2-Dichloroethene	0.140	0.010		0.010		
1,2-Dibromoethane	0.169	0.010		0.010	0.010	
1,1,2,2-Tetrachloroethane	0.151	0.010	0.010	0.010	0.010	0.010
1,1-Dichloroethene	0.190	0.006	0.000	0.000	0.000	0.010
1,1-Dichloroethane	0.089	0.020	0.020			
1,2-Dichloropropane	0.128	0.010				
Cis-1,3-Dichloro-1-Propene	0.091					
Trans-1,3-Dichloro-1-Propene	0.147	0.010				
1,1,2-Trichloroethane	0.202					

Key: Colors relate to the percentage of non-detects or 0 samples. Black = no substitutions (no 0 or non-detects); Teal= lowest reported value or 1/2 MDL substituted for 0 (where 0%-50% of samples are non-detects or 0). Percentiles are used where 50%-90% of samples are non-detects or 0 as follows: Blue= 75th percentile Sky Blue= 80th percentile Violet= 90th percentile. No mean is generated if more than 90% of samples are non-detects or 0.

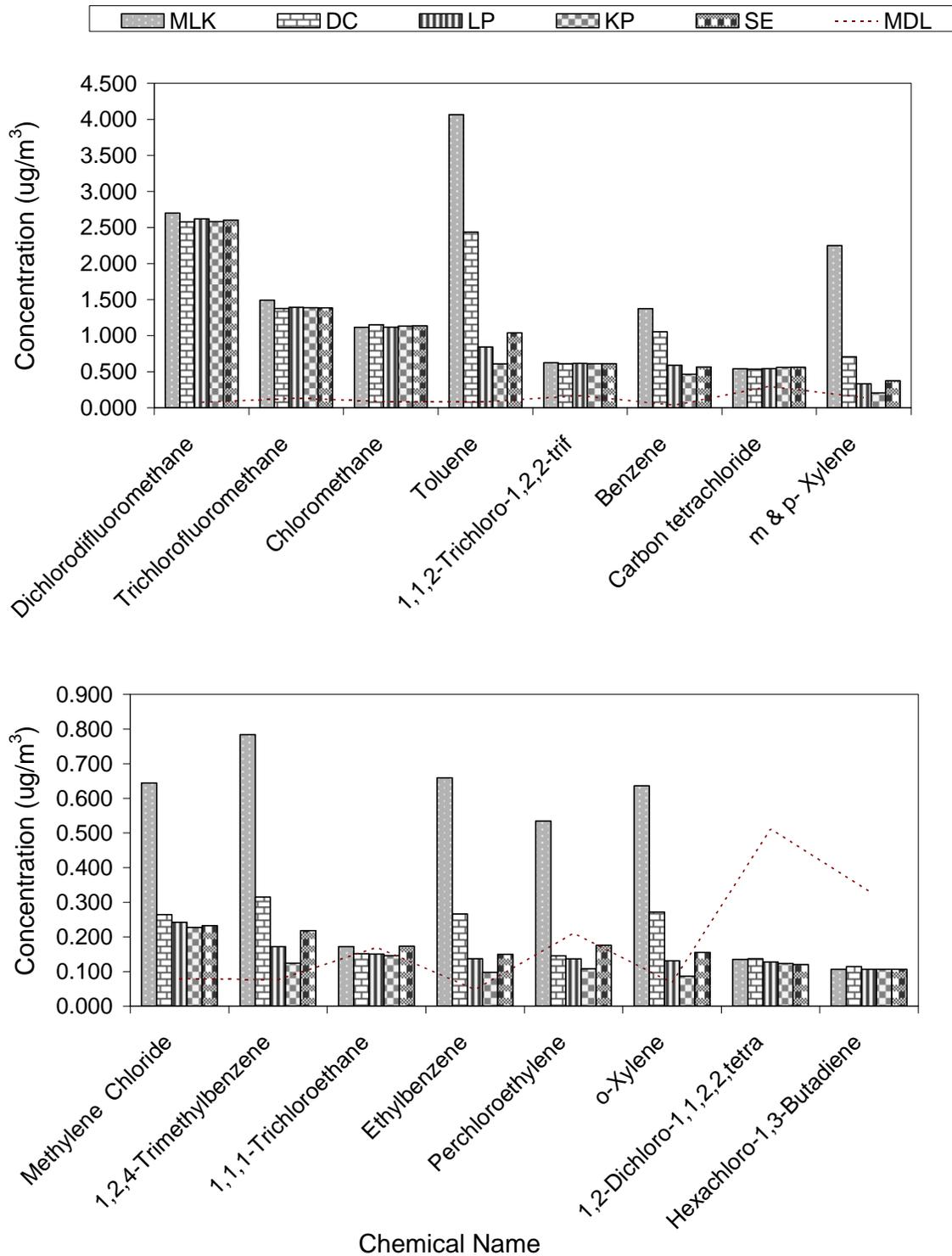


Figure 5.1(a): Annual average VOC concentrations in the air at five Delaware Sites shown in Table 2.1. Dotted line represents MDLs for the shown compounds. These are independent of each other but are connected via dotted lines on the plots only to make them noticeable.

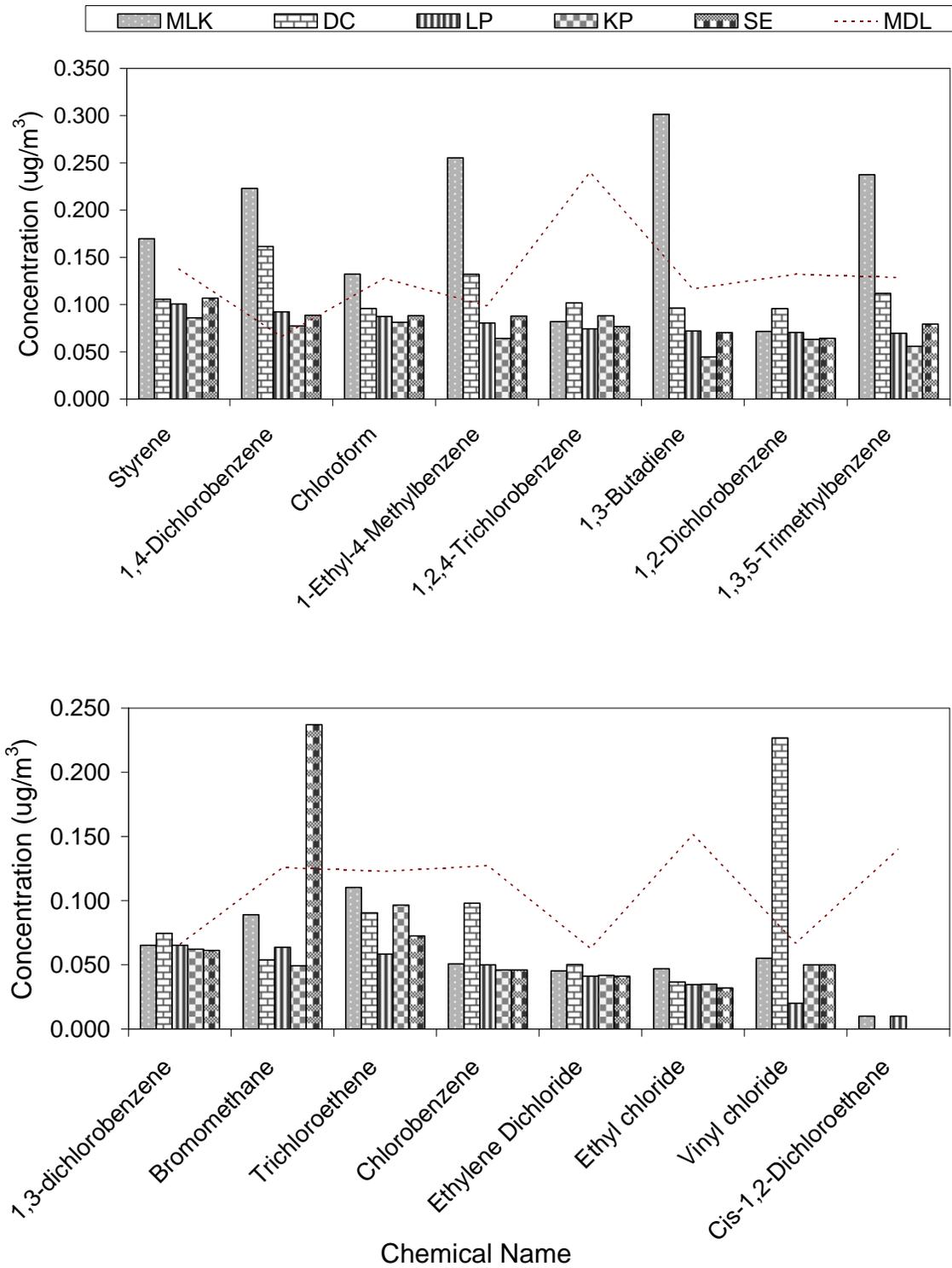


Figure 5.1(b): Annual average VOC concentrations in the air at five Delaware Sites shown in Table 2.1. Dotted line represents MDLs for the shown compounds. These are independent of each other but are connected via dotted lines on the plots only to make them noticeable.

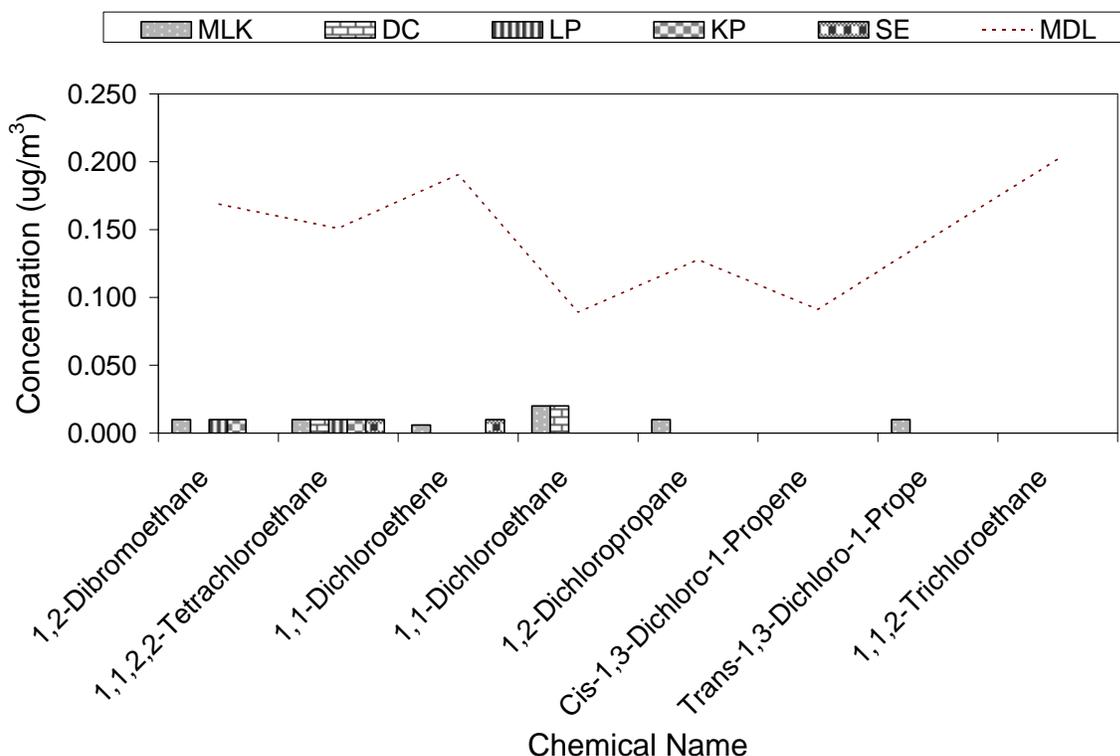


Figure 5.1(c): Annual average VOC concentrations in the air at five Delaware Sites shown in Table 2.1. Dotted line represents MDLs for the shown compounds. These are independent of each other but are connected via dotted lines on the plots only to make them noticeable.

5.1.2 Metals

Annual average metal concentrations are summarized by compound and monitoring site in Table 5.2, and plotted in Figure 5.2. The average method detection limits are also shown for each compound. Additional summary results can be found in Table B2 (Appendix).

Preliminary assessment of the metals data follows.

- Annual average concentrations for all metals, except beryllium are above the minimum detection limit,
- Arsenic and cadmium concentrations appear to be very similar throughout the state indicating that these may be regional pollutants,
- Beryllium concentrations are extremely low throughout the state,

- Chromium, manganese, nickel and lead are lowest at the rural and semi-rural sites (KP and SE, respectively). Chromium concentrations at LP and DC are also comparable to levels at KP and SE,
- Chromium, manganese, nickel and lead concentrations are higher at MLK and DC. Local sources of these metals may be responsible for the higher concentrations, but further analyses are required, and
- Rural and small urban sites (KP and SE) show the lowest concentrations of all metals while the urban site (MLK) shows the highest.

Table 5.2: Metal concentrations ($\mu\text{g}/\text{m}^3$) at five Delaware sites

Metal	AvgMDL	MLK	DC	LP	KP	SE
Arsenic	0.000036	0.001118	0.001021	0.000906	0.000884	0.000884
Beryllium	0.000002	0.000017	0.000011	0.000009	0.000010	0.000008
Cadmium	0.000024	0.000285	0.000236	0.000197	0.000198	0.000205
Chromium	0.000049	0.003809	0.002252	0.002133	0.001770	0.001672
Manganese	0.000049	0.018846	0.006909	0.006561	0.004942	0.004569
Nickel	0.000073	0.006940	0.004583	0.002982	0.002068	0.002309
Lead	0.000073	0.009837	0.005192	0.003951	0.003392	0.003378

Key: Colors relate to the percentage of non-detects or 0 samples. Black = no substitutions (no 0 or non-detects); Teal= lowest reported value or 1/2 MDL substituted for 0 (where 0%-50% of samples are non-detects or 0). Percentiles are used where 50%-90% of samples are non-detects or 0 as follows: Blue= 75th percentile Sky Blue= 80th percentile Violet= 90th percentile. No mean is generated if more than 90% of samples are non-detects or 0.

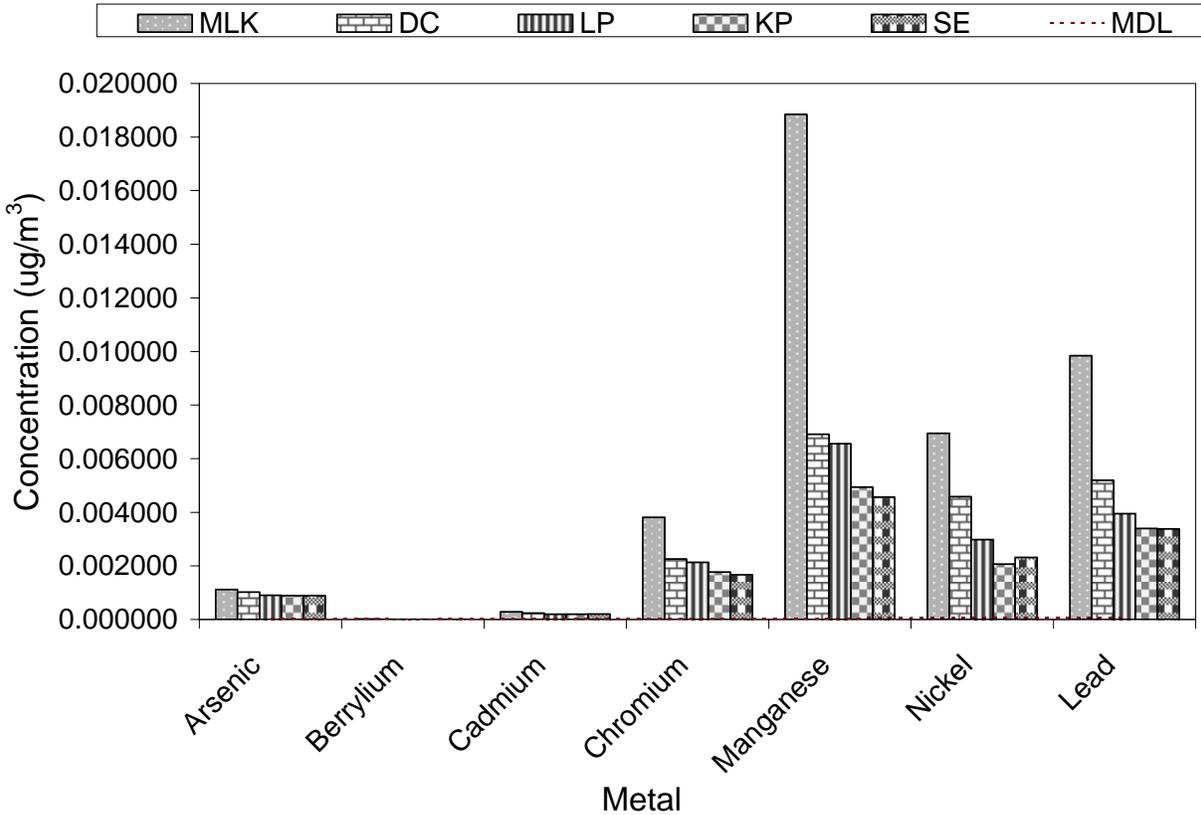


Figure 5.2: Annual average metal concentrations in the air at five Delaware Sites shown in Table 2.1

5.1.3 Carbonyls

The annual average concentrations for all carbonyls are summarized in Table 5.3 and plotted in Figure 5.3. Average method detection limits are also shown for each compound. Additional summary results can be found in Table B3 (Appendix).

Note on calculation of sample results: Carbonyl monitoring data differ somewhat from the other data in this study. Because there are known problems with contaminant interferences with the sampling method used, field blanks were collected with each sample. The laboratory results for the blanks were subtracted from the associated sample results to adjust the final concentration to account for any contamination. The sampling method [14] also includes collection of back-up cartridges. Final sample concentrations were calculated by adding the primary and back-up cartridge results and subtracting the field blank results.

Preliminary assessment of the carbonyl data follows.

- Annual average concentrations for all carbonyls, except methyl isobutyl ketone, were above the MDL,

- Formaldehyde, acetone, and acetaldehyde concentrations are highest at MLK and SE. The high concentration of formaldehyde at SE is a subject for future investigation,
- Methyl isobutyl ketone was detected only at MLK,
- The annual averages for propionaldehyde are comparable for all five monitoring locations.

Table 5.3: Carbonyl concentrations ($\mu\text{g}/\text{m}^3$) at five Delaware sites

Compound	Avg MDL	MLK	DC	LP	KP	SE
Acetaldehyde	0.0001	0.0018	0.0014	0.0014	0.0013	0.0016
Acetone	0.0002	0.0051	0.0039	0.0043	0.0038	0.0049
Formaldehyde	0.0002	0.0024	0.0013	0.0014	0.0012	0.0049
Methyl Ethyl Ketone	0.0002	0.0010	0.0007	0.0007	0.0007	0.0006
Methyl Isobutyl Ketone	0.0001	0.0001				
Propionaldehyde	0.0002	0.0004	0.0006	0.0006	0.0005	0.0003

Key: Colors relate to the percentage of non-detects or 0 samples. Black = no substitutions (no 0 or non-detects); Teal= lowest reported value or 1/2 MDL substituted for 0 (where 0%-50% of samples are non-detects or 0). Percentiles are used where 50%-90% of samples are non-detects or 0 as follows: Blue= 75th percentile Sky Blue= 80th percentile Violet= 90th percentile. No mean is generated if more than 90% of samples are non-detects or 0.

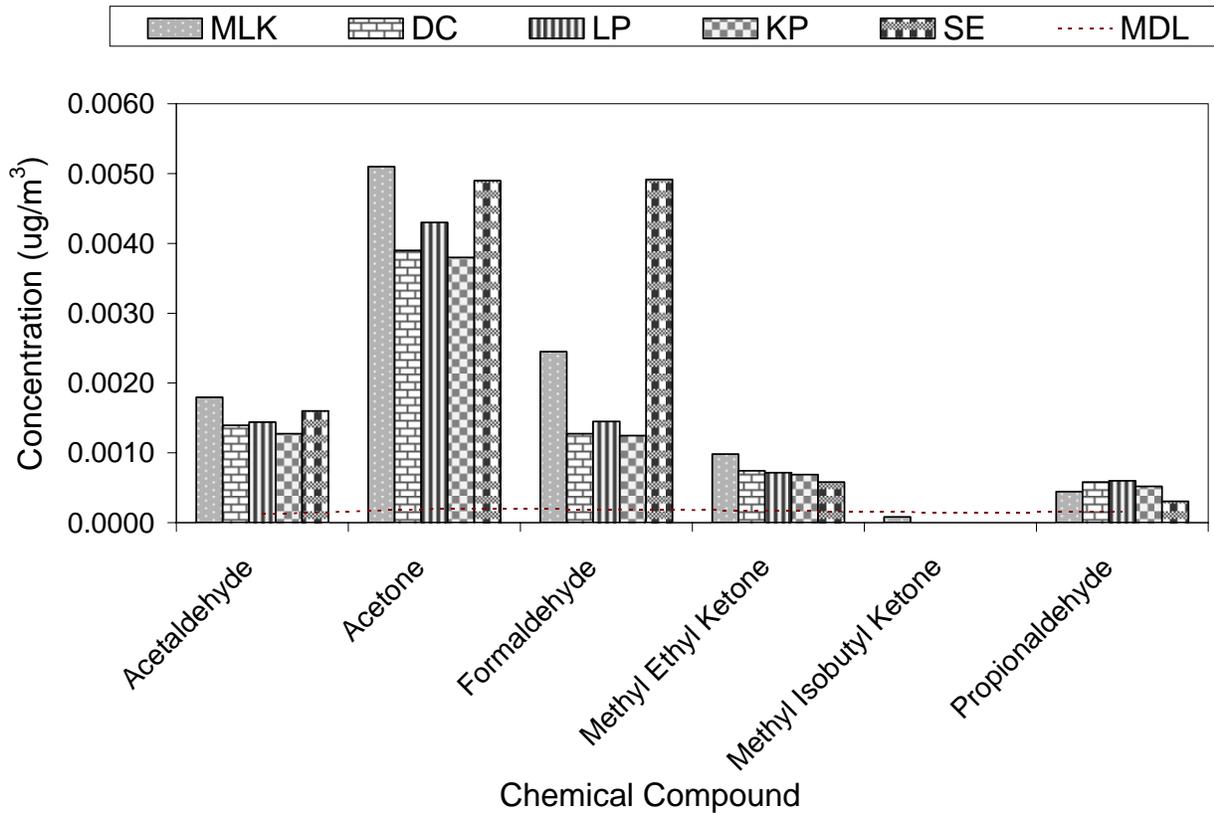


Figure 5.3: Annual average carbonyl concentrations in the air at five Delaware Sites shown in Table 2.1. Dotted line represents MDLs for the shown compounds. These are independent of each other, but are connected via dotted lines on the plots only to make them noticeable.

5.1.4 PAHs

The annual average concentration for all PAH compounds are presented in Table 5.4 and plotted in Figure 5.4 (a, and b). Average minimum detection limits are also shown for each compound. Additional summary results can be found in Tables B4-1 to B4-3 (Appendix).

Preliminary assessment of the PAH data follows.

- Many PAH compounds show the highest concentrations at DC, MLK, and SE monitoring sites,
- A few compounds have somewhat higher annual averages at LP,
- The rural site, KP, has the lowest annual averages for most of the PAHs monitored.

Table 5.4: PAH concentrations ($\mu\text{g}/\text{m}^3$) at five Delaware sites

Compound	Avg MDL	MLK	DC	LP	KP	SE
1-Methylnaphthalene	0.0003	0.00252	0.00113	0.00219	0.00062	0.00091
2-Methylnaphthalene	0.0003	0.00449	0.00186	0.00438	0.00099	0.00146
Acenaphthene	0.0003	0.00208	0.00046	0.00041	0.00028	0.00049
Acenaphthylene	0.0003	0.00152	0.00060	0.00041	0.00030	0.00038
Anthracene	0.0003	0.00090				
Benzo(a)anthracene	0.0003	0.00043	0.00024			
Benzo(a)pyrene	0.0003	0.00053				
Benzo(b)fluoranthene	0.0003	0.00107	0.00050	0.00007	0.00014	0.00044
Benzo(e)pyrene	0.0003	0.00058				
Benzo(g,h,i)perylene	0.0003	0.00057				
Benzo(k)fluoranthene	0.0003	0.00025				
Chrysene	0.0003	0.00075	0.00034	0.00033		0.00027
Dibenz(a,h)anthracene	0.0003					
Fluoranthene	0.0003	0.01144	0.00113	0.00255	0.00060	0.00177
Fluorene	0.0003	0.00468	0.00128	0.00277	0.00081	0.00117
Indeno(1,2,3-cd)pyrene	0.0003	0.00059				
Naphthalene	0.0003	0.00317	0.00163	0.00604	0.00097	0.00148
Phenanthrene	0.0003	0.02966	0.00421	0.00875	0.00228	0.00434
Pyrene	0.0003	0.00589	0.00081	0.00173	0.00039	0.00027

Key: Colors relate to the percentage of non-detects or 0 samples. Black = no substitutions (no 0 or non-detects); Teal= lowest reported value or 1/2 MDL substituted for 0 (where 0%-50% of samples are non-detects or 0). Percentiles are used where 50%-90% of samples are non-detects or 0 as follows: Blue= 75th percentile Sky Blue= 80th percentile Violet= 90th percentile. No mean is generated if more than 90% of samples are non-detects or 0.

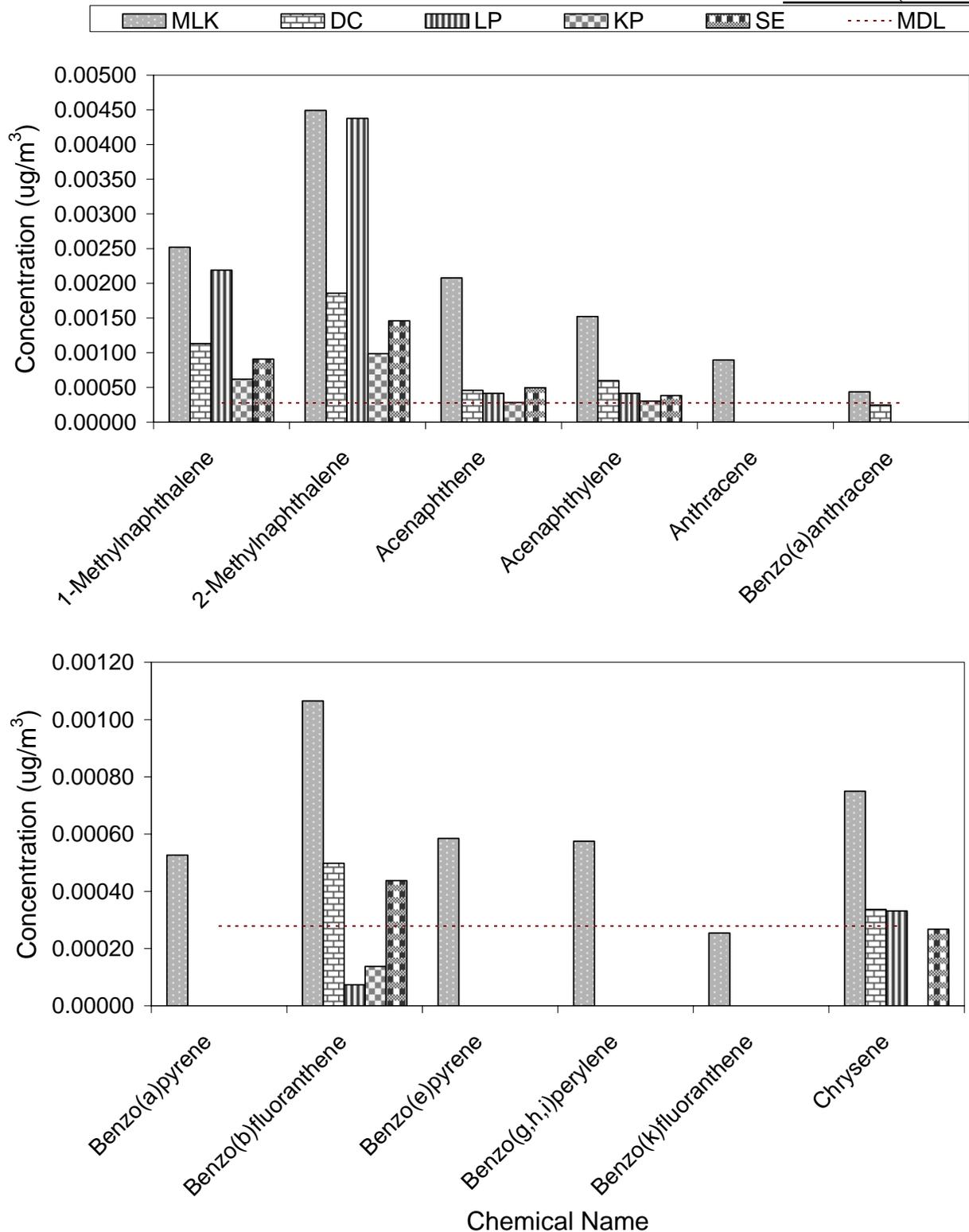


Figure 5.4(a): Annual average PAH concentrations in the air at five Delaware Sites shown in Table 2.1. Dotted line represents MDLs for the shown compounds. These are independent of each other, but are connected via dotted lines on the plots only to make them noticeable.

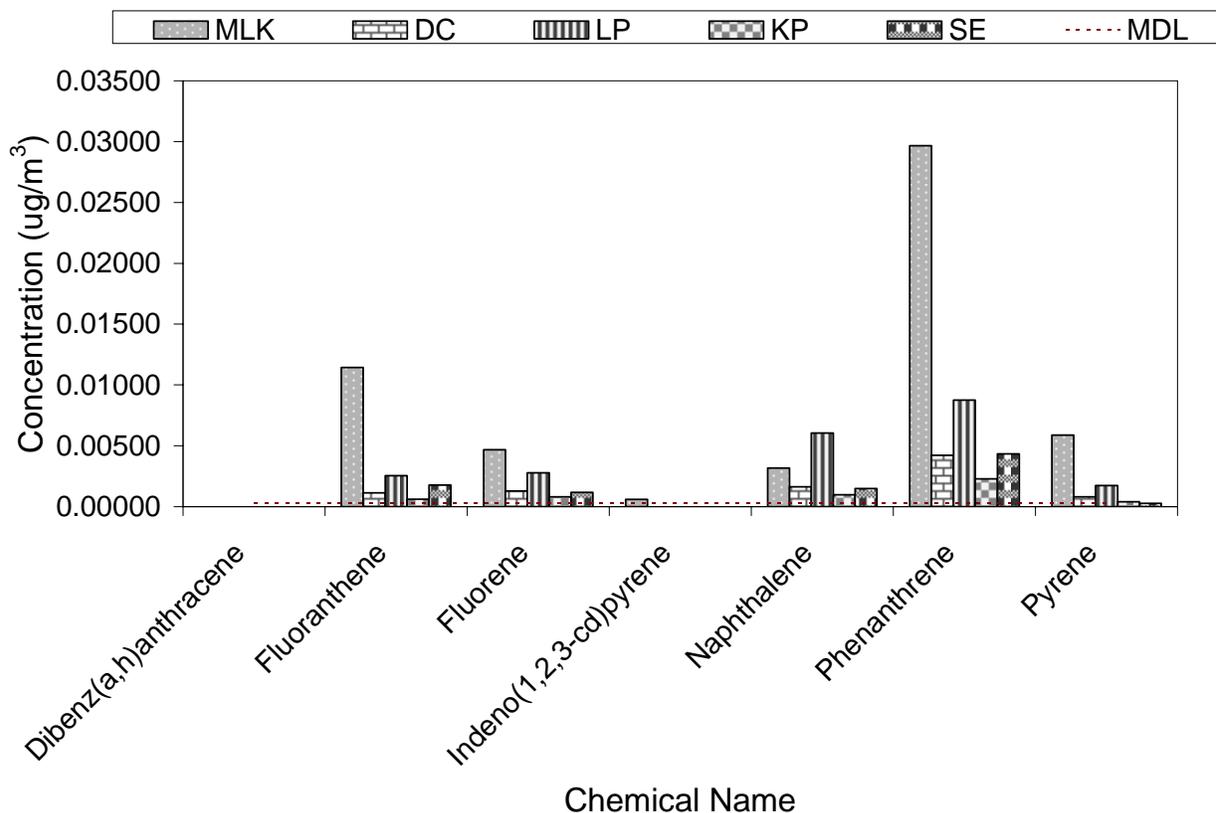


Figure 5.4(b): Annual average PAH concentrations in the air at five Delaware Sites shown in Table 2.1. Dotted line represents MDLs for the shown compounds. These are independent of each other, but are connected via dotted lines on the plots only to make them noticeable.

5.1.5 Dioxins/Furans

Annual average concentrations for each dioxin/furan compound are presented in Table 5.5 and plotted in Figure 5.5 (a, and b). Average minimum detection limits are presented for each congener. Additional summary results can be found in Tables B5-1 through B5-3 (Appendix).

Preliminary assessment of the dioxin/furan data follows.

- Concentrations of most D/F compounds are near the MDL,
- Analysis of the monitoring data is complicated by the relatively low number of samples (23 – 25 versus 47 – 52 for other groups of compounds) as well as the generally low concentrations,
- The concentrations as well as toxicity for D/F is usually expressed as TEQ of 2, 3, 7, 8-TCDD.

Table 5.5: Dioxin/Furan concentration (ng/m³) at five Delaware sites

Compound	Avg MDL	MLK	DC	LP	KP	SE
1,2,3,4,6,7,8-HpCDD	0.000010	0.000134	0.000089	0.000122	0.000236	0.000088
1,2,3,4,6,7,8-HpCDF	0.000010	0.000090	0.000034	0.000041	0.000070	0.000037
1,2,3,4,7,8,9-HpCDF	0.000010	0.000006	0.000004	0.000000	0.000004	0.000004
1,2,3,4,7,8-HxCDD	0.000010	0.000007	0.000005	0.000007	0.000004	0.000006
1,2,3,4,7,8-HxCDF	0.000010	0.000011	0.000007	0.000006	0.000013	0.000007
1,2,3,6,7,8-HxCDD	0.000010	0.000013	0.000008	0.000010	0.000017	0.000007
1,2,3,6,7,8-HxCDF	0.000010	0.000011	0.000007	0.000006	0.000005	0.000006
1,2,3,7,8,9-HxCDD	0.000010	0.000011	0.000008	0.000010	0.000016	0.000007
1,2,3,7,8,9-HxCDF	0.000010	0.000005	0.000002	0.000000	0.000002	
1,2,3,7,8-PeCDD	0.000010	0.000008	0.000005	0.000007	0.000004	0.000006
1,2,3,7,8-PeCDF	0.000010	0.000007	0.000004	0.000005	0.000004	0.000005
2,3,4,6,7,8-HxCDF	0.000010	0.000012	0.000007	0.000007	0.000006	0.000006
2,3,4,7,8-PeCDF	0.000010	0.000009	0.000005	0.000005	0.000007	0.000005
2,3,7,8-TCDD	0.000002	0.000002	0.000001	0.000001	0.000005	0.000001
2,3,7,8-TCDF	0.000002	0.000008	0.000004	0.000004	0.000005	0.000003
OCDD	0.000019	0.000483	0.000296	0.000375	0.000828	0.000279
OCDF	0.000019	0.000040	0.000022	0.000021	0.000046	0.000020

Key: Colors relate to the percentage of non-detects or 0 samples. Black = no substitutions (no 0 or non-detects); Teal= lowest reported value or ½ MDL substituted for 0 (where 0%-50% of samples are non-detects or 0). Percentiles are used where 50%-90% of samples are non-detects or 0 as follows: Blue= 75th percentile Sky Blue= 80th percentile Violet= 90th percentile. No mean is generated if more than 90% of samples are non-detects or 0.

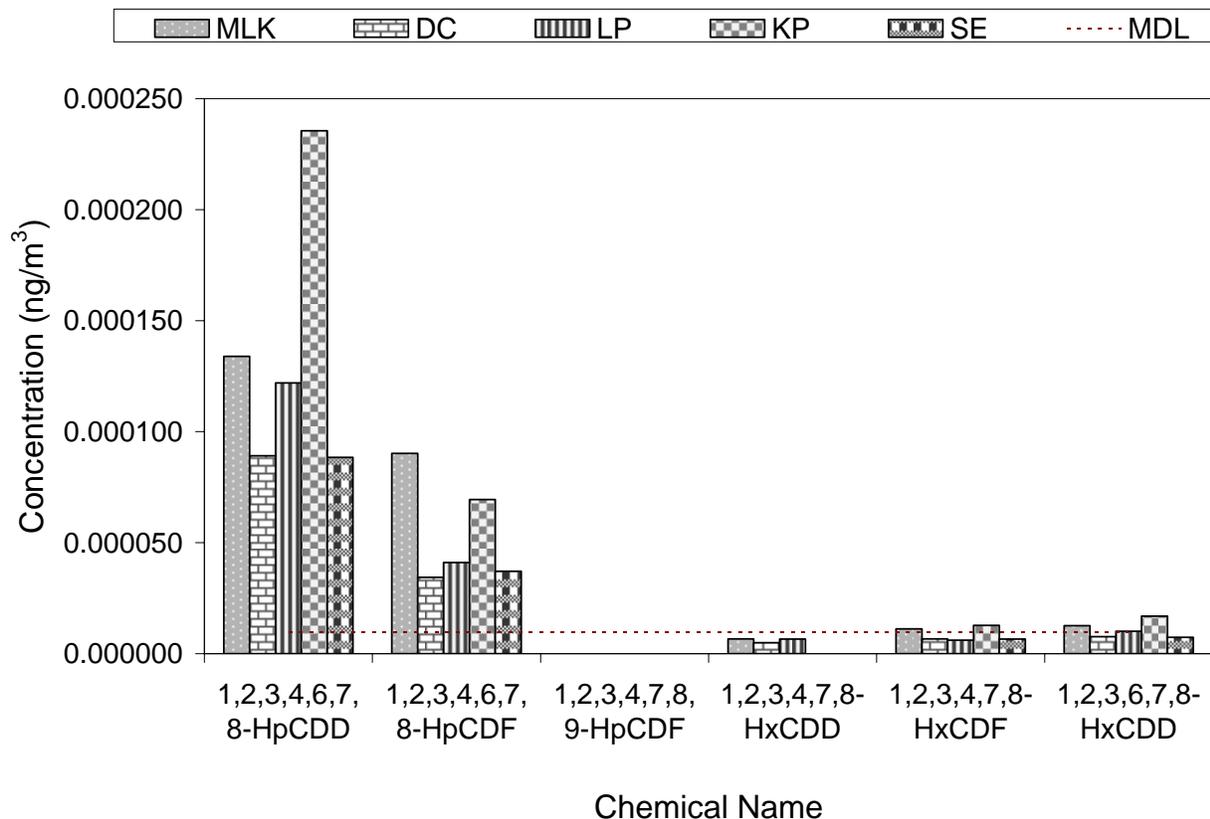


Figure 5.5(a): Annual average Dioxins/Furans concentrations in the air at five Delaware Sites shown in Table 2.1. Dotted line represents MDLs for the shown compounds. These are independent of each other, but are connected via dotted lines on the plots only to make them noticeable.

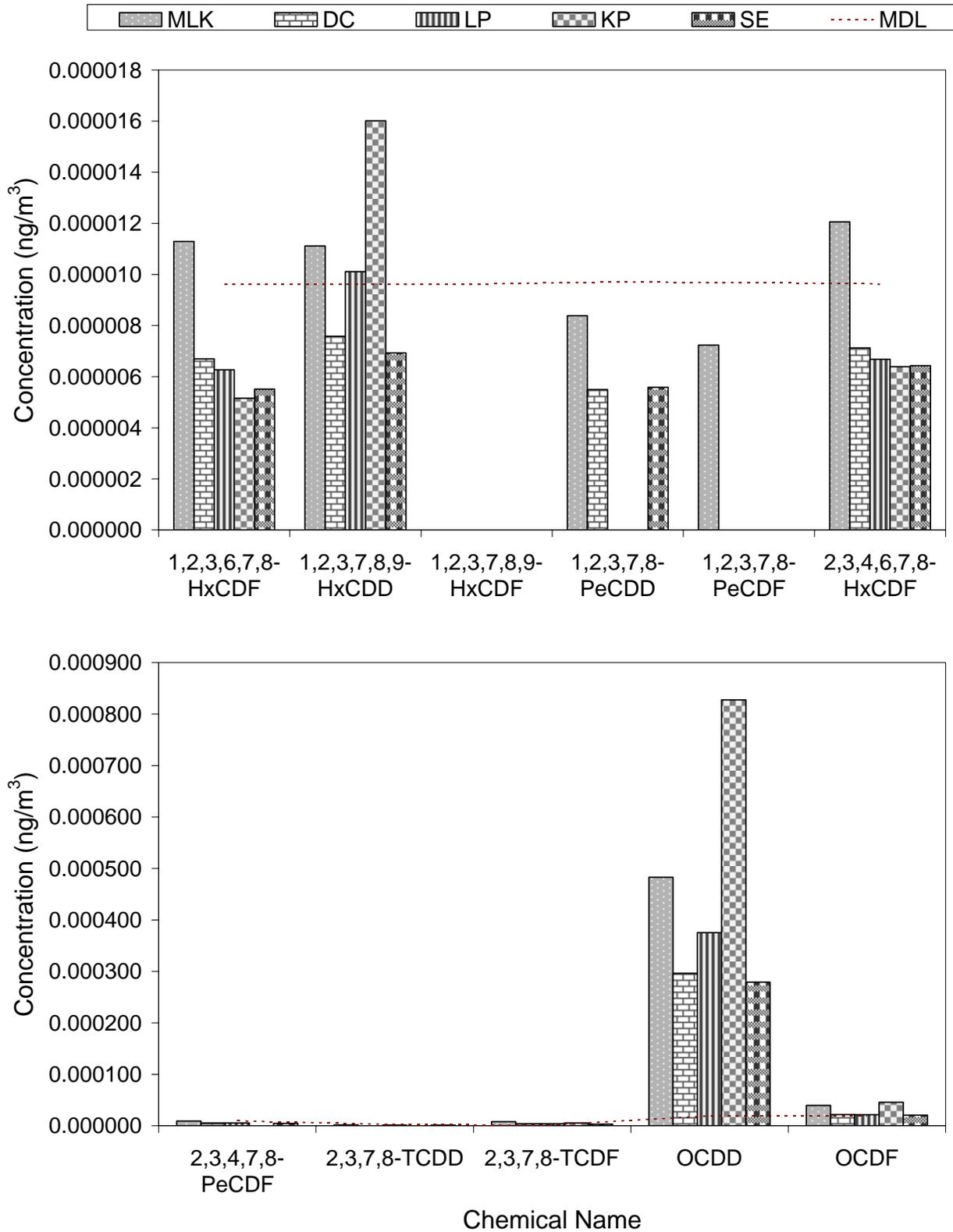


Figure 5.5(b): Annual average Dioxins/Furans concentrations in the air at five Delaware Sites shown in Table 2.1. Dotted line represents MDLs for the shown compounds. These are independent of each other, but are connected via dotted lines on the plots only to make them noticeable.

5.1.6 Additional analysis and findings

To gain additional understanding of the data, some further analyses of selected compounds were performed. These analyses form a preliminary basis for an in-depth study to be undertaken in the second phase of the DATAS project.

From the exhaustive list of monitored, inventoried and modeled compounds (Table 1A, Appendix), several compounds for this preliminary study were selected based upon comparison with other nationwide studies that identified certain compounds as prevalent across the country and presenting some degree of concern due to potential adverse health effects. These compounds were compared with the monitoring data for Delaware. Based on that comparison, the following compounds were chosen:

- Benzene,
- 1,3-Butadiene,
- Carbon Tetrachloride,
- Chloroethene (vinyl chloride),
- Chloroform,
- 1,2-Dibromoethane,
- Tetrachloroethylene (Perchloroethylene), and
- Arsenic.

The data used for the following statistical analyses were drawn from the actual sample results dataset. This is slightly different from the dataset used for risk assessment and presented in Tables B1-1 through B1-5 (Appendix). The actual sample dataset does not have any replacement values for the not detected or 0 results. The annual averages for the data used for the statistical analyses are shown in Table 5.6.

Table 5.6 Annual average concentrations ($\mu\text{g}/\text{m}^3$) (no replacements for “not detected” or 0 samples)

Compound	Avg MDL	MLK	DC	LP	KP	SE
Benzene	0.035	1.375	1.055	0.597	0.465	0.566
1,3-Butadiene	0.117	0.301	0.095	0.070	0.037	0.068
Carbon tetrachloride	0.296	0.541	0.536	0.543	0.560	0.561
Chloroethene (vinyl chloride)	0.067	0.044	0.221	0.048	0.004	0.003
Chloroform	0.128	0.132	0.096	0.088	0.081	0.088
1,2-Dibromoethane	0.169	0.012	0.006	0.008	0.009	0.007
Tetrachloroethylene	0.211	0.534	0.144	0.136	0.103	0.175
Arsenic	0.000036	0.001118	0.001021	0.000906	0.000884	0.000884

1. Box-Whisker Plots

Box-whisker plots are presented in Figure 5.6 for each of the selected compounds previously identified. The plots are organized by compound and monitoring location.

The components to the box-whisker plots include:

- The box, which represents the middle 50% of the concentration value,
- A line marking in the box, identifying the median,
- Upper and lower whiskers, representing the maximum and minimum values, respectively,
- Representation of the 1st and 3rd quartiles (which are the same as the 25th and 75th percentiles).

Also included for comparison purposes are the 2003 results reported for the Urban Air Toxics Monitoring Program (UATMP) [17]. The UATMP is a program designed to characterize the magnitude and composition of potentially toxic air pollution in, or near, urban locations. The UATMP data used in the box-whisker plots is drawn from summary statistics.

2. Seasonal patterns

Some air pollutants have been shown to follow a seasonal pattern. For DATAS project, the data was examined for seasonality by grouping the individual sample results by seasons as follows:

- December through February - Winter,
- March through May – Spring,
- June through August – Summer, and
- September through November – Fall.

An arithmetic mean was calculated for each selected compound for each season at each monitoring site. Because only one year of data was available for analyses, these comparisons can only be considered as preliminary. Results are presented as bar graphs in Figure 5.7.

Preliminary findings from this comparison are summarized as follows:

- Not all pollutants exhibit a seasonal pattern, and
- Benzene, 1,3 butadiene, and tetrachloroethylene exhibit wintertime maximum and summertime minimum concentrations.

One possible explanation for the higher winter concentrations of some compounds is related to weather conditions. Winter conditions tend to reduce mixing in the atmosphere because of stronger and more frequent temperature inversions. As a result, pollutants get trapped in a shallow layer at ground level and reach higher ground level concentrations. This is often compounded by calm wind conditions, further limiting dispersion of the pollutants.

For compounds produced by motor vehicles such as benzene, cold starts in winter lead to longer periods of incomplete combustion and longer warm-up times for catalytic converters, which generates more pollution. This could also play a role in higher winter concentrations for these compounds.

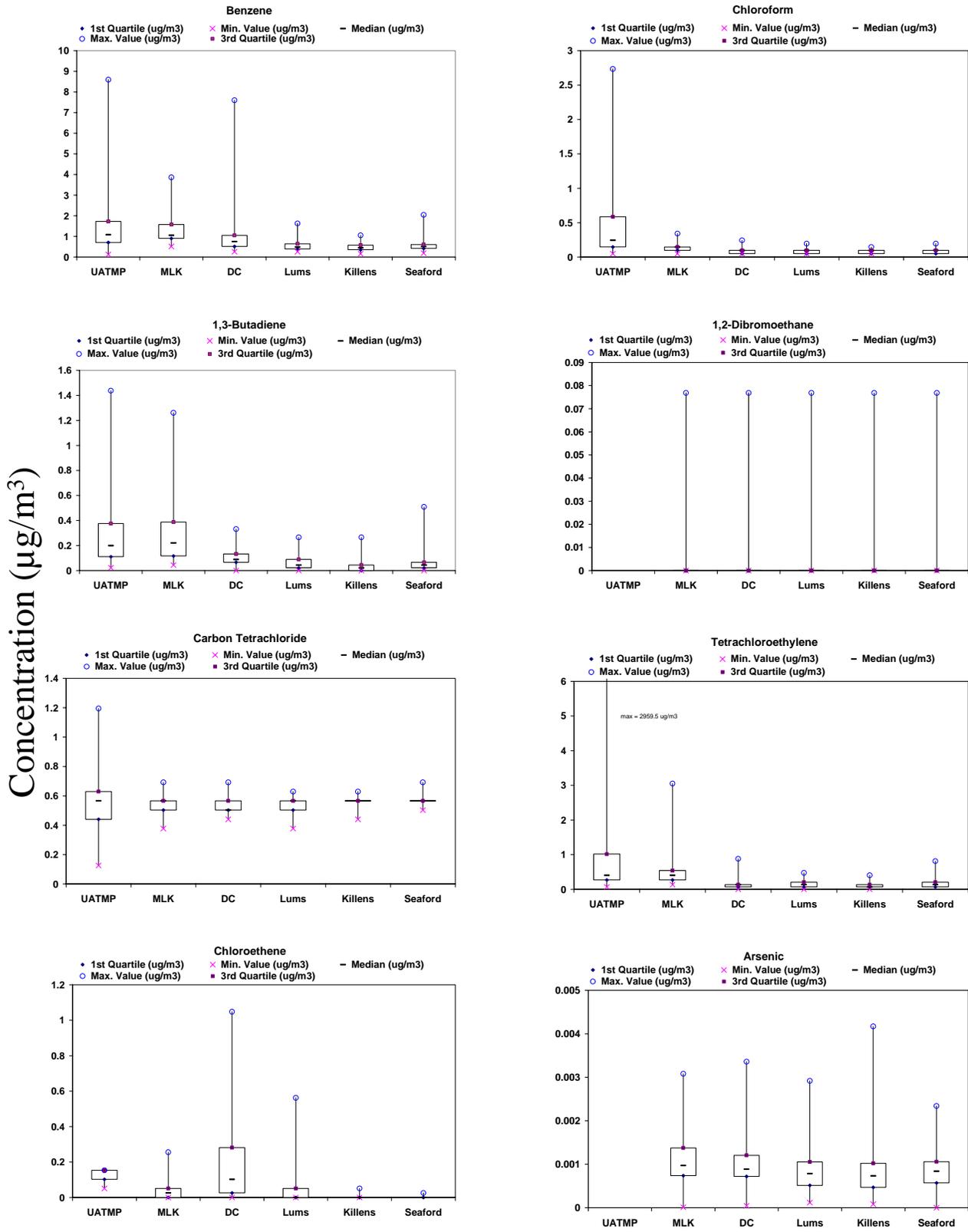


Figure 5.6: Distributions of UATMP and Delaware data

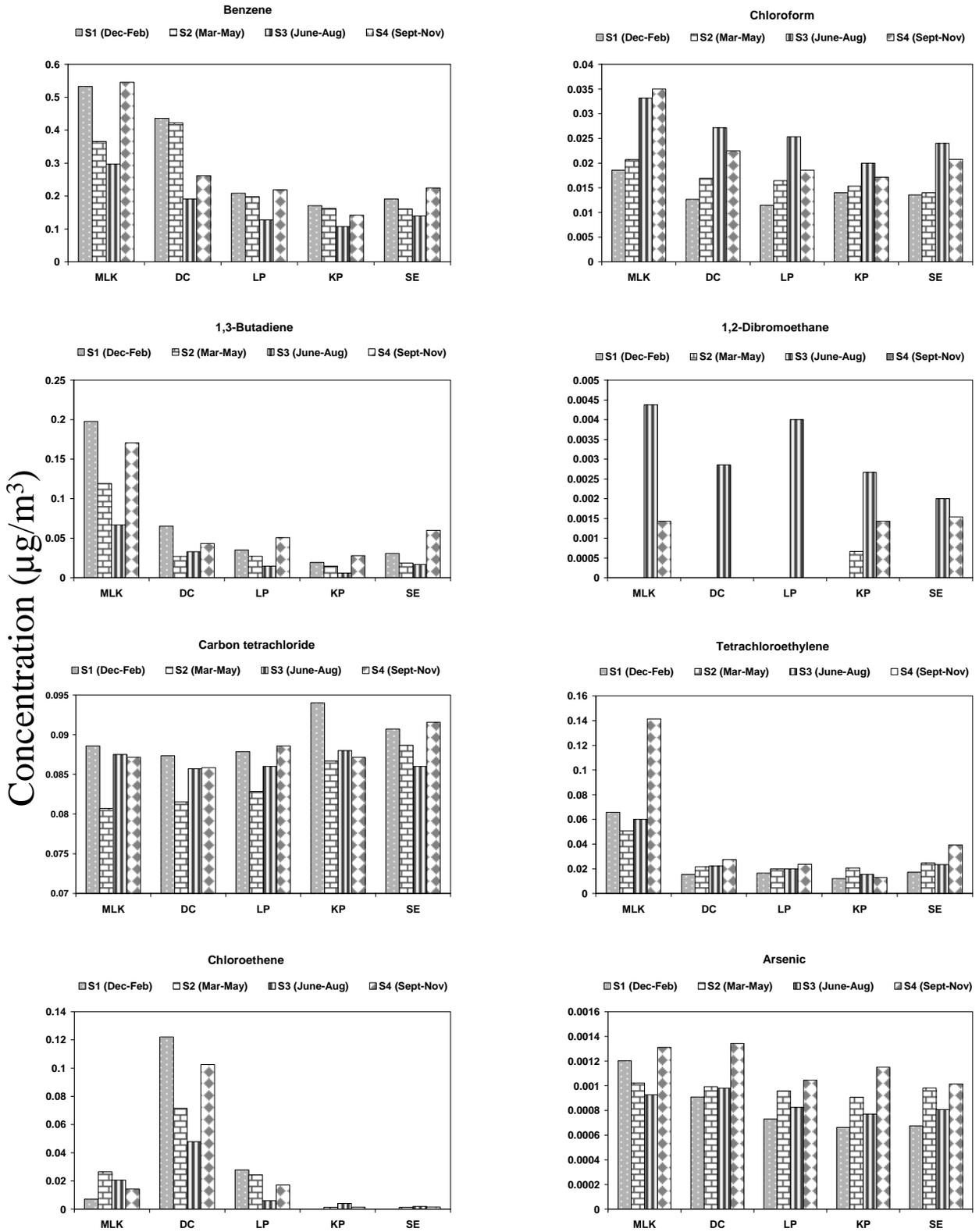


Figure 5.7: Seasonal variations

5.2 Emissions inventory results:

The 2003 air toxics emission inventory represents all known sources in Delaware of each of the DATAS chemicals. The inventory was developed from emission information that was provided to DNREC from Delaware specific sources. Emission factors available as of 2003 were used to estimate emissions from sources that did not directly report their emissions. The inventory was completed in late 2004 and modeling input files were created in early 2005.

The emission inventory quantifies the mass of emissions of each pollutant on a tons per year basis. Mass of emissions must be placed in context with each pollutant’s associated risks (i.e., cancer and/or non-cancer chronic effects.) For example, one ton of Toluene (a common VOC) may pose less risk than one gram of a dioxin. Thus, the ultimate value of the emission inventory is for use as an input to the modeling effort, which will utilize inventory values to estimate ambient concentrations. A risk assessment will then be performed on the ambient concentrations in order to quantify the predicted model output to the pollutants’ potential cancer/non-cancer risk. For this Phase I report, the results of the emissions are presented by mass amount only. The risk associated with these emissions will be determined in Phase II of DATAS.

For analysis purposes, emissions are grouped in two ways, by pollutant categories and by source sectors. The pollutant categories are the same as those used for the monitoring data. These include Volatile Organic Compounds (VOCs), Carbonyls, Metals, Polycyclic Aromatic Hydrocarbons (PAHs), and Dioxins/Furans (D/Fs). In addition to these five pollutant categories, diesel particulate matter (dPM) is treated separately. The source sectors include point sources (large facilities that report their emissions), area sources, on-road mobile sources, and off-road vehicles and equipments. These sectors were described in detail in Section 3 of this report.

5.2.1 Emissions by pollutant

The inventory development suggested seven DATAS chemicals have no identified emission sources in Delaware. These chemicals are listed in Table 5.7, none of which were included in the monitoring effort.

Table 5.7: DATAS pollutants with no identified emission sources

Chemical Name
Chloromethylbenzene
3,3-Dichlorobenzidine
Hexachloroethane
Pentachlorophenol
Quinoline
2,4,6-Trichlorophenol
Coke Oven Emissions

Of the remaining DATAS chemicals, 9 had more than 100 tons/year of emissions statewide for 2003. Six of these are VOCs, two carbonyls, and one dPM. Seven of these were

emitted predominantly by mobile sources (on- and off-road vehicles and equipments.) These high-volume chemicals are listed in Table 5.8. For further reference to the sources of these chemicals, see Table A1 in Appendix.

Table 5.8: DATAS chemicals with emissions greater than 100 tons/year for 2003

Chemical	Statewide Emissions (TPY)	Significant Source Sector
Toluene	1,501	76% mobile
Xylenes (all isomers)	1,321	67% mobile
Diesel Particulate Matter	770	95% mobile
Benzene	561	74% mobile
Formaldehyde	349	83% mobile
Ethylbenzene	265	68% mobile
Methylene Chloride	211	90% area ¹
Acetaldehyde	147	67% mobile
Perchloroethylene	143	99+% area ²

¹Commercial/Consumer Products, ²Dry Cleaning and Commercial/Consumer Products

The total mass of chemicals emitted in Delaware as tracked by DATAS in 2003 is 5,725 tons. The breakdown by chemical group, on a mass basis, is as follows:

VOCs	75.9%	4346 tons
Diesel particulate matter	13.4%	770 tons
Carbonyls	9.2%	525 tons
Metals, PAHs and D/Fs	1.5%	84 tons

A detailed discussion for each pollutant group is presented in the following

5.2.2 Volatile organic compounds

Emissions from 31 VOCs on the DATAS list of chemicals were quantified. No emissions were identified for seven VOCs. A large majority of VOC emissions were from chemicals that are components of fuel, in particular gasoline. These include Toluene, Xylenes, Ethylbenzene, and Benzene. These chemicals are primarily released through evaporation and incomplete combustion by on-road and off-road mobile sources. Refineries and supertanker crude oil lightering operations that take place in the lower Delaware Bay are other significant sources of these chemicals. Additional emissions of these chemicals occur during the marketing of gasoline, in particular at retail gasoline stations.

Other high-emission VOCs are split in dominance between area and point sources. Most are reported by only one or a few facilities, or within one or a few area source categories. For those chemicals emitted by only point sources, ambient concentrations will be dependent on the location of the facilities. Two examples of point source-driven emission totals include the

emission of Acrylonitrile from Dow Reichhold located near Cheswold (87% of statewide emissions) and Vinyl Chloride from Formosa Plastics and Kaneka (closed down in the late 2003) located in the Delaware City Industrial Complex (99% of statewide emissions.)

Several area sources are responsible for large emissions of certain VOCs. These include the category that quantifies emissions from Consumer/Commercial Products (1,4-Dichlorobenzene, Methylene Chloride, and Perchloroethylene), the Degreasing category (Trichloroethylene), and the Residential Wood Combustion category (Phenol). Consumer/Commercial Products use and Degreasing operations are activities concentrated in populated areas, while Residential Wood Combustion is an activity more prevalent in suburban and rural areas of the State. The breakdown of VOC emissions by source sector is shown in Figure 5.8.

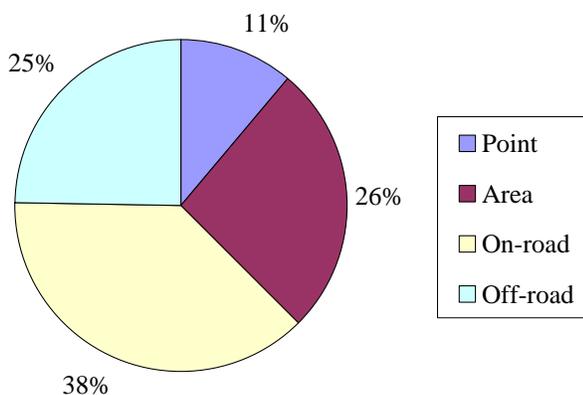


Figure 5.8: VOC Emissions by Source Sector
(Total 4346 tons)

5.2.3 Carbonyls

The DATAS carbonyls of concern include Acetaldehyde, Acrolein, and Formaldehyde. These chemicals are the result of combustion activities, including mobile source engine emissions, stationary combustion sources, residential wood combustion, open burning activities and commercial cooking. Since the mobile sources represent a majority of carbonyl emissions, emissions are increased in areas of New Castle County with a high density of roads and a large amount of traffic. Structure Fires is the largest source of Acrolein emissions. Finally, Residential Wood Combustion is a large source of all three carbonyls. The breakdown of Carbonyl emissions by source sector is shown in Figure 5.9.

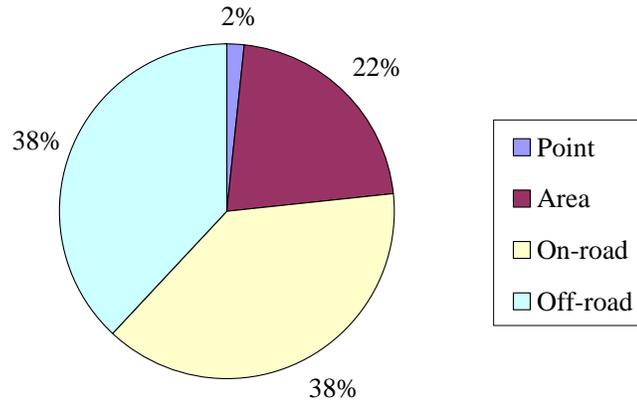


Figure 5.9: Carbonyl Emissions by Source Sector
(Total 525 tons)

5.2.4 Metals

Since Delaware has few metals processing facilities, the largest emissions of metal compounds come from the combustion of fossil fuels, in particular, coal-fired units. Metals are an impurity in coal and other fuels. Small amounts of metals are contained in crude oil, too, and are emitted from the Premcor Refinery, especially Nickel Compounds. Metal emissions were also quantified from re-entrained road dust. Dust emission categories are contained within the area source sector, although re-entrained road dust is related to on-road mobile sources. The breakdown of Metal emissions by source sector is shown in Figure 5.10.

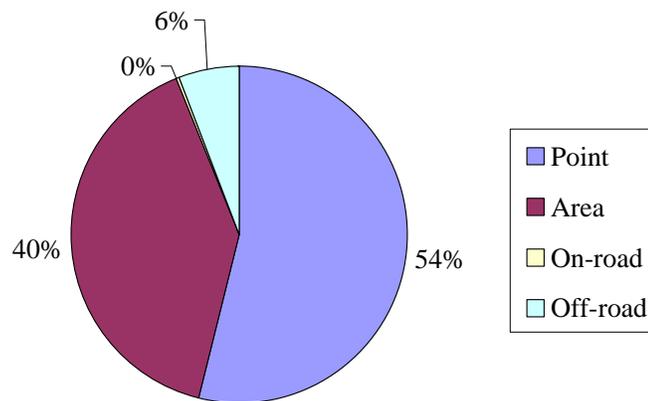


Figure 5.10: Metal Emissions by Source Sector
(Total <24 tons)

5.2.5 Polycyclic aromatic hydrocarbons

PAHs are mostly a result of combustion processes, in particular lower temperature combustion such as Residential Wood Combustion, Prescribed Fires, and Wildfires. These sources are widespread and not concentrated in population centers. PAH emissions from these sources represent over 80% of the statewide annual total. Stationary and mobile fuel combustion, while contributing less than 20% of the statewide total is more concentrated to urban and populated areas of the State. Concentrations may be higher in these areas as compared to rural areas. The breakdown of PAH emissions by source sector is shown in Figure 5.11.

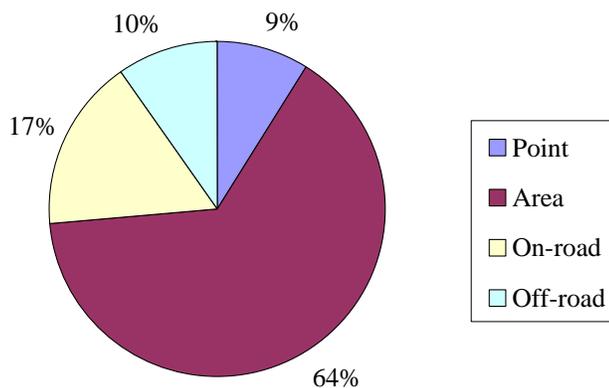


Figure 5.11: PAH Emissions by Source Sector
(Total <60 tons)

5.2.6 Dioxins/Furans

Similar to PAHs, Dioxins and Furans are combustion by-products. Emissions of D/Fs are fairly evenly split between large industrial combustion units, area source fuel combustion (including Residential Wood Combustion), mobile sources, and open burning activities. Even though the open burning of trash is considered a significant source of D/Fs nationwide, no emission factors were located during the inventory development process. Therefore, emissions are probably under-estimated in the inventory. This issue will be addressed in future inventories. The breakdown of D/F emissions by source sector is shown in Figure 5.12.

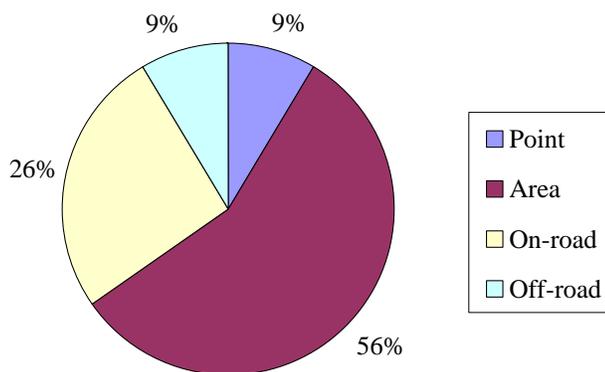


Figure 5.12: D/F Emissions by Source Sector
(Total <30 grams)

5.2.7 Diesel particulate matter

Diesel particulate matter (dPM) is a conglomeration of compounds that stick together as the combustion gases are released from an internal combustion engine. dPM is a very fine particulate, often less than one micron in diameter. The small size of diesel particulate enables it to penetrate the smallest airway passages of the lungs, and is one of the reasons that the health impacts of dPM are so great.

Mobile sources (on- and off-road vehicles and equipment) contribute 95% of the 770 tons total annual estimated dPM emitted in Delaware. Internal combustion diesel engines are used to a small degree as stationary sources.

5.2.8 Emissions by source sector

Source sector contributions to the five pollutant categories were presented in Figures 5.8 through 5.12. Considering all DATAS chemicals, 68% of the mass of emissions are attributed to mobile sources. Area sources contribute another 23%, while point sources contribute only 9%. Although point source emissions represent a small portion of the overall contribution of air toxics, owing essentially to their release pattern from one or only a few sources, point source emissions may pose greater health risks in areas close to those facilities. Area and mobile source emissions, on the other hand, occur over wide areas of Delaware, thus reducing the likelihood of concentration spikes. However, in areas of high population density and high economic activity, pollutant concentrations resulting from area and mobile sources may still pose a health risk, especially when considering the cumulative impact of all pollutants combined.

Ten DATAS pollutants with emissions exceeding 10 tons per year from point sources statewide are presented in Table 5.9 along with their major contributors.

Table 5.9: DATAS Pollutants with Statewide Point Source Emissions exceeding 10 tons/year for 2003

Chemical	Emissions (TPY)	Significant Sources (in order of contribution)
Xylenes (all isomers)	169.3	Maritrans, General Motors, DaimlerChrysler
Vinyl Chloride	63.0	Formosa Plastics, Kaneka
Toluene	55.3	Maritrans, DaimlerChrysler, Premcor
Ethylbenzene	53.6	Maritrans, DaimlerChrysler, Premcor
Benzene	50.2	Maritrans, Premcor, Sunoco,
Styrene	24.7	Justin Tanks, Dow Reichhold, Spatz Fiberglass
Methylene Chloride	20.3	Premcor, Sunoco
Diesel PM	20.1	Seaford Power Plant, Premcor
Ethylene Dichloride	15.4	Premcor, Sunoco
1,3-Butadiene	10.7	Dow Reichhold, Proctor & Gamble, Premcor

Fourteen DATAS pollutants with emissions exceeding 10 tons per year from area sources statewide are presented in Table 5.10 along with their source categories that had highest contributions.

Table 5.10: DATAS Pollutants with Statewide Area Source Emissions exceeding 10 tons/year for 2003

Chemical	Emissions (TPY)	Significant Sources (in order of contribution)
Toluene	298.2	Graphic Arts, Industrial Surface Coatings
Xylenes (all isomers)	267.5	Industrial Surface Coatings, AIM Coatings
Methylene Chloride	190.9	Consumer/Commercial Products
Perchloroethylene	142.7	Consumer/Commercial Products, Dry Cleaning
Benzene	97.3	Residential Wood Combustion, Ag. Pesticides
Formaldehyde	51.5	Residential Wood Combustion
Acetaldehyde	47.6	Residential Wood Combustion
1,4-Dichlorobenzene	40.0	Consumer/Commercial Products
Ethylbenzene	30.2	Gasoline Marketing
Phenol	30.1	Residential Wood Combustion
Trichloroethylene	28.3	Degreasing
Naphthalene	19.9	Residential Wood Combustion
Diesel PM	18.7	Industrial and Commercial Diesel Fuel Use ¹
Acrolein	14.1	Structure Fires

¹ Conservative assumption was applied to the burning of diesel fuel in the industrial and commercial fuel combustion categories. Not all diesel consumed in these categories is within an internal combustion engine.

5.3 Risk assessment results

Risk assessment was performed for a wide range of chemicals from the DATAS study. Potential risk was calculated for chemicals which were provided to EHEB by DNREC. The chemicals were compared to the October 2004 RBC Table and for IRIS from EPA Region III. Risk was calculated for detected chemicals with either inhalation or oral toxicity factors.

Risk values were calculated for individual chemicals detected at each of the five monitoring locations. The calculations are separated by chemical effect into cancer risk and non-cancer risk. The total risk of each cancer and non-cancer category was approximated by summing the individual chemical risks at each site for each receptor group. The total procedure provides for a conservative approach since it is unlikely that all chemicals would target the same organ for cancer or add to the same adverse health effect.

The level of risk posed by an individual chemical at a particular monitoring location, as well as the total risk for that location, was determined by a three level risk scale. Tables of risk values included in this report include values color coded as green, yellow and red. This color coding is a visual way to represent the approximate risk based on the exposure scenarios and the monitored concentrations. Chemicals capable of causing cancer that showed concentrations with the potential to cause one or less additional cancer case in 100,000 exposed people are shown in green. Cancer-causing chemicals showing increased, incremental, lifetime cancer risk with concentrations that could cause between one and ten additional cancer cases in 100,000 exposed people are shown in yellow. Chemicals with concentrations and potential to cause over ten additional increased, incremental lifetime cancer cases per 100,000 exposed people are shown in red. Table C4-1 (Appendix) is an example of the results for Killens Pond, listing the chemicals, estimated dose, and the calculated risk for each of the three scenarios.

The same color-coding scheme was applied to chemicals with non-cancer effects, though the numerical values each color represents are different. Hazard quotients of 1 or less than 1 were considered low adverse health effect scenarios (displayed in green). Compounds with a hazard quotient of between 1 and 10 were considered to be an increased level of adverse effects (displayed in yellow). Compounds with a hazard quotient of greater than 10 were considered at a high level (displayed in red).

The risk analysis of the individual chemicals in this study showed no individual chemical with risk above the low risk levels at any of the monitoring stations. There are several chemicals however, detected at concentrations close to the value triggering a yellow color (increased risk). Chromium was the chemical where the calculated risk was closest to its elevated risk value. The cumulative risks for each monitoring location showed some areas with levels of elevated risk. These levels are coded in yellow on the Tables included with this report (See Appendix, Tables C1 through C5).

The monitoring station located at Martin Luther King Boulevard in Wilmington showed the highest overall chemical concentrations. The risk analysis showed that cancer and non-cancer causing chemicals were present in sufficient concentrations to reach an increased risk level when summed. The increased risk for cancer was seen in all three age scenarios. At MLK

the chemicals that contributed most to the summed risk are chromium, trichloroethene, benzene, 1,3 butadiene, and carbon tetrachloride. For the non-cancer level (Hazard Index), the chemicals that contributed most to risk were manganese, carbon tetrachloride, 1,3 butadiene, and 1,2,4 trimethylbenzene.

The remaining monitoring stations differed in the concentrations and types of individual chemicals detected. However, when summed the color-coded results appear visually similar. The non-cancer causing chemicals detected at each of the sites showed an increased adverse effect level for the child exposure scenario. This scenario is the most susceptible to a non-cancer exposure, based on a chemical intake per unit of body weight basis. The reverse was seen in the results of the cancer causing chemical risk analysis. The child scenario in these four monitoring stations was green. The adult and age-adjusted scenarios were yellow in each location. The age-adjusted scenario is the most susceptible to cancer causing chemicals with the adult scenario being slightly less susceptible. The susceptibility to cancer-causing chemical exposure is determined mostly by exposure time and for the age-adjusted calculations the factor of a child's intake per body weight playing a role.

The National Air Toxics Assessment (NATA) study of 1996 included 33 chemicals for assessment. See Table 5.11 for a list of the chemicals. DATAS included these, except for PCBs and acrolein, and expanded the assessment to include additional chemicals totaling 89.

Table 5.11: 1996 National Air Toxics Assessment Study Chemicals [33]

Acetaldehyde	Dioxin	Mercury compounds
Acrolein	1,2 dibromoethane	Methylene chloride
Acrylonitrile	Propylene dichloride	Nickel compounds
Arsenic compounds	1,3 dichloropropene	Polychlorinated biphenyls
Benzene	Ethylene dichloride	Polycyclic organic matter
1,3 butadiene	Ethylene oxide	Quinoline
Cadmium compounds	Formaldehyde	1,1,2,2 tetrachloroethane
Carbon tetrachloride	Hexachlorobenzene	Tetrachloroethylene
Chloroform	Hydrazine	Trichloroethylene
Chromium compounds	Lead compounds	Vinyl chloride (chloroethene)
Coke oven emissions	Manganese compounds	

The NATA grouped chemicals into cancer and non-cancer risk drivers and cancer and non-cancer risk contributors. The distinction of drivers and contributors is noted below along with the chemicals found. In general, risk drivers have a greater risk to more people than contributors.

5.3.1 Important national cancer risk chemicals

The NATA found the known carcinogens benzene and chromium to be a national cancer risk driver. National drivers are those chemicals with a risk greater than 10 in a million to more

than 25 million people. For probable carcinogens formaldehyde had the greatest risk. In DATAS benzene, chromium and formaldehyde were present in the air, though none were deemed high risk or increased risk levels based on the monitored data.

5.3.2 Regional cancer risk chemicals

The NATA found arsenic and coke oven emissions to be chemicals of regional concern. A region is an area where there is estimated to be 10 in a million risk to more than 1 million people. For probable carcinogens 1,3 butadiene and polycyclic organic matter showed the greatest risk. For DATAS arsenic, 1,3 butadiene and PAHs were present. However, coke oven emissions were not present. Again based on the monitored data none were high or at an increased risk level.

5.3.3 National cancer risk contributors

Cancer risk contributors are those chemicals exceeding a 1 in a million risk to more than 25 million people. Nickel is the known carcinogen for this category. Probable carcinogens are acetaldehyde, carbon tetrachloride, chloroform, ethylene dibromide, ethylene dichloride, and perchloroethylene. DATAS found nickel, acetaldehyde, carbon tetrachloride, chloroform, and ethylene dichloride (1,2-dichloroethane). None of the chemicals had a high or an increased risk level in DATAS.

5.3.4 Regional cancer risk contributors

The NATA cancer risk contributors have a risk exceeding 1 in a million to more than one million people. In this category the probable carcinogens acrylonitrile, beryllium, cadmium, ethylene oxide, 1,3-dichloropropene, hydrazine, and trichloroethylene show such risks. The possible carcinogens quinoline and 1,1,2,2-tetrachloroethane show such risks. DATAS found cadmium, trichloroethylene, and 1,1,2,2-tetrachloroethane were present but none at high or increased risk levels.

5.3.5 National non-cancer hazard drivers

For the non-cancer chemicals NATA found acrolein to be a hazard driver. A non-cancer hazard driver is where the hazard quotient exceeds 1 for more than 25 million people. Acrolein was not part of the DATAS sampling routine.

5.3.6 Regional non-cancer drivers

Regional non-cancer hazard drivers are those where the hazard quotient exceeds 1 for more than 10,000 people. Chemicals in this category were acetaldehyde, arsenic, 1,3 butadiene, formaldehyde, and manganese. DATAS found these chemicals but none were at a high or increased non-cancer risk levels.

5.3.7 Health effects of pollutants of concern

As noted above, on an individual basis no chemical reached the level of increased risk (yellow shading). When risk was summed for a particular monitoring site some summations were in the yellow range for cancer risk and non-cancer adverse health effect level. A few chemicals contributed to a greater part of the risk value. These were chromium, benzene, 1,3 butadiene, trichloroethene, carbon tetrachloride, chloroethene (vinyl chloride), arsenic, and 1,2,4 trimethylbenzene. Table 5.12 lists these chemicals. Table 5.13 shows some of the health effects of these chemicals.

Table 5.12: Chemicals contributing 10 percent or more to total risk by monitoring location.

Martin Luther King Monitoring Station	
Carcinogens	Non-Carcinogens
Chromium	Manganese
Trichloroethene	Carbon tetrachloride
Benzene	1,3-Butadiene
1,3-Butadiene	1,2,4-Trimethylbenzene
Carbon tetrachloride	
Delaware City Monitoring Station	
Carcinogens	Non-Carcinogens
Trichloroethene	Carbon tetrachloride
Chloroethene	Manganese
Chromium	
Benzene	
Carbon tetrachloride	
Lums Pond Monitoring Station	
Carcinogens	Non-Carcinogens
Chromium	Carbon tetrachloride
Carbon tetrachloride	Manganese
Trichloroethene	
Killens Pond Monitoring Station	
Carcinogens	Non-Carcinogens
Trichloroethene	Carbon tetrachloride
Carbon tetrachloride	Manganese
Chromium	
Seaford Monitoring Station	
Carcinogens	Non-Carcinogens
Carbon tetrachloride	Carbon tetrachloride
Trichloroethene	Manganese
Chromium	

Table 5.13: Health effects of pollutants of concern

Chemical of concern	Chronic health hazards [34-37]
Chromium VI (C, NC)	<p>Chronic inhalation exposure to chromium (VI) in humans results in effects on the respiratory tract, with perforations and ulcerations of the septum, bronchitis, decreased pulmonary function, pneumonia, asthma, and nasal itching and soreness reported.</p> <p>Chronic human exposure to high levels of chromium (VI) by inhalation or oral exposure may produce effects on the liver, kidney, gastrointestinal and immune systems, and possibly the blood.</p> <p>Rat studies have shown that, following inhalation exposure, the lung and kidney have the highest tissue levels of chromium.</p> <p>Dermal exposure to chromium (VI) may cause contact dermatitis, sensitivity, and ulceration of the skin.</p> <p>Epidemiological studies of workers have clearly established that inhaled chromium is a human carcinogen, resulting in an increased risk of lung cancer. EPA has concluded that only chromium (VI) should be classified as a human carcinogen. EPA has classified chromium (VI) as a Group A, known human carcinogen by the inhalation route of exposure.</p>
Manganese (NC)	<p>Manganese at low levels is nutritionally essential in humans. The recommended daily intake of manganese is 2 to 5 mg/day for adults and adolescents.</p> <p>Chronic inhalation exposure of humans to manganese results primarily in effects on the nervous system. Slower visual reaction time, poorer hand steadiness, and impaired eye-hand coordination were reported in several studies of workers occupationally exposed to manganese dust in air.</p> <p>Chronic inhalation exposure of humans to high levels may result in a syndrome called manganism and typically begins with feelings of weakness and lethargy and progresses to other symptoms such as gait disturbances, clumsiness, tremors, speech disturbances, a mask-like facial expression, and psychological disturbances.</p> <p>Other chronic effects reported in humans from inhalation exposure to manganese are respiratory effects such as an increased incidence of cough, bronchitis, dyspnea during exercise, and an increased susceptibility to infectious lung disease.</p> <p>EPA has classified manganese as a Group D, not classifiable as to carcinogenicity in humans.</p>

Chemical of concern	Chronic health hazards [34-37]
	<p>Reproductive effects, such as impotence and loss of libido, have been noted in male workers afflicted with manganism attributed to occupational exposure to high levels of manganese by inhalation.</p>
<p>Trichloroethene (trichloroethylene) (C, NC)</p>	<p>Chronic exposure to trichloroethylene by inhalation can affect the human central nervous system. Case reports of intermediate and chronic occupational exposures included effects such as dizziness, headache, sleepiness, nausea, confusion, blurred vision, facial numbness, and weakness.</p> <p>Effects to the liver, kidneys, and immune and endocrine systems have also been seen in humans exposed to trichloroethylene occupationally or from contaminated drinking water.</p> <p>Studies have shown that simultaneous alcohol consumption and trichloroethylene inhalation increases the toxicity of trichloroethylene in humans.</p> <p>Neurological, liver, and kidney effects were reported in chronically-exposed animals.</p> <p>Epidemiological studies strongest demonstrate an association between trichloroethylene exposure and kidney cancer.</p> <p>Animal studies have reported increases in lung, liver, kidney, and testicular tumors and lymphoma from inhalation and oral exposures in rats and mice.</p> <p>EPA does not currently have a consensus classification for the carcinogenicity of trichloroethylene. However, the Agency is currently reassessing its potential carcinogenicity, and new data suggest that trichloroethylene is a likely human carcinogen. However, no cancer classification has been assigned yet.</p>
<p>Benzene (C, NC)</p>	<p>Chronic inhalation of certain levels of benzene causes disorders in the blood in humans. Benzene specifically affects bone marrow (the tissues that produce blood cells). Aplastic anemia, excessive bleeding, and damage to the immune system (by changes in blood levels of antibodies and loss of white blood cells) may develop.</p> <p>Benzene causes both structural and numerical chromosomal aberrations in humans.</p> <p>Increased incidence of leukemia (cancer of the tissues that form white blood cells) has been observed in humans occupationally exposed to</p>

Chemical of concern	Chronic health hazards [34-37]
	<p>benzene.</p> <p>EPA has classified benzene as a Group A, known human carcinogen</p>
Carbon tetrachloride (C, NC)	<p>Carbon tetrachloride is reasonably anticipated to be a human carcinogen based on sufficient evidence of carcinogenicity in experimental animals. When administered by gavage, animal studies demonstrated an increase the incidences of hepatomas and hepatocellular carcinomas, and an increase in the incidence of neoplastic nodules of the liver. When administered intrarectally, it also induced hepatocellular carcinomas in male rats and mammary adenocarcinomas and fibroadenomas in female rats. When administered intrarectally to male mice, the compound induced nodular hyperplasia of the liver. When administered by inhalation, carbon tetrachloride induced liver carcinomas in rats.</p> <p>No adequate data were available from human studies to evaluate the carcinogenicity of carbon tetrachloride in humans. Occasional reports have noted the occurrence of liver cancer in workers who had been exposed to carbon tetrachloride by inhalation exposure; however, the data are not sufficient to establish a cause-and-effect relationship.</p> <p>Chronic inhalation or oral exposure to carbon tetrachloride produces liver and kidney damage in humans and animals. EPA has not established a Reference Concentration (<u>RfC</u>) for carbon tetrachloride. The California Environmental Protection Agency (CalEPA) has established a chronic reference exposure level of 0.04 milligrams per cubic meter (mg/m³) for carbon tetrachloride based on liver effects in guinea pigs.</p> <p>ATSDR has established an acute duration (1-14 days) inhalation minimal risk level (MRL) of 1.3 mg/m³ (0.2 parts per million [ppm]) based on liver effects in rats, and an intermediate duration (14-365 days) MRL of 0.3 mg/m³ (0.05 ppm) also based on liver effects in rats. The 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.</p> <p>EPA has classified carbon tetrachloride as a Group B2, probable human carcinogen.</p>
Chloroethene (vinyl chloride) (C)	<p>Liver damage may result in humans from chronic exposure to vinyl chloride, through both inhalation and oral exposure.</p> <p>A small percentage of individuals occupationally exposed to high levels of vinyl chloride in air have developed a set of symptoms termed "vinyl chloride disease," which is characterized by Raynaud's phenomenon</p>

Chemical of concern	Chronic health hazards [34-37]
	<p>(fingers blanch and numbness and discomfort are experienced upon exposure to the cold), changes in the bones at the end of the fingers, joint and muscle pain, and scleroderma-like skin changes (thickening of the skin, decreased elasticity, and slight edema).</p> <p>CNS effects (including dizziness, drowsiness, fatigue, headache, visual and/or hearing disturbances, memory loss, and sleep disturbances) as well as peripheral nervous system symptoms (peripheral neuropathy, tingling, numbness, weakness, and pain in fingers) have also been reported in workers exposed to vinyl chloride.</p> <p>Animal studies have reported effects on the liver, kidney, and CNS from chronic exposure to vinyl chloride.</p> <p>Testicular damage and decreased male fertility have been reported in rats exposed to low levels for up to 12 months.</p> <p>Inhaled vinyl chloride has been shown to increase the risk of a rare form of liver cancer (angiosarcoma of the liver) in humans.</p> <p>Animal studies have shown that vinyl chloride, via inhalation, increases the incidence of angiosarcoma of the liver and cancer of the liver.</p> <p>Several rat studies show a pronounced early-life susceptibility to the carcinogenic effect of vinyl chloride, i.e., early exposures are associated with higher liver cancer incidence than similar or much longer exposures that occur after maturity.</p> <p>EPA has classified vinyl chloride as a Group A, human carcinogen.</p>
1,3-butadiene (C, NC)	<p>One epidemiological study reported that chronic (long-term) exposure to 1,3-butadiene via inhalation resulted in an increase in cardiovascular diseases, such as rheumatic and arteriosclerotic heart diseases, while other human studies have reported effects on the blood.</p> <p>Animal studies have reported effects on the respiratory and cardiovascular systems, blood, and liver from chronic, inhalation exposure to 1,3-butadiene.</p> <p>Animal studies using mice have reported developmental effects, such as skeletal abnormalities and decreased fetal weights, and reproductive effects, including an increased incidence of ovarian atrophy and testicular atrophy from inhalation exposure to 1,3-butadiene.</p> <p>A large epidemiological study of synthetic rubber industry workers demonstrated a consistent association between 1,3-butadiene exposure</p>

Chemical of concern	Chronic health hazards [34-37]
	<p>and occurrence of leukemia.</p> <p>Animal studies have reported tumors at a variety of sites from inhalation exposure to 1,3-butadiene.</p> <p>1,3-Butadiene is metabolized into genotoxic metabolites by experimental animals and humans.</p> <p>EPA has classified 1,3-butadiene as a Group B2, probable human carcinogen. However, based on recently available human data, EPA is reevaluating the cancer classification.</p>
<p>1,2,4-trimethylbenzene (NC)</p>	<p>Inhalation of 1,2,4-trimethylbenzene has been reported to cause headache, fatigue, drowsiness, decreases in levels of red and white blood cells (anemia), impairment of blood coagulation, and bronchitis. Direct contact with 1,2,4-trimethylbenzene can cause skin irritation.</p> <p>1,2,4-trimethylbenzene depresses the central nervous system. Exposure to 1,2,4-trimethylbenzene causes headache, fatigue, nervousness, drowsiness, tension, and bronchitis</p> <p>No information on the carcinogenicity of 1,2,4-trimethylbenzene is available.</p>
<p>Arsenic (C)</p>	<p>Chronic inhalation exposure to inorganic arsenic in humans is associated with irritation of the skin and mucous membranes (dermatitis, conjunctivitis, pharyngitis, and rhinitis).</p> <p>Chronic oral exposure to inorganic arsenic in humans has resulted in gastrointestinal effects, anemia, peripheral neuropathy, skin lesions, hyperpigmentation, gangrene of the extremities, vascular lesions, and liver or kidney damage.</p> <p>Human, inhalation studies have reported inorganic arsenic exposure to be strongly associated with lung cancer.</p> <p>Ingestion of inorganic arsenic in humans has been associated with an increased risk of non-melanoma skin cancer and also to an increased risk of bladder, liver, and lung cancer.</p> <p>EPA has classified inorganic arsenic as a Group A, human carcinogen.</p>

Section 6

Modeling

Air toxics modeling is an essential element of DATAS which is expected to provide a better understanding of ambient concentrations of air toxics in communities of interest, their exposure to those air toxics, and help evaluating related health risks. Specifically, it will help assessing air toxics health impacts in communities other than those monitored, defining risk-based air quality standards, developing control strategies for meeting these standards, improving emissions inventory development, and assisting permitting process base its decisions on cumulative impacts.

Modeling objectives for DATAS include utilizing regional-scale modeling to account for long-range transport and secondary production of many air toxic compounds due to photochemistry, and local-scale modeling to address impact from nearby sources. This effort is based on 2003 monitoring data, emissions inventory and meteorology. In this section, results of a direct comparison between several existing models are presented. Air quality modeling for determining ambient air toxic concentrations for 2003 will be conducted in Phase II of DATAS. A prototype modeling simulation has been completed collaboratively with EPA (details in Section 6.2). The effort proved to be invaluable for defining the DATAS modeling objectives. The prototype demonstrated the ability of the regional-scale models to:

- Capture spatial and temporal variability of the air toxics compounds,
- Identify and characterize hot spots of air toxics compounds,
- Characterize photochemical mechanisms specific to carbonyl compounds, and
- Address long-range transport.

This section explains the significance of regional-scale modeling for assessing impacts of air toxics on neighborhood communities, the experience gained from a recent regional-scale modeling effort in collaboration with EPA, and the DATAS approach to assess the impact of air toxic emissions for the five selected communities in Delaware.

6.1 Significance of regional-scale modeling for air toxics assessment

Ambient concentrations of air toxics (AT) or hazardous air pollutants (HAPs) in an urban area vary spatially and temporally. Toxic hot spots, which are the areas that experience high levels of air toxics, are impacted by local sources as well as by the secondary production of many air toxics compounds due to photochemistry and long range transport of air toxics [38-42]. For example, formaldehyde and acetaldehyde have secondary components produced due to oxidant photochemistry. Local-scale models, such as the Industrial Source Complex (ISC3) Dispersion Model [43], AMS/EPA Regulatory Model (AERMOD) [44], and the CALPUFF Dispersion Model [45], do not have in-built chemical mechanisms to account for the production of secondary components due to photochemistry; ISC3 and AERMOD are not recommended for

assessing the impacts for distant sources. Therefore, regional-scale modeling is essential to capture the secondary production and long-range transport of air toxics compounds.

The EPA’s Community Multi-scale Air Quality (CMAQ) [46] model is a regional-scale model that is widely used to study the spatial and temporal variability of air toxics (AT) or hazardous air pollutants (HAP) for identifying toxic hot spots [38-42]. CMAQ modeling system has recently been used in a number of air toxic studies [38-42]. Air toxics version of CMAQ utilizes a modified Carbon Bond IV chemical mechanism commonly used in the CMAQ modeling system to explicitly treat a number of gas-phase air toxics compounds. Resulting modeling system is generally identified as CMAQ-AT. Furthermore, CMAQ-AT has the ability to track primary and secondary components of the carbonyl compounds – formaldehyde, acetaldehyde, and acrolein. Primary species are due to direct emissions from sources while the secondary species result from photochemical reactions. Secondary formaldehyde results especially from the reaction of isoprene with the hydroxyl radical. Figure 6.1 shows the annual time series of the primary and secondary formaldehyde for the central Philadelphia grid cell. Primary formaldehyde varies less on a seasonal basis than the secondary species. Furthermore, the contribution of the primary species to total formaldehyde is typically larger than that of the secondary species during the winter months, but the opposite is true during the summer months. This behavior is attributed to the increase in photochemical activity during warm, sunny periods.

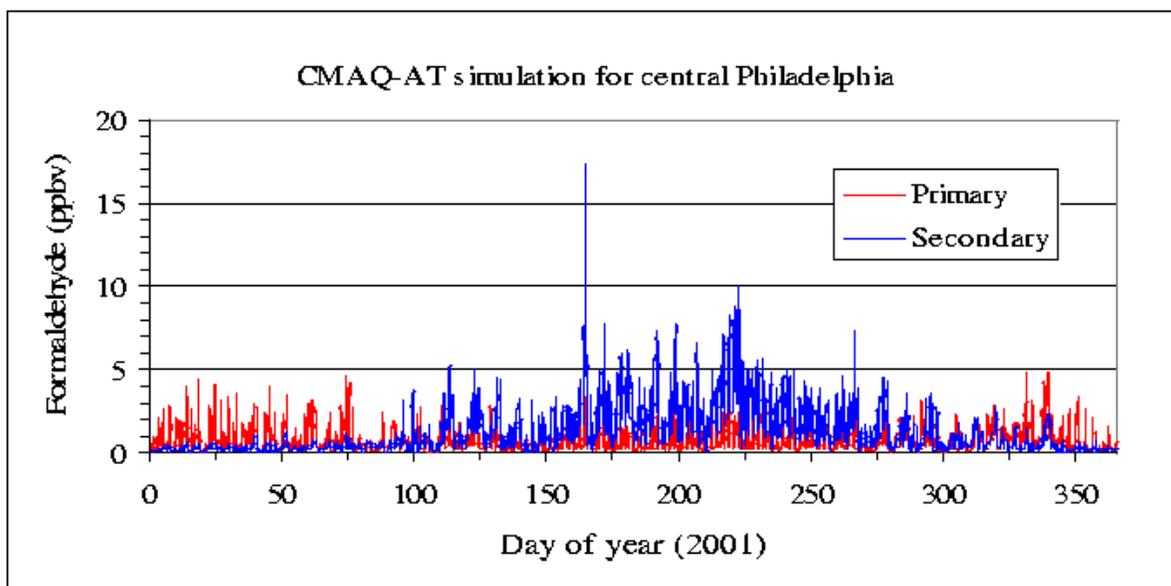


Figure 6.1: Annual variability in primary and secondary formaldehyde as modeled by CMAQ-AT at a 4 km central Philadelphia grid cell

6.2 Delaware prototype study

The EPA/National Exposure Research Laboratory (NERL) has been pursuing its Community Multi-scale Air Quality (CMAQ) modeling system at neighborhood scales to drive the human exposure models for risk assessment due to air toxics. As part of its research program, EPA/NERL has been investigating the application of air toxics version of CMAQ to perform

annual simulations at grid resolutions of 36 km, 12 km, and 4 km with 12- and 4-km nests centered over Philadelphia. The EPA/NERL and DNREC/AQMS agreed to extend the scope of the Philadelphia Study to investigate CMAQ's capabilities to assist in air toxics modeling assessment at fine scales. EPA/NERL, EPA/Region III, and DNREC/AQMS signed a memorandum of collaboration in March 2004. To assist in this model activity, EPA/NERL expanded the 4 km modeling domain to encompass the state of Delaware, and it was decided that the CMAQ modeling system would be exercised at a fine scale of 1 km grid resolution. The source distribution, photochemistry, dry and wet deposition of the air toxics compounds are scale dependent. Therefore, in order to capture the spatial and temporal variability of these compounds and to identify air toxic hot spots, it is necessary to perform air quality simulations at fine scales.

The CMAQ modeling system has been selected because it can perform multiscale and multipollutant simulations for air toxics at a neighborhood scale of 1 km. Modeling for this prototype study was based on 1999 emissions and 2001 meteorology. CMAQ'S preprocessors, the NCAR-PSU Mesoscale Model Version 5 (MM5) [47] and the Sparse Matrix Operator Kernel Emissions Model (SMOKE) [48] provided the meteorological and emission fields respectively, at 1 km grid cell size. 1999 National Emissions Inventory (NEI) and 2001 meteorological databases are chosen for this study. The simulations are assessed to examine the spatial and temporal variability of air toxics compounds, the characteristics and nature of hot spots and their scale resolution requirements. The Carbon Bond IV chemical mechanism commonly used in the CMAQ modeling system has been modified to explicitly treat a number of gas-phase air toxics compounds. The resulting modeling system is identified as CMAQ-AT, and it permits us to model ozone as well as air toxics compounds.

Modeling from regional to finer scales is accomplished by nesting of CMAQ-AT. Simulations for this study are focused on Delaware area. Figure 6.2 shows Delaware modeling domain at 1 km grid resolution nested within the 4 km modeling domain. MM5 and CMAQ-AT simulations have been performed for the entire calendar year of 2001 using nests with 36, 12, and 4 km grid sizes at successively smaller domains, respectively, to provide the initial and boundary conditions for the simulations at 1 km grid size [38]. Simulations with 1 km grids were not performed on an annual basis; however, a one month period (July 2001) for operational purposes was selected for the 1 km simulations to serve as a complement to the annual coarser grid size runs. A one month period is longer than a typical meteorological episode and serves as a surrogate for representing "seasonal" outputs and still be operationally expedient consistent with current computational speed and storage capabilities. For the 1 km grid runs, the emissions were spatially resolved for 1 km grids for the month of July 2001. CMAQ-AT produced enhancement in spatial structure and concentrations of various pollutant species such as ozone, CO, NO_x, and HAPs.

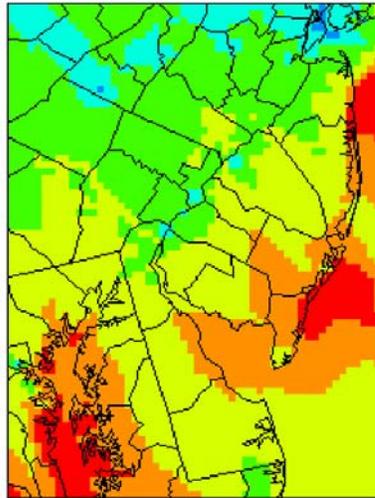


Figure 6.2: Two different modeling domains (4 and 1 km) for Delaware prototype study

The next set of Figures are based on the 1 km simulations. CMAQ-AT tracks primary and secondary species of three carbonyl compounds - formaldehyde, acetaldehyde and acrolein. Primary species are the result of direct emissions from both anthropogenic and natural sources, whereas the secondary are the result of photochemical reactions. Figures 6.3a and 6.3b show that the primary formaldehyde emissions are negligible, and that most of the formaldehyde is due to the secondary species. Isoprene reaction with hydroxyl radical contributes significantly to the secondary formaldehyde during the summer season (Figure 6.3c). Note that formaldehyde hotspots occur where isoprene emissions are significant (Figure 6.3b). The secondary component is likely to be considerably smaller in the winter periods when photochemical activity is diminished [39]. CMAQ-AT predictions for acetaldehyde are similar to formaldehyde, see Figures 6.4a and 6.4b. The peaks in secondary acetaldehyde occur where the concentrations in benzene (Figure 6.4(c)) and CO (Figure 6.4(d)) are high, which can be attributed primarily to emissions from mobile sources. This indicates that the peaks of secondary acetaldehyde are related to mobile source emissions. Figure 6.4(b) indicates that the acetaldehyde hotspots occur where benzene emissions are significant. The behavior of acrolein (Figure 6.5) is different from formaldehyde and acetaldehyde. The primary contribution to acrolein dominates; primary acrolein is attributed primarily to mobile source emissions. Figure 6.5(a) indicates that acrolein hotspots are generally collocated where benzene emissions are high (Figure 6.4(c)). A comparison of Figures 6.3 – 6.5 indicates that formaldehyde and acetaldehyde are impacted by long-range transport, while acrolein more from local contributions.

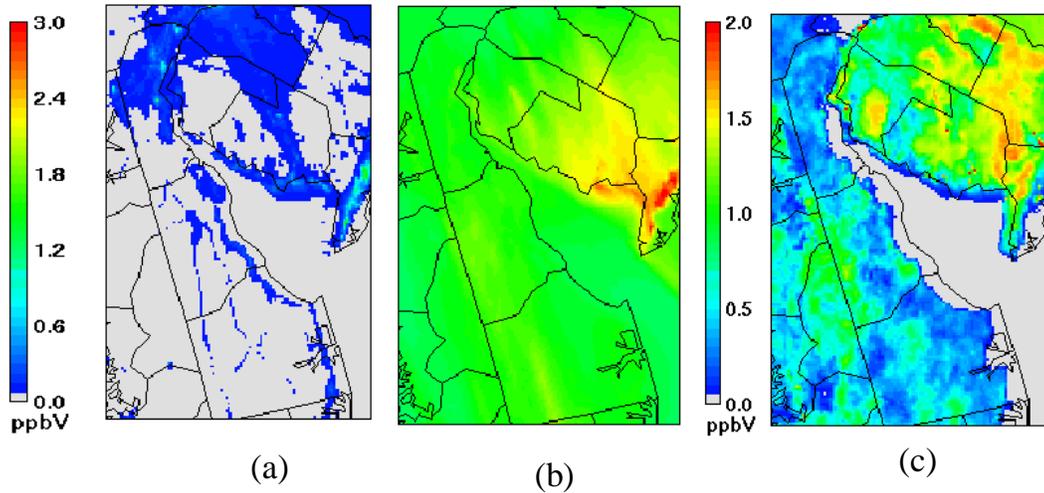


Figure 6.3: (a) Primary formaldehyde, (b) Secondary formaldehyde, and (c) Isoprene at 4:00 PM EST (July 2nd, 2001)

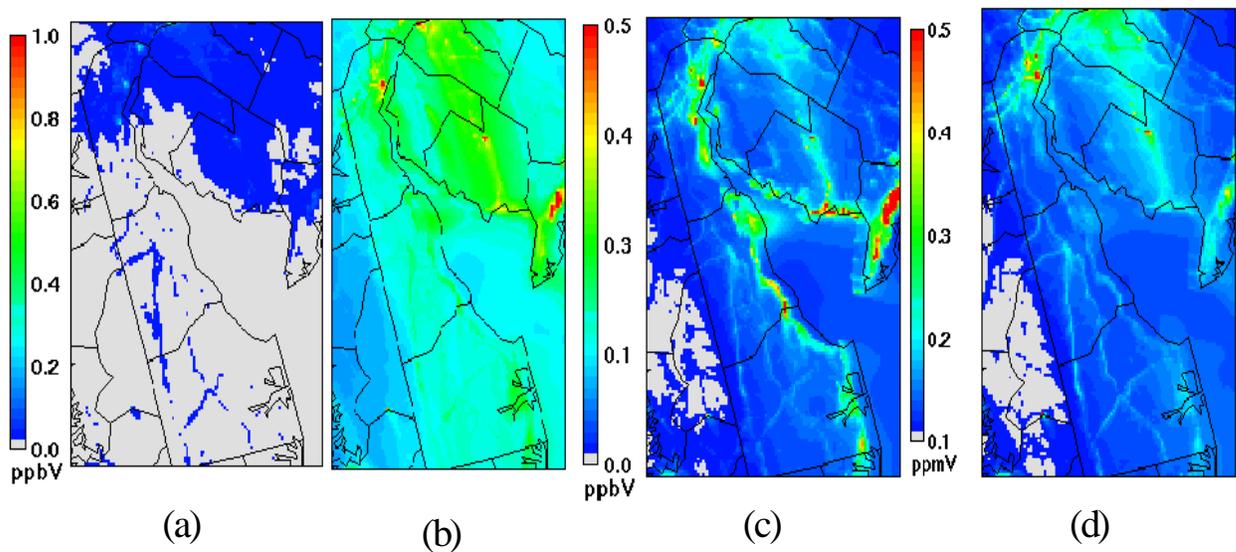


Figure 6.4: (a) Primary acetaldehyde, (b) Secondary acetaldehyde, (c) Benzene, and (d) CO (5:00 PM EST July 2nd, 2001)

Regional-scale models estimate uniform concentration fields within a receptor grid of several square kilometers resulting from multiple emission sources: point, area, mobile, and biogenic. DATAS proposes to apply and test two grid-based models for regional-scale modeling – CAMQ [39] and CMAQ (core part of Models-3 [40]). Both CAMx and Models-3 (June 2002 release) use the SAPRC-99 [41] chemical mechanism modified to incorporate toxic chemistry. The CAMx has been traditionally applied for ozone modeling, while Models-3 represents the

state-of-science model that has been developed by EPA. The mechanism designated as SAPRC-99 is a complete update of the SAPRC mechanism released in 1990. Condensed versions of the SAPRC-99 mechanism have been developed for use in air quality modeling.

Local-scale models provide detailed concentration fields near emission sources. We recommend the use of more than one model to estimate ambient concentrations – they are ISCST [44], AERMOD [45] and CALPUFF [46] for point and arcs, and CALINE [46] for mobile sources.

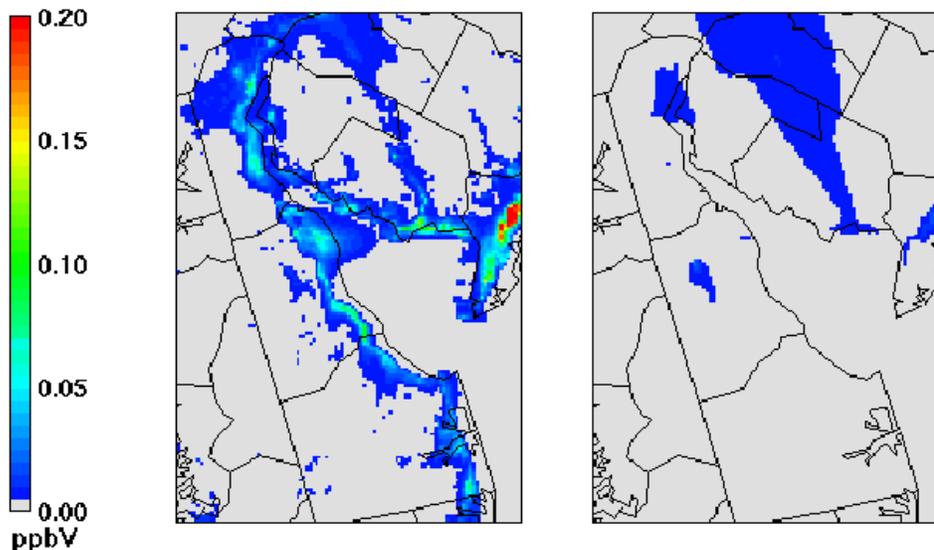


Figure 6.5:(a) Primary acrolein, (b) Secondary acrolein

This prototype study with CMAQ-AT fine-scale modeling demonstrated that fine-scale modeling captures spatial and temporal variability of the air toxics compounds, and also fine-scale modeling provides the spatial gradients and concentration magnitudes which help identify and characterize the hot spots of air toxics compounds. The CMAQ multiscale simulation results provide evidence of the important role of photochemistry in modeling of the carbonyl species. Secondary components of formaldehyde and acetaldehyde dominate the primary component during the summer months.

Model performance for CMAQ is assessed by evaluating the modeling results for a number of species against the monitored data; performance results for a number of species at Lums Pond are presented below. Time series of the monitored isoprene, and CMAQ modeled isoprene concentrations from 4 km and 1 km grid resolution modeling are shown in Figure 6.6. CMAQ model reproduces the diurnal pattern observed in the monitored isoprene data. It is not able to produce, however, the same levels in the peaks observed in the monitored data.

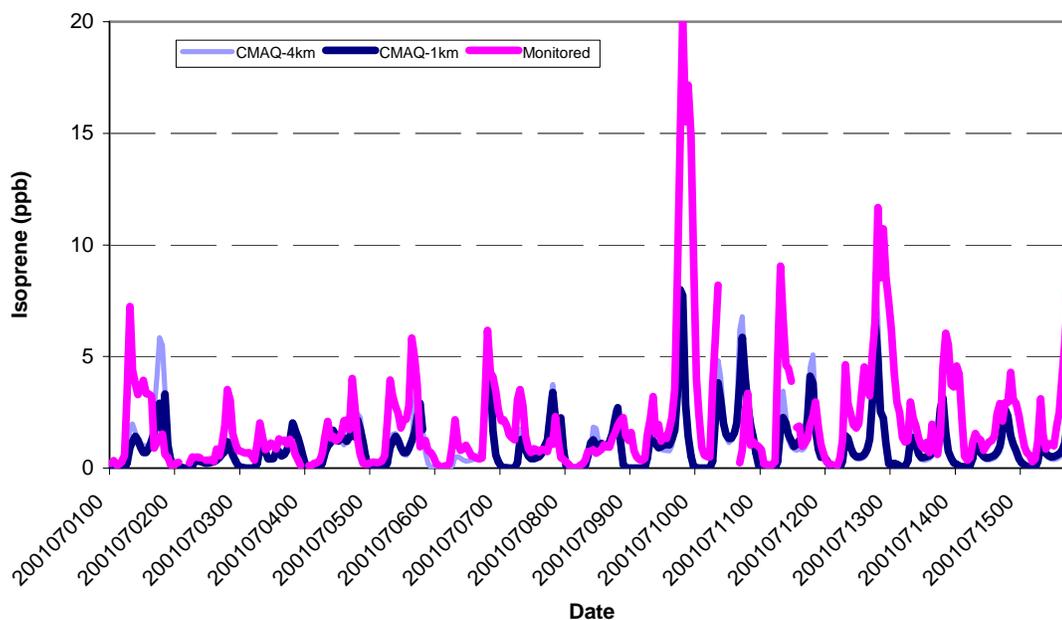


Figure 6.6: Time series for modeled and monitored isoprene at Lums Pond

Time series of the monitored benzene, and CMAQ modeled benzene concentrations from 4 km and 1 km grid resolution modeling are shown in Figure 6.7. Even though CMAQ model reproduces the diurnal pattern observed in the monitored benzene data, it constantly under predicts benzene. Since the model performance for ozone and isoprene is satisfactory, this may indicate that there may not be a concern with the model but the under prediction might be due to under estimation of benzene emissions in the 1999 emissions inventory.

This modeling exercise helped us identify some possible abnormalities in the monitoring data. Figure 6.8 shows time series for monitored formaldehyde at five monitoring stations – Delaware City (DC), Killens Pond (KIL), Lums Pond (LP), Martin Luther King Blvd. (MLK), and Seaford (SE). Formaldehyde observed at Seaford monitoring stations is the highest. Modeling, however, seem to indicate that it should be high at MLK not at the Seaford site. It is not clear yet whether it is an unidentified source close to the monitor or an artifact that is contributing to high formaldehyde readings at the Seaford site.

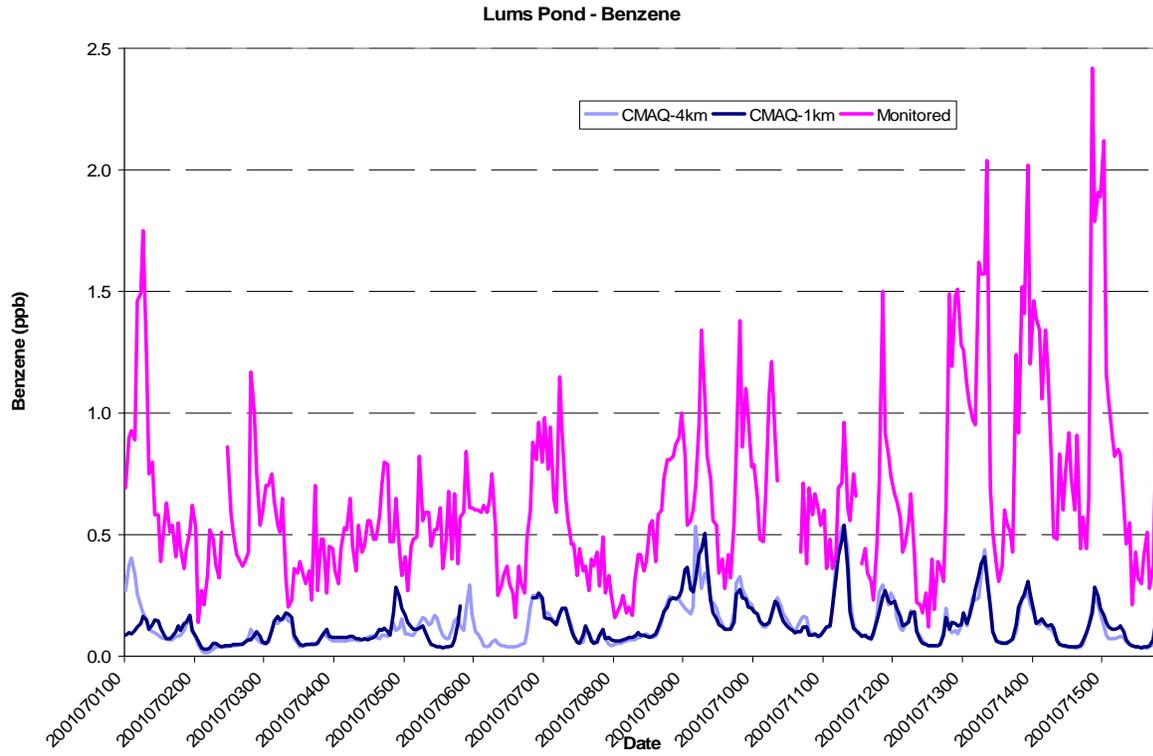


Figure 6.7: Time series for modeled and monitored benzene at Lums Pond

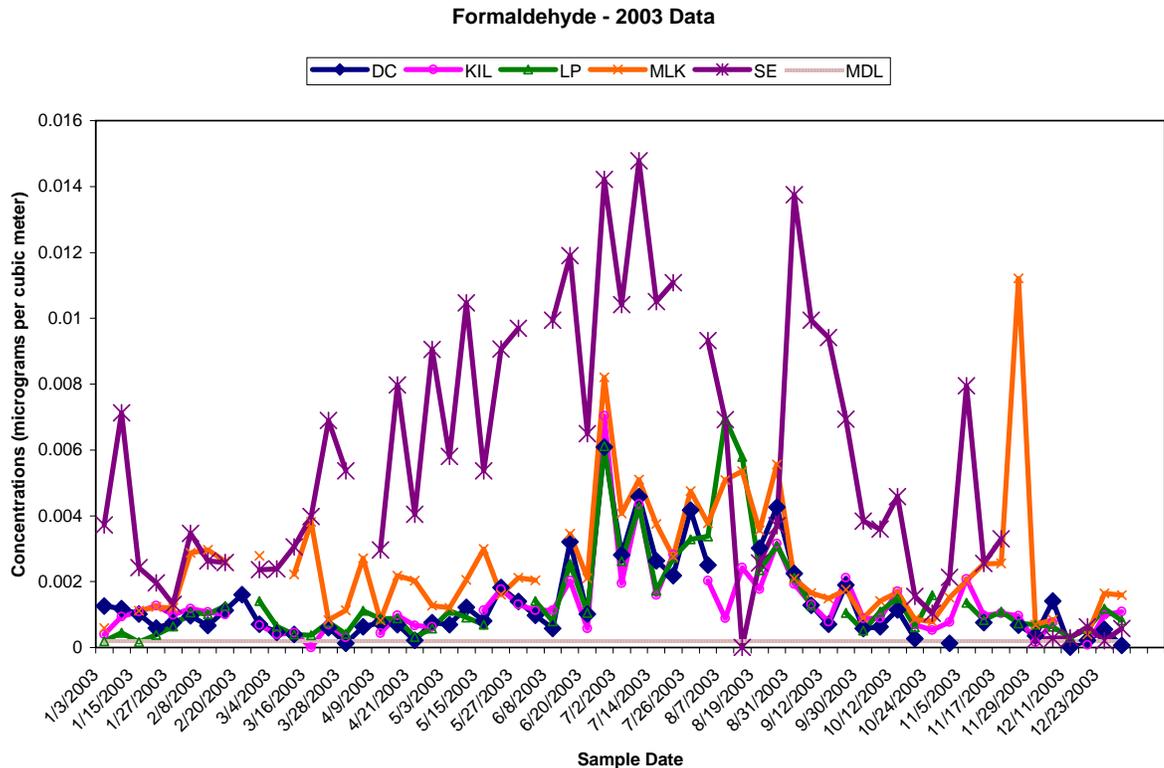


Figure 6.8: Time series for 2003 monitored formaldehyde at 5 monitoring stations

6.1 DATAS modeling approach

As discussed above, recent modeling studies for assessing the impact of air toxics [38-42] and the experience gained from the Delaware Prototype Study [38] indicated a modeling approach that would require both regional- and local-scale modeling. Local-scale modeling will be used to predict impacts for receptors located near emission sources, and the regional-scale modeling for assessing long-range transport and the secondary production of compounds due to photochemistry.

Because the EPA-identified list of 188 HAP compounds is too comprehensive to be modeled, DATAS selected to model more than 33 urban HAPs for both local- and regional-scale modeling, which contribute to the majority of the health risk. The list of HAPs to be modeled will consist of the 33 urban HAPs consisting of volatile organic compounds (VOCs) and semi-volatile organic compounds (SVOCs), carbonyl compounds, and metals, and also the compounds that are identified as of importance to EPA Region III states based on a recent study [49]. The compounds that will be modeled are flagged in a table given in Appendix A. For regional-scale modeling it may not be possible to model all these flagged compounds because of the limitations of the chemical mechanisms adopted in the regional-scale models and the computational demands, and therefore, regional-scale modeling will be limited to the compounds that were modeled in the Delaware Prototype Study.

6.3.1 Local-scale modeling

For the purposes of this study, local-scale study areas are chosen based on population density, roadway network, industrial density, location of monitoring sites, and sensitive receptors such as schools, hospitals, etc. Based on the above criteria, AQMS selected three communities in the New Castle County, and one each in the Kent and Sussex Counties. The selected modeling domains in each of the counties are as depicted in Figure 1.1. Each of the domains will cover an area of approximately 10 x 10 km, and will include a network of receptors placed at 250 – 500 m apart. Depending upon the availability of time and resources, one or more of local-scale models (ISCST [43], AERMOD [44] and CALPUFF [45]) will be used to assess the impact of HAPs emissions. The local-scale modeling study will utilize the meteorology data - hourly surface observations from local airports in Delaware and upper air data from Sterling, Virginia. Local-scale models have the ability to model emissions from point, stationary area, and mobile sources, but not the natural/biogenic emissions. Therefore, local-scale modeling will be limited to emissions from point, stationary area, and mobile sources.

6.3.2 Regional-scale modeling

Regional-scale models estimate uniform concentration fields within a receptor grid of several square kilometers resulting from multiple emission sources: point, area, mobile, and biogenic. Depending upon the availability of time and resources, one or both of CMAQ [46] or California Photochemical Grid Model (CALGRID) will be utilized for this Study. A nested-grid approach will be adopted for regional-scale modeling of meteorology and air quality. The outer modeling domain will extend beyond the state boundary lines, with the inner domain centered on Delaware. The outer domain is defined based on a number of factors - the transport of pollutants from distant sources, capture of major emissions sources, reasonable turn around time of the modeling runs, etc. The size of the domain, however, is compromised by the model selected and resources available.

A limitation of the regional-scale air quality models is their inability to simulate an entire year of data for meteorology. The photochemical grid models CMAQ [47] or CAMx [50] are resource intensive, and therefore, are generally modeled for selected episodes or seasons. Simulating these models for an entire year of meteorology is impractical. CALGRID, however, is less resource intensive and is likely to allow us to model an entire year of meteorology. Therefore, the size of the domain and the length of simulation for the 2003 meteorology are dictated by the model we choose for the simulations. Depending upon the time and resources available, CALGRID or CMAQ will be used for regional-scale modeling of this Study.

The meteorological inputs for the photochemical grid models will be developed by the meteorological models – CALMET [51] or MM5 [47]. CALMET will be used to generate meteorological data for CALGRID and MM5 to generate data for CMAQ. CALMET is a diagnostic model and is the simpler of the two models. It requires inputs from observational data for surface and aloft winds as well as temperature data and generates three-dimensional meteorology fields to drive air quality models. The surface and aloft data from the National Weather Service (NWS) stations, all other surface stations and airports, and upper air rawinsonde data are the inputs to the CALMET model. The model performance of CALMET can also be

improved by initializing it with MM5 output. MM5 is an advanced state-of-science prognostic meteorological model that solves the conservation equations to simulate winds and temperatures. The MM5 model will be initialized from the files generated by the National Climate and Environmental Prediction (NCEP) center and National Center for Atmospheric Research (NCAR).

Boundary conditions for both regional-scale models will be determined from all available data and special modeling studies. Performance evaluation for each model is necessary to ensure that the models are working properly and that modeling results are reliable. Model performance will be evaluated to compare model estimates of concentrations with measured hourly concentrations for criteria pollutants and toxic pollutants where measurements are available. Standard statistical techniques, such as bias and gross error, will also be calculated for annual model estimates as well as for monthly and seasonal averaging times.

6.3.3 Emissions inventory and emissions modeling

A comprehensive air toxics emissions inventory has already been developed for the calendar year 2003, which is the year of study for DATAS. Spatial and temporal allocation of emissions inventory is a necessity for regional- and local-scale modeling. Emissions will be spatially disaggregated using gridded surrogates. MCNC's Sparse Matrix Operator Kernel Emissions [48] (SMOKE) for regional-scale modeling and Emissions Modeling System for Hazardous Air Pollutants (EMS-HAP) [52] for local-scale modeling or other appropriate emissions processing tools will be used for processing of emissions from point, area, mobile and biogenic sources.

Section 7

Communications

DATAS represents the most comprehensive study of its kind in the state, therefore, communications for the study include not only the initial outreach for the five monitoring sites (Phase I), but also a comprehensive statewide outreach effort for the modeling results (Phase II). As both carcinogenic and non-carcinogenic risks are addressed in the final study, outreach will be focused as a coordinated effort between DNREC, DPH, and the Cancer Consortium to establish priorities for the state.

The scope and results of DATAS will be communicated to the public in conjunction with DPH and the Cancer Consortium to build community awareness and involvement. In addition, as part of the risk management plan (RMP), an advisory committee and community-based stakeholder groups will be developed consisting of informed participants (environmental and civic groups). Community workshops/informational forums will be held, during which DNREC will discuss the risk associated with air toxics and ways to decrease risk, as well as solicit responses and ideas about DATAS. Voluntary action materials for public involvement will be developed to decrease emissions of, and exposure to, air toxics. Long-term communications and outreach will include building air toxics reduction partnerships with established organizations and community groups (e.g., Wilmington Area Planning Council [WILMAPCO], American Lung Association of Delaware [ALADE], Community Involvement Advisory Council [CIAC], etc.).

7.1 Community outreach

Effective communications explaining the purpose, method, and results of the DATAS project are important to the success of the project. Working in cooperation with DPH and the Cancer Consortium, DNREC will focus outreach efforts for Phase I on the monitoring station sites and the surrounding communities. Community workshops/informational forums will be held in these areas to present the results of Phase I, and develop an understanding of the entire study and the upcoming Phase II results. As part of the planned outreach for both phases, DPH will develop fact sheets on the specific pollutants. In addition, a comprehensive fact sheet summarizing the DATAS program will be developed for general distribution. Local legislators, and municipal and county officials will be briefed prior to conducting the community workshops/informational forums for Phase I. In conjunction with targeted outreach to communities, information will also be made available to the general public through press briefings and releases at key project junctures.

Outreach and communications for Phase II will focus on the modeling results for the state. Outreach efforts will continue to be coordinated with DPH and the Cancer Consortium to establishing long-term outreach priorities and air toxics reduction partnerships. An internet website and a mail/e-mail list for interested parties and community organizations will be

developed. Public workshops/informational forums for Phase II will be conducted in each of the three counties. Other opportunities for outreach are incorporated in the RMP (Section 9.4.3).

7.2 Communications timeline

Communications for the DATAS project will be completed quarterly over the next year (Table 7.1). As part of Phase I, outreach efforts will introduce the project and provide the monitoring and risk assessment results for the five monitoring stations to the surrounding communities. The outreach efforts for Phase II will focus on providing the statewide air quality modeling and risk assessment results to the public, and building community awareness and air toxics reduction partnerships.

Table 7.1 Communication and outreach activities for DATAS

Activity	Lead	Dates
Internal Meetings on Communications and Reporting (Phase I)	AQM/DPH/OTS	Spring 05
Review PowerPoint Presentation and Outline Display Materials	AQM/DPH/OTS	Spring 05
Internal Presentations/ Governor's Office/Cancer Consortium <ul style="list-style-type: none"> - DAWM Director/Agency Secretaries/Office of the Governor - Cancer Consortium – Environ. Subcom. Meeting - DNREC Enforcement 	AQM/DPH/OTS	Spring/Summer 05 (April 25, 2005) (August 15, 2005)
Prepare Executive Summary Report for Phase I	AQM	Summer 05
Develop Fact Sheets Develop Related Distribution Materials <ul style="list-style-type: none"> - Map of Study Area - Chemicals Compound Information - Isopleth Maps/Related Information - Charts/Graphs - Diesel Information - Explanatory Materials for Risk Colors Create News Releases, Media Stories	DPH/AQM AQM	Summer/Fall 05
Risk Communication Training for Staff	DAWM/DPH	Summer 05
Develop Neighborhood Outreach (Phase I) <ul style="list-style-type: none"> - Letters to Local Groups and Civic Associations - Add Legislators/Interested Persons and Local Governments to list of Neighborhood Contacts) Develop Display Materials	AQM	Fall /Winter 05
Local Community Workshops/Informational Forums (Phase I)	AQM/DPH/OTS	Winter 05
Develop Website Area for DATAS and EPA Data Links	AQM/ITO	2006
External Workshops/Informational Forums, (As Needed) For example: <ul style="list-style-type: none"> - Interested Local Groups and Civic Associations - Counties, Local Municipalities - Community Involvement Advisory Council (CIAC) - Urban Health Environmental Learning Project (UHELP) - Other Stakeholder Groups 	AQM/DPH	2006

Activity	Lead	Dates
Internal Meetings, Preparation of Presentations for Phase II	DAWM/DPH/OTS	2006
Prepare Summary Report for Phase II	AQM/DPH	2006
Phase II Workshops/Informational Forums - Governmental Agencies/Interested Groups (Focus on Air Toxics Reduction Partnerships) For example: -Spring State and Territorial Air Pollution Program Administrators / Association of Local Air Pollution Control Officials (STAPPA/ALAPCO) -Environmental Education Seminars -Chemical Industry Council (CIC) -Odor Roundtable Group -DeIDOT -WILMAPCO -American Lung Association of DE -Mid-Atlantic Regional Air Management Association (ARAMA)	DPH/AQM	2006/2007
Additional Outreach Implemented through RMP	DAWM/DPH	2007

- AQM – Air Quality Management Section of DNREC’s DAWM
- DAWM – Division of Air and Waste Management
- DPH – Division of Public Health (Delaware Department of Health and Social Services)
- ITO – Information and Technology Office (DNREC’s OTS)
- OTS – Office of the Secretary (DNREC)

Section 8

Conclusions

This section provides overall conclusions that can be drawn from the work presented in this report. These are based on the DATAS efforts in air toxics monitoring measurements, emissions inventory estimates, prototype modeling work, and the health risk analyses. They are summarized in the following groups.

8.1 Monitoring

- High sample collection rate for air toxics monitoring is achieved. For all five classes of chemicals monitored at each of our monitoring location, valid sample collection rates ranged from 68% to 95%.
- Toxics concentration levels for many samples, especially PAHs and dioxins/furans, are far below MDL such that they could not be quantified.
- Urban Wilmington site generally shows the highest concentrations for most air toxics compounds.
- Preliminary comparison of results for selected compounds shows the range of monitored data to be comparable to results reported for other urban sites in the UATMP.
- A few compounds display seasonal variations: benzene, 1,3 butadiene, and tetrachloroethylene show wintertime maximum and summertime minimum concentrations while formaldehyde shows summertime maximum and wintertime minimum concentrations.
- Continued monitoring for VOCs, carbonyls, and metals is needed in order to develop sufficient datasets for more detailed evaluation of trends, spatial/temporal analyses, and in-depth exploration of associations with other datasets such as emissions and wind trajectories.

8.2 Inventory

- Mobile sources (on-road and off-road) account for two-thirds of the mass of emissions of reported DATAS chemicals,
- Mobile sources account for the majority of VOC and carbonyl emissions,
- Point sources account for a majority of the metal emissions,

- Residential wood combustion and the several open burning categories dominate the emissions of PAHs and D/Fs,
- Significant inventoried sources of the risk driver pollutants are the following

Emission Inventory Sources of Risk Drivers (Cancer and Non-cancer)

Chemical	Significant Source Contributor
Benzene	Mobile sources
1,3-Butadiene	Mobile sources
Carbon Tetrachloride	No large or widespread sources; appears to be background
Chromium (hexavalent)	Point and area sources; 35% Cr 6+ to total Cr appears an overly conservative ratio for Delaware
Manganese	Road dust and point sources (large combustion units)
Trichloroethylene	Mostly area sources and one point source
1,2,4-Trimethylbenzene	Typically from mobile sources (not inventoried)
Vinyl Chloride	Point sources at Delaware City

8.3 Modeling

- Regional-scale modeling plays an important role in correctly estimating concentrations of carbonyl compounds,
- Regional-scale modeling is essential to addressing the long-range transport of pollutants,
- High resolution regional-scale modeling can capture spatial and temporal variability of the air toxics compounds, and possibly identify and characterize hot spots,
- Although model performance for many of the air toxic compounds show satisfactory trends, a few need further improvement.

8.4 Risk assessment

- Risk assessment of monitoring results from the first phase of DATAS estimates that there is no chemical present in sufficient concentration in the ambient air in Delaware which exceeds the HSCA defined risk level of one additional cancer case per 100,000 people. This means that beyond the usual occurrence of cancer, long-term exposure to the chemical would not cause more than one additional case per 100,000 people.
- The cumulative risk, summed for all chemicals with viable data, on a per site basis, for the five monitoring locations in Delaware estimates that the total risk falls into the increased risk category (yellow color of the tables) of between one and ten

additional cancer cases per 100,000 people. The highest estimated cumulative risk was 4.3 additional cases per 100,000 exposed people.

- Risk assessment of monitoring results performed on chemicals with potential for non-carcinogenic effects indicate that there is no single chemical with a hazard quotient greater than one. An exceedance of a hazard quotient of one would result in increased level for adverse health effects.
- The hazard quotient summations, for each site, for estimated adverse health effects posed by chemicals with the potential for non-carcinogenic effects showed some sampling locations with a hazard index greater than one. This indicates for the total non-cancer effect an increased adverse health effect.
- Existing programs seem to have provided a reasonable level of protection for human exposure to individual compounds. This picture however, may change as the study expands its geographical scope and expands the list of compounds during its further monitoring and modeling phase.

Section 9

On-going Activities

This section describes continuing DATAS activities, some of which extend beyond the scope of the Phase I report. These activities will be completed for the Phase II report in 2006.

9.1 DATAS modeling activities and timeline

As discussed earlier in Section 6, recent modeling studies for assessing the impact of air toxics [38-42] and the experience gained from the Delaware Prototype Study [38] indicated a modeling approach that would require both local- and regional- scale modeling. Therefore, for DATAS, local-scale modeling will be used to predict impacts for receptors located near emission sources, and the regional-scale modeling for assessing long-range transport and the secondary production of compounds due to photochemistry.

9.1.1 Timeline for DATAS modeling

Major activities of DATAS modeling are - meteorological inputs for applications in regional- and local-scale modeling, emissions processing for regional- and local-scale air quality modeling, regional- and local-scale air quality modeling, model performance and quality assurance, documentation. The projected timeline for these activities are presented in Figure 9.1.

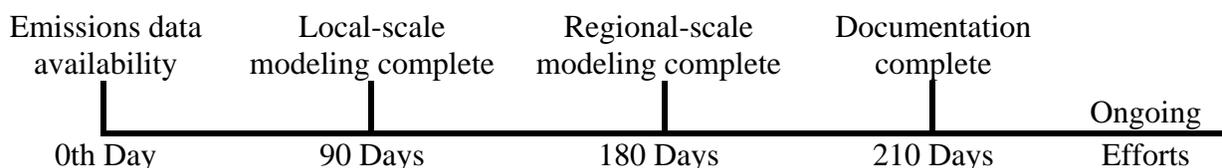


Figure 9.1: Projected timeline of modeling activities for 2003 DATAS

The above projected timeline does not provide enough time to complete local- and regional-scale modeling using more than one model for each. DATAS modeling is likely to identify a number of issues, which may require resolving differences in monitoring and modeled data, finding ways to improve emissions inventories and modeling, refining the modeling to improve the results, etc. The timeline identified above does not account for the time needed for addressing these issues. Therefore, additional 6 to 9 months will be required to address these issues, and also to perform local- and regional-scale modeling with the models that have been identified but not utilized.

9.2 Risk assessment activities

Section 4 described the methodology that the Division of Public Health employed to conduct the risk assessments for Phase I of DATAS. Those risk assessments used 2003 monitored concentrations observed at the five monitoring sites described in Section 2. The risk assessment results for these five sites have been presented in Section 5.3.

During Phase II of DATAS, the Division of Public Health will conduct additional risk assessments based on model generated toxics concentrations in Delaware, employing the methodologies described in Section 4. As with the Phase I of DATAS, the Phase II risk assessments will determine the potential cancer and non-cancer health effects using the three scenarios of adults, children and the age-adjusted population.

The notable differences between the Phase I and Phase II risk assessments are as follows:

1. The concentrations used in the Phase II risk assessments will be generated from models as described above, rather than monitoring results,
2. The number of potential areas for which risk assessments may be performed could be numerous, rather than limited to the finite number of monitoring sites,
3. The chemicals or chemical compounds included in the Phase II risk assessments could be broadened to include chemicals for which both 2003 emissions inventory and qualified risk information exists.

Risk assessment activities will commence as soon as the modeled information becomes available. The projected timeline for these activities are presented in Figure 9.2.

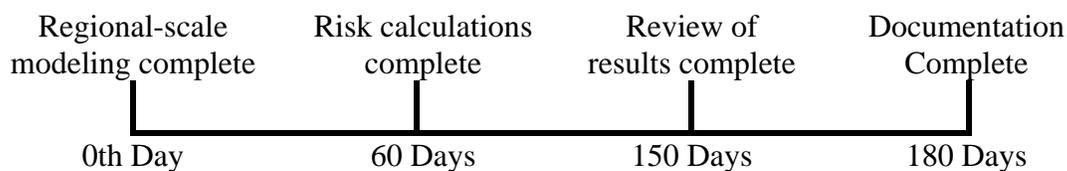


Figure 9.2: Projected timeline of risk assessment activities for 2003 DATAS

9.3 Communications and outreach activities

Section 7 described the outreach and communication efforts underway to build community awareness and involvement based on the risk results for the five monitoring sites (DATAS-Phase I). This section addresses the outreach and communication activities that will be undertaken for Phase II. In addition to supporting DATAS, similar outreach and communication activities are an integral part of the risk management planning activities, described in Section

9.4.3. These activities provide a process for identifying, evaluating, selecting, and implementing actions to reduce risk to human health and to the environment.

9.3.1 Community outreach

Outreach and communications for Phase II will focus on the modeling results for the state. Outreach efforts will continue to be coordinated with DPH and the Cancer Consortium, as described in Section 7, to establishing long-term outreach priorities and air toxics reduction partnerships. An internet website and a mail/e-mail list for interested parties and community organizations will be developed. Public workshops/informational forums for Phase II will be conducted in each of the three counties. Other opportunities for outreach are incorporated into the risk management planning activities (Section 9.4.3).

9.3.2 Communications timeline

The outreach efforts for Phase II will focus on providing the statewide air quality modeling and risk assessment results to the public, and building community awareness and air toxics reduction partnerships. The DATAS Phase I activities will transition into the communication and outreach activities for Phase II and risk management planning, as shown in the timeline, Figure 7.1.

9.4 Continued AQM air toxics activities and plans beyond DATAS

This section describes ongoing AQM's air toxics activities to reduce the adverse health impacts of air toxics to Delaware residents. In what follows, several activities, planned and practiced, are described towards this goal.

9.4.1 Monitoring activities

AQM will continue to meet the normal monitoring needs of Delaware, as funding allows. Analysis of the ambient monitoring data to date has focused largely on data validation and verification, and can best be considered to be initial descriptive analysis. The work done so far includes time-series descriptions, identification of outliers, and some preliminary comparisons with the Toxics Release Inventory data.

The next phase of data analysis will focus on more in-depth research to generate a complete description of the ambient concentrations in Delaware in 2003. Towards this goal, more thorough statistical analysis will be carried out to identify and characterize seasonal trends for various VOCs and carbonyls, compare and correlate concentration trends among different sites and compounds, perform spatial and temporal variation analysis, and compare with monitored concentrations in other states. Other areas of interest include evaluation of local emissions and meteorological data to examine relationships with monitored concentrations.

In addition to the continuing data analysis, VOCs, carbonyls and metals will continue to be monitored at all sites for the next several years.

During 2005/2006 there will be several special monitoring projects coordinated with the University of Delaware and Duke University to enhance understanding of ambient air toxics concentrations in Wilmington. Some of these will include:

1. Intensive measurements – Single Particle Analysis (University of Delaware)

Ambient single particle measurements will be performed at the MLK site by the University of Delaware with RSMS-3 (rapid single particle mass spectrometer, version 3) during four 1-month intensive periods in 2005-2006. It is expected that on the order of 200,000 particles will be obtained during each period. The tentative monitoring dates are: 4/15-5/15, 7/15-8/15, 10/15-11/15 and 12/15-1/15. The data will provide information on the composition of aerosol particles, including toxic compounds, at several different particle sizes.

2. Additional measurements during Intensive Periods (Duke University)

The Duke University team will perform mobile measurements of formaldehyde and water-soluble chromium species, and total particle count of the aerosol at the monitoring site at Wilmington (DE) for one week during each intensive period. The data will provide information on spatial distribution of these components on a scale of the order of 1 km.

3. Additional measurements in between Intensive Periods (University of Delaware)

Between the intensive periods, the single particle analyzer will remain at the MLK site and be available on an “as-needed” basis to supplement intensive measurements. Examples of measurements include:

- Source sampling – key sources will be characterized by inflating a Teflon bag with ambient air at the source, then transporting the bag back to the instrument for analysis,
- Ambient measurements during unusual PM “events”; examples include road construction, snow/road salt, and unusual point source emissions.

9.4.2 Emission inventory activities

Activities of this section will cover four main tasks.

1. Compare EI data to monitoring results

For this Phase I report the emission inventory results were looked at to determine the mass of emissions for each DATAS pollutant and the source or sources that are the largest contributors of that mass on a statewide basis. The next step is to review the monitoring results to determine if the pollutants observed at the monitors are also quantified within the emission inventory. The suite and concentrations of pollutants seen

at each monitor may be characteristic of a certain source sector (i.e., point sources, stationary area sources or mobile sources) which may be corroborated by the spatial distribution of emissions sources near to the monitoring sites.

2. Feedback from modeling/monitoring comparison

Once the local- and regional-scale modeling is performed, modeled concentrations at the monitoring sites can be compared to the concentrations observed at the monitoring sites. While there are a number of reasons the modeled and the monitored concentrations could be different, one reason could be due to an inaccurate or incomplete inventory. Feedback on certain pollutants will enable AQM to employ additional scrutiny to the inventory to determine areas for improvement.

3. Identify sources contributing to elevated risk

Once the risk assessment has been completed for the modeled concentrations statewide (Phase II), areas of elevated risk (“hot spots”), on a cumulative basis, will be identified and prioritized according to the magnitude of the potential risk. For each hot spot, the pollutant(s) contributing most to the potential risk will be identified. Next, through analysis of the modeling effort, the sources that contribute to the pollutant concentrations at the hot spots will be identified.

4. Perform a 2005 air toxics inventory

Air toxics inventories are needed on an on-going basis to identify emission trends, new sources, and new emissions data (emission factors, models, speciation profiles, etc.). Since Delaware is already required to develop a comprehensive inventory for criteria pollutants every three years, the toxics inventory will be combined with this effort.

9.4.3 Risk management planning activities

With the completion of Phase I of DATAS, AQM has information on the ambient concentrations for well over 89 chemical compounds (air toxics) measured for the five monitoring sites identified in Section 2 (Table A1, Appendix). Using this information, the Division of Public Health (DPH) conducted the cancer and non-cancer risk assessments identified in Section 4.

Anticipating that one or more of these five risk assessments may indicate a potential risk that is unacceptable, AQM has prepared a community-based risk management plan to reduce unacceptable risks. The risk management planning activities are described below.

1. Establish and utilize an advisory committee

The implementation of these risk management planning activities are the first major community-based initiatives undertaken by AQM. To that end, AQM plans to establish an advisory committee structured to provide support and give balanced, expert advice on

healthcare, scientific, community, and funding problems AQM might face while undertaking these activities. In addition, the advisory committee could make recommendations regarding acceptable risks and appropriate actions to reduce unacceptable risks. Members of the advisory committee should represent the following interests.

- State Elected Officials
- Medical Community
- Health Advocates
- Academia
- Umbrella Civic Organizations
- Chamber of Commerce
- State (DNREC, DHSS, DeIDOT) Leaders
- Federal (EPA) Leaders

The advisory committee can be instrumental in identifying the appropriate participants (risk managers and stakeholders) for those communities in which the risk management process will be undertaken.

2. Identify participants and assemble community-based committee

Risk managers are those that have the authority and responsibility to take needed actions. Stakeholders are those affected by the risk posed. Together, these participants bring important information, knowledge, expertise and diverse perspectives to the risk management process.

The initial step in the community-based risk management process is to identify and engage recognized local leaders representing the following interests.

Risk Managers

- Local (City/County) Elected Officials
- Air Quality Management
- Division of Public Health
- Other DNREC and State agencies
- EPA Region 3
- Representatives of Local Industry
- Representatives of Local Mobile Sources

Stakeholders

- Local Community Associations
- Local Civic Associations
- Local Religious Leaders
- Local Interested Citizens
- Local Health Care Providers
- Local Health Advocates
- Local Environmental Advocates
- Media

3. Define the problem in context of the local community

AQM recognizes that the participants have different interpretations about the nature and significance of risk and different levels of understanding of risk assessment practices. This step includes discussions to overcome these differences. The participants will discuss risk, including the role risk assessments play in using science and judgment to draw conclusions about the likelihood of effects to human health. The participants will learn about DATAS and the information that it provides. Likewise, they will learn the limitations of DATAS; i.e., not every chemical included and only the effects from inhalation of outdoor air are being evaluated.

The expected result from these discussions is the establishment of the community-based committee's risk management goals. These goals can include reducing risk to an acceptable level, reducing risk with minimal job loss, protecting the most-sensitive population, etc. These risk management goals serve as benchmarks in future activities in the risk management process.

4. Define the potential risks in context of the local community

In order to identify risk reducing options and make effective risk management decisions, participants need to know the potential harm the chemical and chemical compounds pose to the community, the likelihood that people in the community will be harmed, and the most probable sources of the chemicals of most concern. During this step in the risk management process, the participants will analyze the DATAS risk assessment information in light of the risk management goals developed above. In addition, the relative contribution of the various stationary and mobile sources to the community's risk can be identified using the DATAS modeling information.

The end-product of these analyses is a determination as to whether the community's potential risk of adverse health effects exceeds the acceptable risk identified in the risk management goals, which chemical or chemicals contribute the greater risk and which sources provide the greatest impact. The participants use this information to establish priorities during the next risk management planning activity.

Note – Upon completion of this step, the participants may decide that additional information, e.g. more monitoring is needed before proceeding to the next step.

5. Identify and examine the options for reducing air toxics in the environment

During this step of the risk management process, the participants identify potential options that would effectively control the emission of air toxics from contributing sources. It is likely that many of the chemical or chemical compounds that contribute to adverse health effects are emitted from more than one stationary source. In some instances, some of the chemicals, e.g. benzene, are also emitted from mobile sources.

Typically, regulatory actions are considered for managing or reducing adverse health effects. The community-based committee should consider non-regulatory options during this step, as well. The following are examples of some non-regulatory options that may be appropriate in given situations.

- Voluntary Controls by Sources
- Public Awareness
- Education of Sources
- Education of Health Providers
- Technical Assistance
- Economic Assistance
- Public Health Initiatives
- Pollution Prevention
- Political Action

Participants assess the effectiveness, feasibility, benefits, and cost of the options identified.

The end-product from these analyses is a variety of the activities that would reduce or eliminate various adverse health effects in the community.

Note – As the emission sources are identified and the analyses of the options are completed, it is likely that additional risk managers may be added to the community-based committee.

6. Decide which options should be undertaken

During this decision-making step, the community-based committee reviews the information developed during risk and option identification steps to determine the appropriate options that achieve the risk management goals. In order to arrive at sound risk management decisions, the committee should consider the following.

- Adequacy of the information available
- Multiple sources and chemicals
- Feasibility with a reasonable cost to benefit ratio
- Priority to preventing risks
- Non-regulatory alternatives, where applicable, and
- Incentives for innovation

7. Implement appropriate decision to reduce the adverse effects

Virtually all of the participants in the risk management process could be an action taker, given the variety of risk management activities that could be selected. Activities can range from changing manufacturing operations to influencing traffic or driving patterns to educating communities. Action takers are responsible for implementing and reporting on the following activities.

- Developing and implementing the plan of action
- Explaining the decision and action plan to the community
- Progress made

8. Evaluate the effectiveness of the actions taken

This step is probably the most difficult one in the risk management process. Once the selected activities are completed or in place, the community-based committee should track key indicators to determine whether the expected results (reduced rates of disease or death) are achieved.

As the risk management process strives to reduce chronic (long term exposure to low concentrations) health effects, it is often difficult to identify direct indicators of risk reduction, such as a reduction in the incidences of leukemia. In this case, the committee

needs to identify and monitor indirect indicators, such as ambient concentrations in the community or vehicle miles traveled.

Note – The risk management process is an iterative process. Should direct or indirect indicators ever suggest that the expected results are not being achieved, the committee needs to revisit the risk management process by again defining the risk and identifying and implementing additional risk reducing options.

Upon completion of Phase II of DATAS, AQM expects to extend its ability to identify the potential adverse health effects beyond the five monitoring sites. Risk assessments, again conducted by DPH, will provide calculated probabilities for cancer and non-cancer health effects based on emissions inventory and modeling. AQM will use the same community-based committee approach summarized above in communities subject to unacceptable risks.

9.4.4 Air toxics strategic planning activities

In order to develop and implement risk-based programs to address the adverse health effects posed by the emission of air toxics from both stationary and mobile sources, AQM recently developed its first long-term air toxics strategic plan (ATSP). The 2005 AQM Air Toxics Strategic Plan captured and organized the combined considerations and contributions of management and professionals from the various branches across the AQM Section. This five-year plan, encompassing 2005 through 2009, defines the activities, their timing and responsible persons. The 2005 ATSP is organized into the following six major components.

1. Build greater understanding of the ambient air toxics environment

The monitoring aspects of DATAS were completed in 2004. These monitoring activities provided information on the concentrations of various air toxics in five communities of interest. Future activities in this major area will focus on continuing to improve our knowledge of air toxics present in Delaware's environment, to support the modeling of ambient concentrations in other communities of interest, and to aid in evaluating the success of past and future AQM actions taken to reduce health risks.

2. Identify potential harm from exposure to air toxics

The health risk assessment aspect of DATAS will be conducted by DPH. DPH will assess the quantitative data from the DATAS monitoring and the results from the DATAS modeling. Future activities will focus on improving the risk assessment tools, finding risk assessment alternatives and supporting the various risk management plans (RMPs) implemented in communities of interest, if unacceptable levels of health risk are found. In order to measure progress in reducing health risk, a second DATAS-like project is planned in 2009.

3. Gather information related to air toxics sources

Beginning in 2005, the DATAS risk assessments conducted by DPH will be used to identify those chemicals of concern, i.e. those chemicals that are major contributors of increased health risk. Future activities in this area will focus on identifying and tracking Delaware stationary and mobile sources that emit identified chemicals of concern. Special attention will focus on those sources that are major contributors to elevated health risk for identified “hot spots.” This future information is needed to carry out the various RMPs undertaken in the communities subject to an unacceptably high level of risk.

4. Identify options for reducing air toxics in the environment

Based on the DATAS health risk assessment results, AQM will develop and implement various long-term RMPs involving stakeholders from the affected communities and regulated sources that contribute to the unacceptable risk. In the event that multiple communities are identified, priority communities will be identified and the stakeholders will focus on identifying and implementing risk reducing actions, i.e. voluntary controls, public education, regulatory actions, etc.

Several new categories of risk-based federal regulations are anticipated over the next several years. Unrelated to the RMP process, additional activities will be undertaken to evaluate and communicate the impact of these risk-based regulations on Delaware air toxics and AQM resources.

5. Implement appropriate actions to reduce the harm from exposure to air toxics

In order to minimize unacceptable health risk from exposure to air toxics, the Engineering and Compliance (E&C) Branch will ensure continued or improved compliance by stationary sources by revising operating permits consistent with new air toxics regulations and voluntary controls and through timely compliance inspections. Likewise, the Planning Branch (PB) will continue to work closely with DelDOT to reduce mobile source emissions of volatile organic compounds (VOCs), many of which are air toxics. PB will adopt new and amend existing mobile and stationary source air regulations to further reduce air toxics emissions. Air toxics regulatory actions driven by the various RMPs will be identified, if voluntary reductions are not forthcoming. Educational initiatives will be considered and implemented as a result of the risk assessments to increase the public awareness of actions that the public can take to reduce their exposure, including appropriate Pollution Prevention (P2) programs.

6. Evaluate the effectiveness of the actions taken to reduce harm from exposure to air toxics

AQM will evaluate and determine the effectiveness of the actions taken to date, as well as future actions that are implemented. In addition to air toxics monitoring and emissions inventory reporting for chemicals of concern, a set of “air toxics indicators” will be sought and tracked. As mentioned above, a more focused DATAS project will also be undertaken in 2009.

In addition to these six major planning areas, the ATSP identifies activities needed to further develop the air toxics program and to deploy AQM resources over the course of the next five years.

9.5 Improving the planning process

The development of the air toxics strategic plan (ATSP) discussed in Section 9.4.4 was not an isolated activity. AQM views this as being an on-going process of development, implementation, review, and evaluation. To improve the usefulness of this tool, AQM will broaden the planning process to identify the personnel and funding requirements needed to achieve the ATSP activities, and will seek the participation of outside interests during future planning cycle.

Section 10

Recommendations for Future Work

10.1 Implement monitoring DATAS-identified air toxics

Results of continuing data analyses (including modeling) will guide changes to the monitoring network, including the possibility of short-term monitoring in targeted areas. Future functions could include tracking trends, evaluating impacts on specific communities, and tracking progress of control measures. Availability of resources will also play a role in future monitoring efforts.

10.2 Improve and expand emissions inventory process

Due to inaccuracies with, or lack of, emission factors, speciation profiles, and activity data, it may be necessary to provide for Delaware-specific surveys or other emission inventory studies to accurately characterize emissions for certain source categories. As an example, emissions for the Residential Wood Combustion (RWC) category were derived from a Mid-Atlantic and Northeast regional study, but may be improved if a Delaware-specific survey of residential wood use is conducted. The RWC category was responsible for the largest emissions of both polycyclic aromatic hydrocarbons (PAHs) and dioxins/furans.

10.3 Refine air toxics modeling capabilities

AQM will be developing control strategies for the 8-hr ozone and PM-2.5 State Implementation Plans (SIPs). Many of these strategies are likely to bring incidental benefits to reduce air toxics. Therefore, the future air toxics modeling will attempt to integrate ozone and PM-2.5 modeling efforts so that AQM can assess the synergistic effect impacts of ozone and PM-2.5 control strategies.

10.4 Provide risk assessment support for risk management plan implementations

The Environmental Health Evaluation Branch of DPH will continue to provide risk assessment and health information support for the ongoing activities, including risk management plan implementations.

10.5 Expand and improve air toxics communications

The scope of the communications and outreach activities supporting the DATAS project and the risk management planning activities are described in Section 9.4.3. In addition to these two areas, AQM will work with the advisory committee, community groups, Division of Public Health and the Cancer Consortium to educate communities on adverse health effects. Funding needs may include, but are not limited to, the following:

- Contractual support for future risk assessments and related communication,
- Evaluating and communicating the impact of future risk-based regulations on Delaware air toxics and AQM resources,
- Contractual support for evaluating cumulative risks and associated risk reduction strategies, and
- Evaluating communication needs for the air toxics risk-based permitting process.

10.6 Expand the community-based risk management plan

AQM recommends implementing the process outlined in the development of a risk management framework (Section 9.4.3) where risk assessments prepared by the Environmental Health Evaluation Branch of DPH identify unacceptable levels of risk for adverse cancer or non-cancer health effects. The need to implement multiple community-based plans for the State is likely. Therefore, to the extent that the resources are available, AQM will prioritize the communities with unacceptable risks. Additional funding will be needed to address multiple communities. Funded needs may include, but are not limited to, the following:

- Risk management planning consultation,
- Third-party contracting and facilitation of community-based plans,
- Third-party contracting and facilitation of community workshops/informational forums,
- Meeting refreshments,
- Risk communication training, and
- Development and implementation of additional communication and outreach activities, as needed.

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Appendix

Table A1: A comprehensive list of Air Toxic compounds, monitored, inventoried, modeled and/or used for risk assessment purposes (Y = yes, N = no). Values provided in modeling column relate to Phase II study only.

Chemical Compound	CAS Number	Major Sources/Uses	Monitoring	Emission Inventory	Risk Assessment		Modeling	
					Phase I	Phase II	Local Scale	Regional Scale
Volatile Organic Compounds (VOCs)								
1,1,1-Trichloroethane	71556	Used as a solvent and degreasing agent in metal degreasing agents, paints, glues, and cleaning products, formerly used as a food and grain fumigant	Y	Y	Y	Y	Y	N
1,1,1,2-Tetrachloroethane	79345	Used as a solvent, and in the production of wood stains and varnishes	Y	Y	Y	Y	Y	N
1,1,2-Trichloro-1,2,2-trifluoroethane (CFC-113)	76131	Used to clean metal surfaces, in the manufacture of lubricants and of fluorocarbon resins, and in aerosol formulations	Y	N	Y	N	N	N
1,1,2-Trichloroethane	79005	Used as a solvent and in the production of vinylidene chloride and 1,1-dichloroethane	Y	Y	1	Y	Y	N
1,1-Dichloroethane (Ethylenedichloride)	75343	Used to as solvent, to remove grease, make plastic products, and adhesive applications	Y	Y	Y	Y	Y	N
1,1-Dichloroethene	75354	Used to make polyvinylidene chloride, in flame-retardant coatings for fiber and carpet backing	Y	Y	Y	Y	Y	N
1,2,4-Trichlorobenzene	120821	Used in manufacturers of electronic components, wood preservatives, and in septic tank cleaner	Y	Y	Y	Y	Y	N
1,2,4-Trimethylbenzene (Pseudocumene)	95636	Used in petroleum refining, foundries and plastic manufacturer, detected in motor vehicle exhaust	Y	N	Y	N	N	N
1,2-Dibromoethane (Ethylene Dibromide)	106934	Used as wood preservative, insecticide, solvent for dyes and waxes, it was used as pesticide	Y	Y	Y	Y	Y	N
1,2-Dichloro-1,1,2,2-tetrafluoroethane (Freon 114)	76142	Used in spray cans, in the production of polystyrene and polyurethane foam plastics	Y	N	N	N	N	N
1,2-Dichlorobenzene	95501	Used as a solvent, fumigant, deodorizer, insecticide/fungicide on crops, and chemical intermediate	Y	N	Y	N	N	N
1,2-Dichloropropane (Propylene Dichloride)	78875	Used in manufacture of perchloroethylene and related chemicals. In past, used as a soil fumigant	Y	Y	Y	Y	Y	N
1,3,5-Trimethylbenzene (Mesitylene)	108678	It is present in petroleum and coal tar and is used as an industrial solvent	Y	N	Y	N	N	N
1,3-Butadiene	106990	Released from combustion of gasoline and diesel, biomass combustion, and industrial on-site uses	Y	Y	Y	Y	Y	Y
1,3-Dichlorobenzene	541731	Used to make herbicides, insecticides, medicine, and dyes	Y	N	Y	N	N	N
1,3-Dichloropropene	542756	Predominant component of several formulations used in agriculture as soil fumigants	N	Y	N	Y	Y	N
1,4-Dichlorobenzene	106467	A registered pesticide. Also used in mothballs and in toilet-deodorizer blocks	Y	Y	Y	Y	Y	Y
1-Ethyl-4-methylbenzene	622968	Used in preparation of paints, certain rubbers, and wood furnishings	Y	N	N	N	N	N
2-Methylphenol (o-cresol)	95487	Used as solvent, disinfectant, and chemical intermediate, released from automobile exhaust, power plants, municipal solid waste incinerators, oil refineries, and cigarettes	N	Y	N	Y	Y	N
Acrylonitrile	107131	Used in the manufacture of acrylic fibers, acrylamide, and nitrile rubbers and barrier resins	N	Y	N	Y	Y	N

Chemical Compound	CAS Number	Major Sources/Uses	Monitoring	Emission Inventory	Risk Assessment		Modeling	
					Phase I	Phase II	Local Scale	Regional Scale
Benzene	71432	Generated from various refinery operations, dry cleaning operation and vehicle exhaust	Y	Y	Y	Y	Y	Y
Bis(2-ethylhexyl)phthalate	117817	Used in the production of polyvinyl chloride (PVC)	N	Y	N	Y	Y	N
Bromomethane	74839	An ingredient in some fertilizer and pesticides, also used in fumigation for controlling fungi	Y	Y	Y	Y	Y	N
Carbon Tetrachloride	56235	Used in the synthesis of CFCs, in petroleum refining, pharmaceutical manufacturing, pesticide products	Y	Y	Y	Y	Y	Y
Chlorobenzene	108907	Released from crude petroleum and natural gas extraction and petroleum refining, plastic and synthetics manufacturing	Y	Y	Y	Y	Y	N
Chloroethane (Ethyl chloride)	75003	Used in the production of cellulose, dyes, medicinal drugs, as a solvent and refrigerant. Also used to numb the skin before medical procedures such as ear piercing and as a treatment in sports injuries	Y	Y	Y	Y	Y	N
Chloroform	67663	Released from pulp and paper mills, hazardous waste sites, sanitary landfills, chlorination of water	Y	Y	Y	Y	Y	Y
Chloromethane	74873	Released from industries, cigarette smoke, wood/coal/plastics smoke, and chlorinated swimming pools	Y	Y	Y	Y	Y	N
Cis-1,2-Dichloroethene	156592	Used as a solvent and refrigerant, in the manufacture of rubber, pharmaceuticals and artificial pearls	Y	N	Y	N	N	N
Cis-1,3-Dichloro-1-propene	10061015	Used as farm pesticide and in manufacture of other chemicals	Y	N	N	N	N	N
Dichlorodifluoromethane	75718	Used as a refrigerant, production phase-out	Y	N	Y	N	N	N
Ethylbenzene	100414	Used in the production of styrene, petroleum refining, as a solvent, detected in auto emissions and cigarette smoke	Y	Y	Y	Y	Y	N
Ethylene Dichloride (1,2-Dichloroethane)	107062	Used in the production of vinyl chloride, as solvent, fumigant, degreaser, paint remover	Y	Y	Y	N	Y	Y
Ethylene Oxide	75218	in the manufacture of textiles, detergents, PUF, antifreeze, solvents, adhesives, and other products, as a fumigant, sterilant for food and cosmetics, and in hospital sterilization of surgical equipment and plastic devices, detected in automobile exhaust	N	Y	N	Y	Y	Y
Hexachloro-1,3-butadiene	87683	Used to make rubber compounds, lubricant, as a solvent, and as hydraulic fluid	Y	N	Y	Y	N	N
Hexachlorobenzene (perchlorobenzene)	118741	Was used as a pesticide, in the production of rubber, aluminum, and dyes, and in wood preservation until 1965, currently formed as a byproduct during the manufacture of other chemicals (mainly solvents) and pesticides	N	Y	N	Y	Y	N
Hydrazine	302012	Used in pesticides, pharmaceutical intermediates, photography chemicals, boiler water treatment for corrosion protection, textile dyes, and as fuel for rockets and spacecraft, detected in tobacco smoke	N	Y	N	Y	Y	N
Isophorone	78591	Used in printing and the metal coating industries, released by coal-fired power plants and ship and boat building facilities	N	Y	N	Y	Y	N
Methylene Chloride (dichloromethane)	75092	Used in pesticide products, as industrial solvent, paint stripper and PolyurethaneFoam manufacture	Y	Y	Y	Y	Y	Y
m-Xylene	108383	Released into the atmosphere from industrial sources and auto exhaust	Y	Y	2	2	Y	N
Nitrobenzene	98953	Used to manufacture aniline, lubricating oils, dyes, drugs, pesticides, and synthetic rubber	N	Y	N	Y	Y	N
o-Xylene	95476	Released into the atmosphere from industrial sources and auto exhaust	Y	Y	2	2	Y	N

Chemical Compound	CAS Number	Major Sources/Uses	Monitoring	Emission Inventory	Risk Assessment		Modeling	
					Phase I	Phase II	Local Scale	Regional Scale
Perchloroethylene (tetrachloroethylene)	127184	Used in dry cleaning, textile processing, as a chemical intermediate, and in metal-cleaning operations	Y	Y	Y	Y	Y	Y
Phenol	108952	Used in the production of phenolic resins, in the manufacture of nylon and epoxy resins, as a slimicide, disinfectant, and in medicinal products, present in tobacco smoke	N	Y	N	Y	Y	N
p-Xylene	106423	Released into the atmosphere from industrial sources and auto exhaust	Y	Y	2	2	Y	N
Styrene	100425	Used in the production of polystyrene plastics and resins, has been detected in motor vehicle exhaust	Y	Y	Y	Y	Y	Y
Toluene	108883	Automobile emissions, cigarette smoke, exhaust from industries that use toluene as solvent (paint/print)	Y	Y	Y	Y	Y	Y
Trans-1,3-Dichloro-1-propene	10061026	Used in dry cleaning and degreasing processes, limited agricultural use	Y	N	N	N	N	N
Trichloroethylene (Trichloroethene)	79016	Used in metal degreasing, as an extraction solvent, a chemical intermediate, as a refrigerant, in typewriter correction fluids, paint removers/strippers, adhesives, spot removers, and rug-cleaning fluids	Y	Y	Y	Y	Y	Y
Trichlorofluoromethane	75694	Used as a foaming agent, refrigerant, and solvent, or use in the manufacture of fluoropolymers. Production phase-out in process	Y	N	Y	N	N	N
Vinyl Chloride (Chloroethene)	75014	Released from plastics industries, hazardous waste sites, and landfills	Y	Y	Y	Y	Y	Y
Xylenes (mixed isomers)	1330207	Released into the atmosphere from industrial sources and auto exhaust	N	Y	2	2	Y	Y
Carbonyls								
Acetaldehyde	75070	Natural and artificial sources. Wood combustion, coffee roasting, burning of tobacco, vehicle exhaust fumes, and coal refining and waste processing. During hot summer months, secondary formation of acetaldehyde is a major source	Y	Y	Y	Y	Y	Y
Acetone	67641	Natural and artificial sources. Released during its manufacture and use, in exhaust from automobiles, and from tobacco smoke, landfills, and certain kinds of burning waste materials	Y	Y	Y	Y	Y	N
Acrolein	107028	Used as a pesticide to control algae, weeds, bacteria, and mollusks, formed from the breakdown of certain pollutants found in outdoor air, from coal power plants, burning tobacco and gasoline	N	Y	N	Y	Y	Y
Formaldehyde	50000	Natural and artificial sources. Released from power plants, manufacturing facilities, incinerators, automobile exhaust, and cigarette smoke. During hot summer months, secondary formation of formaldehyde is a major source	Y	Y	Y	Y	Y	Y
Methyl Ethyl Ketone	78933	Used as a solvent, manufacture of synthetic rubber & paraffin wax, and in household products Found in motor vehicle exhaust. Secondary formation is important source	Y	Y	Y	Y	Y	N
Methyl Isobutyl Ketone	108101	Released from its manufacture and use, automobile exhaust, and landfill/hazardous waste sites	Y	Y	Y	Y	Y	N
Propionaldehyde	123386	Released from manufacturing facilities, municipal waste incinerators, and combustion of wood, gasoline, & diesel fuel. Tobacco smoke also contains propionaldehyde	Y	Y	N	N	Y	N
Polycyclic Aromatic Hydrocarbons (PAHs): Released from vehicle exhausts, asphalt roads, burning of wood, coal, oil, forest fires, coal tar production plants, coking plants, coal-gasification sites, smokehouses, municipal trash incinerators, cigarette smoke, agricultural burning and hazardous waste sites. Cooking meat or other foods at high temperatures also release PAHs								
1-Methylnaphthalene	90120		Y	N	N	N	N	N

Chemical Compound	CAS Number	Major Sources/Uses	Monitoring	Emission Inventory	Risk Assessment		Modeling	
					Phase I	Phase II	Local Scale	Regional Scale
2-Methylnaphthalene	91576		Y	N	Y	N	N	N
Acenaphthene	83329		Y	Y	Y	Y	Y	N
Acenaphthylene	208968		Y	Y	N	N	Y	N
Anthracene	120127		Y	Y	Y	Y	Y	N
Benzo(a)anthracene	56553		Y	Y	Y	Y	Y	N
Benzo(a)pyrene	50328		Y	Y	Y	Y	Y	N
Benzo(b)fluoranthene	205992		Y	Y	Y	Y	Y	N
Benzo(e)pyrene	192972		Y	N	N	N	N	N
Benzo(g,h,i)perylene	191242		Y	Y	N	N	Y	N
Benzo(k)fluoranthene	207089		Y	Y	Y	Y	Y	N
Chrysene	218019		Y	Y	Y	Y	Y	N
Dibenz(a,h)anthracene	53703		Y	Y	1	Y	Y	N
Fluoranthene	206440		Y	Y	Y	Y	Y	N
Fluorene	86737		Y	Y	Y	Y	Y	N
Indeno(1,2,3-cd)pyrene	193395		Y	Y	Y	Y	Y	N
Naphthalene	91203		Y	Y	Y	Y	Y	N
Phenanthrene	85018		Y	Y	N	N	Y	N
Pyrene	129000		Y	Y	Y	Y	Y	N
Dioxin/Furan: Released during the combustion of fossil fuels (including motor vehicles) and wood, the incineration of municipal and industrial wastes and backyard burning of trash. May be formed during the chlorine bleaching process at pulp and paper mills and in the manufacture of chlorinated organic chemicals								
1,2,3,4,6,7,8-HpCDD	35822469		Y	Y	Y	Y	Y	N
1,2,3,4,6,7,8-HpCDF	67562394		Y	Y	Y	Y	Y	N
1,2,3,4,7,8,9-HpCDF	55673897		Y	Y	Y	Y	Y	N
1,2,3,4,7,8-HxCDD	39227286		Y	Y	Y	Y	Y	N
1,2,3,4,7,8-HxCDF	70648269		Y	Y	Y	Y	Y	N
1,2,3,6,7,8-HxCDD	57653857		Y	Y	Y	Y	Y	N
1,2,3,6,7,8-HxCDF	57117449		Y	Y	Y	Y	Y	N
1,2,3,7,8,9-HxCDD	19408743		Y	Y	Y	Y	Y	N
1,2,3,7,8,9-HxCDF	72918219		Y	Y	Y	Y	Y	N
1,2,3,7,8-PeCDD	40321764		Y	Y	Y	Y	Y	N

Chemical Compound	CAS Number	Major Sources/Uses	Monitoring	Emission Inventory	Risk Assessment		Modeling	
					Phase I	Phase II	Local Scale	Regional Scale
1,2,3,7,8-PeCDF	57117416		Y	Y	Y	Y	Y	N
2,3,4,6,7,8-HxCDF	60851345		Y	Y	Y	Y	Y	N
2,3,4,7,8-PeCDF	57117314		Y	Y	Y	Y	Y	N
2,3,7,8-TCDD	1746016		Y	Y	Y	Y	Y	N
2,3,7,8-TCDD TEQ	600		N	Y	Y	Y	Y	N
2,3,7,8-TCDF	51207319		Y	Y	Y	Y	Y	N
OCDD	3268879		Y	Y	Y	Y	Y	N
OCDF	39001020		Y	Y	Y	Y	Y	N
Metals:								
Arsenic	7440382	Used in wood preservation, decolorizing glass and metal smelting operations. Released form burning of coal and diesel that is high in arsenic	Y	Y	Y	Y	Y	Y
Beryllium	7440417	Used in manufacture of electrical components, tools and other metal-fabricating operations	Y	Y	Y	Y	Y	Y
Cadmium	7440439	Released from metal smelting operations, burning of coal or oil and the incineration of municipal waste	Y	Y	Y	Y	Y	Y
Chromium (Total)	7440473	Released from ore refining, automobile brake lining and catalytic converters, chemical and refractory processing, cement-producing plants, ferrochrome production, leather tanneries, and chrome pigment	Y	Y	3	3	Y	N
Chromium Compounds (Chromium VI)	18540299	Source of chromium (VI) include chrome plating, electrical services, aircraft and parts manufacturing, and steam and air conditioning supply, from dyes and pigments, leather tanning, and wood preserving	N	Y	3	3	Y	Y
Lead	7439921	Released from combustion of solid waste, coal, and oils, emissions from iron and steel production, battery manufacture, ammunition manufacture, and lead smelters	Y	Y	N	N	Y	Y
Manganese	7439965	Released into the air by iron and steel production plants, power plants, coke ovens, dry-cell batteries manufacture, matches, fireworks, and the production of other manganese compounds	Y	Y	Y	Y	Y	Y
Mercury Compounds	7439976	Elemental mercury used in thermometers, barometers, and pressure-sensing devices, batteries, lamps, industrial processes, refining, lubrication oils, and dental amalgams, most agricultural and pharmaceutical uses of inorganic mercury have been discontinued in the United States, mercuric chloride is still used as a disinfectant and pesticide	N	Y	N	Y	Y	Y
Nickel	7440020	Released from oil and coal combustion, nickel metal refining and sewage sludge incineration	Y	Y	Y	Y	Y	Y
Other								
1,4-Dioxane	123911	Used as a solvent for cellulose acetate, ethyl cellulose, benzyl cellulose, resins, oils, waxes, some dyes, and other organic and inorganic compounds	N	Y	N	Y	Y	N
4,4'-Methylenediphenyl Diisocyanate	101688	Used to produce polyurethane foams	N	Y	N	Y	Y	N
Acetophenone	98862	Used as a fragrance ingredient in soaps, detergents, creams, lotions, and perfumes, as a flavoring agent in foods, beverages, and tobacco, as a industrial solvent and catalyst	N	Y	N	Y	Y	N

Chemical Compound	CAS Number	Major Sources/Uses	Monitoring	Emission Inventory	Risk Assessment		Modeling	
					Phase I	Phase II	Local Scale	Regional Scale
Antimony	7440360	Antimony is found at very low levels throughout the environment, Antimony is alloyed with other metals for lead acid storage batteries, solder, sheet and pipe, bearing metals, castings, and type metal.	N	Y	N	Y	Y	N
Biphenyl	92524	Used in organic syntheses, heat transfer fluids, dye carriers, food preservatives, as an intermediate for polychlorinated biphenyls, and as a fungistat in the packaging of citrus fruits.	N	Y	N	Y	Y	N
Chlorine	7782505	Used household cleaner and disinfectant, used as an oxidizing agent in water treatment and chemical processes, in the bleaching process of wood pulp in pulp mills.	N	Y	N	Y	Y	N
Cobalt	7440484	Cobalt is a natural element found throughout the environment, used to make superalloys and in pigment manufacture.	N	Y	N	Y	Y	N
Diesel Particulate Matter	Not applicable	Exposure to diesel particulate matter comes from both on road and off road engine exhaust that is either directly emitted from the engines (locomotives, marine vessels and heavy duty equipment) or aged through lingering in the atmosphere	N	Y	N	Y	Y	Y
Hexamethylene-1,6-diisocyanate	822060	Used as polymerizing agent in polyurethane spray paint formulations and coatings, in the preparation of dental materials, contact lenses, and medical adsorbents.	N	Y	N	Y	Y	N
Hydrochloric Acid	7647010	Used in the production of chlorides, fertilizers, dyes, in electroplating, and in the photographic, textile, and rubber industries.	N	Y	N	Y	Y	N
Hydrogen Sulfide	7783064	Occurs naturally. Released municipal sewers and sewage treatment plants, swine containment and manure-handling operations, and pulp and paper operations, petroleum refineries, natural gas plants, petrochemical plants, coke oven plants, food processing plants, and tanneries.	N	Y	N	Y	Y	N
PCBs	1336363	PCBs are no longer produced in the United States and are no longer used in the manufacture of new products; the major source of air exposure is the redistribution of PCBs already present in soil and water. Smaller amounts of PCBs may be released to the air from disposal sites containing transformers, capacitors, and other PCB wastes, incineration of PCB-containing wastes, and improper disposal of the compounds to open areas	N	Y	N	Y	Y	N
Phosgene	75445	Used in the preparation synthesis of isocyanate-based polymers, carbonic acid esters, and acid chlorides, manufacture of dyestuffs, some insecticides, and pharmaceuticals and in metallurgy	N	Y	N	Y	Y	N
Phosphorous	7723140	Used in rodenticides, smoke screens, tracer bullets, fertilizers, gas analysis, manufacture phosphoric acid and other phosphorus compounds, phosphor bronzes, and metallic phosphides, and as an additive to semiconductors, electroluminescent coatings, safety matches, and fertilizers	N	Y	N	Y	Y	N

Notes:

- 1 Risk assessment possible for compound however monitoring data was insufficient to produce mean value for risk calculations.
- 2 Risk assessment values are available for total xylenes, but not for individual isomers. Mean values for individual isomers were added prior to risk assessment calculations.
- 3 Risk assessment for Chromium based on assumption that 35% of total chromium would be present as hexavalent chromium.

Table B1-1: VOC data summary at MLK

Compound	Concentration (µg/m ³)							ND %	Valid	SD	CV
	MDL	Max	Min	Low Qnt	Mean	Median	UCL				
1,1,1-Trichloroethane	0.170	0.327	0.109	0.109	0.172	0.164	0.194	0	59	0.038	0.221
1,1,2,2-Tetrachloroethane	0.151	0.137	0.000	0.069	0.010 ^P	0.000	NA	67	59	0.038	3.793
1,1,2-Trichloro-1,2,2-trifluoroethane	0.168	0.843	0.460	0.460	0.625	0.613	0.667	0	59	0.073	0.117
1,1,2-Trichloroethane	0.202	0.055	0.000	0.055	NA	0.000	NA	91	59	0.015	NA
1,1-Dichloroethane	0.089	0.040	0.000	0.040	0.020	0.000	NA	88	59	0.013	0.665
1,1-Dichloroethene	0.190	0.162	0.000	0.040	0.006 ^P	0.000	NA	79	59	0.034	5.749
1,2,4-Trichlorobenzene	0.240	0.148	0.000	0.074	0.082	0.074	0.095	21	59	0.041	0.500
1,2,4-Trimethylbenzene	0.076	4.228	0.098	0.098	0.784	0.639	1.178	0	59	0.689	0.879
1,2-Dibromoethane	0.169	0.077	0.000	0.077	0.010	0.000	NA	84	59	0.028	2.806
1,2-Dichloro-1,1,2,2-tetrachloroethane	0.511	0.280	0.070	0.070	0.135	0.140	0.163	0	59	0.049	0.361
1,2-Dichlorobenzene	0.132	0.180	0.000	0.060	0.072	0.060	0.087	16	59	0.037	0.521
1,2-Dichloropropane	0.128	0.046	0.000	0.046	0.010 ^P	0.000	NA	86	59	0.016	1.607
1,3,5-Trimethylbenzene	0.129	1.131	0.049	0.049	0.237	0.197	0.344	0	59	0.186	0.785
1,3-Butadiene	0.117	1.261	0.044	0.044	0.301	0.221	0.442	0	59	0.245	0.815
1,3-dichlorobenzene	0.066	0.120	0.000	0.060	0.065	0.060	0.075	29	59	0.035	0.539
1,4-Dichlorobenzene	0.066	0.541	0.060	0.060	0.223	0.210	0.291	0	59	0.119	0.532
1-Ethyl-4-Methylbenzene	0.099	1.327	0.049	0.049	0.255	0.197	0.375	0	59	0.209	0.820
Benzene	0.035	3.866	0.511	0.511	1.375	1.054	1.832	0	59	0.798	0.581
Bromomethane	0.126	1.126	0.000	0.039	0.089	0.039	0.177	7	59	0.155	1.739
Carbon tetrachloride	0.296	0.692	0.377	0.377	0.541	0.566	0.576	0	59	0.060	0.111
Chlorobenzene	0.127	0.092	0.000	0.046	0.051	0.046	0.059	33	59	0.029	0.564
Chloroform	0.128	0.342	0.049	0.049	0.132	0.146	0.165	0	59	0.057	0.432
Chloromethane	0.083	1.507	0.826	0.826	1.117	1.094	1.189	0	59	0.126	0.113
Cis-1,2-Dichloroethene	0.140	0.079	0.000	0.040	0.010	0.000	NA	84	59	0.019	1.887
Cis-1,3-Dichloro-1-Propene	0.091	0.045	0.000	0.045	NA	0.000	NA	90	59	0.014	NA
Dichlorodifluoromethane	0.077	3.363	2.127	2.127	2.702	2.670	2.837	0	59	0.236	0.087
Ethyl Chloride	0.152	0.158	0.000	0.026	0.047	0.026	0.066	29	59	0.041	0.865
Ethylbenzene	0.048	3.387	0.130	0.130	0.660	0.434	1.035	0	59	0.657	0.996
Ethylene Dichloride	0.063	0.081	0.000	0.040	0.045	0.040	0.053	19	59	0.023	0.498
Hexachloro-1,3-Butadiene	0.331	0.107	0.000	0.107	0.107	0.107	0.107	7	59	0.027	0.256
m & p- Xylene	0.143	12.244	0.304	0.304	2.250	1.433	3.618	0	59	2.391	1.063
Methylene Chloride	0.079	4.829	0.174	0.174	0.644	0.486	1.029	0	59	0.672	1.043
o-Xylene	0.067	2.953	0.130	0.130	0.636	0.478	0.942	0	59	0.534	0.839
Perchloroethylene	0.211	3.052	0.136	0.136	0.534	0.407	0.806	0	59	0.474	0.887
Styrene	0.138	0.469	0.000	0.043	0.170	0.170	0.222	2	59	0.093	0.546
Toluene	0.083	43.074	0.565	0.565	4.066	2.808	7.414	0	59	5.850	1.439
Trans-1,3-Dichloro-1-Propene	0.147	0.045	0.000	0.045	0.010 ^P	0.000	NA	88	59	0.015	1.491
Trichloroethene	0.123	0.322	0.000	0.054	0.110	0.107	0.146	10	59	0.069	0.629
Trichlorofluoromethane	0.133	2.584	1.124	1.124	1.493	1.461	1.630	0	59	0.240	0.161
Vinyl chloride	0.067	0.256	0.000	0.026	0.055	0.026	0.088	43	59	0.064	1.165

MDL – Average Minimum Detection Limit; **Max** – highest sampled concentration; **Min** – lowest sampled concentration

Low Qnt – lowest quantitated concentration used to replace 0 in calculating mean and UCL for risk assessment

ND % – percent of samples where compound was reported as 0

UCL – 95% upper confidence limit; **Valid** – number of valid samples collected

SD – standard deviation; **CV** – coefficient of variation; **NA** – not applicable

^P Percentile values used in place of arithmetic mean

Table B1-2: VOC data summary at Delaware City

Compound	Concentration (µg/m ³)							ND %	Valid	SD	CV
	MDL	Max	Min	Low Qnt	Mean	Median	UCL				
1,1,1-Trichloroethane	0.170	0.218	0.109	0.109	0.152	0.164	0.169	0	54	0.029	0.194
1,1,2,2-Tetrachloroethane	0.151	0.069	0.000	0.069	0.010	0.000	NA	80	54	0.028	2.791
1,1,2-Trichloro-1,2,2-trifluoroethane	0.168	0.766	0.460	0.460	0.610	0.613	0.643	0	54	0.056	0.091
1,1,2-Trichloroethane	0.202	0.218	0.000	0.055	NA	0.000	NA	93	54	0.032	NA
1,1-Dichloroethane	0.089	0.040	0.000	0.040	0.020 ^P	0.000	NA	89	54	0.013	0.642
1,1-Dichloroethene	0.190	0.121	0.000	0.040	0.000 ^P	0.000	NA	69	54	0.027	NA
1,2,4-Trichlorobenzene	0.240	0.371	0.000	0.074	0.102	0.074	0.136	19	54	0.071	0.696
1,2,4-Trimethylbenzene	0.076	4.179	0.000	0.049	0.315	0.197	0.658	2	54	0.578	1.835
1,2-Dibromoethane	0.169	0.077	0.000	0.077	NA	0.000	NA	93	54	0.020	NA
1,2-Dichloro-1,1,2,2-tetrachloroethane	0.511	0.909	0.070	0.070	0.137	0.140	0.206	0	54	0.116	0.845
1,2-Dichlorobenzene	0.132	0.481	0.000	0.060	0.096	0.060	0.146	19	54	0.093	0.972
1,2-Dichloropropane	0.128	0.046	0.000	0.046	NA	0.000	NA	94	54	0.011	NA
1,3,5-Trimethylbenzene	0.129	1.229	0.000	0.049	0.112	0.049	0.213	4	54	0.171	1.528
1,3-Butadiene	0.117	0.332	0.000	0.022	0.096	0.088	0.136	7	54	0.069	0.718
1,3-dichlorobenzene	0.066	0.240	0.000	0.060	0.075	0.060	0.094	11	54	0.041	0.543
1,4-Dichlorobenzene	0.066	0.782	0.000	0.060	0.161	0.120	0.250	7	54	0.153	0.950
1-Ethyl-4-Methylbenzene	0.099	1.524	0.000	0.049	0.132	0.098	0.254	2	54	0.207	1.567
Benzene	0.035	7.604	0.256	0.256	1.055	0.751	1.758	0	54	1.184	1.122
Bromomethane	0.126	0.388	0.000	0.039	0.054	0.039	0.084	11	54	0.054	1.003
Carbon tetrachloride	0.296	0.692	0.440	0.440	0.536	0.503	0.568	0	54	0.054	0.101
Chlorobenzene	0.127	0.598	0.000	0.046	0.098	0.046	0.160	41	54	0.115	1.177
Chloroform	0.128	0.244	0.049	0.049	0.096	0.098	0.123	0	54	0.045	0.474
Chloromethane	0.083	2.251	0.888	0.888	1.151	1.146	1.268	0	54	0.198	0.172
Cis-1,2-Dichloroethene	0.140	0.079	0.000	0.040	NA	0.000	NA	93	54	0.014	NA
Cis-1,3-Dichloro-1-Propene	0.091	0.045	0.000	0.045	NA	0.000	NA	93	54	0.012	NA
Dichlorodifluoromethane	0.077	3.165	2.275	2.275	2.577	2.572	2.673	0	54	0.162	0.063
Ethyl chloride	0.152	0.237	0.000	0.026	0.037	0.026	0.056	33	54	0.038	1.032
Ethylbenzene	0.048	3.300	0.043	0.043	0.266	0.174	0.550	0	54	0.478	1.796
Ethylene Dichloride	0.063	0.121	0.000	0.040	0.050	0.040	0.063	4	54	0.023	0.453
Hexachloro-1,3-Butadiene	0.331	0.213	0.000	0.107	0.115	0.107	0.131	15	54	0.050	0.438
m & p- Xylene	0.143	8.163	0.130	0.130	0.708	0.391	1.423	0	54	1.204	1.700
Methylene Chloride	0.079	1.251	0.139	0.139	0.264	0.208	0.377	0	54	0.190	0.717
o-Xylene	0.067	3.083	0.043	0.043	0.272	0.174	0.537	0	54	0.448	1.647
Perchloroethylene	0.211	0.882	0.000	0.068	0.146	0.136	0.222	2	54	0.129	0.886
Styrene	0.138	0.469	0.000	0.043	0.106	0.085	0.150	6	54	0.078	0.737
Toluene	0.083	54.530	0.301	0.301	2.437	0.961	6.857	0	54	7.451	3.058
Trans-1,3-Dichloro-1-Propene	0.147	0.045	0.000	0.045	NA	0.000	NA	94	54	0.010	NA
Trichloroethene	0.123	0.699	0.000	0.054	0.091	0.054	0.147	19	54	0.102	1.122
Trichlorofluoromethane	0.133	1.966	1.180	1.180	1.378	1.348	1.454	0	54	0.129	0.093
Vinyl chloride	0.067	1.048	0.000	0.026	0.227	0.102	0.391	24	54	0.281	1.238

MDL – Average Minimum Detection Limit; **Max** – highest sampled concentration; **Min** – lowest sampled concentration
Low Qnt – lowest quantitated concentration used to replace 0 in calculating mean and UCL for risk assessment
ND % – percent of samples where compound was reported as 0
UCL – 95% upper confidence limit; **Valid** – number of valid samples collected
SD – standard deviation; **CV** – coefficient of variation; **NA** – not applicable
^P Percentile values used in place of arithmetic mean

Table B1-3: VOC data summary at Lums Pond

Compound	Concentration (µg/m ³)							ND %	Valid	SD	CV
	Avg MDL	Max	Min	Low Qnt	Mean	Median	95% UCL				
1,1,1-Trichloroethane	0.170	0.218	0.055	0.055	0.150	0.164	0.168	0	58	0.031	0.209
1,1,2,2-Tetrachloroethane	0.151	0.137	0.000	0.069	0.010 ^P	0.000	NA	74	58	0.034	3.372
1,1,2-Trichloro-1,2,2-trifluoroethane	0.168	0.766	0.307	0.307	0.617	0.613	0.661	0	58	0.076	0.123
1,1,2-Trichloroethane	0.202	0.055	0.000	0.055	NA	0.000	NA	93	58	0.014	NA
1,1-Dichloroethane	0.089	0.040	0.000	0.040	NA	0.000	NA	91	58	0.012	NA
1,1-Dichloroethene	0.190	0.081	0.000	0.040	0.000 ^P	0.000	NA	72	58	0.026	NA
1,2,4-Trichlorobenzene	0.240	0.074	0.000	0.074	0.074	0.074	0.074	23	58	0.031	0.423
1,2,4-Trimethylbenzene	0.076	0.639	0.000	0.049	0.172	0.147	0.235	2	58	0.113	0.655
1,2-Dibromoethane	0.169	0.077	0.000	0.077	0.010	0.000		89	58	0.024	2.379
1,2-Dichloro-1,1,2,2-tetrachloroethane	0.511	0.210	0.070	0.070	0.128	0.140	0.153	0	58	0.044	0.345
1,2-Dichlorobenzene	0.132	0.240	0.000	0.060	0.070	0.060	0.089	19	58	0.043	0.603
1,2-Dichloropropane	0.128	0.046	0.000	0.046	NA	0.000	NA	91	58	0.013	NA
1,3,5-Trimethylbenzene	0.129	0.147	0.000	0.049	0.070	0.049	0.087	11	58	0.037	0.537
1,3-Butadiene	0.117	0.265	0.000	0.022	0.072	0.044	0.108	16	58	0.066	0.915
1,3-dichlorobenzene	0.066	0.120	0.000	0.060	0.065	0.060	0.075	37	58	0.037	0.571
1,4-Dichlorobenzene	0.066	0.421	0.000	0.060	0.092	0.060	0.126	9	58	0.065	0.708
1-Ethyl-4-Methylbenzene	0.099	0.246	0.000	0.049	0.081	0.049	0.106	5	58	0.048	0.599
Benzene	0.035	1.629	0.256	0.256	0.590	0.479	0.755	0	58	0.289	0.490
Bromomethane	0.126	0.505	0.000	0.039	0.064	0.039	0.103	7	58	0.071	1.112
Carbon tetrachloride	0.296	0.629	0.377	0.377	0.542	0.566	0.574	0	58	0.055	0.102
Chlorobenzene	0.127	0.184	0.000	0.046	0.050	0.000	NA	60	58	0.034	0.676
Chloroform	0.128	0.195	0.049	0.049	0.087	0.098	0.111	0	58	0.042	0.481
Chloromethane	0.083	1.384	0.867	0.867	1.118	1.115	1.187	0	58	0.120	0.107
Cis-1,2-Dichloroethene	0.140	0.079	0.000	0.040	0.010	0.000	NA	89	58	0.015	1.512
Cis-1,3-Dichloro-1-Propene	0.091	0.045	0.000	0.045	NA	0.000	NA	91	58	0.013	NA
Dichlorodifluoromethane	0.077	3.017	2.077	2.077	2.621	2.621	2.735	0	58	0.198	0.076
Ethyl chloride	0.152	0.211	0.000	0.026	0.035	0.026	0.050	35	58	0.032	0.939
Ethylbenzene	0.048	0.521	0.043	0.043	0.137	0.130	0.181	0	58	0.077	0.561
Ethylene Dichloride	0.063	0.081	0.000	0.040	0.041	0.040	0.044	33	58	0.020	0.497
Hexachloro-1,3-Butadiene	0.331	0.107	0.000	0.107	0.107	0.107		18	58	0.041	0.384
m & p- Xylene	0.143	1.433	0.087	0.087	0.334	0.304	0.461	0	58	0.222	0.664
Methylene Chloride	0.079	0.521	0.139	0.139	0.242	0.208	0.288	0	58	0.081	0.333
o-Xylene	0.067	0.521	0.000	0.043	0.131	0.130	0.176	2	58	0.080	0.614
Perchloroethylene	0.211	0.475	0.000	0.068	0.137	0.136	0.186	4	58	0.088	0.645
Styrene	0.138	0.213	0.000	0.043	0.101	0.085	0.133	5	58	0.060	0.600
Toluene	0.083	3.806	0.188	0.188	0.844	0.678	1.208	0	58	0.635	0.752
Trans-1,3-Dichloro-1-Propene	0.147	0.045	0.000	0.045	NA	0.000	NA	91	58	0.013	NA
Trichloroethene	0.123	0.107	0.000	0.054	0.058	0.054	0.067	46	58	0.035	0.596
Trichlorofluoromethane	0.133	1.910	0.843	0.843	1.394	1.405	1.484	0	58	0.158	0.114
Vinyl chloride	0.067	0.562	0.000	0.026	0.020 ^P	0.000	NA	56	58	0.096	4.820

MDL –Average Minimum Detection Limit; **Max** – highest sampled concentration; **Min** – lowest sampled concentration

Low Qnt – lowest quantitated concentration used to replace 0 in calculating mean and UCL for risk assessment

ND % – percent of samples where compound was reported as 0

UCL – 95% upper confidence limit; #Valid – number of valid samples collected

SD – standard deviation; **CV** – coefficient of variation; **NA** – not applicable

^P Percentile values used in place of arithmetic mean

Table B1-4: VOC data summary at Killens Pond

Compound	Concentration (µg/m ³)							ND %	Valid	SD	CV
	Avg MDL	Max	Min	Low Qnt	Mean	Median	95% UCL				
1,1,1-Trichloroethane	0.170	0.218	0.055	0.055	0.146	0.164	0.164	0	59	0.031	0.213
1,1,2,2-Tetrachloroethane	0.151	0.069	0.000	0.069	0.010 ^P	0.000	NA	76	59	0.029	2.946
1,1,2-Trichloro-1,2,2-trifluoroethane	0.168	0.766	0.536	0.536	0.610	0.613	0.641	0	59	0.053	0.087
1,1,2-Trichloroethane	0.202	0.055	0.000	0.055	NA	0.000	NA	93	59	0.014	NA
1,1-Dichloroethane	0.089	0.040	0.000	0.040	NA	0.000	NA	90	59	0.012	NA
1,1-Dichloroethene	0.190	0.121	0.000	0.040	0.000 ^P	0.000	NA	68	59	0.028	NA
1,2,4-Trichlorobenzene	0.240	0.223	0.000	0.074	0.088	0.074	0.108	20	59	0.051	0.575
1,2,4-Trimethylbenzene	0.076	0.344	0.000	0.049	0.124	0.098	0.162	2	59	0.067	0.542
1,2-Dibromoethane	0.169	0.077	0.000	0.077	0.010 ^P	0.000	NA	88	59	0.025	2.506
1,2-Dichloro-1,1,2,2-tetrachloroethane	0.511	0.210	0.070	0.070	0.123	0.140	0.148	0	59	0.044	0.355
1,2-Dichlorobenzene	0.132	0.120	0.000	0.060	0.063	0.060	0.071	22	59	0.030	0.473
1,2-Dichloropropane	0.128	0.046	0.000	0.046	NA	0.000	NA	93	59	0.012	NA
1,3,5-Trimethylbenzene	0.129	0.147	0.000	0.049	0.056	0.049	0.068	19	59	0.031	0.553
1,3-Butadiene	0.117	0.265	0.000	0.022	0.045	0.022	0.069	34	59	0.048	1.083
1,3-dichlorobenzene	0.066	0.120	0.000	0.060	0.062	0.060	0.068	32	59	0.032	0.510
1,4-Dichlorobenzene	0.066	0.180	0.000	0.060	0.077	0.060	0.094	12	59	0.039	0.503
1-Ethyl-4-Methylbenzene	0.099	0.147	0.000	0.049	0.064	0.049	0.080	10	59	0.034	0.528
Benzene	0.035	1.054	0.160	0.160	0.465	0.447	0.575	0	59	0.193	0.416
Bromomethane	0.126	0.272	0.000	0.039	0.049	0.039	0.068	15	59	0.038	0.774
Carbon tetrachloride	0.296	0.629	0.440	0.440	0.560	0.566	0.584	0	59	0.043	0.077
Chlorobenzene	0.127	0.046	0.000	0.046	0.046	0.000	NA	75	59	0.020	0.439
Chloroform	0.128	0.146	0.049	0.049	0.081	0.098	0.099	0	59	0.031	0.381
Chloromethane	0.083	2.127	0.909	0.909	1.132	1.115	1.227	0	59	0.168	0.148
Cis-1,2-Dichloroethene	0.140	0.079	0.000	0.040	NA	0.000	NA	95	59	0.012	NA
Cis-1,3-Dichloro-1-Propene	0.091	0.045	0.000	0.045	NA	0.000	NA	92	59	0.013	NA
Dichlorodifluoromethane	0.077	3.116	2.275	2.275	2.583	2.572	2.676	0	59	0.164	0.063
Ethyl chloride	0.152	0.158	0.000	0.026	0.035	0.026	0.047	22	59	0.026	0.739
Ethylbenzene	0.048	0.261	0.043	0.043	0.098	0.087	0.127	0	59	0.051	0.518
Ethylene Dichloride	0.063	0.081	0.000	0.040	0.042	0.040	0.046	36	59	0.022	0.522
Hexachloro-1,3-Butadiene	0.331	0.107	0.000	0.107	0.107	0.107	0.107	27	59	0.048	0.448
m & p- Xylene	0.143	0.695	0.043	0.043	0.206	0.174	0.271	0	59	0.114	0.556
Methylene Chloride	0.079	0.903	0.104	0.104	0.227	0.208	0.295	0	59	0.119	0.524
o-Xylene	0.067	0.261	0.043	0.043	0.087	0.087	0.113	0	59	0.046	0.533
Perchloroethylene	0.211	0.407	0.000	0.068	0.108	0.068	0.145	7	59	0.071	0.653
Styrene	0.138	0.170	0.000	0.043	0.086	0.085	0.108	8	59	0.044	0.511
Toluene	0.083	7.612	0.188	0.188	0.609	0.415	1.155	0	59	0.962	1.581
Trans-1,3-Dichloro-1-Propene	0.147	0.045	0.000	0.045	NA	0.000	NA	93	59	0.012	NA
Trichloroethene	0.123	2.257	0.000	0.054	0.097	0.054	0.259	44	59	0.292	3.020
Trichlorofluoromethane	0.133	2.135	1.124	1.124	1.387	1.348	1.470	0	59	0.146	0.105
Vinyl chloride	0.067	0.051	0.000	0.026	0.050	0.000	NA	85	59	0.011	0.215

MDL –Average Minimum Detection Limit; **Max** – highest sampled concentration; **Min** – lowest sampled concentration

Low Qnt – lowest quantitated concentration used to replace 0 in calculating mean and UCL for risk assessment

ND % – percent of samples where compound was reported as 0

UCL – 95% upper confidence limit; **Valid** – number of valid samples collected

SD – standard deviation; **CV** – coefficient of variation; **NA** – not applicable

^P Percentile values used in place of arithmetic mean

Table B1-5: VOC data summary at Seaford

Compound	Concentration (µg/m ³)							ND %	Valid	SD	CV
	Avg MDL	Max	Min	Low Qnt	Mean	Median	95% UCL				
1,1,1-Trichloroethane	0.170	0.491	0.109	0.109	0.173	0.164	0.210	0	57	0.063	0.363
1,1,2,2-Tetrachloroethane	0.151	0.069	0.000	0.069	0.010	0.000	NA	84	57	0.025	2.526
1,1,2-Trichloro-1,2,2-trifluoroethane	0.168	0.766	0.536	0.536	0.610	0.613	0.643	0	57	0.056	0.092
1,1,2-Trichloroethane	0.202	0.055	0.000	0.055	NA	0.000	NA	93	57	0.014	NA
1,1-Dichloroethane	0.089	0.040	0.000	0.040	NA	0.000	NA	91	57	0.012	NA
1,1-Dichloroethene	0.190	0.081	0.000	0.040	0.010	0.000	NA	86	57	0.017	1.674
1,2,4-Trichlorobenzene	0.240	0.148	0.000	0.074	0.077	0.074	0.085	16	57	0.032	0.411
1,2,4-Trimethylbenzene	0.076	1.573	0.000	0.049	0.218	0.147	0.355	2	57	0.237	1.087
1,2-Dibromoethane	0.169	0.077	0.000	0.077	NA	0.000	NA	91	57	0.022	NA
1,2-Dichloro-1,1,2,2-tetrachloroethane	0.511	0.210	0.070	0.070	0.120	0.140	0.140	0	57	0.034	0.286
1,2-Dichlorobenzene	0.132	0.120	0.000	0.060	0.064	0.060	0.073	25	57	0.032	0.503
1,2-Dichloropropane	0.128	0.046	0.000	0.046	NA	0.000	NA	91	57	0.013	NA
1,3,5-Trimethylbenzene	0.129	0.393	0.000	0.049	0.079	0.049	0.114	12	57	0.064	0.812
1,3-Butadiene	0.117	0.509	0.000	0.022	0.070	0.044	0.118	12	57	0.084	1.197
1,3-dichlorobenzene	0.066	0.120	0.000	0.060	0.061	0.060	0.066	35	57	0.031	0.503
1,4-Dichlorobenzene	0.066	0.301	0.000	0.060	0.089	0.060	0.117	7	57	0.053	0.600
1-Ethyl-4-Methylbenzene	0.099	0.492	0.000	0.049	0.088	0.049	0.132	7	57	0.079	0.895
Benzene	0.035	2.045	0.192	0.192	0.566	0.511	0.731	0	57	0.286	0.507
Bromomethane	0.126	2.718	0.000	0.039	0.237	0.039	0.543	7	57	0.531	2.238
Carbon tetrachloride	0.296	0.692	0.503	0.503	0.561	0.566	0.586	0	57	0.043	0.077
Chlorobenzene	0.127	0.046	0.000	0.046	0.046	0.000	NA	77	57	0.019	0.423
Chloroform	0.128	0.195	0.049	0.049	0.088	0.098	0.108	0	57	0.035	0.397
Chloromethane	0.083	1.425	0.950	0.950	1.138	1.115	1.193	0	57	0.096	0.084
Cis-1,2-Dichloroethene	0.140	0.040	0.000	0.040	NA	0.000	NA	93	57	0.010	NA
Cis-1,3-Dichloro-1-Propene	0.091	0.045	0.000	0.045	NA	0.000	NA	93	57	0.012	NA
Dichlorodifluoromethane	0.077	3.017	2.275	2.275	2.604	2.572	2.695	0	57	0.158	0.061
Ethyl chloride	0.152	0.079	0.000	0.026	0.032	0.026	0.039	35	57	0.021	0.652
Ethylbenzene	0.048	0.825	0.043	0.043	0.150	0.130	0.220	0	57	0.122	0.811
Ethylene Dichloride	0.063	0.081	0.000	0.040	0.041	0.040	0.044	42	57	0.021	0.521
Hexachloro-1,3-Butadiene	0.331	0.107	0.000	0.107	0.107	0.107	0.107	28	57	0.048	0.453
m & p- Xylene	0.143	2.649	0.043	0.043	0.376	0.261	0.595	0	57	0.379	1.010
Methylene Chloride	0.079	1.216	0.104	0.104	0.232	0.208	0.317	0	57	0.147	0.632
o-Xylene	0.067	0.912	0.043	0.043	0.155	0.130	0.231	0	57	0.131	0.840
Perchloroethylene	0.211	0.814	0.000	0.068	0.176	0.136	0.264	2	57	0.153	0.870
Styrene	0.138	0.256	0.000	0.043	0.107	0.085	0.138	5	57	0.057	0.533
Toluene	0.083	12.285	0.226	0.226	1.041	0.641	2.005	0	57	1.669	1.603
Trans-1,3-Dichloro-1-Propene	0.147	0.045	0.000	0.045	NA	0.000	NA	93	57	0.012	NA
Trichloroethene	0.123	0.269	0.000	0.054	0.073	0.054	0.096	33	57	0.055	0.760
Trichlorofluoromethane	0.133	1.742	1.180	1.180	1.386	1.348	1.455	0	57	0.120	0.087
Vinyl chloride	0.067	0.026	0.000	0.026	0.050 ^P	0.000	NA	88	57	0.008	0.169

MDL –Average Minimum Detection Limit; **Max** – highest sampled concentration; **Min** – lowest sampled concentration

Low Qnt – lowest quantitated concentration used to replace 0 in calculating mean and UCL for risk assessment

ND % – percent of samples where compound was reported as 0

UCL – 95% upper confidence limit; **Valid** – number of valid samples collected

SD – standard deviation; **CV** – coefficient of variation; **NA** – not applicable

^P Percentile values used in place of arithmetic mean

Table B2: Metals data summary at MLK, Delaware City, Lums Pond, Killens Pond and Seaford

Element	Concentration (ng/m ³)							ND %	Valid	SD	CV
	MDL	Max	Min	½ MDL	Mean	Median	UCL				
MLK:											
Arsenic	0.036	3.082	0.012	0.018	1.118	0.971	1.451	0	55	0.567	0.507
Beryllium	0.002	0.047	0.000	0.001	0.017	0.014	0.023	2	55	0.010	0.583
Cadmium	0.024	0.939	0.092	0.012	0.285	0.231	0.390	0	55	0.178	0.626
Chromium (Total)	0.049	10.122	1.787	0.025	3.809	3.326	4.765	0	55	1.625	0.427
Manganese	0.049	68.751	4.366	0.025	18.846	14.298	26.703	0	55	13.367	0.709
Nickel	0.073	26.029	1.813	0.036	6.940	5.762	9.443	0	55	4.259	0.614
Lead	0.073	50.186	1.682	0.036	9.837	8.099	14.144	0	55	7.327	0.745
Delaware City:											
Arsenic	0.036	3.361	0.046	0.018	1.021	0.889	1.364	0	48	0.551	0.540
Beryllium	0.002	0.024	0.000	0.001	0.011	0.010	0.015	13	48	0.006	0.538
Cadmium	0.024	0.926	0.077	0.012	0.236	0.167	0.340	0	48	0.166	0.705
Chromium (Total)	0.049	3.423	1.522	0.025	2.252	2.254	2.554	0	48	0.485	0.215
Manganese	0.049	22.652	2.045	0.025	6.909	6.340	9.273	0	48	3.758	0.544
Nickel	0.073	17.610	1.505	0.036	4.583	4.121	6.404	0	48	2.895	0.632
Lead	0.073	13.482	2.179	0.036	5.192	4.905	6.869	0	48	2.666	0.513
Lums Pond:											
Arsenic	0.036	2.919	0.119	0.018	0.906	0.787	1.253	0	52	0.574	0.633
Beryllium	0.002	0.025	0.000	0.001	0.009	0.009	0.013	10	52	0.005	0.576
Cadmium	0.024	0.853	0.054	0.012	0.197	0.175	0.276	0	52	0.130	0.659
Chromium (Total)	0.049	5.152	1.317	0.025	2.133	1.991	2.489	0	52	0.588	0.276
Manganese	0.049	22.061	1.373	0.025	6.561	5.337	9.148	0	52	4.278	0.652
Nickel	0.073	10.480	1.089	0.036	2.982	2.425	4.151	0	52	1.935	0.649
Lead	0.073	10.343	1.618	0.036	3.951	3.807	4.980	0	52	1.703	0.431
Killens pond:											
Arsenic	0.036	4.171	0.084	0.018	0.884	0.734	1.269	0	57	0.667	0.755
Beryllium	0.002	0.055	0.000	0.001	0.010	0.008	0.014	14	57	0.008	0.847
Cadmium	0.024	1.078	0.046	0.012	0.198	0.149	0.294	0	57	0.166	0.841
Chromium (Total)	0.049	3.647	1.284	0.025	1.770	1.717	1.977	0	57	0.359	0.203
Manganese	0.049	24.274	0.884	0.025	4.942	4.052	7.286	0	57	4.059	0.821
Nickel	0.073	4.321	0.871	0.036	2.068	1.968	2.515	0	57	0.775	0.375
Lead	0.073	8.636	0.723	0.036	3.392	2.931	4.556	0	57	2.015	0.594
Seaford:											
Arsenic	0.036	2.340	0.000	0.018	0.884	0.852	1.147	2	57	0.460	0.520
Beryllium	0.002	0.026	0.000	0.001	0.008	0.007	0.011	4	57	0.005	0.631
Cadmium	0.024	0.986	0.025	0.012	0.205	0.167	0.300	0	57	0.164	0.802
Chromium (Total)	0.049	2.590	1.070	0.025	1.672	1.636	1.845	0	57	0.296	0.177
Manganese	0.049	16.214	0.419	0.025	4.569	3.744	6.195	0	57	2.825	0.618
Nickel	0.073	6.774	0.796	0.036	2.309	2.022	3.007	0	57	1.198	0.519
Lead	0.073	8.554	0.460	0.036	3.378	3.284	4.392	0	57	1.771	0.525

MDL –Average Minimum Detection Limit; **Max** – highest sampled concentration; **Min** – lowest sampled concentration
½ MDL – ½ Minimum Detection Limit used to replace not detected/0 in calculating mean and UCL for risk assessment
ND % – percent of samples where compound was reported as not detected
UCL – 95% upper confidence limit; **Valid** – number of valid samples collected
SD – standard deviation; **CV** – coefficient of variation; **NA** – not applicable

Table B3: Carbonyl data summary at MLK, Delaware City, Lums Pond, Killens Pond and Seaford

Compound	Concentration ($\mu\text{g}/\text{m}^3$) ($\times 10^{-2}$)							ND %	Valid	SD ($\times 10^{-2}$)	CV
	MDL	Max	Min	Low Qnt	Mean	Median	UCL				
MLK:											
Acetaldehyde	0.012	0.331	0.052	0.052	0.180	0.169	0.210	0	56	0.065	0.364
Acetone	0.020	1.054	0.000	0.049	0.510	0.570	0.680	0	48	0.266	0.521
Formaldehyde	0.019	1.251	0.000	0.009	0.245	0.213	0.358	2	56	0.191	0.779
Methyl Ethyl Ketone	0.017	0.272	0.000	0.008	0.098	0.101	0.134	2	56	0.061	0.625
Methyl Isobutyl Ketone	0.015	0.015	0.000	0.010	0.010 ^P	0.000	NA	71	56	0.016	1.631
Propionaldehyde	0.015	0.155	0.000	0.001	0.044	0.009	0.070	45	56	0.049	1.091
Delaware City:											
Acetaldehyde	0.012	0.295	0.025	0.025	0.139	0.130	0.168	0	59	0.053	0.382
Acetone	0.020	1.015	0.032	0.032	0.390	0.360	0.520	0	49	0.208	0.533
Formaldehyde	0.019	0.681	0.000	0.009	0.127	0.090	0.200	3	59	0.125	0.978
Methyl Ethyl Ketone	0.017	0.363	0.000	0.004	0.074	0.073	0.108	3	59	0.113	1.527
Methyl Isobutyl Ketone	0.015	0.000	0.000	NA	NA	NA	NA	100	59	NA	NA
Propionaldehyde	0.015	0.264	0.000	0.008	0.058	0.041	0.093	39	59	0.061	1.052
Lums Pond:											
Acetaldehyde	0.012	0.442	0.007	0.007	0.144	0.130	0.185	0	58	0.070	0.488
Acetone	0.020	1.403	0.000	0.166	0.430	0.397	0.617	0	48	0.267	0.621
Formaldehyde	0.019	0.681	0.000	0.009	0.145	0.090	0.230	2	56	0.143	0.985
Methyl Ethyl Ketone	0.017	0.185	0.000	0.005	0.072	0.063	0.097	2	57	0.042	0.583
Methyl Isobutyl Ketone	0.015	0.000	0.000	NA	NA	NA	NA	100	57	NA	NA
Propionaldehyde	0.015	0.350	0.000	0.005	0.060	0.042	0.102	39	57	0.066	1.100
Killens pond:											
Acetaldehyde	0.012	0.261	0.020	0.020	0.128	0.125	0.159	0	59	0.055	0.429
Acetone	0.020	1.168	0.062	0.062	0.380	0.387	0.510	0	49	0.197	0.519
Formaldehyde	0.019	0.755	0.000	0.009	0.125	0.094	0.193	3	59	0.117	0.938
Methyl Ethyl Ketone	0.017	0.236	0.000	0.005	0.069	0.046	0.104	9	58	0.058	0.841
Methyl Isobutyl Ketone	0.015	0.000	0.000	NA	NA	NA	NA	100	59	NA	NA
Propionaldehyde	0.015	0.158	0.000	0.002	0.050 ^P	0.000	NA	51	59	0.039	0.786
Seaford:											
Acetaldehyde	0.012	0.393	0.033	0.033	0.160	0.157	0.210	0	58	0.076	0.475
Acetone	0.020	1.310	0.070	0.070	0.490	0.560	0.660	0	47	0.246	0.502
Formaldehyde	0.019	1.435	0.000	0.006	0.491	0.362	0.724	5	58	0.395	0.804
Methyl Ethyl Ketone	0.017	0.188	0.000	0.006	0.058	0.051	0.087	12	58	0.044	0.760
Methyl Isobutyl Ketone	0.015	0.000	0.000	NA	NA	NA	NA	100	58	NA	NA
Propionaldehyde	0.015	0.122	0.000	0.008	0.030 ^P	0.000	NA	67	58	0.030	1.012

MDL – Average Minimum Detection Limit; **Max** – highest sampled concentration; **Min** – lowest sampled concentration

Low Qnt – lowest quantitated concentration used to replace 0 in calculating mean and UCL for risk assessment

ND % – percent of samples where compound was reported as not detected

UCL – 95% upper confidence limit; **Valid** – number of valid samples collected

SD – standard deviation; **CV** – coefficient of variation; **NA** – not applicable

^P Percentile values were used for arithmetic mean calculations

Table B4-1: PAH data summary at MLK and Delaware City

Compound	Concentration ($\mu\text{g}/\text{m}^3$) ($\times 10^{-2}$)							ND %	Valid	SD ($\times 10^{-2}$)	CV
	MDL	Max	Min	Low Qnt	Mean	Median	UCL				
MLK:											
1-Methylnaphthalene	0.028	1.7433	0.000	0.030	0.252	0.130	0.436	2	50	0.299	1.185
2-Methylnaphthalene	0.028	1.889	0.031	0.031	0.449	0.264	0.727	0	50	0.450	1.002
Acenaphthene	0.028	0.662	0.000	0.046	0.208	0.168	0.295	4	50	0.142	0.684
Acenaphthylene	0.028	0.951	0.000	0.029	0.152	0.054	0.273	38	50	0.196	1.287
Anthracene	0.028	0.331	0.000	0.029	0.090	0.059	0.138	28	50	0.079	0.878
Benzo(a)anthracene	0.028	0.698	0.000	0.028	0.043 ^P	0.000	NA	54	50	0.103	2.386
Benzo(a)pyrene	0.028	0.166	0.000	0.030	0.053 ^P	0.000	NA	62	50	0.045	0.850
Benzo(b)fluoranthene	0.028	0.354	0.000	0.031	0.107	0.089	0.156	14	50	0.079	0.745
Benzo(e)pyrene	0.028	0.215	0.000	0.030	0.058	0.041	0.085	36	50	0.043	0.736
Benzo(g,h,i)perylene	0.028	0.185	0.000	0.027	0.057	0.038	0.084	36	50	0.043	0.747
Benzo(k)fluoranthene	0.028	0.149	0.000	0.030	0.044 ^P	0.000	NA	64	50	0.039	0.895
Chrysene	0.028	0.381	0.000	0.020	0.075	0.049	0.115	29	50	0.067	0.889
Dibenz(a,h)anthracene	0.028	0.033	0.000	0.027	NA	0.000	NA	96	50	0.006	NA
Fluoranthene	0.028	8.663	0.121	0.121	1.144	0.613	2.108	0	50	1.564	1.367
Fluorene	0.028	1.242	0.069	0.069	0.468	0.390	0.639	0	50	0.277	0.592
Indeno(1,2,3-cd)pyrene	0.028	0.231	0.000	0.028	0.059	0.032	0.088	44	50	0.048	0.806
Naphthalene	0.028	1.347	0.027	0.027	0.317	0.191	0.504	0	50	0.302	0.951
Phenanthrene	0.028	14.439	0.338	0.338	2.966	2.056	4.593	0	50	2.639	0.890
Pyrene	0.028	2.888	0.065	0.065	0.589	0.451	0.911	0	50	0.523	0.888
Delaware City:											
1-Methylnaphthalene	0.028	0.505	0.027	0.027	0.113	0.071	0.191	29	42	0.116	1.027
2-Methylnaphthalene	0.028	0.814	0.030	0.030	0.186	0.133	0.316	12	42	0.194	1.044
Acenaphthene	0.028	0.190	0.028	0.028	0.046	0.030	0.068	45	42	0.032	0.706
Acenaphthylene	0.028	0.228	0.000	0.030	0.060 ^P	0.000	NA	52	42	0.058	0.974
Anthracene	0.028	0.114	0.000	0.031	NA	0.000	NA	93	42	0.019	NA
Benzo(a)anthracene	0.028	0.084	0.000	0.027	0.024 ^P	0.000	NA	88	42	0.016	0.686
Benzo(a)pyrene	0.028	0.092	0.000	0.045	NA	0.000	NA	95	42	0.016	NA
Benzo(b)fluoranthene	0.028	0.264	0.000	0.030	0.050 ^P	0.000	NA	64	42	0.045	0.908
Benzo(e)pyrene	0.028	0.137	0.000	0.028	NA	0.000	NA	90	42	0.023	NA
Benzo(g,h,i)perylene	0.028	0.172	0.000	0.030	NA	0.000	NA	93	42	0.027	NA
Benzo(k)fluoranthene	0.028	0.087	0.000	0.087	NA	0.000	NA	98	42	0.013	NA
Chrysene	0.028	0.238	0.000	0.033	0.034 ^P	0.000	NA	86	42	0.038	1.111
Dibenz(a,h)anthracene	0.028	0.000	0.000	0.000	NA	0.000	NA	100	42	0.000	NA
Fluoranthene	0.028	0.898	0.034	0.034	0.113	0.095	0.199	2	42	0.128	1.130
Fluorene	0.028	0.290	0.030	0.030	0.128	0.095	0.185	2	42	0.083	0.649
Indeno(1,2,3-cd)pyrene	0.028	0.108	0.000	0.033	NA	0.000	NA	90	42	0.020	NA
Naphthalene	0.028	0.968	0.027	0.027	0.163	0.095	0.297	19	42	0.200	1.229
Phenanthrene	0.028	2.614	0.119	0.119	0.421	0.351	0.673	2	42	0.374	0.889
Pyrene	0.028	0.687	0.026	0.026	0.081	0.065	0.147	7	42	0.098	1.211

MDL –Average Minimum Detection Limit; **Max** – highest sampled concentration; **Min** – lowest sampled concentration

Low Qnt – lowest quantitated concentration used to replace 0 in calculating mean and UCL for risk assessment

ND % – percent of samples where compound was reported as 0

UCL – 95% upper confidence limit; **Valid** – number of valid samples collected

SD – standard deviation; **CV** – coefficient of variation; **NA** – not applicable

^P Percentile values used in place of arithmetic mean

Table B4-2: PAH data summary at Lums Pond and Killens Pond

Compound	Concentration ($\mu\text{g}/\text{m}^3$) ($\times 10^{-2}$)							ND %	Valid	SD ($\times 10^{-2}$)	CV
	MDL	Max	Min	Low Qnt	Mean	Median	UCL				
Lums Pond:											
1-Methylnaphthalene	0.028	5.789	0.000	0.031	0.219	0.041	0.775	36	45	0.856	3.905
2-Methylnaphthalene	0.028	13.10	0.000	0.028	0.438	0.080	1.697	20	45	1.938	4.428
Acenaphthene	0.028	1.676	0.000	0.032	0.041 ^P	0.000	NA	58	45	0.248	6.054
Acenaphthylene	0.028	3.961	0.000	0.033	0.041 ^P	0.000	NA	73	45	0.589	14.365
Anthracene	0.028	0.000	0.000	0.000	NA	0.000	NA	100	45	0.000	NA
Benzo(a)anthracene	0.028	0.050	0.000	0.029	NA	0.000	NA	93	45	0.010	NA
Benzo(a)pyrene	0.028	0.047	0.000	0.047	NA	0.000	NA	98	45	0.007	NA
Benzo(b)fluoranthene	0.028	2.742	0.000	0.029	0.007 ^P	0.000	NA	75	45	0.412	58.898
Benzo(e)pyrene	0.028	0.040	0.000	0.038	NA	0.000	NA	96	45	0.008	NA
Benzo(g,h,i)perylene	0.028	0.000	0.000	0.000	NA	0.000	NA	100	45	0.000	NA
Benzo(k)fluoranthene	0.028	0.000	0.000	0.000	NA	0.000	NA	100	45	0.000	NA
Chrysene	0.028	2.133	0.000	0.027	0.033 ^P	0.000	NA	84	45	0.317	9.620
Dibenz(a,h)anthracene	0.028	0.000	0.000	0.000	NA	0.000	NA	100	45	0.000	NA
Fluoranthene	0.028	7.922	0.000	0.029	0.255	0.072	1.015	20	45	1.170	4.583
Fluorene	0.028	8.379	0.000	0.026	0.277	0.078	1.081	13	45	1.237	4.460
Indeno(1,2,3-cd)pyrene	0.028	1.828	0.000	0.077	NA	0.000	NA	93	45	0.272	NA
Naphthalene	0.028	19.805	0.000	0.030	0.604	0.074	2.511	22	45	2.936	4.863
Phenanthrene	0.028	25.899	0.000	0.039	0.875	0.318	3.358	4	45	3.820	4.364
Pyrene	0.028	5.484	0.000	0.028	0.173	0.043	0.700	27	45	0.810	4.680
Killens Pond:											
1-Methylnaphthalene	0.028	0.206	0.000	0.027	0.062	0.033	0.095	40	47	0.052	0.843
2-Methylnaphthalene	0.028	0.387	0.000	0.030	0.099	0.054	0.153	30	47	0.086	0.872
Acenaphthene	0.028	0.077	0.000	0.027	0.028 ^P	0.000	NA	70	47	0.020	0.699
Acenaphthylene	0.028	0.127	0.000	0.029	0.030 ^P	0.000	NA	77	47	0.025	0.819
Anthracene	0.028	0.238	0.000	0.238	NA	0.000	NA	98	47	0.035	NA
Benzo(a)anthracene	0.028	0.068	0.000	0.030	NA	0.000	NA	96	47	0.011	NA
Benzo(a)pyrene	0.028	0.042	0.000	0.042	NA	0.000	NA	98	47	0.006	NA
Benzo(b)fluoranthene	0.028	0.062	0.000	0.028	0.014 ^P	0.000	NA	74	47	0.019	1.389
Benzo(e)pyrene	0.028	0.041	0.000	0.030	NA	0.000	NA	96	47	0.007	NA
Benzo(g,h,i)perylene	0.028	0.000	0.000	0.000	NA	0.000	NA	100	47	0.000	NA
Benzo(k)fluoranthene	0.028	0.000	0.000	0.000	NA	0.000	NA	100	47	0.000	NA
Chrysene	0.028	0.041	0.000	0.027	NA	0.000	NA	96	47	0.007	NA
Dibenz(a,h)anthracene	0.028	0.000	0.000	0.000	NA	0.000	NA	100	47	0.000	NA
Fluoranthene	0.028	0.298	0.000	0.027	0.060	0.046	0.089	6	47	0.046	0.758
Fluorene	0.028	0.229	0.000	0.027	0.081	0.066	0.113	11	47	0.050	0.621
Indeno(1,2,3-cd)pyrene	0.028	0.042	0.000	0.042	NA	0.000	NA	96	47	0.006	NA
Naphthalene	0.028	0.580	0.000	0.027	0.097	0.060	0.162	34	47	0.103	1.055
Phenanthrene	0.028	0.487	0.087	0.087	0.228	0.196	0.293	0	47	0.102	0.450
Pyrene	0.028	0.157	0.000	0.027	0.039	0.029	0.054	40	47	0.024	0.605

MDL –Average Minimum Detection Limit; **Max** – highest sampled concentration; **Min** – lowest sampled concentration

Low Qnt – lowest quantitated concentration used to replace 0 in calculating mean and UCL for risk assessment

ND % – percent of samples where compound was reported as 0

UCL – 95% upper confidence limit; **Valid** – number of valid samples collected

SD – standard deviation; **CV** – coefficient of variation; **NA** – not applicable

^P Percentile values used in place of arithmetic mean

Table B4-3: PAH data summary at Seaford

Compound	Concentration ($\mu\text{g}/\text{m}^3$) ($\times 10^{-2}$)							ND %	Valid	SD ($\times 10^{-2}$)	CV
	MDL	Max	Min	Low Qnt	Mean	Median	UCL				
Seaford:											
1-Methylnaphthalene	0.028	0.517	0.000	0.029	0.091	0.045	0.155	30	43	0.097	1.065
2-Methylnaphthalene	0.028	0.544	0.000	0.030	0.146	0.097	0.233	12	43	0.132	0.904
Acenaphthene	0.028	0.209	0.000	0.027	0.049	0.035	0.076	37	43	0.040	0.801
Acenaphthylene	0.028	0.248	0.000	0.030	0.038 ^P	0.000	NA	65	43	0.063	1.646
Anthracene	0.028	0.043	0.000	0.043	NA	0.000	NA	98	43	0.007	NA
Benzo(a)anthracene	0.028	0.029	0.000	0.027	NA	0.000	NA	95	43	0.007	NA
Benzo(a)pyrene	0.028	0.043	0.000	0.043	NA	0.000	NA	98	43	0.006	NA
Benzo(b)fluoranthene	0.028	0.072	0.000	0.027	0.044 ^P	0.000	NA	56	43	0.026	0.587
Benzo(e)pyrene	0.028	0.044	0.000	0.030	NA	0.000	NA	91	43	0.011	NA
Benzo(g,h,i)perylene	0.028	0.054	0.000	0.038	NA	0.000	NA	95	43	0.010	NA
Benzo(k)fluoranthene	0.028	0.000	0.000	0.000	NA	0.000	NA	100	43	0.000	NA
Chrysene	0.028	0.044	0.000	0.030	0.027 ^P	0.000	NA	88	43	0.012	0.456
Dibenz(a,h)anthracene	0.028	0.000	0.000	0.000	NA	0.000	NA	100	43	0.000	NA
Fluoranthene	0.028	0.892	0.000	0.033	0.177	0.116	0.286	2	43	0.164	0.928
Fluorene	0.028	0.299	0.000	0.035	0.117	0.095	0.159	5	43	0.063	0.537
Indeno(1,2,3-cd)pyrene	0.028	0.074	0.000	0.044	NA	0.000	NA	91	43	0.017	NA
Naphthalene	0.028	0.787	0.000	0.032	0.148	0.077	0.258	19	43	0.166	1.119
Phenanthrene	0.028	1.030	0.000	0.083	0.434	0.399	0.585	0	43	0.226	0.522
Pyrene	0.028	0.446	0.000	0.027	0.027	0.071	0.138	14	43	0.075	2.775

MDL –Average Minimum Detection Limit; **Max** – highest sampled concentration; **Min** – lowest sampled concentration

Low Qnt – lowest quantitated concentration used to replace 0 in calculating mean and UCL for risk assessment

ND % – percent of samples where compound was reported as 0

UCL – 95% upper confidence limit; **Valid** – number of valid samples collected

SD – standard deviation; **CV** – coefficient of variation; **NA** – not applicable

^P Percentile values used in place of arithmetic mean

Table B5-1: Dioxins/Furans data summary at MLK and Delaware City

Compound	Concentration (pg/m ³)							ND %	Valid	SD	CV
	MDL	Max	Min	Low Qnt	Mean	Median	UCL				
MLK:											
1,2,3,4,6,7,8-HpCDD	0.010	0.474	0.014	0.014	0.134	0.114	0.226	0	24	0.103	0.771
1,2,3,4,6,7,8-HpCDF	0.010	0.788	0.004	0.004	0.090	0.048	0.227	0	24	0.154	1.704
1,2,3,4,7,8,9-HpCDF	0.010	0.019	0.000	0.004	0.006 ^P	0.004	NA	50	24	0.004	0.584
1,2,3,4,7,8-HxCDD	0.010	0.020	0.000	0.001	0.007	0.006	0.010	8	24	0.004	0.594
1,2,3,4,7,8-HxCDF	0.010	0.021	0.000	0.001	0.011	0.011	0.016	4	24	0.005	0.476
1,2,3,6,7,8-HxCDD	0.010	0.032	0.000	0.001	0.013	0.011	0.019	4	24	0.007	0.593
1,2,3,6,7,8-HxCDF	0.010	0.032	0.000	0.001	0.011	0.010	0.017	4	24	0.007	0.606
1,2,3,7,8,9-HxCDD	0.010	0.032	0.000	0.001	0.011	0.010	0.017	4	24	0.007	0.619
1,2,3,7,8,9-HxCDF	0.010	0.010	0.000	0.001	0.005 ^P	0.002	NA	63	24	0.003	0.627
1,2,3,7,8-PeCDD	0.010	0.019	0.000	0.001	0.008	0.008	0.013	8	24	0.005	0.556
1,2,3,7,8-PeCDF	0.010	0.017	0.000	0.001	0.007	0.007	0.011	4	24	0.005	0.647
2,3,4,6,7,8-HxCDF	0.010	0.030	0.000	0.001	0.012	0.010	0.019	4	24	0.008	0.632
2,3,4,7,8-PeCDF	0.010	0.027	0.000	0.001	0.009	0.008	0.015	8	24	0.007	0.735
2,3,7,8-TCDD	0.002	0.003	0.000	0.000	0.002	0.002	0.002	13	24	0.001	0.579
2,3,7,8-TCDF	0.002	0.030	0.001	0.001	0.008	0.007	0.013	0	24	0.005	0.660
OCDD	0.019	1.927	0.052	0.052	0.483	0.389	0.826	0	24	0.385	0.797
OCDF	0.019	0.107	0.000	0.003	0.040	0.037	0.061	4	24	0.024	0.591
Delaware City:											
1,2,3,4,6,7,8-HpCDD	0.010	0.462	0.019	0.019	0.089	0.075	0.164	0	25	0.086	0.960
1,2,3,4,6,7,8-HpCDF	0.010	0.158	0.009	0.009	0.034	0.028	0.060	0	25	0.029	0.849
1,2,3,4,7,8,9-HpCDF	0.010	0.158	0.000	0.002	0.004 ^P	0.003	NA	56	25	0.002	0.506
1,2,3,4,7,8-HxCDD	0.010	0.016	0.000	0.003	0.005	0.004	0.007	44	25	0.003	0.540
1,2,3,4,7,8-HxCDF	0.010	0.012	0.000	0.002	0.007	0.006	0.009	8	25	0.003	0.417
1,2,3,6,7,8-HxCDD	0.010	0.034	0.000	0.004	0.008	0.006	0.013	8	25	0.006	0.785
1,2,3,6,7,8-HxCDF	0.010	0.011	0.000	0.004	0.007	0.007	0.008	12	25	0.002	0.293
1,2,3,7,8,9-HxCDD	0.010	0.032	0.000	0.004	0.008	0.007	0.13	12	25	0.006	0.745
1,2,3,7,8,9-HxCDF	0.010	0.006	0.000	0.001	0.002 ^P	0.002	NA	88	25	0.001	0.590
1,2,3,7,8-PeCDD	0.010	0.018	0.000	0.004	0.005	0.005	0.008	28	25	0.003	0.545
1,2,3,7,8-PeCDF	0.010	0.010	0.000	0.002	0.004 ^P	0.003	NA	52	25	0.002	0.539
2,3,4,6,7,8-HxCDF	0.010	0.013	0.000	0.004	0.007	0.006	0.009	20	25	0.003	0.377
2,3,4,7,8-PeCDF	0.010	0.012	0.000	0.003	0.005	0.005	0.007	20	25	0.002	0.378
2,3,7,8-TCDD	0.002	0.003	0.000	0.000	0.001 ^P	0.001	NA	56	25	0.001	0.623
2,3,7,8-TCDF	0.002	0.009	0.003	0.003	0.004	0.003	0.005	0	25	0.001	0.342
OCDD	0.019	1.387	0.067	0.067	0.296	0.268	0.516	0	25	0.252	0.851
OCDF	0.019	0.044	0.000	0.007	0.022	0.019	0.032	8	25	0.011	0.520

MDL –Average Minimum Detection Limit; **Max** – highest sampled concentration; **Min** – lowest sampled concentration

Low Qnt – lowest quantitated concentration used to replace 0 in calculating mean and UCL for risk assessment

ND % – percent of samples where compound was reported as 0

UCL – 95% upper confidence limit; **Valid** – number of valid samples collected

SD – standard deviation; **CV** – coefficient of variation; **NA** – not applicable

^P Percentile values used in place of arithmetic mean

Table B5-2: Dioxins/Furans data summary at Lums Pond and Killens Pond

Compound	Concentration (pg/m ³)							ND %	Valid	SD	CV
	MDL	Max	Min	Low Qnt	Mean	Median	UCL				
Lums Pond:											
1,2,3,4,6,7,8-HpCDD	0.010	0.688	0.015	0.015	0.122	0.081	0.263	0	23	0.151	1.240
1,2,3,4,6,7,8-HpCDF	0.010	0.276	0.016	0.016	0.041	0.026	0.093	0	23	0.055	1.348
1,2,3,4,7,8,9-HpCDF	0.010	0.008	0.000	0.000	0.003 ^P	0.002	NA	70	23	0.003	0.907
1,2,3,4,7,8-HxCDD	0.010	0.029	0.000	0.003	0.007	0.003	0.013	43	23	0.007	1.073
1,2,3,4,7,8-HxCDF	0.010	0.013	0.000	0.004	0.006	0.005	0.008	17	23	0.002	0.353
1,2,3,6,7,8-HxCDD	0.010	0.046	0.000	0.004	0.010	0.006	0.020	22	23	0.010	1.004
1,2,3,6,7,8-HxCDF	0.010	0.011	0.000	0.004	0.006	0.006	0.008	9	23	0.002	0.377
1,2,3,7,8,9-HxCDD	0.010	0.046	0.000	0.004	0.010	0.006	0.020	35	23	0.011	1.045
1,2,3,7,8,9-HxCDF	0.010	0.005	0.000	0.000	0.002 ^P	0.002	NA	78	23	0.002	0.758
1,2,3,7,8-PeCDD	0.010	0.023	0.000	0.000	0.007 ^P	0.003	NA	52	23	0.007	1.001
1,2,3,7,8-PeCDF	0.010	0.006	0.000	0.000	0.005 ^P	0.003	NA	52	23	0.003	0.507
2,3,4,6,7,8-HxCDF	0.010	0.016	0.000	0.004	0.007	0.006	0.010	22	23	0.003	0.507
2,3,4,7,8-PeCDF	0.010	0.011	0.000	0.004	0.005	0.004	0.007	30	23	0.002	0.373
2,3,7,8-TCDD	0.002	0.005	0.000	0.001	0.001	0.001	0.002	43	23	0.001	0.852
2,3,7,8-TCDF	0.002	0.010	0.000	0.000	0.004	0.003	0.005	0	23	0.002	0.442
OCDD	0.019	2.025	0.000	0.000	0.375	0.267	0.772	0	23	0.427	1.136
OCDF	0.019	0.061	0.000	0.008	0.021	0.018	0.033	9	23	0.012	0.577
Killens Pond:											
1,2,3,4,6,7,8-HpCDD	0.010	3.775	0.004	0.004	0.236	0.053	0.883	0	25	0.742	3.152
1,2,3,4,6,7,8-HpCDF	0.010	0.689	0.003	0.003	0.070	0.023	0.205	0	25	0.156	2.243
1,2,3,4,7,8,9-HpCDF	0.010	0.009	0.000	0.004	0.004 ^P	0.002	NA	76	25	0.003	0.683
1,2,3,4,7,8-HxCDD	0.010	0.012	0.000	0.003	0.004 ^P	0.002	NA	56	25	0.004	0.951
1,2,3,4,7,8-HxCDF	0.010	0.181	0.000	0.004	0.013	0.005	0.043	24	25	0.035	2.746
1,2,3,6,7,8-HxCDD	0.010	0.246	0.000	0.004	0.017	0.004	0.059	36	25	0.048	2.843
1,2,3,6,7,8-HxCDF	0.010	0.015	0.000	0.003	0.005	0.004	0.008	44	25	0.003	0.591
1,2,3,7,8,9-HxCDD	0.010	0.230	0.000	0.004	0.016	0.005	0.055	36	25	0.045	2.801
1,2,3,7,8,9-HxCDF	0.010	0.005	0.000	0.000	0.002 ^P	0.002	NA	88	25	0.001	0.497
1,2,3,7,8-PeCDD	0.010	0.014	0.000	0.000	0.004 ^P	0.002	NA	68	25	0.003	0.855
1,2,3,7,8-PeCDF	0.010	0.009	0.000	0.000	0.004 ^P	0.002	NA	72	25	0.002	0.477
2,3,4,6,7,8-HxCDF	0.010	0.016	0.000	0.004	0.006	0.005	0.010	28	25	0.004	0.615
2,3,4,7,8-PeCDF	0.010	0.011	0.000	0.000	0.007 ^P	0.004	NA	52	25	0.003	0.395
2,3,7,8-TCDD	0.002	0.004	0.000	0.000	0.005 ^P	0.001	NA	60	25	0.001	0.177
2,3,7,8-TCDF	0.002	0.064	0.001	0.001	0.005	0.002	0.016	0	25	0.012	2.293
OCDD	0.019	14.117	0.028	0.028	0.828	0.186	3.249	0	25	2.778	3.356
OCDF	0.019	0.640	0.000	0.008	0.046	0.017	0.154	4	25	0.124	2.729

MDL –Average Minimum Detection Limit; **Max** – highest sampled concentration; **Min** – lowest sampled concentration

Low Qnt – lowest quantitated concentration used to replace 0 in calculating mean and UCL for risk assessment

ND % – percent of samples where compound was reported as 0

UCL – 95% upper confidence limit; **Valid** – number of valid samples collected

SD – standard deviation; **CV** – coefficient of variation; **NA** – not applicable

^P Percentile values used in place of arithmetic mean

Table B5-3: Dioxins/Furans data summary at Seaford

Compound	Concentration (pg/m ³)							ND %	Valid	SD	CV
	MDL	Max	Min	Low Qnt	Mean	Median	UCL				
Seaford:											
1,2,3,4,6,7,8-HpCDD	0.010	0.292	0.000	0.024	0.088	0.065	0.145	4	25	0.063	0.712
1,2,3,4,6,7,8-HpCDF	0.010	0.208	0.005	0.005	0.037	0.028	0.074	0	25	0.041	1.115
1,2,3,4,7,8,9-HpCDF	0.010	0.007	0.000	0.004	0.004 ^P	0.002	NA	76	25	0.002	0.493
1,2,3,4,7,8-HxCDD	0.010	0.013	0.000	0.004	0.006 ^P	0.004	NA	52	25	0.004	0.608
1,2,3,4,7,8-HxCDF	0.010	0.020	0.000	0.003	0.007	0.006	0.010	20	25	0.004	0.559
1,2,3,6,7,8-HxCDD	0.010	0.019	0.000	0.004	0.007	0.006	0.011	16	25	0.004	0.498
1,2,3,6,7,8-HxCDF	0.010	0.010	0.000	0.003	0.006	0.006	0.007	16	25	0.002	0.334
1,2,3,7,8,9-HxCDD	0.010	0.019	0.000	0.003	0.007	0.006	0.010	16	25	0.004	0.557
1,2,3,7,8,9-HxCDF	0.010	0.005	0.000	0.003	NA	0.002	0.001	92	25	0.001	NA
1,2,3,7,8-PeCDD	0.010	0.012	0.000	0.003	0.006	0.005	0.008	32	25	0.003	0.464
1,2,3,7,8-PeCDF	0.010	0.013	0.000	0.003	0.005 ^P	0.002	NA	60	25	0.004	0.781
2,3,4,6,7,8-HxCDF	0.010	0.013	0.000	0.003	0.006	0.007	0.009	16	25	0.003	0.433
2,3,4,7,8-PeCDF	0.010	0.010	0.000	0.003	0.005	0.004	0.006	24	25	0.002	0.404
2,3,7,8-TCDD	0.002	0.003	0.000	0.001	0.001	0.001	0.002	32	25	0.001	0.489
2,3,7,8-TCDF	0.002	0.005	0.000	0.001	0.003	0.003	0.004	4	25	0.001	0.338
OCDD	0.019	0.846	0.000	0.058	0.279	0.212	0.461	4	25	0.204	0.730
OCDF	0.019	0.061	0.000	0.011	0.020	0.018	0.030	16	25	0.011	0.531

MDL –Average Minimum Detection Limit; **Max** – highest sampled concentration; **Min** – lowest sampled concentration

Low Qnt – lowest quantitated concentration used to replace 0 in calculating mean and UCL for risk assessment

ND % – percent of samples where compound was reported as 0

UCL – 95% upper confidence limit; **Valid** – number of valid samples collected

SD – standard deviation; **CV** – coefficient of variation; **NA** – not applicable

^P Percentile values were used for arithmetic mean calculations

Table C1-1: Risk of cancer at Martin Luther King Monitoring Station. Last row shows cumulative risk from all listed chemical compounds.

Chemical Compound	Adult Estimated Dose	Adult Cancer Risk	Child Estimated Dose	Child Cancer Risk	Age-adjusted Estimated Dose	Age-adjusted Cancer Risk
1,1,2,2-Tetrachloroethane	1.17E-06	2.35E-07	5.14E-07	1.03E-07	1.60E-06	3.19E-07
1,2,3,4,6,7,8-HpCDD	1.57E-13	2.36E-08	6.88E-14	1.03E-08	2.14E-13	3.21E-08
1,2,3,4,7,8,9-HpCDF	7.48E-15	1.12E-09	3.27E-15	4.91E-10	1.02E-14	1.53E-09
1,2,3,4,7,8-HxCDD	8.23E-14	1.23E-08	3.60E-14	5.40E-09	1.12E-13	1.68E-08
1,2,3,4,7,8-HxCDF	1.32E-13	1.97E-08	5.75E-14	8.63E-09	1.79E-13	2.68E-08
1,2,3,6,7,8-HxCDD	1.48E-13	2.22E-08	6.47E-14	9.71E-09	2.01E-13	3.02E-08
1,2,3,6,7,8-HxCDF	1.33E-13	1.99E-08	5.80E-14	8.71E-09	1.80E-13	2.71E-08
1,2,3,7,8,9-HxCDD	1.30E-13	1.95E-08	5.70E-14	8.55E-09	1.77E-13	2.66E-08
1,2,3,7,8,9-HxCDF	5.77E-14	8.65E-09	2.52E-14	3.78E-09	7.84E-14	1.18E-08
1,2,3,7,8-PeCDD	9.84E-13	1.48E-07	4.30E-13	6.46E-08	1.34E-12	2.01E-07
1,2,3,7,8-PeCDF	4.25E-14	6.38E-09	1.86E-14	2.79E-09	5.78E-14	8.67E-09
1,2-Dibromoethane	1.17E-06	8.92E-07	5.14E-07	3.90E-07	1.60E-06	1.21E-06
1,2-Dichloroethane	5.33E-06	4.85E-07	2.33E-06	2.12E-07	7.25E-06	6.60E-07
1,3-Butadiene	3.53E-05	3.53E-06	1.55E-05	1.55E-06	4.81E-05	4.81E-06
1,4-Dichlorobenzene	2.62E-05	5.76E-07	1.15E-05	2.52E-07	3.56E-05	7.84E-07
2,3,4,6,7,8-HxCDF	1.42E-13	2.13E-08	6.22E-14	9.32E-09	1.93E-13	2.90E-08
2,3,7,8-TCDD	1.83E-13	2.75E-08	8.01E-14	1.20E-08	2.49E-13	3.74E-08
2,3,7,8-TCDF	9.43E-14	1.41E-08	4.13E-14	6.19E-09	1.28E-13	1.92E-08
Acetaldehyde	2.11E-07	1.63E-09	9.25E-08	7.12E-10	2.88E-07	2.21E-09
Arsenic	1.32E-07	1.99E-06	5.75E-08	8.69E-07	1.79E-07	2.70E-06
Benzene	1.62E-04	4.37E-06	7.09E-05	1.91E-06	2.20E-04	5.95E-06
Benzo(a)anthracene	5.11E-09	1.58E-08	2.23E-09	6.93E-09	6.95E-09	2.15E-08
Benzo(a)pyrene	6.19E-08	1.92E-07	2.71E-08	8.39E-08	8.42E-08	2.61E-07
Benzo(b)fluoranthene	1.26E-08	3.89E-08	5.50E-09	1.70E-08	1.71E-08	5.30E-08
Benzo(k)fluoranthene	2.98E-10	9.25E-10	1.30E-10	4.04E-10	4.06E-10	1.26E-09
Cadmium	3.35E-08	2.11E-07	1.46E-08	9.22E-08	4.55E-08	2.87E-07
Carbon tetrachloride	6.35E-05	3.37E-06	2.78E-05	1.47E-06	8.64E-05	4.58E-06
Chloroform	1.55E-05	1.26E-06	6.78E-06	5.49E-07	2.11E-05	1.71E-06
Chromium	1.57E-07	6.42E-06	6.85E-08	2.81E-06	2.13E-07	8.73E-06
Chrysene	8.81E-11	2.73E-10	3.85E-11	1.19E-10	1.20E-10	3.71E-10
Formaldehyde	2.88E-07	1.29E-08	1.26E-07	5.66E-09	3.91E-07	1.76E-08
Hexachloro-1,3-Butadiene	1.26E-05	9.80E-07	5.50E-06	4.29E-07	1.71E-05	1.33E-06
Indeno(1,2,3-cd)pyrene	6.93E-09	2.15E-08	3.03E-09	9.40E-09	9.42E-09	2.92E-08
OCDD	5.67E-15	8.51E-10	2.48E-15	3.72E-10	7.71E-15	1.16E-09
OCDF	4.67E-15	7.01E-10	2.04E-15	3.07E-10	6.36E-15	9.54E-10
Perchloroethylene	6.27E-05	1.25E-06	2.74E-05	5.49E-07	8.53E-05	1.71E-06
Trichloroethene	1.29E-05	5.17E-06	5.65E-06	2.26E-06	1.76E-05	7.03E-06
Vinyl Chloride	6.47E-06	9.70E-08	2.83E-06	8.49E-08	4.24E-05	1.27E-06
		3.15E-05		1.38E-05		4.39E-05

Table C1-2: Risk of non-cancer effects at Martin Luther King Monitoring Station. Last row shows cumulative risk from all listed chemical compounds.

Chemical Compound	Adult Estimated Dose	Adult Non-cancer Risk	Child Estimated Dose	Child Non-cancer Risk	Age-adjusted Estimated Dose	Age-adjusted Non-cancer Risk
1,1,1-Trichloroethane	4.71E-05	7.48E-05	1.03E-04	1.64E-04	6.41E-05	1.02E-04
1,1,2-Trichloro-1,2,2-trifluoroethane	1.71E-04	1.99E-05	3.75E-04	4.36E-05	2.33E-04	2.71E-05
1,1-Dichloroethane	5.48E-06	3.91E-05	1.20E-05	8.56E-05	7.45E-06	5.32E-05
1,1-Dichloroethene	1.64E-06	2.74E-05	3.60E-06	5.99E-05	2.24E-06	3.73E-05
1,2,4-Trichlorobenzene	2.24E-05	2.24E-02	4.91E-05	4.91E-02	3.05E-05	3.05E-02
1,2,4-Trimethylbenzene	2.15E-04	1.26E-01	4.70E-04	2.76E-01	2.92E-04	1.72E-01
1,2-Dibromoethane	2.74E-06	4.81E-02	5.99E-06	1.05E-01	3.73E-06	6.54E-02
1,2-Dichlorobenzene	1.96E-05	4.90E-04	4.29E-05	1.07E-03	2.66E-05	6.66E-04
1,2-Dichloroethane	1.24E-05	8.88E-03	2.72E-05	1.94E-02	1.69E-05	1.21E-02
1,2-Dichloropropane	2.74E-06	2.40E-03	5.99E-06	5.26E-03	3.73E-06	3.27E-03
1,3,5-Trimethylbenzene	6.49E-05	3.82E-02	1.42E-04	8.36E-02	8.83E-05	5.20E-02
1,3-Butadiene	8.25E-05	1.45E-01	1.80E-04	3.16E-01	1.12E-04	1.97E-01
1,3-dichlorobenzene	1.79E-05	5.96E-03	3.91E-05	1.30E-02	2.43E-05	8.11E-03
1,4-Dichlorobenzene	6.11E-05	2.67E-04	1.34E-04	5.84E-04	8.31E-05	3.63E-04
2-Methylnaphthalene	1.23E-06	3.08E-04	2.69E-06	6.73E-04	1.67E-06	4.18E-04
Acenaphthene	5.70E-07	9.50E-06	1.25E-06	2.08E-05	7.75E-07	1.29E-05
Acetaldehyde	4.93E-07	1.92E-04	1.08E-06	4.20E-04	6.71E-07	2.61E-04
Acetone	1.40E-06	1.55E-06	3.06E-06	3.40E-06	1.90E-06	2.11E-06
Anthracene	2.46E-07	8.19E-07	5.38E-07	1.79E-06	3.34E-07	1.11E-06
Benzene	3.78E-04	4.40E-02	8.27E-04	9.62E-02	5.14E-04	5.98E-02
Bromomethane	2.44E-05	1.74E-02	5.33E-05	3.81E-02	3.32E-05	2.37E-02
Cadmium	7.81E-08	1.37E-03	1.71E-07	3.00E-03	1.06E-07	1.86E-03
Carbon tetrachloride	1.48E-04	2.60E-01	3.24E-04	5.68E-01	2.02E-04	3.53E-01
Chlorobenzene	1.39E-05	8.19E-04	3.04E-05	1.79E-03	1.89E-05	1.11E-03
Chloroethane	1.28E-05	4.43E-06	2.81E-05	9.69E-06	1.75E-05	6.03E-06
Chloroform	3.62E-05	2.58E-03	7.91E-05	5.65E-03	4.92E-05	3.51E-03
Chloromethane	3.07E-04	1.18E-02	6.71E-04	2.58E-02	4.17E-04	1.61E-02
Chromium	3.65E-07	1.22E-02	7.99E-07	2.66E-02	4.97E-07	1.66E-02
Cis-1,2-Dichloroethene	2.74E-06	2.74E-04	5.99E-06	5.99E-04	3.73E-06	3.73E-04
Dichlorodifluoromethane	7.40E-04	1.48E-02	1.62E-03	3.24E-02	1.01E-03	2.01E-02
Ethylbenzene	1.81E-04	6.24E-04	3.96E-04	1.36E-03	2.46E-04	8.48E-04
Fluoranthene	3.12E-06	7.81E-05	6.83E-06	1.71E-04	4.25E-06	1.06E-04
Fluorene	1.28E-06	3.21E-05	2.80E-06	7.01E-05	1.74E-06	4.36E-05
Manganese	5.15E-06	3.60E-01	1.13E-05	7.88E-01	7.01E-06	4.90E-01
Methyl Ethyl Ketone	2.69E-07	1.92E-07	5.89E-07	4.20E-07	3.66E-07	2.61E-07
Methyl Isobutyl Ketone	2.19E-08	2.54E-08	4.78E-08	5.56E-08	2.97E-08	3.46E-08
Naphthalene	8.68E-07	4.34E-05	1.90E-06	9.50E-05	1.18E-06	5.91E-05
Naphthalene	8.68E-07	9.65E-04	1.90E-06	2.11E-03	1.18E-06	1.31E-03
Nickel	1.90E-06	9.51E-05	4.16E-06	2.08E-04	2.59E-06	1.29E-04
Perchloroethylene	1.46E-04	1.05E-03	3.20E-04	2.29E-03	1.99E-04	1.42E-03
Styrene	4.66E-05	1.63E-04	1.02E-04	3.56E-04	6.34E-05	2.22E-04
Toluene	1.12E-03	9.78E-03	2.44E-03	2.14E-02	1.52E-03	1.33E-02
Trichloroethene	3.01E-05	3.01E-03	6.59E-05	6.59E-03	4.10E-05	4.10E-03
Trichlorofluoromethane	4.08E-04	2.04E-03	8.93E-04	4.46E-03	5.55E-04	2.78E-03
Vinyl Chloride	1.51E-05	5.39E-04	3.30E-05	1.18E-03	2.05E-05	7.33E-04
Xylenes	7.91E-04	2.64E-02	1.73E-03	5.77E-02	1.08E-03	3.59E-02
		1.17E+00		2.56E+00		1.59E+00

Table C2-1: Risk of cancer at Delaware City Monitoring Station. Last row shows cumulative risk from all listed chemical compounds.

Chemical Compound	Adult Estimated Dose	Adult Cancer Risk	Child Estimated Dose	Child Cancer Risk	Age-adjusted Estimated Dose	Age-adjusted Cancer Risk
1,1,2,2-Tetrachloroethane	1.17E-06	2.35E-07	5.14E-07	1.03E-07	1.60E-06	3.19E-07
1,2,3,4,6,7,8-HpCDD	1.05E-13	1.57E-08	4.58E-14	6.87E-09	1.42E-13	2.13E-08
1,2,3,4,7,8,9-HpCDF	5.06E-15	7.59E-10	2.21E-15	3.32E-10	6.88E-15	1.03E-09
1,2,3,4,7,8-HxCDD	5.75E-14	8.63E-09	2.52E-14	3.78E-09	7.83E-14	1.17E-08
1,2,3,4,7,8-HxCDF	7.87E-14	1.18E-08	3.44E-14	5.16E-09	1.07E-13	1.61E-08
1,2,3,6,7,8-HxCDD	8.98E-14	1.35E-08	3.93E-14	5.89E-09	1.22E-13	1.83E-08
1,2,3,6,7,8-HxCDF	7.86E-14	1.18E-08	3.44E-14	5.15E-09	1.07E-13	1.60E-08
1,2,3,7,8,9-HxCDD	8.90E-14	1.34E-08	3.89E-14	5.84E-09	1.21E-13	1.82E-08
1,2,3,7,8,9-HxCDF	2.43E-14	3.65E-09	1.06E-14	1.60E-09	3.31E-14	4.96E-09
1,2,3,7,8-PeCDD	6.46E-13	9.69E-08	2.83E-13	4.24E-08	8.78E-13	1.32E-07
1,2,3,7,8-PeCDF	2.63E-14	3.95E-09	1.15E-14	1.73E-09	3.58E-14	5.37E-09
1,2-Dichloroethane	5.89E-06	5.36E-07	2.58E-06	2.35E-07	8.02E-06	7.30E-07
1,3-Butadiene	1.13E-05	1.13E-06	4.95E-06	4.95E-07	1.54E-05	1.54E-06
1,4-Dichlorobenzene	1.89E-05	4.16E-07	8.27E-06	1.82E-07	2.57E-05	5.66E-07
2,3,4,6,7,8-HxCDF	8.36E-14	1.25E-08	3.66E-14	5.49E-09	1.14E-13	1.71E-08
2,3,7,8-TCDD	1.30E-13	1.95E-08	5.70E-14	8.55E-09	1.77E-13	2.66E-08
2,3,7,8-TCDF	4.59E-14	6.89E-09	2.01E-14	3.01E-09	6.25E-14	9.37E-09
Acetaldehyde	1.63E-07	1.26E-09	7.14E-08	5.50E-10	2.22E-07	1.71E-09
Arsenic	1.20E-07	1.81E-06	5.24E-08	7.91E-07	1.63E-07	2.46E-06
Benzene	1.24E-04	3.36E-06	5.45E-05	1.47E-06	1.69E-04	4.57E-06
Benzo(a)anthracene	2.88E-09	8.92E-09	1.26E-09	3.90E-09	3.91E-09	1.21E-08
Benzo(b)fluoranthene	5.85E-09	1.81E-08	2.56E-09	7.93E-09	7.95E-09	2.47E-08
Cadmium	2.77E-08	1.75E-07	1.21E-08	7.64E-08	3.77E-08	2.37E-07
Carbon tetrachloride	6.29E-05	3.34E-06	2.75E-05	1.46E-06	8.56E-05	4.54E-06
Chloroform	1.12E-05	9.11E-07	4.92E-06	3.99E-07	1.53E-05	1.24E-06
Chromium	9.25E-08	3.79E-06	4.05E-08	1.66E-06	1.26E-07	5.16E-06
Chrysene	3.96E-11	1.23E-10	1.73E-11	5.37E-11	5.38E-11	1.67E-10
Formaldehyde	1.49E-07	6.71E-09	6.52E-08	2.94E-09	2.03E-07	9.13E-09
Hexachloro-1,3-Butadiene	1.35E-05	1.05E-06	5.91E-06	4.61E-07	1.84E-05	1.43E-06
OCDD	3.48E-15	5.21E-10	1.52E-15	2.28E-10	4.73E-15	7.09E-10
OCDF	2.57E-15	3.86E-10	1.13E-15	1.69E-10	3.50E-15	5.25E-10
Perchloroethylene	1.71E-05	3.43E-07	7.50E-06	1.50E-07	2.33E-05	4.66E-07
Trichloroethene	1.06E-05	4.26E-06	4.65E-06	1.86E-06	1.45E-05	5.79E-06
Vinyl Chloride	2.67E-05	4.00E-07	1.17E-05	3.50E-07	1.75E-04	5.25E-06
		2.20E-05		9.80E-06		3.46E-05

Table C2-2: Risk of non-cancer effects at Delaware City Monitoring Station. Last row shows cumulative risk from all listed chemical compounds.

Chemical Compound	Adult Estimated Dose	Adult Non-cancer Risk	Child Estimated Dose	Child Non-cancer Risk	Age-adjusted Estimated Dose	Age-adjusted Non-cancer Risk
1,1,1-Trichloroethane	4.16E-05	6.61E-05	9.11E-05	1.45E-04	5.66E-05	8.99E-05
1,1,2-Trichloro-1,2,2-trifluoroethane	1.67E-04	1.94E-05	3.66E-04	4.25E-05	2.27E-04	2.64E-05
1,1-Dichloroethane	5.48E-06	3.91E-05	1.20E-05	8.56E-05	7.45E-06	5.32E-05
1,2,4-Trichlorobenzene	2.79E-05	2.79E-02	6.11E-05	6.11E-02	3.80E-05	3.80E-02
1,2,4-Trimethylbenzene	8.63E-05	5.08E-02	1.89E-04	1.11E-01	1.17E-04	6.91E-02
1,2-Dichlorobenzene	2.62E-05	6.56E-04	5.74E-05	1.44E-03	3.57E-05	8.93E-04
1,2-Dichloroethane	1.38E-05	9.82E-03	3.01E-05	2.15E-02	1.87E-05	1.34E-02
1,3,5-Trimethylbenzene	3.07E-05	1.80E-02	6.71E-05	3.95E-02	4.17E-05	2.46E-02
1,3-Butadiene	2.64E-05	4.63E-02	5.77E-05	1.01E-01	3.59E-05	6.30E-02
1,3-dichlorobenzene	2.04E-05	6.81E-03	4.47E-05	1.49E-02	2.78E-05	9.27E-03
1,4-Dichlorobenzene	4.41E-05	1.93E-04	9.65E-05	4.21E-04	6.00E-05	2.62E-04
2-Methylnaphthalene	5.10E-07	1.27E-04	1.11E-06	2.79E-04	6.93E-07	1.73E-04
Acenaphthene	1.26E-07	2.10E-06	2.75E-07	4.58E-06	1.71E-07	2.85E-06
Acetaldehyde	3.81E-07	1.48E-04	8.33E-07	3.24E-04	5.18E-07	2.02E-04
Acetone	1.07E-06	1.19E-06	2.34E-06	2.60E-06	1.45E-06	1.62E-06
Benzene	2.90E-04	3.38E-02	6.35E-04	7.39E-02	3.95E-04	4.59E-02
Bromomethane	1.48E-05	1.05E-02	3.23E-05	2.31E-02	2.01E-05	1.43E-02
Cadmium	6.47E-08	1.13E-03	1.41E-07	2.48E-03	8.80E-08	1.54E-03
Carbon tetrachloride	1.47E-04	2.57E-01	3.21E-04	5.63E-01	2.00E-04	3.50E-01
Chlorobenzene	2.68E-05	1.58E-03	5.87E-05	3.45E-03	3.65E-05	2.15E-03
Chloroethane	1.00E-05	3.46E-06	2.19E-05	7.56E-06	1.36E-05	4.70E-06
Chloroform	2.62E-05	1.87E-03	5.74E-05	4.10E-03	3.57E-05	2.55E-03
Chloromethane	3.15E-04	1.21E-02	6.89E-04	2.65E-02	4.29E-04	1.65E-02
Chromium	2.16E-07	7.19E-03	4.72E-07	1.57E-02	2.93E-07	9.78E-03
Dichlorodifluoromethane	7.07E-04	1.41E-02	1.55E-03	3.09E-02	9.62E-04	1.92E-02
Ethylbenzene	7.29E-05	2.51E-04	1.59E-04	5.50E-04	9.91E-05	3.42E-04
Fluoranthene	3.10E-07	7.74E-06	6.77E-07	1.69E-05	4.21E-07	1.05E-05
Fluorene	3.51E-07	8.77E-06	7.67E-07	1.92E-05	4.77E-07	1.19E-05
Manganese	1.89E-06	1.32E-01	4.14E-06	2.90E-01	2.58E-06	1.80E-01
Methyl Ethyl Ketone	2.03E-07	1.45E-07	4.45E-07	3.18E-07	2.77E-07	1.98E-07
Naphthalene	4.47E-07	2.23E-05	9.77E-07	4.88E-05	6.07E-07	3.04E-05
Naphthalene	4.47E-07	4.96E-04	9.77E-07	1.09E-03	6.07E-07	6.75E-04
Nickel	1.25E-06	6.27E-05	2.74E-06	1.37E-04	1.71E-06	8.53E-05
Perchloroethylene	4.00E-05	2.86E-04	8.75E-05	6.25E-04	5.44E-05	3.89E-04
Styrene	2.90E-05	1.02E-04	6.35E-05	2.22E-04	3.95E-05	1.38E-04
Toluene	6.68E-04	5.86E-03	1.46E-03	1.28E-02	9.09E-04	7.98E-03
Trichloroethene	2.48E-05	2.48E-03	5.43E-05	5.43E-03	3.38E-05	3.38E-03
Trichlorofluoromethane	3.78E-04	1.89E-03	8.27E-04	4.14E-03	5.14E-04	2.57E-03
Vinyl Chloride	6.22E-05	2.22E-03	1.36E-04	4.86E-03	8.46E-05	3.02E-03
Xylenes	2.69E-04	8.95E-03	5.87E-04	1.96E-02	3.65E-04	1.22E-02
		6.56E-01		1.43E+00		8.92E-01

Table C3-1: Risk of cancer at Lums Pond Monitoring Station. Last row shows cumulative risk from all listed chemical compounds.

Chemical Compound	Adult Estimated Dose	Adult Cancer Risk	Child Estimated Dose	Child Cancer Risk	Age-adjusted Estimated Dose	Age-adjusted Cancer Risk
1,1,2,2-Tetrachloroethane	1.17E-06	2.35E-07	5.14E-07	1.03E-07	1.60E-06	3.19E-07
1,2,3,4,6,7,8-HpCDD	1.43E-13	2.15E-08	6.27E-14	9.40E-09	1.95E-13	2.92E-08
1,2,3,4,7,8-HxCDD	7.71E-14	1.16E-08	3.38E-14	5.06E-09	1.05E-13	1.57E-08
1,2,3,4,7,8-HxCDF	7.19E-14	1.08E-08	3.14E-14	4.72E-09	9.78E-14	1.47E-08
1,2,3,6,7,8-HxCDD	1.20E-13	1.80E-08	5.24E-14	7.86E-09	1.63E-13	2.44E-08
1,2,3,6,7,8-HxCDF	7.36E-14	1.10E-08	3.22E-14	4.83E-09	1.00E-13	1.50E-08
1,2,3,7,8,9-HxCDD	1.19E-13	1.78E-08	5.19E-14	7.78E-09	1.61E-13	2.42E-08
1,2,3,7,8-PeCDD	7.68E-13	1.15E-07	3.36E-13	5.04E-08	1.04E-12	1.57E-07
1,2,3,7,8-PeCDF	2.82E-14	4.23E-09	1.23E-14	1.85E-09	3.83E-14	5.75E-09
1,2-Dibromoethane	1.17E-06	8.92E-07	5.14E-07	3.90E-07	1.60E-06	1.21E-06
1,2-Dichloroethane	4.84E-06	4.40E-07	2.12E-06	1.93E-07	6.58E-06	5.99E-07
1,3-Butadiene	8.47E-06	8.47E-07	3.70E-06	3.70E-07	1.15E-05	1.15E-06
1,4-Dichlorobenzene	1.08E-05	2.38E-07	4.74E-06	1.04E-07	1.47E-05	3.24E-07
2,3,4,6,7,8-HxCDF	7.84E-14	1.18E-08	3.43E-14	5.15E-09	1.07E-13	1.60E-08
2,3,7,8-TCDD	1.60E-13	2.40E-08	6.99E-14	1.05E-08	2.17E-13	3.26E-08
2,3,7,8-TCDF	4.57E-14	6.85E-09	2.00E-14	3.00E-09	6.21E-14	9.32E-09
Acetaldehyde	1.69E-07	1.30E-09	7.40E-08	5.70E-10	2.30E-07	1.77E-09
Arsenic	1.06E-07	1.61E-06	4.65E-08	7.03E-07	1.45E-07	2.19E-06
Benzene	6.93E-05	1.87E-06	3.03E-05	8.18E-07	9.42E-05	2.54E-06
Benzo(b)fluoranthene	8.61E-10	2.67E-09	3.77E-10	1.17E-09	1.17E-09	3.63E-09
Cadmium	2.31E-08	1.46E-07	1.01E-08	6.38E-08	3.15E-08	1.98E-07
Carbon tetrachloride	6.36E-05	3.37E-06	2.78E-05	1.48E-06	8.66E-05	4.59E-06
Chloroform	1.03E-05	8.31E-07	4.49E-06	3.64E-07	1.40E-05	1.13E-06
Chromium	8.75E-08	3.59E-06	3.83E-08	1.57E-06	1.19E-07	4.88E-06
Chrysene	3.89E-11	1.20E-10	1.70E-11	5.27E-11	5.29E-11	1.64E-10
Formaldehyde	1.70E-07	7.66E-09	7.45E-08	3.35E-09	2.32E-07	1.04E-08
Hexachloro-1,3-Butadiene	1.26E-05	9.80E-07	5.50E-06	4.29E-07	1.71E-05	1.33E-06
OCDD	4.40E-15	6.60E-10	1.93E-15	2.89E-10	5.99E-15	8.98E-10
OCDF	2.51E-15	3.77E-10	1.10E-15	1.65E-10	3.42E-15	5.13E-10
Perchloroethylene	1.61E-05	3.22E-07	7.04E-06	1.41E-07	2.19E-05	4.38E-07
Trichloroethene	6.86E-06	2.74E-06	3.00E-06	1.20E-06	9.33E-06	3.73E-06
Vinyl Chloride	2.35E-06	3.52E-08	1.03E-06	3.08E-08	1.54E-05	4.62E-07
		1.84E-05		8.07E-06		2.55E-05

Table C3-2: Risk of non-cancer effects at Delaware City Monitoring Station. Last row shows cumulative risk from all listed chemical compounds.

Chemical Compound	Adult Estimated Dose	Adult Non-cancer Risk	Child Estimated Dose	Child Non-cancer Risk	Age-adjusted Estimated Dose	Age-adjusted Non-cancer Risk
1,1,1-Trichloroethane	4.11E-05	6.52E-05	8.99E-05	1.43E-04	5.59E-05	8.87E-05
1,1,2-Trichloro-1,2,2-trifluoroethane	1.69E-04	1.97E-05	3.70E-04	4.30E-05	2.30E-04	2.67E-05
1,2,4-Trichlorobenzene	2.03E-05	2.03E-02	4.45E-05	4.45E-02	2.77E-05	2.77E-02
1,2,4-Trimethylbenzene	4.71E-05	2.77E-02	1.03E-04	6.06E-02	6.41E-05	3.77E-02
1,2-Dibromoethane	2.74E-06	4.81E-02	5.99E-06	1.05E-01	3.73E-06	6.54E-02
1,2-Dichlorobenzene	1.93E-05	4.83E-04	4.23E-05	1.06E-03	2.63E-05	6.57E-04
1,2-Dichloroethane	1.13E-05	8.06E-03	2.47E-05	1.76E-02	1.54E-05	1.10E-02
1,3,5-Trimethylbenzene	1.90E-05	1.12E-02	4.17E-05	2.45E-02	2.59E-05	1.52E-02
1,3-Butadiene	1.98E-05	3.47E-02	4.32E-05	7.58E-02	2.69E-05	4.71E-02
1,3-dichlorobenzene	1.79E-05	5.96E-03	3.91E-05	1.30E-02	2.43E-05	8.11E-03
1,4-Dichlorobenzene	2.53E-05	1.10E-04	5.53E-05	2.42E-04	3.44E-05	1.50E-04
2-Methylnaphthalene	1.20E-06	3.00E-04	2.63E-06	6.56E-04	1.63E-06	4.08E-04
Acenaphthene	1.13E-07	1.88E-06	2.47E-07	4.12E-06	1.54E-07	2.56E-06
Acetaldehyde	3.95E-07	1.54E-04	8.63E-07	3.36E-04	5.37E-07	2.09E-04
Acetone	1.18E-06	1.31E-06	2.58E-06	2.86E-06	1.60E-06	1.78E-06
Benzene	1.62E-04	1.88E-02	3.54E-04	4.11E-02	2.20E-04	2.56E-02
Bromomethane	1.74E-05	1.24E-02	3.81E-05	2.72E-02	2.37E-05	1.69E-02
Cadmium	5.40E-08	9.47E-04	1.18E-07	2.07E-03	7.34E-08	1.29E-03
Carbon tetrachloride	1.48E-04	2.60E-01	3.25E-04	5.69E-01	2.02E-04	3.54E-01
Chlorobenzene	1.37E-05	8.06E-04	3.00E-05	1.76E-03	1.86E-05	1.10E-03
Chloroethane	9.48E-06	3.27E-06	2.07E-05	7.15E-06	1.29E-05	4.45E-06
Chloroform	2.39E-05	1.71E-03	5.24E-05	3.74E-03	3.26E-05	2.33E-03
Chloromethane	3.07E-04	1.18E-02	6.71E-04	2.58E-02	4.17E-04	1.61E-02
Chromium	2.04E-07	6.81E-03	4.47E-07	1.49E-02	2.78E-07	9.26E-03
Cis-1,2-Dichloroethene	2.74E-06	2.74E-04	5.99E-06	5.99E-04	3.73E-06	3.73E-04
Dichlorodifluoromethane	7.18E-04	1.44E-02	1.57E-03	3.14E-02	9.76E-04	1.95E-02
Ethylbenzene	3.75E-05	1.29E-04	8.21E-05	2.83E-04	5.11E-05	1.76E-04
Fluoranthene	6.99E-07	1.75E-05	1.53E-06	3.82E-05	9.50E-07	2.38E-05
Fluorene	7.59E-07	1.90E-05	1.66E-06	4.15E-05	1.03E-06	2.58E-05
Manganese	1.80E-06	1.26E-01	3.93E-06	2.75E-01	2.44E-06	1.71E-01
Methyl Ethyl Ketone	1.96E-07	1.40E-07	4.29E-07	3.07E-07	2.67E-07	1.91E-07
Naphthalene	1.65E-06	8.27E-05	3.62E-06	1.81E-04	2.25E-06	1.13E-04
Naphthalene	1.65E-06	1.84E-03	3.62E-06	4.02E-03	2.25E-06	2.50E-03
Nickel	8.16E-07	4.08E-05	1.79E-06	8.93E-05	1.11E-06	5.55E-05
Perchloroethylene	3.75E-05	2.68E-04	8.21E-05	5.86E-04	5.11E-05	3.65E-04
Styrene	2.77E-05	9.68E-05	6.05E-05	2.12E-04	3.76E-05	1.32E-04
Toluene	2.31E-04	2.03E-03	5.06E-04	4.44E-03	3.15E-04	2.76E-03
Trichloroethene	1.60E-05	1.60E-03	3.50E-05	3.50E-03	2.18E-05	2.18E-03
Trichlorofluoromethane	3.81E-04	1.90E-03	8.33E-04	4.17E-03	5.18E-04	2.59E-03
Vinyl chloride	5.48E-06	1.96E-04	1.20E-05	4.28E-04	7.45E-06	2.66E-04
Xylenes	1.28E-04	4.25E-03	2.79E-04	9.30E-03	1.73E-04	5.78E-03
		6.23E-01		1.36E+00		8.48E-01

Table C4-1: Risk of cancer at Killens Pond Monitoring Station. Last row shows cumulative risk from all listed chemical compounds.

Chemical Compound	Adult Estimated Dose	Adult Cancer Risk	Child Estimated Dose	Child Cancer Risk	Age-adjusted Estimated Dose	Age-adjusted Cancer Risk
1,1,2,2-Tetrachloroethane	1.17E-06	2.35E-07	5.14E-07	1.03E-07	1.60E-06	3.19E-07
1,2,3,4,6,7,8-HpCDD	2.77E-13	4.16E-08	1.21E-13	1.82E-08	3.77E-13	5.65E-08
1,2,3,4,7,8,9-HpCDF	4.74E-15	7.12E-10	2.08E-15	3.11E-10	6.45E-15	9.68E-10
1,2,3,4,7,8-HxCDD	5.25E-14	7.87E-09	2.30E-14	3.44E-09	7.14E-14	1.07E-08
1,2,3,4,7,8-HxCDF	1.50E-13	2.25E-08	6.58E-14	9.86E-09	2.04E-13	3.07E-08
1,2,3,6,7,8-HxCDD	1.98E-13	2.98E-08	8.68E-14	1.30E-08	2.70E-13	4.05E-08
1,2,3,6,7,8-HxCDF	6.06E-14	9.09E-09	2.65E-14	3.98E-09	8.24E-14	1.24E-08
1,2,3,7,8,9-HxCDD	1.88E-13	2.82E-08	8.22E-14	1.23E-08	2.56E-13	3.83E-08
1,2,3,7,8,9-HxCDF	2.40E-14	3.59E-09	1.05E-14	1.57E-09	3.26E-14	4.89E-09
1,2,3,7,8-PeCDD	4.53E-13	6.80E-08	1.98E-13	2.97E-08	6.17E-13	9.25E-08
1,2,3,7,8-PeCDF	2.08E-14	3.13E-09	9.12E-15	1.37E-09	2.84E-14	4.25E-09
1,2-Dibromoethane	1.17E-06	8.92E-07	5.14E-07	3.90E-07	1.60E-06	1.21E-06
1,2-Dichloroethane	4.91E-06	4.47E-07	2.15E-06	1.95E-07	6.68E-06	6.08E-07
1,3-Butadiene	5.24E-06	5.24E-07	2.29E-06	2.29E-07	7.12E-06	7.12E-07
1,4-Dichlorobenzene	9.09E-06	2.00E-07	3.98E-06	8.75E-08	1.24E-05	2.72E-07
2,3,4,6,7,8-HxCDF	7.50E-14	1.13E-08	3.28E-14	4.92E-09	1.02E-13	1.53E-08
2,3,7,8-TCDD	5.59E-13	8.38E-08	2.45E-13	3.67E-08	7.60E-13	1.14E-07
2,3,7,8-TCDF	6.31E-14	9.46E-09	2.76E-14	4.14E-09	8.58E-14	1.29E-08
Acetaldehyde	1.50E-07	1.16E-09	6.58E-08	5.06E-10	2.04E-07	1.57E-09
Arsenic	1.04E-07	1.57E-06	4.54E-08	6.86E-07	1.41E-07	2.13E-06
Benzene	5.46E-05	1.47E-06	2.39E-05	6.45E-07	7.43E-05	2.01E-06
Benzo(b)fluoranthene	1.62E-09	5.02E-09	7.09E-10	2.20E-09	2.20E-09	6.83E-09
Cadmium	2.32E-08	1.46E-07	1.02E-08	6.41E-08	3.16E-08	1.99E-07
Carbon tetrachloride	6.58E-05	3.48E-06	2.88E-05	1.52E-06	8.94E-05	4.74E-06
Chloroform	9.52E-06	7.71E-07	4.17E-06	3.37E-07	1.30E-05	1.05E-06
Chromium	7.27E-08	2.98E-06	3.18E-08	1.30E-06	9.90E-08	4.06E-06
Formaldehyde	1.47E-07	6.60E-09	6.42E-08	2.89E-09	2.00E-07	8.98E-09
Hexachloro-1,3-Butadiene	1.26E-05	9.80E-07	5.50E-06	4.29E-07	1.71E-05	1.33E-06
OCDD	9.72E-15	1.46E-09	4.25E-15	6.38E-10	1.32E-14	1.98E-09
OCDF	5.35E-15	8.03E-10	2.34E-15	3.51E-10	7.28E-15	1.09E-09
Perchloroethylene	1.27E-05	2.54E-07	5.55E-06	1.11E-07	1.73E-05	3.45E-07
Trichloroethene	1.13E-05	4.53E-06	4.96E-06	1.98E-06	1.54E-05	6.17E-06
Vinyl Chloride	5.87E-06	8.81E-08	2.57E-06	7.71E-08	3.85E-05	1.16E-06
		1.89E-05		8.31E-06		2.68E-05

Table C4-2: Risk of non-cancer effects at Killens Pond Monitoring Station. Last row shows cumulative risk from all listed chemical compounds.

Chemical Compound	Adult Estimated Dose	Adult Non-cancer Risk	Child Estimated Dose	Child Non-cancer Risk	Age-adjusted Estimated Dose	Age-adjusted Non-cancer Risk
1,1,1-Trichloroethane	4.00E-05	6.35E-05	8.75E-05	1.39E-04	5.44E-05	8.64E-05
1,1,2-Trichloro-1,2,2-trifluoroethane	1.67E-04	1.94E-05	3.66E-04	4.25E-05	2.27E-04	2.64E-05
1,2,4-Trichlorobenzene	2.41E-05	2.41E-02	5.28E-05	5.28E-02	3.28E-05	3.28E-02
1,2,4-Trimethylbenzene	3.40E-05	2.00E-02	7.43E-05	4.37E-02	4.62E-05	2.72E-02
1,2-Dibromoethane	2.74E-06	4.81E-02	5.99E-06	1.05E-01	3.73E-06	6.54E-02
1,2-Dichlorobenzene	1.73E-05	4.33E-04	3.79E-05	9.47E-04	2.36E-05	5.89E-04
1,2-Dichloroethane	1.15E-05	8.18E-03	2.51E-05	1.79E-02	1.56E-05	1.11E-02
1,3,5-Trimethylbenzene	1.53E-05	8.99E-03	3.34E-05	1.97E-02	2.08E-05	1.22E-02
1,3-Butadiene	1.22E-05	2.14E-02	2.67E-05	4.69E-02	1.66E-05	2.92E-02
1,3-dichlorobenzene	1.70E-05	5.68E-03	3.73E-05	1.24E-02	2.32E-05	7.73E-03
1,4-Dichlorobenzene	2.12E-05	9.26E-05	4.64E-05	2.03E-04	2.88E-05	1.26E-04
2-Methylnaphthalene	2.70E-07	6.75E-05	5.91E-07	1.48E-04	3.67E-07	9.19E-05
Acenaphthene	7.78E-08	1.30E-06	1.70E-07	2.84E-06	1.06E-07	1.76E-06
Acetaldehyde	3.51E-07	1.36E-04	7.67E-07	2.98E-04	4.77E-07	1.86E-04
Acetone	1.04E-06	1.16E-06	2.28E-06	2.53E-06	1.42E-06	1.57E-06
Benzene	1.27E-04	1.48E-02	2.79E-04	3.24E-02	1.73E-04	2.02E-02
Bromomethane	1.35E-05	9.67E-03	2.96E-05	2.11E-02	1.84E-05	1.32E-02
Cadmium	5.42E-08	9.52E-04	1.19E-07	2.08E-03	7.38E-08	1.29E-03
Carbon tetrachloride	1.53E-04	2.69E-01	3.36E-04	5.88E-01	2.09E-04	3.66E-01
Chlorobenzene	1.26E-05	7.41E-04	2.76E-05	1.62E-03	1.71E-05	1.01E-03
Chloroethane	9.56E-06	3.30E-06	2.09E-05	7.21E-06	1.30E-05	4.49E-06
Chloroform	2.22E-05	1.59E-03	4.86E-05	3.47E-03	3.02E-05	2.16E-03
Chloromethane	3.10E-04	1.19E-02	6.77E-04	2.60E-02	4.21E-04	1.62E-02
Chromium	1.70E-07	5.66E-03	3.71E-07	1.24E-02	2.31E-07	7.70E-03
Dichlorodifluoromethane	7.07E-04	1.41E-02	1.55E-03	3.09E-02	9.62E-04	1.92E-02
Ethylbenzene	2.68E-05	9.25E-05	5.87E-05	2.02E-04	3.65E-05	1.26E-04
Fluoranthene	1.65E-07	4.12E-06	3.60E-07	9.00E-06	2.24E-07	5.60E-06
Fluorene	2.22E-07	5.55E-06	4.85E-07	1.21E-05	3.02E-07	7.55E-06
Manganese	1.35E-06	9.46E-02	2.96E-06	2.07E-01	1.84E-06	1.29E-01
Methyl Ethyl Ketone	1.88E-07	1.35E-07	4.12E-07	2.95E-07	2.56E-07	1.83E-07
Naphthalene	2.66E-07	1.33E-05	5.83E-07	2.91E-05	3.62E-07	1.81E-05
Naphthalene	2.66E-07	2.96E-04	5.83E-07	6.47E-04	3.62E-07	4.03E-04
Nickel	5.67E-07	2.84E-05	1.24E-06	6.20E-05	7.71E-07	3.86E-05
Perchloroethylene	2.96E-05	2.11E-04	6.47E-05	4.62E-04	4.03E-05	2.88E-04
Styrene	2.35E-05	8.23E-05	5.15E-05	1.80E-04	3.20E-05	1.12E-04
Toluene	1.67E-04	1.46E-03	3.65E-04	3.20E-03	2.27E-04	1.99E-03
Trichloroethene	2.64E-05	2.64E-03	5.78E-05	5.78E-03	3.60E-05	3.60E-03
Trichlorofluoromethane	3.81E-04	1.90E-03	8.33E-04	4.17E-03	5.18E-04	2.59E-03
Vinyl chloride	1.37E-05	4.89E-04	3.00E-05	1.07E-03	1.86E-05	6.66E-04
Xylenes	8.02E-05	2.67E-03	1.76E-04	5.85E-03	1.09E-04	3.64E-03
		5.70E-01		1.25E+00		7.75E-01

Table C5-1: Risk of cancer at Seaford Monitoring Station. Last row shows cumulative risk from all listed chemical compounds.

Chemical Compound	Adult Estimated Dose	Adult Cancer Risk	Child Estimated Dose	Child Cancer Risk	Age-adjusted Estimated Dose	Age-adjusted Cancer Risk
1,1,2,2-Tetrachloroethane	1.17E-06	2.35E-07	5.14E-07	1.03E-07	1.60E-06	3.19E-07
1,2,3,4,6,7,8-HpCDD	1.04E-13	1.56E-08	4.55E-14	6.82E-09	1.41E-13	2.12E-08
1,2,3,4,7,8,9-HpCDF	4.22E-15	6.32E-10	1.84E-15	2.77E-10	5.73E-15	8.60E-10
1,2,3,4,7,8-HxCDD	6.89E-14	1.03E-08	3.02E-14	4.52E-09	9.38E-14	1.41E-08
1,2,3,4,7,8-HxCDF	7.69E-14	1.15E-08	3.36E-14	5.05E-09	1.05E-13	1.57E-08
1,2,3,6,7,8-HxCDD	8.68E-14	1.30E-08	3.80E-14	5.69E-09	1.18E-13	1.77E-08
1,2,3,6,7,8-HxCDF	6.47E-14	9.70E-09	2.83E-14	4.25E-09	8.80E-14	1.32E-08
1,2,3,7,8,9-HxCDD	8.14E-14	1.22E-08	3.56E-14	5.34E-09	1.11E-13	1.66E-08
1,2,3,7,8-PeCDD	6.55E-13	9.83E-08	2.87E-13	4.30E-08	8.91E-13	1.34E-07
1,2,3,7,8-PeCDF	2.71E-14	4.06E-09	1.18E-14	1.78E-09	3.68E-14	5.52E-09
1,2-Dichloroethane	4.84E-06	4.40E-07	2.12E-06	1.93E-07	6.58E-06	5.99E-07
1,3-Butadiene	8.25E-06	8.25E-07	3.61E-06	3.61E-07	1.12E-05	1.12E-06
1,4-Dichlorobenzene	1.04E-05	2.29E-07	4.55E-06	1.00E-07	1.42E-05	3.11E-07
2,3,4,6,7,8-HxCDF	7.55E-14	1.13E-08	3.30E-14	4.95E-09	1.03E-13	1.54E-08
2,3,7,8-TCDD	1.46E-13	2.18E-08	6.37E-14	9.55E-09	1.98E-13	2.97E-08
2,3,7,8-TCDF	3.73E-14	5.60E-09	1.63E-14	2.45E-09	5.08E-14	7.62E-09
Acetaldehyde	1.88E-07	1.45E-09	8.22E-08	6.33E-10	2.56E-07	1.97E-09
Arsenic	1.04E-07	1.57E-06	4.54E-08	6.86E-07	1.41E-07	2.13E-06
Benzene	6.65E-05	1.79E-06	2.91E-05	7.85E-07	9.04E-05	2.44E-06
Benzo(b)fluoranthene	5.14E-09	1.59E-08	2.25E-09	6.98E-09	7.00E-09	2.17E-08
Cadmium	2.41E-08	1.52E-07	1.05E-08	6.63E-08	3.27E-08	2.06E-07
Carbon tetrachloride	6.59E-05	3.49E-06	2.88E-05	1.53E-06	8.96E-05	4.75E-06
Chloroform	1.04E-05	8.39E-07	4.53E-06	3.67E-07	1.41E-05	1.14E-06
Chromium	6.86E-08	2.81E-06	3.00E-08	1.23E-06	9.34E-08	3.83E-06
Chrysene	3.15E-11	9.75E-11	1.38E-11	4.27E-11	4.28E-11	1.33E-10
Formaldehyde	5.77E-07	2.59E-08	2.52E-07	1.14E-08	7.84E-07	3.53E-08
Hexachloro-1,3-Butadiene	1.26E-05	9.80E-07	5.50E-06	4.29E-07	1.71E-05	1.33E-06
OCDD	3.28E-15	4.91E-10	1.43E-15	2.15E-10	4.46E-15	6.68E-10
OCDF	2.38E-15	3.58E-10	1.04E-15	1.56E-10	3.24E-15	4.86E-10
Perchloroethylene	2.07E-05	4.13E-07	9.04E-06	1.81E-07	2.81E-05	5.62E-07
Trichloroethene	8.52E-06	3.41E-06	3.73E-06	1.49E-06	1.16E-05	4.64E-06
Vinyl Chloride	5.87E-06	8.81E-08	2.57E-06	7.71E-08	3.85E-05	1.16E-06
		1.75E-05		7.71E-06		2.49E-05

Table C5-2: Risk of non-cancer effects at Seaford Monitoring Station. Last row shows cumulative risk from all listed chemical compounds.

Chemical Compound	Adult Estimated Dose	Adult Non-cancer Risk	Child Estimated Dose	Child Non-cancer Risk	Age-adjusted Estimated Dose	Age-adjusted Non-cancer Risk
1,1,1-Trichloroethane	4.74E-05	7.52E-05	1.04E-04	1.65E-04	6.45E-05	1.02E-04
1,1,2-Trichloro-1,2,2-trifluoroethane	1.67E-04	1.94E-05	3.66E-04	4.25E-05	2.27E-04	2.64E-05
1,1-Dichloroethene	2.74E-06	4.57E-05	5.99E-06	9.99E-05	3.73E-06	6.21E-05
1,2,4-Trichlorobenzene	2.10E-05	2.10E-02	4.60E-05	4.60E-02	2.86E-05	2.86E-02
1,2,4-Trimethylbenzene	5.97E-05	3.51E-02	1.31E-04	7.69E-02	8.12E-05	4.78E-02
1,2-Dichlorobenzene	1.76E-05	4.40E-04	3.85E-05	9.63E-04	2.40E-05	5.99E-04
1,2-Dichloroethane	1.13E-05	8.06E-03	2.47E-05	1.76E-02	1.54E-05	1.10E-02
1,3,5-Trimethylbenzene	2.17E-05	1.28E-02	4.75E-05	2.80E-02	2.96E-05	1.74E-02
1,3-Butadiene	1.93E-05	3.38E-02	4.21E-05	7.39E-02	2.62E-05	4.60E-02
1,3-dichlorobenzene	1.68E-05	5.59E-03	3.67E-05	1.22E-02	2.28E-05	7.60E-03
1,4-Dichlorobenzene	2.43E-05	1.06E-04	5.31E-05	2.32E-04	3.30E-05	1.44E-04
2-Methylnaphthalene	4.00E-07	1.00E-04	8.75E-07	2.19E-04	5.44E-07	1.36E-04
Acenaphthene	1.36E-07	2.26E-06	2.97E-07	4.94E-06	1.84E-07	3.07E-06
Acetaldehyde	4.38E-07	1.71E-04	9.59E-07	3.73E-04	5.96E-07	2.32E-04
Acetone	1.34E-06	1.49E-06	2.94E-06	3.26E-06	1.83E-06	2.03E-06
Benzene	1.55E-04	1.80E-02	3.39E-04	3.94E-02	2.11E-04	2.45E-02
Bromomethane	6.49E-05	4.64E-02	1.42E-04	1.01E-01	8.83E-05	6.31E-02
Cadmium	5.62E-08	9.85E-04	1.23E-07	2.16E-03	7.64E-08	1.34E-03
Carbon tetrachloride	1.54E-04	2.69E-01	3.36E-04	5.89E-01	2.09E-04	3.66E-01
Chlorobenzene	1.26E-05	7.41E-04	2.76E-05	1.62E-03	1.71E-05	1.01E-03
Chloroethane	8.74E-06	3.01E-06	1.91E-05	6.59E-06	1.19E-05	4.10E-06
Chloroform	2.42E-05	1.73E-03	5.29E-05	3.78E-03	3.29E-05	2.35E-03
Chloromethane	3.12E-04	1.20E-02	6.83E-04	2.63E-02	4.25E-04	1.63E-02
Chromium	1.60E-07	5.34E-03	3.50E-07	1.17E-02	2.18E-07	7.26E-03
Dichlorodifluoromethane	7.12E-04	1.42E-02	1.56E-03	3.12E-02	9.69E-04	1.94E-02
Ethylbenzene	4.11E-05	1.42E-04	8.99E-05	3.10E-04	5.59E-05	1.93E-04
Fluoranthene	4.85E-07	1.21E-05	1.06E-06	2.65E-05	6.60E-07	1.65E-05
Fluorene	3.21E-07	8.01E-06	7.01E-07	1.75E-05	4.36E-07	1.09E-05
Manganese	1.25E-06	8.76E-02	2.74E-06	1.92E-01	1.70E-06	1.19E-01
Methyl Ethyl Ketone	1.59E-07	1.14E-07	3.49E-07	2.49E-07	2.17E-07	1.55E-07
Naphthalene	4.05E-07	2.03E-05	8.87E-07	4.43E-05	5.52E-07	2.76E-05
Naphthalene	4.05E-07	4.51E-04	8.87E-07	9.86E-04	5.52E-07	6.13E-04
Nickel	6.33E-07	3.16E-05	1.38E-06	6.92E-05	8.61E-07	4.30E-05
Perchloroethylene	4.82E-05	1.03E-04	1.05E-04	1.03E-04	6.56E-05	1.03E-04
Styrene	2.93E-05	1.03E-04	6.41E-05	2.24E-04	3.99E-05	1.39E-04
Toluene	2.85E-04	2.50E-03	6.23E-04	5.47E-03	3.88E-04	3.40E-03
Trichloroethene	1.99E-05	1.99E-03	4.35E-05	4.35E-03	2.71E-05	2.71E-03
Trichlorofluoromethane	3.81E-04	1.90E-03	8.33E-04	4.17E-03	5.18E-04	2.59E-03
Vinyl Chloride	1.37E-05	4.89E-04	3.00E-05	1.07E-03	1.86E-05	6.66E-04
Xylenes	1.45E-04	4.85E-03	3.18E-04	1.06E-02	1.98E-04	6.60E-03
		5.86E-01		1.28E+00		7.97E-01



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