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Health Risk Assessment Posed by the Mobile Source Air Toxics on an Urban to Regional Area

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To the Graduate Council:

I am submitting herewith a dissertation written by Luis Alonso Díaz Robles entitled "Health Risk Assessment Posed by the Mobile Source Air Toxics on an Urban to Regional Area." I have examined the final electronic copy of this dissertation for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy, with a major in Civil Engineering.

Gregory D. Reed, Major Professor

We have read this dissertation and recommend its acceptance:

Wayne T. Davis, Terry L. Miller, Williams L. Seaver

Accepted for the Council:
Carolyn R. Hodges

Vice Provost and Dean of the Graduate School

(Original signatures are on file with official student records.)

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HEALTH RISK ASSESSMENT POSED BY THE MOBILE SOURCE AIR TOXICS ON AN URBAN TO REGIONAL AREA

A Dissertation Presented for the Doctor of Philosophy Degree

The University of Tennessee, Knoxville

Luis Alonso Díaz Robles

December, 2005

DEDICATION

This dissertation is dedicated to my wife Pamela

and daughter Valeria for their love, support, and constant encouragement.

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ABSTRACT

Air toxics are important health concern. The purpose of this research was to develop a protocol to predict exposure concentrations of air toxics and inhalation cancer and non-cancer risk that come from different gasoline and diesel-fueled sources. The protocol was developed by linking the U.S. EPA's Models-3/CMAQ model as the exposure model and toxicological and epidemiological evidence functions. The NEI version 3 for the year 1999 was used in this analysis for point, area, and non-road sources, whereas NMIM was used to create the on-road emissions. The year 2003 was used for meteorological data and as reference to compare the monitored concentrations to model performance. The modeling domain consisted of a 36 km domain. To demonstrate the system's effectiveness, this study was performed on priority mobile sources air toxics (1, 3-butadiene, benzene, formaldehyde, acetaldehyde, acrolein, and DPM), and was applied to Nashville, Tennessee using available air toxics monitored data. Ten emissions scenarios were selected in this study to compare the main results.

This research on air toxics emission scenarios was based on relative analyses and estimates of absolute exposure concentrations and health risk values. The proposed protocol was demonstrated and can be used for decision makers in the quantitative assessment of new policies that will affect the public health and the air quality by air toxics. Eliminating emission source categories is clearly not a policy option, but rather helps gain a better understanding of the total magnitude of the health effects associated with these major sources of air toxics, principally of DPM. Higher formaldehyde and

acetaldehyde exposure concentrations occurred in the summer season, while benzene and 1,3-butadiene occurred in winter. DPM did not show a strong seasonality exposure during the year 2003 in Nashville. DPM generated the higher lifetime cancer risk excess among the other air toxics in Nashville, posing a cancer risk that was 4.2 times higher than the combined total cancer risk from all other air toxics. Those high cancer risk levels were due mainly to non-road sources (57.9%). For the on-road diesel fueled sources (DFS), the principal reductions were due to the DPM contributions generated by HDDVs rather than LDDVs. An evident positive synergism in the cancer risk reduction occurred when reducing diesel on-road and non-road source emissions simultaneously. The main cancer risk reductions from acetaldehyde, benzene, 1,3-butadiene, and formaldehyde (4HAPs) were due to the contribution of biogenic sources with 32.2%. This condition was followed for the scenario that did not consider on-road sources with a 27.5% of reduction. For non-road sources, the main reductions were due to the air toxics contributions generated by gasoline LDVs, principally benzene and 1,3-butadiene. The scenario 2020 showed a DPM and 4HAPs health effect reductions of approximately 32.8 and 19.4 %, respectively in Nashville. Higher cancer and non-cancer risks occurred on Southeastern urban areas due to long-term exposure to DPM, principally in Atlanta, GA, followed by Nashville, TN, Birmingham, AL, Raleigh, NC, and Memphis, TN. This research provided strong evidence that reducing ambient DPM concentrations will lead to improvement in human health more than other air toxics in Nashville, indicating that better technologies and regulations must be applied to mobile diesel engines, principally, over non-road diesel sources.

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NOMENCLATURE

A25I	Aitken mode unspecified anthropogenic mass
A25J	Accumulation mode unspecified anthropogenic mass
AACD	Acetic acid
ACORS	Coarse mode unspecified anthropogenic mass
AECI	Aitken mode elemental carbon mass
AECJ	Accumulation mode elemental carbon mass
AH2OI	Aitken mode water mass
AH2OJ	Accumulation mode water mass
ALD2	High molecular weight aldehydes
ANH4I	Aitken mode ammonium mass
ANH4J	Accumulation mode ammonium mass
ANO3I	Aitken mode aerosol nitrate mass
ANO3J	Accumulation mode nitrate mass
AORGAI	Aitken mode anthropogenic secondary organic mass
AORGAJ	Accumulation mode anthropogenic secondary organic mass
AORGBI	Aitken mode biogenic secondary biogenic organic mass
AORGBJ	Accumulation mode secondary biogenic organic mass
AORGPAI	Aitken mode mode primary organic mass
AORGPAJ	Accumulation mode primary organic mass
ASEAS	Coarse mode marine mass

ASO4I	Aitken mode sulfate mass
ASO4J	Accumulation mode sulfate mass
ASOIL	Coarse mode soil-derived mass
C_2O_3	Peroxyacyl radical ($CH_3C(O)OO\cdot$)
CO	Carbon monoxide
CRO	Methylphenoxy radical
EC	Elemental carbon or black carbon
ETH	Ethene ($CH_2=CH_2$)
FACD	Formic acid
FORM	Formaldehyde ($CH_2=O$)
HNO_3	Nitric acid
HO_2	Hydroperoxy radical
ISOP	Isoprene
MGLY	Methylglyoxal ($CH_3C(O)C(O)H$)
NH3	Ammonia
NO	Nitric oxide
NO_2	Nitrogen dioxide
NO_3	Nitrogen trioxide (nitrate radical)
O	Oxygen atom (triplet)
O_3	Ozone
OC	Organic carbon

OH	Hydroxyl radical
OLE	Olefinic carbon bond
OPEN	High molecular weight aromatic oxidation ring fragment
PACD	Peroxy acetic acid
PAHs	Polycyclic Aromatic Hydrocarbons
PAN	Peroxyacyl nitrate $(CH_3C(O)OONO_2)$
PAR	Paraffin carbon bond (C-C)
ROR	Secondary organic oxy radical
SO2	Sulfur dioxide
SOF	Soluble organic fraction
SVOCs	Semi-volatile organic compounds
TOG	Total organic gases
VOCs	Volatile organic compounds
XO_2	NO-to-NO operation
XO_2N	NO-to-nitrate operation

ABBREVIATIONS

4HAPS   Cumulative cancer risk for acetaldehyde, benzene, 1,3-butadiene, and formaldehyde

A   Incidence rate

ACS   American Cancer Society

ALAPCO   Association of Local Air Pollution Control Officials

ASPEN   Assessment system for population exposure nationwide

ATDA   Air Toxics Data Archive

ATSDR   Agency for Toxic Substances and Disease Registry

NB   Normalized bias

BAAQMD   Bay Area Air Quality Management District

BC   Boundary conditions

BCON   Boundary Condition Processor

BEIS   Biogenic Emission Inventory System

C_i Annual concentration for each compound

CAA   Clean Air Act

CAAA   Clean Air Act Amendment

CalEPA   California Environmental Protection Agency

CAMx   Comprehensive Air Quality Model

CAS   Chemical Abstract Service code

CB-IV   Carbon bond IV chemical mechanism

CDC	Centers for Disease Control
CDT	Central Daylight Savings Time
CG	Conventional gasoline
CI	Confidence interval
CMAQ	Community Multiscale Air Quality Model
CMAS	Community Modeling and Analysis System
CNG	Compressed natural gas
COPD	Chronic obstructive pulmonary disease
C-R	Concentration-response
CVD	Cardiovascular disease
DEOG	Diesel exhaust organic gases
DFS	Diesel-fueled sources
DOT	Department of Transportation
DPM	Diesel particulate matter
EATN	East Nashville Health Clinic HAPs Monitor Station
EC	Elemental carbon
ED	Emergency department
ETS	Environmental tabbaco smoke
FDDA	Four-dimensional data assimilation
NG	Normalized gross error
GDP	Economic growth

GMT	Greenwich Mean Time
GPRA	Government Performance Results Act
HAPEM	Hazardous Air Pollutant Exposure Model
HAPs	Hazardous air pollutants or air toxics
HDDVs	Heavy Duty Diesel Vehicles
HDVs	Heavy Duty Vehicles
IARC	International Agency for Research on Cancer
IC	Initial conditions
ICON	Initial Condition processor
IMPROVE	Interagency Monitoring for Protected Environments
IRIS	Integrated Risk Information System
ISCLT	Industrial Source Complex Long Term model
IUATS	Integrated Urban Air Toxics Strategy
IUR	Inhalation unit risk
JPROC	Photolysis rate processor
LDDVs	Light Duty Diesel Vehicles
LDVs	Light Duty Vehicles
LOTN	Lockeland Middle School HAPs Monitor Station
MACT	Maximum Available Control Technology
MATES	Multiple Air Toxics Exposure Study
MCIP	Meteorology Chemistry Interface Processor

MM5	Mesoscale Meteorological Model
MSATs	Mobile Sources Air Toxics
NASA	National Aeronautics Space Administration
NATA	National-Scale Air Toxics Assessment
NATTS	National Air Toxics Trend Stations
NCAR	National Center for Atmospheric Research
NEI	National Emissions Inventory
NIEHS	National Institute of Environmental Health Sciences
NIOSH	National Institute for Occupational Safety and Health
NLEV	National Low Emissions Vehicle
NMIM	National Mobile Inventory Model
NSCLC	Non-small cell lung cancer
OR	Odds Ratio
PAMS	Photochemical Assessment Monitoring Stations
PBL	Planetary Bounder Layer
PinG	Plume-in-grid
PM_{10}	Particulate matter less than or equal to 10 μm in aerodynamic diameter
$PM_{2.5}$	Particulate matter less than or equal to 2.5 μm in aerodynamic diameter
PMSATs	Priority Mobile Sources Air Toxics
POM	Polycyclic organic matter
PSU	Pennsylvania State University

RADM2	Regional acid deposition model chemical mechanism
R^2	Coefficient of Determination
RfC	Reference concentration
RFG	Reformulated gasoline
RR	Relative Risk
SCC	Source Classification Codes
SCLC	Small cell lung cancer
SMOKE	Sparse Matrix Operator Kernel Emissions Model (inventory processor)
SOA	Secondary organic aerosols
STAPPA	State and Territorial Air Pollution Program Administrators
TDOT	Tennessee Department of Transportation
TOMS	Total Ozone Mapping Spectrometer
U.S.EPA	United States Environmental Protection Agency
UAM	Urban Air-Shed Model
UATMP	Urban Air Toxics Monitoring Program
UATs	Urban Air Toxics
UNC	University of North Carolina
UTK	University of Tennessee
VMT	Vehicle miles traveled
β	Regression coefficient
ΔPM	Change in concentration of PM2.5

σ	Standard deviation
yc	Risk at the control PM2.5 exposure
yo	Risk at the baseline PM2.5 exposure

Units

ppbV	part per billions by volume
TPY	tons per year
ug/cum	micrograms per cubic meter
ug/m^3	micrograms per cubic meter

1.0   INTRODUCTION

Air toxics, which are also called hazardous air pollutants or HAPs, are those pollutants known or suspected to cause cancer and other serious health or environmental effects. They include pollutants like volatile organic compounds (VOCs), metals, semi-volatile organic compounds (SVOCs), organochlorine compounds, diesel particulate matter (DPM), and others HAPs. While the harmful effects of air toxics are of particular concern in areas closest to where they are emitted, they can also be transported and affect the health and welfare of populations in other geographic areas. Once HAPs enter to the body, some persistent air toxics accumulate in body tissues. Predators, such as fish, typically bioaccumulate even greater pollutant concentrations than their contaminated prey. As a result, people and other animals at the top of the food chain are exposed to concentrations that are much higher than the concentrations in the water, air, or soil.

Scientists estimate that millions of tons of toxic pollutants are released into the air each year in the world. Most HAPs originate from anthropogenic sources, including point, area, and mobile sources. These mobile sources contribute approximately 50 percent of the total urban HAPs in the U.S. In addition, some air toxics are released in major amounts from natural sources, called biogenic sources, such as trees.

Because it has not been feasible to attempt to do controlled or epidemiological studies of these many air toxics on humans, the U.S. Environmental Protection Agency (U.S. EPA) regulation required sources to keep the emissions levels as low as possible using

Maximum Available Control Technology (MACT) on point sources, and of existing and newly promulgated mobile source control programs, including its reformulated gasoline (RFG) program, its national low emissions vehicle (NLEV) standards, its Tier 2 motor vehicle emissions standards and gasoline sulfur control requirements, and its 2007 heavy-duty engine, and vehicle standards and on-highway diesel fuel sulfur control requirements (U.S. EPA, 2001a).

However, the fast growth of the mobile sources indicates that some HAPs would increase if the community does not improve the fuels or does not use cleaner vehicle technologies. The American Cancer Society (ACS) estimated that the number of new U.S. cancer cases increased 3.8 percent, to a record of 1.33 million for 2003, where some percentage could be due to air toxics (ATSDR, 2003). ACS estimated that 171,900 cases corresponded to lung cancer, accounting for 13% of all new cancer cases. Also, 30,500 new cancer cases occurred for Tennessee in 2003, of which 4,500 correspond to lung cancer, accounting for 15% of all new cancer cases. The average annual age-adjusted mortality rates for lung cancer deaths per 100,000 persons were 68.9 for Tennessee and 56.8 for Nationwide from 1996 to 2000 (American Cancer Society, 2003). On the other hand, it is estimated that approximately 30,800 individuals were diagnosed with leukemia in the U.S. and 21,700 will die of the disease annually (Xie et al., 2003).

Cardio-respiratory non-cancer diseases also have been significant in the U.S. due to air pollution, mainly in urban areas (American Lung Association, 2003). The most important have been asthma, chronic obstructive pulmonary disease (COPD), and cardiovascular

disease (CVD). Asthma is a chronic illness that has been increasing in prevalence in the

U. S. since 1980 by 75%. In 1999, it was estimated that 24.7 million Americans have

been diagnosed with asthma in their lifetime (National Center for Health Statistics, 1999).

In 2000, asthma accounted for 4,487 deaths, almost 465,000 hospitalizations, 1.8 million

emergency department visits, and approximately 10.4 million physician-office visits

among persons of all ages (CDC, 2001a and 2001b; Brugge et al., 2003).

COPD includes emphysema and chronic bronchitis diseases that are characterized by

obstruction to air flow. Emphysema and chronic bronchitis frequently coexist. According

to a report of the American Lung Association in 2003, COPD claimed the lives of

117,522 Americans. Approximately 80 to 90 percent of COPD cases have been due to

smoking; a smoker is ten times more likely than a nonsmoker to die of COPD. An

estimated of 11 million people were diagnosed with chronic bronchitis in 2001 and 3

million Americans have been diagnosed with emphysema sometime in their life

(American Lung Association, 2003).

Finally, CVD is the leading cause of death in the U.S. According to the American Heart

Association's Heart Disease and Stroke Statistics 2003 Update, in 2000 CVD caused

nearly 40% of all deaths. Traditional risk factors for CVD include lifestyle (such as

smoking, physical inactivity, and diet), serum lipids, gender, race, and family history

(genetics), which account by almost 80% of the total risk factors. However, these risk

factors do not fully explain the etiology or incidence of CVD, and recent data have

indicated that exposure to air pollutants such as particulate matter and diesel exhaust are also risk factors (Oftedal et al., 2003; Lin et al., 2003).

Since 1987, U.S. EPA has sponsored the Urban Air Toxics Monitoring Program (UATMP) to characterize the composition and magnitude of urban air pollution through an extensive ambient air monitoring network (U.S., EPA, 2004a). Currently, there are about 59 air toxics monitoring sites in operation, which include 37 urban locations. Some of this data has been used to assess health risk for particular areas (Tam and Neumann, 2004; Partt et al., 2000). However, this strategy is too expensive to monitor for every pollutants everywhere.

Air quality models are valuable air quality management tools. They estimate the HAPs concentrations at many locations and the number of the locations in a model far exceeds the number of monitors in a typical ambient monitoring network, such as the UATMP. Therefore, the integration of air quality modeling methodologies and health risk assessment techniques is extremely powerful to air quality management, policy, and rulemaking issues, principally to analyze emission scenarios, new strategies, and future growth effects over a wide spatial area where the complex terrain and meteorology factors could be important.

Currently, the U.S. EPA uses a Gaussian plume model and an exposure model to estimate the annual HAPs concentrations (chronic exposure) to assess health risk for cancer and non-cancer effects (U.S. EPA, 2002a). The model has been accurate for local scale

effects on ambient concentrations from emitted HAPs that have short atmospheric lifetimes, slow loss rates, and no photochemical production. For HAPs such as formaldehyde, acetaldehyde, 1,3-butadiene, acrolein, and benzene, several of the assumptions fail.

Air toxics in the atmosphere are difficult to model because they have half-lives varying from a few minutes to over two years. They can be produced in the atmosphere from other HAPs and non-HAPs, for example isoprene. They are temporally variable having large diurnal variations such as secondary formaldehyde and acetaldehyde. Some HAPs are produced and destroyed in a cyclical set of chemical reactions involving VOC, OH$\bullet$, NO$_3\bullet$, O$_3$, and sunlight. They are spatially variable, both vertically and horizontally. Finally, they exit as gases, particles, both gases and particles, or in aqueous phase. As a result, differences in some HAPs and VOC emissions and weather patterns contribute to hourly, daily, seasonal, and annual differences in HAPs concentrations from urban to urban area.

The HAPs ambient concentrations and the health risk assessment can be improved by using a model that better simulates the transport and fate of these compounds, such as the state-of-art Community Multi-scale Air Quality model (Models-3/CMAQ) (Byun and Ching, 1999).

1.1 OBJECTIVE

The overall objective of this study was to develop a model protocol to assess the public health risk caused by the chronic exposure to the mobile source air toxics (MSATs) on an urban to regional area, based on different emissions scenarios by linking the annual air toxics concentrations predicted by the advanced air quality model Models-3/CMAQ, with the risk factors associated to cancer and non-cancer effects.

To demonstrate the system's effectiveness, this study was done on 1, 3-butadiene, benzene, formaldehyde, acetaldehyde, acrolein, and DPM, and was applied to Nashville, Davidson County, Tennessee, using available urban air toxics monitoring data.

The advantage of the proposed modeling approach is that it can be used as a predictive tool to help air quality policy and decision-making. For example, this protocol can be applied to estimate the effect of the new non-road diesel regulations over future scenarios, the effect to use hybrid vehicles, and the emission reduction strategies of open burning sources, among other air quality strategies.

2.0   LITERATURE REVIEW

2.1   AIR TOXICS

2.1.1 Overview

The U.S. EPA classifies pollutants present in the ambient air in two parts; criteria pollutants and HAPs. The 1990 Clean Air Act Amendment (CAAA) defines 188 chemicals as HAP (U.S. EPA, 2002d). According to the CAAA, these HAPs "present, or may present, through inhalation or other routes of exposure, a threat of adverse human health effects (including, but not limited to, substances which are known to be, or may reasonably be anticipated to be, carcinogenic, mutagenic, teratogenic, neurotoxic, which cause reproductive dysfunction, or which are acutely or chronically toxic) or adverse environmental effects whether through ambient concentrations, bioaccumulation, deposition, or otherwise…." They include pollutants like volatile organic compounds (VOCs), metals, other particles, gases adsorbed onto particles, diesel particulate matter (DPM), semi-volatile organic compounds (SVOCs), organochlorine compounds, and others HAPs. About 70% of the pollutants classified as HAPs fall into the category of VOCs (Suh et al., 2000). These compounds are the main components in atmospheric reactions that form ozone and other secondary pollutants, like formaldehyde and acetaldehyde (Seinfeld and Pandis, 1998; Woodruff et al., 1998 and 2000; Atkinson, 2000 and 2003; Pratt et al., 2000; Tam and Neumann 2004; Bloss et al., 2005). While the harmful effects of air toxics are of particular concern in areas closer to where they are

emitted, they can also be transported and affect the ecosystem, the health, and welfare of populations in other geographic areas (Seigneur et al., 2003; Chenier, 2003; Efroymson and Murphy, 2001). Some can persist for considerable time in the environment and/or bioaccumulate through the food chain. This kind of process also affects other organisms, generating a sequence of bioaccumulation steps that occur along a food web, called biomagnification (Offenberg et al., 2005; Baird 2001).

U.S. EPA identified 33 of the 188 air toxics listed in the CAAA, called urban air toxics (UATs), that present the greatest threat to public health in the largest number of urban areas (U.S. EPA, 1999a). At the same time, the agency compared the lists of compounds identified in the motor vehicle emission databases and studied them with the toxic compounds listed in the Integrated Risk Information System, IRIS. Thus, U.S. EPA identified 21 MSATs (U.S. EPA, 2000 and 2001a), each of which has the potential to cause serious adverse health effects as reflected in IRIS and in the ongoing agency scientific assessments. This list, shown in Table 2-1, includes various volatile organic compounds, VOCs, and metals, as well as diesel particulate matter, and diesel exhaust organic gases, collectively called DPM + DEOG.

Mobile sources are one category of air toxic emission sources most relevant to human activities in industrialized societies. According to the U.S. EPA definition for emission inventory purposes, mobile sources include highway vehicles, non-road mobile sources, aircraft, and locomotives. Recreational marine equipment and commercial marine vessels are also classified as non-road mobile sources (U.S. EPA, 2005a).

Table 2-1. List of EPA Mobile Source Air Toxics (Reprinted from U.S EPA, 2001a)

Acetaldehyde	Diesel Particulate Matter + Diesel Exhaust Organic Gases (DPM + DEOG)	MTBE
Acrolein	Ethylbenzene	Naphthalene
Arsenic Compounds	Formaldehyde	Nickel Compounds
Benzene	n–Hexane	POM *
1,3-Butadiene	Lead Compounds	Styrene
Chromium Compounds	Manganese Compounds	Toluene
Dioxin/Furans	Mercury Compounds	Xylene
* Polycyclic Organic Matter includes organic compounds with more than one benzene ring, and which have a boiling point greater than or equal to 100°C.		

Concern about mobile source emissions has been expressed since the 1950s. Hundreds of chemical compounds have been identified in mobile source emissions. Since the mid 1980s, a variety of studies have documented toxic air emissions from mobile sources as a major contributor to overall health risk (U.S. EPA, 1990).

2.1.2 Air Toxics and Health Problems of Concern

People who are exposed to toxic air pollutants at sufficient concentrations and for enough time may increase their risk of getting cancer (Tam and Neumann, 2004; Reynolds et al, 2003; Lloyd and Cackette, 2001; Partt et al, 2000; Morello-Frosch et al., 2000; Woodruff et al., 1998) or experiencing other serious health effects or premature mortality, such as

asthma (Kleeberger and Peden, 2005; Dolinoy and Miranda, 2004; Leikauf, 2002; Weisel, 2002; Delfino, 2002; Peden, 2002) chronic obstructive pulmonary disease (COPD) (Biswas and Wu, 2005; Groneberg and Chung, 2004; White et al., 2003), and cardiovascular disease (CVD) (Krewski et al, 2005a; Delfino et al., 2005; Sioutas et al., 2005; Mills et al., 2005; Zanobetti and Schwartz, 2005; Pope et al., 2002, 2004a and 200b; Brunekreef and Holgate, 2002), among others. Depending upon which air toxics an individual is exposed to, these health effects can include damage to the immune system, as well as neurological, reproductive, developmental, and cardio respiratory problems. The evidence indicates that some air toxics may disturb the endocrine system; in some cases this happens by pollutants either changing or blocking the action of natural hormones. Health effects associated with endocrine disruption include reduced male fertility, birth defects, and breast cancer (Baird, 2001). The evidence continues to associate air pollution with numerous adverse health effects, including mortality and morbidity, especially DPM. Altered respiratory symptoms, altered pulmonary function, bronchodilator usage, school or work absence, and hospital admissions increase in association with exposures to air pollution (U.S. EPA, 2005b). Although local sources are difficult to evaluate rigorously, and long-range transport is recognized to influence ambient concentrations, local sources can augment adverse effects. For example, the Harvard Six-Cities study and its reanalysis found higher mortality in Steubenville, Ohio, and St. Louis, Missouri, locations where the air quality is influenced more by regional stationary sources mixed with long-range transport processes, than in Watertown, Massachusetts, or Kingston/Harriman, Tennessee, locations influenced almost solely by long-range transport processes (Krewski et al., 2005b and 2005c; Dockery et al., 1993).

Because air pollution is a complex mixture, several investigators have postulated that any single exposure variable cannot be solely responsible for observed adverse effects (Williams, 2004; Carpenter et al., 2002; Leikauf, 2002). In fact, people are exposed to mixtures in the real world, not single chemicals, and to multiple exposure pathways. Although several air toxics may have totally independent effects, in many cases two compounds may act at the same site in ways that could be either additive or non-additive impacts (Carpenter et al., 2002), and complex interactions or synergistic effects may occur where the effects of two or more substances together are higher than the sum of either effect alone (Kinney et al., 2002). So far, scientists have not estimated the effects or cancer risk of the HAPs mixtures on human health, which is the reason why in most of the research an additive effect has been assumed (U.S. EPA, 2005b); Tam and Neumann, 2004; Sapkota et al., 2003; Leikauf et al., 2002; Winebrake et al., 2001; Pratt et al., 2000; Woodruff et al., 2000; Morello-Frosch et al., 2000; U.S. EPA, 1990).

Congress did expect that U.S. EPA would be able to estimate the risk to public health from air toxics, mainly for urban areas. However, data on outdoor concentrations of air toxics are not available for many communities. Of the 188 HAPs, only a handful have information on human health effects, derived primarily from animal and occupational studies, except fine particulate matter, to which several epidemiological studies have demonstrated strong linkage with lung cancer, COPD, CVD, asthma, and premature deaths (Krewski et al., 2005a; Delfino et al., 2005; Zanobetti and Schwartz, 2005; Sioutas et al., 2005; Schultz et al., 2005; Riedl et al., 2005; Pope et al., 2004a; Adonis et al., 2003a and 2003b; Pandya et al., 2002; Lloyd and Cackette, 2001; Castranova et al., 2001;

Lipsett and Campleman, 1999; CalEPA, 1998; Steenland et al., 1998). Lack of consistent monitoring data on ambient air toxics makes it difficult to assess the extent of low-level, chronic, ambient exposures to HAPs that could affect human health, and limits attempts to prioritize and evaluate policy initiatives for emissions reduction. One study did attempt to estimate the public health risk from these air toxics (Woofruft et al., 1998), in which approximately 10% of all census tracts had estimated concentrations of one or more carcinogenic urban air toxics greater than 1 in 10,000 risk level.

2.1.2.1   Cancer Risk

Cancer describes a group of related diseases that affect a variety of organs and tissues. Cancer results from a combination of genetic damage and non-genetic factors that favor the growth of damaged cells. The U.S. EPA's 2005 Guidelines for Carcinogen Risk Assessment (U.S. EPA, 2005b) provides guidance on hazard identification for carcinogens. The approach recognizes three broad categories of data: (1) human data (primarily epidemiological); (2) results of long-term experimental animal bioassays; and (3) supporting data, including a variety of short-term tests for genotoxicity and other relevant properties. In hazard identification of carcinogens under the 2005 guidelines, the human data, animal data, and "other" evidence are combined to characterize the weight of evidence regarding the agent's potential as a human carcinogen into one of several hierarchic categories (U.S. EPA, 2005b):

Group A (human carcinogen): These are HAPs compounds for which human data are sufficient to demonstrate a cause and effect relationship between exposure and cancer incidence (rate of occurrence) in humans. In the national-scale assessment, the 7 air toxics classified as human carcinogens are: arsenic compounds, benzene, 1,3-butadiene, chromium compounds (VI), coke oven emissions, nickel compounds, and vinyl chloride.

Group B (probable human carcinogen):

- **Group B1:** These are HAPs compounds for which limited human data suggest a cause and effect relationship between exposure and cancer incidence (rate of occurrence) in humans. In the national-scale assessment, the 5 air toxics classified as probable (B1) human carcinogens are: acrylonitrile, beryllium compounds, cadmium compounds, ethylene oxide, and formaldehyde.

- **Group B2:** These are HAPs compounds for which animal data are sufficient to demonstrate a cause-and-effect relationship between exposure and cancer incidence (rate of occurrence) in animals, and human data are inadequate or absent. In the national-scale assessment, the 15 air toxics classified as probable (B2) human carcinogens are: acetaldehyde, carbon tetrachloride, chloroform, 1,3-dichloropropene, ethylene dibromide, ethylene dichloride, hexachlorobenzene (HCB), hydrazine, lead compounds, methylene chloride, PCBs, polycyclic organic matter (POM), perchloroethylene, propylene dichloride, trichloroethylene.

Group C (possible human carcinogen): These are HAPs compounds for which animal data are suggestive to demonstrate a cause-and-effect relationship between exposure and

cancer incidence (rate of occurrence) in animals. In the national-scale assessment, the 4 air toxics classified as possible human carcinogens are: acrolein, mercury compounds, quinoline and 1,1,2,2-tetrachloroethane. Because unit risk estimates have not been developed for acrolein and mercury compounds, EPA has not estimated cancer risk for these pollutants.

Group D (not classifiable as to human carcinogenicity): These are HAPs compounds for which human and animal data are inadequate to either suggest or refute a cause-and-effect relationship for human carcinogenicity. In the national-scale assessment, only manganese compounds were considered to be not classifiable as to human carcinogenicity.

Group E (evidence of noncarcinogenicity): These are HAPs compounds for which animal data are sufficient to demonstrate the absence of a cause-and-effect relationship between exposure and cancer incidence (rate of occurrence) in animals. In the national-scale assessment, no air toxics were classified as having evidence of noncarcinogenicity.

Air toxics contribute to cancer risk (Tam and Neumann, 2004; Reynolds et al., 2003; Lloyd and Cackette, 2001; Partt et al.,, 2000; Morello-Frosch et al., 2000; Woodruff et al., 1998; U.S. EPA, 2002c), where the main kinds of cancer are lung cancer, leukemia, and nasal cancer. A San Francisco Bay Area Air Toxics study by the Bay Area Air Quality Management District (BAAQMD) estimated that 1,3-butadiene, benzene, formaldehyde, and diesel exhaust particulate matter are responsible for more than 90% of

the potential excess cancer risk associated with urban air toxics (U.S. EPA, 2001a). U.S. EPA and other agencies have conducted a number of air toxics exposures and risk assessment screening studies in the last decade, as well as compiled and analyzed 10 reports, 14 studies, and 2 databases in a document entitled Cancer Risk from Outdoor Exposure to Air Toxics (U.S.EPA, 1990). These reports and studies included a total of 65 source categories, 90 different pollutants, and covered varying geographic areas, ranging from city-specific to nationwide. The results of the U.S.EPA's analysis showed that many types of sources contribute to annual cancer incidence, but the largest contributor found in the document was the motor vehicle source category. According to the U.S. EPA's estimation, 56% of the total cancer cases were attributed to direct emissions from motor vehicles. In addition to the direct emissions, motor vehicles contributed another 2% of the cancer incidences from secondary formaldehyde. It should be noted that approximately 35% of the contribution of secondary formaldehyde was from motor vehicles. Therefore, motor vehicles contributed approximately 58% of the total nationwide annual cancer incidence from exposure to outdoor air toxics (U.S.EPA, 1990). In contrast to motor vehicles, point sources were only estimated to contribute approximately 25% to the total annual cancer incidence.

A preoccupation with the special vulnerability of children to pollutants in the environment has grown over the last decade. According to the body weight, children breath more air, drink more water, and eat more food than adults, increasing relative exposure to pollutants such as air toxics. The risk assessment for children from birth to age one and from birth to age 18 for 10 air toxics in California (Reynolds et al., 2003)

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indicated that in all studied places it exceeded the recommended maximum of one-in-one million cancer risk by age one for three pollutants; 1,3-butadiene, benzene, and diesel particulates.

A.   Lung Cancer

There are two major types of lung cancer: small cell lung cancer (SCLC) and non-small cell lung cancer (NSCLC). Sometimes a lung cancer may have characteristics of both types, which are known as mixed cell/large cell carcinoma (American Lung Association, 2005) The incidence and mortality attributed to lung cancer has been rising steadily since the 1930's in the U.S., mainly due to the increasing popularity of cigarette smoking, which causes between 85 and 90% of lung cancer cases, and therefore, lung cancer has become the leading cause of cancer mortality and morbidity in the U.S. It is estimated that these were 169,400 new cases of lung cancer with 154,900 deaths in 2002, accounting for 28% of all cancer deaths (American Lung Association, 2005).

During a typical day, the average adult inhales about 10,000 L air (Alberg et al., 2003). Consequently, even the carcinogens that are present in the air at low concentrations are of concern as a risk factor for lung cancer. Extrapolation of the risks associated with occupational exposures to the lower concentration of carcinogens in polluted ambient air leads to the conclusion that a small proportion (1-2%) of lung cancer cases could be due to air pollution. Thus, air pollution could cause in the U.S. an estimated of 3,388 new cases of lung cancer and 3,098 deaths in 2002 (American Lung Association, 2005).

In considering respiratory carcinogenesis, the constituents of air pollution will vary by locale and over time depending on the pollution sources (Alberg et al., 2003). Thus, epidemiologic investigations of air pollution and lung cancer have been limited by the difficulty of estimating exposure. Nevertheless, descriptive evidence is consistent with a role for air pollution in causing lung cancer. Urbanization and lung cancer mortality are linked (Buffler et al., 1988; Short et al., 2002; Alberg et al., 2003). This association could increase from differences in the distributions of other lung cancer risk factors, such as smoking and occupational exposures, by degree of urbanization. Adjustment for these factors may considerably attenuate the effect of urban location, but an urban effect persists in a number of studies (Samet et al., 1999). Air pollution has been assessed as a risk factor for lung cancer in both case-control and cohort studies, especially for particulate matter. These studies have been reviewed in detail elsewhere (Krewski et al., 2005a; Lipsett and Campleman, 1999; Samet et al., 1999; CalEPA, 1998; Steenland et al., 1998; Speizer et al., 1994).

B. Leukemia

With the exception of a few established risk factors including benzene and alkylating agents, which account for a fraction of cases, little is known about the causes of leukemia. It is estimated that each year, approximately 30,800 individuals will be diagnosed with leukemia in the United States and 21,700 will die of the disease (Xie et al., 2003).

It has been suggested that these may be an association between increased childhood leukemia rates and high HAPs exposure (Reynolds et al., 2003), but studies involving more comprehensive exposure assessment and individual-level exposure data will be important for elucidating this relationship. Reynolds evaluated the relationship between childhood cancer rates and exposure scores for 25 potentially carcinogenic HAPs emitted from mobile, area, and point sources and from all sources combined in California. Using Poisson regression Reynolds found elevated rate ratios RRs and a significant trend with increasing exposure level for childhood leukemia in tracts ranked highest for exposure to the combined group of 25 HAPs (RR = 1.21; 95% confidence interval, 1.03, 1.42) and in tracts ranked highest for point-source HAP exposure (RR = 1.32; 95% confidence interval, 1.11, 1.57). According to U.S. EPA, benzene and 1,3-butadiene produce leukemia (U.S.EPA, 1998 and 2002e).

2.1.2.2   Non-Cancer Risk

Cancer is commonly used in risk assessment modeling and allows mathematical comparisons of risk estimates among compounds; non-cancer risks also are used in modeling and include reproductive, neurotoxic, and cardio respiratory effects. Among the cardio respiratory effects, the most important chronic diseases are asthma, COPD, and CVD (Delfino et al., 2005; Krewski et al., 2005a; Zanobetti and Schwartz, 2005; Sioutas et al., 2005; Schultz et al., 2005; Riedl et al., 2005; Pope et al., 2004a; Leikauf et al 2002; Delfino et al., 2002).

A. Asthma

Asthma has a high prevalence in the United States (Delfino et al., 2005; Leikauf et al., 2002), and persons with asthma may be at added risk from the adverse effects of HAPs (Luttinger et al., 2003). Complex mixtures, fine particulate matter, and tobacco smoke have been associated with respiratory symptoms and hospital admissions for asthma. The toxic ingredients of these mixtures are HAPs. Certain air toxics are occupational asthma precursors, whereas others may act as adjuncts during sensitization. HAPs may exacerbate asthma because, once sensitized, individuals can respond to remarkably low concentrations, and irritants lower the bronchoconstrictive threshold to respiratory antigens (Leikauf et al., 2002).

Adverse responses after ambient exposures to complex mixtures often occur at concentrations below those producing effects in controlled human exposures to a single compound. In addition, certain HAPs that have been associated with asthma in occupational settings may interact with criteria pollutants in ambient air to aggravate asthma (Leikauf et al., 2002). Based on these observations and past experience with 188 HAPs, a list of 19 compounds that could have the highest impact on the induction or exacerbation of asthma was developed by Leikauf.

Nine additional compounds were identified that might intensify asthma based on their irritancy, respirability, or ability to react with biological macromolecules. Although the ambient levels of these twenty-eight compounds are largely unknown, estimated

exposures from emissions inventories and limited air monitoring suggest that aldehydes, especially acrolein and formaldehyde, and metals, especially nickel and chromium compounds, may have possible health risk indices sufficient for additional attention. According to Leikauf, the most important HAPs that are suspected of inducing or exacerbating asthma are acetaldehyde, acrolein, benzene, cadmium compounds, chromium compounds, coke oven emissions, ethylene oxide, formaldehyde, hydrazine, manganese compounds, and nickel compounds. According to the National Center for Health Statistics (CDC, 2003), in 2000 there were 10.4 million outpatient asthma visits to private physician offices and hospital clinics, or 379 per 10,000 people (Figure 2.1).

Children aged from newborn to seventeen years had 4.6 million visits and an outpatient visit rate of 649 per 10,000, and adults eighteen years and over had a rate of 285 per 10,000. Blacks had an office visit rate 40% higher than whites, and females a 10% higher visit rate compared to males. There were 1.8 million visits to emergency departments (EDs) or 67 per 10,000 people (Figure 2.2). Children aged from newborn to seventeen years had over 728,000 ED visits, a rate of 104 per 10,000. The ED visit rate was highest among children aged from newborn to four years, at 180 per 10,000. Adults 18 years and over had 54 ED visits per 10,000. The ED visit rate for blacks was 125% higher than that for whites, and for females, about 30% higher than for males. There were 465,000 asthma hospitalizations or 17 per 10,000 people (Figure 2.3).
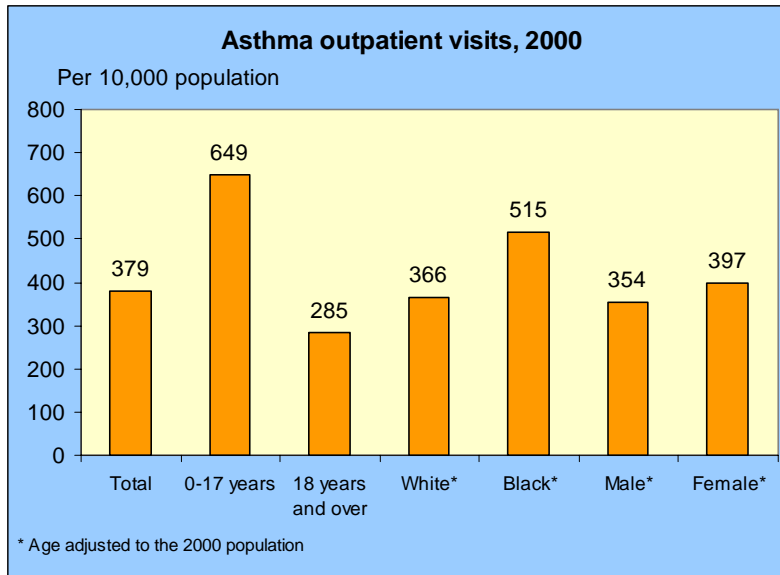
Figure 2.1. Asthma Outpatient Visits, 2000 (Reprinted from CDC, 2001a)
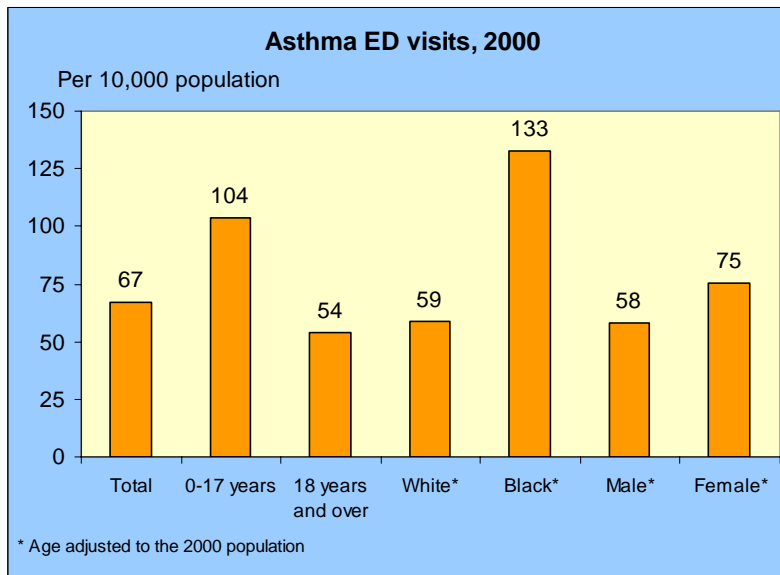
Figure 2.2. Asthma ED visits, 2000 (Reprinted from CDC, 2001a)
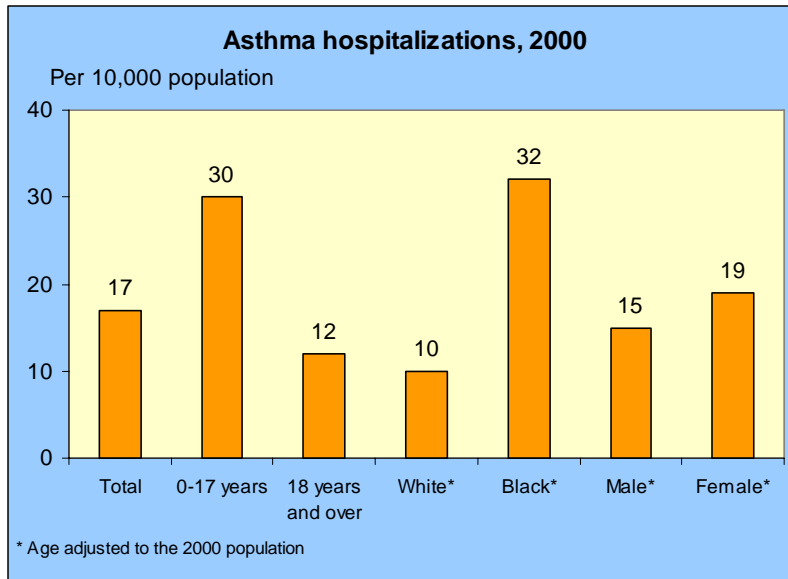
Figure 2.3. Asthma Hospitalizations, 2000 (Reprinted from CDC, 2001a)

Among children from newborn to seventeen years, there were 214,000 hospitalizations (30 per 10,000). Hospitalizations were highest among children from newborn to four years who had 67 hospitalizations per 10,000 populations. The asthma hospitalization rate for blacks was 220% higher than for whites. Females had a hospitalization rate 25% higher than males. In 2000, 4,487 people died from asthma, or 1.6 per 100,000 people (Figure 2.4). Among children, asthma deaths are rare. In that year, 223 children aged from newborn to seventeen years died from asthma, or 0.3 deaths per 100,000 children, compared to 2.1 deaths per 100,000 adults aged 18 and over. Non-Hispanic blacks were the most likely to die from asthma and had an asthma death rate over 200% higher than non-Hispanic whites and 160% higher than Hispanics. Females had an asthma death rate about 40% higher than males.
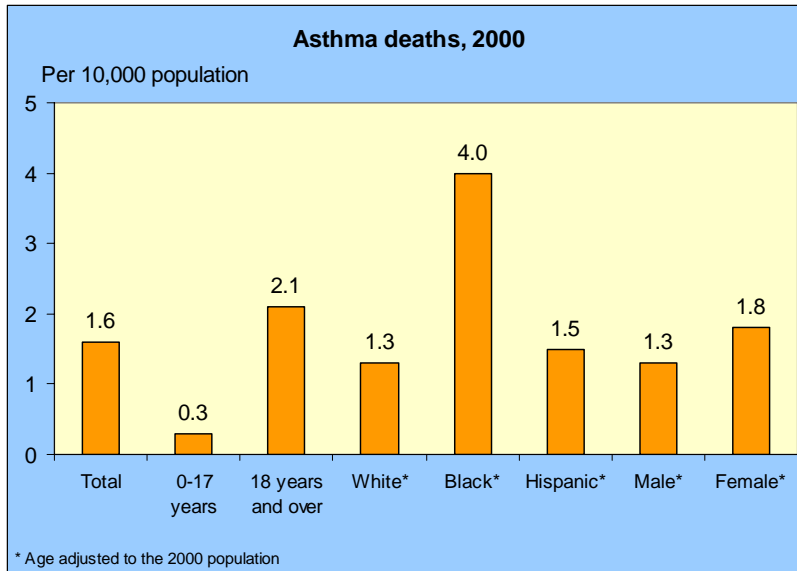
Figure 2.4. Asthma Deaths, 2000 (Reprinted from CDC, 2001a)

In addition, the average annual rates for asthma cases per 100,000 populations were 7,648

for Tennessee and 7,573 nationwide in 2000. Over 301,000 adults and over 71,000

children suffer from asthma every year in Tennessee (American Lung Association, 2003).

Direct health care costs for asthma in the United States total more than $8.1 billion

annually; indirect costs (lost productivity) add another $4.6 billion for a total of $12.7

billion (American Lung Association, 2005). Pharmaceutical costs, or medications,

represent the largest direct cost (Cisternas, 2003). Thus, asthma is in the top tier of

diseases targeted for disease management by managed care organizations because of its

high cost, and persons with asthma could be at added risk from the adverse effects of

hazardous air pollutants (HAPs) (Luttinger et al., 2003).

B. Chronic Obstructive Pulmonary Disease

Another important chronic disease in the United States is COPD, the fourth leading cause of death, illness, and disability (Figure 2.5). COPD includes emphysema and chronic bronchitis diseases that are characterized by obstruction to air flow. Emphysema and chronic bronchitis frequently coexist. COPD does not include other obstructive diseases such as asthma. According to the Centers for Disease Control and Prevention (CDC) and the National Center for Health Statistics (Anderson, 2002), COPD claimed the lives of 119,000 deaths, 726,000 hospitalizations, and 1.5 million hospital emergency department visits in the United States during 2000. Anderson indicated that in Tennessee COPD also is fourth leading cause of death (Figure 2.6).

Leading Causes of Deaths in US, 2000

Other causes 28.0%
CVD 41.0%
Diabetes 3.0%
COPD 4.0%
Cancer 24.0%
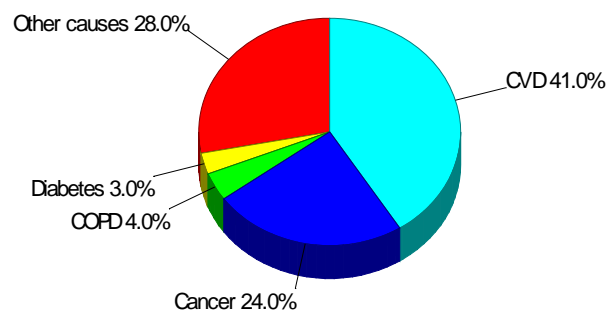
Adults Aged 25 Years and Older

Figure 2.5 Leading Causes of Deaths in US, 2000 (Anderson, 2002)

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**Leading Causes of Deaths in
Tennessee, 2000**

Other causes 29.0%

CVD 41.0%

Diabetes 3.0%

COPD 4.0%

Cancer 23.0%
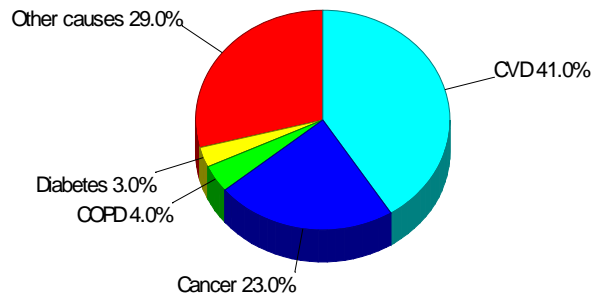
Adults Aged 25 Years and Older

Figure 2.6. Leading Causes of Deaths in Tennessee, 2000 (Anderson, 2002)

Smoking causes approximately 80 to 90 percent of COPD cases; a smoker is 10 times more likely than a nonsmoker to die of COPD. The American Lung Association in 2003 published that the occupational exposure to certain industrial pollutants and urban exposure to certain air pollutants increases the odds for COPD. This report indicated that a recent study found that the fraction of COPD attributed to work was estimated as 19.2%, while the fraction for air toxics was unknown. An estimated 11 million people were diagnosed with chronic bronchitis in 2001. Females have significantly higher rates of chronic bronchitis than males. In 2001, 3.7 million males had a diagnosis of chronic bronchitis compared to 7.5 million females. Finally, an estimated 3 million Americans have been diagnosed with emphysema sometime in their life. Of the emphysema sufferers, 57 percent are male and 43 percent are female (American Lung Association, 2003). According to the Heart, Lung, and Blood Institute (U.S. DHHS, 2003), Tennessee

experienced high death rates from 1996 to 1998, between 41.6 and 45.5 per 100,000 people (Figure 2.7). The annual cost to the nation for COPD is approximately $32.1 billion, including healthcare expenditures of $18.0 billion and indirect costs of $14.1 billion (Anderson, 2002).

C. Cardiovascular Disease

CVD is the leading cause of death in the United Stated. According to the American Heart Association's Heart Disease and Stroke Statistics 2003 Update, in 2000 CVD caused nearly 40% of all deaths (Figures 2.5 and 2.6), and was listed as primary or contributing cause in about 60%. Furthermore, although overall death rates from heart disease and stroke declined in the 1980s and 1990s, heart failure emerged as a major chronic disease for older adults. According to the American Heart Association, 61,800,000 Americans had one or more forms of CVD in 2000, 50,000,000 high blood pressure, 12,900,000 coronary heart disease, 7,600,000 myocardial infarction, 6,600,000 angina pectoris, and stroke 4,700,000. Cardiovascular diseases claimed 945,836 lives in 2000.

Almost 150,000 Americans killed by CVD were under age 65. From 1990 to 2000 death rates from CVD declined 17.0 percent. Despite this decline in the death rate, in the same 10-year period the actual number of deaths declined only 2.5 percent.
Also, heart disease is the leading cause of death in Tennessee, which accounted for 16,174 deaths or approximately 29% of the state's deaths in 2000. The death rate was 292.3 per 100,000 of people (CDC, 2003).

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*Age-adjusted to 2000 U.S. population; rate per 100,000 population
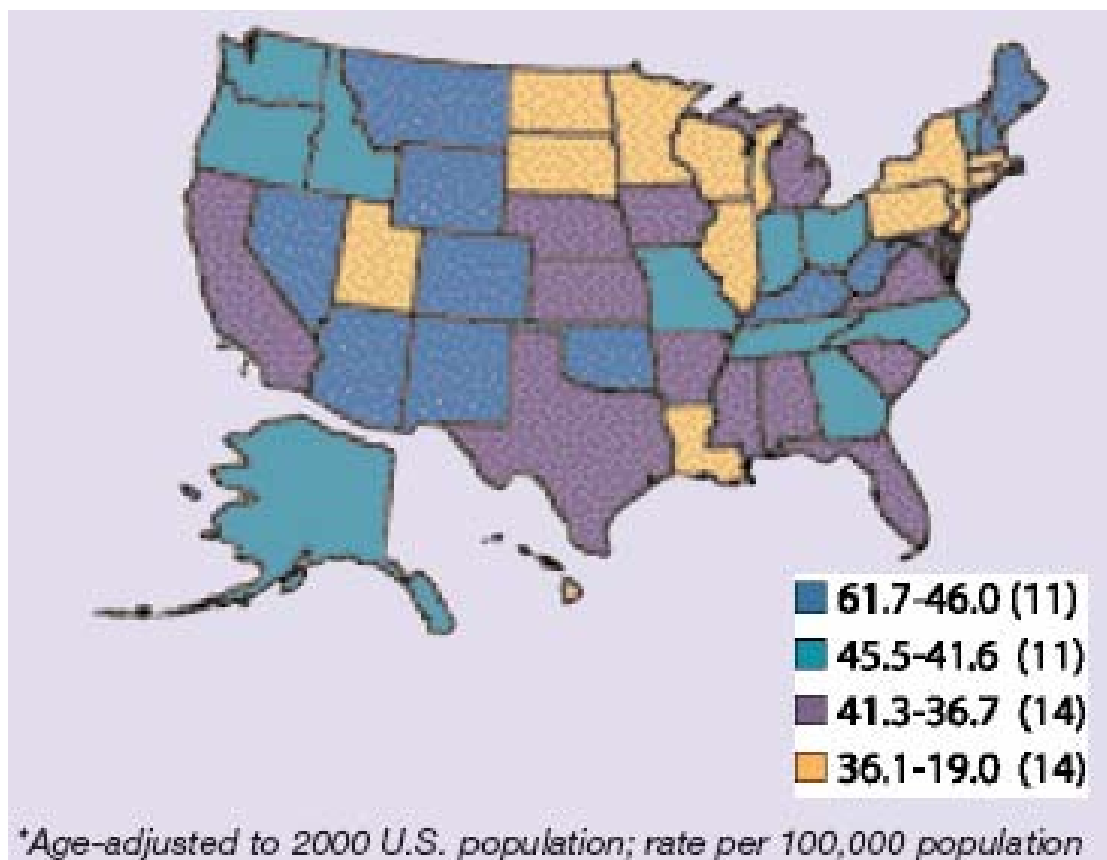
Figure 2.7. Age-Adjusted Death Rates* for COPD by State, U.S., 1996-1998

(Reprinted from U.S. DHHS, 2003)

In addition to age, traditional risk factors for CVD include lifestyle (such as smoking, physical inactivity, and diet), serum lipids, gender, race, and family history (genetics). However, these risk factors do not fully explain the etiology or incidence of CVD. Recent and strong evidence have indicated that exposure to air pollutants is also a CVD risk factor (Riedl et al., 2005; Delfino et al., 2005; Peters, 2000, 2001 and 2002; Lee et al., 2002; Oftedal et al., 2003; Lin et al., 2003; Nemmar et al., 2002; Le Tertre et al., 2002; Yoshizawa et al., 2002; Brunekreef, 2002; Brook et al., 2002; Donaldson et al., 2001; Ghio and Devlin, 2001; Dockery, 2001; Morris, 2001; Braga, 2001; Schwartz, 1999 and 2001; Moolgavkar, 2000; Peters et al., 2000).

The strongest and most consistent association between air pollution exposure and cardiovascular morbidity and mortality has been seen for ambient particulate matter (PM) and diesel exhaust (DE). DE consists of a complex mixture of gaseous and particle-bound chemicals. Chemicals present in the gaseous portion of DE include carbon monoxide and dioxide, nitrogen monoxide and dioxide, aldehydes, benzene, and polycyclic aromatic hydrocarbons (PAHs) (Madl et al., 2002). The particulate phase of diesel exhaust called DPM is composed of a solid carbonaceous core with PAHs, nitr-PAHs, and approximately 1800 other chemicals adsorbed to the surface of particles such as benzene, formaldehyde, and etc. (U.S. EPA, 2002c; Madl et al., 2002).

Large prospective epidemiologic studies have shown that living in areas with higher levels of ambient PM is associated with an increased risk of premature cardiopulmonary death, (Delfino et al., 2005; Zanobetti and Schwartz, 2005; Sioutas et al., 2005; Schultz et

al., 2005; Riedl et al., 2005; Pope et al., 2004a; Oftedal et al., 2003; Lin et al., 2003; Le Tertre et al., 2002; Yoshizawa et al., 2002; Brunekreef, 2002; U.S. EPA, 2002b; Brook et al., 2002; Dockery, 2001; Morris, 2001; Braga, 2001; Schwartz, 1999 and 2001; Moolgavkar, 2000; Peters et al., 2000). Likewise, PAHs such as benzo(a)pyrene have been shown in vitro to alter the redox environment in vascular walls, activating signaling pathways that can lead to proliferation of vascular smooth muscle cells, one of the hallmarks of atherosclerotic changes in the vessel wall. In addition, certain aldehydes, such as acrolein and 1, 3-butadiene, are cardiovascular toxicants and have been shown to induce proliferation of vascular smooth muscle cells as well.

2.1.3 Air Toxics and Confounders

Because cancer, asthma, COPD, and CVD are not single diseases, they do not have a single cause. Many causes or risk factors can contribute to a person's chance of getting those diseases. Risk factors can include such things as age, race, sex, genetic factors, life style, diet, virus, and exposure to chemicals, radiation, and tobacco. Genetic plays a large role for many cancers, such as breast and colon cancer, as well as on asthma and CVD. This means that a family's health history can be a risk factor for some types of cancers. The same occurs for asthma and CVD (Leikauf et al., 2002; Alberg et al., 2003).

Chronic lung cancer and respiratory diseases are due mainly to tobacco smoking, environmental tobacco smoke (ETS), HAPs, particulate matter, radon, ozone, and genetic problems (Leikauf et al., 2002; Beeson et al., 1998; U.S. EPA, 2001a; Wu et al., 2001;

Pope at al., 2002; Alberg et al., 2003; Delfino, 2002; Cohen, 2000; American Lung

Association, 2005). For this reason it is necessary to know which is the contribution of

HAPs or MSATs on those diseases. There is enough evidence that indicates that the

overall contribution of HAPs on total cancers is 0.2% (ACS, 2003), and for lung cancer

HAPs contribute between 1 and 2% (Alberg et al., 2003), however, for asthma, COPD,

and CDV it is not clear how much is an air toxics contribution (Delfino, 2002; Leikauf,

2002). Analyzing some air toxics, it is possible to establish that for the total HAPs, DPM

and 1, 3-butadiene are more important for cancer development (Woodruff, 2000;

CalEPA, 1998). Whereas, acrolein, formaldehyde, and DPM are more important for

asthma (Table 2-2) (Leikauf et al., 2002). One of the most important confounders for

lung cancer is ETS, which is a mixture of exhaled mainstream and side-stream smoke

consisting of over 4,000 chemicals.

Table 2-2. HAPs Contribution on Asthma and Total Cancer Cases

Air Toxic	Contribution	
	Asthma[1]	Cancer[2]
Benzene	1%	7%
1,3-Butadiene	N/A	17%
Formaldehyde	14%	8%
Acetaldehyde	3%	N/A
DPM	13%	40%
Acrolein	61%	N/A

(1):   Leikauf et al., 2002
(2):   Woodruff, 2000; CalEPA, 1998

ETS contains several human respiratory carcinogens (including benzo[a]pyrene, benz[a]anthracene, other polycyclic aromatic hydrocarbons, 4-aminobiphenyl, and nitrosadimethylamine) and irritants (including formaldehyde, acrolein, other aldehydes, cadmium, and other metals) (Leaderer et al., 1990). Twenty-nine of the 49 major components in ETS are HAPs (Leikauf, 2002). Although combustion is a major source of compounds in both ETS and HAPs, the physical and chemical properties of ETS differ from those of the ambient mixture of gaseous and particulate HAPs. Air toxics account for most of the toxicity of ETS because most respiratory irritants that are contained in ETS are HAPs (Leikauf, 2002). The levels of air toxics present in ETS are greater than in urban air.

2.1.4 Air Toxics Emissions and Ambient Concentrations

2.1.4.1   Air Toxics Emissions in the U.S.

The U.S. EPA compiles a HAPs inventory called the National Emissions Inventory (NEI) each 3 years, which has quantitative information concerning the emissions mass of air toxics emitted into the atmosphere from major (sources that emit or have the potential to emit 10 tons per year or more of any listed HAP or 25 tons per year or more of a combination of listed HAPs), area (sources that emit or have the potential to emit less than 10 tons per year of a single HAP and less than 25 tons per year of all HAPs combined), on-road, and non-road sources (U.S. EPA, 2005a) U.S. EPA has compiled

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both a baseline period (1990–1993), as well as 1996 and 1999 emissions estimates for the 188 air toxics as defined in the Clean Air Act (CAA). Actually, the U.S. EPA is compiling the NEI 2002. The NEI 1999 version 3 contains the most complete, up-to-date air toxics emissions estimates available. These emission summaries do not include diesel particulate matter (U.S. EPA, 2005a).

Based on NEI 1999 version 3, the nation wide 188 HAPs and 33 UATs emissions are relatively equally divided between the four types of sources, where the mobile sources account by the 43.8 and 45% respectively (Figures 2.8 and 2.9). However, this distribution varies from state to state and city to city (U.S. EPA, 2005a).

188 HAPs Emissions, 1999

Nonroad 15.3%

Major 25.0%

Onroad 28.5%

Area 31.2%

10,170 Mlbs
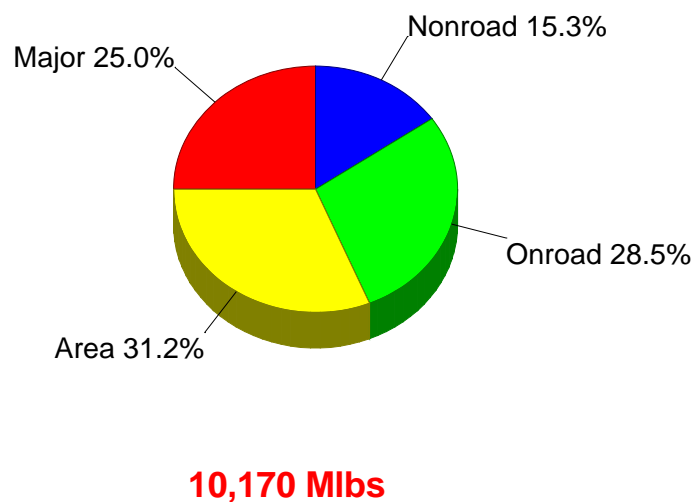
Figure 2.8. 188 National HAPs Emissions, 1999 (U.S. EPA, 2005a)

32

33 UATs Emissions, 1999

2,103 Mlbs
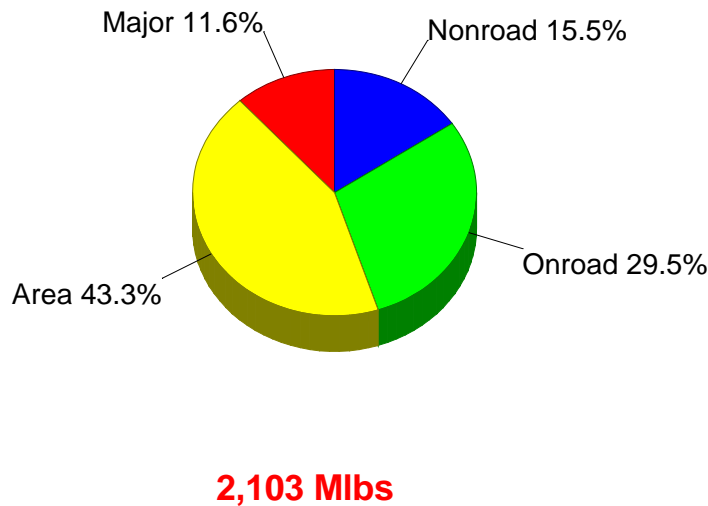
Figure 2.9. 33 National UATs Emissions, 1999 (U.S. EPA, 2005a)

Mobile sources account by 36.5 and 56.8% on the 188 HAPs and 33 UATs respectively in Tennessee, as shown in Figures 2.10 and 2.11 (U.S. EPA, 2005a). While mobile sources account by 49 and 54.4 % on the 188 HAPs and 33 UATs respectively for Davidson County, as illustrated in Figures 2.12 and 2.13 (U.S. EPA, 2005a). It indicates that strong regulations and cleaner technologies on mobile sources is the main challenge to reduce those UATs.

The relative importance of on-road emissions as a participant in air pollutants formation depends in large part on the total vehicle miles traveled (VMT) per day in a given area (Davis et al., 2002), since VMT growth rate is higher than the nationwide annual population growth 1970-1999, as shown in Figures 2.14 and 2.15 (U.S. DOT, 2005).

188 TN HAPs Emissions, 1999

Nonroad 10.3%

Major 45.9%

Onroad 26.2%

Area 17.6%

270.5 Mlbs
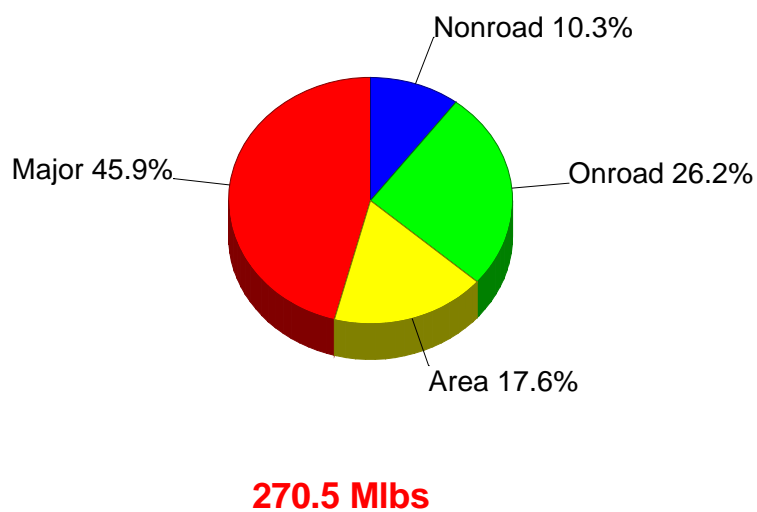
Figure 2.10. 188 Tennessee HAPs Emissions, 1999 (U.S. EPA, 2005a)

33 TN UATs Emissions, 1999

Major 21.1%

Nonroad 15.4%

Area 22.0%

Onroad 41.4%

36.6 Mlbs
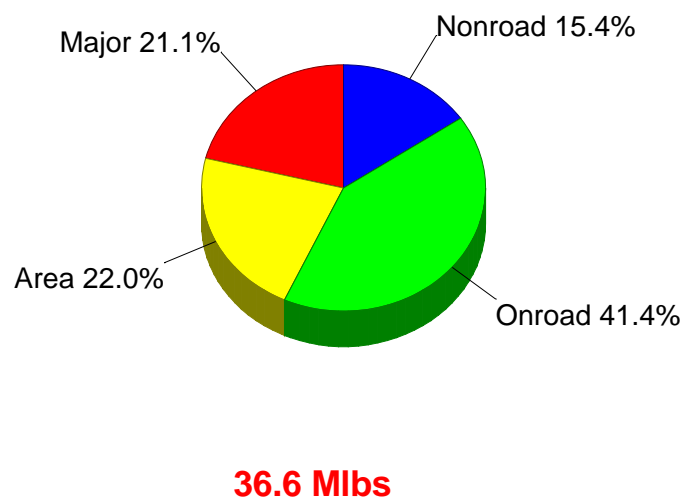
Figure 2.11. 33 Tennessee UATs Emissions, 1999 (U.S. EPA, 2005a)

188 Davidson County HAPs Emissions, 1999

Major 12.8%

Nonroad 10.4%

Onroad 38.6%

Area 38.2%
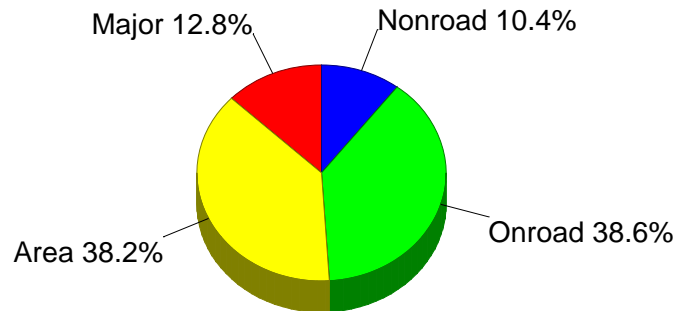
20.7 Mlbs

Figure 2.12. 188 Davidson County HAPs Emissions, 1999 (U.S. EPA, 2005a)

33 Davidson County UATs Emissions, 1999

Major 19.7%

Nonroad 14.0%

Onroad 40.4%

Area 26.0%
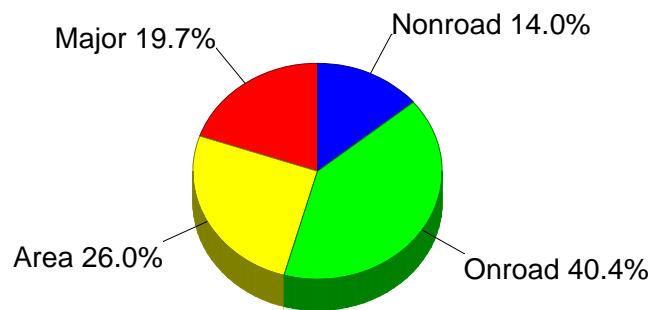
4.1 Mlbs

Figure 2.13. 33 Davidson County UATs Emissions, 1999 (U.S. EPA, 2005a)

Percentage of Change (1970-99)
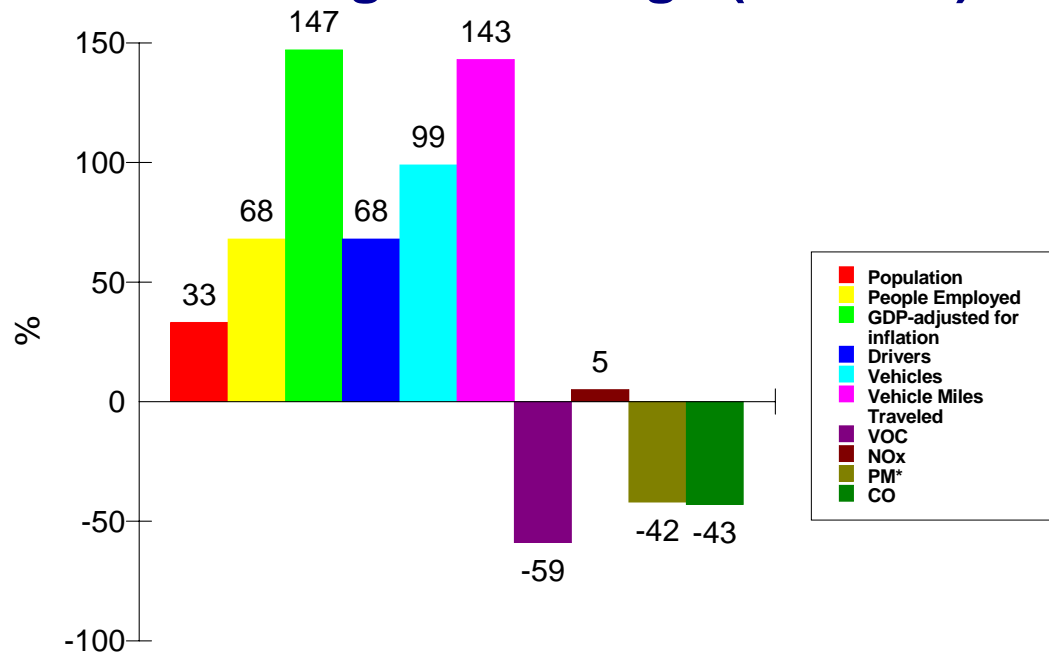
Motor Vehicle Emissions Related to
Demographics and Transportation

Figure 2.14. Percentage of Change (1970-99) (Reprinted from U.S. DOT, 2005)

Travel, Economic Growth and Population (1970-99)
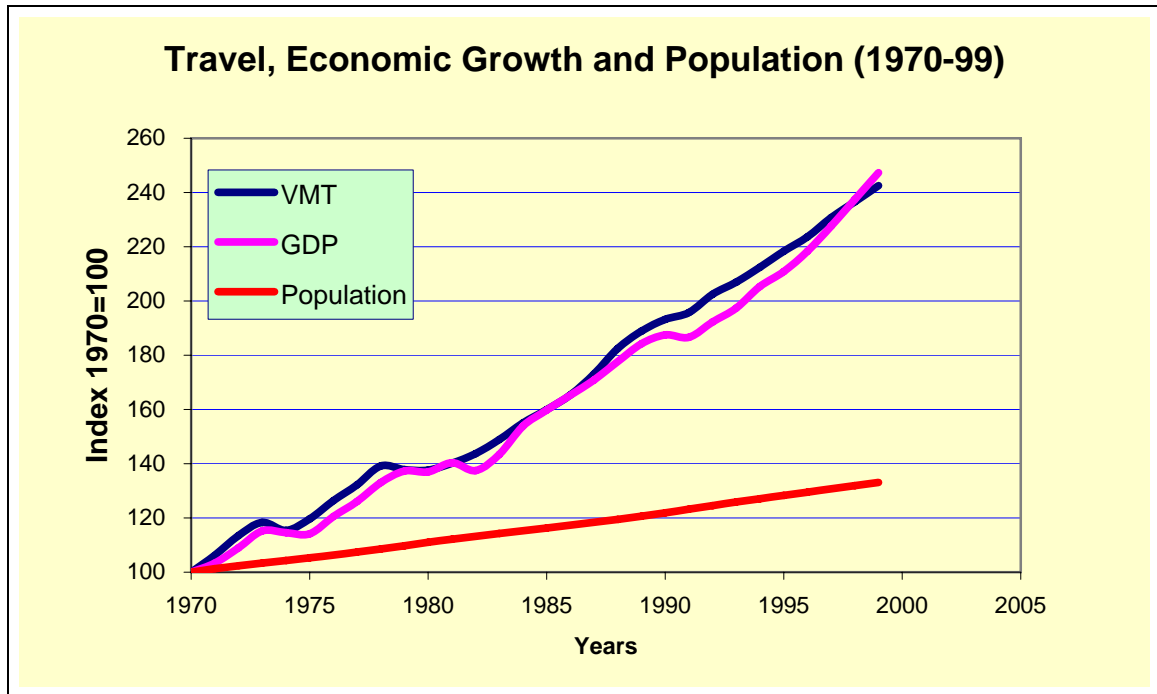
Figure 2.15. VMTs, Economic Growth (GDP), and Population (1970-99) (Reprinted from U.S. DOT, 2005)

In the future, the relative importance of on-road emissions will be affected by the growth in VMT, which results in increased emissions, and the implementation of motor vehicle emissions controls or the use of new fuels, which will reduce the emissions associated with each mile traveled (U.S. EPA, 2001a).

Current VMT data compiled by the U.S. Department of Transportation, DOT, provides the basis for estimating current emissions, and some states have estimated the growth in VMT in order to predict future on-road emissions, as is the case in Tennessee (Davis et

al., 2002), where the statewide annual VMT growth rate was estimated to be 3.9% from 1990 to 1999, which is higher than the statewide annual population growth.

This is consistent with the fact that the state of Tennessee was the thirteenth and fourteenth in on-road 188 HAPs and 33 UATs emissions respectively of the U.S. in 1999 NEI version 3. Whereas, Tennessee was the fifth and fourteenth in major sources 188 HAPs and 33 UATs emissions respectively of the U.S. (U.S. EPA, 2005a).

2.1.4.2 Air Toxics Monitoring Network in the U.S.

The estimated air toxics concentrations in urban areas are typically twice as high as in rural areas (Axelrad et al., 1999), however, as air toxics have been of major concern only recently, limited monitoring data is available.

A. Urban Air Toxics Monitoring Program

Since 1987, U.S. EPA has sponsored the Urban Air Toxics Monitoring Program (UATMP) to characterize the composition and magnitude of urban air pollution through extensive ambient air monitoring (U.S. EPA, 2004a). The original intent of UATMP was to screen ambient air samples for concentrations of toxics VOCs. The UATMP is a year-round sampling program collecting 24-hr integrated ambient air samples every 6 or 12 days at urban sites in the contiguous U.S. and its territories.

The 6- or 12-day sampling schedule permits cost-effective data collection for
characterization (annual-average concentrations) of toxic compounds in ambient air and
ensures that sampling days are evenly distributed among the 7 days of the week to allow
comparison of air quality on weekdays to air quality on weekends. Currently, there are
about 59 air toxics monitoring sites in operation, which include 37 urban locations as
shown in Figure 2.16 (U.S. EPA, 2004a). Some monitors were placed near the centers of
heavily populated cities (e.g., Denver, CO and Phoenix, AZ), while others were placed in
moderately populated areas (e.g., Beulah, ND and Des Moines, IA).
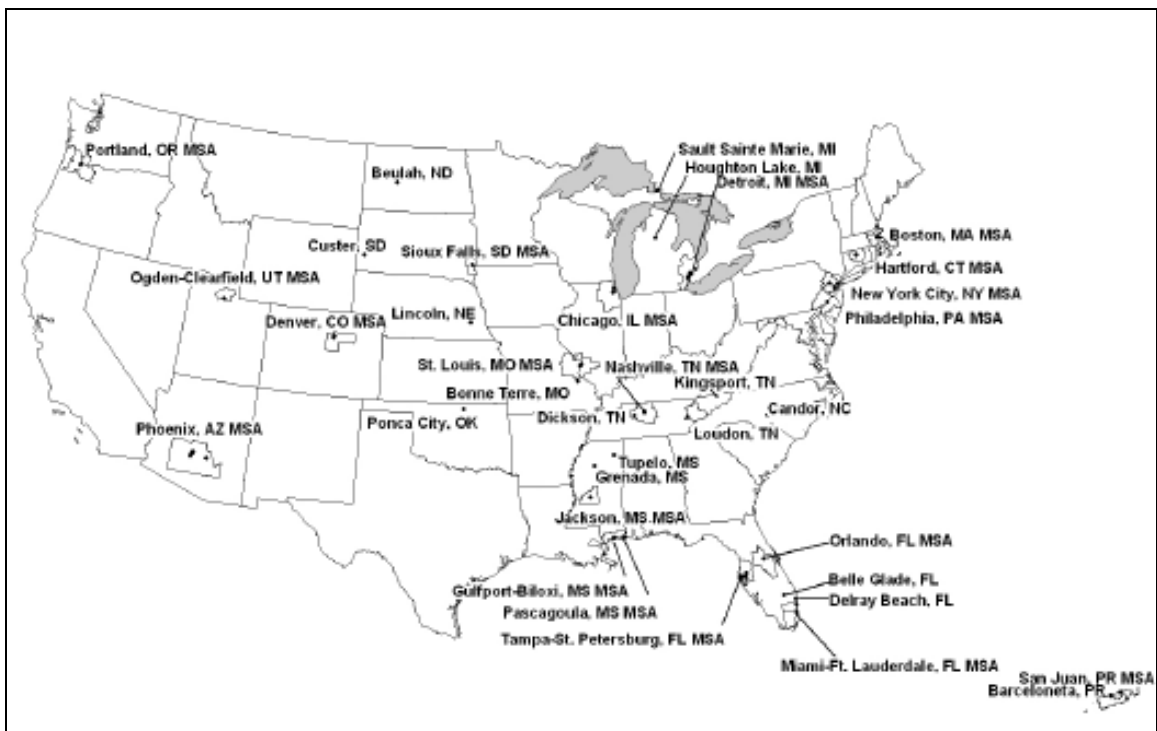
Figure 2.16. UATMP Network, 2003. (Reprinted from U.S EPA, 2004a)

In 1999, the U.S. EPA expanded the UATMP to provide for the measurement of additional HAPs to support Government Performance Results Act (GPRA) and the National Air Toxics Trends Stations (NATTS) (U.S. EPA, 2005c). The central goal of the NATTS network is to detect trends in high-risk air toxics. Among the 33 UATs, 25 pollutants have sufficient historical data for 6-year trends assessment in some urban areas. Although these ambient air toxics data are only available for a limited number of urban areas, the results generally reveal downward trends for most monitored air toxics (U.S. EPA, 2002d and 2004a). The most consistent improvements are apparent for benzene (Fruin et al., 2001).

All UATMP samples are analyzed in a central laboratory for concentrations of selected hydrocarbons, halogenated hydrocarbons, and polar compounds from the canister samples, carbonyl compounds from the cartridge samples, semivolatiles from the XAD-2® thimbles, hexavalent chromium from pre-treated filters, and metal compounds from filters. At every UATMP monitoring location, the air sampling equipment is installed in a small temperature-controlled enclosure (usually a trailer or a shed) with the sampling inlet probe protruding through the roof. With this common setup, every UATMP monitor sampled ambient air at heights approximately 5 to 20 feet above local ground level. As part of the sampling schedule, site operators are instructed to collect duplicate samples (U.S. EPA, 2004a).

In Tennessee, Nashville is the urban area included in the UATMP, which measures 33 UATs in two monitors, East Nashville Health Clinic (EATN) and Lockeland Middle

School (LOTN), working since May and April of 2002 respectively (Figure 2.17). The EATN (47-037-0011) site is located on the roof of East Health Center, which is north (predominately downwind) of downtown Nashville and is a population-oriented site predominantly influenced by primarily commercial and mobile sources. Population residing within 10 miles of the monitoring station is 518,357.

The LOTN (47-037-0023) site is a core site located on the roof of Lockland School, which is in the heart of downtown Nashville. This is also a population-oriented site influenced primarily by commercial and mobile sources. Population residing within 10 miles of the monitoring station is 552,749. These sites were selected for the following reasons: these provided secure locations with the necessary electrical service, represented areas that were not in the immediate vicinity of large air pollution sources, and these were in the proximity and down wind from areas with the highest population density in metropolitan area. It is important to note that these sites are near substantial interstate routes and local traffic corridors, such as I-40. Air toxics concentrations at these sites would not be indicative of average concentrations throughout Nashville nor could specific conclusions be drawn from concentrations at these sites concerning concentrations at any other location. Actual concentrations may be higher adjacent to industrial facilities and may be lower in less densely populated areas. However, the results of the ambient monitoring at these sites provides concentrations to which the majority of the Nashville population would be exposed because monitoring occurred near areas with the densest population in Nashville, as shown in Figure 2.17 (Hissam, 2003).
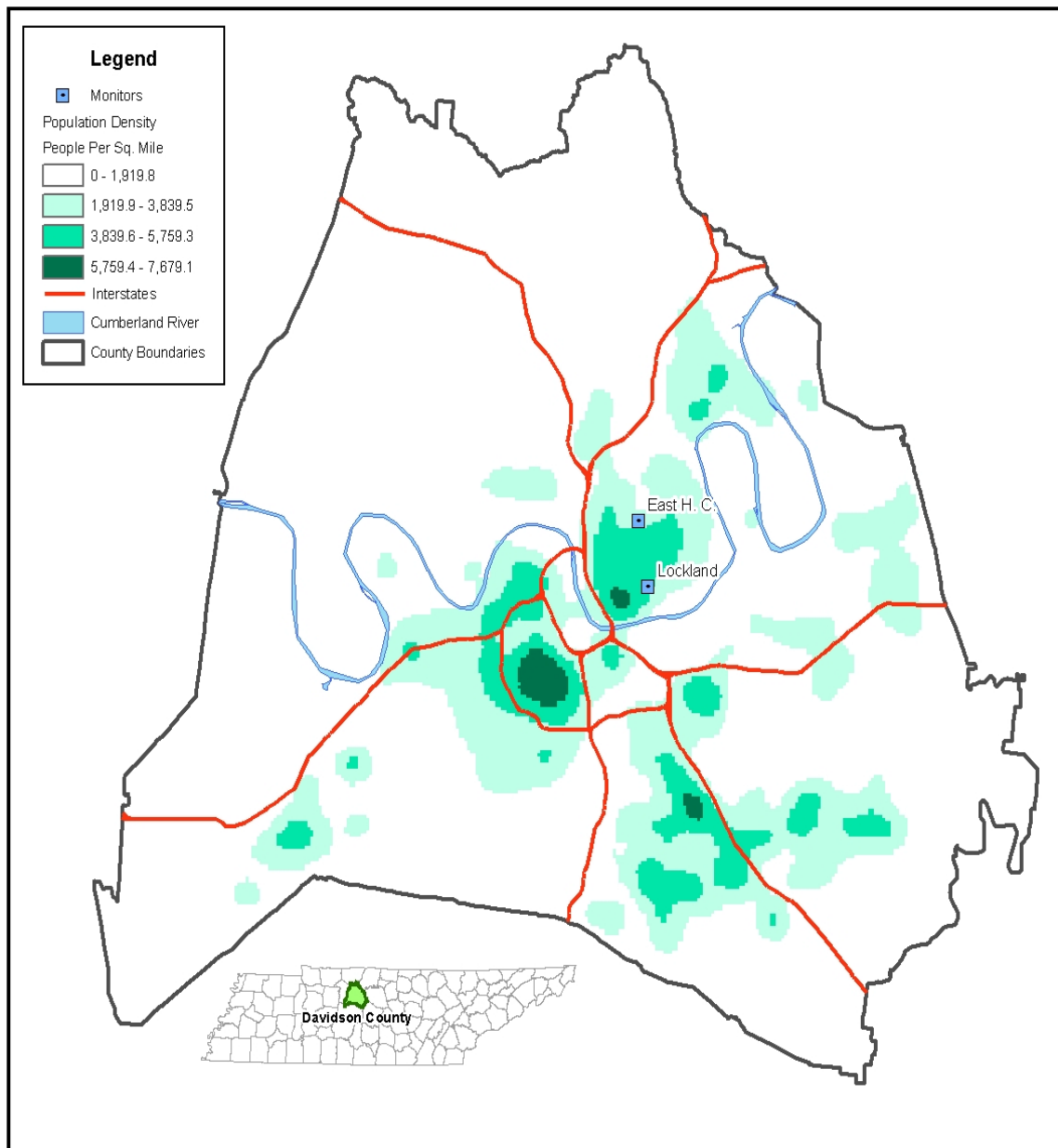
Figure 2.17. Population Density and the Locations of Two Air Toxics Monitors in Davidson County, Tennessee (Reprinted from Hissam, 2003)

Since EATN and LOTN sites are relatively close each other, the UATMP monitoring

data can be merged to represent the air toxics concentrations in Nashville. The available

2002-2003 daily HAPs concentrations for benzene, formaldehyde, and acetaldehyde are

illustrated in the Figures 2.18, 2.19, and 2.20, respectively (Source: Metropolitan Public

Health Department of Nashville and Davidson County, 2004).

These figures show seasonal effect on formaldehyde and acetaldehyde, whose

concentrations are higher in summer season (June, July, August, and September).

Benzene did not show a seasonal pattern.
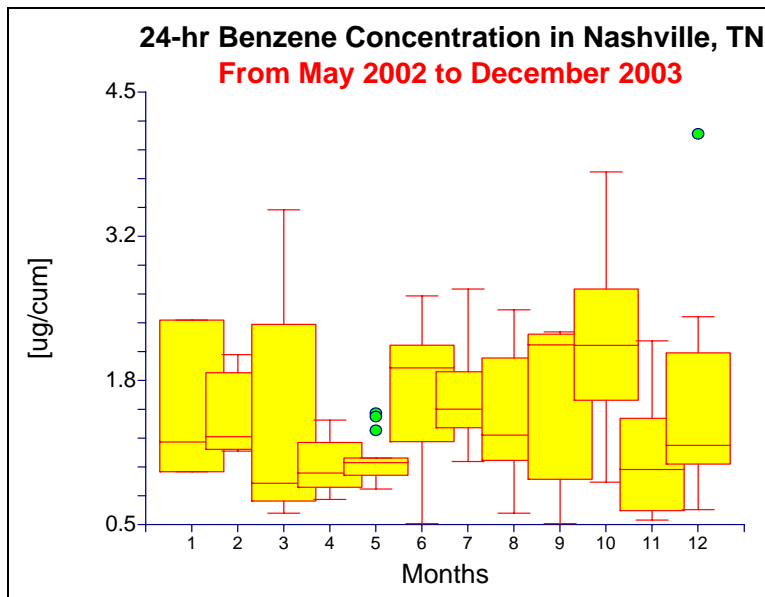
Figure 2.18. 24-hr Benzene Concentration in Nashville, TN. From May 2002 to December 2003
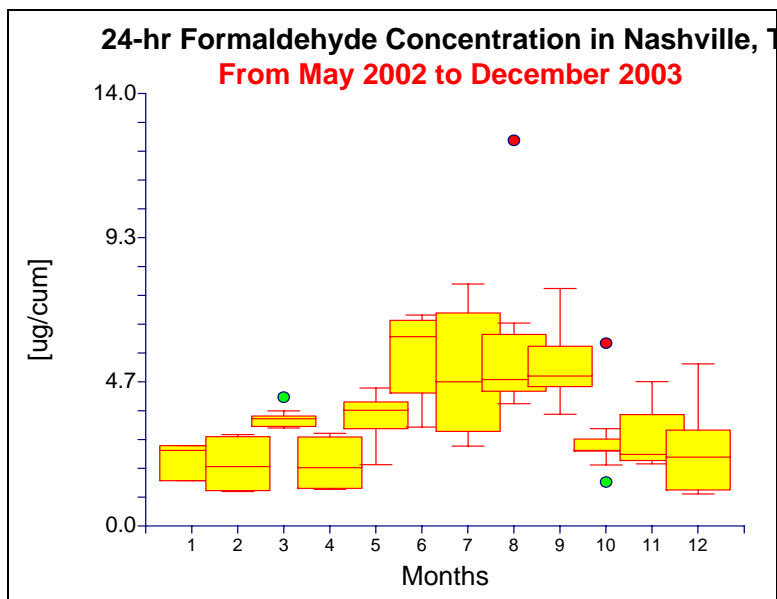
Figure 2.19. 24-hr Formaldehyde Concentration in Nashville, TN. From May 2002 to December 2003
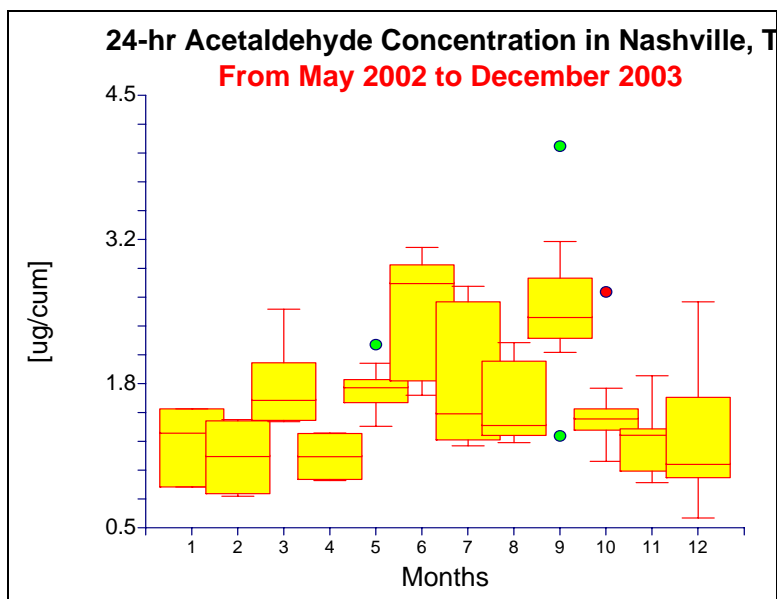
Figure 2.20. 24-hr Acetaldehyde Concentration in Nashville, TN. From May 2002 to December 2003

B. Photochemical Assessment Monitoring Stations

In accordance with the 1990 CAAA, the U.S. EPA has required more extensive

monitoring of ozone and its precursors in areas with persistently high ozone levels

(mostly large metropolitan areas). In these areas, the States have established ambient air

monitoring sites called Photochemical Assessment Monitoring Stations (PAMS) (U.S.

EPA, 2005d), which collect and report detailed data for VOCs, nitrogen oxides, ozone,

and meteorological parameters. This program requires routine year-round measurement

of nine air toxics: acetaldehyde, benzene, ethylbenzene, formaldehyde, n-hexane, styrene,

toluene, xylenes, and 2, 2, 4-trimethlypentane. At the present time, the collection of

current state and local air toxics monitoring data and PAMS data is limited in its

geographic scope and it does not cover many air toxics for most states, including

Tennessee. In addition, the sites are not necessarily at locations that represent the highest

area-wide concentrations. Nevertheless, these can still be used to provide useful

information on the trends in ambient air toxics.

So far, the U.S.EPA is working together with state and local air monitoring agencies to

build upon these sites to develop a monitoring network with the following objectives: to

characterize air toxics problems on a national scale; to provide a means to obtain data on

a more localized basis as appropriate and necessary, and to help evaluate air quality

models (U.S. EPA, 2005c). However, there are a significant number of the 188 air toxics

for which U.S. EPA does not yet have a monitoring method developed, like DPM and

acrolein. For this reason, U.S. EPA is spending its resources on building up the air toxics

monitoring network, as is the case of the Air Toxics Data Archive (ATDA) (U.S. EPA, 2005e). The ATDA web site is sponsored by the STAPPA/ALAPCO/USEPA Air Toxics Monitoring Subcommittee and its purpose is to provide users with the capability to browse and query existing air toxics monitoring data. Such analyses of these data will provide information about the spatial pattern, temporal profile, and general characteristics of various air toxic compounds from UATM, PAMs, and others programs (U.S. EPA, 2005e).

These measurements are used to derive trends in air toxics concentrations to help evaluate the effectiveness of air toxics reduction strategies. They also can provide data to support and evaluate dispersion and deposition models, one of them has been the Assessment System for Population Exposure Nationwide (ASPEN) (U.S. EPA, 2000b and 2002a), which has been used to estimate the national air toxics concentrations and to gain a greater understanding of the spatial distribution of concentrations of these 33 UATs resulting from contributions of multiple emissions sources. As an example, the predicted 1999 benzene concentration for Tennessee is shown in the Figure 2.21; where it is possible see that the maximum benzene concentrations are in the urban areas, such as Nashville, Knoxville, Chattanooga, and Memphis. However, ASPEN has under predicted HAPs' concentrations due to a number of limitations of the Gaussian model formulation, such as neglect of calm wind conditions, secondary air toxics formation, poor representation of stable atmospheric conditions that typically occur at night, and a 50-km downwind distance limit. This would require modeling that accounts for large-scale dispersion associated with three-dimensional wind fields (Rosenbaum et al., 1999).
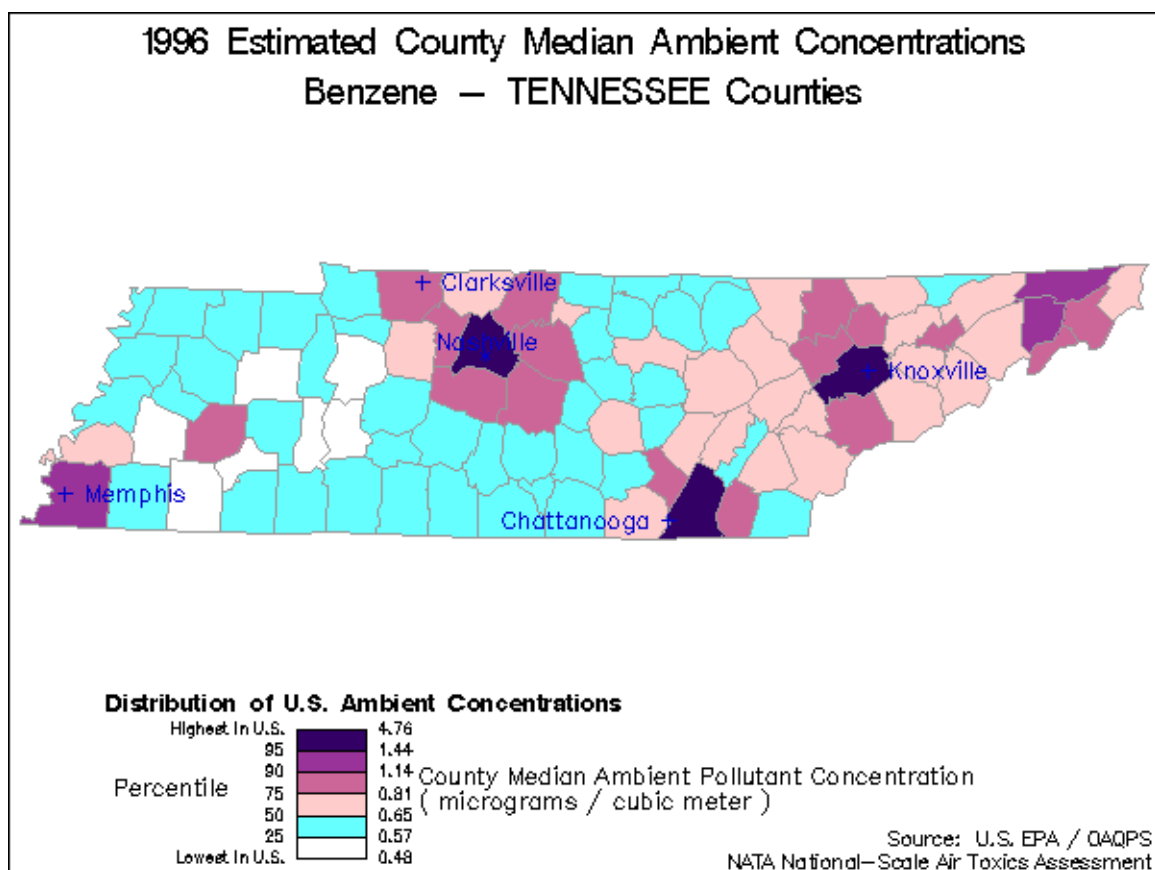
Figure 2.21. 1996 Estimated County Median Ambient Concentrations. Benzene –

Tennessee Counties (U.S. EPA, 2002a)

Although these ambient air toxics data are only available for a limited number of urban areas, the results generally reveal downward trends for most monitored air toxics. The most consistent improvements are apparent for benzene, which is predominantly emitted by mobile sources, oil refineries, and chemical processes (Fruin et al 2001). The urban areas generally have higher levels of benzene than other areas of the country due to the motor vehicles, where U.S. EPA has estimated 85% of ambient benzene comes from mobile sources: 60% from on-road sources and 25% from off-road sources (U.S. EPA, 1998). From 1994 to 2000, annual average concentrations for benzene declined 47 % as shown in Figure 2.22 (U.S. EPA, 2002d and 2004a).

The change in national benzene emissions is attributed to a combination of new car emission standards, use of cleaner fuels in many states, as well as stationary source emission reductions. Ambient concentrations of toluene, emitted primarily from mobile sources, also show a consistent decrease over most reporting locations. Similar to benzene, annual average toluene concentrations dropped 48 % (U.S. EPA, 2002d and 2004a).

Other air toxics, including 1,3-butadiene, styrene, also reveal air quality improvement, but the downward trends are not significant across large numbers of monitoring locations (U.S. EPA, 2002d and 2004a). During this period, the U.S. EPA phased in new car emission standards called TIER 1; required many cities to begin using cleaner burning gasoline; and set standards that required significant reductions in benzene, 1,3-butadiene, and other pollutants emitted from oil refineries and chemical processes.
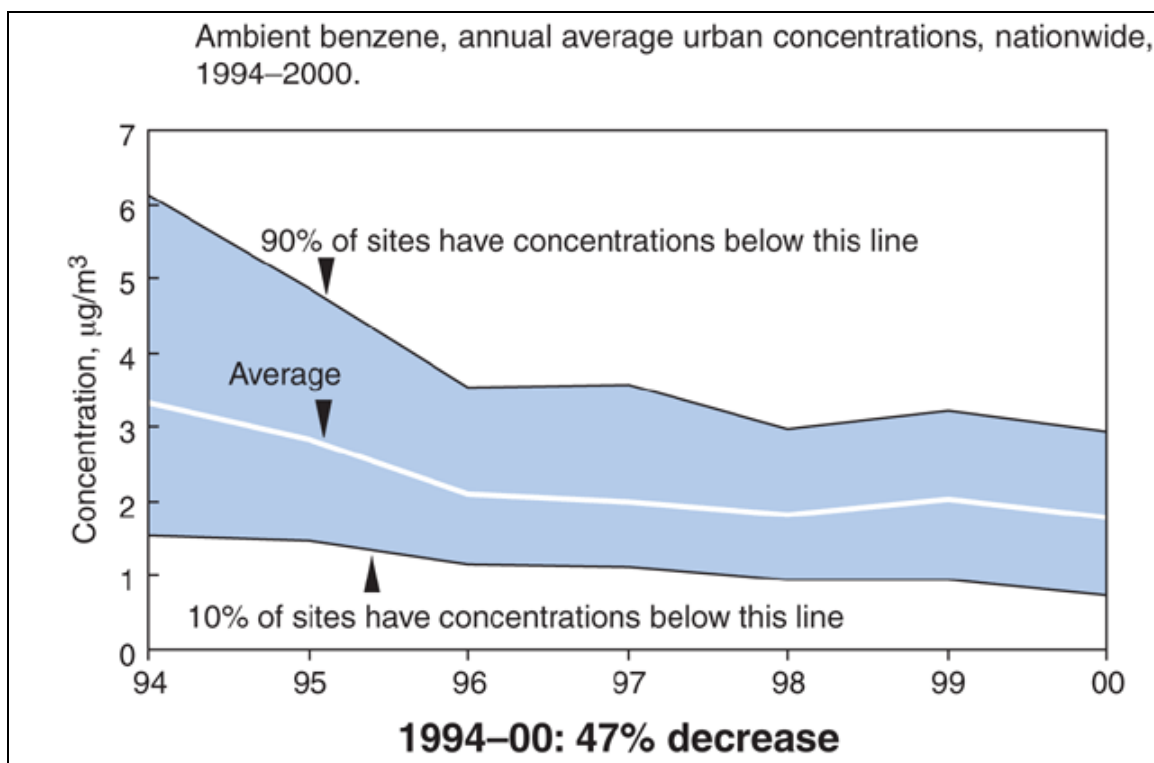
Figure 2.22. Ambient Benzene, Annual Average Urban Concentrations, Nationwide, 1994-2000 (Reprinted from U.S. EPA, 2002d)

The California's improvements in benzene, 1,3-butadiene, and toluene are primarily attributed to the reformulation of gasoline and new-car improvements in terms of emission controls (Morrow, 2001). Ambient air toxics data in rural areas are much more limited, but the results also indicate widespread air quality improvement for many monitored air toxics. Significant downward trends are noted among the few rural sites for benzene and several other VOCs. Lead concentrations in rural areas are also down (U.S.EPA, 1999b and 1999c).

2.1.5 Air Toxics Human Health Risk Assessment

Since most cancer and non-cancer health impacts of air toxics cannot be directly isolated and measured, risk assessment methods and tools have been developed to assist in evaluating them according to the risk assessment paradigm, base on toxicological evidence (U.S. EPA, 2005b; Asante-Duah, 2002; U.S. EPA, 2001c). The risk assessment paradigm includes 4 components: hazard identification, dose-response, exposure assessment, and risk characterization.

Hazard identification involves characterizing the behavior of a chemical within the body, the health effects that may be caused by the chemical, and the exposure conditions associated with those health effects. This typically includes the following: physical-chemical properties, routes and patterns of exposure, metabolic and pharmokinetic data; toxicological studies, including short-term (acute effects) tests and long-term (chronic effects) animal studies, human studies, and ancillary information, including in vitro studies and structure-activity relationships. The availability and accuracy of this information varies widely depending on the substance and the adverse health effects such as cancer and non-cancer (U.S. EPA, 2005b; Williams, 2004; Tam and Neuman, 2004; Asante-Duah, 2002; Teuschler et al., 2001; Partt et al., 2000). All of the information is taken together to establish the weight of evidence that a substance is capable of causing a particulate effect.

Once the potential hazard associated with exposure to a pollutant is established, the dose-response component projects the potential effects on humans at varying exposure concentrations. The nature of quantitative dose-response assessment varies among pollutants (Tam and Neuman, 2004; Asante-Duah, 2002; Teuschler et al., 2001). From this quantitative dose-response relationship, toxicity values are derived for use in risk characterization. Those toxicity values are numerical expressions of the relationship between a given level of exposure to an air toxics and adverse health impacts. The two most common toxicity values for inhalation exposures are the upper-bound inhalation unit risk estimates (IURs) for cancer effects and reference concentrations (RfCs) for non-cancer effects, which include uncertainty factors (U.S. EPA, 2000b, 2001c, 2005b).

An exposure assessment is the quantitative or qualitative evaluation of contact to a specific pollutant of concern and includes such characteristics as magnitude, duration and route of exposure of an organism to a pollutant of concern. The numerical output of an exposure assessment may be either exposure or dose, depending on the purpose of the evaluation and available data (U.S. EPA, 2005b, 2001c; Payne-Sturges et al., 2004). Information important to the assessment of exposure to an air toxics includes: the nature of the source and the releases, the different pathways by which humans can be exposed, transport and fate characterizes of the contaminant, human intake patterns, the number of people potentially exposed, the frequency and duration of the exposures, and the location and activities of the exposed population. All this information can be explicitly identified in an exposure conceptual model (U.S. EPA, 2005b; Payne-Sturges et al., 2004).

In general for an air toxics exposure conceptual model (Figure 2.23), HAPs can enter to the environment by a number of pathways, including release into the air, soil, or water, however, for humans point of view, air and inhalation exposure is the most relevant (Leikauf et al 2002). The most important route of entry into the environment is total air release, which often is the largest source of release, mainly mobile-source releases. Exposure also depends on the intrinsic physical-chemical properties of each compound, including vapor pressure and solubility in various media.

In addition, certain attributes of the manufacturing and generating procedures can influence chemical speciation of stack releases or engine pipes (U.S. EPA, 2005b). Highly volatile substances can more readily escape into the ambient air and thus cause added concern. Many of the HAP compounds persist in the air by processes that dominate the formation of the urban aerosol (Baird, 2001; Asante-Duah, 2002). Another route involves formation through secondary reactions in the atmosphere. For example, several reactive hydrocarbons are formed during combustion and can accumulate in the atmosphere and react to produce some aldehydes such as formaldehyde, acrolein, and acetaldehyde (Baird, 2001).

These compounds are contained in urban photo-oxidant plumes and contribute to ozone formation. Because ozone formation depends on reactive hydrocarbon species, the continuous measurement of ozone concentrations could be useful in estimating the ambient concentrations of precursors that include HAPs (Leikauf et al., 2002).
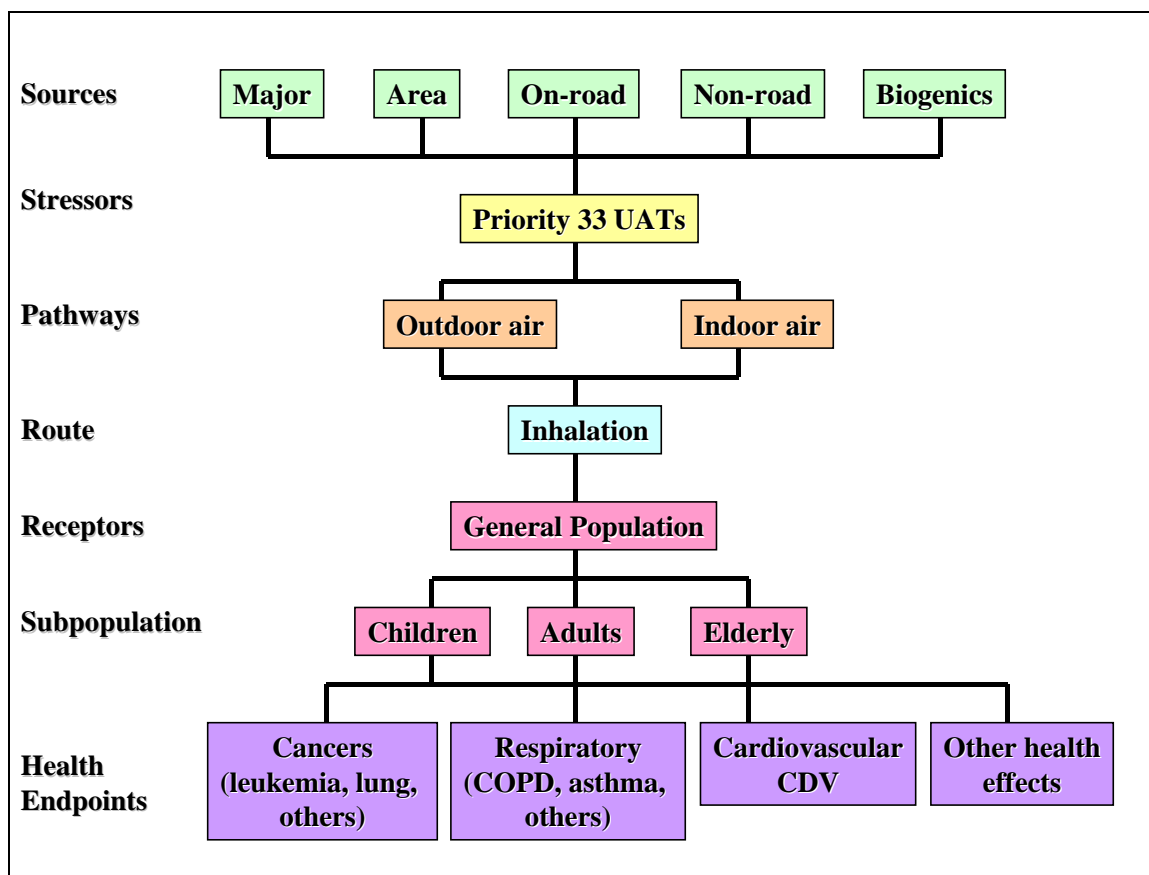
Figure 2.23. Air Toxics Exposure Conceptual Model

Interestingly, once inhaled, some HAPs are likely to react with unsaturated fatty acids in the airway lining fluid or the cell membranes to form aldehydes, hydroxyhydroperoxides, and hydrogen peroxide (Leikauf et al., 2002; Delfino, 2002), thus, the source of these types of HAPs is mixed.

Besides direct release into the air and secondary formation, volatile HAPs can enter the atmosphere through intermediate transport. Even though a chemical is released initially into water, soil, sediment, or biota, if volatile it will enter the atmosphere eventually through evaporation from water or soil (Baird, 2001; Asante-Duah, 2002). Dominated by proximity to mobile sources, intermittent exposure to HAPs mixtures in high concentrations can depend on regional meteorology, atmospheric dispersion, transport, and removal. However, most research on the effects of chemicals on biologic systems is conducted on one chemical at a time. In the real world people are exposed to mixtures, not single chemicals (Carpenter et al., 2002; Teuschler et al., 2001). Although various substances may have totally independent actions, in many cases two substances may act at the same site in ways that can be either additive or non-additive. Many even more complex interactions may occur if two chemicals act at different but related targets. In the extreme case there may be synergistic effects, where the effects of two substances together are greater than the sum of either effect alone. In reality, most persons are exposed to many chemicals, not just one or two, and therefore the effects of a chemical mixture are extremely complex and may differ for each mixture depending on the chemical composition. This complexity is a major reason why mixtures have not been well studied (Carpenter et al 2002; Teuschler et al., 2001).

In risk characterization, the information from hazard identification, exposure assessment, dose-response assessment are summarized and integrated into quantitative and qualitative expressions of risk (U.S. EPA, 2005b, 2001c). To estimate potential non-carcinogenic effects, comparisons are made between projected intakes of substances and toxicity values; to estimate potential carcinogenic effects, probabilities that an individual will develop cancer over a lifetime of exposure are determined from projected intakes and chemical-specific dose-response information. Major assumptions, scientific judgments, and to the extent possible, estimates of the uncertainties embodied in the assessment are also presented (U.S. EPA, 2005b, 2001c).

The toxicity values for cancer risk are calculated using unit risk values (Eq. 2-1). The annual mean HAPs concentration from a location is multiplied by its unit risk to produce a cancer risk. HAPs with cancer risks greater than 1×10^{-6} are considered a potential human health concern (U.S. EPA, 2001c).

$$\text{Cancer Risk} = C_i \left[\frac{\text{ug}}{\text{m}^3} \right] \text{x IUR}_i \left[\frac{\text{m}^3}{\text{ug}} \right] \qquad\qquad \text{Equation 2-1.}$$

Assuming an additive air toxics mixture effect, the cumulative cancer risk is defined by the equation 2-2.

$$\text{Cumulative Cancer Risk} = \sum C_i \left[\frac{\text{ug}}{\text{m}^3} \right] \text{x IUR}_i \left[\frac{\text{m}^3}{\text{ug}} \right] \qquad\qquad \text{Equation 2-2.}$$

Where, C_i is the annual concentration for a particulate air toxic.

Hazard ratios for chronic non-carcinogenic HAPs can be calculated using the RfC toxicity value (Eq. 2-3). For HAPs at a particulate location, the annual mean concentration can be divided by its respective RfC. HAPs with a hazard ratio above 1.0 can be deemed to pose a potential human health concern (U.S. EPA, 2001c).

$$\text{Hazard Ratio} = \frac{C_i \left[\frac{ug}{m^3} \right]}{RfC_i \left[\frac{ug}{m^3} \right]} \qquad \text{Equation 2-3}$$

Hazard ratios for HAPs exceeding 1.0 can be summed to calculate a total hazard index as shown in Equation 2-4.

$$\text{Total Hazard Index} = \sum \frac{C_i \left[\frac{ug}{m^3} \right]}{RfC_i \left[\frac{ug}{m^3} \right]} \qquad \text{Equation 2-4.}$$

From an epidemiological point of view, several long-term exposure epidemiological studies have demonstrated strong evidence that changes in fine particulate matter concentration result in changes in a number of health effects, such as lung cancer mortality, CDV mortality, COPD chronic illness, and asthma hospital admissions, among others. That relationship can be expressed through concentration-response (C-R) functions. For example, the PM$_{2.5}$ concentration can be in ug/m^3 per day or year, and the population response may be the number of premature deaths per 100,000 peoples per day

or year. The researcher chooses a function form, and the parameters of the function are estimated using data on the $PM_{2.5}$ and the health response. There are several different functional forms that have been used for C-R functions, such as linear models, Poisson (log-linear), and logistic regressions, among others. The models most commonly used are the Poisson and logistic regression. For the Poisson regression, the natural logarithm of the health response is a linear function of the $PM_{2.5}$ or other pollutant concentration, while a logistic regression is used to estimate the probability of an occurrence of an adverse health effect, where the natural logarithm of the odds ratio is a linear function of $PM_{2.5}$ or other pollutant concentration.

However, many epidemiological studies do not report the C-R function, but instead report some measure of the change in the population health response associated with a specific change in the pollutant concentration. The most common measure reported are the relative risk and odds ratio associated with a given change in the pollutant concentration. A general relationship between the change in concentration of $PM_{2.5}$, ΔPM, and the corresponding change in the population health response, Δy, can, however, be derived from the relative risk or odds ratio.

For a Poisson regression, the model relationship defines the incidence rate (y) as:

$$y = A \bullet e^{\beta \cdot PM} \hspace{4cm} \text{Equation 2-5.}$$

Where the parameter A is the incidence rate of y when the concentration of $PM_{2.5}$ (PM) is zero, the parameter β is the coefficient of PM.

The relationship between ΔPM and Δy is:

$$\Delta y = yc - yo = Ae^{\beta \cdot PM_c} - Ae^{\beta \cdot PM_o}$$

Equation 2-6.

Where *yo* is the risk (i.e., probability of an occurrence) at the baseline $PM_{2.5}$ exposure and *yc* is the risk at the control $PM_{2.5}$ exposure.

Epidemiological studies often report a relative risk for a given ΔPM, rather than the β coefficient. Thus, the relative risk (RR) is simply the ration of two risks:

$$RR = \frac{yo}{yc} = e^{\beta \cdot \Delta PM}$$

Equation 2-7

Taking the natural log of both sites, the coefficient in the Poisson regression can be derived as:

$$\beta = \frac{\ln(RR)}{\Delta PM}$$

Equation 2-8.

For a logistic regression, the model relationship defines the odds ratio (OR) as:

$$odds\ ratio = \frac{\left(\dfrac{yc}{1-yc}\right)}{\left(\dfrac{yo}{1-yo}\right)} = \frac{e^{PM_c\beta}}{e^{PM_o\beta}} = e^{\Delta PM \cdot \beta}$$

Equation 2-9.

Taking the natural log of both sites, the coefficient in the logistic regression can be derived as:

$$\beta = \frac{\ln(odds\ ratio)}{\Delta PM}$$

Equation 2-10.

Long-term exposure to fine particulate matter, such as DPM, may result in premature death. For lung cancer mortality, Pope et al. (2002) performed an analysis that used data from the largest available prospective cohort study of mortality collected by the American Cancer Society (ACS), which included 116 metropolitan areas, 50 states, and about 1.2 million adults (ages 30 and older), from 1982 to the first three quarters of 2000. Using Poisson regression, Pope et al., (2002) found robust association between ambient $PM_{2.5}$ and elevated risk (RR) of lung cancer mortality, providing the strongest evidence to date that long-term exposure to a change in annual $PM_{2.5}$ mean average exposure of 10.0 ug/m^3 is an important health risk. Overall, they found a 13.5% (95% CI, 4.4-23.4%) increasing risk of lung cancer mortality. The estimated coefficient and standard error for $PM_{2.5}$ from their regression were (Pope et al., 2002):

Lung cancer mortality: β= 0.012663 and σ= 0.004265

In 2004, using the same ACS data and Poisson regression, Pope et al. (2004a) found strong association between ambient $PM_{2.5}$ and elevated risk (RR) of CVD (ischemic heart disease) mortality among never smokers, providing also the strongest evidence to date that long-term exposure to a change in annual $PM_{2.5}$ mean average exposure of 10.0 ug/m^3 is an important health risk on CVD mortality. Overall, they found a 22.0% (95% CI, 14.0-29.0%) increasing risk of ischemic heart disease mortality. The estimated coefficient and standard error for $PM_{2.5}$ from their regression were (Pope et al., 2004a):

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CVD mortality: $\beta= 0.019885$ and $\sigma= 0.002553$

Abbey et al. (1995) examined the relationship between the annual mean $PM_{2.5}$

concentration from 1966 to 1977 and the chronic respiratory symptoms in a sample

population of 1,868 Californian Seventh Day Adventists. The initial survey was

conducted in 1977 and the final survey in 1987. To ensure a better estimate of exposure,

the study participants had to have been living in the same area for an extended period of

time. Using logistic regression, they found strong association between ambient $PM_{2.5}$ and

development of COPD (chronic bronchitis) among adults over 27 year old exposed to a

long-term exposure to a change in annual $PM_{2.5}$ mean average of 45 ug/m^3. Other

pollutants were not examined. Overall, they found an OR of 1.81 (95% CI, 0.98-3.25)

increasing risk of chronic illness for COPD. The estimated coefficient and standard error

for $PM_{2.5}$ from their regression were (Abbey et al., 1995):

COPD (chronic bronchitis): $\beta= 0.01370$ and $\sigma= 0.00680$

Sheppard et al. (1999) found a significant relation between air pollution in Seattle and

non-elderly (<65) hospital admissions for asthma from 1987 to 1994. They used air

quality data for $PM_{2.5}$, PM_{10}, CO, O_3, and SO_2 in a Poisson regression model with control

for time trends, seasonal variations, and temperature-related weather effects. In response

to concerns that the work by Sheppard et al. (1999) may be biased, Sheppard (2003)

reanalyzed some of this work. In particular, Sheppard reanalyzed the original study's

$PM_{2.5}$ single pollutant model. He found strong association between ambient $PM_{2.5}$ and

hospital admissions for asthma among adults less than 65 year old exposed to a long-term

60

exposure to a change in daily $PM_{2.5}$ mean average of 11.8 ug/m^3. Overall, he found a

4.0% (95% CI, 1.0-6.0%) increasing risk of hospital admissions for asthma. The

estimated coefficient and standard error for $PM_{2.5}$ from their regression were (Sheppard,

2003):

Hospital admissions for asthma: $\beta= 0.003324$ and $\sigma= 0.001045$

2.1.5.1 Previous Air Toxics Risk Assessment

In recent times, the U.S. EPA's National-Scale Air Toxics Assessment (NATA) has

modeled those 33 UATs across the U.S. for 1996 and 1999 (U.S. EPA, 2002a). NATA is

a nationwide analysis of air toxics and has used a computer modeling called Assessment

System for Population Exposure Nationwide (ASPEN) of the 1996 and 1999 NEI air

toxics data as the basis for developing health risk estimates on census tracts (U.S. EPA,

2002a). Census tracts are land areas defined by the U.S. Bureau of the Census and

typically contain about 4,000 residents each. Census tracts are usually smaller than 2

square miles in size in cities, but much larger in rural areas (U.S. EPA, 2002a).

The NATA is intended to provide state, local, and tribal agencies and others with a better

understanding of the risks from inhalation exposure to HAPs from outdoor sources. It

helps U.S. EPA and states prioritize data and research needs to better assess risk in the

future and provides a baseline to help measure future trends in estimated health risks.

ASPEN model is based on the U.S. EPA's Industrial Source Complex Long Term model

(ISCLT), which simulates the behavior of the pollutants after they are emitted into the

atmosphere. ASPEN uses estimates of toxic air pollutant emissions and meteorological data from National Weather Service Stations to estimate air toxics concentrations nationwide (U.S. EPA, 2000b).

ASPEN model takes into account important determinants of pollutant concentrations, such as: rate of release, location of release, the height from which the pollutants are released, wind speeds and directions from the meteorological stations nearest to the release, breakdown of the pollutants in the atmosphere after being released (i.e., reactive decay), and settling of pollutants out of the atmosphere (i.e., deposition). However, ASPEN has several limitations mainly in a large scale and a three-dimensional arena and chemical reactions, since a Gaussian model is limited for this kind of analysis.

The NATA consists of 4 steps that produce nationwide estimates of: (1) the release of these pollutants into the air from various sources, (2) the concentration of these compounds in the air, (3) the exposure of populations to this air, and (4) the risk of both cancer and noncancer health effects resulting from this exposure (U.S. EPA, 2002a).

Rosenbaum et al., (1999) assessed the accuracy of the U.S. EPA's nationwide NATA estimates by comparing modeled HAPs concentrations to monitored concentrations. Its results indicated that when compared to monitoring data, model estimates underestimated HAPs concentrations to both carcinogenic and non-carcinogenic concentrations.

Payne-Sturges et al. (2004) examined the extent of exposure misclassification and its impact on risk for exposure estimated by the U.S. EPA's ASPEN model relative to monitoring results from a community-based exposure assessment conducted in Baltimore, Maryland. They found the ASPEN model estimates were generally lower than measured personal exposures and the estimated health risks. ASPEN's lower exposures resulted in proportional underestimation of cumulative cancer risk when pollutant exposures were combined to estimate cumulative risk.

Median cumulative lifetime cancer risk based on personal exposures was 3-fold greater than estimates based on ASPEN-modeled concentrations, demonstrating the significance of indoor exposure sources and the importance of indoor and/or personal monitoring for accurate assessment of risk. Results from their study concluded that environmental health policies may not be sufficient in reducing exposures and risks if these are based solely on modeled ambient VOC concentrations, which emphasize the need for a coordinated multimedia approach to exposure assessment for setting public health policy (Payne-Sturges et al., 2004).

In recognition of the limitations of ASPEN to estimate risk and the need to account for the time that people spend indoors and outdoors, the U.S. EPA recently developed the exposure module Hazardous Air Pollutant Exposure Model (HAPEM) (U.S. EPA, 2005f), and included it in the second phase of NATA. The HAPEM model was designed to predict the "apparent" inhalation exposure for specified population groups and air toxics (U.S., EPA, 2005f and 2002a). The HAPEM exposure model estimates inhalation

exposure for selected population groups to various air toxics. Through a series of

calculation routines, the model makes use of ambient air concentration predicted by

ASPEN, indoor/outdoor microenvironment concentration relationship data, population

data, and human activity pattern data to estimate an expected range of inhalation

exposure concentrations for groups of individuals. It also predicts nationwide census-

tract-level annual average human exposures and is to be used in a screening-level

inhalation risk assessment.

As the U.S. EPA continues to apply the ASPEN and HAPEM models to identify air

toxics of greatest public health concern, and assess progress in reducing exposures across

the United States, comparison of exposure measurements with modeling estimates

provides the basis for continued model development and refinement. A review of the

HAPEM data versus ASPEN estimate showed that, for a number of target VOCs, the

HAPEM estimates were lower than those from ASPEN (Payne-Sturges et al., 2004). The

authors concluded that risks based on HAPEM would be underestimated compared with a

measured personal exposure.

With all those limitations and including the second phase of the NATA for 1996

emissions data set, the highest ranking 20 percent of counties in terms of risk (622

counties) contained almost three-fourths of the U.S. population. Three air toxics,

chromium, benzene, and formaldehyde, appeared to pose the greatest nationwide

carcinogenic risk.

Exposure to DPM was widespread, and the U.S. EPA has concluded that DPM is a likely

human carcinogen and ranks with the other substances that the national-scale assessment

suggests pose the greatest relative risk, but the U.S. EPA has not defined an inhalation

cancer risk. In contrast, CalEPA has defined that DPM is a carcinogen (CalEPA, 1998).

One air toxic, acrolein, was estimated to pose the highest potential nationwide for

significant chronic adverse effects other than cancer (U.S. EPA, 2002a). This technical

assessment represented an important step toward characterizing air toxics nationwide. For

example, a nationwide assessment using NATA modeled HAPs estimated 56,000 excess

cancer cases or 800 cases annually, based on a 70-year life span (Woodruff et al., 2000).

According to the Clean Air Task Force in its modeling study over the 1999 National

Emissions Inventory Version 3 and based on the NATA's modeled concentrations, DPM

posed a cancer risk that was 7.5 times higher than the combined total cancer risk from all

other air toxics in the whole U.S. (Conrad et al., 2005). In addition, and based on

epidemiological evidence (Pope et al., 2002 and 2004a), this study concluded that fine

particle pollution from diesels shortens the lives of nearly 21,000 people each year. This

includes almost 3,000 early deaths from lung cancer. Finally, this study indicated that

tens of thousands of Americans suffer each year from asthma attacks (over 400,000),

heart attacks (27,000), and respiratory problems associated with fine particles from diesel

vehicles.

These illnesses result in thousands of emergency room visits, hospitalizations, and lost

workdays. This important report did not estimate secondary formation of PM that may

occur from gaseous diesel exhaust, such as sulfur or nitrogen compounds; instead, it used directly emitted DPM simulated by ASPEN (U.S. EPA, 2000b).

According to the U.S. EPA, NATA has been designed to help identify general patterns in air toxics exposure and risk across the country and is not recommended as a tool to characterize or compare risk at local levels using ASPEN as a dispersion model and HAPEM as an exposure model. Figures 2.24, 2.25, 2.26, and 2.27 show a pattern of the distribution of relative cancer and noncancer risk across the continental United States and Tennessee as estimated by NATA 1996 including all sources except diesel particulate matter (U.S. EPA, 2002a).

A number of statewide assessments have been conducted using monitored HAPs concentrations and/or NATAs modeling data (Tam and Neumann, 2004; Pratt et al., 2000; Morello-Frosch et al., 2000). Tam and Neumann (2004) used monitored data to assess 43 air toxics in Portland, OR. Seventeen HAPs exceeded a cancer risk level of 1 x 10^{-6} at all five monitoring sites. Nineteen HAPs exceeded this level at one or more site.

Carbon tetrachloride, 1,3-butadiene, formaldehyde, and 1,1,2,2-tetrachloroethane contributed more than 50% to the cumulative cancer risk. Mobile sources contributed the greatest HAPs emissions percentage (68%) (Tam and Neumann, 2004).
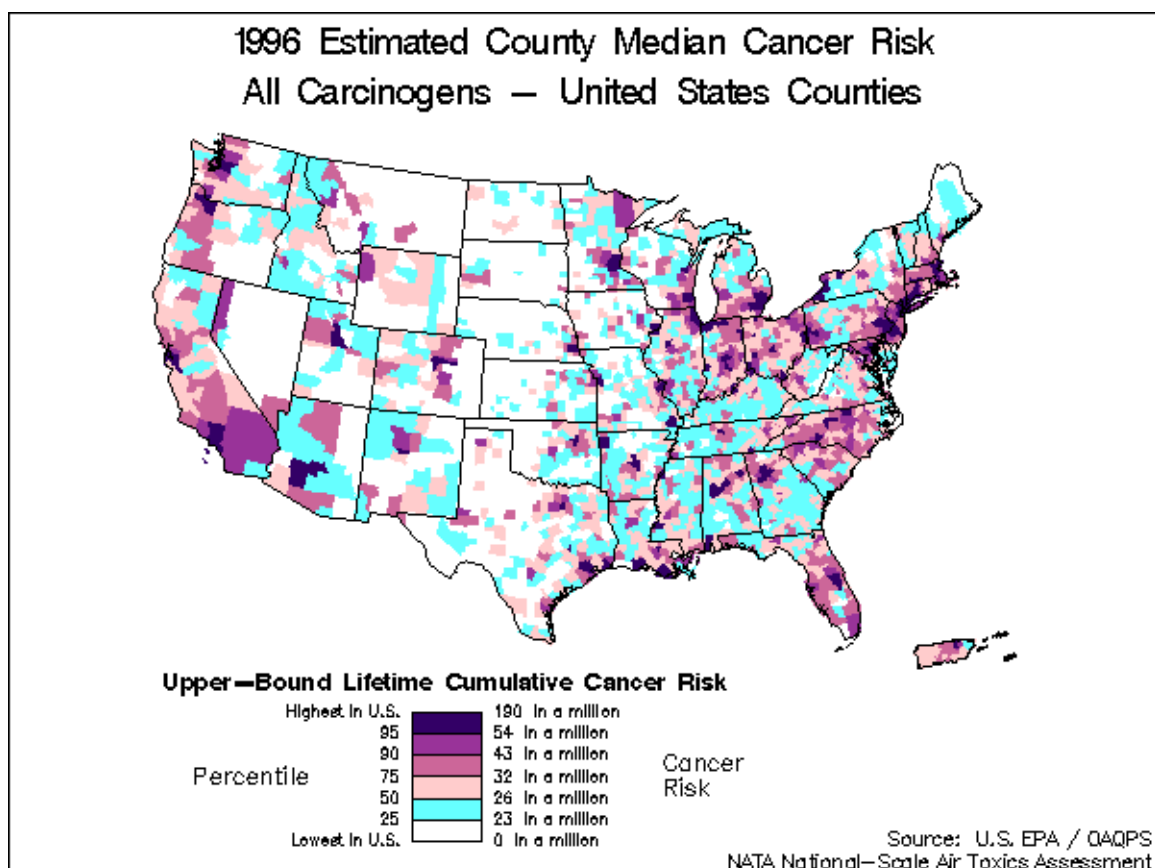
Figure 2.24. 1996 Estimated County Median Cancer Risk, All Carcinogens - United States Counties. (This MAP does not include DPM).
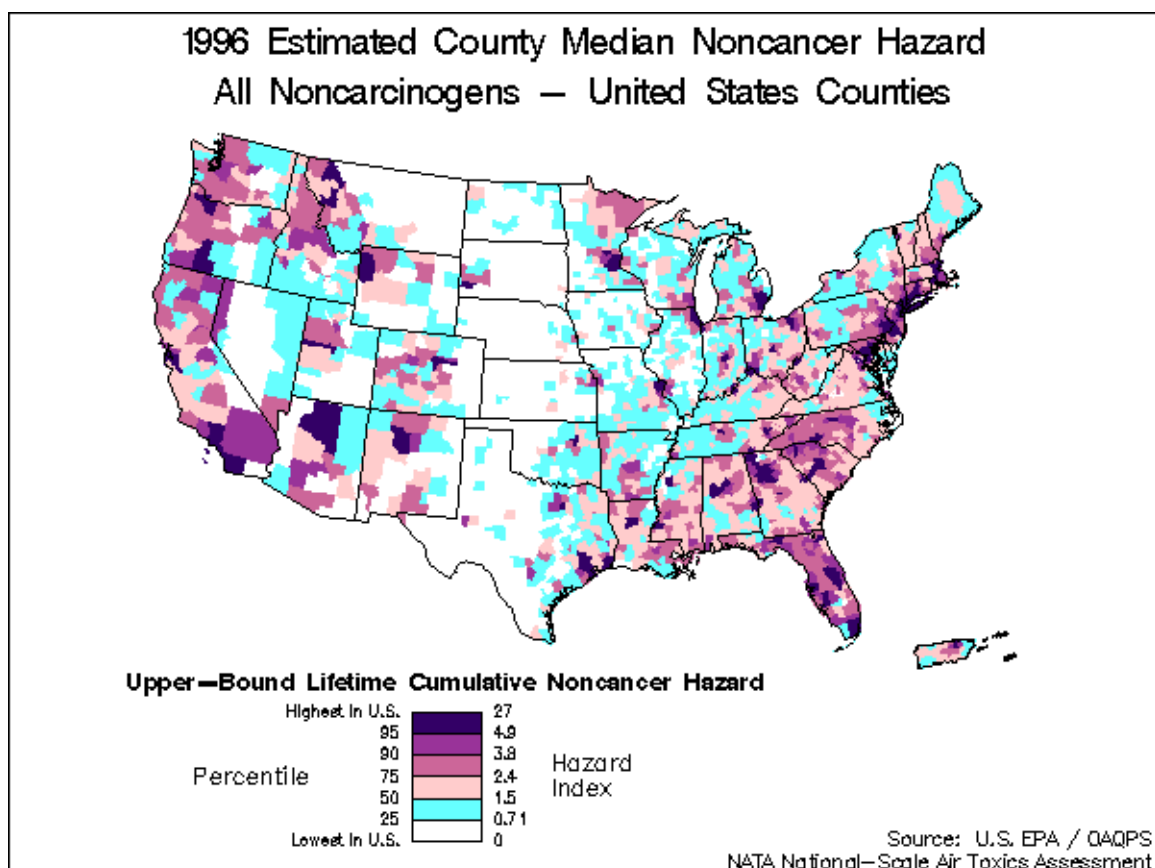
Figure 2.25. 1996 Estimated County Median Noncancer Risk, All Noncarcinogens - United States Counties. (This MAP does not include DPM).
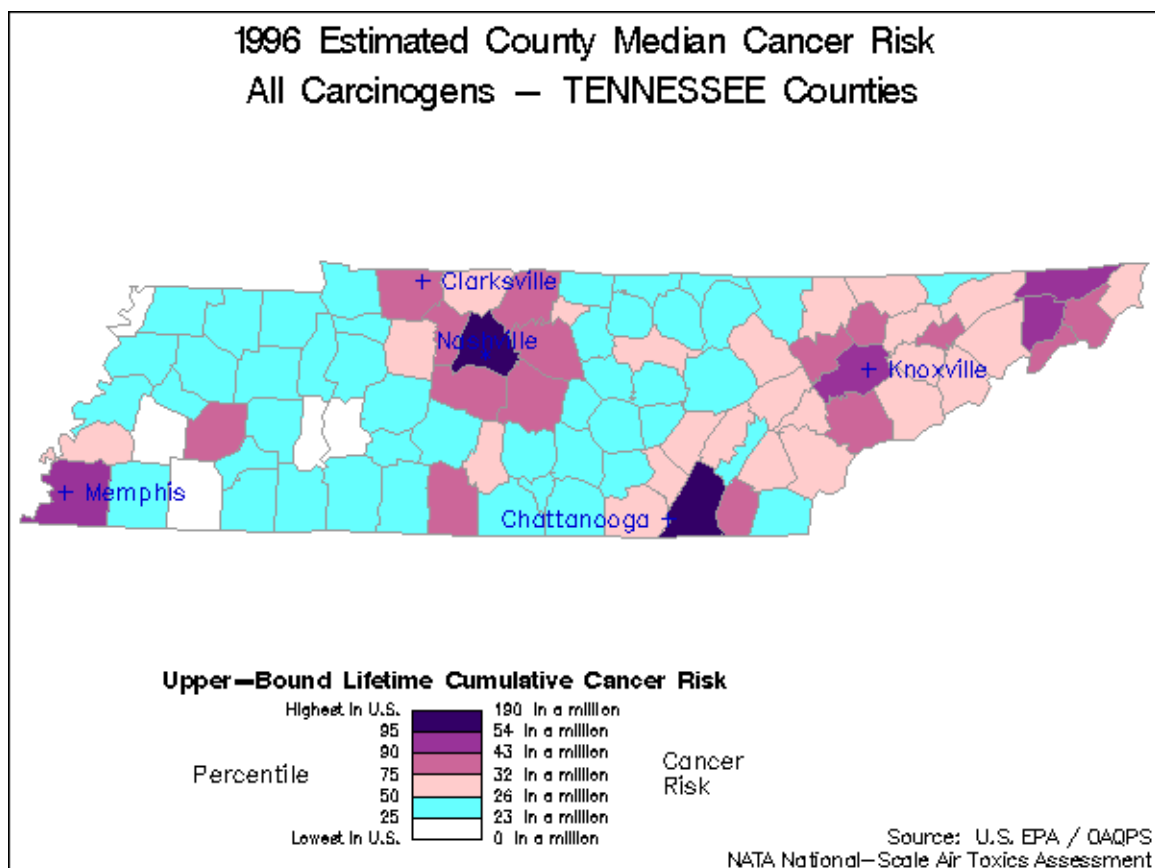
Figure 2.26. 1996 Estimated County Median Cancer Risk, All Carcinogens-Tennessee Counties. (This MAP does not include DPM).
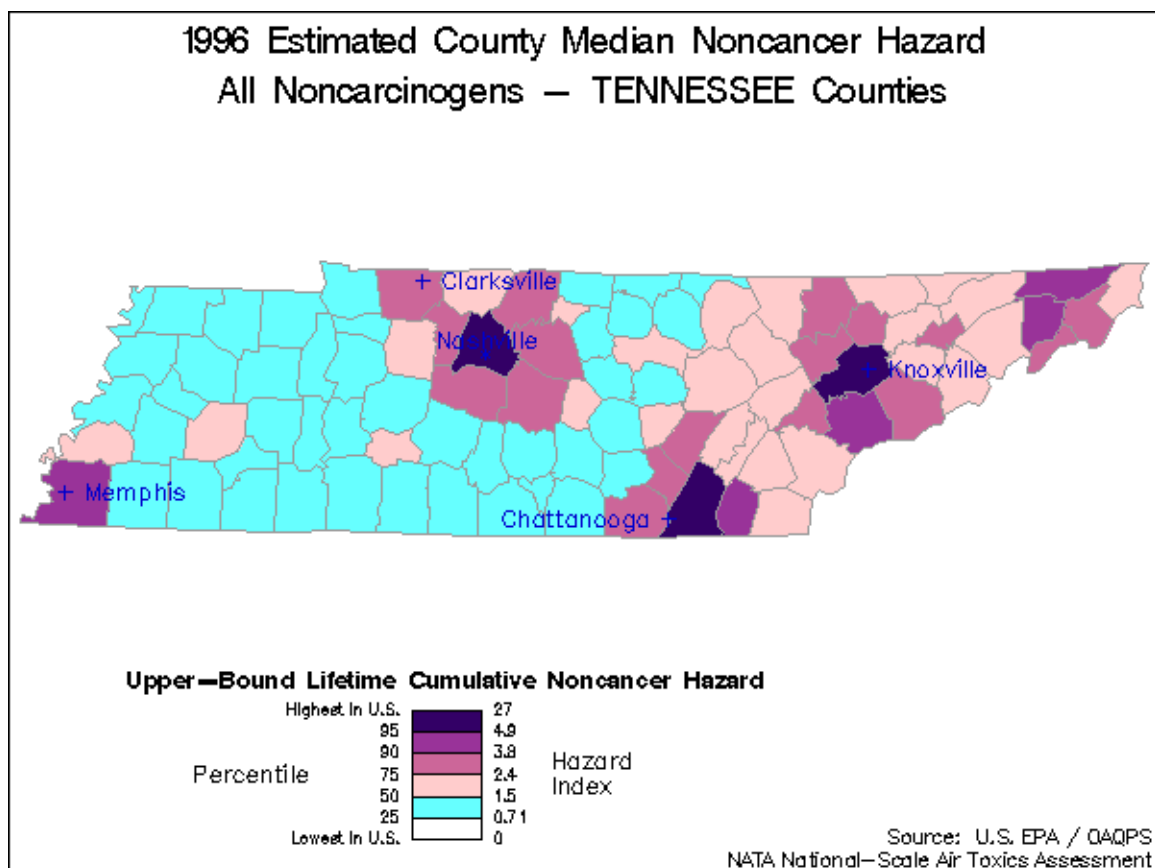
Figure 2.27. 1996 Estimated County Median Noncancer Risk, All Noncarcinogens - Tennessee Counties. (This MAP does not include DPM).

On the other hand, Pratt et al., (2000) used both HAPs modeled data from NATA and monitored concentrations to assess air toxics in Minnesota. Eleven pollutants exceeded health benchmark concentrations at one or more site by modeling, monitoring, or both. These HAPs included acrolein, arsenic, benzene, 1,3-butadiene, carbon tetrachloride, chromium, chloroform, ethylene dibromide, formaldehyde, nickel, and polycyclic organic matter (POM). Based on NATA data, POM, 1,3-butadiene, chromium, formaldehyde, carbon tetrachloride, and benzene contributed approximately 95% to the cumulative cancer risk (Pratt et al., 2000).

In California, NATA modeling data was used to screen for HAPs that exceeded health benchmark concentrations, and to identify emission sources (Morello-Frosch et al., 2000). Five HAPs exceeded a cancer risk level of 1 x 10^{-5} of all California census tracts. Chromium, 1, 3-butadiene, formaldehyde, POM, and benzene contributed more than 75% to the cumulative cancer risk. Acrolein and chromium contributed the greatest to the hazard indices. Mobile and area sources were estimated to account for the greater part of HAPs emissions (Morello-Frosch et al., 2000).

2.1.6   Air Toxics from Mobile Sources

MSATs come from four sources (U.S. EPA, 2001a). First, some air toxics are present in fuel and are emitted to the air when the fuel evaporates during refueling operation or passes through the engine unburned. Benzene, for example, is a component of gasoline. Cars emit small quantities of benzene in unburned fuel, or as vapor when gasoline

evaporates. Second, MSATs are formed through engine combustion processes. A significant amount of automotive benzene comes from the incomplete combustion of compounds in gasoline such as toluene and xylene that are chemically very similar to benzene. Like benzene itself, these compounds occur naturally in petroleum and become more concentrated when petroleum is refined to produce high-octane gasoline. Diesel exhaust emissions, as well as formaldehyde, acrolein, acetaldehyde, and 1, 3-butadiene, are also by-products of incomplete combustion. Third, some compounds, like acrolein, formaldehyde, and acetaldehyde, are also formed through a secondary process when other mobile source pollutants undergo chemical reactions in the atmosphere. Finally, metal air toxics result from engine wear or from impurities in oil or gasoline. These can also be present in fuel additives (U.S., EPA, 2001a and 2000a).

2.1.6.1   Regulations

In 1993, in compliance with Section 202(l)(1) of the Clean Air Act, the U.S. EPA released a study of motor vehicle-related air toxics (U.S. EPA, 1993). The study provided estimates of motor vehicle emissions of several pollutants believed to pose the greatest risk to public health, including benzene, formaldehyde, and 1,3-butadiene, as required by the Act, as well as acetaldehyde, DPM, gasoline particulate matter, and gasoline vapors. Exposure and risk were evaluated for four different years: 1990, 1995, 2000, and 2010. A total of three scenarios were modeled to explore the exposure and risk attributable to motor vehicle emissions: a baseline case reflecting motor vehicle related Clean Air Act

requirements, expanded use of reformulated gasoline, and expanded adoption of the

California Low Emissions Vehicles (LEV) standards. The study also explored air toxics

emissions from alternative fuel vehicles and non-road engines, but for the baseline cases

only and not for control scenarios (Table 2-3). It should be noted that this study did not

address whether to promulgate air toxics standards or suggest what those standards

should be (U.S. EPA, 1993). The U.S. EPA also developed a 1996 inventory estimates

for several gaseous MSATs (DPM, acetaldehyde, benzene, 1,3-butadiene, formaldehyde,

MTBE) as part of the 1999 study "Analysis of the Impacts of Control Programs on Motor

Vehicle Toxic Emissions and Exposure in Urban Areas and Nationwide" (U.S. EPA,

1999b and 1999c). The pollutants examined in the 1999 study were chosen because U.S.

EPA had adequate data to perform a rigorous modeling analysis for those pollutants.

**Table 2-3. Annual Expected Cancer Deaths due to Exposure to Selected Mobile
Source Air Toxics in the U.S.**

Annual Expected Cancer Deaths (U.S. Total)				
Pollutant	1990	1995	2000	2010
Acetaldehyde	5.3	3.6	2.8	3.0
Benzene	70	43	35	35
1,3-Butadiene	304	209	176	204
Formaldehyde	44	28	21	22
Diesel PM	1923	1165	688	476
Total	2,346.3	1,448.6	922.8	740

The 1999 study examined the impact of a variety of parameters including fuel properties, emission control technologies, and type of in-use operation on the 1990 and 1996 emissions inventories for six pollutants: acetaldehyde, benzene, 1,3-butadiene, DPM, formaldehyde, and MTBE. Those selected MSATs shown in Table 2-4 decremented their emissions, except MTBE due to the MTBE prohibition in the gasoline.

The 1996 NEI also contained emissions estimates for several other MSATs, and included data for non-road as well as on-road sources (Table 2-5). Between the 1999 U.S. EPA's Motor Vehicle Air Toxics Study and the 1996 NEI, U.S. EPA had baseline inventory data for all of the 21 MSATs except naphthalene. For DPM + DEOG, U.S. EPA did not have inventory data on the DEOG portion. For this analysis, U.S. EPA used DPM as a surrogate for DPM + DEOG (U.S. EPA, 1999b and 1999c).

Table 2-4. Annual Emissions from On-Highway Vehicles for Selected Air Pollutants [Short tons per year]

Compound	1990 Emissions	1996 Emissions (a)
1,3-Butadiene	36,000	24,000
Acetaldehyde	41,000	31,000
Benzene	257,000	171,000
Formaldehyde	139,000	93,000
DPM	235,000	182,000
MTBE	55,000	67,000

(a)   The 1996 estimates are based on updated inventories taking into consideration the proposed 2007 and later model year heavy-duty engine standards.

Table 2-5. On-Road and Non-Road Emission Inventories of Some MSATs from the 1996 NEI in [Short tons]

Compound	On-road		Non-Road		Mobile Sources	
	Tons	%	Tons	%	Tons	%
1,3-Butadiene (a)	23,500	42	9,900	18	33,400	60
Acetaldehyde (a)	28,700	29	40,800	41	69,500	70
Acrolein (a)	5,000	16	7,400	23	12,400	39
Arsenic Compounds (a)	0.25	0.06	2.01	0.51	2.26	0.57
Benzene (a)	168,200	48	98,700	28	266,900	76
Chromium Compounds (a)	14	1.2	35	3	49	4.2
Dioxins/Furans (a, b)	0.0001	0.2	N.A.	N.A.	0.0001	0.2
Ethylbenzene	80,800	47	62,200	37	143,000	84
Formaldehyde (a)	83,000	24	86,400	25	169,400	49
Lead Compounds (a)	19	0.8	546	21.8	565	22.6
Manganese Compounds (a)	5.8	0.2	35.5	1.3	41.3	1.5
Mercury Compounds (a)	0.2	0.1	6.6	4.1	6.8	4.2
MTBE	65,100	47	53,900	39	119,000	86
n-Hexane	63,300	26	43,600	18	106,600	44
Naphthalene	N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
Nickel Compounds (a)	10.7	0.9	92.8	7.6	103.5	8.5
POM (as sum of 7 PAH) (a)	42	4	19.3	2	61.3	6
Styrene	16,300	33	3,500	7	19,800	40
Toluene	549,900	51	252,200	23	802,100	74
Xylene	311,000	43	258,400	36	569,400	79

(a)   These compounds are also on the list of urban HAPs for the Integrated Urban Air Toxics Strategy.

(b)   Mass given in tons of TEQ (toxic equivalency quotient). The EPA Office of Research and Development (ORD) has recently developed an inventory for dioxin and dioxin-like compounds using different methods than those used in the 1996 NTI. For 1995, the EPA-ORD estimate of on-highway emissions of dioxin compounds is 0.00005 tons TEQ, comprising 1.5 percent of the national inventory in that year. (The TEQ rates the toxicity of each dioxin and furan relative to that of 2,3,7,8-TCDD, which is assigned a TEQ of 1.0.)

The 1996 inventory data reflected certain interesting characteristics of mobile source air toxics emissions. First, mobile sources accounted for the majority of the national inventory of three of the gaseous MSATs that are included on the urban HAP list. These three were 1, 3-butadiene (60 %), acetaldehyde (70 %), and benzene (76 %). Mobile sources accounted for 39 % of the national inventory of acrolein, and 49 % of the national inventory of formaldehyde.

All of these MSATs were formed as part of the combustion process except for benzene, which is also released through evaporative emissions from gasoline (U.S. EPA, 2001a). Second, with regard to the other MSATs that are included on the UATs list, the mobile source contribution generally was small (arsenic compounds, chromium compounds, manganese compounds, mercury compounds, nickel compounds, POM, and dioxins/furans). The sole exception was lead compounds. Mobile sources contributed 23 % to national inventories of lead emissions, due primarily to non-road sources and, more specifically, to the use of a lead additive package used to boost the octane of aviation gasoline. The mobile source contribution to the other metals on the UATs list came primarily from engine wear, some fuel additives, or impurities in engine oil (U.S. EPA, 1999b and 1999c).

With regard to the gaseous MSATs that are not included on the UATs list (ethylbenzene, MTBE, n-hexane, toluene, and xylene), mobile source contributions were high because of the presence of these compounds in gasoline (U.S. EPA, 1999b and 1999c). In addition, mobile sources accounted for almost all DPM emissions.

A limited number of stationary sources, such as large generators, did operate on diesel fuel. Because there were relatively few stationary sources that operated on diesel fuel, U.S. EPA believes that DPM from stationary sources is relatively small compared to DPM from mobile sources (U.S. EPA, 1999b and 1999c).

From 1990 to 2020, the U.S. EPA's study projected these programs will reduce the levels of on-highway emissions of benzene by 73 %, formaldehyde by 76 %, 1, 3-butadiene by 72 %, and acetaldehyde by 67 %. In addition, by 2020, on-highway DPM emission reductions of 94 % from 1990 levels were projected for heavy-duty engines. This action also finalized new gasoline toxic emissions baseline requirements, which require refiners to maintain current levels of over-compliance with toxic emissions performance standards that apply to reformulated gasoline (RFG) and anti-dumping standards that apply to conventional gasoline, (CG).

The new baseline requirements were designed to ensure that existing over compliance with current standards continues. The U.S. EPA was not setting additional vehicle-based air toxics controls at that time because the technology-forcing Tier 2 light-duty vehicle standards and those standards being developed in response to its proposal for heavy-duty engine and vehicle standards represented the greatest degree of toxics control achievable at that time considering existing standards, the availability and cost of the technology, energy, safety factors, and lead time (U.S.EPA, 2001a)

Federal and state governments are promoting the use of alternative fuel vehicles and new engine technologies as a means to improve local air pollution. So far, the impact of this initiative toward alternative fuels with respect to MSATs has been studied for several pollutants using emission analysis (Winebrake et al., 2001; Sapkota et al., 2003). To alternative fuel, Winebrake found that almost all of the fuels studied reduce 1,3-butadiene emissions compared with conventional gasoline; however the use of ethanol in E85 (fuel made with 85% ethanol) or reformulated gasoline leads to increased acetaldehyde emissions, and the use of methanol, ethanol, and compressed gas natural may result in increased formaldehyde emissions, finally, when the modeling resulted for the risk factors, all the fuels and vehicle technologies showed air toxic emission reduction benefits (Winebrake et al., 2001).

Because of the U.S. EPA's continuing concern about the potential health impacts of public exposure to air toxics, it was appropriate to establish additional mobile source controls their fuels that are specifically designed to reduce further or minimize increases in national inventories of these pollutants. By 2010, the U.S. EPA's existing programs will reduce MSATs by over one million tons from 1996 levels. In addition to controlling pollutants such as hydrocarbons, particulate matter, and nitrogen oxides, the U.S. EPA's recent regulations controlling emissions from highway vehicles and non-road equipment also result in large air toxic reductions. Reformulated gasoline and anti-dumping standards (U.S. EPA, 2005h), along with anti-backsliding provisions of the 2001 MSATs rule (U.S. EPA, 2001a), the clean diesel trucks and buses rule and (U.S., EPA, 2001b), and the clean non-road diesel rule (U.S. EPA, 2004b) also result in large reductions.

Today's action also describes a technical analysis plan through which U.S. EPA will continue to improve its understanding of the risk posed by air toxics to public health and welfare.

In 1999, the U.S. EPA created the Integrated Urban Air Toxics Strategy (IUATS) (U.S. EPA, 1999a). The overarching goal of the IUATS is to reduce cancer and non-cancer risks associated with all sources of air toxics in urban areas, principally mobile sources. In urban areas, toxic air pollutants raise special concerns because sources of emissions and people are concentrated in the same geographic areas, leading to large numbers of people being exposed to the emissions of many air toxics from many sources.

The goals of the strategy reflected both the statutory requirements stated in section 112(k) of the Act and the goals of the U.S. EPA's overall air toxics program. These goals consist of the following:

- ✓ Attain a 75 percent reduction from 1990 incidence of cancer attributable to exposure to HAPs emitted by stationary sources. This is relevant to all HAPs from both major and area stationary sources, in all urban areas nationwide. Reductions can be the result of actions by Federal, State, local and/or Tribal governments, achieved by any regulations or voluntary actions.
- ✓ Attain a substantial reduction from 1990 levels in public health risks posed by HAP emissions from area sources. This includes health effects other than cancer posed by all HAPs (e.g., birth defects and reproductive effects). Reductions can be the result of

actions by Federal, State, local and/or Tribal governments, achieved by any

regulations or voluntary actions.

✓ Address disproportionate impacts of air toxics hazards across urban areas. This will

necessarily involve consideration of both stationary and mobile source emissions of

all HAPs, as well as sources of HAPs in indoors air. The U.S. EPA intends to

characterize exposure and risk distributions both geographically and

demographically. This will include particular emphasis on highly exposed individuals

and specific population subgroups (e.g., children, the elderly, and low-income

communities).

Summarizing, so far the existing and newly promulgated mobile source control programs

are the reformulated gasoline (RFG) program, the national low emissions vehicle

(NLEV) standards, the Tier 2 motor vehicle emissions standards and gasoline sulfur

control requirements, the 2007 heavy-duty engine, the vehicle standards and on-highway

diesel fuel sulfur control requirements, and the clean non-road diesel standards (U.S.

EPA, 2001a, 2001b, and 2004b).

2.1.6.2   Priority Mobile Sources Air Toxics

According to the U.S. EPA's IUATS and the MSATs regulations, the following 6 air

toxics can be considered as priority mobile sources air toxics (PMSATs) due to their

higher risk: acrolein, acetaldehyde, benzene, 1,3-butadiene, formaldehyde, and DPM

(U.S. EPA, 2001a). For those HAPs Table 2-6 shows the carcinogenicity classification

schemes and rankings, Table 2-7 shows their main diseases for cancer and non-cancer

effects, and Table 2-8 shows the carcinogenicity and chronic effects of PMSATs (U.S.

EPA, 2001a). The effects on human health and the photochemical mechanism of those

PMSATs are described as follow:

Table 2-6. Carcinogenicity Classification Schemes and Rankings for the PMSAT's

Classification Scheme	Acetaldehyde	Benzene	Acrolein	1,3-Butadiene	DPM	Formaldehyde
USEPA	$B2^3$	A^1	C^4	A	Not available	$B1^2$
NIEHS	Reasonably Anticipated	Known	Not available	Known	Reasonably Anticipated	Reasonably Anticipated
IARC	$2B^7$	1^5	Not available	$2A^6$	2A	2A
NIOSH	Potential	Potential	Not available	Potential	Not available	Potential

(1): Human carcinogen

(2): Probable human carcinogen. Limited human data

(3): Probable human carcinogen. Limited anaimal data

(4): Possible human carcinogen.

(5): Human carcinogen

(6): Probably carcinogenic to humans.

(7): Possible human carcinogens

Table 2-7. PMSATs Health Effects

Compound	Cancer Effects	Non-cancer Effects
Acetaldehyde	Nasal and nasopharyngeal tumors	Upper respiratory tract irritation, dysplasia and squamous metaplasia of respiratory and olfactory epithelia; papillomas in the forestomach of rats, skin irritation at high doses.
Acrolein	No effects	Asthma, COPD, respiratory tract irritation
Benzene	Acute myeloid leukemia	Hematological effects, effects on red blood cells, white blood cells, platelets, bone marrow damage leading to a plastic anemia
1,3-Butadiene	Lymphohaematopoietic system (leukemia, lymph sarcoma, and reticular call sarcoma)	Cardiovascular, hematopoietic reproductive and developmental effects, respiratory diseases such as asthma and COPD.
DPM	Lung Cancer	CVD, asthma, and COPD.
Formaldehyde	Nasal and nasopharyngeal tumors	Upper respiratory tract irritation, dysplasia and squamous metaplasia of respiratory and olfactory epithelia; papillomas in the forestomach of rats, skin irritation at high doses.

Table 2-8. Carcinogenicity & Chronic Effects of PMSATs

MSATs	IURs $[m^3/ug] \times 10^{-6}$	RfC $[ug/m^3]$	Reference
Acetaldehyde	2.2	9.00	IRIS
Acrolein	Not applicable	0.02	IRIS, NATA
Benzene	7.8	30.00	IRIS
1,3-Butadiene	30.0	2.00	IRIS
Diesel PM	300.0	5.00	CalEPA/IRIS
Formaldehyde	13.0	4.00	IRIS, ATSDR

A. Acrolein

According to the Agency for Toxic Substances and Disease Registry (ATSDR), acrolein is primarily used as an intermediate in the manufacture of acrylic acid. It can be formed from the photo-oxidation of certain organic pollutants in outdoor air or from burning wood, tobacco, gasoline, or diesel (ATSDR, 1999). It is extremely toxic to humans from inhalation and dermal exposure. Acute (short-term) inhalation exposure may result in upper respiratory tract irritation and congestion; even exposure to high levels (10 parts per million [ppm]) of acrolein in humans may result in death. Effects on the lung, such as upper respiratory tract irritation and congestion have been noted at acrolein levels ranging from 0.17 ppm to 0.43 ppm (U.S. EPA, 2005i). The major effects from chronic (long-term) inhalation exposure to acrolein in humans consist of general respiratory congestion and eye, nose, and throat irritation. The Reference Concentration (RfC) for acrolein is 0.00002 mg/m^3 based on squamous metaplasia and neutrophilic infiltration of nasal epithelium in rats (U.S. EPA, 2005i). No information is available on its reproductive, developmental, or carcinogenic effects in humans. The animal cancer data are limited, with one study reporting an increased incidence of adrenocortical tumors in rats exposed to acrolein in the drinking water. U.S. EPA considers acrolein a possible human carcinogen (Group C), however, has not estimated an inhalation cancer risk factor (ATSDR, 1999; U.S. Department of Health and Human Services, 2005; U.S. EPA, 2005i) Acrolein is unlikely to be transported over long distances because of its high reactivity and estimated short half-lives in air and water. This HAPs is rapidly metabolized by

organisms and does not bioaccumulate. Its chemical formula, Chemical Abstracts Service (CAS), and molecular weight are C_3H_4O, 107-02-8, and 56.06 g/mol respectively (ATSDR, 1999). Acrolein belongs to the α,β-Unsatured Carbonyls compounds, which are known to react with ozone, with oxygen radical, and with OH radical. Photolysis and NO_3 radical reaction are of minor importance (Seinfeld and Pandis, 1998). Under atmospheric conditions the O_3 reactions are also of minor significance, leaving the OH radical reaction as the major loss process, principally during summer season. Summarizing, the CB-IV photochemical acrolein reactions are:

Acrolein decomposition

OH radical reaction

ACROLEIN + OH = 0.709*FORM + 0.709*ALD2 + 0.709*XO_2 + 0.709*HO_2 - 0.709*PAR + 0.814*C_2O_3

Ozone reaction

ACROLEIN + O_3 = 10.326*ALD2 + 15.282*FORM + 6.815*CO + 9.087*HO_2 + 4.543*XO_2 + 2.065*OH + 4.130*FACD + 4.130*AACD - 20.651*PAR

NO_3 radical reaction

ACROLEIN + NO_3 = 1.192*XO_2 + 0.118*XO_2N + 1.310*FORM + 1.310*ALD2 - 1.310*PAR + 1.310*NO_2 + 0.851*C_2O_3 + 0.851*HNO_3
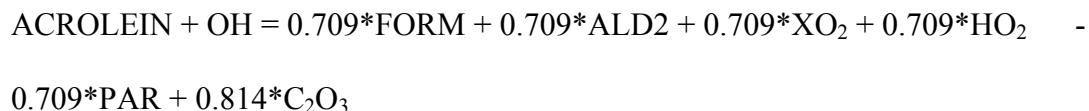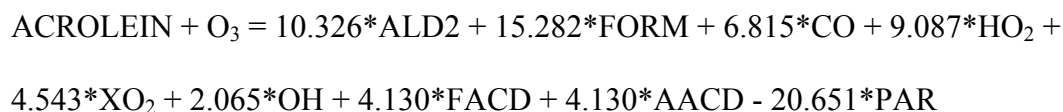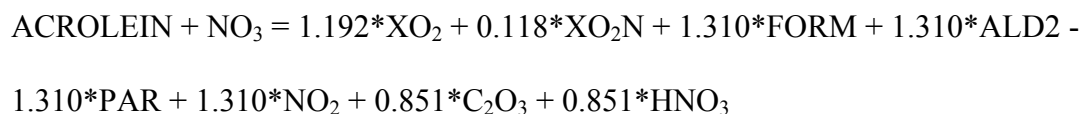
Oxygen atom reaction

ACROLEIN + O = 0.538*ALD2 + 0.325*HO_2 + 0.239*XO_2 + 0.257*CO +

0.171*FORM + 0.017*XO_2N + 0.188*PAR + 0.356*OH + 0.185*C_2O_3

Acrolein photolysis

ACROLEIN = 6.3*XO_2 + 12.6*HO_2 +6.3*CO+ 6.3*FORM
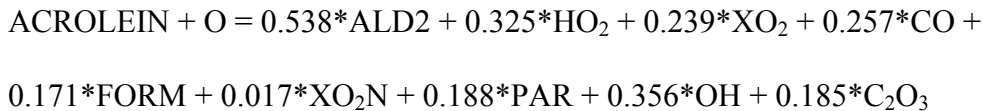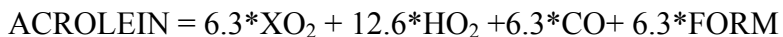
Secondary acrolein formation

1, 3-BUTADIENE + OH = 0.85*FORM + 0.85*ACROLEIN + 0.85*XO_2+ 0.85*HO_2 -

0.85*PAR

1, 3-BUTADIENE + O_3 = 0.50*ACROLEIN + 2.81*FORM +1.26*CO + 1.67*HO_2 +

0.84*XO_2 + 0.38*OH +0.76*FACD + 0.76*AACD -3.8*PAR

1, 3-BUTADIENE + O = 0.230*ACROLEIN + 0.155*HO_2 + 0.115*XO_2 + 0.123*CO +

0.082*FORM + 0.008*XO_2N +0.090*PAR + 0.082*OH
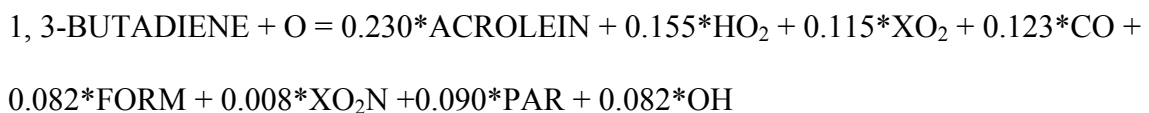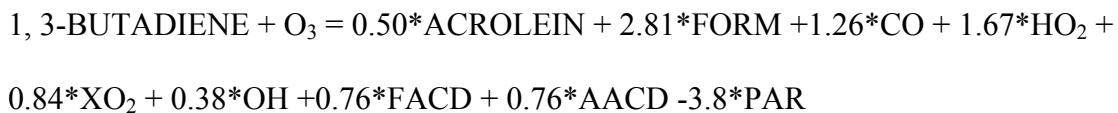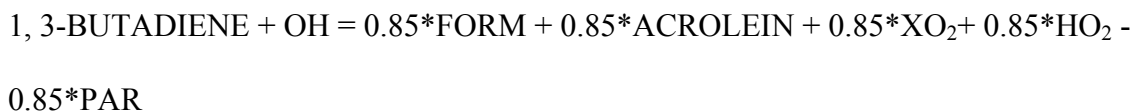
The species description is illustrated in Table 2-9.

Table 2-9. Chemical Species in the CB-IV Mechanism for Air Toxics

Representation	Species Name
AACD	Acetic acid
ACROLEIN	Acrolein
ALD2	High molecular weight aldehydes
1,3-BUTADIENE	1, 3-Butadiene
C_2O_3	Peroxyacyl radical ($CH_3C(O)OO\bullet$)
CO	Carbon monoxide
CRO	Methylphenoxy radical
ETH	Ethene ($CH_2=CH_2$)
FACD	Formic acid
FORM	Formaldehyde ($CH_2=O$)
HNO_3	Nitric acid
HO_2	Hydroperoxy radical
ISOP	Isoprene
MGLY	Methylglyoxal ($CH_3C(O)C(O)H$)
NO	Nitric oxide
NO_2	Nitrogen dioxide
NO_3	Nitrogen trioxide (nitrate radical)
O	Oxygen atom (triplet)
O_3	Ozone
OH	Hydroxyl radical
OLE	Olefinic carbon bond
OPEN	High molecular weight aromatic oxidation ring fragment
PACD	Peroxy acetic acid
PAN	Peroxyacyl nitrate ($CH_3C(O)OONO_2$)
PAR	Paraffin carbon bond (C-C)
ROR	Secondary organic oxy radical
XO_2	NO-to-NO operation
XO_2N	NO-to-nitrate operation

B. Acetaldehyde

Acetaldehyde is ubiquitous in the ambient environment. It is mainly used as an intermediate in the synthesis of other chemicals, as well as it is an intermediate product of higher plant respiration and formed as a product of incomplete wood combustion in fireplaces and woodstoves, coffee roasting, burning of tobacco, vehicle exhaust gases, coal refining, and waste processing. Hence, many individuals are exposed to acetaldehyde by breathing ambient air, principally due to the secondary formation during summer season (Seinfeld and Pandis, 1998). Acute exposure to acetaldehyde results in effects including irritation of the eyes, respiratory tract, and skin. At higher exposure levels, erythema, coughing, pulmonary edema, and necrosis may also occur (U.S. EPA, 1987). Symptoms of chronic intoxication of acetaldehyde are similar to those of alcoholism, whose Reference Concentration is 0.009 mg/m^3 based on degeneration of olfactory epithelium in rats (U.S. Department of Health and Human Services, 2005; U.S. EPA, 2005j). Acetaldehyde is considered a probable human carcinogen (Group B2) based on inadequate human cancer studies and animal studies that have shown nasal tumors in rats and laryngeal tumors in hamsters. The U.S. EPA calculated an inhalation unit risk of 2.2 x 10^{-6} m^3/μg. (U.S. Department of Health and Human Services, 2005; U.S. EPA, 2005j).

The acetaldehyde lifetime due to reaction with OH radical, NO3 radical, and *hv* are 11 h, 17 days, and 5 days respectively (Seinfeld and Pandis, 1998). Its chemical formula, CAS

code, and molecular weight are CH$_3$CHO, 75-07-0, and 44.06 g/mol respectively (U.S. Department of Health and Human Services, 2005). Acetaldehyde belongs to the aldehyde compounds, which are known to decompose by photolysis and react with oxygen atom and with OH radical. The NO$_3$ radical reaction is of minor importance (Seinfeld and Pandis, 1998). Under atmospheric conditions the oxygen reaction is also of minor significance, leaving the photolysis and OH radical reaction as the major loss process, principally during summer season. Summarizing, the CB-IV photochemical acetaldehyde reactions are:

Acetaldehyde (ALD2) decomposition

Oxygen radical reaction

$$ALD2 + O = C_2O_3 + OH$$

OH radical reaction

$$ALD2 + OH = C_2O_3$$

NO$_3$ radical reaction

$$ALD2 + NO_3 = C_2O_3 + HNO_3$$
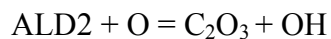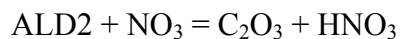
Acetaldehyde photolysis

$$ALD2 = XO_2 + 2*HO_2 + CO + FORM$$

Secondary acetaldehyde formation (ALD2)

PAR + OH= 0.87*XO$_2$ +0.13*XO$_2$N +0.11*HO$_2$ + 0.11* ALD2+ 0.76*ROR - 0.11*PAR

ROR= 1.1* ALD2 + 0.96*XO$_2$ + 0.94*HO$_2$ - 2.10*PAR + 0.04*XO$_2$N + 0.02*ROR

OLE + O= 0.63*ALD2+ 0.38*HO$_2$ + 0.28*XO$_2$+ 0.3*CO+ 0.2*FORM+ 0.02*XO$_2$N+
0.22*PAR + 0.2*OH

OLE + OH= FORM+ ALD2+ XO$_2$+ HO$_2$– PAR

OLE + O$_3$= 0.5*ALD2+ 0.74*FORM+ 0.33*CO+ 0.44*HO$_2$+ 0.22*XO$_2$+ 0.1*OH+
0.20*FACD+ 0.20*AACD– PAR

OLE + NO$_3$ = 0.91*XO$_2$ + 0.09*XO$_2$N + FORM + ALD2 - PAR + NO$_2$

ETH + OH= XO$_2$ + 1.56*FORM + HO$_2$ + 0.22*ALD2

OPEN + O$_3$ = 0.03*ALD2+ 0.62*C$_2$O$_3$ + 0.7*FORM+ 0.03*XO$_2$ + 0.69*CO+ 0.08*OH
+ 0.76*HO$_2$ + 0.2*MGLY

ISOP + O$_3$ = 0.65*ISPD+ 0.60*FORM+ 0.20*XO$_2$+ 0.066*HO$_2$+ 0.266*OH+
0.20*C$_2$O$_3$+ 0.15*ALD2+ 0.35*PAR + 0.066*CO

ISOP+ NO$_3$ = 0.20*ISPD+ 0.80*NTR+ 1.0*XO$_2$+ 0.80*HO$_2$+ 0.20*NO$_2$+ 0.80*ALD2+ 2.4*PAR

ISPD + OH = 1.565*PAR+ 0.167*FORM+ 0.713*XO$_2$+ 0.503*HO$_2$+ 0.334*CO + 0.168*MGLY + 0.273*ALD2 + 0.498*C$_2$O$_3$

ISPD + O$_3$ = 0.114*C$_2$O$_3$+ 0.150*FORM+ 0.850*MGLY+ 0.154*HO$_2$+ 0.268*OH+ 0.064*XO$_2$+ 0.020*ALD2+ 0.360*PAR+ 0.225*CO

ISPD + NO$_3$ = 0.357*ALD2 + 0.282*FORM+ 1.282*PAR+ 0.925*HO$_2$+ 0.643*CO+ 0.850*NTR+ 0.075*C$_2$O$_3$+ 0.075*XO$_2$ + 0.075*HNO$_3$

ISPD = 0.333*CO+ 0.067*ALD2+ 0.900*FORM+ 0.832*PAR+ 1.033*HO$_2$+ 0.700*XO$_2$+ 0.967*C$_2$O$_3$

ISOP + NO2 = 0.20*ISPD+ 0.80*NTR+ 1.00*XO$_2$+ 0.80*HO$_2$+ 0.20*NO+ 0.80*ALD2 + 2.4*PAR

ACROLEIN + OH = 0.709*FORM+ 0.709*ALD2+ 0.709*XO$_2$+ 0.709*HO$_2$- 0.709*PAR+ 0.814*C$_2$O$_3$

ACROLEIN + O$_3$ = 10.326*ALD2+ 15.282*FORM+ 6.815*CO+ 9.087*HO$_2$+ 4.543*XO$_2$+ 2.065*OH+ 4.130*FACD+ 4.130*AACD- 20.651*PAR

ACROLEIN + NO$_3$ = 1.192*XO$_2$+ 0.118*XO$_2$N+ 1.310*FORM+ 1.310*ALD2 -

1.310*PAR+ 1.310*NO$_2$+ 0.851*C$_2$O$_3$+ 0.851*HNO$_3$

ACROLEIN + O = 0.538*ALD2 + 0.325*HO$_2$ + 0.239*XO$_2$ + 0.257*CO +

0.171*FORM + 0.017*XO$_2$N + 0.188*PAR + 0.356*OH + 0.185*C$_2$O$_3$

1,3-BUTADIENE + NO3 = 0.140*XO$_2$+ 0.014*XO$_2$N+ 0.154*FORM+ 0.154*ALD2 -

0.154*PAR+ 0.154*NO$_2$
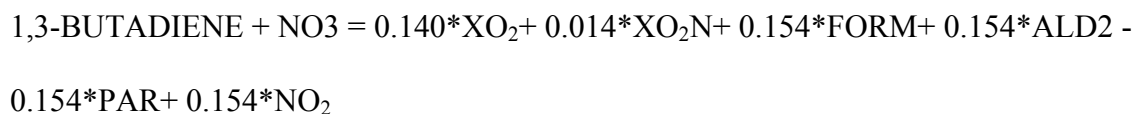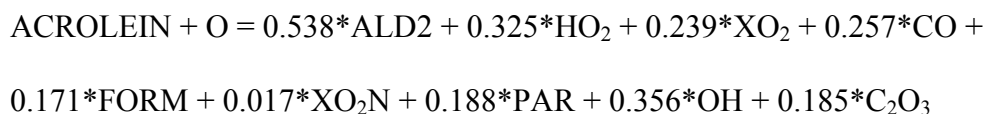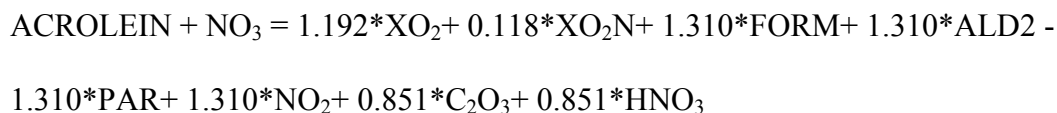
The species description is illustrated in Table 2-9.

C. Benzene

Benzene is found in the air from emissions of burning coal and oil, gasoline service

stations, and on-road sources. Acute inhalation exposure of humans to benzene may

cause drowsiness, dizziness, headaches, as well as eye, skin, and respiratory tract

irritation, and, at high levels, unconsciousness (ATSDR, 1997). Chronic inhalation

exposure has caused various disorders in the blood, including reduced numbers of red

blood cells and anemia (ATSDR, 1997). Reproductive effects have been reported for

women exposed by inhalation to high levels, and adverse effects on the developing fetus

have been observed in animal tests. The U.S. EPA has not established a Reference

Concentration, however, the California Environmental Protection Agency (CalEPA) has

established a chronic reference exposure level of 0.06 mg/m^3 for benzene based on hematological effects in humans (CalEPA, 1999). Increased incidence of leukemia has been observed in humans exposed to benzene (ATSDR, 1997). U.S. EPA has classified benzene as a Group A, and calculated a range inhalation cancer risk of 2.2 x 10^{-6} to 7.8 x 10^{-6} as the increase in the lifetime risk of an individual who is continuously exposed to 1 µg/m^3 of benzene in the air over their lifetime (U.S. EPA, 2005k).

Benzene chemical formula, CAS code, and molecular weight are C_6H_6, 71-43-2, and 78.11 g/mol (U.S. Department of Health and Human Services, 2005). The atmospheric sink for benzene is the reaction with OH radical, whose lifetime is 12 days as shown in the following reaction (Seinfeld and Pandis, 1998).

(C(C=CC2C(COO2)O)O[O.])

(O=CCC(C=CC=O)O)
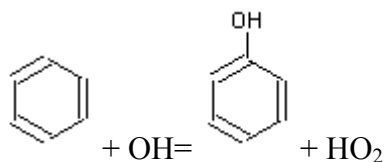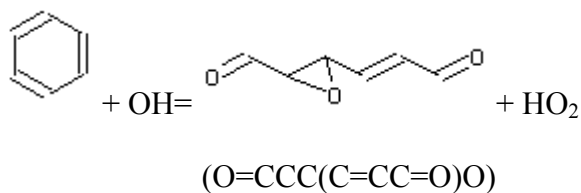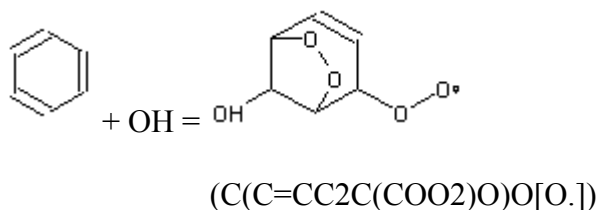
D. 1,3-Butadiene

Mobile sources are the main 1, 3-butadiene emissions. Although 1, 3-butadiene breaks down quickly in the atmosphere, it is usually found in ambient air at low levels in urban and suburban areas (Seinfeld and Pandis, 1998). Acute exposure to 1 ,3-butadiene by inhalation in humans results in irritation of the eyes, nasal passages, throat, and lungs. Neurological effects, such as blurred vision, fatigue, headache, and vertigo, have also been reported at very high exposure levels (ATSDR, 1995; U.S. Department of Health and Human Services, 2005). Epidemiological studies have reported a possible association between 1, 3-butadiene exposure and cardiovascular diseases, such as rheumatic and arteriosclerotic heart diseases, while other human studies have reported effects on the blood (ATSDR, 1995). The U.S.EPA is currently developing a Reference Concentration for 1,3-butadiene, while CalEPA has established a chronic reference level of 0.008 mg/m^3 for 1,3-butadiene based on reproductive effects in mice (CalEPA, 1999) Epidemiological studies of workers in rubber plants have shown an association between 1,3-butadiene exposure and increased incidence of leukemia (Delzell et al, 1996; Macaluso et al., 1996). U.S. EPA has classified 1,3-butadiene as a Group B2, and calculated inhalation cancer risk of 3×10^{-5} m^3/μg (U.S. EPA, 2005g). 1, 3-Butadiene chemical formula, CAS code, and molecular weight are C_4H_6, 106-99-0, and 54.09 g/mol (U.S. Department of Health and Human Services, 2005). The atmospheric sinks for 1, 3-butadiene are the reaction with OH radical, oxygen atom, O_3, and NO_3. NO_3 radical reaction is of minor importance, and under atmospheric conditions the O_3 reactions is

also of minor significance, leaving the OH radical and O atom reactions as the major loss process, principally during summer season. Summarizing, the CB-IV photochemical 1, 3-Butadiene reactions are:

1, 3-Butadiene decomposition

OH radical reaction
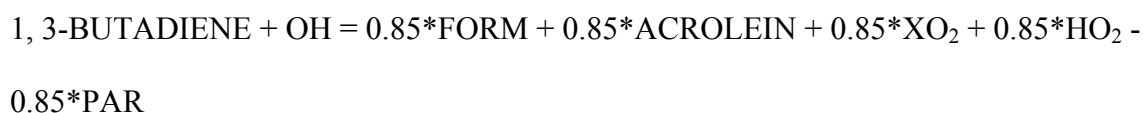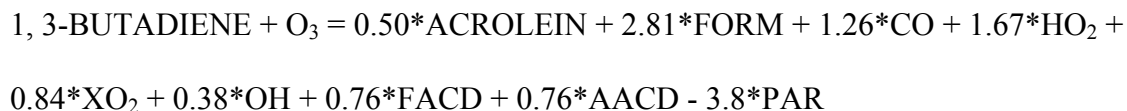
1, 3-BUTADIENE + OH = 0.85*FORM + 0.85*ACROLEIN + 0.85*XO_2 + 0.85*HO_2 - 0.85*PAR

Ozone reaction

1, 3-BUTADIENE + O_3 = 0.50*ACROLEIN + 2.81*FORM + 1.26*CO + 1.67*HO_2 + 0.84*XO_2 + 0.38*OH + 0.76*FACD + 0.76*AACD - 3.8*PAR

NO_3 radical reaction

1, 3-BUTADIENE + NO_3 = 0.140*XO_2 + 0.014*XO_2N + 0.154*FORM + 0.154*ALD2 - 0.154*PAR + 0.154*NO_2

Oxygen atom reaction

1, 3-BUTADIENE + O = 0.230*ACROLEIN + 0.155*HO_2 + 0.115*XO_2 + 0.123*CO+ 0.082*FORM + 0.008*XO_2N + 0.090*PAR + 0.082*OH
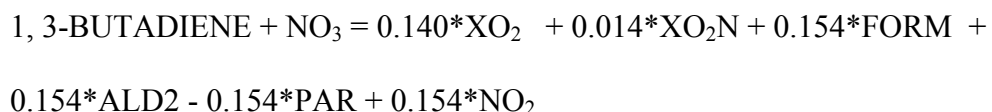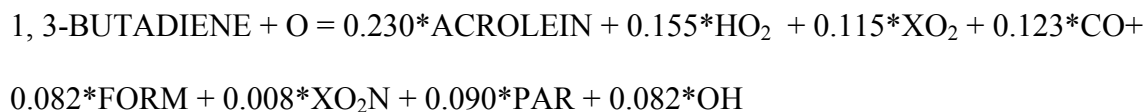
The species description is illustrated in Table 2-9.

E. Formaldehyde

Formaldehyde is used mainly to produce resins and as an intermediate in the synthesis of other chemicals. Mobile sources are the main primary formaldehyde emissions (Seinfeld and Pandis, 1998). Acute inhalation exposure to formaldehyde in humans can result in respiratory symptoms, and eye, nose, and throat irritation. Other effects seen from exposure to high levels of formaldehyde in humans are coughing, wheezing, chest pains, and bronchitis (U.S. EPA, 1988; WHO, 1989). Chronic exposure to formaldehyde by inhalation in humans has been associated with respiratory symptoms and eye, nose, throat irritation, asthma (Delfino, 2002; Leikauf, 2002; U.S. EPA, 1988; WHO, 1989). The U.S. EPA has not established a Reference Concentration for formaldehyde (U.S. EPA, 2005l) Limited human studies have reported an association between formaldehyde exposure and lung and nasopharyngeal cancer. Animal inhalation studies have reported an increased incidence of nasal squamous cell cancer (U.S. EPA, 2005l). The U.S. EPA considers formaldehyde a probable human carcinogen (Group B1) and calculated an inhalation unit risk estimate of 1.3×10^{-5} m^3/μg (U.S. EPA, 2005l).

The formaldehyde lifetime due to reaction with OH radical, NO$_3$ radical, and *hv* are 1.5 days, 80 days, and 4 h respectively (Seinfeld and Pandis, 1998). Its chemical formula, CAS code, and molecular weight are CH2O, 50-00-0, and 30.03 g/mol respectively (U.S. Department of Health and Human Services, 2005). Formaldehyde is known to be decomposed rapidly by photolysis and react with OH radical and with oxygen radical.

NO_3 radical reaction is of minor importance (Seinfeld and Pandis, 1998). Under atmospheric conditions the oxygen reaction is also of minor significance, leaving the photolysis and OH radical reaction as the major loss process, principally during summer season. Summarizing, the CB-IV photochemical acetaldehyde reactions are:

Formaldehyde (FORM) decomposition

OH radical reaction

$FORM + OH = HO_2 + CO$

Formaldehyde photolysis

$FORM = 2*HO_2 + CO$

$FORM = CO$

Oxygen atom reaction

$FORM + O = OH + HO_2 + CO$

NO_3 radical reaction

$FORM + NO_3 = HNO_3 + HO_2 + CO$

Secondary formaldehyde formation (FORM)

$ALD2 = XO_2 + 2*HO_2 + CO + FORM$

$C_2O_3 + NO = NO_2 + XO_2 + FORM + HO_2$

$C_2O_3 + C_2O_3 = 2*XO_2 + 2*FORM + 2*HO_2$

$C_2O_3 + HO_2 = 0.79*FORM + 0.79*XO_2 + 0.79*HO_2 + 0.79*OH + 0.21*PACD$

$CH4 + OH = XO_2 + FORM + HO_2$

$OLE + O = 0.63*ALD2 + 0.38*HO_2 + 0.28*XO_2 + 0.3*CO + 0.2*FORM + 0.02*XO_2N + 0.22*PAR + 0.2*OH$

$OLE + OH = FORM + ALD2 + XO_2 + HO_2 - PAR$

$OLE + O_3 = 0.5*ALD2 + 0.74*FORM + 0.33*CO + 0.44*HO_2 + 0.22*XO_2 + 0.1*OH + 0.20*FACD + 0.20*AACD - PAR$

$OLE + NO_3 = 0.91*XO_2 + 0.09*XO_2N + FORM + ALD2 - PAR + NO_2$

$ETH + O = FORM + 0.7*XO_2 + CO + 1.7*HO_2 + 0.3*OH$

$ETH + OH = XO_2 + 1.56*FORM + HO_2 + 0.22*ALD2$

$ETH + O_3 = FORM + 0.42*CO + 0.12*HO_2 + 0.40*FACD$

OPEN + OH= XO_2 + 2*CO + 2*HO_2 + C_2O_3+ FORM

OPEN + O_3 = 0.03*ALD2 + 0.62* C_2O_3 + 0.7*FORM + 0.03*XO_2 +0.69*CO +

0.08*OH + 0.76*HO_2 + 0.2*MGLY

ISOP + O = 0.75*ISPD + 0.50*FORM + 0.25*XO_2 + 0.25*HO_2 + 0.25* C_2O_3+

0.25*PAR

ISOP + OH = 0.912*ISPD + 0.629*FORM + 0.991*XO_2+ 0.912*HO_2 + 0.088*XO_2N

ISOP + O_3 = 0.65*ISPD+ 0.60*FORM+ 0.20*XO_2+ 0.066*HO_2+ 0.266*OH+ 0.20*

C_2O_3 + 0.15*ALD2 + 0.35*PAR+ 0.066*CO

ISPD + OH= 1.565*PAR+ 0.167*FORM+ 0.713*XO_2+ 0.503*HO_2 + 0.334*CO+

0.168*MGLY + 0.273*ALD2+ 0.498* C_2O_3

ISPD + O_3 = 0.114* C_2O_3 + 0.150*FORM + 0.850*MGLY + 0.154*HO_2+ 0.268*OH+

0.064*XO_2 + 0.020*ALD2+ 0.360*PAR+ 0.225*CO

ISPD + NO_3 = 0.357*ALD2 + 0.282*FORM + 1.282*PAR+ 0.925*HO_2 + 0.643*CO+

0.850*NTR + 0.075* C_2O_3 + 0.075*XO_2+ 0.075*HNO_3

ISPD= 0.333*CO + 0.067*ALD2 + 0.900*FORM + 0.832*PAR + 1.033*HO_2+

0.700*XO_2 + 0.967* C_2O_3

ACROLEIN+ OH = 0.709*FORM + 0.709*ALD2 + 0.709*XO_2 + 0.709*HO_2-

0.709*PAR + 0.814* C_2O_3

ACROLEIN + O_3 = 10.326*ALD2 + 15.282*FORM + 6.815*CO+ 9.087*HO_2 +

4.543*XO_2+ 2.065*OH + 4.130*FACD+ 4.130*AACD - 20.651*PAR

ACROLEIN + NO_3 = 1.192*XO_2 + 0.118*XO_2N+ 1.310*FORM + 1.310*ALD2 -

1.310*PAR+ 1.310*NO_2+ 0.851* C_2O_3+ 0.851*HNO_3

ACROLEIN + O = 0.538*ALD2 + 0.325*HO_2 + 0.239*XO_2+ 0.257*CO+ 0.171*FORM

+ 0.017*XO_2N+ 0.188*PAR+ 0.356*OH+ 0.185* C_2O_3

ACROLEIN = 6.3*XO_2 + 12.6*HO_2 + 6.3*CO + 6.3*FORM

1, 3-BUTADIENE+ OH = 0.85*FORM + 0.85*ACROLEIN + 0.85*XO_2+ 0.85*HO_2-

0.85*PAR

1, 3-BUTADIENE+ O_3 = 0.50*ACROLEIN+ 2.81*FORM+ 1.26*CO+ 1.67*HO_2

+0.84*XO_2+ 0.38*OH+ 0.76*FACD+ 0.76*AACD - 3.8*PAR

1, 3-BUTADIENE+NO$_3$ = 0.140*XO$_2$+ 0.014*XO$_2$N + 0.154*FORM + 0.154*ALD2 - 0.154*PAR+ 0.154*NO$_2$

1, 3-BUTADIENE+ O = 0.230*ACROLEIN + 0.155*HO$_2$ + 0.115*XO$_2$+ 0.123*CO+ 0.082*FORM+ 0.008*XO$_2$N + 0.090*PAR+ 0.082*OH
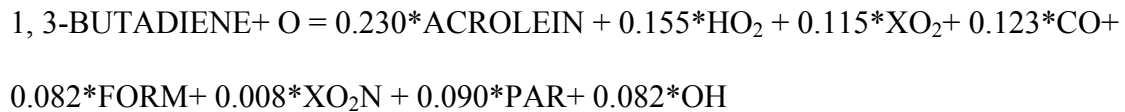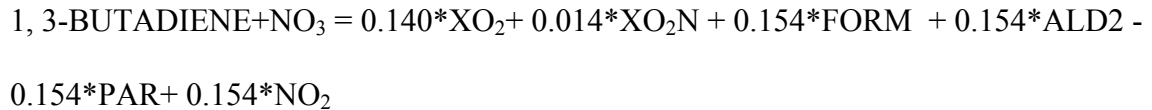
The species description is illustrated in Table 2-9.

F. Diesel Particulate Matter

Diesel particulate matter (DPM) is currently a topic of great concern from both pollution and public health standpoints. The U.S. EPA finalized strict new regulations on diesel particle emissions, and a number of other countries are considering regulatory action as well. Although specific output depends on operating conditions, the largest single component of DPM emissions is carbonaceous soot produced by the incomplete combustion of diesel fuel. A great research effort is currently being devoted to reducing the amount of DPM emissions. These efforts include reducing the diesel fuel sulfur content, making the diesel engine combustion process more efficient, as well as removing particles from the exhaust stream, such as particle traps and catalytic converters on particle properties (Burtscher, 2005; U.S. EPA, 2001b and 2004b).

DPM is part of a complex mixture. The sizes of diesel particulates, which are of greatest health concern, are in the categories of fine, ultra fine, and nano particles (Biswas and

Wu, 2005; Lloyd and Cackette 2001; Lighty et al., 2000; Kittelson, 1998), as shown in Figure 2.28. The mixture of these fine, ultra fine, and nano particles is composed mainly of elemental carbon (EC) or black carbon with adsorbed compounds (generally described as the soluble organic fraction, SOF), such as organic carbon (OC), sulfate, nitrate, metals, and other trace elements (Kleeman et al., 2000; Kittelson, 1998), as illustrated in Figure 2.29. The elemental fraction stems from fuel droplet pyrolysis, while the organic fraction originates from unburned fuel, lubricating oil, and combustion byproducts (Shah et al., 2004).
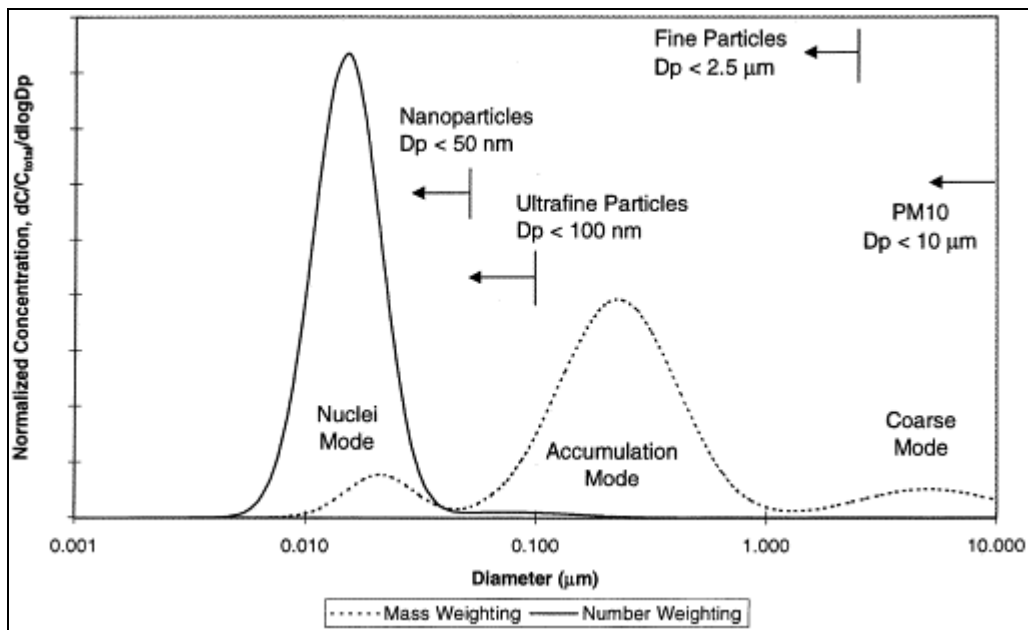
Figure 2.28. Typical Engine Exhaust Size Distribution: Both in Mass and in Number. (Reprinted from Kittelson et al., 1998)

Particles consist mainly of highly agglomerated solid carbonaceous material and ash and volatile organic and sulfur compounds.
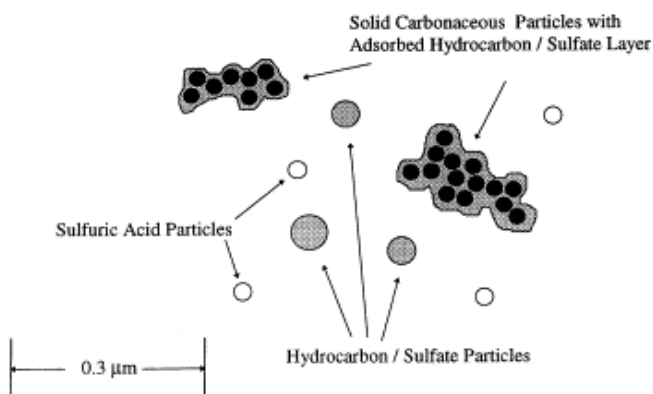
Figure 2.29. Typical Composition and Structure of Engine Exhaust Particles (Reprinted from Kittelson et al., 1998)

Many carcinogenic and mutagenic compounds have been measured in the SOF of DPM, such as polycyclic aromatic hydrocarbons (PAHs) and nitroarenes, as well as irritants or inflammatory agents such as acrolein and other toxins that cause a range of diverse health effects (Jasco Inc, 2004; Kittelson, 1998; Rosenkranz, 1996). A diesel particle initially consists of an agglomeration of EC spheres coated with organic and inorganic compounds that are adsorbed or absorbed at the surface of this agglomerate, as illustrated in Figure 2.29 (Vouitsis et al., 2005; Kim et al., 2002a, 2002b; Kittelson, 1998). Diesel particles lose their identity rapidly as they coagulate with other particles and act as condensation sites for secondary aerosol species (Ning et al., 2004). It may be noted that for some time, the diesel exhaust nano-particles are formed in part due to the continuation of in-stack coagulation and adsorption, along with the condensation of significant quantities of organic and inorganic compounds present in diesel exhaust (Biswas and Wu, 2005).

DPM composition is variable, which typically has a composition of 25-60% of EC (Seigneur et al., 2003; Moosmuller et al., 2001; Schauer et al., 1999; Kittelson, 1998) and 20-50% of OC (Shi et al., 2000). Sulfate and nitrate may account for concentrations lower than 4%, depending on the sulfur diesel content and vehicles type (Shi et al., 2000). Figure 2.30 shows a typical particle composition for a heavy-duty diesel engine tested in a heavy-duty transient cycle published by Kittelson et al., (1998). This composition and emissions quantity is strongly dependent of the diesel fuel sulfur content, because the lower sulfur concentration is in the fuel, the lower will be the particulate matter emissions from the diesel engines (Saiyasitpanich et al., 2005; Liang et al., 2005; U.S. EPA, 2001b and 2004b).
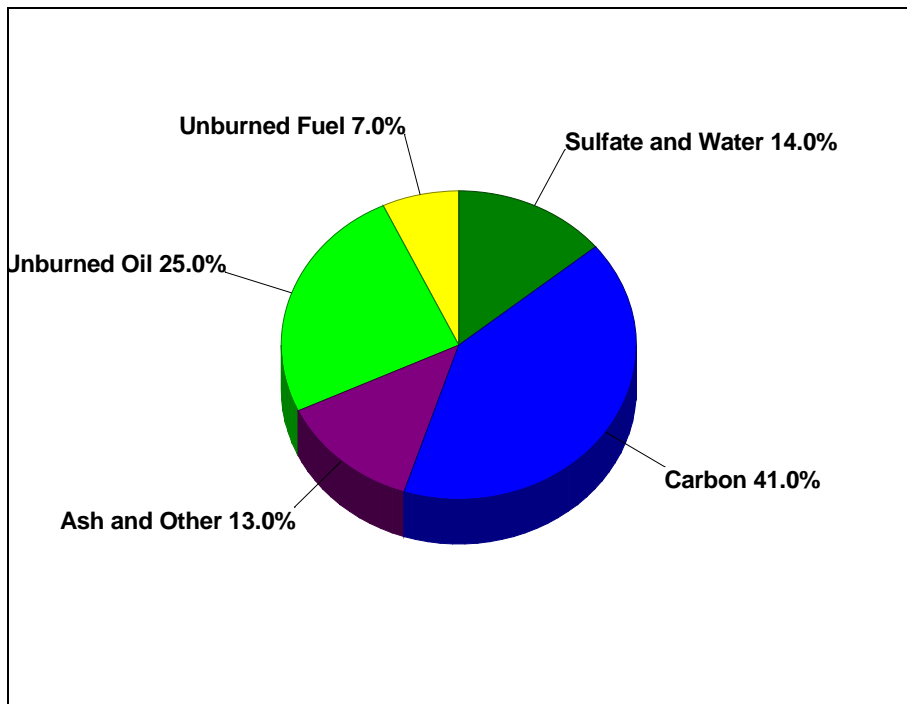
Figure 2.30. Typical Particle Composition for a Heavy-Duty Diesel Engine Tested in a Heavy-Duty Transient Cycle (Reprinted from Kittelson et al., 1998)

For this reason, in January 2001 and in June 2004, the U.S. EPA finalized the Clean Diesel Trucks and Buses Rule and the Clean Non-road Diesel Rule, respectively, with more stringent standards for new diesel engines and fuels. The rules require the use of sulfur content as low as 15 ppm beginning in 2006 for highway diesel fuel, and 2007 for non-road diesel fuel. The diesel fuel sulfur content was in the range of 500 ppm in 1999 (U.S. EPA, 2001b). These fuels will enable the use of after treatment technologies for new diesel engines, which can reduce harmful emissions by 90 percent or more. After treatment technologies will start phasing into the diesel sector beginning in 2007 for highway and 2011 for non-road (U.S. EPA, 2001b and 2004b).

The diesel sulfur content is oxidized in the engine combustion chamber to give sulfur dioxide vapors, and as the exhaust gases cool in the ambient condenses to form H_2SO_4 droplets or nuclei particles (Liang et al., 2005; Kim et al., 2002a and 2002b; Kittelson, 1998). This sulfuric acid provides the condensation surface to the DPM formation (Kim et al., 2002b). However, if lower sulfur content is on the diesel fuel, nano-particle number concentrations may increase due to the lower availability of condensation surfaces (Biswas and Wu, 2005).

The smallest DPM can deeply penetrate in the lungs and enter the blood stream, carrying the toxins in the rest of the body (Nemmar et al., 2002; Donaldson et al., 2001; U.S. EPA, 2002c) where they can affect the respiratory and cardiovascular systems, and other organs, causing a high number of premature deaths (Delfino et al., 2005; Zanobetti and Schwartz, 2005; Sioutas et al., 2005; Schultz et al., 2005; Riedl et al., 2005; Pope et al.,

2004a; Adonis et al., 2003a and 2003b; Pandya et al., 2002; Lloyd and Cackette, 2001; Castranova et al., 2001). The fine particles aggravate cardiovascular diseases increasing heart and brain attacks, since they invade the blood stream and start an inflammatory response, interrupting the heart beats and increasing the sanguineous coagulation (Riedl et al., 2005; Delfino et al., 2005; Peters, 2001 and 2002; Lee et al., 2002; Donaldson et al., 2001; Ghio and Devlin, 2001; Nemmar et al., 2002). On the other hand, a consistent and evident relationship between the exposure to the DPM and lung cancer has been published in more than 32 epidemiologic studies in humans (Krewski et al., 2005; CalEPA, 1998; Lipsett and Campleman, 1999; Steenland et al., 1998). In 1989, the International Agency for Research on Cancer (IARC) concluded that DPM is a probable human carcinogen, classifying it in the group 2A. In 1990, California through its Environmental Protection Agency (CalEPA) identified the DPM as a chemical that causes cancer (CalEPA, 2005) and defined an inhalation cancer risk factor of 3×10^{-4} [m^3/ug]. The U.S. EPA has not defined an inhalation cancer risk yet because data are not sufficient to develop a numerical estimate of carcinogenic potency for this pollutant. However, the U.S. EPA has concluded that diesel exhaust ranks with the other substances that the national-scale assessment suggests pose the greatest relative risk. In addition to the potential for lung cancer risk, the U.S. EPA indicates that there is a significant potential for non-cancer health effects as well, based on the contribution of diesel particulate matter to ambient levels of fine particles (U.S. EPA, 2000a and 2002c).

High morbidity and mortality levels of respiratory diseases have been reported in communities with high DPM concentrations (Krewski et al., 2005; Conrad et al., 2005;

Fruin et al., 2004; Adonis et al., 2003a). Adverse effects also are observed when breathing airborne particles in controlled acute human exposure studies, including cough, respiratory symptoms of asthma, and reduced lung function, especially over susceptible groups of the population, such as children and the elderly (Tainio et al., 2005; Pope et al., 2004b; Schwartz, 2004). The DPM also affects the environment, decreasing visibility mainly in urban areas (Lloyd and Cackette, 2001), while EC or black carbon is considered one of the more important green house pollutants after carbon dioxide (Figure 2.31), which is causing as much as a quarter of all observed global warming by reducing the ability of snow and ice to reflect sunlight (Conrad et al., 2005; Hansen and Sato, 2001; Jacobson, 2001).
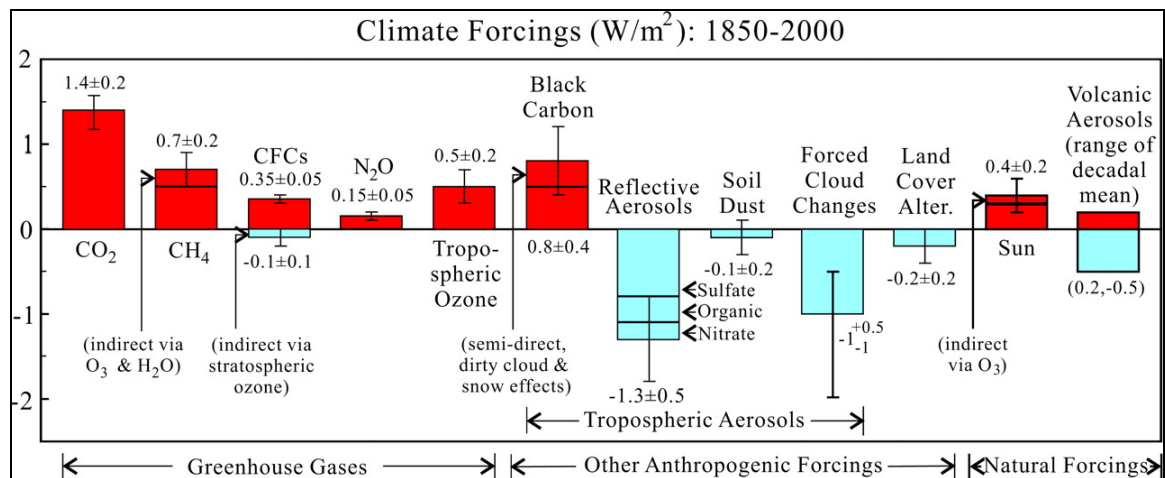
Figure 2.31. Radiative Forcing since 1850 due to Changes in Green House Gases, Aerosols, Land Use, Solar Activity, and Volcanoes (Reprinted from Hansen and Sato, 2001)

The cancer risk estimate by Conrad et al., (2005), as explained above, could be higher in European urban areas and developing countries, because those countries have higher light duty diesel vehicle (LDDV) and diesel buses percentages than the U.S. (Adonis et al., 2003b; Walker, 2004). In fact, diesel engines are being more popular each day in the LDDV sector, principally in Europe and some South American countries, where almost 30% of the market is diesel-vehicles; this proportion will increase until 50% by 2010 (Walker, 2004), while in the U.S. the LDDV market will increase only 7% (Greene et al., 2004).

Because DPM is the major source of EC in the atmosphere (Schauer, 2003) and so far there is no analytical method to measure ambient DPM (Lloyd and Cackette, 2001), this has led to the use of EC as a marker for assessing human exposure to diesel exhaust, for determining the contribution of diesel engines to ambient particulate concentrations, and as a surrogate for DPM (Shah et al., 2004; Tamura et al., 2003; Birch et al., 1996; Schauer, 2003). The ability to use EC accurately as a tracer for DPM in either the environmental or the occupational setting critically relies on a clear understanding of relative contributions of other sources to EC concentrations; however, it approximation generates important uncertainty, because those studies used an average EC contribution to come up the DPM concentration in any place between 50 and 80% (Seigneur et al., 2003; MATES II, 2000). In addition, EC is not a unique tracer for ambient DPM, and efforts to utilize EC as an indicator of DPM must properly address other sources of EC as well as utilize a consistent measurement technique for EC when comparing source and ambient EC measurements to avoid significant biases. Those sources include gasoline

vehicles, heating wood combustion, restaurant kitchens, agriculture biomass burning, and forest wildfires (Lloyd and Cackette, 2001). In order to better manage air quality, it is important to know the sources or source categories that contribute to the concentrations of DPM at a particular area or receptor. Receptor models have been widely used to characterize the sources that contribute to specific pollutants, such as PM2.5 and PM10 (Zheng et al., 2002; Kavouras et al., 2001; Koutrakis et al., 2005). However, they do not fully take into account the chemical reactions involved in the formation of secondary fine particles nor distinguish among a heavy diesel engine vehicle, a light diesel engine vehicle, or a diesel non-road engine (U.S. National Research Council, 1999).

In a CMAQ DPM modeling between August 27th and September 9th, 1999, one of the worst ozone episodes that occurred in the Southeast U.S. between 1997 and 2000, Diaz estimated that in general the main EC contribution came from goods transportation; however, for some particulate urban areas, the main contribution came from construction engines and marine vessels, as was the case in Memphis, Tennessee, which is located close to the Mississippi river (Diaz et al., 2005). In addition, in this study it was estimated that Altanta, GA, presented diesel EC emissions and concentrations 40 and 4 times higher than a rural area, respectively. As well as, the sources that use diesel contributed in average with a 74% of EC emissions and a 70% in ambient EC concentrations on the analyzed geographic area. These contributions were close to the values obtained by Zheng et al., 2002, who employed a molecular marker chemical mass balance model to apportion the sources of atmospheric particulate matter in eight cities in the Southeastern U.S. for one-month of each season between the spring of 1999 and the winter of 2000.

The calculated value, for January, April, July, and October were 74, 84, 92, and 85%

respectively. His results demonstrated the seasonal impact of wood smoke on EC

concentrations. The differences between the results obtained by Diaz and Zheng could be

due to Zheng not considering the photochemical reaction's effect on the SOA formation,

while the Diaz's results could be affected by a sub estimation of the emission inventory

provided for the U.S. EPA (U.S. EPA, 2005a).

2.2   AIR TOXICS MODELING APPROACHES

2.2.1 Advanced Air Quality Models

The advanced air quality models (Zolghadri et al., 2004; Russell et al., 2000; Byun and

Ching, 1999) combine and systematize the information and the knowledge of emissions,

meteorology, and the atmospheric photochemical mechanism to estimate ambient

concentrations of several pollutants, even over complex terrains. Those models can be

used to simulate and explain old episode of pollution, to assess the potential effects of

different strategies of emissions reductions, or to forecast the air quality. The more

common air quality models are: the Models-3/ Community Multiscale Air Quality

(CMAQ) model (Byun and Ching, 1999) and the Comprehensive Air quality Model

(CAMx) model (ENVIRON, 2005). The Models-3/CMAQ model is a third generation

model able to simulate the tropospheric ozone, acid deposition, particulate matter, air

toxics, visibility, and other contaminants over one atmosphere context. CMAQ is the model used by EPA and has been widely adopted by the expert air quality modeling community (CMAS, 2005a). This model is able to execute the following photochemical mechanisms: CB-IV (Gery et al., 1989), RADM2 (Stockwell et al., 1990), and SAPRC99 (Carter, 2000). On the other hand, the CAMx model is based on the old Urban Air-Shed Model (UAM) (Reynolds, 1973, 1974) and has been widely used in California and more recently in Houston, Texas. These advanced air quality models contain modules to secondary organic aerosols (SOA) and inorganic aerosols using modal and sectional representations to particle sizes. For inorganic aerosols, the ISORROPIA equilibrium model is used by CMAQ and CAMx (Nenes et al., 1998), while CAMx uses the proposed model by Koo to simulate the SOA (Koo et al., 2003), and CMAQ uses the formulation proposed by Schell (Schell et al., 2001).

2.2.1.1   Models-3/CMAQ

Models-3/CMAQ is designed to be a multipollutant multiscale air quality model that incorporates the latest scientific algorithms for simulating all atmospheric and land process that affect transport, chemical/physical transformation and deposition of atmospheric pollutants both on a regional and urban scale (Byun and Ching, 1999). The CMAQ air quality model is driven for the MM5 meteorological model, and the Sparse Matrix Operator Kernel Emissions (SMOKE) model (Figure 2.32).

Health Risk Conceptual Model

Meteorological Data → MM5 3.7 2003
Land Use Geophysical data → MM5 3.7 2003
MM5 3.7 2003 → MCIP 2.2
MCIP 2.2 → Models-3 CMAQ
Initial & Boundary Conditions → Models-3 CMAQ
Emission Inventory 1999 → Modeling Inventory SMOKE 2.0
Modeling Inventory SMOKE 2.0 → Models-3 CMAQ
Models-3 CMAQ → Modeled ambient concentrations
Ambient Monitored Pollutants 2003 → Modeled ambient concentrations
Modeled ambient concentrations → Health Risk Assessment

Figure 2.32. Models-3/CMAQ Layout

In these latest versions of the modeling system, improvements have been made to all major components. A new land-surface and soil moisture model was added to the MM5 version 3.7 meteorological model to produce better representations of the evolution of the atmospheric mixed layer containing most of the pollutant burden. Also, a new interface processor was created to allow meteorological parameters to pass through directly from the MM5 model to the CMAQ air quality model. Previous model versions had required some meteorological parameters to be re-derived for CMAQ. A new biogenic emissions model, Biogenic Emissions Inventory System-Version 3.09, was included in the SMOKE emissions model version 2.0. Emission factors of naturally-occurring hydrocarbons have been improved and refined in this model. Within the CMAQ model, a new efficient numerical solution routine for the chemical equations was also implemented, resulting in faster CMAQ model run times. Refinements in the treatment of particulate matter were also included in this model version, including new aerosol yields from organic gas species and a new thermodynamics sub-model that has already seen community-wide use in other particulate matter models. Finally, a new routine for vertical diffusion, the Asymmetric Convective Model, was added as an option to Models-3/CMAQ. Thus, the Community Modeling and Analysis System Center, CMAS, in cooperation with USEPA Office of Research and Development have developed an air toxics Models-3/CMAQ version (Ching et al., 2003).

In the near future, the U.S. EPA and the states will be conducting air quality assessments and health risk exposures to a host of semi-volatile toxic compounds, and modeling will be a key aspect of these assessments. Extension of CMAQ modeling to the toxics arena

will help to provide tools for these assessments over a fine scale, as dioxins and diesel exhaust (Ching et al., 2004). The U.S. EPA has planned future work in two areas. First, comparisons will be made between model results and observations and atmospheric deposition of air toxics. Second, based on the work of extending the CMAQ model to this initial toxic compound, other toxic and semi-volatile compounds will be included in CMAQ's simulation capabilities, and tested with new ambient toxics monitoring data (Cooter et al 2002a and 2002b). These advances air quality models can be integrated with health risk assessment techniques (Ching et al., 2004), which together can be a powerful and indispensable tool to optimize the air quality management, air pollution control policy, and to generate air quality standards, especially on some MSATs, such as DPM and benzene (Molina et al., 2004; Lloyd and Cackette, 2001).

A.   SMOKE and Air Toxics Emissions Processing

SMOKE version 2.0 is primarily an emissions processing system designed to create gridded, speciated, hourly emissions for input into a variety of air quality models such as CMAQ, REMSAD, CAMx, and UAM. SMOKE supports area, biogenic, mobile (both onroad and nonroad), and point source emissions processing for criteria, particulate, and air toxics (UNC, 2004). For biogenic emissions modeling, SMOKE uses the Biogenic Emission Inventory System, version 2.3 (BEIS2) and version 3.09 (BEIS3). SMOKE is also integrated with the on-road emissions model MOBILE6.2. The emissions processing paradigm implemented in SMOKE is shown in Figure 2.33.

113
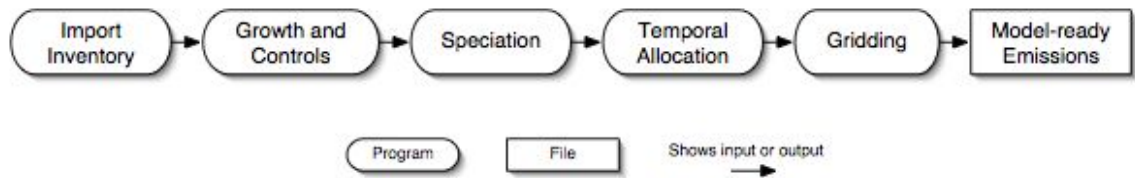
Figure 2.33. Serial Approach to Emissions Processing

For each SMOKE processing category (i.e., area, biogenic, mobile, and point sources), the following tasks are performed:

✓ Read emissions inventory data files

✓ Optionally grow emissions from the base year to the (future or past) modeled year (except biogenic sources)

✓ Transform inventory species into chemical mechanism species defined by an air quality model

✓ Optionally apply emissions controls (except for biogenic sources)

✓ Model the temporal distribution of the emissions, including any meteorology effects

✓ Model the spatial distribution of the emissions

✓ Merge the various source categories of emissions to form input files for the air quality model

✓ At every step of the processing, perform quality assurance on the input data and the results

The emissions processing is managed by the core SMOKE programs. Those core SMOKE version 2.0 programs are: Cntlmat, Elevpoint, Emisfac, Grdmat, Grwinven, Laypoint, Mbsetup, Mrggrid, Normbeis3, Premobl, Rawbio, Smkinven, Smkmerge, Spcmat, Temporal, Tmpbeis3, and Tmpbio (Figure 2.34).

The Smkinven program is responsible for importing the stationary area, non-road, on-road mobile, and point source inventory emissions (Figure 2.35a). The output from Smkinven is used as input to nearly every other core SMOKE programs. Grdmat creates the gridding matrix for the anthropogenic source categories.

Also, for mobile sources the Grdmat program creates an ungridding matrix used by Mbsetup, Premobl, and Emisfac when emission factors are being generated. If meteorology data are input for mobile-source processing, Premobl processes these data, creating hourly temperature and relative humidity profiles by county that are used by Emisfac to compute mobile-source emission factors. Emisfac computes emission factors for a variety of emission processes; these factors are needed when VMT data are used as input for mobile sources (Figure 2.35b). Mbsetup, Premobl, and Emisfac are skipped if only emissions data are imported for mobile sources.

The Temporal program is used to create an hourly emissions file for the anthropogenic source categories. It can read in day-specific and hour-specific data, and merge this with estimated daily and hourly data created from the annual emissions data using temporal profiles.
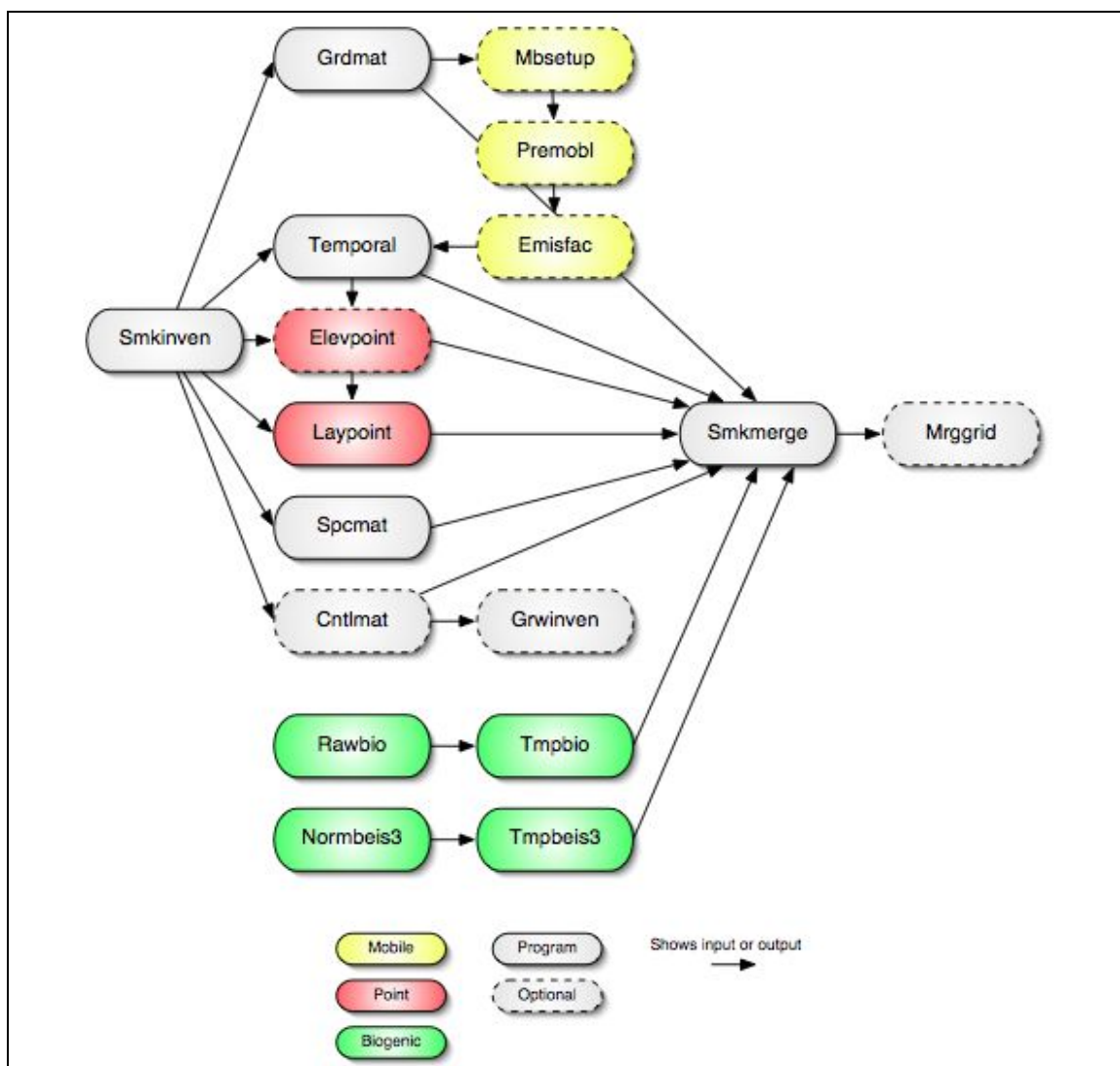
Figure 2.34. Core SMOKE version 2.0 Programs

a

b
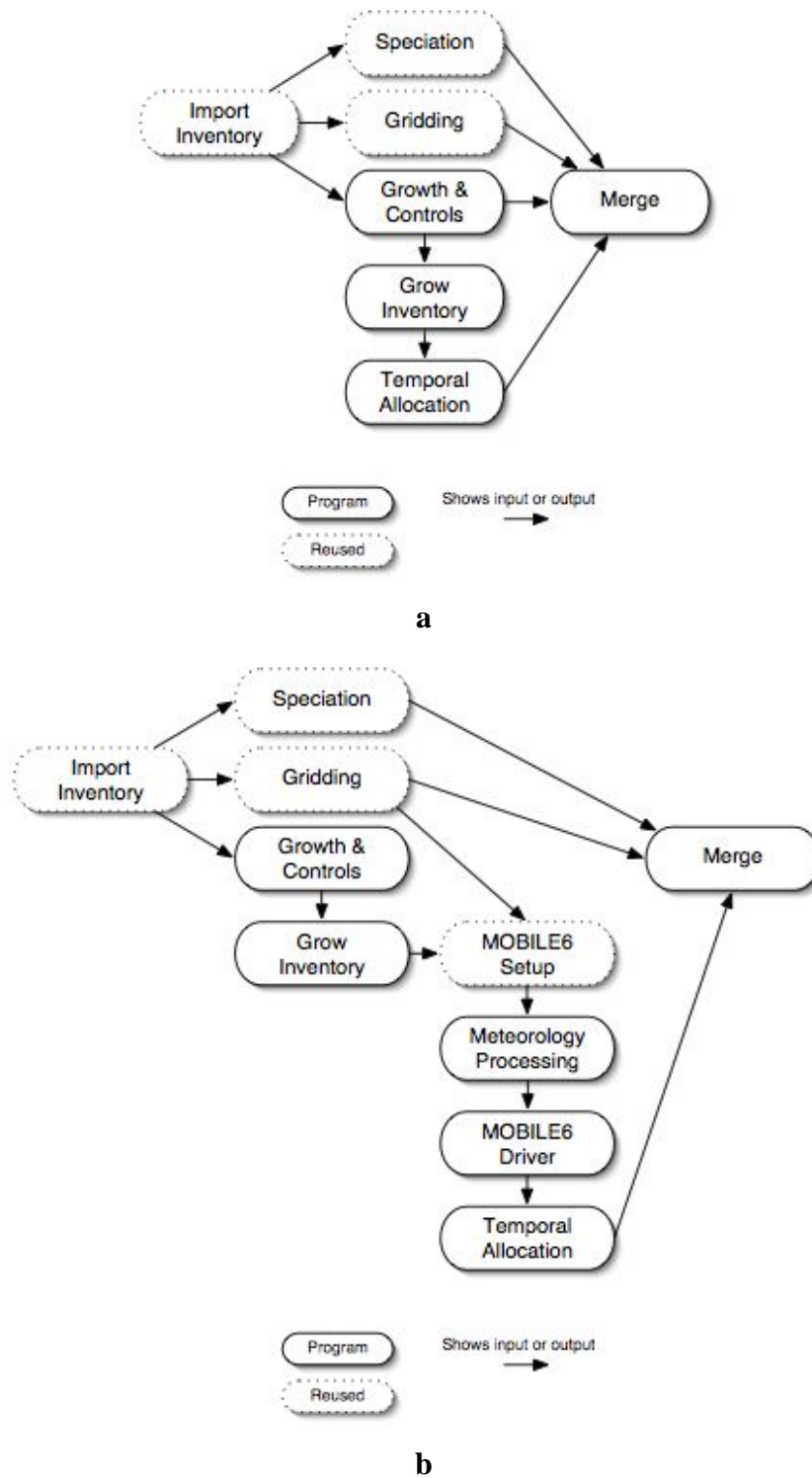
Figure 2.35. Base Case Sources Processing Steps. Area (a) and Mobile Sources (b)

c

d
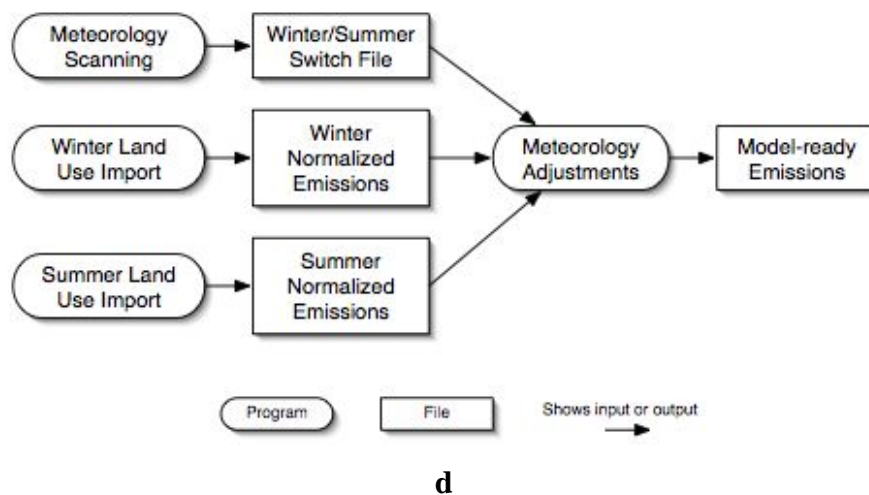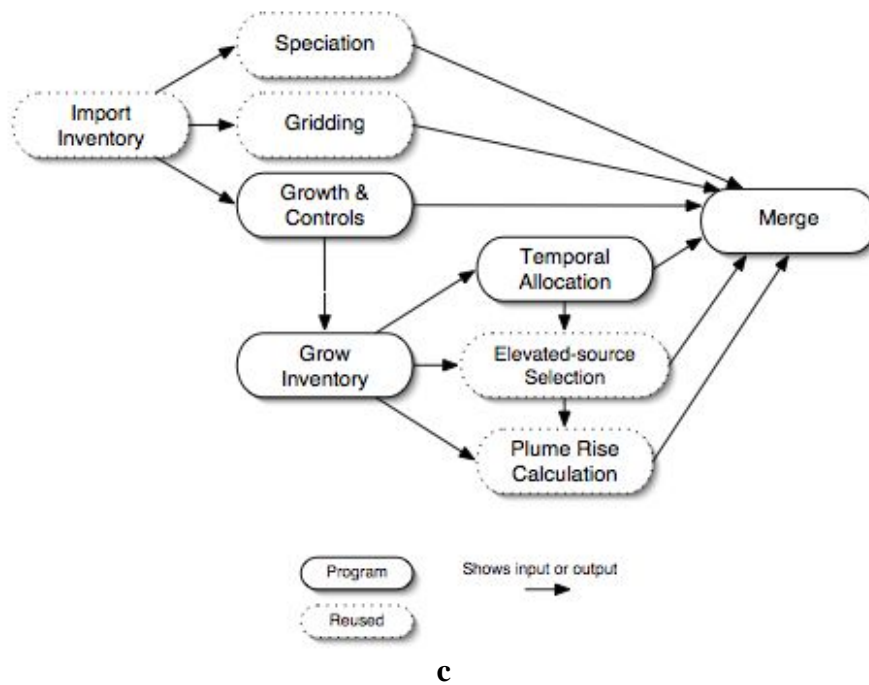
Figure 2.35. Continued. Point (c) and Biogenic Sources (d)

For mobile sources, Temporal can compute hourly emissions using the daily VMT, temporal factors (monthly, day-of-week, and hourly), and the emission factors from Emisfac. If VMT are not used, then the emissions from Smkinven are combined with the temporal factors, and the emission factors are not used at all. Elevpoint preprocesses the selected Plume-in-grid (PinG) and major point sources (Figure 2.35c). Elevpoint is not used when no PinG or major point sources need to be defined. In this case, SMOKE can create elevated emissions for all point sources, so there is no need to specifically indicate the major point sources. Laypoint computes the plume rise for all point sources based on the meteorology data. Spcmat creates the speciation matrices (both mass and molar) for the anthropogenic source categories. It uses the user-selected chemical mechanism. The Rawbio program imports the county or gridded land use data and computes normalized (time-independent) biogenic emissions. As an alternative to BEIS2 processing using Rawbio, SMOKE is capable of using the BEIS3 model. In this type of processing, Normbeis3 creates gridded, normalized biogenic emissions from land use and biogenic emissions factors. Tmpbio applies meteorology adjustments to the gridded, normalized biogenic emissions from Rawbio. It also applies the speciation factors needed for the user-selected chemical mechanism to create gridded, hourly, model-species biogenic emission files for use in Smkmerge or Mrggrid. Tmpbeis3 applies meteorology adjustments to the normalized emissions created by Normbeis3. Tmpbeis3 also applies the speciation profiles needed for the user-selected chemical mechanism to create gridded, hourly, model-species biogenic emissions data for use in Smkmerge and Mrggrid. The Normbeis3 and Tmpbeis3 programs taken together are the equivalent of SMOKE-BEIS3 (Figure 2.35d).

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Cntlmat creates the growth matrices, multiplicative control matrices, and reactivity control matrices for the anthropogenic source categories. This program is not used for base-year emissions without controls. Grwinven is used to grow the emissions to past or future years using the growth matrix created by Cntlmat and the imported inventory data from Smkinven. The output from Grwinven is used in place of the original output from Smkinven when processing past or future years. Smkmerge is used to combine all emissions and matrices to create the gridded, hourly, model-species emissions needed for an advanced air quality model like CMAQ. It can merge for one source category at a time or all source categories at once, and it can read in the model-ready biogenic emissions to merge with the anthropogenic source categories. It can merge the matrices with the inventory data output from Smkinven or the hourly emissions from Temporal, and it can optionally merge the speciation, gridding, or control matrices, or any combination. It also writes state and county emissions totals. Finally, Mrggrid is used to combine gridded emission data files, which can be speciated or non-speciated, and hourly or time-independent. It can combine a 3-D point source file with any number of 2-D files from other source categories. This program is optional and provides a convenient way to merge model-ready output files outside of Smkmerge (UNC, 2004).

SMOKE allows integrating high-performance-computing sparse-matrix algorithms. The SMOKE system is a significant addition to the available resources for decision-making about emissions controls for both urban and regional applications. It provides a mechanism for preparing specialized inputs for air quality modeling research, and it makes air quality forecasting possible (UNC, 2004).

Because of the large amount of computing necessary to calculate a mobile sources emission inventory to be processed on SMOKE, the National Mobile Inventory Model (NMIM) model was designed by U.S. EPA to utilize multiple computers over a computer network (U.S. EPA, 2005m). NMIM develops estimates of current and future emission inventories for on-road motor vehicles and non-road equipment. It uses current versions of MOBILE6.2 and NONROAD models to calculate emission inventories, based on multiple input scenarios to enter into the system, including future scenarios (U.S. EPA, 2005m). However, there are a number of significant limitations in the MOBILE6.2 and NMIM models based highway vehicle HAP inventory for 1999 (Cook et al 2002).

Among these limitations are:

1) The toxic to VOC ratios used to estimate gaseous HAP emissions from heavy-duty gasoline and diesel vehicles are based on tests from only a few engines. Thus, emission estimates for heavy-duty vehicle classes are highly uncertain.

2) MOBILE6.2 and NMIM do not account for impacts of fuel formation on toxics to VOC ratios for diesel-powered vehicles.

3) The adjustments to toxic to VOC ratios applied to account for off-cycle emissions are based on tests from only 12 vehicles in one study.

4) Toxic to VOC ratios are assumed to be the same, regardless of speed, due to a lack of modal emissions data.

5) The modeling used default assumptions about the vehicle mix for various roadway types.

6) All metal emissions estimates are based on only a few tests, and estimates for arsenic and mercury were based on one-half of the detection limit used in various studies, which measured metal emissions.

B.   Description of Models-3 Aerosol Module

The CMAQ version 4.3 aerosol module considers PM2.5 and PM10 and process primary and secondary species. CMAQ's aerosol module adopts a modal approach to represent the ambient particles (Binkowski and Roselle, 2003; Mebust et al., 2003). This module uses the superposition of 3 lognormal sub-distributions to represent the size distribution of these particles. The PM2.5 species are represented by two of these sub-distributions called Aitken (i) mode, which are particles have diameters up to 0.1 μm, and the Accumulation (j) mode, which are particles have diameters between 0.1 and 2.5 μm. The third modal sub-distribution represents particles of the coarse mode, which are particles that have diameters between 2.5 to 10 μm.

Aitken mode represents particles recently formed from nucleation or from direct emissions that have a short lifetime (few minutes), while Accumulation mode represents old particles that have a long lifetime (weeks). Coarse mode represents particles with short lifetime (one day or less). The PM2.5 chemical species considered in Aitken and Accumulation modes are: sulfate, nitrate, ammonia, primary organics, secondary anthropogenic organics, secondary biogenic organics, EC, water, and unspecified anthropogenic mass, as shown in Table 2.10. The primary species considered on CMAQ

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Table 2.10. Speciation and Variable Name Used in CMAQ Aerosol Module

Name	Species Description
ASO4J	Accumulation mode sulfate mass
ASO4I	Aitken mode sulfate mass
ANH4J	Accumulation mode ammonium mass
ANH4I	Aitken mode ammonium mass
ANO3J	Accumulation mode nitrate mass
ANO3I	Aitken mode aerosol nitrate mass
AORGAJ	Accumulation mode anthropogenic secondary organic mass
AORGAI	Aitken mode anthropogenic secondary organic mass
AORGPAJ	Accumulation mode primary organic mass
AORGPAI	Aitken mode mode primary organic mass
AORGBJ	Accumulation mode secondary biogenic organic mass
AORGBI	Aitken mode biogenic secondary biogenic organic mass
AECJ	Accumulation mode elemental carbon mass
AECI	Aitken mode elemental carbon mass
A25J	Accumulation mode unspecified anthropogenic mass
A25I	Aitken mode unspecified anthropogenic mass
ACORS	Coarse mode unspecified anthropogenic mass
ASEAS	Coarse mode marine mass
ASOIL	Coarse mode soil-derived mass
AH2OJ	Accumulation mode water mass
AH2OI	Aitken mode water mass

module are elemental and organic carbon (AEC and AORGPA), dust and other species not further specified (A25). Secondary species considered are sulfate (ASO4), nitrate (ANO3), ammonium (ANH4), water (AH2O), and organic from precursors of anthropogenic (AORGA) and biogenic (AORGB) sources (Binkowski and Roselle, 2003).

In general, the CMAQ module includes the coagulation processes, the particles growth for the new mass addition, and the particles formation due to the gas-gas interaction. The chemical transformations of gaseous emissions and particles are processed by the chemical mechanism available on CMAQ.

The aerosols module that considers the nucleation, coagulation, and other phenomena of particles growth processes the formed results. The outcomes estimated by CMAQ contain the information over the primary and secondary hourly PM2.5 concentrations (Byun and Ching, 1999; Binkowski and Roselle, 2003).

According to Binkowski and Roselle, the following is the translation of CMAQ output species into PM2.5 (Binkowski and Roselle, 2003):

PM2.5= ASO4I + ASO4J + ANH4I + ANH4J + ANO3I +ANO3J +   Equation 2-11
AORGAI + AORGAJ + 1.167*(AORGPAI + AORGPAJ) +
AORGBI + AORGBJ + AECI + AECJ + A25I + A25J

C.   Description of Models3 Air Toxics Module

U.S. EPA's Models-3/CMAQ version Air Toxics (CMAQ-AT) modeling system relates

source emissions to ambient air concentrations through simulation of the relevant

physical and chemical processes. It can predict the chemistry and fate of 20 gaseous high

priority toxic air pollutants. High priority HAPs can exist in the gas phase, aqueous phase

or aerosol phase, or partially in all of these phases, which makes their behavior in the

atmosphere difficult to model. The CMAQ-AT, SMOKE version 2.0, and NMIM through

MOBILE version 6.2 have the capability to model some semi-volatile air toxic

compounds such as atrazine (Cooter et al, 2002), benzene, MTBE, 1,3-butadiene, and

primary and secondary formaldehyde, acetaldehyde, and acrolein, among others (Majeed

et al., 2004; Hutzell et al., 2004; Ching et al., 2004 and 2003; Seigneur et al., 2003). For

secondary HAPs formation, CMAQ-AT provides two names speciation for acetaldehyde,

acrolein, and formaldehyde, as shown in Table 2.11.

Table 2.11. Primary and secondary HAPs species on CMAQ-AT

HAPs	Primary	Secondary
Acetaldehyde	ALD2_PRIMARY	ALD2
Acrolein	ACROLEIN_PRIMARY	ACROLEIN
Formaldehyde	FORM_PRIMARY	FORM

3.0   METHODOLOGY

The methodology explained in this chapter has the objective of giving a structure to the development of an analytical protocol to assess the health risk of emission scenarios and of regulatory actions over future on-road sources scenarios to reduce HAPs concentrations. The overall approach included running CMAQ version 4.3 and CMAQ-AT models with and without the following sources categories: on-road, light-duty vehicles (LDVs), heavy-duty vehicles (HDVs), diesel fueled sources (DFS), on-road DFS, and biogenic sources, as well as a future 2020 year with the effects of MSATs regulations. This chapter explains the methodology followed to estimate the emissions inventory required for the model runs, the base case modeling run, the emissions sources scenarios, and the health risk estimations considered in this study. The year 2020 was selected as a future scenario to compare the projections estimated by the U.S. EPA in its study Analysis of the Impacts of Control Programs on Motor Vehicle Toxic Emissions and Exposure Nationwide (U.S. EPA, 1999b and 1999c).

3.1   CONCEPTUAL MODEL'S DEVELOPMENT

The protocol developed here allows the estimation of the chronic health effects based on toxicological and epidemiological evidence under different emission scenarios, by taking into account the modeled HAPs annual concentrations obtained as output of the Models-3/CMAQ and linked with toxicological cancer risk and non-cancer risk equations, as well

as with C-R functions based on epidemiological studies. With those components, the cancer risk, non-cancer risk, premature mortality risk, lung cancer mortality risk, and CVD mortality risk, expected in a particular area of the modeling domain can be estimated and compared for different emission scenarios. It may be noted that Models-3/CMAQ was assumed as the inhalation exposure model, i.e., the population is exposed to the outdoor ambient HAPs concentrations without taking into account for the time that people spend indoors and outdoors. In addition, the risk assessment is designed to be a picture for measuring progress in reducing risks from exposure to air toxics. For this reason, this study is based on a 1999 inventory of air toxics emissions. It then assumes individuals spend their entire lifetimes (70 years) exposed to these air toxics. Therefore, it does not account for the reductions in emissions that have occurred since 1999 or those that will happen in the near future due to new regulations for mobile and industrial sources.

These assumptions could be considered satisfactory to do emission scenarios analysis for pollutants, since the analysis approach intended by this proposed modeling involves considering the difference in mass concentrations and health risk values among the proposed emission scenarios as compared to the base case scenario rather than the absolute mass concentration or health risk values. This assumes that the factors that contributed to the under and over prediction of those air toxics concentrations would contribute similarly in all the scenarios considered in the analysis, causing minimal effects on the differences among the scenarios.

The 2003 daily and annual ground level diesel aerosols and HAPs concentrations were predicted using the advanced air quality model CMAQ version 4.3 and CMAQ-AT, respectively, through a 36-km modeling domain and compared with available 2003 monitored data from Nashville, TN. Those area, point, on-road, non-road, and biogenic emissions were temporal and spatially allocated using the advanced emissions model SMOKE version 2.0. On-road sources were predicted using NMIM for the whole modeling domain, whereas the 1999 UTK emission inventory and the NEI99 were used for point, area, and non-road sources for Tennessee and for the rest of the 23 states in the modeling domain, respectively. The meteorological variables were generated for March, June, September, and December of 2003 through the mesoscale model (MM5) version 3.7 developed by the National Center for Atmospheric Research (NCAR) at the Pennsylvania State University, PSU (PSU/NCAR, 2005) and processed by the meteorology-chemistry interface processor (MCIP) version 2.2. This general conceptual model is shown in Figure 3.1.

3.2   TIME PERIOD SELECTION

The time period selected for this study consisted of the following months of 2003: March, June, September, and December, which were used to estimate an annual concentration for the following pollutants: acetaldehyde, acrolein, benzene, 1,3-butadiene, formaldehyde, EC, NOx, and DPM (EC and NOx were selected to compare the diesel fueled sources emission of DPM).

Health Risk Conceptual Model
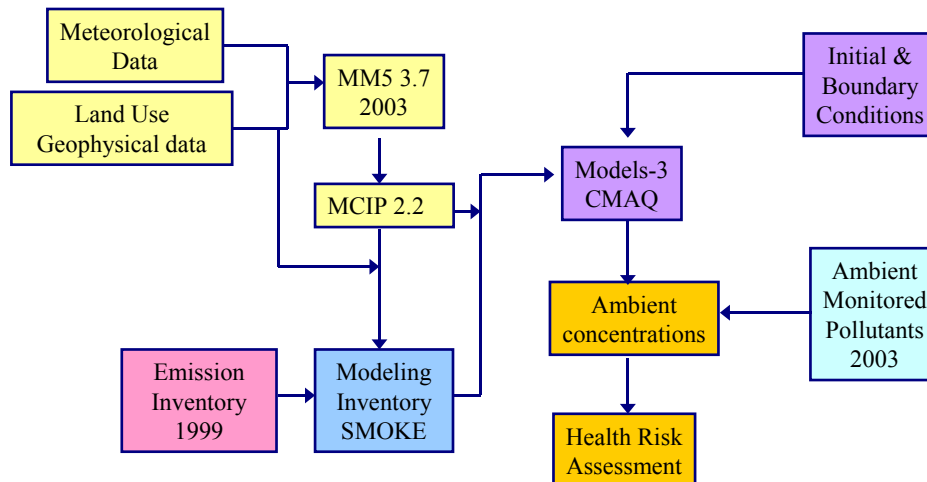
Figure 3.1 Health Risk Conceptual Model

These months were selected because they represented the seasonal concentrations of some HAPs for spring, summer, fall, and winter, respectively. In fact, an analysis of the daily formaldehyde and acetaldehyde collected at EATN and LOTN monitors in Nashville during 2002 and 2003, showed that the formaldehyde and acetaldehyde concentrations peaked during the months of June, July, August, and September, and were minimum during December, January, and February, as shown in Figures 2.19 and 2.20 (other pollutants did not show seasonal patterns). Therefore, it was assumed that each month represented a season; March represented spring, June represented summer, September represented fall, and December represented the winter season.

3.3   MODELS-3/CMAQ MODELING DOMAIN

A modeling domain represents the geographical boundaries of the region to be modeled. It is selected such that the area of interest is situated at the center of the domain with enough area surrounding the analyzed region, to minimize the effect of boundary conditions and pollution transport (Doraiswamy, 2004; Sanhueza, 2002). The size of the domain of 36-km was selected due to the unavailability of the computer sources required and the input data necessary. Since the results of this study were focused principally in the Nashville metropolitan area and Tennessee, the domain was selected such that Nashville and Tennessee were approximately at the center of the domain surrounded by other states.

This domain included most of the central eastern region of the U.S., counting the following 24 states: Alabama, Arkansas, Florida, Georgia, Illinois, Indiana, Iowa, Kansas, Kentucky, Louisiana, Michigan, Mississippi, Missouri, Nebraska, New York, North Carolina, Ohio, Oklahoma, Pennsylvania, South Carolina, Tennessee, Texas, Virginia, and West Virginia, as shown in Figure 3.2. This domain had 48x42 grid cells, each one with 1,728 km x 1512 km, base on Lambert Conformal map projection. A vertical resolution of eleven layers was configured following the sigma-pressure structure with denser grids at lower levels in order to better resolve the boundary layer (Sanhueza, 2002).
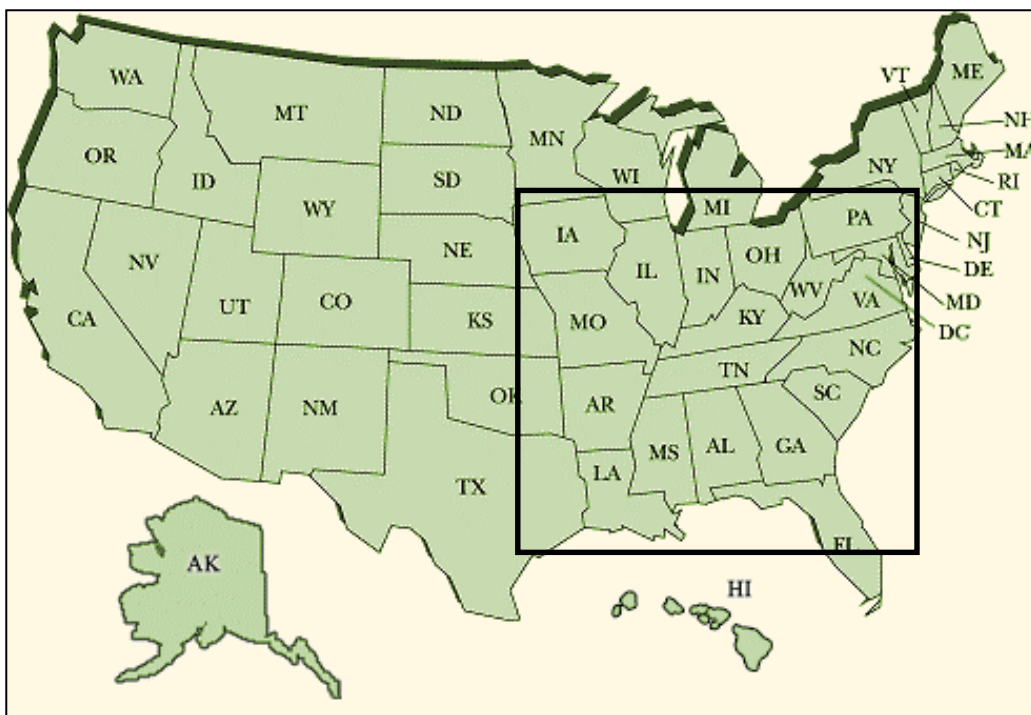
Figure 3.2   36-km Modeling Domain

3.4   METEOROLOGICAL INPUTS

The meteorological inputs to the SMOKE and CMAQ models were processed by the

MM5 model version 3.7, which is a mesoscale prognostic meteorological model and a

limited-area, nonhydrostatic, terrain-following sigma-coordinate model designed to

simulate or predict mesoscale atmospheric circulation, such as temperature, wind speed,

wind field, humidity, and planetary boundary layer. The model is supported by several

pre- and post-processing programs (Figure 3.3), which are referred to collectively as the

MM5 modeling system (PSU/NCAR, 2005).

The MM5 Modeling System Flow Chart
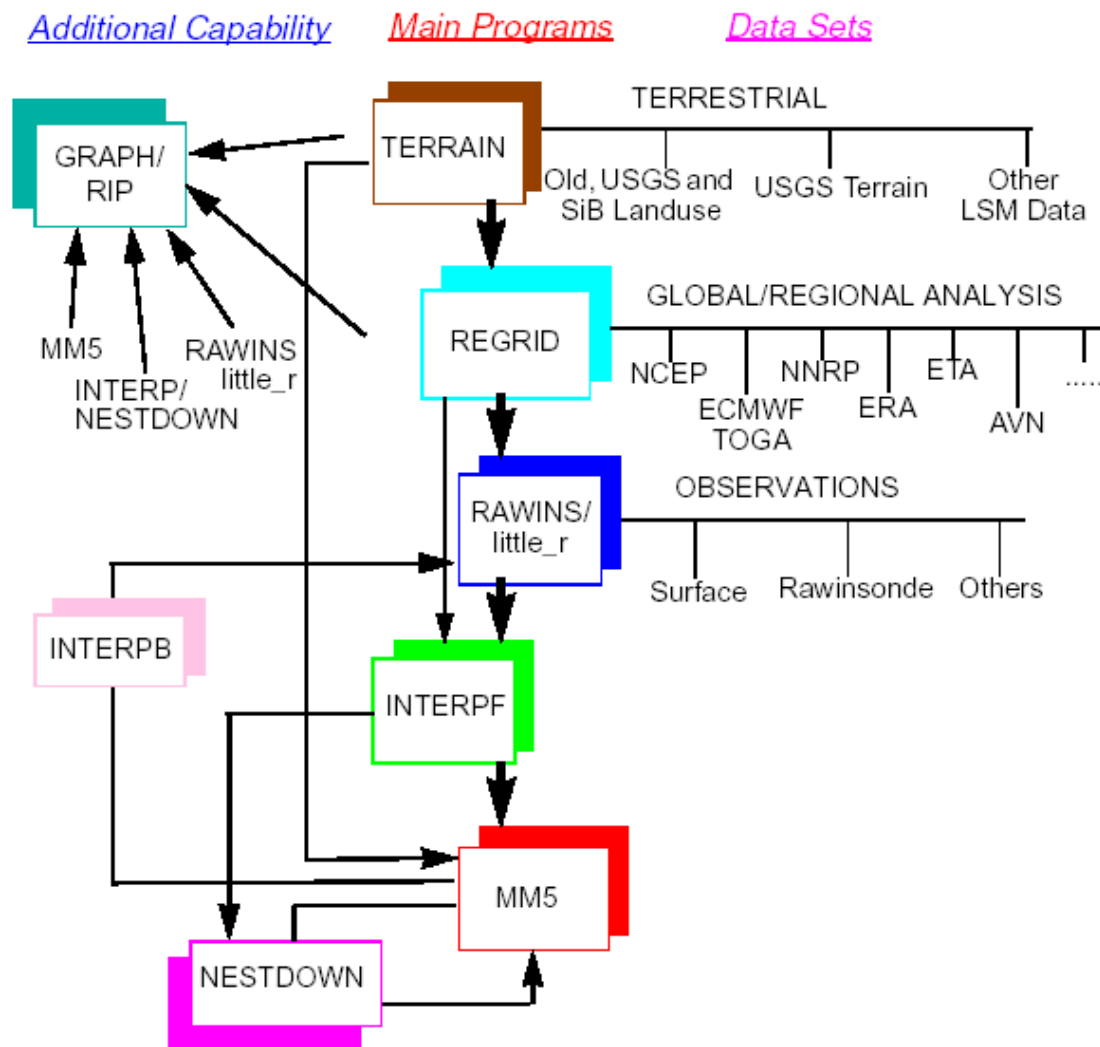
Figure 3.3 The MM5 Modeling System Flow Chart (PSU/NCAR, 2005)

Terrestrial and isobaric meteorological data are horizontally interpolated through the programs TERRAIN and REGRID from a latitude-longitude mesh to a variable high-resolution domain on a Mercator, Lambert conformal, or polar stereographic projection. Since the interpolation does not provide mesoscale detail, the interpolated data may be enhanced through the programs RAWINS or little_r with observations from the standard network of surface and rawinsonde stations. Program INTERPF performs the vertical interpolation from pressure levels to the sigma coordinate system of MM5. Sigma surfaces near the ground closely follow the terrain, and the higher-level sigma surfaces tend to approximate isobaric surfaces. Since MM5 is a regional model, it requires an initial condition as well as lateral boundary condition to run. To produce lateral boundary condition for a model run, it is necessary to use gridded data to cover the entire time period that the model is integrated (PSU/NCAR, 2005).

For this particulate study, the following scheme was used to run MM5:

TERRAIN $\rightarrow$ REGRID $\rightarrow$ LITTLE_R $\rightarrow$ INTERPF $\rightarrow$ MM5.

The program TERRAIN is the first program used to run in the suite of MM5 system programs, which allows to design the mesoscale model configuration: grid place, grid size, and the resolution data to use to generate terrain elevation, landuse category, and other datasets to run MM5 with the land-surface model option. This program creates meteorological fields on the mesoscale grid designed in program TERRAIN. This is also the first place to select the time period of the case (PSU/NCAR, 2005).

The program REGRID is the second program in the suite of MM5 system programs. REGRID has two sequential programs: pregrid and regridder. The program pregrid pre-processes gridded, pressure-level meteorological fields from sources such as the NCAR archive, and NCEP's ftp server, and it puts the data in an intermediate format. The program regridder takes the intermediate-format data and output file from TERRAIN, and creates a "first-guess" for subsequent programs. The output file from REGRID has 3-dimensional meteorological fields of wind, temperature, relative humidity, geopotential height, and 2-dimensional fields like sea-level pressure and sea-surface temperature (PSU/NCAR, 2005).

The program LITTLE_R is the third program in the suite of MM5 system programs. This program reads in output from program REGRID and observations (radiosonde and surface reports), performs an objective analysis, which blends first guess with observations, and outputs data on pressure levels again. The program functions of LITTLE_R are as those in RAWINS, but RAWINS is an older Fortran 77 package. The advantages of LITTLE_R are that it is more users friendly and makes meteorological observations inputs easier to deal with for users who don't have access to NCAR's archived ADP data (PSU/NCAR, 2005).

The program INTERPF is the fourth program in the suite of MM5 system programs. This program takes pressure-level meteorological fields produced by LITTLE_R; the user's definition of model sigma levels, and interpolates pressure level data to sigma levels (PSU/NCAR, 2005).

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The program MM5 is the last program in the suite of MM5 system programs. This is the numerical weather prediction part of the modeling system. MM5 can be used for a broad spectrum of theoretical and real-time studies, including applications of both predictive simulation and four-dimensional data assimilation to monsoons, hurricanes, and cyclones (PSU/NCAR, 2005).

A 36-km domain was used in this study. The model was run in a PSU/NCAR super computer called bluesky using the following physics options to get better MM5 performance:

✓ An explicit moisture scheme of warm rain for June and September and simple ice (Dudhia) for March and December were used.

✓ The Planetary Boundar Layer (PBL) scheme and diffusion of Hong-Pan (or MRF) was used for all months, which is suitable for high-resolution in PBL (as for Blackadar scheme).

✓ The cloud-radiation scheme was used for all months, which is sophisticated enough to account for long wave and short wave interactions with explicit cloud and clear-air, as well as atmospheric temperature tendencies, providing surface radiation fluxes.

✓ A five-layer soil model was used for all months. The temperature is predicted in 1, 2, 4, 8, 16 cm layers (approx.) with fixed substrate below using vertical diffusion equation.

✓ A non-hydrostatic mode processed with cloud cover was used for all months. The Kain-Fritsch convective parameterization scheme accounted for sophisticated cloud

mixing schemes to determine entrainment/detrainment, and removing all available buoyant energy in the relaxation time. The planetary boundary layer (PBL) processing was performed according to the Blackadar scheme that allowed for high resolution PBL.

This work also utilized the gridded four-dimensional data assimilation (FDDA) scheme available in MM5. The coarse domain consisted of 48 by 42 grid cells with 36 km horizontal grid resolution. A vertical resolution of 23 sigma layers was used. Because SMOKE 2.0 cannot process a tremendous meteorological input file, like one-month data, each month was separated into 4 batches of 10 or 5 days, starting 5 days before the 1st of each month to avoid the initial conditions effects on the CMAQ runs, accounting for 16 runs and outputs generated through MM5 version 3.7. Since the outputs from the MM5 model cannot be used directly with chemistry and emissions models, they were processed using the Meteorology Chemistry Interface Processor (MCIP) version 2.2. The MCIP transforms the MM5 output to a form that can be used by the emissions processor and by the air quality model. Furthermore, the MCIP processor performs the collapsing of the 23-vertical layer resolution to an 11-layer resolution used in the CMAQ modeling system (Doraiswamy, 2004; Sanhueza, 2002).

3.5   EMISSIONS INVENTORY DEVELOPMENT

Emissions inventories are used as inputs to the advanced air quality models, which consist of county-based annual emission estimates based on algorithms developed by the

U.S. EPA. The CMAQ model requires speciated, spatial, and temporal emissions for each grid cell of the modeling domain and time period being modeled. These emissions can be hourly processed through the Sparse Matrix Operated Kernel Emissions processor (SMOKE) version 2.0. This section explains the steps followed in the emission inventory development and processing.

The emission inventories are usually categorized into four major categories: point, area, mobile, and biogenic sources

3.5.1 Point Sources

For criteria pollutants, this category includes all emission sources that can be attributed to emissions points, usually a stack. These are identified by name, latitude, and longitude. The research group at the University of Tennessee, Knoxville, developed the criteria pollutants point source inventory for the state of TN. For other states in the domain, the NEI99 version 3 was used (Doraiswamy, 2004). For HAPs, this category is called major sources, which emit or have the potential to emit 10 tons per year or more of any listed HAP or 25 tons per year or more of a combination of listed HAPs. For the 24 states in the domain, the NEI99 version 3 was used. These HAPs emissions inventory were checked and fixed carefully for mass units in tons per year, since each state or each county provided the annual emissions using the following units: tons, pounds, kilograms, grams, and milligrams. To do that, the JMP statistics software version 5.1 was used over the NEI99 version 3 for each state and SCC code.

3.5.2 Area Sources

For criteria pollutants, this includes emission sources that are spread over an area including many small point sources that are not accounted for in the point source inventory. This category also includes non-road mobile sources such as airplanes, marine vessels, locomotives, etc. The research group at the University of Tennessee, Knoxville, developed the ammonia area source inventory and fugitive particulate emissions from paved roads for the state of TN (Doraiswamy, 2004). For other states in the domain and other pollutants for Tennessee the NEI99 version 2.0 was used (Doraiswamy, 2004). For HAPs, they are defined as stationary sources that emit or have the potential to emit less than 10 tons per year of a single HAP and less than 25 tons per year of all HAPs combined. This category for HAPs does not include non-road sources. The CAA defines "area source" as any stationary source of HAPs that does not qualify as a major source (U.S. EPA, 1990). For the 24 states in the domain, the NEI99 version 3 for area sources was used. Like Point sources, these HAPs emissions inventory were checked and fixed carefully for mass units to tons per year using the JMP statistics software version 5.1 over the NEI99 version 3 for each state and SCC code.

In addition and since open burning sources are important HAPs emissions contributors, a spatial distribution over the modeling domain was performed in SMOKE 2.0 for the NEI99 HAPs open burning sources to check for inconsistence between Tennessee and surrounding states.

3.5.3 Mobile Sources

For criteria pollutants, this category includes all on-road mobile sources. While for HAPs, this category is defined as any kind of vehicle or equipment with a gasoline or diesel engine, which is subdivided in on-road and non-road sources. The on-road criteria pollutants were estimated through NMIM and compared with the NEI99 version 3 and the 1999 UTK emission inventory (Doraiswamy, 2004) to get a more realistic and accurate on-road emissions inventory. The VMTs used for Tennessee to run NMIM were those developed by the research group at the University of Tennessee, Knoxville, (UTK Project) for the year 1999 (Davis et al., 2002; Doraiswamy, 2004). Those VMTs were adjusted to 28 vehicles types as required on the NMIM model instead of 16 used in the UTK project. For the rest of the 23 states the NEI99 version 3's VMTs were used. The diesel sulfur content used on NMIM varied by county, based on fuel survey information provided for the U.S. EPA (2005m), which on average were less than the standard of 500 ppm for 1999. The BAROMETRIC PRES command was always used in NMIM, since this value interacts with the relative humidity values. Average speed distributions were always specified on NMIM using the SPEED VMT command, rather than the AVERAGE SPEED command. Finally, NMIM used hourly relative humidity and hourly temperatures for each county provided by the U.S. EPA (2005m).

HAPs emissions for acetaldehyde, acrolein, benzene, 1,3-butadiene, formaldehyde, and MTBE were generated through NMIM for the 24 states in the modeling domain using the same VMTs and setting used to create the criteria pollutants emissions. However,

gasoline and diesel fuel parameters, provided by the U.S. EPA by county for each month of each calendar day, required the maximum amount of detail to properly model toxic emissions. The OXYGENATE command is required when modeling air toxics, and supersedes the OXYGENATED FUELS command used to model winter oxygenate programs in MOBILE6. The OXYGENATE command requires that oxygen content be expressed as volume percent instead of weight percent, as required by the OXYGENATED FUELS command. GAS AROMATIC%, GAS OLEFIN%, GAS BENZENE%, E200, and E300 were always specified using the default data provided in NMIM. RVP OXY WAIVER command was always set to 1 (no waiver), because Reid vapor pressure (RVP) values from the fuel surveys are assumed to already account for any RVP effect from oxygenated fuels. The FUEL RVP command was always required. The GASOLINE SULFUR command and FUEL PROGRAM command Option 4 were always used to explicitly set the default sulfur content of gasoline provided on NMIM for 1999. For gasoline, Eastern Research Group, Inc., (ERG) determined the fuel properties under contract to the U.S. EPA using gasoline survey data (U.S. EPA, 2005m).

The on-road criteria pollutants and HAPs emissions inventories were run on NMIM for the year 2020 and for Compressed Natural Gas (CNG) on LDVs and all vehicles over all the 24 states in the modeling domain assuming 100% penetration from 1994. The Tennessee VMTs growth adjusted to 28 vehicle types for the scenario 2020 was obtained from the UTK project (Davis et al, 2002). For the rest of the domain annual VMTs growths were used, which were provided by U.S. EPA. For the year 2020, all the in effect on-road sources regulations were applied on NMIM, including the reformulated gasoline

(RFG) program, national low emission vehicle (NLEV) program, emissions standards for passenger vehicles and gasoline sulfur control requirements (Tier 2), and the 2007 heavy-duty vehicle standards and highway diesel fuel sulfur control requirements, which are expected to yield significant reductions of mobile source air toxics (U.S. EPA, 2001a and 2001b). The non-road HAPs emissions were obtained from the NEI99 version 3.

3.5.4 Biogenic Sources

This includes emissions from natural sources such as vegetation. This inventory was created using BEIS 3.09 on SMOKE2.0, base on land use and meteorological data for each month analyzed for 1999.

3.6 SMOKE 2.0 MODEL RUNS

The methodology consisted of running the SMOKE 2.0 model with and without the source emissions scenarios as illustrated in Table 3-1. The base case was run with all sources included. The scenarios NO DFS, NO ONROAD_DFS, NO LDVs, NO HDVs, were estimated through control matrices for the corresponding source (s) as described in section 2.2.1.1 (A). Those source categories were eliminated using the source classification codes (SCC) through a control matrix for each scenario. The scenarios NO ONROAD and NO BIO were estimated running the SMOKE 2.0 and merging all the emissions sources without on-road and biogenic emissions, respectively. Finally, the scenario for the year 2020 was estimated merging the point, area, non-road, and biogenic

Table 3-1 Emissions Scenarios on SMOKE 2.0

Year	Emission Scenario	Name Scenario	Objective
1999	All Sources Present (Base Case)	BC	Base Case
1999	Base Case Without On-Road Sources	NO ONROAD	Contribution of on-road sources to air toxics, EC, and NOx emissions
1999	Base Case Without DFS	NO DFS	Contribution of DFS to air toxics, EC, DPM, and NOx emissions
1999	Base Case Without On-Road DFS	NO ONROAD_DFS	Contribution of on-road DFS to air toxics, EC, DPM, and NOx emissions
1999	Base Case Without LDVs	NO LDVs	Contribution of LDVs sources to air toxics, EC, and NOx emissions
1999	Base Case Without HDVs	NO HDVs	Contribution of HDVs sources to air toxics, EC, and NOx emissions
1999	Base Case Without Biogenic Emissions	NO BIO	Contribution of biogenic sources to air toxics emissions
2020	In effect MSATs regulation for 2020	YEAR 2020	Contribution of in effect MSATs regulations to air toxics, EC, and NOx emissions
1999	Base Case Without HDDVs	NO HDDVS	Contribution of HDVs sources to DPM emissions
1999	Base Case with CNG on LDVs	CNG on LDVs	Contribution of CNG on LDVs to air toxics emissions
1999	Base Case with CNG on all vehicles	CNG on All Vehicles	Contribution of CNG on all vehicles to air toxics emissions
2020	In effect MSATs regulation for 2020 without DFS	YEAR 2020_NODFS	Contribution of in effect MSATs regulations to DPM emissions

1999 emissions with the 2020 on-road emissions estimated by NMIM. Two extra scenarios were run to consider the DPM effect on heavy-duty vehicles and over the year 2020. Those scenarios included running the base case without heavy-duty diesel vehicles (NO HDDVs) and the year 2020 without diesel fueled sources from point, area, and non-road 1999 emissions, and without diesel fueled sources from the 2020 on-road emissions generated by NMIM (YEAR 2020_NODFS). These two scenarios were also estimated through control matrices for the corresponding source (s) as described in section 2.2.1.1 (A). The difference between the base case scenario and the NO DFS scenario were the DPM emissions for the base case run, whereas the difference between the scenario YEAR 2020 and the YEAR 2020_DFS scenario were the DPM emissions for the year 2020. Those DPM emissions were estimated considering the addition of following PM2.5 species defined in SMOKE2.0: Elemental carbon (EC), primary fine particulate matter (PMFINE), primary nitrate (PNO3), primary organic aerosols (POA), and primary sulfate (PSO4) (UNC, 2004).

The difference between the NO ONROAD DFS scenario and the NO DFS were the DPM emissions from the on-road DFS. Whereas, the difference between the NO HDDVs scenario and the NO DFS scenario were the DPM emissions from the HDDVs. Finally, the Equation 3-1 allows estimating the DPM emissions from LDDVs.

DPM (LDDVs) = [BC – (NO HDDVs-NO ONROAD_DFS)]- NO DFS   Equation 3-1

Once the base case run was completed, the model results were plotted in order to determine the contribution of each scenario for the area analyzed on the modeling domain. A future 2020 scenario for non-road, area, and point sources were not simulated since no activity growth data were available for the 24 states at the time this study was conducted, as well as the in effect non-road regulations on diesel engines were not available in the model NMIM or NONROAD by the time when this analysis was conducted. Summarizing, batches of 5 days were run for each month of 30 days plus an extra batch of 5 days early for each month to avoid the boundary conditions, in other words 35 days, doing a total of 7 batches for each scenario. Since this study considered 10 scenarios (Table 3-1), then the total batches to run for each month were 70 batches. Considering that 4 months were analyzed, and then a total of 280 batches were run in SMOKE2.0 to merge each point, area, on-road, non-road, and biogenic run scenarios.

3.6.1 Inventory Speciation

SMOKE 2.0 processes criteria pollutants and air toxics inventories. The EPA's criteria pollutants inventory typically includes emissions of carbon monoxide (CO), nitrogen oxides (NOx), and volatile organic compounds (VOC) or total organic gases (TOG). Particulate inventories contain ammonia (NH_3), sulfur dioxide (SO_2), particulate matter (PM) of size 10 microns or less (PM_{10}), and PM of size 2.5 microns or less ($PM_{2.5}$). The VOC classification is an umbrella for all organic compounds and the NO_x is the sum of NO and NO_2 emissions.

The toxics inventories that SMOKE can process are data from the NEI that includes 188 HAPs. These aggregate emissions need to be broken down into the constituent species for the model to process them appropriately in the chemical reactions. For point, nonpoint, on-road mobile, and non-road mobile sources, the toxics inventory contains emissions for VOC pollutants that are provided as explicit chemical compounds (for example, benzene, formaldehyde, etc). These same VOC emissions are also included as an aggregated VOC value in the criteria emissions inventory. To use these inventories together, Smkinven tool provides the necessary options to ensure that double counting of VOC emissions will not occur. These two options are the "integrate" and "no-integrate" options.

The "integrate" option involves subtracting toxic VOC emissions from the criteria VOC emissions to avoid double counting of VOC when the emissions are speciated. With this option the user must ensure that the sources in the toxics and criteria inventories match up one-to-one, so that Smkinven can properly compute the emissions. For this study the "integrate" option was used (UNC, 2004).

Only two chemical mechanisms support combining toxics and criteria inventories, and both are for the CMAQ model. The first is the "current-CB4" mechanism, which can refer to either the released version of CMAQ with CB4, or to an unreleased version that includes mercury. The second chemical mechanism is the "toxics-CB4" mechanism, which refers to the CMAQ version that includes toxics species that have been integrated into the chemical mechanism; these "explicit" toxics species are formaldehyde, acetaldehyde, acrolein, and 1,3-butadiene. This CMAQ version also includes benzene as

a separate species (UNC, 2004). For this study the "toxics-CB4" chemical mechanism was used.

During import of both toxics and criteria emission inventories, SMOKE matches the area, on-road mobile, and nonroad mobile emission inventories by country/state/county code and SCC. SMOKE also matches the toxics and criteria records for the point sources, provided that the point sources in the two inventories use identical fields for their source characteristics. It was necessary to ensure that the source characteristics for all source categories match between the two inventories for any sources that were required to have matched. Once they were matched, SMOKE had both a criteria VOC emissions value and toxics emission values for individual VOC chemical compounds.

To do that, SMOKE can optionally compute a NONHAPVOC value by subtracting the sum of toxics VOC from the criteria VOC value. This same approach can be used to create a NONHAPTOG value if the inventory uses a TOG value instead of a VOC value. The case of computing NONHAPVOC is called the "integrate" case. Likewise, the case of not computing NONHAPVOC is called the "no-integrate" case (UNC, 2004).

With the "integrate" approach, the NONHAPVOC mass and the toxics VOC mass are independent from one another and will not double count emissions. The calculation must be performed for each source, and Smkinven will set the criteria VOC value to zero when it computes the NONHAPVOC value. Smkinven determines which pollutants should be

subtracted from VOC using the "VOC or TOG" column in the inventory table (INVTABLE) file (UNC, 2004).

3.6.2 Spatial Allocation of Emissions

The research group at the University of Tennessee, Knoxville, developed the spatial allocation for the state of Tennessee, whose county-based emissions were allocated to each grid cell based on spatial surrogates (Doraiswamy, 2004). For the rest of the 23 states the default spatial allocations of SMOKE2.0 were used. Spatial surrogates represent the percentage of emissions from each county that are allocated to each grid.

The spatial processing operation, or gridding, combines the grid specification for the air-quality modeling domain with source locations from the SMOKE inventory file. The resulting gridding matrix is a sparse matrix that describes in which grid cells the emissions for each source occur within the modeling domain. The gridding matrix is applied to the inventory emissions to transform source-based inventory emissions to gridded emissions. The SMOKE Grdmat program creates the gridding matrix for area, mobile, and point sources. The gridding step is different depending on the type of source being processed (UNC, 2004).

3.6.3 Temporal Processing

The emissions in the inventory were annual emissions fixed to tons/year. These were converted to hourly emissions by use of appropriate default profiles within the SMOKE2.0 model. The temporal profiles describe the variation in emissions as function of time-period for each source category. Profiles are available for month of the year, day of the week, and hour of the day periods in SMOKE2.0. The final processed inventory contained hourly emissions for each grid cell in the domain. This was used as input to the CMAQ models.

3.7 INITIAL AND BOUNDARY CONDITIONS

Initial conditions (IC) refer to the concentrations of all the species at the start of the simulation. For this study, the following date were set to start five day earlier for March, June, September, and December to allow for the "spin-up" period:

✓ March; February 24, 2003, 00 hours GMT

✓ June; May 27, 2003, 00 hours GMT

✓ September; August 27, 2003, 00 hours GMT

✓ December; November 26, 2003, 00 hours GMT

Boundary conditions (BC) refer to the concentrations of all species at the boundary of the domain for each time step of the air quality simulation. Both IC and BC specific conditions are required on the CMAQ modeling system. For the 36-km domain, the IC

and BC were generated using the Initial Condition processor (ICON) and the Boundary Condition processor (BCON) on CMAQ modeling, respectively. The ICON and BCON processors performed the needed spatial interpolation, gridding, species processing, and mechanism conversions for each month analyzed. This domain used a set of default time invariant concentrations provided as part of the CMAQ 4.3, and zero time invariant concentrations for the CMAQ-AT model, representing the background atmosphere (Byun and Ching, 1999). IC and BC can have a significant effect on predicted ambient concentrations, but it is not expected to significantly affect the results of the emission sources scenarios, since the analyzed area is located in the center of the modeling domain, the runs were set to start five days early, and the difference approach performed in this study allows to have better results.

3.8   PHOTOLYSIS RATES

Many chemical reactions in the atmosphere are initiated by the photodissociation of numerous trace gases. In order to accurately model and predict the effects of air pollution, good photodissociation reaction rate (or photolysis rate) estimates must be made. Photodissociation is the conversion of solar radiation into chemical energy to activate and dissociate chemical species. Photolysis reactions play an important role in air quality modeling for air toxics, principally on formaldehyde, acetaldehyde, and acrolein. The photolysis rates processor (JPROC) in CMAQ calculates the photolysis rates for the required species. The extraterrestrial radiation data and the latest available oxygen and

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ozone absorption cross-section data, which corresponded to the default CMAQ data from

NASA for the year 1994, were used. The total vertical ozone column depth data for the

year 2003 was obtained from the Total Ozone Mapping Spectrometer (TOMS) (NASA,

2005). Then the estimated vertical ozone profiles were normalized and uniformly

rescaled to match the TOMS total ozone column data through JPROC (Byun and Ching,

1999).

3.9   CMAQ MODEL RUNS

3.9.1 Base Case Modeling

The methodology consisted of running the CMAQ 4.3 and CMAQ-AT with all sources

included. The default set of boundary conditions available in CMAQ was used for the 36

km domain run. Since the first day of each month under consideration was the 1st, the

model runs were set to start five days earlier to allow for the "spin-up" period over each

month. This is to avoid the influence of the initial conditions on the model results. These

results were used to assess the modeling performance, analyze the spatial distribution in

the Southeaster of the U.S., and temporal patterns of air toxics and EC exposure in

Nashville, TN, for 2003.

Since each month included 35 days (5 days for spin-up), the base case runs for CMAQ

4.3 and CMAQ-AT were 280 runs (35 days x 4 months x 2 models).

3.9.2 Model Performance Assessment

Once the base case run was completed, the model results for the BC were compared to the monitoring data of acetaldehyde, benzene, 1,3-butadiene, formaldehyde, and EC to evaluate the model performance. This performance assessment was conducted base on the draft modeling guidance published by the U.S. EPA (U.S., EPA, 2001d). To compare the 2003 CMAQ modeled concentrations with the 2003 UATMP's monitored data for Nashville, the cell containing the monitored data was used.

The normalized bias NB of concentrations at the sites EATN and LOTN and the temperature at the Nashville International Airport were estimated using the Equation 3-2

$$NB = \frac{1}{n}\sum\left[\frac{Predicted - Observed}{Observed}\right]$$
Equation 3-2

Where n is the number of days with monitored data. The predicted concentrations that were used in the equation were actually the average 24-hr concentration predicted by the models CMAQ 4.3 and CMAQ-AT in the cell (24, 23) of the 36-km modeling domain. While, the predicted temperature that was used in the equation was the hourly temperature predicted by MM5 over the same cell. The observed concentrations and temperature were the monitored data at those sites.

The normalized gross error NG was estimated through the Equation 3-3.

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$$NG = \frac{1}{n} \sum ABS \left[\frac{Predicted - Observed}{Observed} \right]$$

Equation 3-3

Where the "ABS" stands for the absolute value of term.

To evaluate the overall model performance, 45° scatter plots and time series of modeled and observed daily concentrations were prepared for EATN and LOTN together, and hourly temperature for Nashville International Airport. For each month, 30 days were evaluated.

3.9.3 Emissions Sources Scenarios

After the model performance evaluation was completed, the specific scenarios from Tables 3-1 and 3-2 were modeled. A series of model runs with and without a specific source category were performed in order to answer the specific questions listed in the objectives. Those scenarios were estimated by SMOKE2.0 according to section 3.6. Because of secondary diesel particulate matter is formed when gaseous emissions from diesel engines interact with other compounds in the atmosphere (Ning et al., 2004), then for a health risk point of view, diesel particulate matter can be estimated base on primary PM2.5 (Conrad et al., 2005). Thus, the difference between the BC scenario and the NO DFS scenario were the DPM concentrations for the base case run, whereas the difference between the scenario YEAR 2020 and the YEAR 2020_DFS scenario were the DPM concentrations for the year 2020. The difference between the NO ONROAD_DFS

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Table 3-2 Emissions Scenarios Run in CMAQ

Year	Emission Scenario	Name Scenario	Objective
1999	All Sources Present (Base Case)	BC	Base Case air toxics exposure and health risk
1999	Base Case Without On-Road Sources	NO ONROAD	Contribution of on-road sources to air toxics exposure and health risk
1999	Base Case Without DFS	NO DFS	Contribution of DFS to DPM and health risk
1999	Base Case Without On-Road DFS	NO ONROAD_DFS	Contribution of on-road DFS to air toxics and DPM exposure and health risk
1999	Base Case Without LDVs	NO LDVs	Contribution of LDVs sources to air toxics exposure and health risk
1999	Base Case Without HDVs	NO HDVs	Contribution of HDVs sources to air toxics exposure and health risk
1999	Base Case Without Biogenic Emissions	NO BIO	Contribution of biogenic sources to air toxics exposure and health risk
2020	In effect MSATs regulation for 2020	YEAR 2020	Contribution of in effect MSATs regulations to air toxics exposure and health risk
1999	Base Case Without HDDVs	NO HDDVS	Contribution of HDVs sources to DPM exposure and health risk
1999	Base Case with CNG on LDVs	CNG on LDVs	Contribution of CNG on LDVs to air toxics exposure and health risk
1999	Base Case with CNG on all vehicles	CNG on All Vehicles	Contribution of CNG on all vehicles to air toxics exposure and health risk
2020	In effect MSATs regulation for 2020 without DFS	YEAR 2020_NODFS	Contribution of in effect MSATs regulations to DPM exposure and health risk

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scenario and the NO DFS scenario were the DPM concentrations contributed by the on-road DFS.

Whereas, the difference between the NO HDDVs scenario and the NO DFS scenario were the DPM concentrations contributed by the HDDVs. Finally, the Equation 3-1 allows estimating the DPM concentrations from the LDDVs. Since those DPM concentrations were assumed primary PM2.5, they were estimated considering the Equation 2-5 for the following primary PM2.5 Aitken and accumulation mode species defined in CMAQ 4.3 (Table 2-10): AECI, ACEJ, A25I, A25J, AORGPAI, and AORGPAJ.

DPM= 1.167*(AORGPAI + AORGPAJ) + AECI + AECJ + A25I +   Equation 3-4
      A25J

The factor 1.167 is estimated in CMAQ as the conversion factor between organic carbon to organic mass is 1.2 for primary organic aerosol emission.

This approach did not account for the PM sources that are apportioned between primary and secondary sulfate and nitrate aerosols, since there currently is no way to determine how much of the sulfate and nitrate are primary and how much secondary in the Aitken and accumulative modes on CMAQ 4.3. However, the sulfate and nitrate concentrations in the DPM emissions modeled by Diaz et al. (2005) on Atlanta, Birmingham, Nashville, Memphis, and Knoxville for summer 1999 were in average as low as 1.82% and 0.16%, respectively

Since the most of the SO_2 and part of the NOx from the diesel exhaust engines are converted to secondary sulfate and nitrate respectively, it is possible assume that the contribution of primary sulfate and nitrate on DPM concentration are approximately 1.82% and 0.16% also, respectively, depending on the sulfur content in the diesel fuel used.

Considering that the primary sulfate and nitrate contributions on DPM concentrations are small, the Equation 3-4 can be considered adequate to estimate the health risk effects and its reductions generated by the analyzed emission scenarios. In addition, the analysis approach involves considering the difference in mass DPM concentrations and health risk values among the proposed emission scenarios and the base case scenario rather than the absolute mass concentration or health risk value.

Future DPM studies could consider the CMAQ emissions mapping to see which emitted pollutants are going into the CMAQ species. One way to do this is to redo the sensitivity analysis but only zero out the diesel PM emissions and leave the diesel exhaust gas phase constant to account for the secondary sulfate and nitrate formation.

Once the scenarios were run, the model results were plotted in order to determine the exposure contribution and seasonal effect of each scenario for the area analyzed on the modeling domain.

3.9.4 Health Effects Estimation

With the cancer risk and non-cancer risk equations (Equations 2-1, 2-2, 2-3, and 2-4), the IUR and RfC values from the Table 2.8 of the Chapter 2, and the annual acetaldehyde, acrolein, benzene, 1,3-butadiene, formaldehyde, and DPM concentrations from CMAQ, the individual and cumulative cancer risk and non-cancer risk can be estimated for the base case and for each analyzed scenario described in Table 3-2. The cumulative cancer risk posed by gaseous air toxics (acetaldehyde, benzene, 1,3-butadiene, and formaldehyde) was called 4HAPs, and the cumulative cancer risk for those 4 air toxics and DPM was called 4HAPs+DPM. The cumulative non-cancer risk (Total Hazard Index) was estimated if one individual hazard ratio was higher than 1, as explained in section 2.1.5. Those cancer risk and non-cancer risk for the base case and the reductions due to the emissions scenarios were estimated for Nashville and were plotted to see the spatial distribution in the Southeastern U.S.

With the Equations 2-7 and 2-9 for lung cancer mortality, CDV mortality, and hospital admissions for asthma, and COPD chronic illness, respectively, plus their β coefficients described in section 2.1.5, and the annual DPM concentrations predicted from CMAQ, the respective risks were estimated for the base case and for each analyzed scenario described in Table 3-2 for Nashville, TN. In addition, those health risks for the base case and the reductions due to the emission scenario of 2020 were estimated and plotted to see the spatial distribution in the Southeastern U.S.

4.0   RESULTS AND DISCUSSIONS

4.1   EMISSIONS INVENTORY

The 1999 annual emissions for criteria pollutants inventory were obtained from the UTK

project (Doraiswami, 2004) for point and ammonia area sources. For the rest of the area

and non-road sources, NEI99 version 3 was used. Moreover, the HAPs emissions for

major, area, and non-road sources were obtained from the NEI99 version 3. The on-road

criteria pollutants emissions inventory was developed for the whole modeling domain

through NMIM using the same 1999 VMTs estimated by the UTK project for Tennessee

and the NEI99 version 3 VMTs for the rest of the states. The NMIM results were

compared with the UTK project and NEI99 version 3 to define the best inventory to be

used as an efficient 1999 base case scenario. Later, the selected criteria emissions

inventory was used to generate the scenarios for the year 2020 and CNG on-road

vehicles. The 1999 criteria pollutants emissions for on-road sources for the all states in

the 36 km domain are shown in Table 4-1 and Table 4-2, which list the NMIM results

versus the UTK emissions, and the NMIM results versus the NEI99 version 3 emissions

in TPY respectively before any processing by the emission processor SMOKE2.0.

Table 4-1. 1999 UTK Criteria Emission Inventory versus NMIM Emission Inventory for On-Road Sources [TPY]

FIPSS	State Name	Inventory	CO	NH3	NOX	PM10	PM25	SO2	VOC
01	Alabama	NMIM	1,376,362	5,252	166,345	4,636	3,525	5,215	123,556
		UTK	1,475,033	5,296	190,680	5,840	4,546	7,348	123,642
		Error	-6.7%	-0.8%	-12.8%	-20.6%	-22.5%	-29.0%	-0.1%
05	Arkansas	NMIM	766,692	2,867	102,969	2,963	2,307	3,048	63,414
		UTK	719,543	2,866	114,645	3,153	2,471	3,857	67,043
		Error	6.6%	0.0%	-10.2%	-6.0%	-6.6%	-21.0%	-5.4%
12	Florida	NMIM	3,333,569	14,174	415,636	12,307	9,369	17,327	334,396
		UTK	3,400,235	12,364	360,954	13,201	10,123	17,555	264,744
		Error	-2.0%	14.6%	15.1%	-6.8%	-7.4%	-1.3%	26.3%
13	Georgia	NMIM	2,438,883	9,794	317,185	9,119	6,997	10,011	217,035
		UTK	2,341,646	8,673	311,703	9,767	7,563	12,542	177,760
		Error	4.2%	12.9%	1.8%	-6.6%	-7.5%	-20.2%	22.1%
17	Illinois	NMIM	2,624,416	10,207	339,428	8,861	6,722	9,783	219,201
		UTK	1,980,899	6,323	237,178	8,964	6,828	12,667	163,610
		Error	32.5%	61.4%	43.1%	-1.2%	-1.5%	-22.8%	34.0%
18	Indiana	NMIM	1,887,019	6,912	247,266	6,796	5,268	8,593	157,635
		UTK	1,677,721	6,371	238,752	7,160	5,571	9,015	132,012
		Error	12.5%	8.5%	3.6%	-5.1%	-5.4%	-4.7%	19.4%
19	Iowa	NMIM	824,461	2,857	107,850	2,933	2,280	2,826	65,544
		UTK	789,271	2,925	129,238	3,214	2,519	3,933	65,831
		Error	4.5%	-2.3%	-16.5%	-8.7%	-9.5%	-28.2%	-0.4%
20	Kansas	NMIM	774,719	2,732	97,061	2,664	2,058	2,844	60,022
		UTK	698,030	2,770	110,646	2,877	2,241	3,612	57,358
		Error	11.0%	-1.4%	-12.3%	-7.4%	-8.2%	-21.3%	4.6%
21	Kentucky	NMIM	1,241,509	4,705	169,629	4,762	3,706	5,944	100,412
		UTK	1,097,666	4,245	178,585	4,982	3,884	6,219	88,088
		Error	13.1%	10.8%	-5.0%	-4.4%	-4.6%	-4.4%	14.0%

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Table 4-1. Continued

FIPSS	State Name	Inventory	CO	NH3	NOX	PM10	PM25	SO2	VOC
22	Louisiana	NMIM	1,050,198	4,059	138,363	4,006	3,100	4,246	93,498
		UTK	1,093,743	4,128	156,234	4,646	3,662	5,471	99,370
		Error	-4.0%	-1.7%	-11.4%	-13.8%	-15.3%	-22.4%	-5.9%
26	Michigan	NMIM	2,931,633	9,497	333,563	8,843	6,810	12,679	222,737
		UTK	2,002,324	8,050	258,996	8,753	6,629	12,002	141,929
		Error	46.4%	18.0%	28.8%	1.0%	2.7%	5.6%	56.9%
28	Mississippi	NMIM	789,184	3,408	127,375	3,840	3,040	3,521	72,596
		UTK	897,056	3,481	142,544	3,906	3,069	4,738	86,369
		Error	-12.0%	-2.1%	-10.6%	-1.7%	-1.0%	-25.7%	-15.9%
29	Missouri	NMIM	1,723,005	6,606	223,587	6,154	4,716	6,042	139,434
		UTK	1,173,670	5,887	187,123	5,839	4,414	8,386	101,879
		Error	46.8%	12.2%	19.5%	5.4%	6.8%	-28.0%	36.9%
31	Nebraska	NMIM	510,984	1,768	66,152	1,802	1,400	1,802	39,258
		UTK	444,681	1,785	79,234	1,938	1,517	2,384	36,123
		Error	14.9%	-1.0%	-16.5%	-7.0%	-7.7%	-24.4%	8.7%
36	New York	NMIM	3,361,830	12,619	408,599	10,651	8,018	9,178	263,869
		UTK	2,196,577	11,324	293,454	10,085	7,545	15,295	173,980
		Error	53.0%	11.4%	39.2%	5.6%	6.3%	-40.0%	51.7%
37	North Carolina	NMIM	2,218,901	8,668	292,084	8,341	6,433	9,147	198,327
		UTK	1,604,035	8,579	252,681	7,534	5,684	11,192	135,880
		Error	38.3%	1.0%	15.6%	10.7%	13.2%	-18.3%	46.0%
39	Ohio	NMIM	2,756,741	10,471	354,599	9,674	7,429	12,495	230,227
		UTK	2,414,855	9,468	307,473	10,325	7,982	13,345	169,692
		Error	14.2%	10.6%	15.3%	-6.3%	-6.9%	-6.4%	35.7%
40	Oklahoma	NMIM	1,109,143	4,204	144,231	4,094	3,168	5,039	93,968
		UTK	1,052,960	3,600	143,377	4,443	3,458	5,596	81,143
		Error	5.3%	16.8%	0.6%	-7.8%	-8.4%	-9.9%	15.8%

Table 4-1. Continued

FIPSS	State Name	Inventory	CO	NH3	NOX	PM10	PM25	SO2	VOC
42	Pennsylvania	NMIM	2,668,754	10,100	345,624	9,309	7,114	8,049	210,842
		UTK	1,975,095	9,440	294,301	10,358	8,058	12,764	158,774
		Error	35.1%	7.0%	17.4%	-10.1%	-11.7%	-36.9%	32.8%
45	South Carolina	NMIM	1,190,876	4,332	157,773	4,454	3,467	4,759	98,351
		UTK	1,110,152	4,358	159,062	4,744	3,715	5,829	103,975
		Error	7.3%	-0.6%	-0.8%	-6.1%	-6.7%	-18.4%	-5.4%
47	Tennessee	NMIM	1,635,155	6,278	212,291	5,894	4,530	6,478	139,693
		UTK	1,718,296	6,584	291,976	6,611	5,134	8,433	154,585
		Error	-4.8%	-4.7%	-27.3%	-10.8%	-11.7%	-23.2%	-9.6%
48	Texas	NMIM	4,793,542	20,992	648,470	18,552	14,120	21,209	462,675
		UTK	3,723,477	16,240	515,685	22,211	17,364	26,445	298,176
		Error	28.7%	29.3%	25.7%	-16.5%	-18.7%	-19.8%	55.2%
51	Virginia	NMIM	1,748,204	7,560	203,411	5,225	3,800	6,301	152,087
		UTK	1,351,135	6,899	235,046	7,508	5,843	9,274	121,018
		Error	29.4%	9.6%	-13.5%	-30.4%	-35.0%	-32.1%	25.7%
54	West Virginia	NMIM	486,404	1,887	59,809	1,585	1,205	2,300	38,311
		UTK	465,908	1,857	81,882	2,106	1,657	2,542	41,648
		Error	4.4%	1.7%	-27.0%	-24.7%	-27.3%	-9.5%	-8.0%
	Total	NMIM	44,242,184	171,952	5,679,299	157,464	120,584	178,836	3,797,091
		UTK	37,404,007	153,514	5,271,450	170,165	131,478	220,444	3,044,629
		Error	**18.3%**	**12.0%**	**7.7%**	**-7.5%**	**-8.3%**	**-18.9%**	**24.7%**

Table 4-2. NEI99 version 3 Criteria Emission Inventory versus NMIM Emission Inventory for On-Road Sources [TPY]

FIPSS	State Name	Inventory	CO	NH3	NOX	PM10	PM25	SO2	VOC
01	Alabama	NMIM	1,376,362	5,252	166,345	4,636	3,525	5,215	123,556
		NEI-V3	1,412,342	5,247	163,024	4,705	3,591	6,279	121,201
		Error	-2.5%	0.1%	2.0%	-1.5%	-1.9%	-17.0%	1.9%
05	Arkansas	NMIM	766,692	2,867	102,969	2,963	2,307	3,048	63,414
		NEI-V3	798,252	2,863	101,043	3,004	2,348	3,735	64,710
		Error	-4.0%	0.1%	1.9%	-1.4%	-1.7%	-18.4%	-2.0%
12	Florida	NMIM	3,333,569	14,174	415,636	12,307	9,369	17,327	334,396
		NEI-V3	3,379,564	14,160	424,968	12,253	9,310	16,581	328,412
		Error	-1.4%	0.1%	-2.2%	0.4%	0.6%	4.5%	1.8%
13	Georgia	NMIM	2,438,883	9,794	317,185	9,119	6,997	10,011	217,035
		NEI-V3	2,526,592	9,782	313,568	9,247	7,123	12,027	207,562
		Error	-3.5%	0.1%	1.2%	-1.4%	-1.8%	-16.8%	4.6%
17	Illinois	NMIM	2,624,416	10,207	339,428	8,861	6,722	9,783	219,201
		NEI-V3	2,680,827	10,195	319,326	9,015	6,874	12,085	214,399
		Error	-2.1%	0.1%	6.3%	-1.7%	-2.2%	-19.0%	2.2%
18	Indiana	NMIM	1,887,019	6,912	247,266	6,796	5,268	8,593	157,635
		NEI-V3	1,917,221	6,905	234,672	6,796	5,266	8,677	150,727
		Error	-1.6%	0.1%	5.4%	0.0%	0.0%	-1.0%	4.6%
19	Iowa	NMIM	824,461	2,857	107,850	2,933	2,280	2,826	65,544
		NEI-V3	855,171	2,853	102,693	2,985	2,332	3,714	62,442
		Error	-3.6%	0.2%	5.0%	-1.7%	-2.2%	-23.9%	5.0%
20	Kansas	NMIM	774,719	2,732	97,061	2,664	2,058	2,844	60,022
		NEI-V3	768,862	2,727	93,125	2,696	2,089	3,439	58,584
		Error	0.8%	0.2%	4.2%	-1.2%	-1.5%	-17.3%	2.5%
21	Kentucky	NMIM	1,241,509	4,705	169,629	4,762	3,706	5,944	100,412
		NEI-V3	1,225,414	4,700	162,160	4,760	3,703	6,005	97,287
		Error	1.3%	0.1%	4.6%	0.0%	0.1%	-1.0%	3.2%

Table 4-2. Continued

FIPSS	State Name	Inventory	CO	NH3	NOX	PM10	PM25	SO2	VOC
22	Louisiana	NMIM	1,050,198	4,059	138,363	4,006	3,100	4,246	93,498
		NEI-V3	1,077,314	4,055	137,706	4,063	3,156	5,148	91,550
		Error	-2.5%	0.1%	0.5%	-1.4%	-1.8%	-17.5%	2.1%
26	Michigan	NMIM	2,931,633	9,497	333,563	8,843	6,810	12,679	222,737
		NEI-V3	2,845,998	9,487	311,621	8,760	6,725	11,511	212,941
		Error	3.0%	0.1%	7.0%	0.9%	1.3%	10.2%	4.6%
28	Mississippi	NMIM	789,184	3,408	127,375	3,840	3,040	3,521	72,596
		NEI-V3	830,477	3,403	126,344	3,898	3,095	4,477	74,579
		Error	-5.0%	0.2%	0.8%	-1.5%	-1.8%	-21.4%	-2.7%
29	Missouri	NMIM	1,723,005	6,606	223,587	6,154	4,716	6,042	139,434
		NEI-V3	1,670,245	6,597	215,990	6,291	4,852	8,154	138,187
		Error	3.2%	0.1%	3.5%	-2.2%	-2.8%	-25.9%	0.9%
31	Nebraska	NMIM	510,984	1,768	66,152	1,802	1,400	1,802	39,258
		NEI-V3	519,741	1,764	63,061	1,828	1,426	2,285	38,333
		Error	-1.7%	0.2%	4.9%	-1.4%	-1.8%	-21.2%	2.4%
36	New York	NMIM	3,361,830	12,619	408,599	10,651	8,018	9,178	263,869
		NEI-V3	3,372,453	12,607	388,409	11,050	8,413	14,874	260,299
		Error	-0.3%	0.1%	5.2%	-3.6%	-4.7%	-38.3%	1.4%
37	North Carolina	NMIM	2,218,901	8,668	292,084	8,341	6,433	9,147	198,327
		NEI-V3	2,252,671	8,660	285,380	8,453	6,541	10,829	187,345
		Error	-1.5%	0.1%	2.3%	-1.3%	-1.6%	-15.5%	5.9%
39	Ohio	NMIM	2,756,741	10,471	354,599	9,674	7,429	12,495	230,227
		NEI-V3	2,916,658	10,460	335,490	9,686	7,437	12,714	219,814
		Error	-5.5%	0.1%	5.7%	-0.1%	-0.1%	-1.7%	4.7%
40	Oklahoma	NMIM	1,109,143	4,204	144,231	4,094	3,168	5,039	93,968
		NEI-V3	1,122,894	4,199	139,118	4,104	3,177	5,257	95,342
		Error	-1.2%	0.1%	3.7%	-0.2%	-0.3%	-4.1%	-1.4%

Table 4-2. Continued

FIPSS	State Name	Inventory	CO	NH3	NOX	PM10	PM25	SO2	VOC
42	Pennsylvania	NMIM	2,668,754	10,100	345,624	9,309	7,114	8,049	210,842
		NEI-V3	2,754,719	10,091	332,142	9,614	7,413	12,448	211,000
		Error	-3.1%	0.1%	4.1%	-3.2%	-4.0%	-35.3%	-0.1%
45	South Carolina	NMIM	1,190,876	4,332	157,773	4,454	3,467	4,759	98,351
		NEI-V3	1,207,337	4,328	153,347	4,510	3,522	5,615	98,010
		Error	-1.4%	0.1%	2.9%	-1.2%	-1.5%	-15.2%	0.3%
47	Tennessee	NMIM	1,635,155	6,278	212,291	5,894	4,530	6,478	139,693
		NEI-V3	1,697,778	6,388	211,133	6,097	4,705	7,876	138,629
		Error	-3.7%	-1.7%	0.5%	-3.3%	-3.7%	-17.7%	0.8%
48	Texas	NMIM	4,793,542	20,992	648,470	18,552	14,120	21,209	462,675
		NEI-V3	4,954,993	20,967	647,036	18,807	14,370	25,061	465,254
		Error	-3.3%	0.1%	0.2%	-1.4%	-1.7%	-15.4%	-0.6%
51	Virginia	NMIM	1,748,204	7,560	203,411	5,225	3,800	6,301	152,087
		NEI-V3	1,894,832	7,551	195,039	5,329	3,903	7,967	148,112
		Error	-7.7%	0.1%	4.3%	-2.0%	-2.6%	-20.9%	2.7%
54	West Virginia	NMIM	486,404	1,887	59,809	1,585	1,205	2,300	38,311
		NEI-V3	495,400	1,885	56,935	1,566	1,186	2,093	37,034
		Error	-1.8%	0.2%	5.0%	1.2%	1.6%	9.9%	3.4%
	Total	NMIM	44,242,184	171,952	5,679,299	157,464	120,584	178,836	3,797,091
		NEI-V3	45,177,756	171,874	5,513,331	159,520	122,556	208,850	3,721,755
		Error	**-2.1%**	**0.0%**	**3.0%**	**-1.3%**	**-1.6%**	**-14.4%**	**2.0%**

It is important to emphasize that the on-road criteria pollutants emissions from NMIM were significantly different than those from UTK emissions, which are shown in Table 4-1. This difference was mainly because the UTK project used daily ozone season emissions to develop the annual emissions and NMIM generated annual emissions directly, as well as NMIM used 28 vehicle types instead of 16 used in the UTK project. Another difference is that the UTK project used a single diesel sulfur content value of 500 ppm for all counties, based on federal standards for diesel fuel sulfur content, while NMIM was more reasonable and used diesel sulfur values that varied by county, based on fuel survey information provided for the U.S. EPA (U.S. EPA, 2005m), which on average were less than the standard. Also, UTK project used a single set of temperature min/max for all counties and NMIM used hourly temperatures for each county. Finally, NMIM used humidity values for each county, which affect NOx significantly (U.S. EPA, 2005m), while the UTK project used default humidity values for all counties.

In general and according to Table 4-2, the total on-road emissions from NMIM for the whole domain were slightly lower than those from the NEI99 version 3, i.e., lower than 2.1 %, except for NOx and VOCs emissions, which were somewhat higher than NEI99 version 3 with 3.0 and 2.0 % respectively. In the same way, the emission differences between NMIM and NEI99 version 3 in Tennessee were similar to those from the all-modeling domain. It may be noted that both the NEI99 version 3 and NMIM run on-road estimates for every month, however, the difference is that the NEI99 version 3 used a single set of temperature min/max for all counties within a state and NMIM used hourly temperatures for each county. Another difference is that NMIM used humidity values for

each county (U.S. EPA, 2005m), while the NEI99 version 3 used default humidity values for all counties. The lowest NMIM emissions were for SO_2 emissions, which were 14.4% lower than those from the NEI99 version 3 in the whole domain and 17.7 % in Tennessee. The reason for this difference is because the SO_2 is strictly a function of fuel sulfur levels (U.S. EPA, 2002f). The NEI99 version 3 used a single diesel sulfur content value of 500 ppm for all counties of each state, based on federal standards for diesel fuel sulfur content (U.S. EPA, 2005a), and NMIM now had diesel sulfur values that varied by county, based on fuel survey information provided for the U.S. EPA (U.S. EPA, 2005m), which on average were less than the standard.

Although NMIM and NEI99 version 3 showed some differences, the on-road criteria emissions for the whole domain and Tennessee estimated by NMIM were considered accurate and good enough to be used as base case scenario and as inputs to run SMOKE2.0.

Since NMIM was estimated for criteria pollutants, it also was used to develop the base case for the on-road HAPs emissions for acetaldehyde, acrolein, benzene, 1,3-butadiene, and formaldehyde. The NEI99 version 3 was not used for HAPs emissions since they were estimated base on season-basis run on MOBILE6.2 (U.S. EPA, 2005a) instead of monthly-basis run used in this study through NMIM. In addition, the NEI99 version 3 used a single diesel sulfur content value of 500 ppm for all counties of each state instead of survey values used in NMIM (U.S. EPA, 2005m), as well as NEI99 version 3 used a single set of temperature min/max for all counties within each state and NMIM used

hourly temperatures for each county (U.S. EPA, 2005m). Finally, NMIM used humidity values for each county instead of default humidity values for all counties as used in NEI99 version 3. Those on-road HAPs emissions by state estimated by NMIM are shown in Table 4-3.

Before running the emission inventories on SMOKE2.0, the relative contribution of the criteria pollutants sources classification for Tennessee was similar to that of the emissions from all states in the domain with some small differences. However, an adjustment and improvement of the open burning area sources was done by Diaz et al., (2005) for acetaldehyde, acrolein, benzene, 1,3-butadiene, and formaldehyde in Tennessee before doing any emissions apportioning analysis and running SMOKE2.0.

4.1.1 Area Sources Improvement in Tennessee

Using more realistic activity data of acres burned, Diaz et al., (2005) found that the main open burning area sources emissions improvements in Tennessee were for wildfire sources, mainly in rural counties such as Grundy County, because for this county the estimated acetaldehyde, acrolein, benzene, 1,3-butadiene, and formaldehyde were 69 times higher than those estimated in the NEI 1999 version 3, i.e., 43.991 TPY instead of 0.6381 TPY estimated by NEI99. The information by county is shown in the Figure 4.1 (Diaz et al., 2005).

Table 4-3. 1999 HAPs Emission Inventory for On-Road Sources in [TPY] Using

NMIM Model

FIPSS	State Name	Acetaldehyde	Acrolein	Benzene	1,3-Butadiene	Formaldehyde	Total
01	Alabama	513.2	78.1	3,895.0	505.6	1,533.0	6,524.9
05	Arkansas	280.9	41.9	2,046.4	269.3	830.0	3,468.6
12	Florida	1,305.3	200.7	10,298.8	1,284.5	3,900.2	16,989.5
13	Georgia	914.1	138.9	6,604.8	879.2	2,712.8	11,249.8
17	Illinois	1,931.0	154.0	6,504.8	882.9	3,263.5	12,736.2
18	Indiana	1,006.0	107.7	5,027.1	630.6	2,215.2	8,986.6
19	Iowa	439.7	45.0	2,123.7	264.0	920.1	3,792.5
20	Kansas	290.9	41.8	2,292.1	267.3	831.7	3,723.8
21	Kentucky	492.2	69.5	3,367.2	422.2	1,449.5	5,800.5
22	Louisiana	376.6	57.1	2,895.3	360.4	1,116.6	4,805.9
26	Michigan	1,092.2	150.9	7,830.8	942.6	2,901.0	12,917.5
28	Mississippi	313.5	46.4	2,246.2	289.7	901.5	3,797.3
29	Missouri	660.2	95.8	4,720.3	598.9	1,932.8	8,008.0
31	Nebraska	232.7	27.8	1,405.7	171.9	562.2	2,400.3
36	New York	1,217.2	183.4	7,825.7	1,185.0	3,909.5	14,320.9
37	North Carolina	837.6	127.4	6,130.9	795.9	2,474.8	10,366.7
39	Ohio	1,453.0	148.6	7,054.3	870.9	3,067.5	12,594.3
40	Oklahoma	429.0	64.5	3,114.3	425.8	1,265.4	5,299.0
42	Pennsylvania	968.3	146.8	6,276.5	925.7	2,981.0	11,298.3
45	South Carolina	429.3	64.5	3,092.7	408.5	1,267.7	5,262.7
47	Tennessee	613.5	92.8	4,552.9	592.9	1,809.2	7,661.2
48	Texas	1,978.8	280.3	13,504.4	1,776.0	5,978.3	23,517.9
51	Virginia	962.0	87.6	4,139.8	592.2	2,094.9	7,876.6
54	West Virginia	152.8	22.9	1,359.7	151.7	457.1	2,144.2
	Total	**18,890.0**	**2,474.5**	**118,309.4**	**15,493.8**	**50,375.4**	**205,543.2**
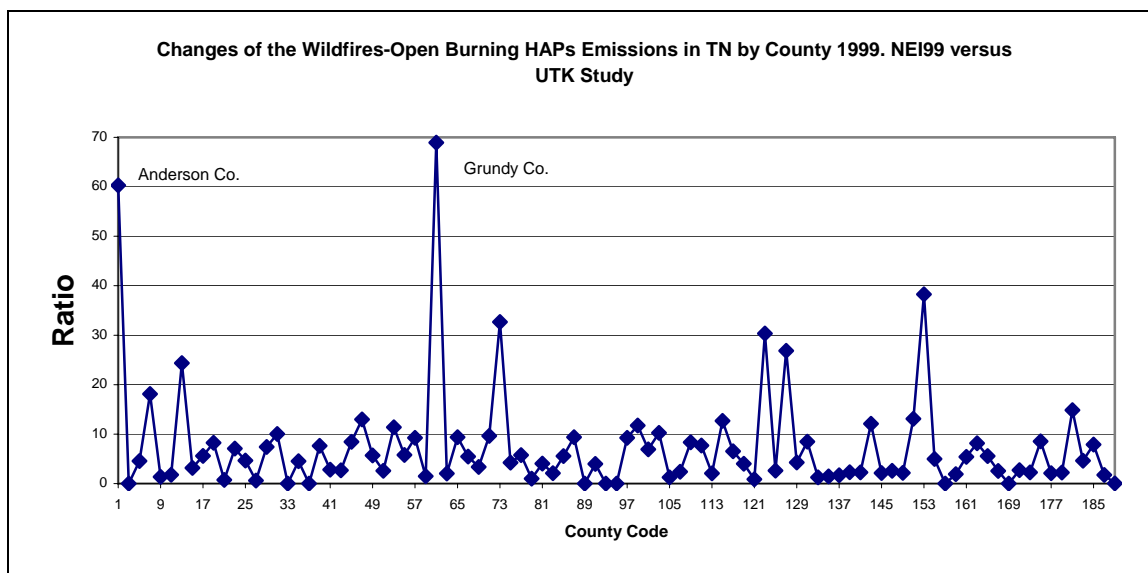
Figure 4.1. Changes of the Wildfire Open Burning HAPs Emissions in TN by County 1999, NEI99 versus UTK Study (Diaz et al., 2005)

The emissions increment for Municipal Solid Waste open burning of those air toxics occurred for rural counties, where the maximum increment was for Hardeman County with 1.15 times higher emissions than the NEI99. In general, for urban counties the Diaz et al., (2005) emissions were lower than the NEI99, where the maximum difference was for Knox County, which was 0.18 times lower than NEI99. The exceptions were for the following urban counties: Davidson, Hamilton, and Shelby counties, which showed emissions instead of zero emissions in the NEI99 (Figure 4.2).
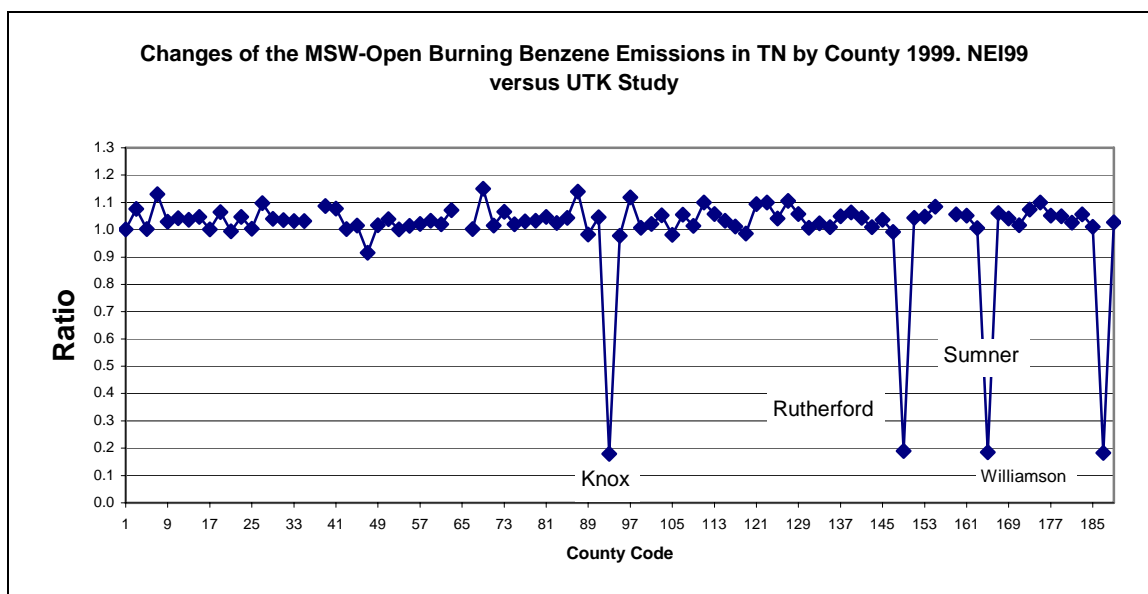
Figure 4.2. Changes of the MSW Open Burning Benzene Emissions in TN by County 1999, NEI99 versus UTK Study (Diaz et al., 2005)

The explanation of this difference is that this study used a fraction of county population that was rural from the Census bureau 1990, which was a little different of those used in the NEI99. For urban counties, the emissions were lower than those from the NEI99, since this study used a more realistic waste generated fraction that is burned of 5% for those counties instead of 28% used by NEI99 (Diaz et al., 2005). The total Open Burning emissions estimated by Diaz et al., (2005) (Wild Fires and Municipal Solid Waste Burnings) increased to 1,186 TPY instead of 833 TPY estimated by NEI99, which was 42.4 % higher than the NEI99. Important Open Burning sources that generates HAPs as yard waste and construction land clearing could be included in future emissions inventories, however, the AP-42 database (US EPA, 1996) and its expanded EIIP documents (US EPA, 2001e and f) do not have any speciated VOCs, Semi volatile

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organic compounds (SVOCs), metals, or dioxins and furans (PCDD/F) data. To solve the problem, a recent publication "Emissions of organic air toxics from open burning: a comprehensive review" written by Lemieux could be used in the future open burning emissions inventory development (Lemieux et al., 2004).

4.1.2   Emissions After the Open Burning Improvement

After improving the HAPs open burning emissions, the 1999 HAPs emissions from major, area, on-road, and non-road sources for the all States in the 36 km domain for the base case are shown in Table 4-4, which lists the emissions in TPY before any processing by the emission processor SMOKE2.0.

The emissions for each pollutant were higher in Florida and Georgia than surrounding states, which could indicate that those emissions were over estimated for Florida and Georgia or under estimated for those surrounding states in the NEI99. The respective pollutants and the fraction of emissions classified as major, area, on-road, and non-road sources for all the States in the 36 km domain, Tennessee, and improved area sources for Tennessee are shown in the Figure 4.3, Figure 4.4, and Figure 4.5.

A majority of the acetaldehyde emissions were contributed by on-road and non-road mobile sources for the all States in the domain and Tennessee, which was approximately 62.0%, followed by area and major sources with 18.8 and 17.6%, respectively.

Table 4-4. Total 1999 HAPs Anthropogenic Emissions in [TPY] on the all States in

the 36-km Domain

State Name	State Abbr.	Total Anthropogenic Emissions [TPY]						
		ACET	ACRO	BENZ	BUTA	FORM	188 HAPs	33 HAPs
Alabama	AL	1,468	387	6,936	858	4,249	103,856	17,736
Arkansas	AR	1,282	257	3,755	493	2,899	64,331	11,685
Florida	FL	5,337	2,842	23,419	4,284	22,373	275,870	67,819
Georgia	GA	3,213	898	11,414	1,752	8,312	177,221	32,812
Illinois	IL	2,910	332	9,818	1,483	7,320	171,882	30,256
Indiana	IN	1,607	199	7,766	806	3,590	132,589	20,743
Iowa	IA	918	102	3,420	418	2,027	58,138	8,692
Kansas	KS	726	97	3,799	358	2,399	42,988	9,105
Kentucky	KY	1,350	265	5,816	898	5,048	102,319	18,032
Louisiana	LA	2,312	332	6,817	807	9,931	99,919	22,636
Michigan	MI	1,938	314	13,373	1,410	7,382	200,656	34,993
Mississippi	MS	1,221	328	4,308	567	3,520	80,140	14,113
Missouri	MO	1,228	204	7,080	813	3,732	106,122	17,594
Nebraska	NE	628	110	2,145	275	1,884	34,032	5,946
New York	NY	2,140	424	14,743	5,395	6,738	203,535	40,066
North Carolina	NC	2,411	433	10,267	1,197	5,855	164,753	26,277
Ohio	OH	2,485	299	11,577	1,448	5,680	193,782	28,524
Oklahoma	OK	820	176	4,917	628	2,746	58,830	10,947
Pennsylvania	PA	1,739	306	10,768	1,255	5,348	166,124	27,973
South Carolina	SC	1,434	275	5,457	688	3,540	87,214	14,319
Tennessee	TN	1,630	241	7,164	842	3,550	135,250	18,278
Texas	TX	9,532	2,008	28,083	3,849	20,158	340,809	73,721
Virginia	VA	2,068	289	6,710	883	4,448	112,701	18,824
West Virginia	WV	475	84	2,752	241	1,296	59,731	6,187
Total		**50,872**	**11,203**	**212,304**	**31,649**	**144,023**	**3,172,790**	**577,278**
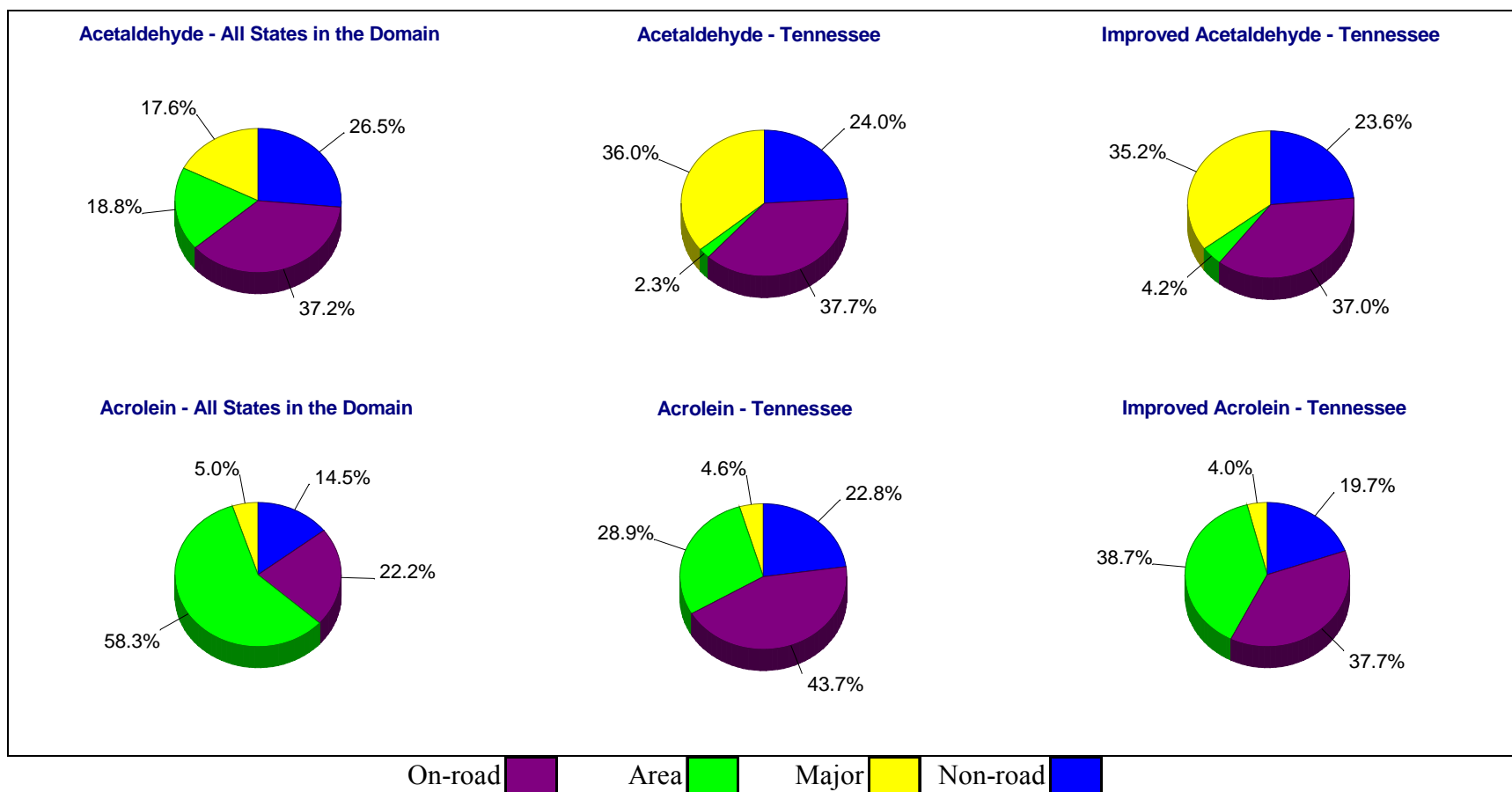
Figure 4.3. Fraction of Acetaldehyde and Acrolein Emissions from Major, Area, On-Road, and Non-Road Categories (36 km Domain and Tennessee Before and After the Open Burning Improvement Emissions) During 1999
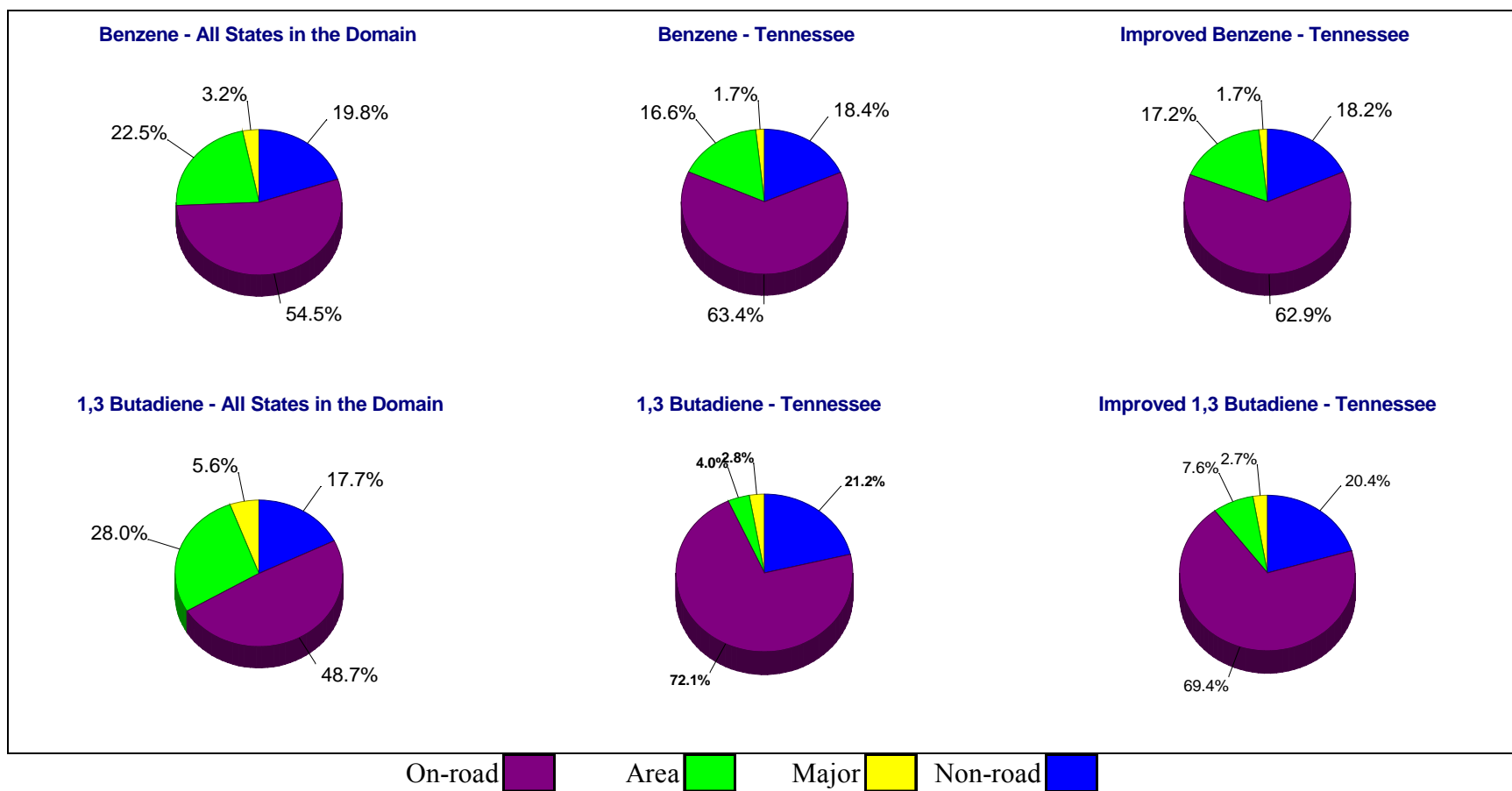
Figure 4.4. Fraction of Benzene and 1,3 Butadiene Emissions from Major, Area, On-Road, and Non-Road Categories (36 km Domain and Tennessee Before and After the Open Burning Improvement Emissions) During 1999

Formaldehyde - All States in the Domain

16.6%
21.6%
26.8%
35.0%

Formaldehyde - Tennessee

12.7%
7.3%
26.7%
53.2%

Improved Formaldehyde - Tennessee

12.0%
12.5%
25.2%
50.2%

On-road Area Major Non-road
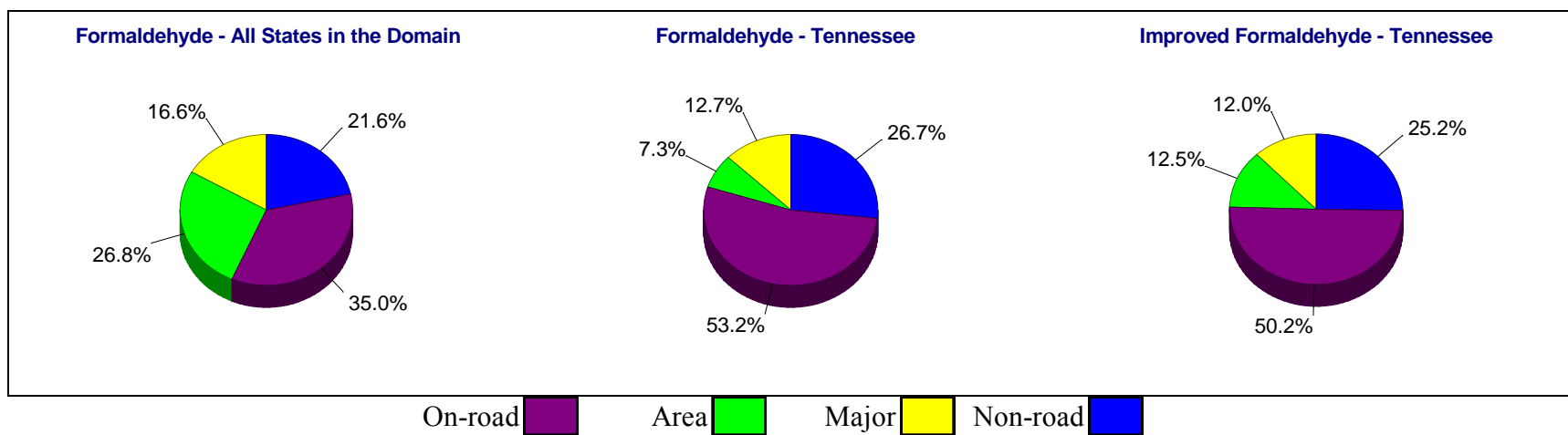
Figure 4.5. Fraction of Formaldehyde Emissions from Major, Area, On-Road, and Non-Road Categories (36 km Domain and Tennessee Before and After the Open Burning Improvement Emissions) During 1999

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However, after improving open burning emissions, area sources accounted for 4.2% in Tennessee, which could indicate that acetaldehyde area emissions were still under estimated for Tennessee. Or Florida's area emissions increased the total area sources contribution in the modeling domain, whose contribution value in Florida was as high as 43.1%.

Emissions of acrolein presented more variability over each analyzed region, whose higher contribution were due to area sources for the all States in the domain with 58.3% and followed by on-road sources with 22.2%. On the other hand and after improving the open burning emissions in Tennessee, the main acrolein sources were area sources followed by on-road sources with 38.7 and 37.7% respectively, which were significantly different from those before the open burning emissions improvement and significantly different from the all States in the domain.

The Florida's acrolein area emissions influentied the acrolein emissions in the total 24 states, increasing the total area sources contribution in the modeling domain. The area sources contribution on acrolein was as high as 86.6% in Florida.

The relative contribution of the source classifications on benzene for Tennessee was similar to that from all States in the domain with some differences, the on-road sources contributions were as high as 54.5 and 62.9 % for the whole domain and Tennessee, respectively.

Emissions of 1,3-butadiene presented different distribution among the analyzed regions, whose higher contribution were due to on-road sources on the all States in the domain, contributing 48.7 %, followed by area sources with 28.0%. In Tennessee the main 1,3 butadiene sources were on-road followed by non-road sources with 69.4 and 20.4 % respectively after the open burning emissions improvement. This significant distribution difference between all States in the domain and the Tennessee area could also indicate that the U.S. EPA's 1,3 butadiene area sources inventory for Tennessee plus the Diaz et al., (2005) improvement could still be under estimated.

The on-road sources contributed by 35.0 % of the formaldehyde, followed by area sources with 26.8 % and non-road sources with 21.6 % for the all States in the domain. This distribution was different from that of the Tennessee area, where the on-road and non-road sources contributed 50.2 and 25.2 % respectively and area sources with 12.5 %. Although, the formaldehyde area emissions were improved, this difference could indicate that U.S. EPA's formaldehyde area inventory could still be under estimated for Tennessee also. In general, the distribution in Davidson County for each pollutant, after improving the open burning emissions in Tennessee, was similar to the distribution before improving those emissions, since this county is an urban area. It is important to give emphasis to the high contribution of on-road and non-road mobile sources to each air toxic in all analyzed regions, which were as high as 89.8% for 1,3 butadiene in Tennessee, although this value may be not true, since 1,3 butadiene area sources could be under estimated in this state.

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4.1.3 Temporal Distribution of Emissions in Davidson County, Tennessee

The following analysis was focused on air toxics and some criteria pollutants species, EC and NOx, in order to compare the trends and behavior between HAPs and criteria pollutants in the base case. The NOx and EC species were selected because they are related to diesel-fueled sources (DFS). Thus, after improving the HAPs open burning emissions, processing, and merging the air toxics, criteria pollutants, and biogenic emissions by the emission processor SMOKE 2.0, the temporal distribution of area, point, on-road, non-road, and biogenic emissions for Davidson County are shown in the Figures 4.6, 4.7, 4.8, and 4.9 for acetaldehyde.
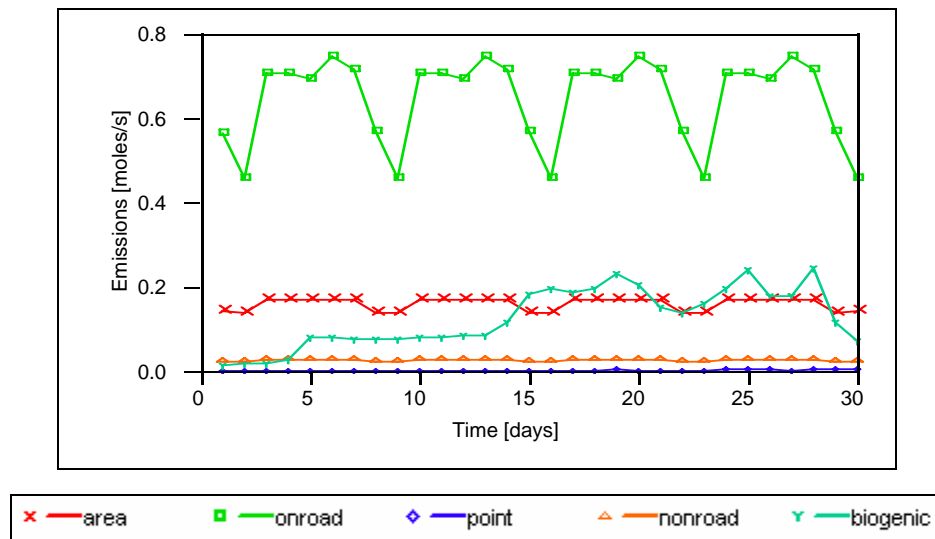
Figure 4.6. Acetaldehyde Emissions for March 1999
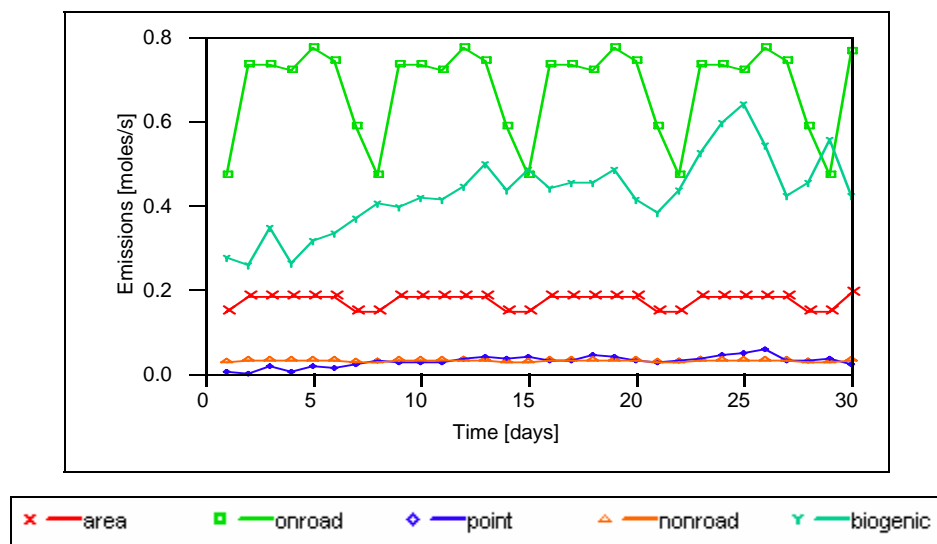
Figure 4.7. Acetaldehyde Emissions for June 1999
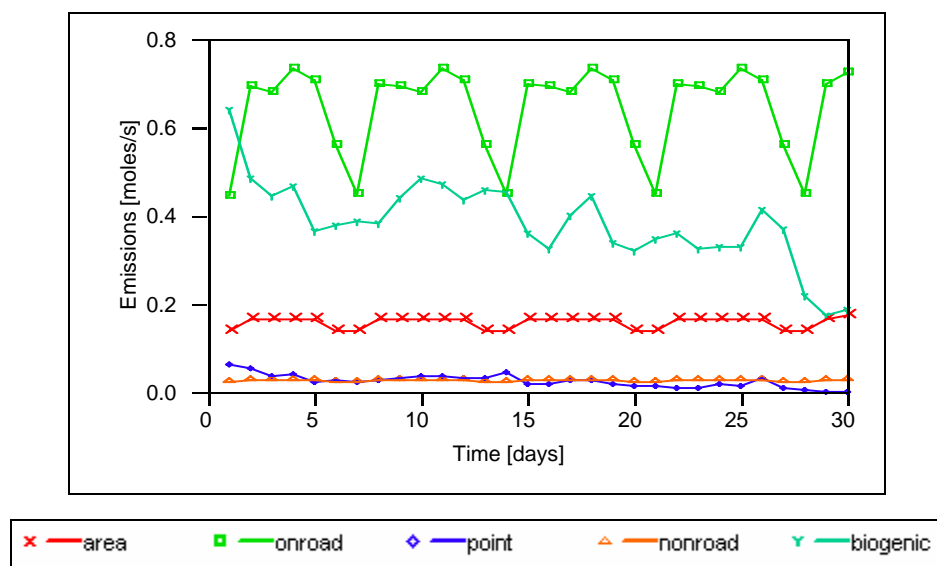
Figure 4.8. Acetaldehyde Emissions for September 1999

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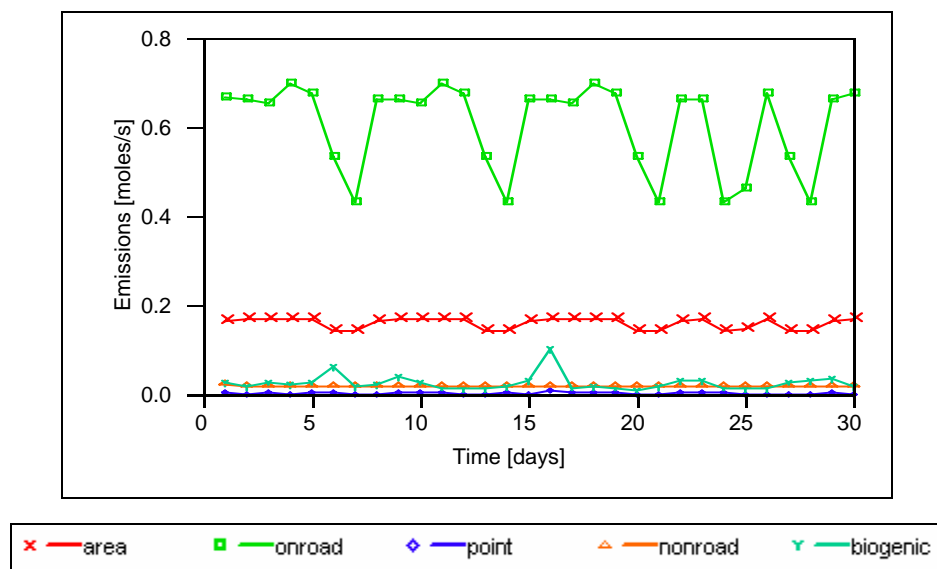
Figure 4.9. Acetaldehyde Emissions for December 1999

Area, point, on-road, and non-road sources data indicated a cyclic weekly pattern, where more emissions were generated on weekdays for most of the pollutants. In addition, June showed the highest emissions among the other analyzed months, which is explained because in the summer season the construction activity is higher than other seasons, as well as, more vehicles are on the roads due to the vacation season.

The acetaldehyde biogenic emissions increased as the summer was closer and the daily-high temperatures were higher, where more foliage there is on trees. In the same way, they declined as the cold months became evident, where most of the foliage was gone from the trees. Thus the highest biogenic emissions occurred in June and the lowest in December.

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4.1.4 Spatial Distribution of Air Toxics Emissions

Figures 4.10, 4.11, 4.12, 4.13, 4.14, 4.15, and 4.16 show the spatial distribution of merged annual average acetaldehyde, acrolein, benzene, 1,3 butadiene, formaldehyde, elemental carbon, and NOx emissions for 1999. Each plot shows that the highest emissions occurred in the Atlanta metropolitan area, Georgia, followed by Nashville (Davidson County), Memphis (Shelby County), Tennessee, and Birmingham (Jefferson County), Alabama in the Southeaster of the U.S., which had higher VMTs and non-road engines among the analyzed Southeastern counties. In addition, acrolein, 1, 3-butadiene, and formaldehyde emissions were clearly high in Florida compared with surrounding states.
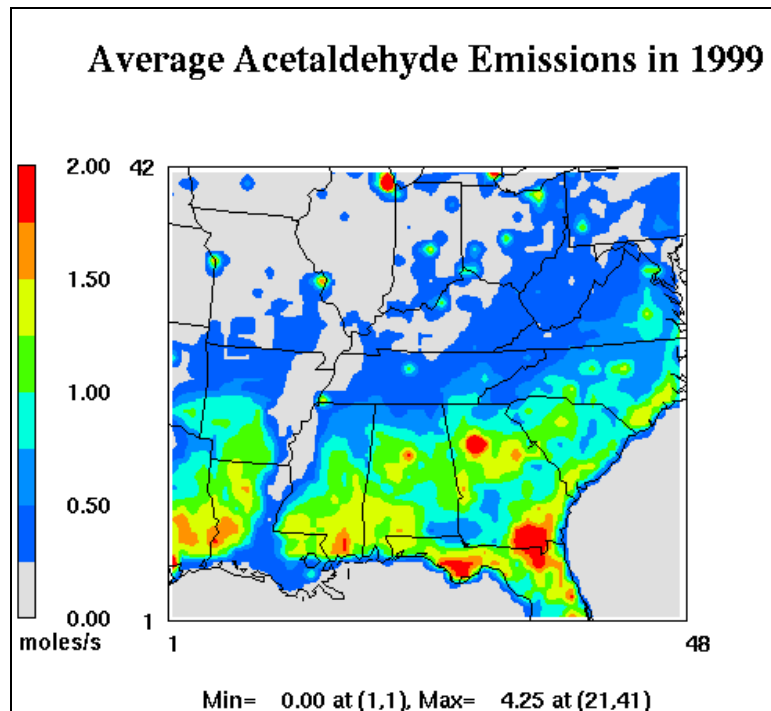
Figure 4.10. Average Acetaldehyde Emissions

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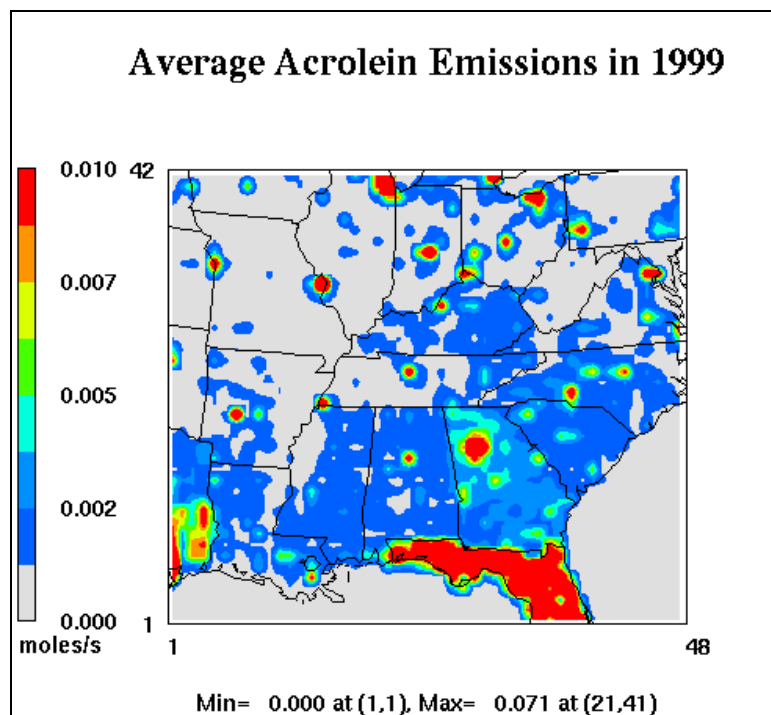
Figure 4.11. Average Acrolein Emissions
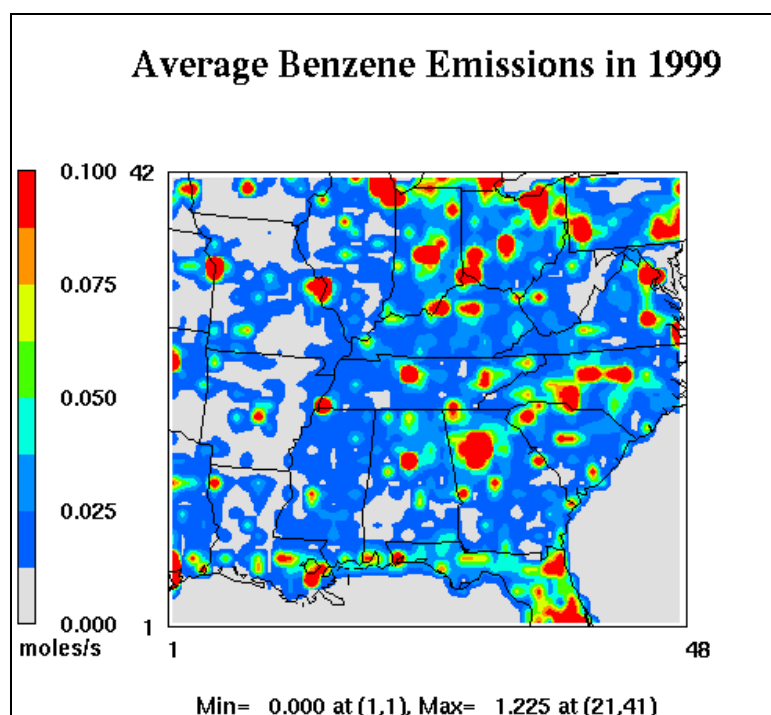
Figure 4.12. Average Benzene Emissions

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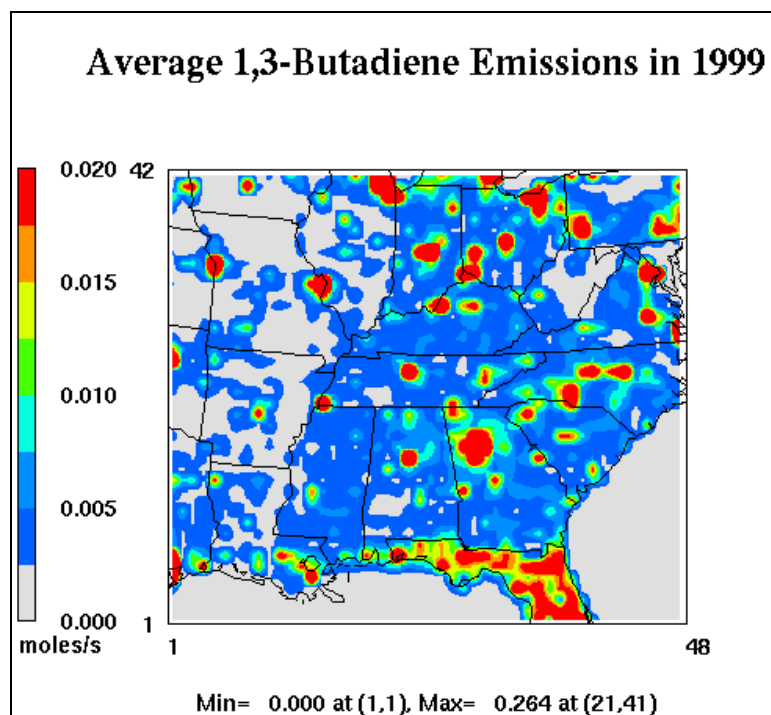
Figure 4.13. Average 1,3 Butadiene Emissions
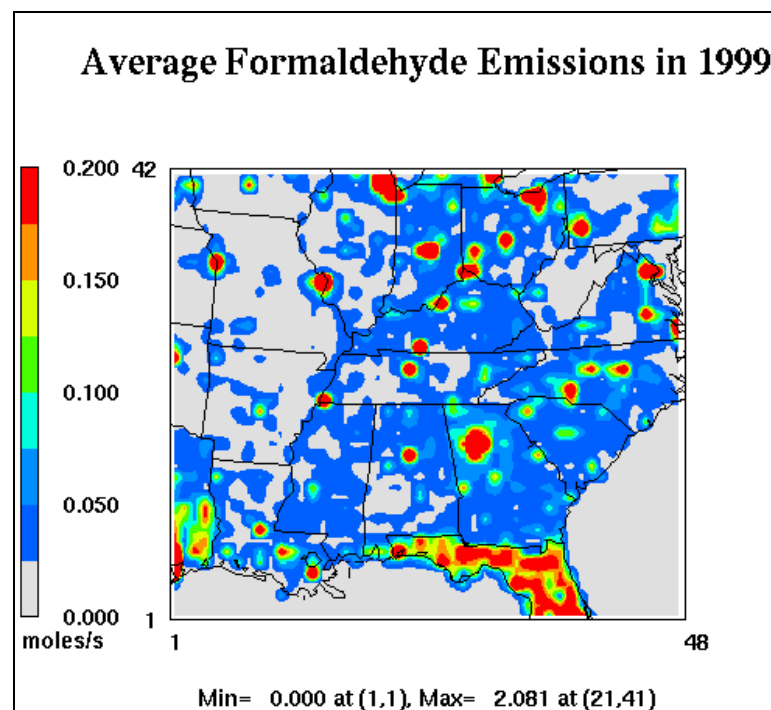
Figure 4.14. Average Formaldehyde Emissions

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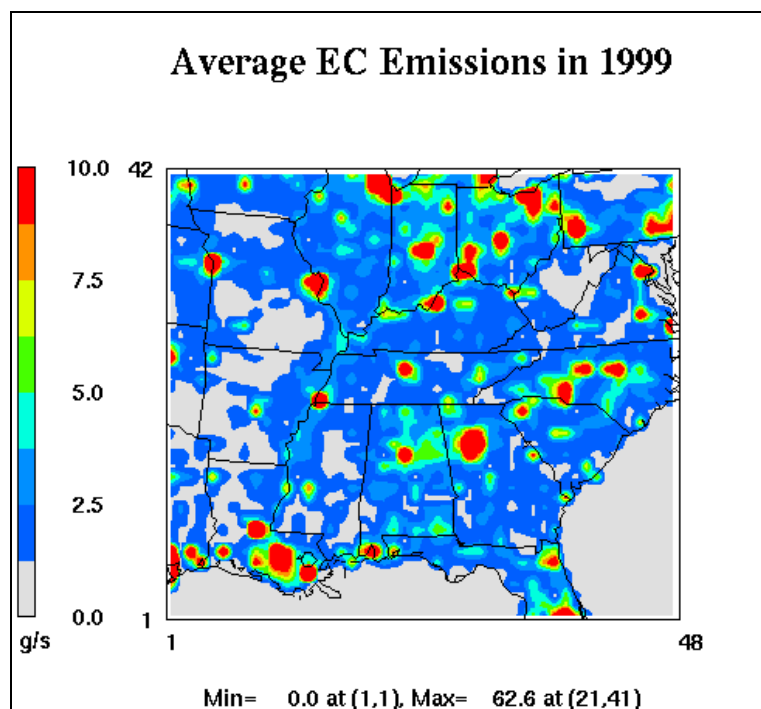
Figure 4.15. Average EC Emissions
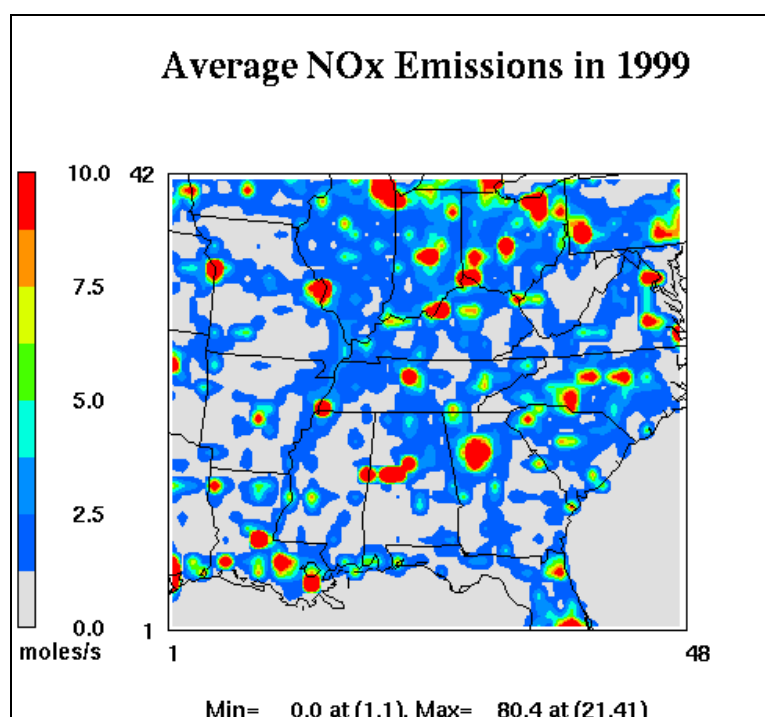
Figure 4.16. Average NOx Emissions

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Finally and according to Figure 4.17, acetaldehyde emissions were highly affected by biogenic emissions at the Southeastern U.S., which were more significant in the summer season, June and September of 1999.

4.1.5 Source Emission Scenarios

The on-road criteria pollutants and HAPs emissions inventories were run on NMIM for the year 2020 scenario and the scenarios that used CNG on LDVs and all vehicles over all States in the modeling domain. The rest of the scenarios were run on SMOKE2.0 using control matrices on SCC codes for the whole domain.

The Tennessee VMTs growth adjusted to 28 vehicle types for the scenario 2020 was obtained from the UTK project (Davis et al., 2002), which was approximately 3.1% per year. For the rest of the domain an annual average VMTs growth of 2.1% was used, which were provided by the U.S. EPA (U.S. EPA, 2004c). The VMTs and growth for each state in the domain are shown in the Table 4-5.

Each run scenario was estimated on a monthly-basis, where the diesel sulfur values varied by county and hourly temperatures and humidity values for each county were used (U.S. EPA, 2005m). The annual emissions are shown in Tables 4-6, 4-7, 4-8, and 4-9 for 2020 criteria pollutant emissions, 2020 HAPs emissions, CNG on LDVs emissions, and CNG on all vehicles, respectively.
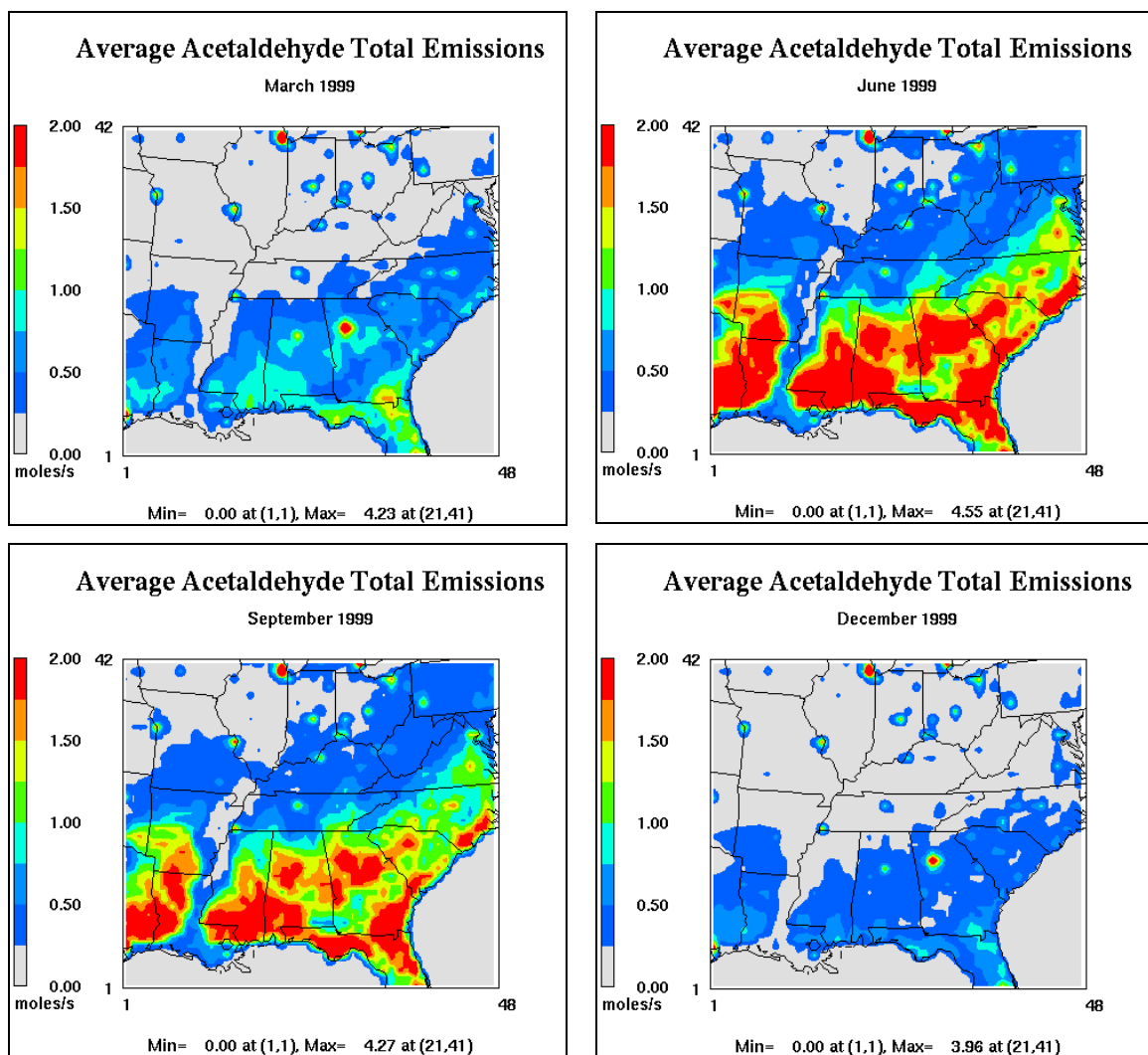
Figure 4.17. Average Merged Acetaldehyde Emissions for March, June, September, and December 1999

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Table 4-5. Annual Million VMTs by State in 1999 and 2020

FIPS	State	1999[1]	2020[2]	Total Growth	Annual Growth
1	Alabama	52,914.0	71,741.1	35.6%	1.6%
5	Arkansas	29,246.9	42,578.5	45.6%	2.1%
12	Florida	141,902.8	214,149.3	50.9%	2.3%
13	Georgia	98,859.1	159,267.1	61.1%	2.8%
17	Illinois	102,394.2	150,103.4	46.6%	2.1%
18	Indiana	70,040.6	102,051.2	45.7%	2.1%
19	Iowa	29,137.7	39,481.7	35.5%	1.6%
20	Kansas	27,699.1	39,486.7	42.6%	1.9%
21	Kentucky	47,816.1	67,236.9	40.6%	1.8%
22	Louisiana	41,204.9	58,346.0	41.6%	1.9%
26	Michigan	95,643.6	136,636.7	42.9%	1.9%
28	Mississippi	34,955.0	40,916.7	17.1%	0.8%
29	Missouri	66,735.4	97,988.1	46.8%	2.1%
31	Nebraska	18,011.0	25,247.1	40.2%	1.8%
36	New York	126,491.7	179,801.7	42.1%	1.9%
37	North Carolina	87,759.3	130,199.5	48.4%	2.2%
39	Ohio	105,486.4	151,622.7	43.7%	2.0%
40	Oklahoma	42,569.2	62,050.2	45.8%	2.1%
42	Pennsylvania	102,013.9	147,508.6	44.6%	2.0%
45	South Carolina	44,145.7	63,605.0	44.1%	2.0%
47	Tennessee	64,569.8	108,883.2	68.6%	3.1%
48	Texas	210,871.1	330,453.5	56.7%	2.6%
51	Virginia	74,162.4	105,350.6	42.1%	1.9%
54	West Virginia	18,638.0	20,554.3	10.3%	0.5%
	Total	**1,733,267.8**	**2,545,260.1**	**46.8%**	**2.1%**

(1). Source: U.S. EPA, 2005a, except for Tennessee.

(2). Source: U.S. EPA, 2004c, except for Tennessee.

Table 4-6. 2020 NMIM Criteria Emissions Inventory versus 1999 NMIM Emissions Inventory for On-Road Sources [TPY]

FIPSS	State Name	Inventory	CO	NH3	NOX	PM10	PM25	SO2	VOC
01	Alabama	1999	1,376,362	5,252	166,345	4,636	3,525	5,215	123,556
		2020	625,264	7,327	39,804	2,354	1,177	694	39,025
		Reduction	54.6%	-39.5%	76.1%	49.2%	66.6%	86.7%	68.4%
05	Arkansas	1999	766,692	2,867	102,969	2,963	2,307	3,048	63,414
		2020	365,313	4,297	24,164	1,451	733	414	21,150
		Reduction	52.4%	-49.8%	76.5%	51.0%	68.2%	86.4%	66.6%
12	Florida	1999	3,333,569	14,174	415,636	12,307	9,369	17,327	334,396
		2020	1,489,377	22,057	107,482	6,950	3,464	2,064	112,683
		Reduction	55.3%	-55.6%	74.1%	43.5%	63.0%	88.1%	66.3%
13	Georgia	1999	2,438,883	9,794	317,185	9,119	6,997	10,011	217,035
		2020	1,228,045	16,269	79,086	5,273	2,642	1,541	76,544
		Reduction	49.6%	-66.1%	75.1%	42.2%	62.2%	84.6%	64.7%
17	Illinois	1999	2,624,416	10,207	339,428	8,861	6,722	9,783	219,201
		2020	1,332,153	15,436	74,176	4,888	2,438	1,447	62,607
		Reduction	49.2%	-51.2%	78.1%	44.8%	63.7%	85.2%	71.4%
18	Indiana	1999	1,887,019	6,912	247,266	6,796	5,268	8,593	157,635
		2020	969,058	10,374	58,739	3,420	1,720	989	50,197
		Reduction	48.6%	-50.1%	76.2%	49.7%	67.4%	88.5%	68.2%
19	Iowa	1999	824,461	2,857	107,850	2,933	2,280	2,826	65,544
		2020	405,065	3,984	24,165	1,343	679	383	19,963
		Reduction	50.9%	-39.4%	77.6%	54.2%	70.2%	86.4%	69.5%
20	Kansas	1999	774,719	2,732	97,061	2,664	2,058	2,844	60,022
		2020	380,352	4,009	23,176	1,324	666	382	19,538
		Reduction	50.9%	-46.8%	76.1%	50.3%	67.6%	86.6%	67.4%
21	Kentucky	1999	1,241,509	4,705	169,629	4,762	3,706	5,944	100,412
		2020	583,306	6,814	36,577	2,268	1,143	652	30,517
		Reduction	53.0%	-44.8%	78.4%	52.4%	69.2%	89.0%	69.6%

Table 4-6. Continued

FIPSS	State Name	Inventory	CO	NH3	NOX	PM10	PM25	SO2	VOC
22	Louisiana	1999	1,050,198	4,059	138,363	4,006	3,100	4,246	93,498
		2020	482,161	5,919	31,892	1,965	989	566	30,487
		Reduction	54.1%	-45.8%	77.0%	51.0%	68.1%	86.7%	67.4%
26	Michigan	1999	2,931,633	9,497	333,563	8,843	6,810	12,679	222,737
		2020	1,461,902	13,982	82,102	4,507	2,256	1,321	70,535
		Reduction	50.1%	-47.2%	75.4%	49.0%	66.9%	89.6%	68.3%
28	Mississippi	1999	789,184	3,408	127,375	3,840	3,040	3,521	72,596
		2020	302,340	3,979	22,141	1,484	769	368	18,270
		Reduction	61.7%	-16.7%	82.6%	61.4%	74.7%	89.6%	74.8%
29	Missouri	1999	1,723,005	6,606	223,587	6,154	4,716	6,042	139,434
		2020	851,630	9,993	50,826	3,256	1,633	949	44,573
		Reduction	50.6%	-51.3%	77.3%	47.1%	65.4%	84.3%	68.0%
31	Nebraska	1999	510,984	1,768	66,152	1,802	1,400	1,802	39,258
		2020	251,618	2,538	15,289	852	430	244	12,373
		Reduction	50.8%	-43.6%	76.9%	52.7%	69.3%	86.5%	68.5%
36	New York	1999	3,361,830	12,619	408,599	10,651	8,018	9,178	263,869
		2020	1,719,041	17,640	173,860	5,839	2,896	1,618	89,945
		Reduction	48.9%	-39.8%	57.4%	45.2%	63.9%	82.4%	65.9%
37	North Carolina	1999	2,218,901	8,668	292,084	8,341	6,433	9,147	198,327
		2020	1,063,127	13,251	65,959	4,353	2,188	1,262	64,485
		Reduction	52.1%	-52.9%	77.4%	47.8%	66.0%	86.2%	67.5%
39	Ohio	1999	2,756,741	10,471	354,599	9,674	7,429	12,495	230,227
		2020	1,356,534	15,511	78,596	5,004	2,504	1,466	68,959
		Reduction	50.8%	-48.1%	77.8%	48.3%	66.3%	88.3%	70.0%
40	Oklahoma	1999	1,109,143	4,204	144,231	4,094	3,168	5,039	93,968
		2020	541,163	6,311	35,339	2,076	1,043	601	31,645
		Reduction	51.2%	-50.1%	75.5%	49.3%	67.1%	88.1%	66.3%

Table 4-6. Continued

FIPSS	State Name	Inventory	CO	NH3	NOX	PM10	PM25	SO2	VOC
42	Pennsylvania	1999	2,668,754	10,100	345,624	9,309	7,114	8,049	210,842
		2020	1,430,586	15,050	141,085	4,904	2,460	1,428	75,358
		Reduction	46.4%	-49.0%	59.2%	47.3%	65.4%	82.3%	64.3%
45	South Carolina	1999	1,190,876	4,332	157,773	4,454	3,467	4,759	98,351
		2020	576,312	6,427	36,769	2,165	1,094	619	32,622
		Reduction	51.6%	-48.4%	76.7%	51.4%	68.5%	87.0%	66.8%
47	Tennessee	1999	1,635,155	6,278	212,291	5,894	4,530	6,478	139,693
		2020	901,912	10,905	60,215	3,767	1,914	1,081	53,442
		Reduction	44.8%	-73.7%	71.6%	36.1%	57.7%	83.3%	61.7%
48	Texas	1999	4,793,542	20,992	648,470	18,552	14,120	21,209	462,675
		2020	2,377,370	33,906	156,062	10,822	5,403	3,191	157,226
		Reduction	50.4%	-61.5%	75.9%	41.7%	61.7%	85.0%	66.0%
51	Virginia	1999	1,748,204	7,560	203,411	5,225	3,800	6,301	152,087
		2020	1,003,408	11,071	97,252	3,275	1,599	993	54,998
		Reduction	42.6%	-46.4%	52.2%	37.3%	57.9%	84.2%	63.8%
54	West Virginia	1999	486,404	1,887	59,809	1,585	1,205	2,300	38,311
		2020	185,921	2,084	10,454	687	345	184	8,859
		Reduction	61.8%	-10.4%	82.5%	56.7%	71.4%	92.0%	76.9%
	Total	1999	44,242,184	171,952	5,679,299	157,464	120,584	178,836	3,797,091
		2020	21,882,956	259,134	1,525,211	84,224	42,184	24,458	1,246,002
		Reduction	**50.5%**	**-50.7%**	**73.1%**	**46.5%**	**65.0%**	**86.3%**	**67.2%**

Table 4-7. 2020 NMIM HAPs Emissions Inventory versus 1999 NMIM Emissions Inventory for On-Road Sources [TPY]

FIPSS	State Name	Inventory	Acetaldehyde	Acrolein	Benzene	1,3-Butadiene	Formaldehyde	Total
01	Alabama	1999	513.2	78.1	3,895.0	505.6	1,533.0	6,524.9
		2020	170.8	22.5	1,280.5	138.7	468.8	2,081.3
		Reduction	66.7%	71.2%	67.1%	72.6%	69.4%	68.1%
05	Arkansas	1999	280.9	41.9	2,046.4	269.3	830.0	3,468.6
		2020	99.5	13.0	708.3	78.0	270.3	1,168.9
		Reduction	64.6%	69.0%	65.4%	71.1%	67.4%	66.3%
12	Florida	1999	1,305.3	200.7	10,298.8	1,284.5	3,900.2	16,989.5
		2020	454.3	59.2	3,435.7	359.5	1,256.4	5,565.0
		Reduction	65.2%	70.5%	66.6%	72.0%	67.8%	67.2%
13	Georgia	1999	914.1	138.9	6,604.8	879.2	2,712.8	11,249.8
		2020	335.4	43.8	2,327.0	257.0	914.7	3,878.0
		Reduction	63.3%	68.4%	64.8%	70.8%	66.3%	65.5%
17	Illinois	1999	1,931.0	154.0	6,504.8	882.9	3,263.5	12,736.2
		2020	490.0	38.7	1,788.5	201.0	857.4	3,375.7
		Reduction	74.6%	74.9%	72.5%	77.2%	73.7%	73.5%
18	Indiana	1999	1,006.0	107.7	5,027.1	630.6	2,215.2	8,986.6
		2020	318.5	31.4	1,648.8	172.6	665.2	2,836.6
		Reduction	68.3%	70.8%	67.2%	72.6%	70.0%	68.4%
19	Iowa	1999	439.7	45.0	2,123.7	264.0	920.1	3,792.5
		2020	135.1	12.7	688.7	70.3	270.6	1,177.5
		Reduction	69.3%	71.6%	67.6%	73.4%	70.6%	69.0%
20	Kansas	1999	290.9	41.8	2,292.1	267.3	831.7	3,723.8
		2020	99.7	12.5	788.7	75.1	263.5	1,239.4
		Reduction	65.7%	70.1%	65.6%	71.9%	68.3%	66.7%
21	Kentucky	1999	492.2	69.5	3,367.2	422.2	1,449.5	5,800.5
		2020	153.9	19.5	1,038.7	107.7	415.8	1,735.6
		Reduction	68.7%	72.0%	69.2%	74.5%	71.3%	70.1%

Table 4-7. Continued

FIPSS	State Name	Inventory	Acetaldehyde	Acrolein	Benzene	1,3-Butadiene	Formaldehyde	Total
22	Louisiana	1999	376.6	57.1	2,895.3	360.4	1,116.6	4,805.9
		2020	134.8	17.8	1,006.8	105.4	367.1	1,631.9
		Reduction	64.2%	68.9%	65.2%	70.8%	67.1%	66.0%
26	Michigan	1999	1,092.2	150.9	7,830.8	942.6	2,901.0	12,917.5
		2020	379.4	44.9	2,663.0	263.6	913.4	4,264.4
		Reduction	65.3%	70.3%	66.0%	72.0%	68.5%	67.0%
28	Mississippi	1999	313.5	46.4	2,246.2	289.7	901.5	3,797
		2020	97.9	12.7	567.7	65.1	268.4	1,012
		Reduction	68.8%	72.6%	74.7%	77.5%	70.2%	73.4%
29	Missouri	1999	660.2	95.8	4,720.3	598.9	1,932.8	8,008.0
		2020	218.3	27.7	1,527.1	158.8	590.1	2,521.9
		Reduction	66.9%	71.1%	67.6%	73.5%	69.5%	68.5%
31	Nebraska	1999	232.7	27.8	1,405.7	171.9	562.2	2,400.3
		2020	75.5	8.2	471.9	47.3	172.9	775.7
		Reduction	67.6%	70.7%	66.4%	72.5%	69.2%	67.7%
36	New York	1999	1,217.2	183.4	7,825.7	1,185.0	3,909.5	14,320.9
		2020	551.6	56.5	2,790.3	385.9	1,190.1	4,974.4
		Reduction	54.7%	69.2%	64.3%	67.4%	69.6%	65.3%
37	North Carolina	1999	837.6	127.4	6,130.9	795.9	2,474.8	10,366.7
		2020	284.4	37.2	1,938.9	211.8	771.6	3,243.8
		Reduction	66.0%	70.8%	68.4%	73.4%	68.8%	68.7%
39	Ohio	1999	1,453.0	148.6	7,054.3	870.9	3,067.5	12,594.3
		2020	434.2	41.5	2,137.3	222.1	881.6	3,716.7
		Reduction	70.1%	72.1%	69.7%	74.5%	71.3%	70.5%
40	Oklahoma	1999	429.0	64.5	3,114.3	425.8	1,265.4	5,299.0
		2020	149.3	19.4	1,062.8	121.1	406.0	1,758.5
		Reduction	65.2%	70.0%	65.9%	71.6%	67.9%	66.8%

Table 4-7. Continued

FIPSS	State Name	Inventory	Acetaldehyde	Acrolein	Benzene	1,3-Butadiene	Formaldehyde	Total
42	Pennsylvania	1999	968.3	146.8	6,276.5	925.7	2,981.0	11,298.3
		2020	365.6	48.3	2,482.0	294.0	1,004.7	4,194.5
		Reduction	62.2%	67.1%	60.5%	68.2%	66.3%	62.9%
45	South Carolina	1999	429.3	64.5	3,092.7	408.5	1,267.7	5,262.7
		2020	151.3	19.8	1,067.2	117.8	409.3	1,765.4
		Reduction	64.8%	69.2%	65.5%	71.2%	67.7%	66.5%
47	Tennessee	1999	613.5	92.8	4,552.9	592.9	1,809.2	7,661.2
		2020	240.4	31.3	1,644.6	182.0	653.8	2,752.1
		Reduction	60.8%	66.3%	63.9%	69.3%	63.9%	64.1%
48	Texas	1999	1,978.8	280.3	13,504.4	1,776.0	5,978.3	23,517.9
		2020	679.7	83.7	4,366.8	490.6	1,848.5	7,469.2
		Reduction	65.7%	70.1%	67.7%	72.4%	69.1%	68.2%
51	Virginia	1999	962.0	87.6	4,139.8	592.2	2,094.9	7,876.6
		2020	351.7	29.7	1,665.3	196.1	673.8	2,916.7
		Reduction	63.4%	66.1%	59.8%	66.9%	67.8%	63.0%
54	West Virginia	1999	152.8	22.9	1,359.7	151.7	457.1	2,144.2
		2020	40.8	5.4	316.3	30.4	111.5	504.4
		Reduction	73.3%	76.3%	76.7%	80.0%	75.6%	76.5%
	Total	1999	18,890.0	2,474.5	118,309.4	15,493.8	50,375.4	205,543.2
		2020	6,412.1	737.2	39,412.8	4,351.9	15,645.4	66,559.5
		Reduction	**66.1%**	**70.2%**	**66.7%**	**71.9%**	**68.9%**	**67.6%**

Table 4-8. CNG on LDVs versus 1999 NMIM Emissions for On-Road Sources in [TPY]

FIPSS	State Name	Inventory	CO	NH3	NOX	PM10	PM25	SO2	VOC
01	Alabama	1999	1,376,362	5,252	166,345	4,636	3,525	5,215	123,556
		CNG on LDVs	1,159,296	5,252	155,743	4,544	3,433	3,413	102,582
		Reduction	15.8%	0.0%	6.4%	2.0%	2.6%	34.5%	17.0%
05	Arkansas	1999	766,692	2,867	102,969	2,963	2,307	3,048	63,414
		CNG on LDVs	640,391	2,867	97,121	2,915	2,259	2,010	52,465
		Reduction	16.5%	0.0%	5.7%	1.6%	2.1%	34.1%	17.3%
12	Florida	1999	3,333,569	14,174	415,636	12,307	9,369	17,327	334,396
		CNG on LDVs	2,760,483	14,174	386,953	11,944	9,006	10,881	271,605
		Reduction	17.2%	0.0%	6.9%	2.9%	3.9%	37.2%	18.8%
13	Georgia	1999	2,438,883	9,794	317,185	9,119	6,997	10,011	217,035
		CNG on LDVs	2,052,425	9,794	298,347	8,948	6,827	6,679	179,557
		Reduction	15.8%	0.0%	5.9%	1.9%	2.4%	33.3%	17.3%
17	Illinois	1999	2,624,416	10,207	339,428	8,861	6,722	9,783	219,201
		CNG on LDVs	2,165,824	10,207	318,111	8,671	6,532	6,308	183,084
		Reduction	17.5%	0.0%	6.3%	2.1%	2.8%	35.5%	16.5%
18	Indiana	1999	1,887,019	6,912	247,266	6,796	5,268	8,593	157,635
		CNG on LDVs	1,554,027	6,912	231,681	6,635	5,107	5,418	129,995
		Reduction	17.6%	0.0%	6.3%	2.4%	3.1%	37.0%	17.5%
19	Iowa	1999	824,461	2,857	107,850	2,933	2,280	2,826	65,544
		CNG on LDVs	685,244	2,857	101,715	2,887	2,234	1,870	54,711
		Reduction	16.9%	0.0%	5.7%	1.6%	2.0%	33.8%	16.5%
20	Kansas	1999	774,719	2,732	97,061	2,664	2,058	2,844	60,022
		CNG on LDVs	610,335	2,732	85,669	2,457	1,888	1,814	47,049
		Reduction	21.2%	0.0%	11.7%	7.8%	8.2%	36.2%	21.6%
21	Kentucky	1999	1,241,509	4,705	169,629	4,762	3,706	5,944	100,412
		CNG on LDVs	1,026,467	4,705	159,448	4,658	3,602	3,781	82,802
		Reduction	17.3%	0.0%	6.0%	2.2%	2.8%	36.4%	17.5%

Table 4-8. Continued

FIPSS	State Name	Inventory	CO	NH3	NOX	PM10	PM25	SO2	VOC
22	Louisiana	1999	1,050,198	4,059	138,363	4,006	3,100	4,246	93,498
		CNG on LDVs	878,041	4,059	130,368	3,934	3,028	2,796	77,207
		Reduction	16.4%	0.0%	5.8%	1.8%	2.3%	34.2%	17.4%
26	Michigan	1999	2,931,633	9,497	333,563	8,843	6,810	12,679	222,737
		CNG on LDVs	2,433,179	9,497	310,923	8,599	6,566	7,978	184,297
		Reduction	17.0%	0.0%	6.8%	2.8%	3.6%	37.1%	17.3%
28	Mississippi	1999	789,184	3,408	127,375	3,840	3,040	3,521	72,596
		CNG on LDVs	657,550	3,408	121,090	3,788	2,987	2,427	60,016
		Reduction	16.7%	0.0%	4.9%	1.4%	1.7%	31.1%	17.3%
29	Missouri	1999	1,723,005	6,606	223,587	6,154	4,716	6,042	139,434
		CNG on LDVs	1,446,321	6,606	210,801	6,056	4,619	4,080	116,326
		Reduction	16.1%	0.0%	5.7%	1.6%	2.1%	32.5%	16.6%
31	Nebraska	1999	510,984	1,768	66,152	1,802	1,400	1,802	39,258
		CNG on LDVs	427,264	1,768	62,377	1,773	1,372	1,217	32,955
		Reduction	16.4%	0.0%	5.7%	1.6%	2.0%	32.4%	16.1%
36	New York	1999	3,361,830	12,619	408,599	10,651	8,018	9,178	263,869
		CNG on LDVs	2,793,388	12,619	383,808	10,493	7,860	6,230	223,527
		Reduction	16.9%	0.0%	6.1%	1.5%	2.0%	32.1%	15.3%
37	North Carolina	1999	2,218,901	8,668	292,084	8,341	6,433	9,147	198,327
		CNG on LDVs	1,859,298	8,668	275,070	8,187	6,279	6,120	163,403
		Reduction	16.2%	0.0%	5.8%	1.9%	2.4%	33.1%	17.6%
39	Ohio	1999	2,756,741	10,471	354,599	9,674	7,429	12,495	230,227
		CNG on LDVs	2,266,083	10,471	331,720	9,434	7,189	7,794	188,866
		Reduction	17.8%	0.0%	6.5%	2.5%	3.2%	37.6%	18.0%
40	Oklahoma	1999	1,109,143	4,204	144,231	4,094	3,168	5,039	93,968
		CNG on LDVs	924,636	4,204	135,301	4,004	3,078	3,245	77,688
		Reduction	16.6%	0.0%	6.2%	2.2%	2.9%	35.6%	17.3%

Table 4-8. Continued

FIPSS	State Name	Inventory	CO	NH3	NOX	PM10	PM25	SO2	VOC
42	Pennsylvania	1999	2,668,754	10,100	345,624	9,309	7,114	8,049	210,842
		CNG on LDVs	2,240,812	10,100	325,821	9,178	6,982	5,492	176,611
		Reduction	16.0%	0.0%	5.7%	1.4%	1.8%	31.8%	16.2%
45	South Carolina	1999	1,190,876	4,332	157,773	4,454	3,467	4,759	98,351
		CNG on LDVs	997,850	4,332	148,937	4,380	3,394	3,230	81,370
		Reduction	16.2%	0.0%	5.6%	1.6%	2.1%	32.1%	17.3%
47	Tennessee	1999	1,635,155	6,278	212,291	5,894	4,530	6,478	139,693
		CNG on LDVs	1,418,508	6,278	206,861	5,785	4,422	4,431	118,813
		Reduction	13.2%	0.0%	2.6%	1.9%	2.4%	31.6%	14.9%
48	Texas	1999	4,793,542	20,992	648,470	18,552	14,120	21,209	462,675
		CNG on LDVs	3,969,473	20,992	607,509	18,159	13,727	13,579	379,190
		Reduction	17.2%	0.0%	6.3%	2.1%	2.8%	36.0%	18.0%
51	Virginia	1999	1,748,204	7,560	203,411	5,225	3,800	6,301	152,087
		CNG on LDVs	1,433,709	7,560	187,649	5,103	3,679	3,871	126,132
		Reduction	18.0%	0.0%	7.7%	2.3%	3.2%	38.6%	17.1%
54	West Virginia	1999	486,404	1,887	59,809	1,585	1,205	2,300	38,311
		CNG on LDVs	399,585	1,887	55,903	1,546	1,166	1,423	31,280
		Reduction	17.8%	0.0%	6.5%	2.5%	3.2%	38.1%	18.4%
	Total	1999	44,242,184	171,952	5,679,299	157,464	120,584	178,836	3,797,091
		CNG on LDVs	36,800,190	171,952	5,328,927	154,077	117,236	116,087	3,141,535
		Reduction	**16.8%**	**0.0%**	**6.2%**	**2.2%**	**2.8%**	**35.1%**	**17.3%**

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Table 4-9. CNG on all Vehicles versus 1999 NMIM Emissions for On-Road Sources in [TPY]

FIPSS	State Name	Inventory	CO	NH3	NOX	PM10	PM25	SO2	VOC
01	Alabama	1999	1,376,362	5,252	166,345	4,636	3,525	5,215	123,556
		CNG on AVs	1,148,572	5,252	121,410	4,479	3,367	2,451	101,434
		Reduction	16.6%	0.0%	27.0%	3.4%	4.5%	53.0%	17.9%
05	Arkansas	1999	766,692	2,867	102,969	2,963	2,307	3,048	63,414
		CNG on AVs	634,849	2,867	72,705	2,876	2,220	1,427	51,897
		Reduction	17.2%	0.0%	29.4%	3.0%	3.8%	53.2%	18.2%
12	Florida	1999	3,333,569	14,174	415,636	12,307	9,369	17,327	334,396
		CNG on AVs	2,730,710	14,174	296,865	11,762	8,824	8,172	268,260
		Reduction	18.1%	0.0%	28.6%	4.4%	5.8%	52.8%	19.8%
13	Georgia	1999	2,438,883	9,794	317,185	9,119	6,997	10,011	217,035
		CNG on AVs	1,981,545	9,794	220,731	8,606	6,530	4,586	173,527
		Reduction	18.8%	0.0%	30.4%	5.6%	6.7%	54.2%	20.0%
17	Illinois	1999	2,624,416	10,207	339,428	8,861	6,722	9,783	219,201
		CNG on AVs	2,148,827	10,207	248,963	8,555	6,416	4,595	181,094
		Reduction	18.1%	0.0%	26.7%	3.5%	4.6%	53.0%	17.4%
18	Indiana	1999	1,887,019	6,912	247,266	6,796	5,268	8,593	157,635
		CNG on AVs	1,540,927	6,912	177,966	6,543	5,015	4,050	128,555
		Reduction	18.3%	0.0%	28.0%	3.7%	4.8%	52.9%	18.4%
19	Iowa	1999	824,461	2,857	107,850	2,933	2,280	2,826	65,544
		CNG on AVs	679,850	2,857	77,495	2,849	2,197	1,322	54,142
		Reduction	17.5%	0.0%	28.1%	2.8%	3.7%	53.2%	17.4%
20	Kansas	1999	774,719	2,732	97,061	2,664	2,058	2,844	60,022
		CNG on AVs	645,539	2,732	69,909	2,577	1,971	1,323	49,803
		Reduction	16.7%	0.0%	28.0%	3.2%	4.2%	53.5%	17.0%
21	Kentucky	1999	1,241,509	4,705	169,629	4,762	3,706	5,944	100,412
		CNG on AVs	1,017,713	4,705	121,075	4,591	3,535	2,798	81,918
		Reduction	18.0%	0.0%	28.6%	3.6%	4.6%	52.9%	18.4%

Table 4-9. Continued

FIPSS	State Name	Inventory	CO	NH3	NOX	PM10	PM25	SO2	VOC
22	Louisiana	1999	1,050,198	4,059	138,363	4,006	3,100	4,246	93,498
		CNG on AVs	869,871	4,059	97,526	3,879	2,974	1,989	76,329
		Reduction	17.2%	0.0%	29.5%	3.2%	4.1%	53.2%	18.4%
26	Michigan	1999	2,931,633	9,497	333,563	8,843	6,810	12,679	222,737
		CNG on AVs	2,413,294	9,497	243,966	8,465	6,432	5,978	182,366
		Reduction	17.7%	0.0%	26.9%	4.3%	5.6%	52.9%	18.1%
28	Mississippi	1999	789,184	3,408	127,375	3,840	3,040	3,521	72,596
		CNG on AVs	652,045	3,408	87,219	3,736	2,936	1,673	59,447
		Reduction	17.4%	0.0%	31.5%	2.7%	3.4%	52.5%	18.1%
29	Missouri	1999	1,723,005	6,606	223,587	6,154	4,716	6,042	139,434
		CNG on AVs	1,433,667	6,606	161,701	5,970	4,532	2,816	115,077
		Reduction	16.8%	0.0%	27.7%	3.0%	3.9%	53.4%	17.5%
31	Nebraska	1999	510,984	1,768	66,152	1,802	1,400	1,802	39,258
		CNG on AVs	423,815	1,768	47,581	1,748	1,346	840	32,626
		Reduction	17.1%	0.0%	28.1%	3.0%	3.9%	53.4%	16.9%
36	New York	1999	3,361,830	12,619	408,599	10,651	8,018	9,178	263,869
		CNG on AVs	2,769,673	12,619	301,442	10,359	7,726	4,276	221,116
		Reduction	17.6%	0.0%	26.2%	2.7%	3.6%	53.4%	16.2%
37	North Carolina	1999	2,218,901	8,668	292,084	8,341	6,433	9,147	198,327
		CNG on AVs	1,841,642	8,668	210,451	8,061	6,153	4,275	161,542
		Reduction	17.0%	0.0%	27.9%	3.4%	4.4%	53.3%	18.5%
39	Ohio	1999	2,756,741	10,471	354,599	9,674	7,429	12,495	230,227
		CNG on AVs	2,247,769	10,471	256,465	9,306	7,061	5,898	186,757
		Reduction	18.5%	0.0%	27.7%	3.8%	4.9%	52.8%	18.9%
40	Oklahoma	1999	1,109,143	4,204	144,231	4,094	3,168	5,039	93,968
		CNG on AVs	916,021	4,204	103,214	3,944	3,018	2,369	76,841
		Reduction	17.4%	0.0%	28.4%	3.7%	4.7%	53.0%	18.2%

Table 4-9. Continued

FIPSS	State Name	Inventory	CO	NH3	NOX	PM10	PM25	SO2	VOC
42	Pennsylvania	1999	2,668,754	10,100	345,624	9,309	7,114	8,049	210,842
		CNG on AVs	2,220,963	10,100	250,542	9,058	6,863	3,748	174,626
		Reduction	16.8%	0.0%	27.5%	2.7%	3.5%	53.4%	17.2%
45	South Carolina	1999	1,190,876	4,332	157,773	4,454	3,467	4,759	98,351
		CNG on AVs	989,186	4,332	111,413	4,311	3,325	2,219	80,475
		Reduction	16.9%	0.0%	29.4%	3.2%	4.1%	53.4%	18.2%
47	Tennessee	1999	1,635,155	6,278	212,291	5,894	4,530	6,478	139,693
		CNG on AVs	1,406,248	6,278	157,161	5,696	4,333	3,132	117,519
		Reduction	14.0%	0.0%	26.0%	3.4%	4.4%	51.7%	15.9%
48	Texas	1999	4,793,542	20,992	648,470	18,552	14,120	21,209	462,675
		CNG on AVs	3,840,456	20,992	455,063	17,532	13,192	9,759	366,592
		Reduction	19.9%	0.0%	29.8%	5.5%	6.6%	54.0%	20.8%
51	Virginia	1999	1,748,204	7,560	203,411	5,225	3,800	6,301	152,087
		CNG on AVs	1,426,214	7,560	152,823	5,044	3,619	3,000	125,151
		Reduction	18.4%	0.0%	24.9%	3.5%	4.8%	52.4%	17.7%
54	West Virginia	1999	486,404	1,887	59,809	1,585	1,205	2,300	38,311
		CNG on AVs	397,103	1,887	42,805	1,525	1,145	1,112	30,988
		Reduction	18.4%	0.0%	28.4%	3.8%	5.0%	51.6%	19.1%
	Total	1999	44,242,184	171,952	5,679,299	157,464	120,584	178,836	3,797,091
		CNG on AVs	36,376,499	171,952	4,086,494	151,472	114,731	83,807	3,098,078
		Reduction	**17.8%**	**0.0%**	**28.0%**	**3.8%**	**4.9%**	**53.1%**	**18.4%**

The 2020 on-road criteria pollutants emissions shown in Table 4-6 were significantly lower than those of 1999 for the all-domain and Tennessee runs, except ammonia emissions, which were 50.7 % higher than the year 1999 for the all States in the domain and 73.7 % higher in Tennessee with respect to the year 1999. Those NH_3 emissions in the year 2020 were higher than those of the year 1999 because the on-road regulations consider reducing the NOx emissions through a catalytic system that generates more NH_3 emissions (U.S. EPA, 2004m). Tennessee showed more NH_3 emissions increase due mainly to the highest VMTs growth in this state compared with the values provided by EPA for the rest of the states in the modeling domain.

Although the 2020 VMTs were significantly higher than those of 1999, the effect of the on-road regulations will be to reduce those total criteria pollutants emissions by 53.9 % in the whole domain and 48.6 % in Tennessee. The main reductions in all 24 states occurred for SO_2 with 86.3 %, followed by NOx with 73.1 %, VOC with 67.2%, $PM_{2.5}$ with 65.0 %, CO with 50.5 %, and PM_{10} with 46.5 %. For Tennessee, the main reductions were SO_2 with 83.3 %, followed by NOx with 71.6 %, VOC with 61.7 %, $PM_{2.5}$ with 57.7 %, CO with 44.8 %, and PM_{10} with 36.1 %. The main reduction on the whole domain was SO_2 in West Virginia with a reduction as high as 92.0 %.

According to Table 4-7, the 2020 HAPs emissions from on-road sources were significantly lower than those from 1999, which resulted in a 67.6% reduction in the whole domain, and 64.1% in Tennessee.

The maximum reduction was for 1,3-butadiene, which accounted for 71.9 % in the whole domain and 69.3 % in Tennessee. The reductions in 2020 from 1999 for acetaldehyde, acrolein, benzene, 1,3-butadiene, and formaldehyde in the whole domain were 66.1, 70.2, 66.7, 71.9, and 68.9 % respectively. Those reductions were higher than those estimated and proposed by the U.S. EPA from 1996 to 2020 (U.S. EPA, 1999b, 1999c, 2001a), which were 57, 60, 57, 64 % for acetaldehyde, benzene, 1,3-butadiene, and formaldehyde respectively in the whole nation.

The criteria pollutant emissions from the scenarios that used CNG on LDVs and CNG on all vehicles versus the base case scenario are shown in Tables 4-8 and 4-9 respectively. In general, the total emissions reductions from those CNG scenarios were not significant in the modeling domain, with 15.7 % attributed to the scenario that used CNG on LDVs and 18.9 % to the scenario that used CNG on all vehicles in 1999. The exception was SO_2, with total reductions of 35.1 % in the scenario that used CNG on LDVs and of 53.1 % in the scenario that used CNG on all vehicles, with behavior similar to Tennessee. This significant reduction was because CNG fuel has lower sulfur content than gasoline and diesel fuels. Ammonia did not present variations between the base case and the CNG scenarios through all states, since NMIM and MOBILE6.2 have not incorporated that function, as well as, those models assume similar particulate matter emission factors on NGVs as conventional vehicles. MOBILE6.2 and NMIM assume that the exhaust particulate emissions of NGVs are the same as gasoline-fueled vehicles operating on very low sulfur fuel content. This assumption was based on comparisons between NGV and gasoline vehicle hydrocarbon emission test results (U.S. EPA, 2001a). These test results

suggested that NGVs generally had equivalent or lower emissions than gasoline vehicles. Based on the similarity between hydrocarbon and particulate emission formation, the general assumption of rough equivalence between these vehicle types was extended to their particulate emission factors. The tire and brake wear emissions of NGVs were assumed to be the same as gasoline-fueled vehicles (U.S. EPA, 2002f). This would explain why there would be no PM difference between changing vehicles from gasoline to CNG in NMIM and why NGVs scenarios showed small PM_{10} and $PM_{2.5}$ reductions. Finally, particulate matter from diesel vehicles has not been tested when switching to NGVs in the model.

The VOC reductions were not significant to the NGVs when compared to the base case scenario, with 17.3 % for the scenario that used CNG on LDVs and 18.4 % for the scenario that used CNG on all vehicles, reason why it is expectable that the vapor air toxics reductions from NGVs are not significant. Unfortunately, NMIM and MOBILE6.2 do not generate HAPs emissions to NGVs; therefore, it was not possible to model those HAPs CNG scenarios on SMOKE2.0 and CMAQ. Finally, it may be noted that CNG on HDVs generated lower reductions than use CNG on LDVs, since when subtracting the total reductions of the CNG on LDVs scenario from the CNG on all vehicles scenario, the CNG on HDVs reductions were as low as 3.2%. All vehicles term includes HDVs and LDVs.

The following analysis was focused on air toxics and some criteria pollutants species, such us EC and NOx, in order to compare the reductions among HAPs and criteria

pollutants. Thus, after running each emission scenario on SMOKE version 2.0, the merged daily acetaldehyde, elemental carbon, and benzene emissions from major, area, on-road, non-road, and biogenic emissions for March, June, September, and December in Davidson County are shown in the Figures 4.18, 4.19, and 4.20.

For the acetaldehyde emissions, a seasonal pattern was evident during the year, since biogenic sources generate acetaldehyde as primary pollutant and SMOKE provided a temporal profile for those sources according to the daily temperature and the respective season. In this particulate case, the acetaldehyde biogenic emissions had a positive trend during March and June, a negative trend in September, and were almost constant in December in Davidson County, where the biogenic activity decreases in the winter season.

Also, without biogenic emissions, the positive or negative trend disappeared for each month to each scenario; only a weekly cyclic pattern remained for each time series plot. The main daily reductions occurred if there were no on-road sources in the domain, there were no LDVs sources in the domain, and the year 2020 scenarios for all months, except for June and September, where important reductions occurred if biogenic emissions were not in the domain, mainly during the weekends. For the rest of the scenarios small reductions occurred in Davidson County. Daily elemental carbon emissions performed differently than acetaldehyde, which did not have positive nor negative trends during each month.
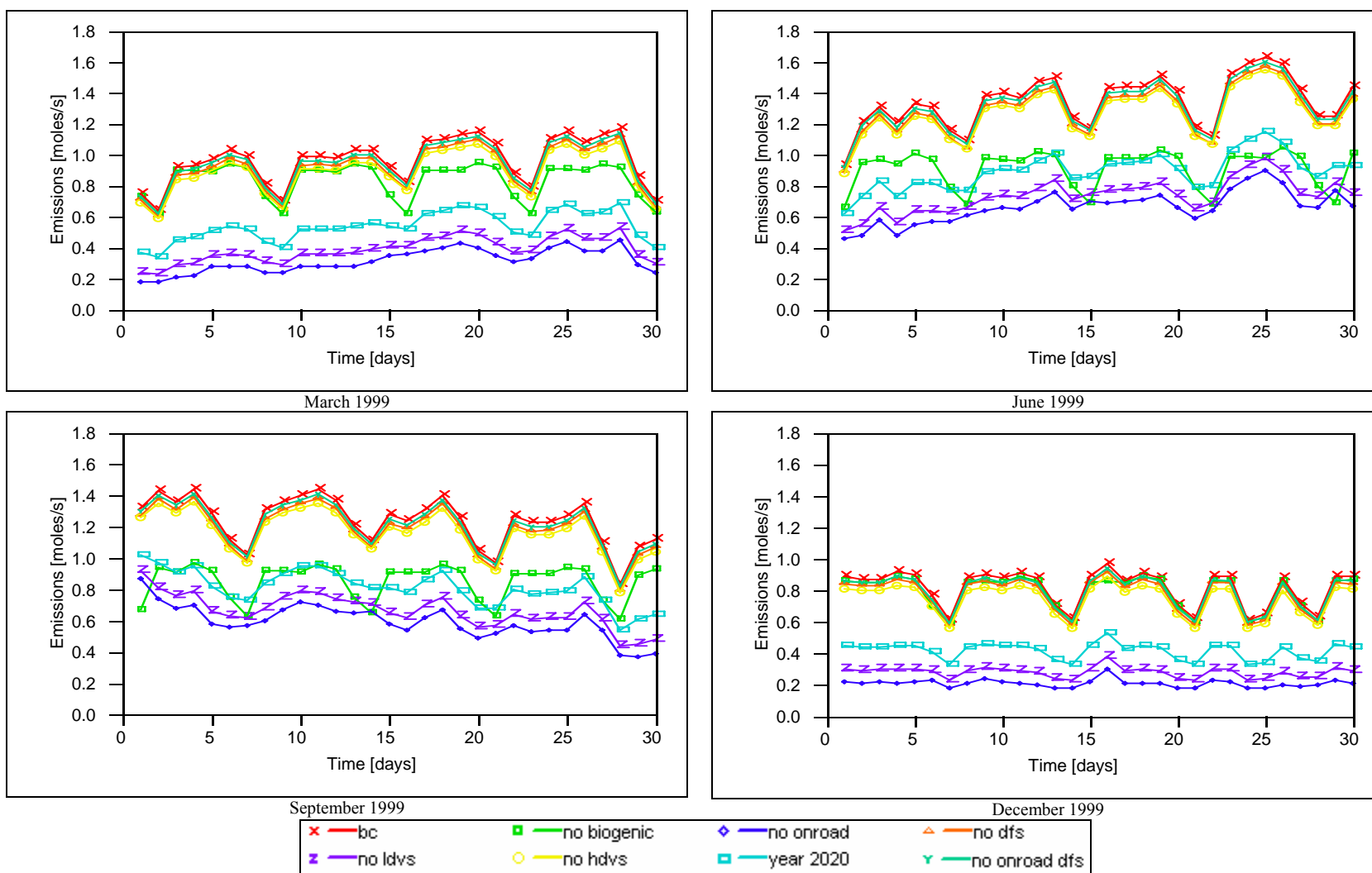
Figure 4.18. Daily Acetaldehyde Emissions in Davidson Co. for March, June, September, and December by each Scenario

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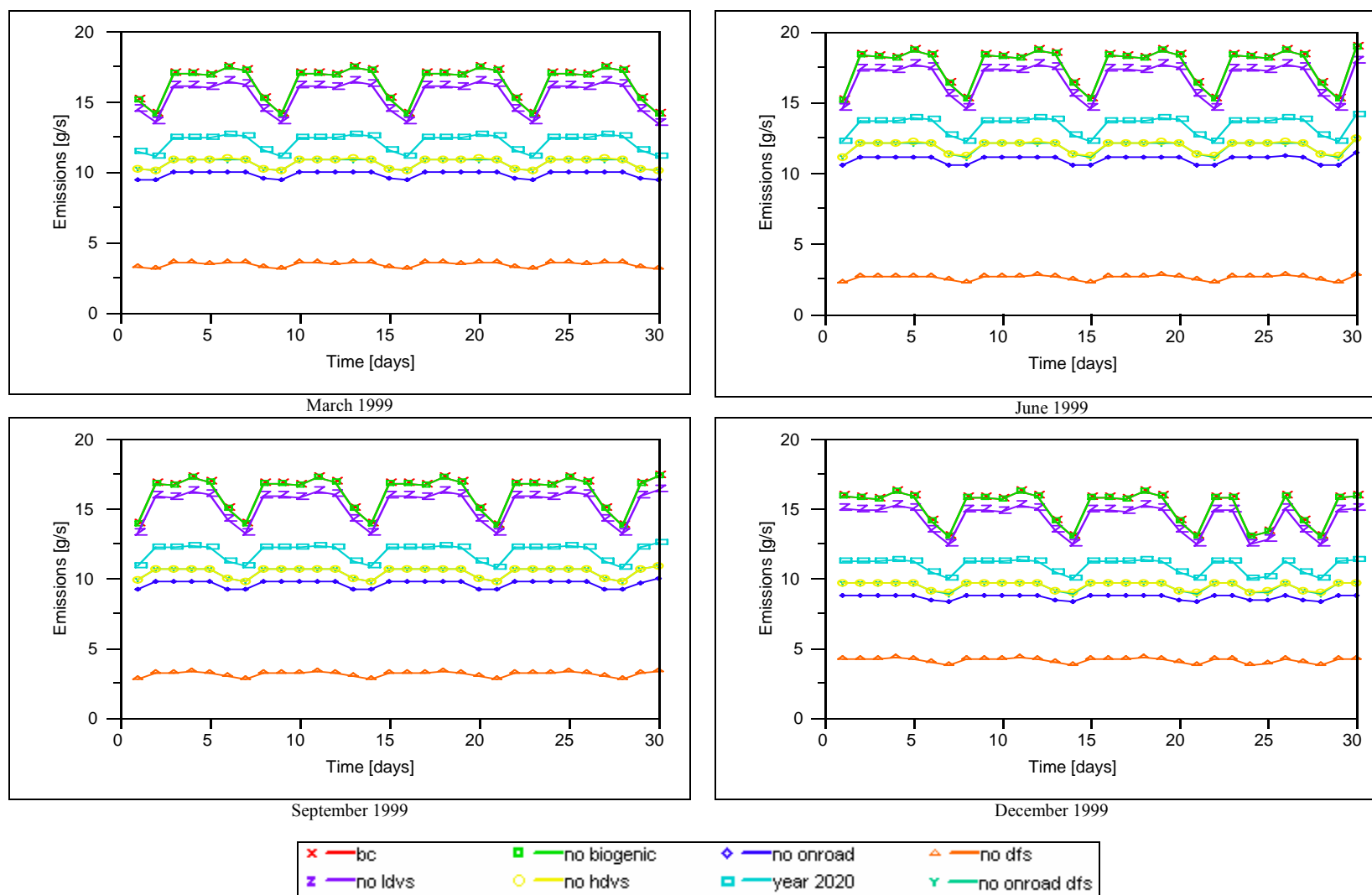
Figure 4-19. Daily EC Emissions in Davidson Co. for March, June, September, and December by each Scenario
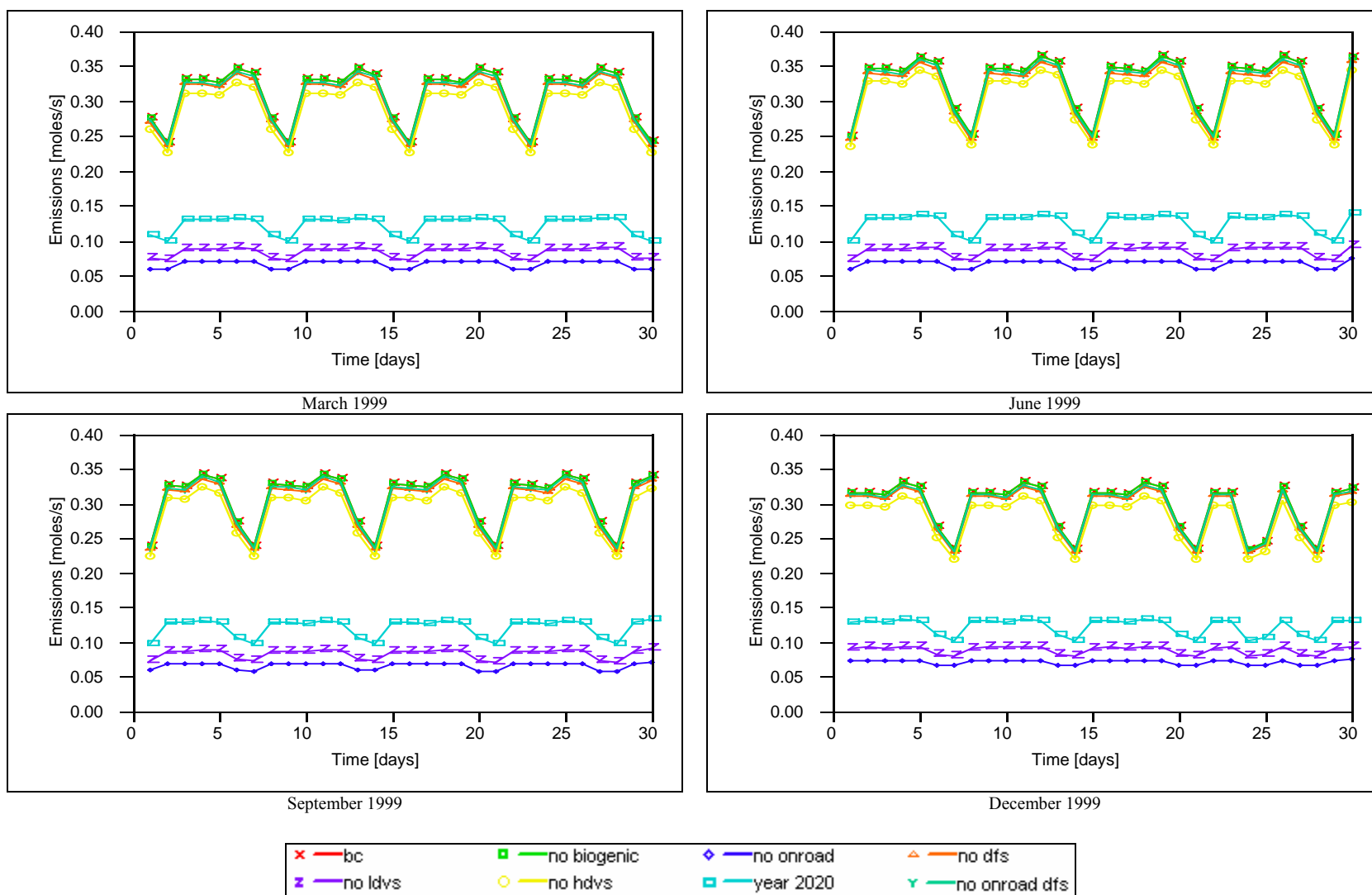
Figure 4.20. Daily Benzene Emissions in Davidson Co. for March, June, September, and December by each Scenario

The main reductions occurred when there were no diesel-fueled sources in Davidson County and they were similar through each analyzed month, followed by the scenario without on-road sources, without HDVs, and the year 2020 scenario. The cyclic SMOKE temporal profile showed that during weekends there were less EC emissions than weekdays. For the rest of the scenarios small EC emission reductions occurred in Davidson County.

Benzene emissions also did not show positive or negative trends during each analyzed month. The main reduction occurred when there were no non-road sources in Davidson County and they were similar through each month, followed by the scenario without LDVs sources, and the year 2020 scenario. For the rest of the scenarios small reductions occurred in Davidson County. The benzene time series plots showed similar patterns and reductions for acrolein, formaldehyde, 1,3 butadiene, and NOx emissions. However, the scenario without diesel-fueled sources achieved the third reduction level after the year 2020 scenario for NOx emissions. Those time series plots for Davidson County are shown in Appendix A.

The maximum daily emissions reduction for each month in Davidson County are shown in Figures 4.21, 4.22, 4.23, 4.24, 4.25, 4.26, and 4.27 for EC, acetaldehyde, acrolein, benzene, 1,3-butadiene, formaldehyde, and NOx respectively, and the annual reductions are shown in Table 4-10.
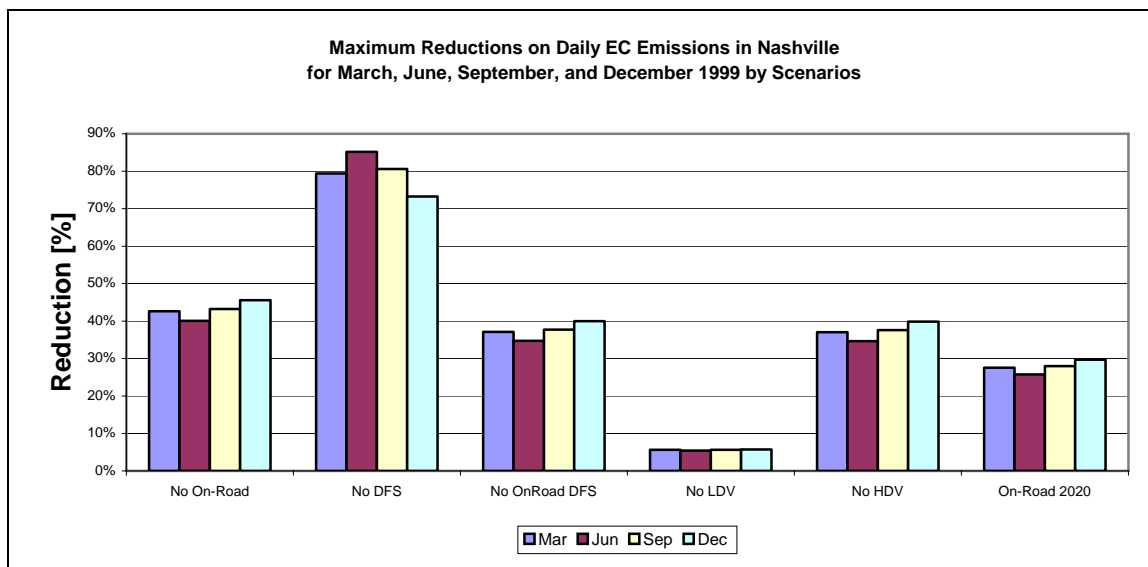
Figure 4.21. Maximum Reductions on Daily EC Emissions in Nashville for March, June, September, and December 1999 by each Scenario
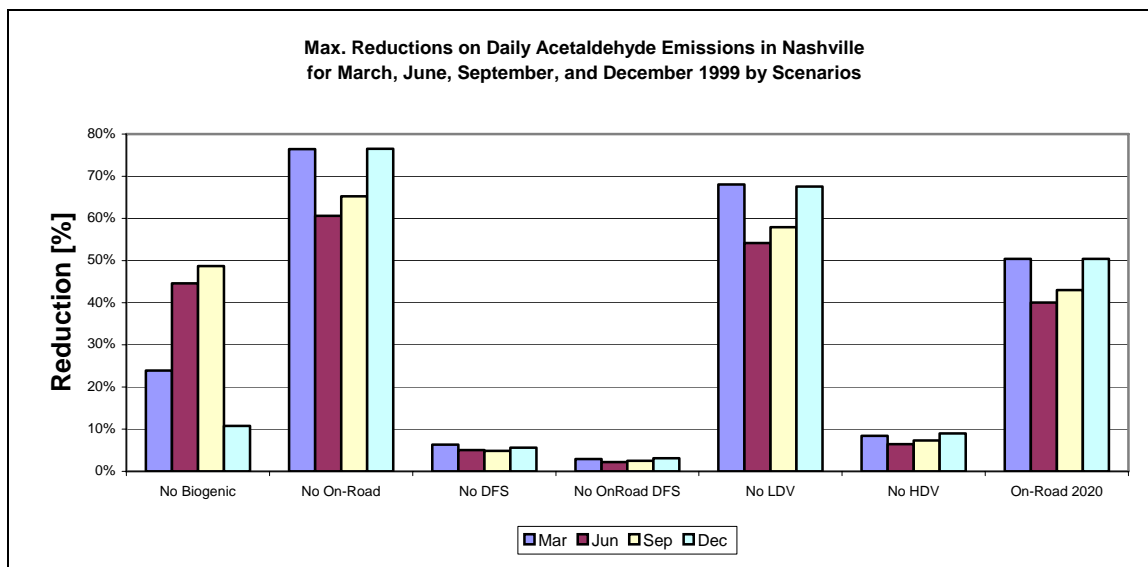
Figure 4.22. Maximum Reductions on Daily Acetaldehyde Emissions in Nashville for March, June, September, and December 1999 by each Scenario
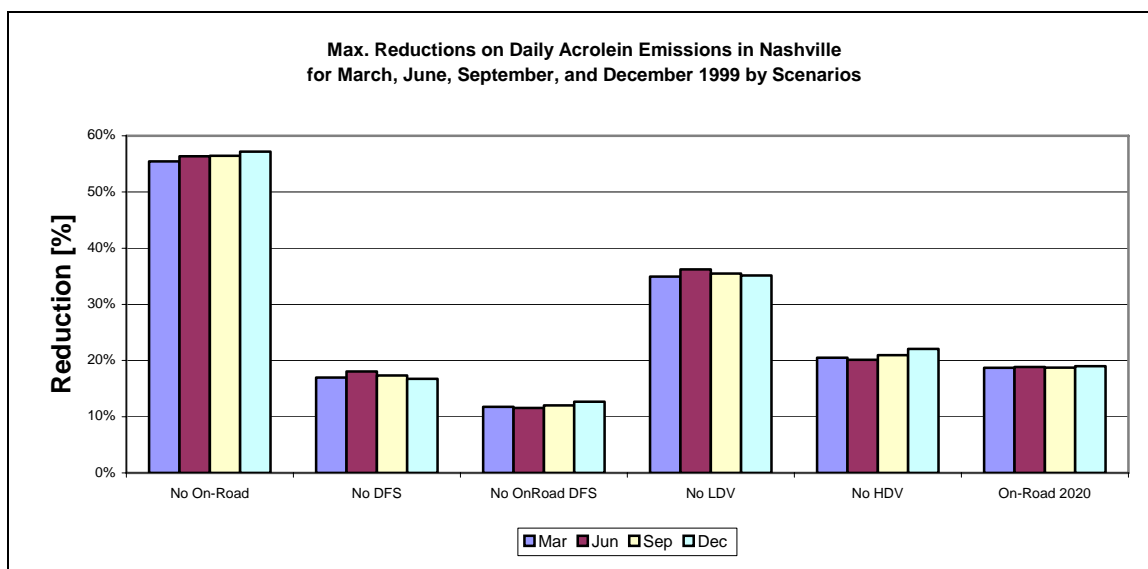
Figure 4.23. Maximum Reductions on Daily Acrolein Emissions in Nashville for

March, June, September, and December 1999 by each Scenario
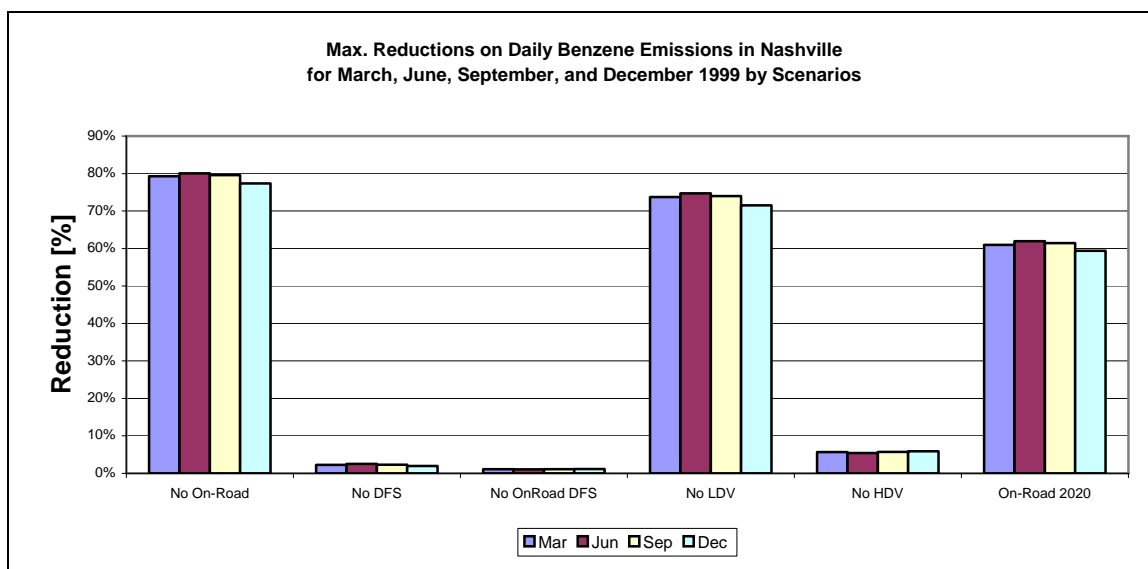
Figure 4.24. Maximum Reductions on Daily Benzene Emissions in Nashville for

March, June, September, and December 1999 by each Scenario
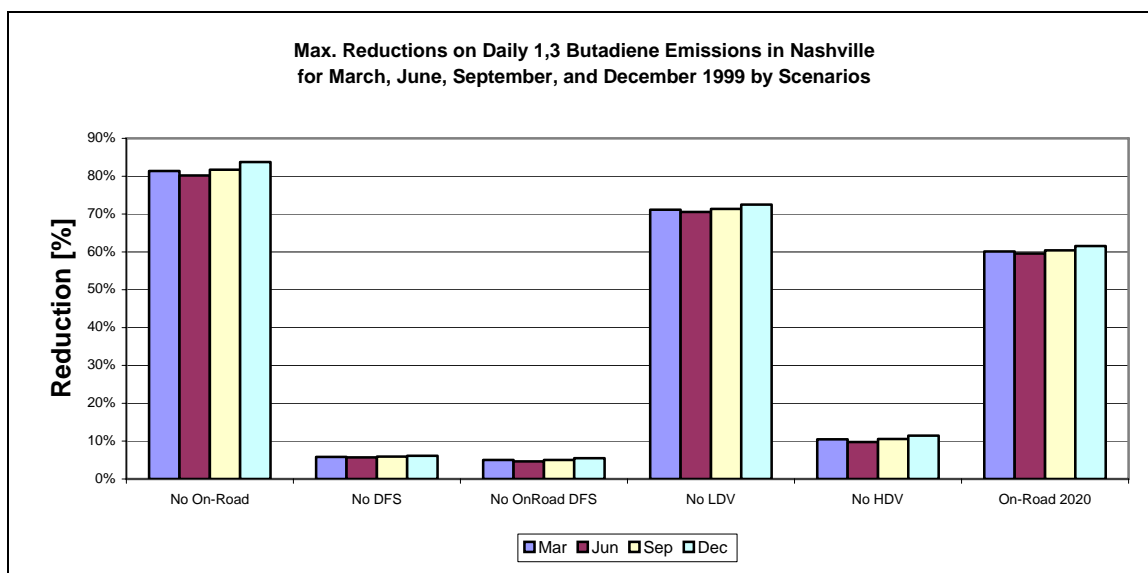
Figure 4.25. Maximum Reductions on Daily 1,3 Butadiene Emissions in Nashville

for March, June, September, and December 1999 by each Scenario
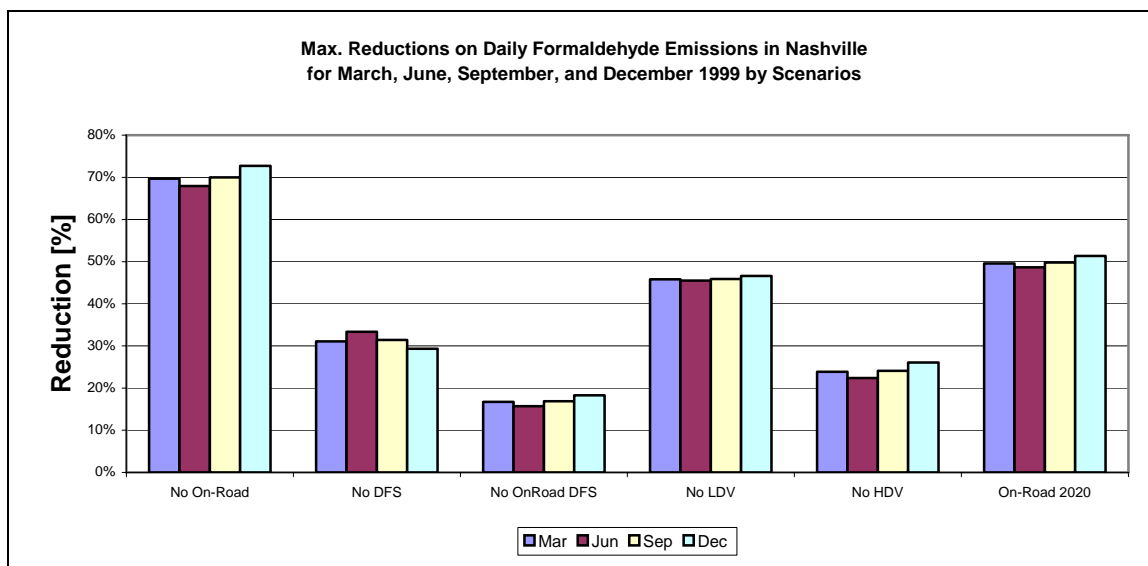
Figure 4.26. Maximum Reductions on Daily Formaldehyde Emissions in Nashville

for March, June, September, and December 1999 by each Scenario
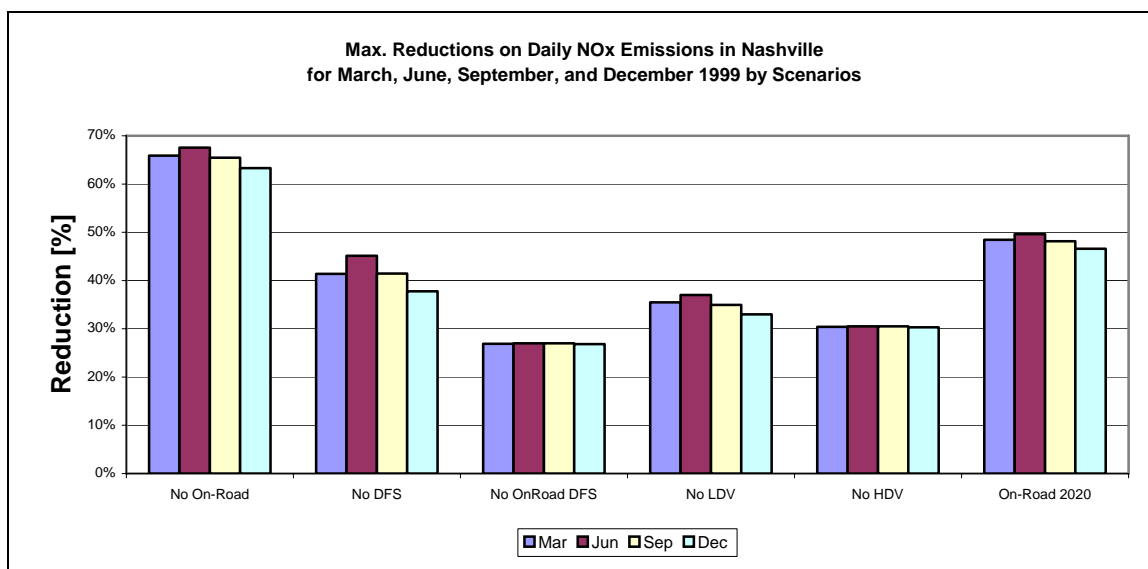
Figure 4.27. Maximum Reductions on Daily NOx Emissions in Nashville for March, June, September, and December 1999 by each Scenario

Table 4-10. Maximum Reductions on Daily Emissions in Nashville 1999

Scenario	Acrolein	Acetaldehyde	Benzene	1,3 Butadiene	Formaldehyde	EC	NOx
No Biogenic		32.0%					
No On-Road	56.3%	69.7%	79.1%	81.8%	70.1%	42.9%	65.6%
No DFS	17.3%	5.4%	2.3%	5.9%	31.3%	79.6%	41.4%
No OnRoad DFS	12.0%	2.7%	1.1%	5.1%	16.9%	37.4%	26.9%
No LDV	35.4%	61.9%	73.5%	71.4%	46.0%	5.6%	35.1%
No HDV	20.9%	7.8%	5.7%	10.6%	24.1%	37.3%	30.4%
On-Road 2020	18.8%	46.0%	60.9%	60.4%	49.8%	27.7%	48.2%

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The maximum daily reduction of EC emissions occurred in June for the hypothetic

scenario without DFS (Figure 4.21), which generate mainly EC and accounted for 85% of

reductions. During March and September the maximum reduction was 80% and

December 73%. That maximum reduction occurred in June, because in the summer

season construction activity becomes more active in Tennessee and less active in the

winter season, which includes December when the impact of wood smoke becomes

important. These DFS contributions were close to the values obtained for Zheng (Zheng

et al., 2002) where they employed a molecular marker chemical mass balance model to

apportion the sources of atmospheric particulate matter in eight cities in the Southeastern

U.S. for one-month of each season between the spring of 1999 and the winter of 2000.

The calculated value for January, April, July, and October were 74, 84, 92, and 85%

respectively. Their results demonstrated the seasonal impact of wood smoke on EC

concentrations.

During June, the maximum EC reduction due to the scenario without non-road sources

was as high as 50%, and during December only 32%, which were obtained subtracting

the on-road DFS from the DFS scenario. In addition, that maximum reduction was not

affected significantly by on-road DFS, since the amount of truck VMTs is similar in each

season. The maximum EC reduction generated by the scenario without on-road sources

was similar during each month, which was 43% on average (Table 4-10), followed by the

scenario without on-road diesel-fueled sources with an average reduction of 37%, the

scenario without HDVs with 37%, the year 2020 scenario with 28%, and the scenario

without LDVs with an average reduction of 6%. Although the scenario 2020 included the

on-road sources regulations only, the EC maximum reductions were important, but not enough to achieve a strong reduction compared with the scenario without DFS, which were close to 80% on average. In a future 2020 scenario that includes the non-road sources with all fuel and technological regulations, the maximum daily EC reduction could be important but not strong enough compared with an hypothetic scenario that not consider DFS sources.

It may be noted that the maximum reductions of daily acetaldehyde emissions for Davidson County were due to the scenario that did not consider on-road sources in the modeling domain with an average reduction of 70% (Figure 4.22 and Table 4-10), followed by the scenario without LDVs with 62%, the scenario for the year 2020 with 46%, and the scenario without biogenic emissions with an average reduction of 32%. The rest of the scenarios performed a reduction lower than 8%, mainly for those scenarios without DFS and on-road DFS, which accounted for 5 and 3% in emission reductions respectively. It indicates that LDVs were the main acetaldehyde contributors in Davidson County, mainly in March and December, where the biogenic activity is low. In fact, during June and September the reduction due to the scenario without on-road sources were 60 and 65% respectively. For months in which the reductions due to the scenario that did not consider biogenic sources, reductions were higher than March and December with 44 and 49% respectively, while March and December performed a daily maximum reduction of 22 and 10% respectively for Davidson County. Although the scenario 2020 included the on-road sources regulations only, the acetaldehyde maximum reductions

were important, but not enough to achieve a strong emissions reduction compared with the scenario without on-road sources.

The maximum daily reduction of acrolein emissions did not show a significant seasonal variation for any scenario as shown in Figure 4.23, and the highest reductions occurred for the hypothetic scenario without on-road sources, which accounted on average for 56% (Table 4-10), followed by the scenario without LDVs with 35%, the scenario without HDVs emissions with 21%, the scenario for the year 2020 with 19%, the scenario without DFS with 17%, and the scenario without on-road DFS with 12%. Although the scenario 2020 included the on-road sources regulations, the acrolein maximum reductions were not significant, since the main emissions contribution came from open burning sources, which must be strongly controlled for the Southeastern U.S.

According to Figure 4.24, no scenario showed a significant seasonal variation to the maximum daily reduction of benzene emissions in Davidson County, and the highest reductions occurred for the hypothetic scenario without on-road sources, which accounted in average for 79 % (Table 4-10), followed by the scenario without LDVs with 74 %, and the scenario for the year 2020 with 61 %. The rest of the scenarios showed reductions lower than 6 %, including the scenario without on-road DFS, which generated a reduction as low as 1 %. The greatest benzene sources were the gasoline LDVs sources and therefore, the on-road mobile sources regulations will significantly reduce the benzene emissions in 2020.

The maximum daily reductions of 1,3 butadiene emissions showed a behavior similar to the benzene emissions, where no scenario showed a significant seasonal variation in Davidson County (Figure 4.25), and the highest reductions occurred for the hypothetic scenario without on-road sources, which accounted on average for 82 % reduction (Table 4-10), followed by the scenario without LDVs with 71 %, and the scenario for the year 2020 with 60 %. The rest of the scenarios showed reductions lower than 11 %, including the scenario without on-road DFS, which generated a reduction as low as 5 %. The greatest 1,3-butadiene sources were the gasoline LDVs sources like benzene and therefore, the on-road mobile sources regulations will significantly reduce the 1,3-butadiene emissions in 2020.

It may be noted that the maximum reductions of daily formaldehyde emissions for Davidson County were due to the scenario that did not consider on-road sources in the modeling domain with an average reduction of 70% (Figure 4.26 and Table 4-10), followed by the scenario for the year 2020 with 50%, the scenario without LDVs with 46%, the scenario without DFS with 31%, the scenario without HDVs emissions with 24%, and the scenario without on-road DFS with 17%. It indicates that LDVs were the main formaldehyde contributors in Davidson County followed by HDVs, mainly in December with 47 and 26 % respectively.

Although the scenario 2020 included the on-road sources regulations only, the formaldehyde maximum reductions were significant, but not enough to achieve a strong reduction compared with the scenario without on-road sources, which was close to 70%

in average. However, it is reasonable to expect that if a future 2020 scenario includes the non-road sources with strong fuel and technological regulations, the maximum daily formaldehyde reduction could be higher than 50% for Davidson County. When subtracting the reductions from the scenario without on-road DFS from the scenario without DFS the results were that maximum reductions came from non-road sources, which were of 14% for Davidson County. Finally, according to the Figure 4.27, the maximum daily NOx reductions showed a slight seasonality, except for the scenario without on-road DFS, and the scenario without HDVs.

The maximum daily NOx reductions occurred in June, followed by March, September, and December for the scenarios without on-road sources, followed by the scenario for the year 2020, the scenario without DFS, and the scenario without LDVs. Those maximum daily NOx reductions in June were the highest since in the summer season more light duty vehicles are on-road because of vacations, and construction becomes more active at this season generating more non-road NOx emissions.

The maximum daily NOx reductions were almost the same for each month for the scenario without on-road DFS, with the source mainly by diesel trucks. The maximum average daily NOx reductions were performed for the scenario without on-road sources with 66% (Table 4-10), followed by the scenario for the year 2020 with 48%, the scenario without DFS with 41%, the scenario without LDVs with 35%, the scenario that did not consider HDVs with 30%, and the scenario without on-road DFS with an average reduction of 27%.

4.2   MODELING PERFORMANCE

4.2.1 MM5 Modeling Performance

The MM5 performance for Nashville, TN is shown in Figures 4.28 (a), 4.29, 4.30, 4.31, and 4.32. Figures 4.28 (a), 4.29, 4.30, and 4.31 depict the time series of the MM5 predicted temperature at 10 m and the monitored temperature at Nashville International Airport for March, June, September, and December of 2003. Figure 4.32 shows the scatter plot of monitored versus modeled temperature for those months, and the 45-degree line corresponds to points where the predicted values equal the monitored values. If the model predicted perfectly, all points would lie on the 45-degree line. Points above the line indicate that the model under predicted the results and points below the line indicate that the model over predicted the results. Finally, the metrics and statistics are tabulated in Table 4-11.

In general, the MM5 model version 7 tracked well with the monitored temperature, except the first 15 days of March, as shown in Figure 4.28 (a), which could have been affected for the modeling grid size of 36 km and its input data. In fact, the uncertainty increases when the grid resolution increases (Majeed et al., 2004), mainly on the input information such as land use, land cover, and roughness lengths.

(a)
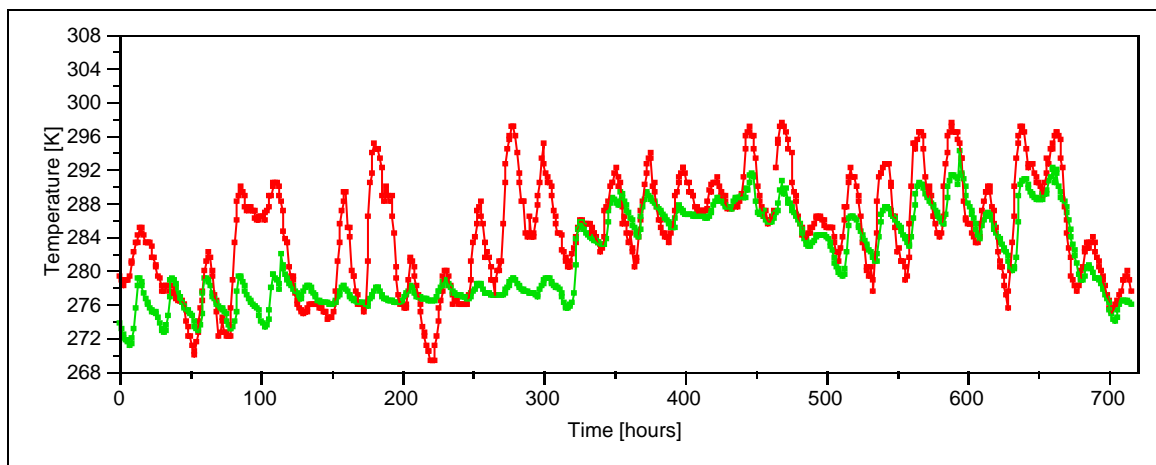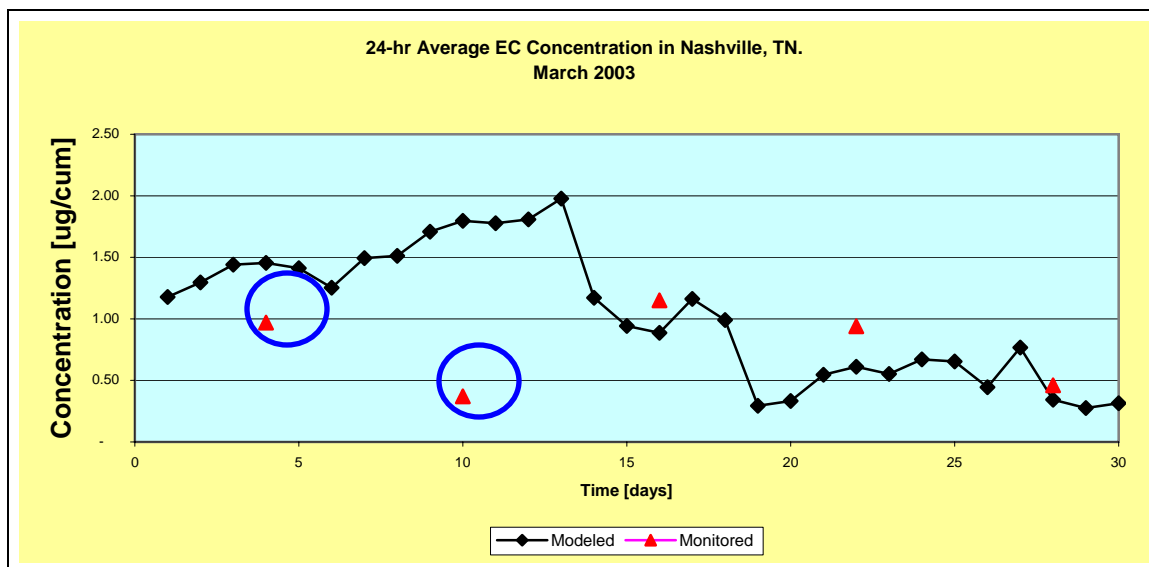
(b)

Figure 4.28. MM5 Modeling Performance in Nashville, TN, Match 2003. (a) MM5 versus Monitored Temperature (b) 24-hr Average EC Concentration

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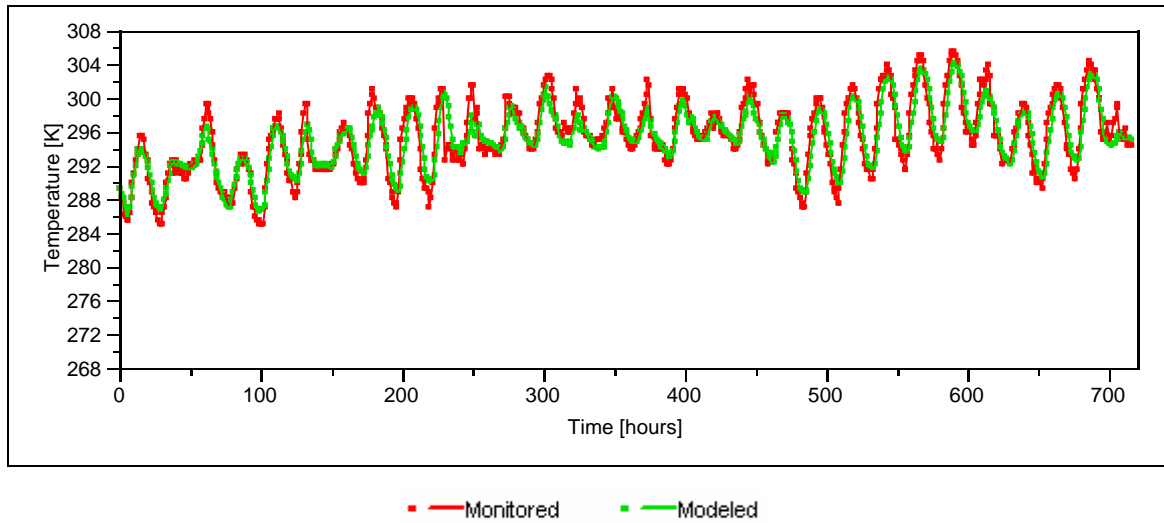
Figure 4.29. MM5 versus Monitored Temperature at Nashville, TN, June 2003
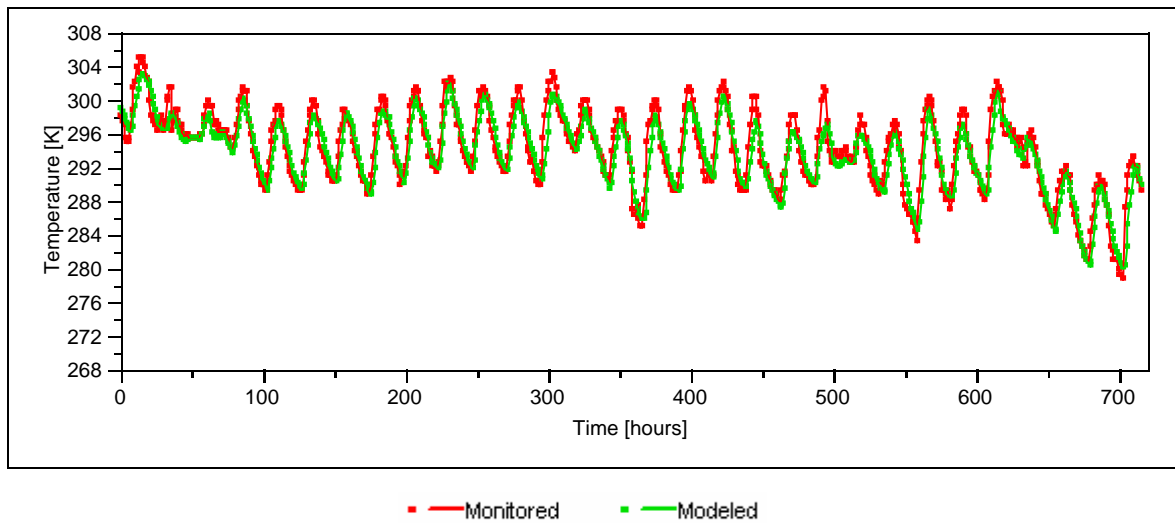
Figure 4.30. MM5 versus Monitored Temperature at Nashville, TN, September 2003
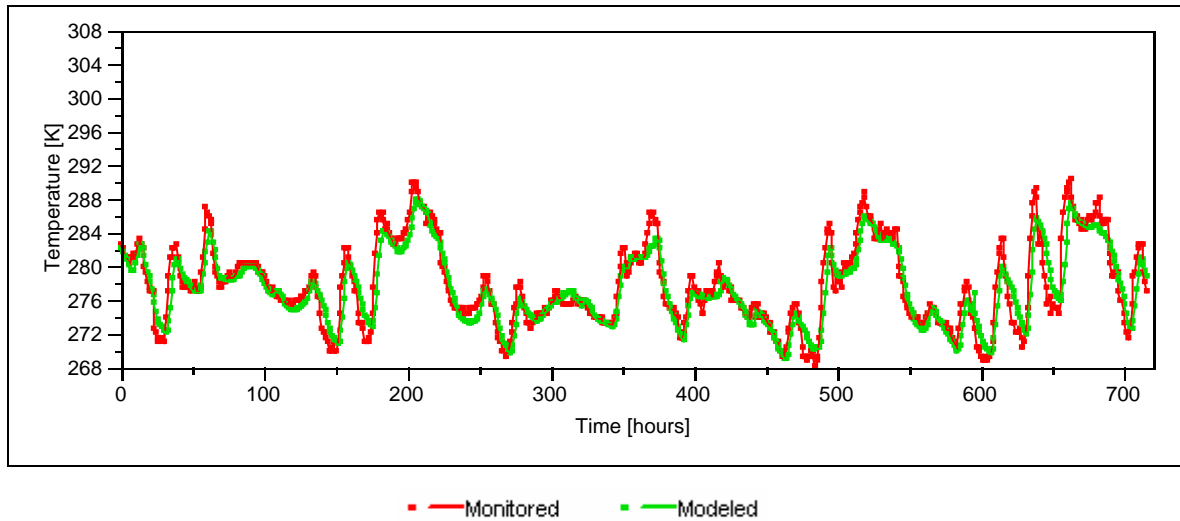
Figure 4.31. MM5 versus Monitored Temperature at Nashville, TN, December 2003

(a) All four months

(b) All four months without the first 15

days of March
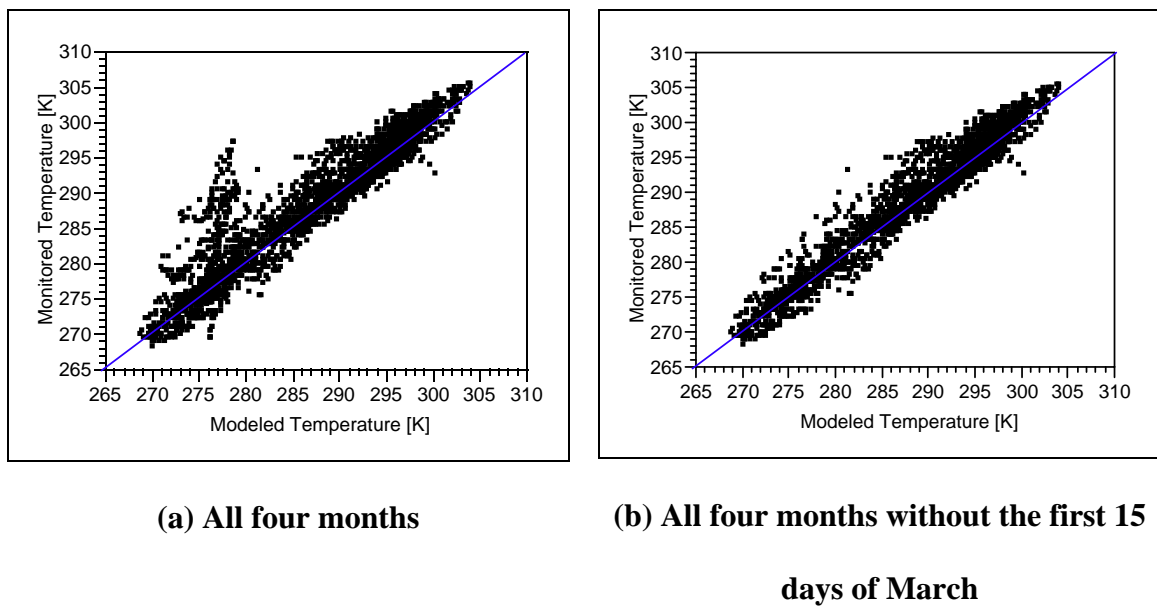
Figure 4.32. Observed Versus Predicted Hourly Temperature at Nashville, TN, 2003

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Table 4-11. Comparison of Hourly Temperature Measured at the Nashville International Airport, Nashville, Davidson County, TN, to MM5 (layer 1).

Monitored data	Mean T [K]		σ T [K]		NB [%]	NG [%]	R^2
	MM5	Obs.	MM5	Obs.			
2497	288.3	288.8	8.6	8.9	-0.19	0.55	0.945

The patterns and distribution of other meteorological variables, such as incoming radiation, air temperature, wind speed, are also affected and differ among different grid size resolutions; these different grid sizes impact the modeling of the emission fields and the final air quality performance (Majeed et al., 2004). Thus, the modeled temperature during the first 15 days of March could wrongly simulate a prolonged inversion and therefore, higher pollutant concentrations, mainly on aerosols. In fact, the modeled daily EC concentrations in March were considerably higher than the first two monitored values at Nashville, as shown in Figure 4.28 (b).

This behavior was not produced on air toxics, since the HAPs emission inventory could be underestimated for Tennessee. As result, the correct emissions scenarios analysis were considered from March 15 to 30, and the complete June, September, and December for the next analyses. For the rest of March and the other months, occasionally the monitored daily maximum temperatures were slightly higher than the predicted daily maximum, which was considered good enough to be used on air quality modeling as shown in Figures 4.29, 4.30, and 4.31.

Finally, most of the MM5 predicted temperature were over or close to the 45-degree line in the scatter plot as shown in Figure 4.32 (b), and the predicted temperature had a normalized bias of –0.19 %, a normalized gross error of 0.55 %, and a linear correlation of 0.945, which are statistically very significant. Therefore, although the modeling considered a 36-km grid size, the model performance can be considered robust and similar to the performance seen by other researchers (Majeed et al., 2004) to model CMAQ and emission scenarios analysis for HAPs and criteria pollutants.

4.2.2 CMAQ Modeling Performance

Although detailed field observations for the Nashville modeling domain were lacking, air toxics concentration measurements were available from two monitors in downtown, EATN and LOTN sites, which are part of EPA's Urban Air Toxics Monitoring Program (UATMP) (U.S. EPA, 1999d). Those two Nashville sites are very close to each other. The EATN site is located on the roof of East Health Center, which is north (predominately downwind) of downtown Nashville and is a population oriented site predominantly influenced by primarily commercial and mobile sources.

The LOTN site is located on the roof of Lockland School, which is located in the heart of downtown Nashville. This is also a population-oriented site influenced primarily by commercial and mobile sources. Observations taken as part of the UATMP are made over a 24-hour period every 6 to 12 days. Additional details on the site and the sampling

protocol are available from UATMP. Concentrations from those monitoring sites for a few air toxics and EC were compared by matching modeled 2003 concentrations simulated with CMAQ-AT for vapor toxics and CMAQ version 4.3 for EC. Comparisons for acetaldehyde, benzene, 1,3-butadiene, formaldehyde, and EC are shown in Table 4-12 and Table C-13 (Appendix C). Scatter plots of predicted and observed 24-hour average of those air toxics and EC are shown in Figure 4-33. Finally, the metrics for each pollutant are tabulated in Table 4-13, which were calculated for all monitored and modeled days.

In general, the modeled daily mean values compared reasonably well against the observed values, mainly for EC and formaldehyde. However, the model did not perform reasonably for 1,3-butadiene (Table 4-12). A possible cause is because of the lack of monitored data. For EC concentrations, the model performed better, since the 1999 criteria emissions inventory could be more accurate than the HAPs emission inventory. Finally, the highest correlation and metrics for EC may have occurred because the pollutant has a relatively long lifetime in the atmosphere and accurately represented emissions as well as no aerosol formation. EC is part of the fine particles in the accumulation mode that ranges from 0.1 to 1.0 μm particles, which have long lifetimes in suspension because both diffusion and inertial removal mechanisms are slowest in this size range (Lighty et al., 2000). On the other hand, EC is part of the ultrafine particles also, which have a short lifetime in the atmosphere of typically 15 min for 10-nm, but they can become fine particles through nucleation and accumulation mechanisms and can be transported over long distances adding regional air quality degradation (Biswas and Wu, 2005).

Table 4-12. Comparison of Air Toxics and EC Daily Concentrations of 2003 Measured at the EATN and LOTN sites, Nashville, Davidson County, TN, to CMAQ (layer 1).

Compound	Monitored data	Mean [ug/m³]		σ [ug/m³]		R²
		CMAQ	Obs.	CMAQ	Obs.	
Acetaldehyde	17	1.12	1.66	0.40	0.61	0.37
Benzene	18	0.93	1.69	0.53	0.90	0.27
1,3-Butadiene	4	0.11	0.29	0.06	0.10	0.15
Formaldehyde	17	1.72	3.20	1.04	1.14	0.62
EC	17	0.55	0.72	0.20	0.36	0.65

Table 4-13. Modeled Performance Metrics for EATN and LOTN sites at Nashville, Davidson County, TN, 2003.

Compound	NB (%)	NG (%)
Acetaldehyde	-25	42
Benzene	-39	43
1,3-Butadiene	-57	57
Formaldehyde	-47	47
EC	-13	28

Observed Versus Predicted Daily EC Concentrations

Observed Versus Predicted Daily Benzene Concentrations

Observed Versus Predicted Daily Formaldehyde Concentrations

Observed Versus Predicted Daily Acetaldehyde Concentrations

Observed Versus Predicted Daily 1,3-Butadiene Concentrations
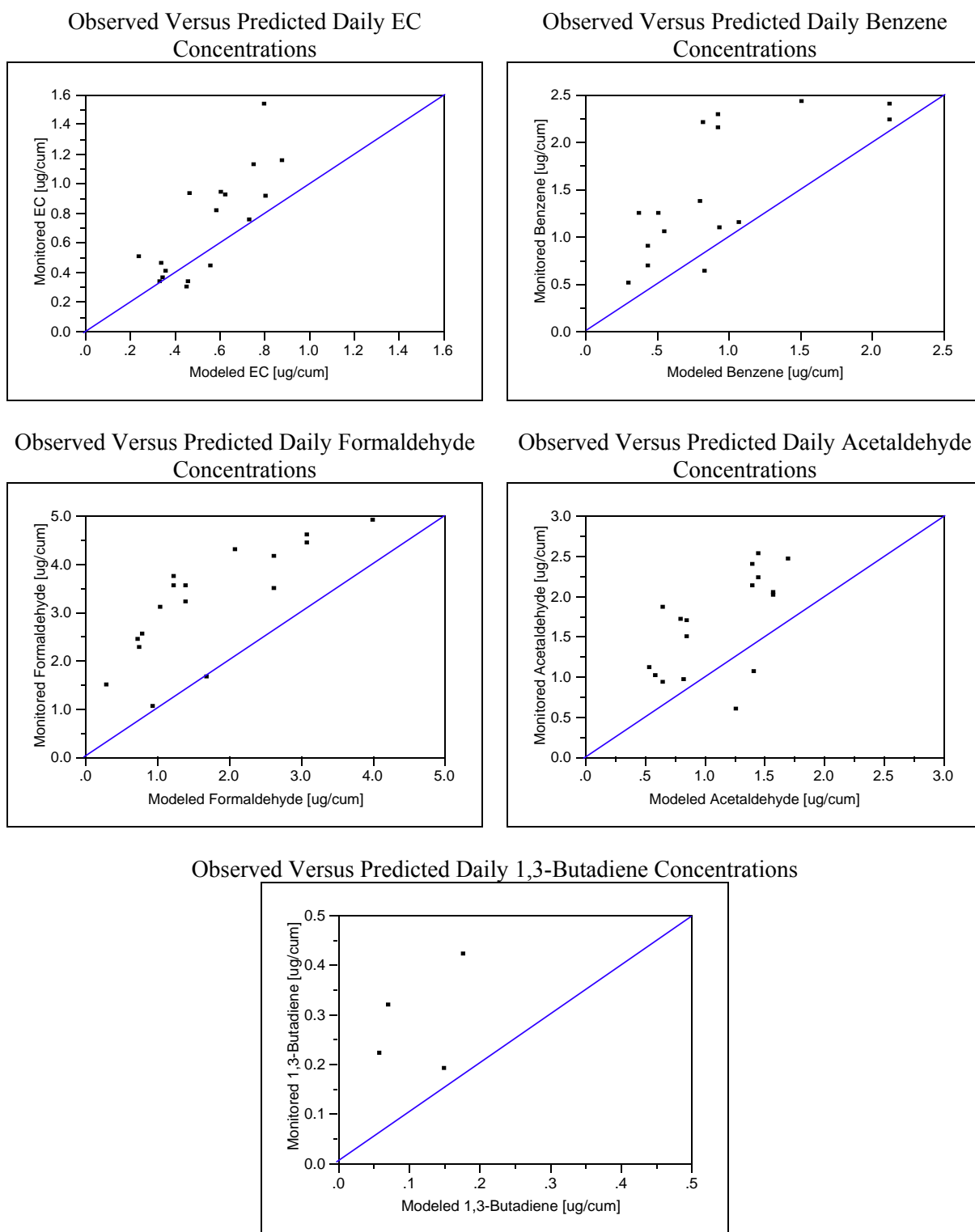
Figure 4.33. Scatter Plots of Monitored vs. Predicted Daily Acetaldehyde, Benzene, 1,3-Butadiene, Formaldehyde, and EC in Nashville, Davidson County, TN, 2003.

The lack of gravitational settling results in a long lifetime in the air and increases the chances of a better modeling performance. The scatter plots also showed that the model under predicted the acetaldehyde, benzene, 1,3-butadiene, formaldehyde, and EC daily concentrations for most days of 2003, since a majority of the points fell above the 45-degree line. This can be corroborated in Table 4-13, since the model under predicted about 13 to 57% (normalized bias) over those four months. The normalized gross error ranged from 28 to 57% for those HAPs. The U.S. EPA guidance (U.S. EPA, 2001d) recommends that the normalized bias has to be less than or equal to 20%, and less or equal to 30% for normalized gross error. As discussed above, a possible cause may have been due to the modeling assumed zero boundary conditions in the simulations; however, the main errors could have resulted from an inaccurate emissions inventory, that the inventory and meteorological data were base on different years, and for the modeling grid size of 36 km used.

Modeling MM5 and CMAQ for 36 km grid resolution produces inaccurate meteorology, dispersion fields, spatial, and temporal variability of the pollutants; however, this assumption is good enough to analyze the proposed emission scenarios on ground level concentrations and health risk. In fact, even though the model performance was not very strong and similar to the performance seen by other researchers (Ching et al., 2004), it could be considered satisfactory to do emission scenarios analysis for those pollutants, since the analysis approach involves considering the difference in mass concentrations and health risk values among the proposed emission scenarios and the base case scenario rather than the absolute mass concentration or health risk value. This assumes that the

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factors that contributed to the under and over prediction of those air toxics and EC concentrations for March, June, September, and December would contribute similarly in all the scenarios considered in the analysis, causing minimal effects on the differences among the scenarios. Future simulations could include running fine-scale modeling on air toxics to better capture spatial and temporal variability, as well as, concentration magnitudes, which could help identify and characterize the hot spots of air toxics compounds from an exposure point of view.

4.3   BASE CASE CMAQ MODELING RESULTS

The base case CMAQ modeling results are summarized and discussed for Nashville, Davidson County, TN, showing the temporal variation for the analyzed months. In addition, results are also presented as tile plots to show the spatial variation of March, June, September, and December in the whole modeling domain. The 36 km grid size almost entirely covered Davidson County and the results were assumed to be representative of the concentration in the whole urban area of Nashville.

The column 23 and row 24 of the 36 km grid cell approximately covered all of Nashville. For each month under analysis, the model runs were set to start five days earlier to allow for the "spin-up" period, which was done to avoid the influence of the initial conditions on the model results.

4.3.1 Air Toxics Concentrations in Davidson County, Tennessee

4.3.1.1   Acetaldehyde

Hourly acetaldehyde concentrations at Nashville produced similar seasonal patterns for most of the 24-hour time periods for each month as shown in Figure 4.34. Maximum concentrations occurred between 6 and 9 PM and the minimum concentrations occurred between 3 and 6 AM, and a second maximum occurred between 7 and 9 AM. It may be noted that acetaldehyde is generated as a primary and secondary pollutant. The photochemical reaction at the troposphere for this kind of aldehyde is very complex, mainly during summer season, where the high temperatures speed up its overall reaction and the overall reaction of its precursors. In fact, the anthropogenic acetaldehyde is a primary pollutant generated mainly from mobile sources, whose maximum emissions occurred at traffic pick hours in Davidson County, i.e, from 7 AM to 7 PM, with the highest emissions at 5 PM as shown in Figure 4.36. This primary acetaldehyde was also produced from biogenic sources mainly in spring and summer seasons. Some emitted primary acetaldehyde at morning rush-hour traffic in June reacted rapidly with $OH^\bullet$ to generate CH_3CO_3 radical. Simultaneously, it was decomposed by potholysis into $HCO^\bullet$ and $CH_3^\bullet$ (alkyl) free radicals, as well as, dispersed due to higher wind speeds during afternoon at Davison county as shown in Figures 4.35 (June) and 4.37.

227
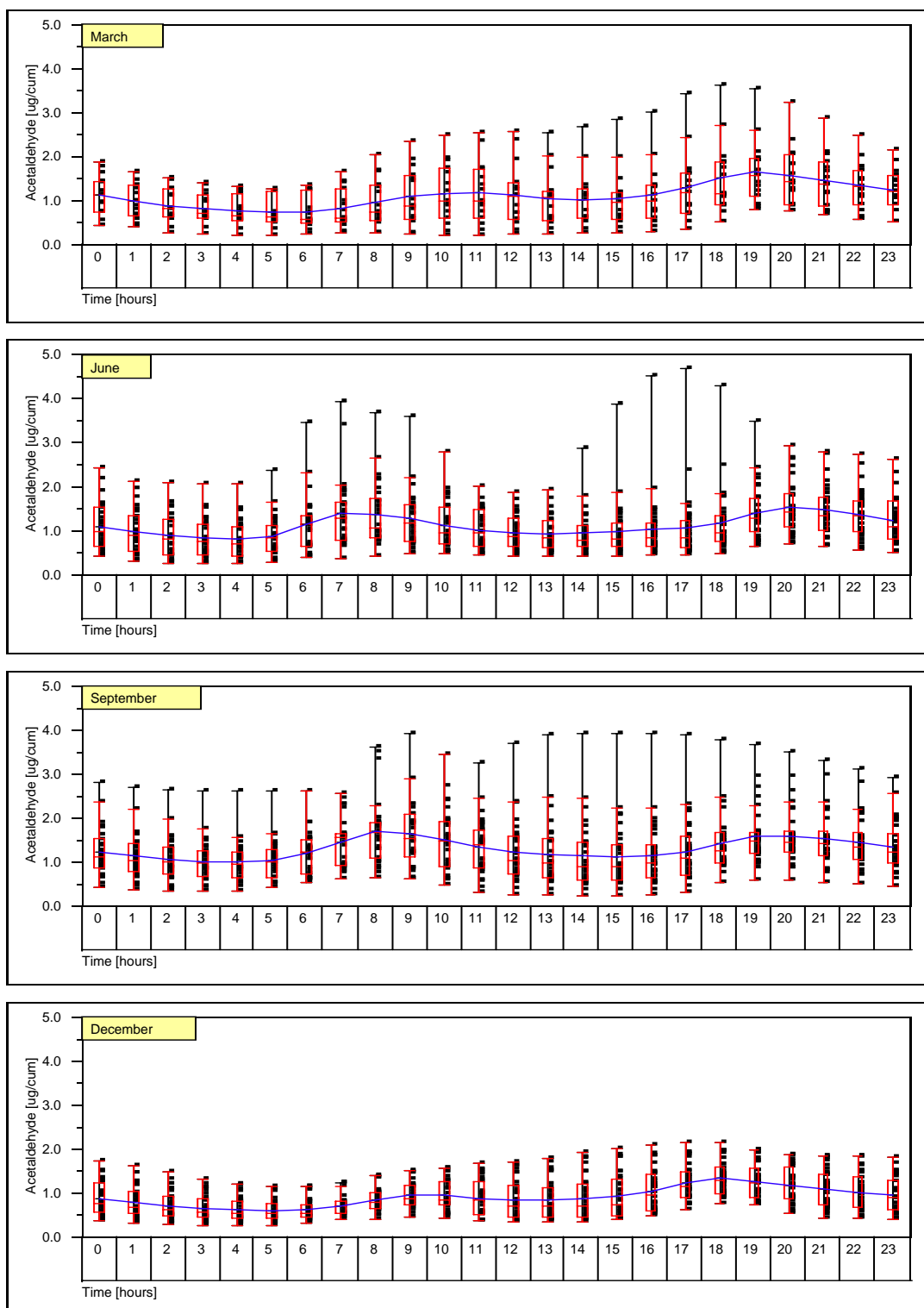
Figure 4.34. Box-Plots of the Modeled Hourly Average Acetaldehyde Concentration at Nashville, TN, for March, June, September, and December 2003

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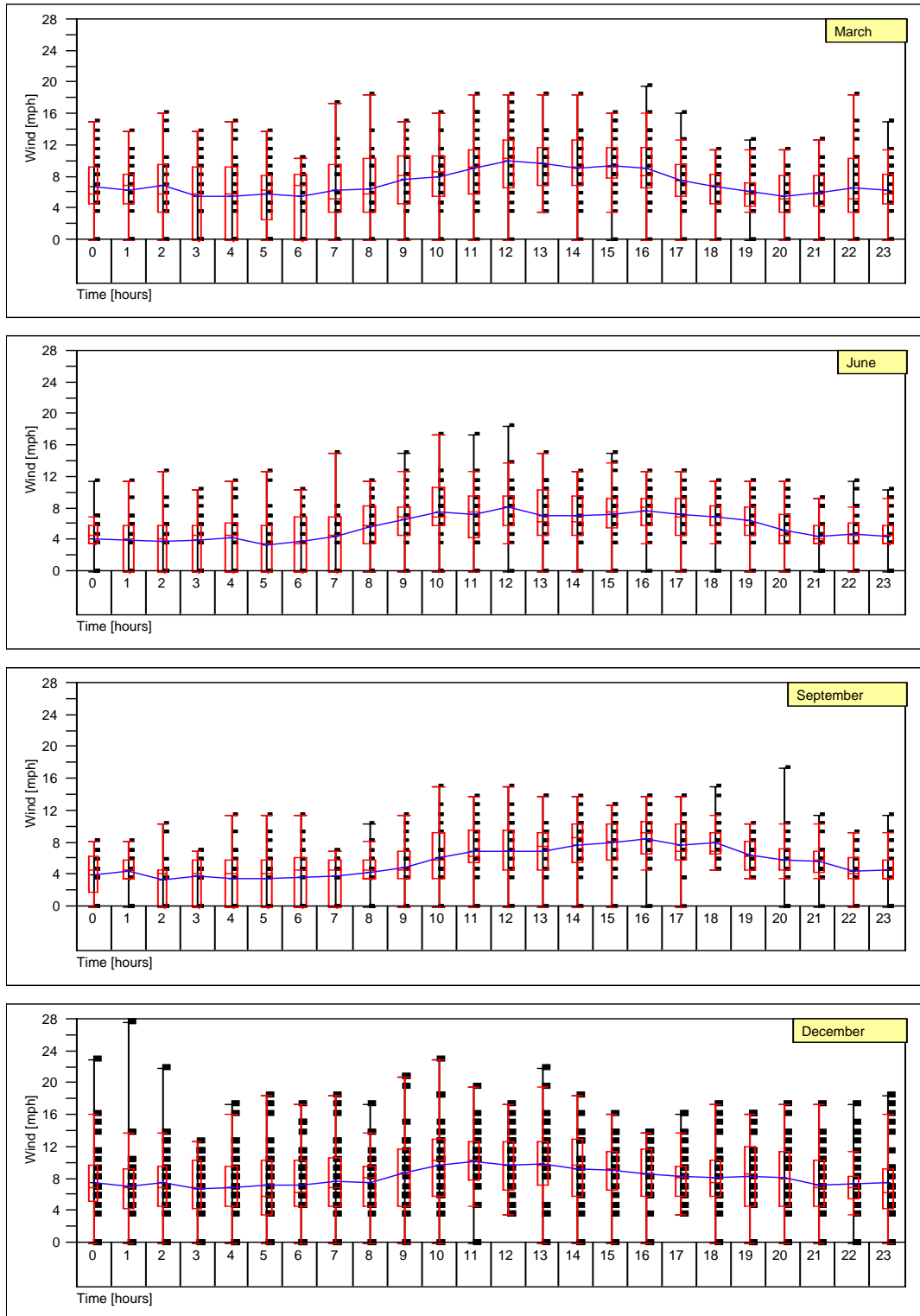
Figure 4.35. Box-Plots of the Monitored Hourly Average Wind Speed at Nashville, TN, for March, June, September, and December 2003
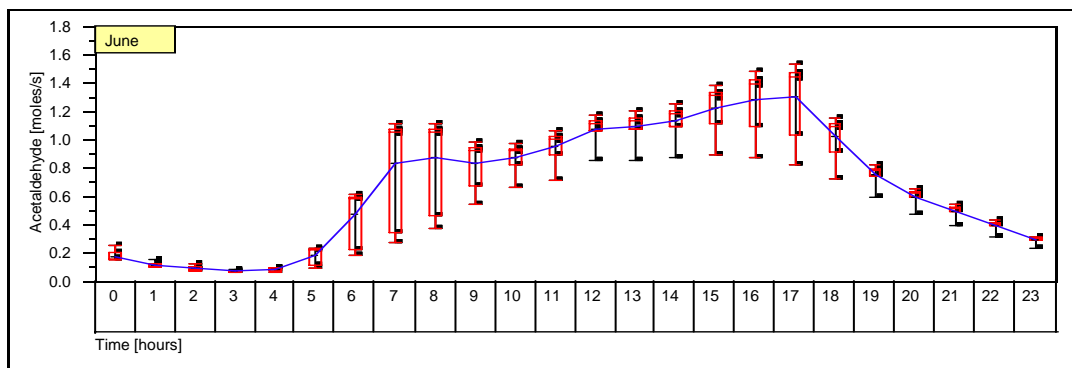
Figure 4.36. Box-plot of the On-road Hourly Average Acetaldehyde Emissions in Nashville, TN, June 2003
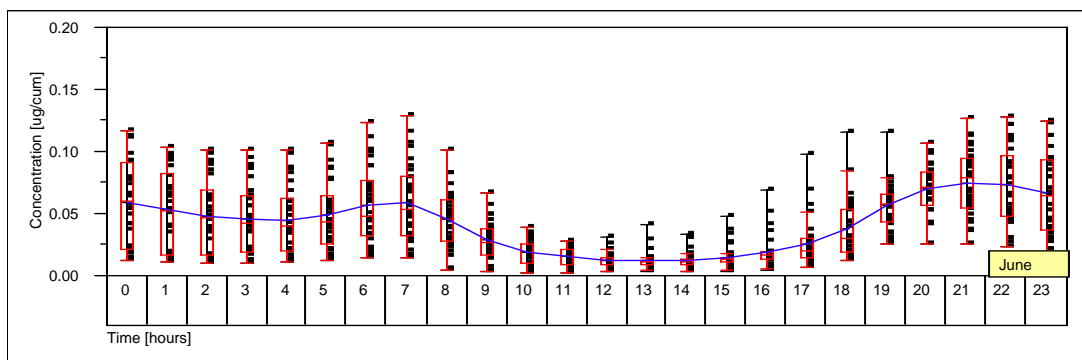
Figure 4.37. Box-plot of the Primary Hourly Average Acetaldehyde Concentration in Nashville, TN, June 2003

Those figures show that most of the primary acetaldehyde emitted at morning rush-hour traffic was decomposed and dispersed at around 1PM. At night, the acetaldehyde emitted during afternoon rush-hour traffic reacted slowly with $NO_3^{\bullet}$ to generate HNO_3 and CH_3CO_3 radical, and was slowly dispersed as a result of a greater atmospheric stability as shown in Figure 4.35 (June) and 4.37. This chemical mechanism is explained also for some authors (Bloss et al., 2005; Seinfeld and Pandis, 1998; Baird, 2001).

On the other hand, at least 136 VOCs are secondary acetaldehyde precursors in the troposphere, especially isoprene (Bloss et al., 2005). Most of those reactive VOCs emitted into the air by morning and afternoon rush-hour traffic were converted to secondary acetaldehyde in summer season, but at the same time, it was dispersed as a result of an unstable atmosphere by about noon in Davidson County, as shown in Figure 4.38.
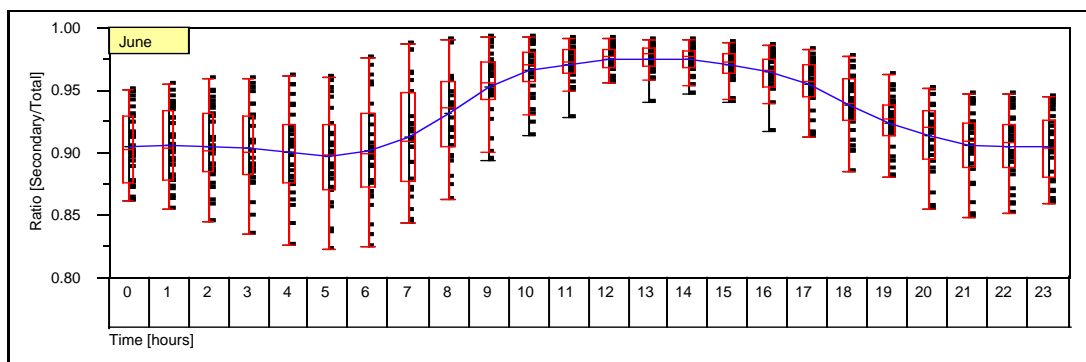
Figure 4.38. Box-plot of the Secondary to Total Acetaldehyde Ratio on Hourly Concentrations at Nashville, TN, June 2003

This box-plot shows that the maximum secondary acetaldehyde contribution was 98% around 1 PM. By the end of the day and at night, an important amount of acetaldehyde disappeared, since it was photochemically decomposed into HCO$^\bullet$ and CH$_3$$^\bullet$ free radicals, as well as, it was dispersed and reacted with OH$^\bullet$ radical. The rest of the acetaldehyde concentration was primary acetaldehyde emitted from mobile and biogenic sources and its decomposition with NO$_3$$^\bullet$ at nighttime. Simultaneously, acetaldehyde was added through the secondary formation generated by the oxidation of some VOCs with NO$_3$$^\bullet$ rather than OH$^\bullet$, which also was slowly dispersed as a result of a greater atmospheric stability, as shown in Figures 4.35 (June) and 4.39. During cold months, a reduced amount of secondary acetaldehyde was formed through the reaction of its VOCs precursors with less available OH$^\bullet$ radical and with NO$_3$$^\bullet$ radical rather than photolysis. At the same time, less primary acetaldehyde was decomposed into CH$_3$CO$_3$$^\bullet$, HCO$^\bullet$, and CH$_3$$^\bullet$ radicals, and more acetaldehyde was dispersed due to the higher wind speed during December.
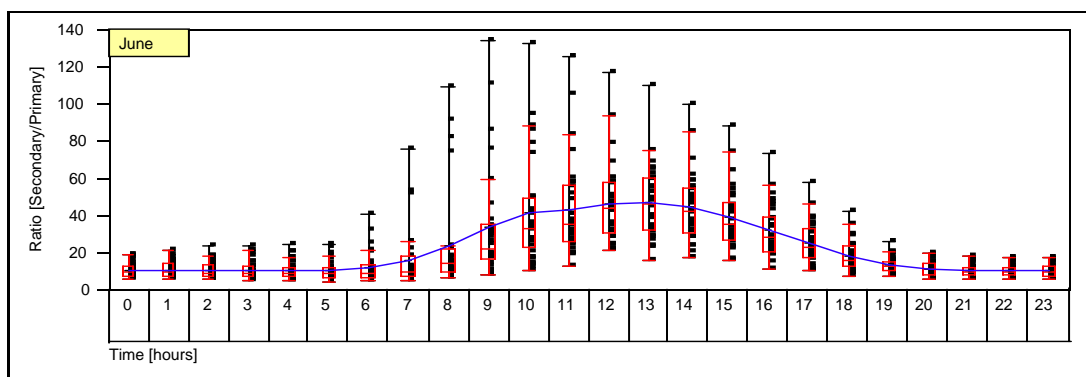
Figure 4.39. Box-plot of the Secondary to Primary Acetaldehyde Ratio on Hourly Concentrations at Nashville, TN, June 2003

This behavior is observed by the different seasonal patterns in cold months as shown in Figures 4.35 (December), 4.40, 4.41, and 4.42. As result, the total acetaldehyde concentration during December was lower than June. In fact, the maximum secondary acetaldehyde contribution in December was around 85% instead of 98% in June, and the maximum mean secondary to primary hourly acetaldehyde ratio was 7 times in December instead of 40 times in June, as shown in Figures 4.39 and 4.42. Those secondary acetaldehyde contributions and secondary to primary ratio were almost flat during December instead of seasonal pattern during June. This explains the importance of the overall photolysis and the OH$^\bullet$ availability in the secondary acetaldehyde formation and primary acetaldehyde decomposition. Wind speed was also important on the acetaldehyde dispersion. It may be noted that in the summer season the population of Nashville was exposed to slightly higher daily acetaldehyde concentrations, mainly due to secondary formation. Finally, the annual modeled acetaldehyde concentration was 1.11 ug/m^3 at Nashville, TN, 2003, as shown in Figure 4.43 and Table 4.14.
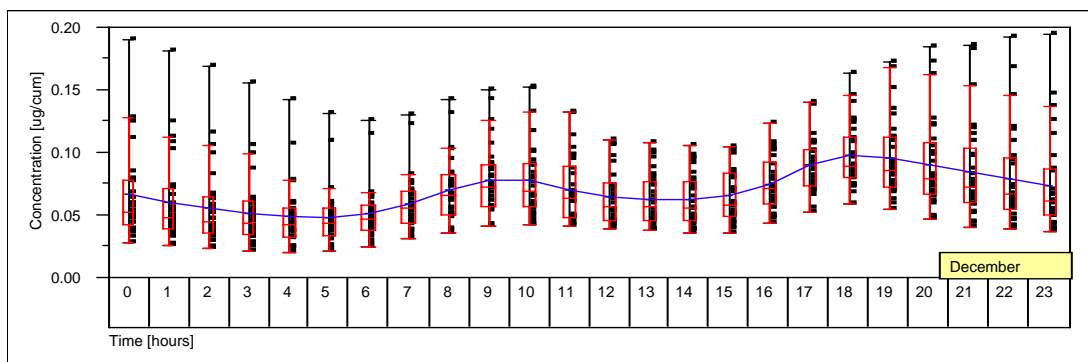
Figure 4.40. Box-plot of thePprimary Hourly Average Acetaldehyde Concentration in Nashville, TN, December 2003
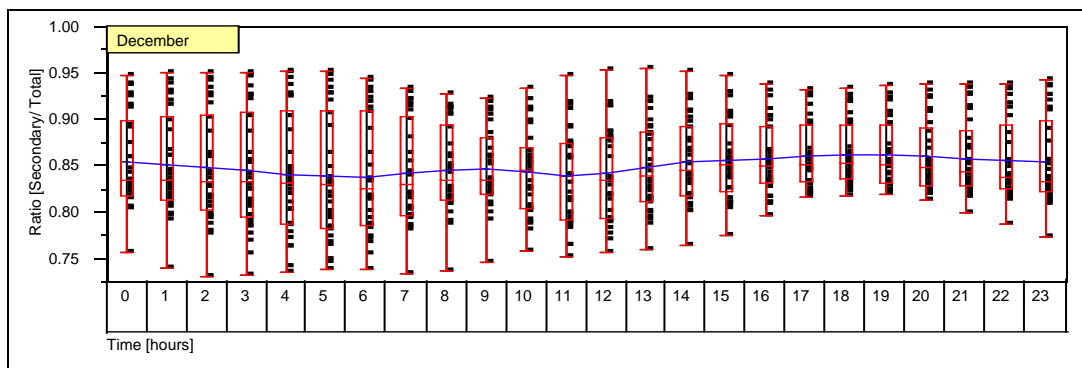
Figure 4.41. Box-plot of the Secondary to Total Acetaldehyde Ratio on Hourly Concentrations at Nashville, TN, December 2003
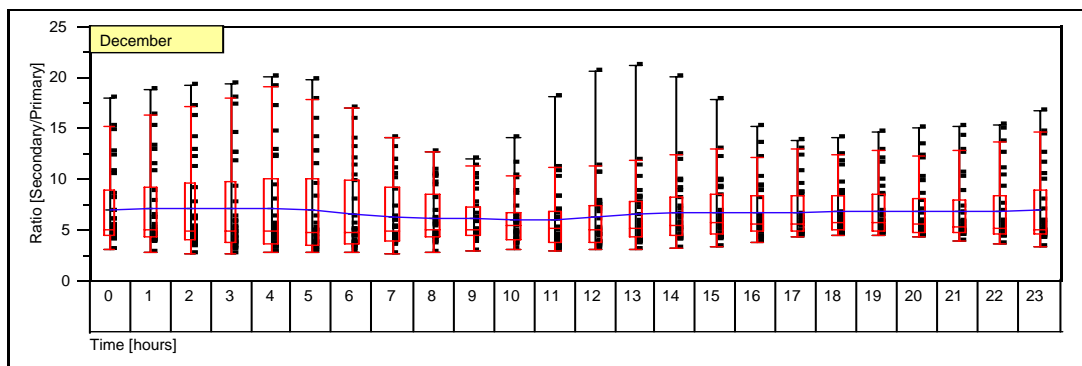
Figure 4.42. Box-plot of the Secondary to Primary Acetaldehyde Ratio on Hourly Concentrations at Nashville, TN, December 2003
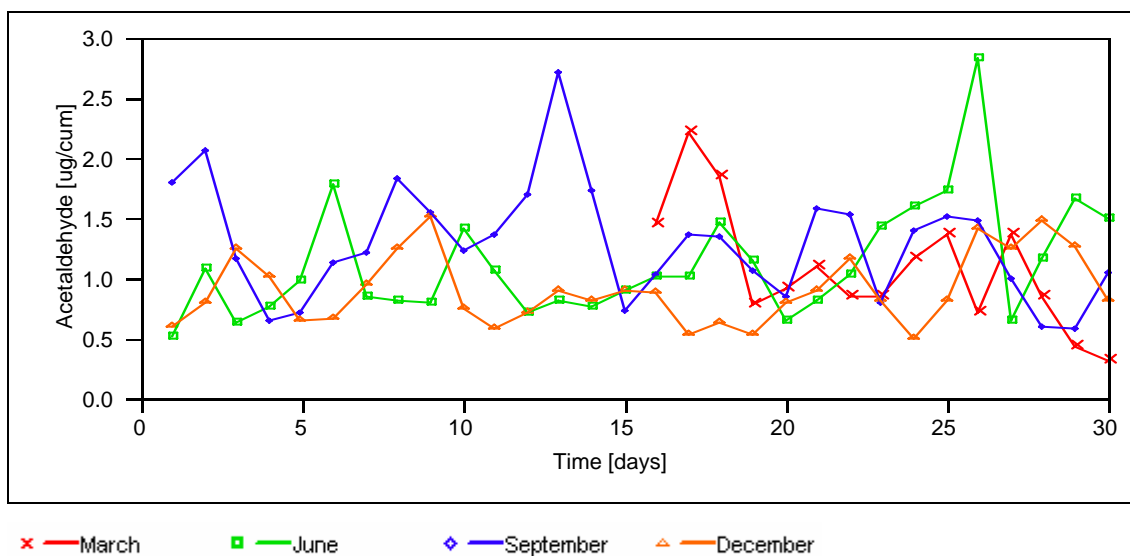
Figures 4.43. Modeled Daily Acetaldehyde Concentration in Nashville, TN, 2003

Table 4-14. Modeled Acetaldehyde Concentration at Nashville, Davidson County, TN, 2003

Period	Concentration [ug/m^3]
March	1.09
June	1.13
September	1.30
December	0.91
Annual	**1.11**

4.3.1.2   Formaldehyde

Hourly formaldehyde concentrations in Nashville where higher in June and September than December and March as shown in Figure 4.44, however, it was not easy to see the seasonal patterns for the 24-hour time periods for each month, since during some particulate days in the summer season, the daily formaldehyde concentration was as high as 8 and 7 ug/m^3 on June 26th and September 13th 2003, respectively. Those high values generated more variability on the box-plots shown in Figure 4.44. To analyze the seasonal pattern for each month, those high values were removed. Thus the modified modeled hourly average formaldehyde concentration at Nashville is shown in Figure 4.45. Those concentrations produced similar seasonal patterns for the most of the 24-hour time periods for each month as shown in Figure 4.45, whose maximum concentrations occurred between 6 and 9 PM and the minimum concentrations occurred between 4 and 7 AM, with a second maximum occurred between 8 and 10 AM.

Formaldehyde is emitted mainly from mobile sources as shown in Figure 4.5, and also is an oxidation product of at least 240 VOCs, especially isoprene (Bloss et al., 2005). It is an essential component of tropospheric photochemistry predominantly in the summer season. In fact, like acetaldehyde, some emitted anthropogenic primary formaldehyde at morning rush-hour traffic reacted rapidly with OH$^\bullet$ to generate HCO$^\bullet$ radical and H$_2$O during the daytime.
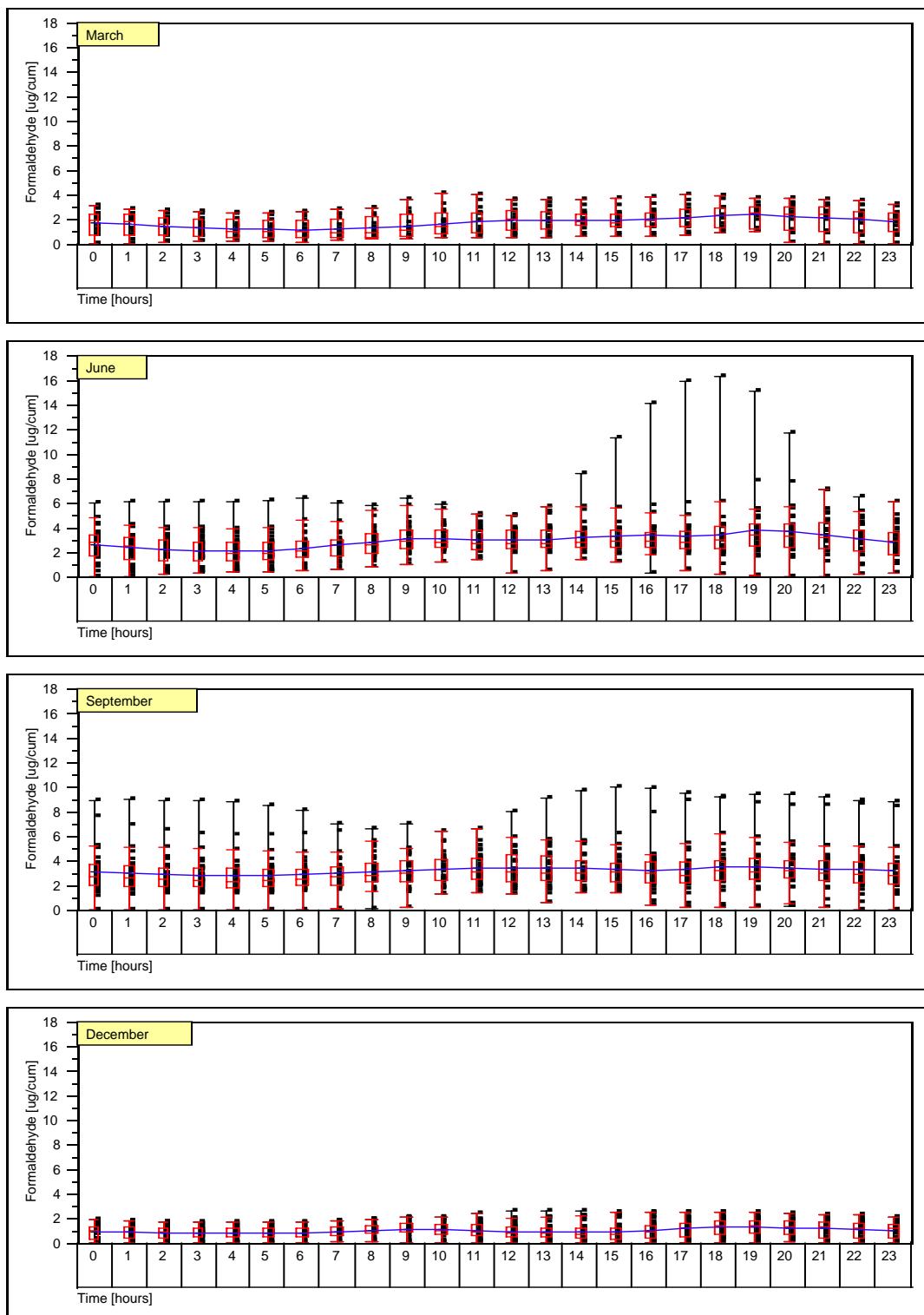
Figure 4.44. Box-Plots of the Modeled Hourly Average Formaldehyde Concentration at Nashville, TN, for March, June, September, and December 2003

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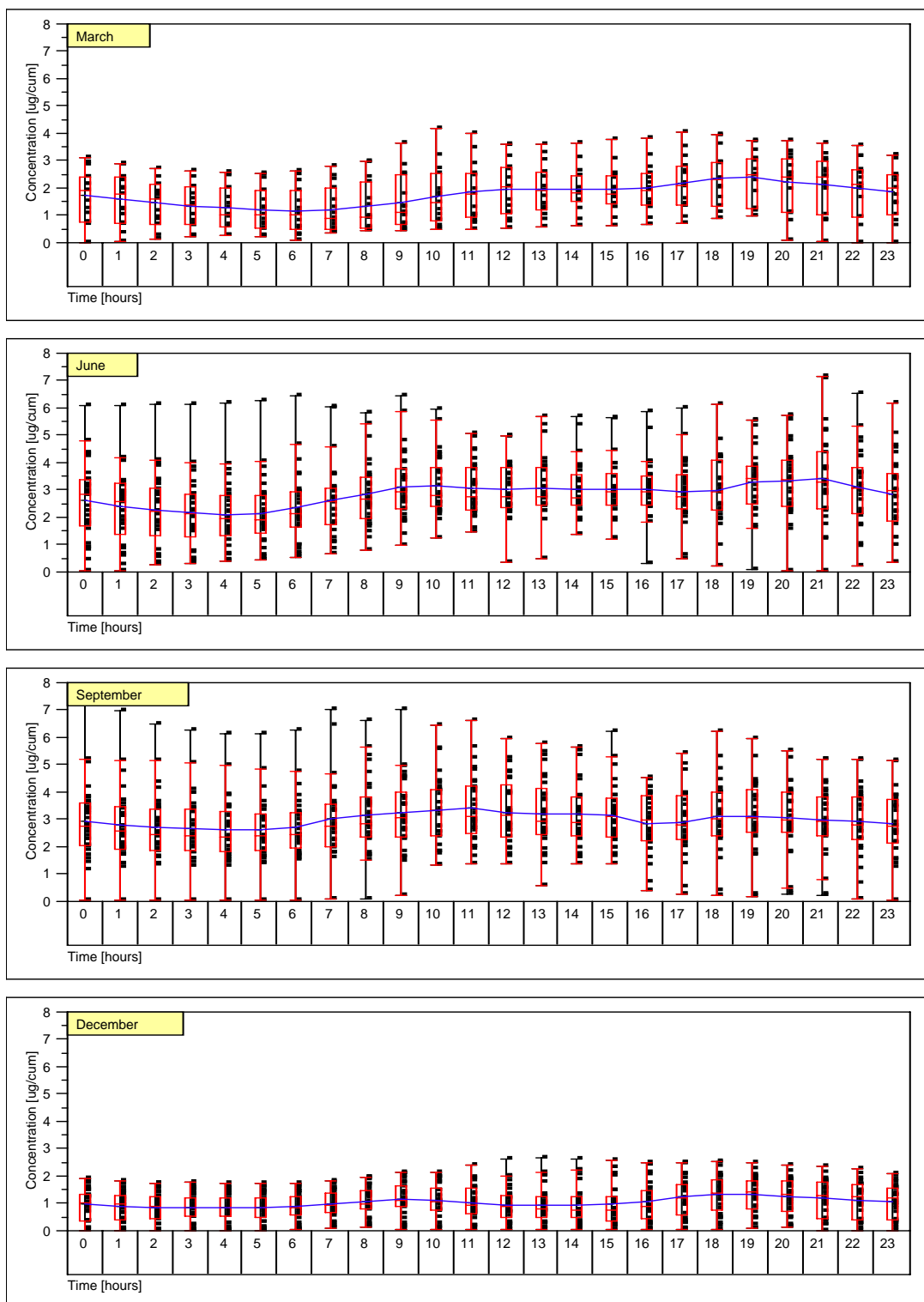
Figure 4.45. Box-Plots of the Modified Modeled Hourly Average Formaldehyde Concentration at Nashville, TN, for March, June, September, and December 2003

Simultaneously, it was decomposed photochemically into $H^\bullet$ and $HCO^\bullet$ free radicals, reacted with O_2 to generate CO and HO_2 radical, as well as, was dispersed due to higher wind speeds during afternoon hours at Davidson County, as shown in Figures 4.35 (June) and 4.46. Those figures show that most of the primary formaldehyde emitted at morning traffic congestion was decomposed and dispersed at noon. At night, the formaldehyde emitted at afternoon rush-hour traffic reacted slowly with $NO_3^\bullet$ to generate HNO_3, CO, and HO_2 radical, and was slowly dispersed as a result of a greater atmospheric stability, as shown in Figures 4.35 (June) and 4.46. This chemical mechanism is explained also for some authors (Bloss et al., 2005; Seinfeld and Pandis, 1998; Baird, 2001).

On the other hand and like acetaldehyde, several of those 240 reactive VOCs emitted into the air by morning rush-hour traffic were converted to secondary formaldehyde, but at the same time, it was dispersed as a result of an unstable atmosphere by about noon in Davidson County, as shown in Figures 4.47 and 4.48, where the maximum secondary formaldehyde contribution was almost 98%. By the end of the day and at night, an important amount of formaldehyde disappeared, since it was photochemically decomposed into $HCO^\bullet$ and $H^\bullet$ free radicals and later to H_2 and CO, as well as, dispersed and reacted with $OH^\bullet$ radical.
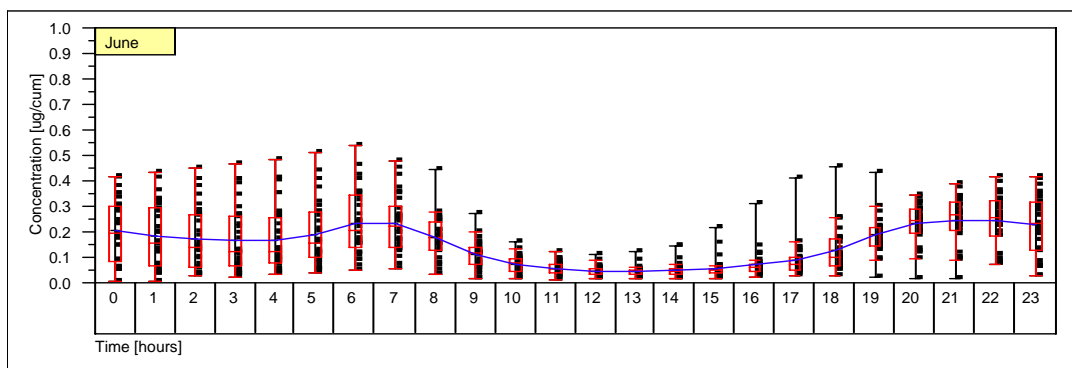
Figure 4.46. Box-plot of the Primary Hourly Average Formaldehyde Concentration in Nashville, TN, June 2003
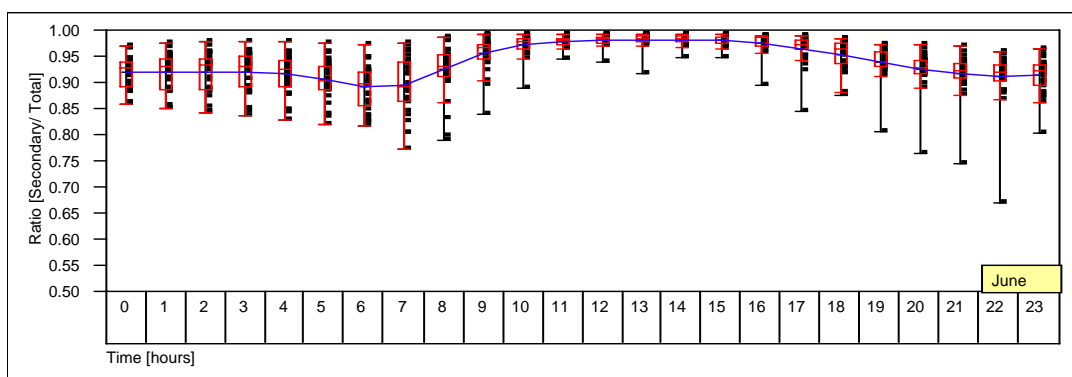
Figure 4.47. Box-plot of the Secondary to Total Formaldehyde Ratio on Hourly Concentrations at Nashville, TN, June 2003
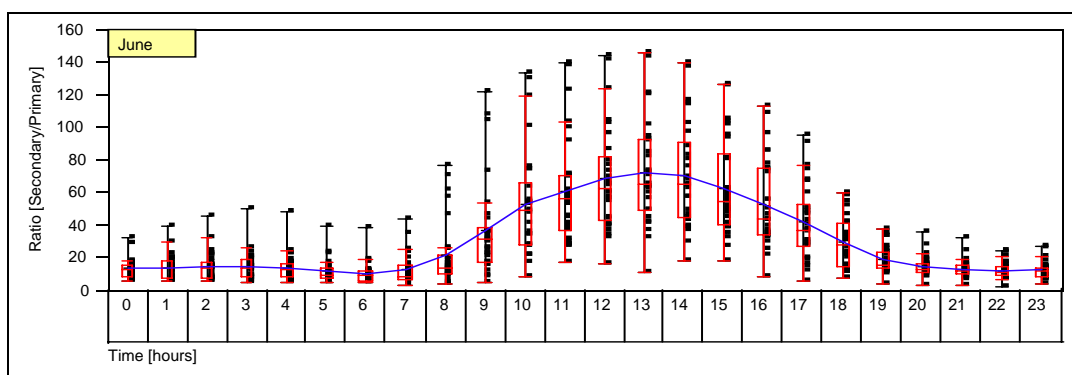
Figure 4.48. Box-plot of the Secondary to Primary Formaldehyde Ratio on Hourly Concentrations at Nashville, TN, June 2003

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The remaining formaldehyde concentration was contributed by primary acetaldehyde emitted from mobile sources and its decomposition with $NO_3^{\bullet}$ at nighttime. Concurrently, formaldehyde was contributed through the secondary formaldehyde generated by the oxidation of some VOCs with $NO_3\bullet$ rather than $OH^{\bullet}$, which also was slowly dispersed as result of a greater atmospheric stability, as shown in Figures 4.35 (June) and 4.48.

During cold months, similar to acetaldehyde, a reduced amount of secondary formaldehyde was generated due to the reaction of its VOCs precursors with $OH^{\bullet}$ radical, O_2, and with $NO_3^{\bullet}$ radical rather than photolysis. Simultaneously, less primary formaldehyde was decomposed, and more formaldehyde was dispersed due to the higher wind speeds during December.

This performance is represented through the different seasonal patterns in cold months as shown in Figures 4.35 (December), 4.49, 4.50, and 4.51. As a result, the total formaldehyde concentration during December was lower than June at Nashville. In fact, the maximum secondary formaldehyde contribution in December was around 73% instead of 98% in June, and the maximum mean secondary to primary hourly formaldehyde ratio was 4 times instead of 70 times as in June, as shown in Figures 4.48 and 4.51. It explains the importance of the overall photolysis and the $OH^{\bullet}$ availability in the secondary formaldehyde formation and primary formaldehyde decomposition. Wind speed was also an important factor on the formaldehyde dispersion.
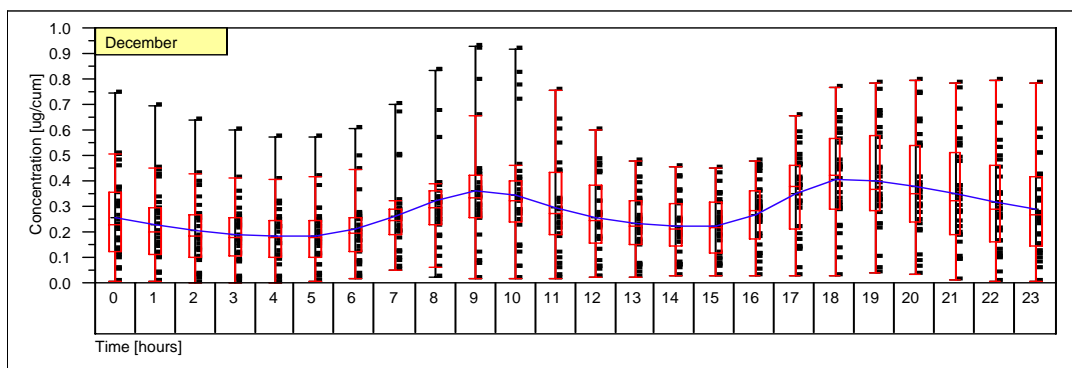
Figure 4.49. Box-plot of the Primary Hourly Average Formaldehyde Concentration in Nashville, TN, December 2003
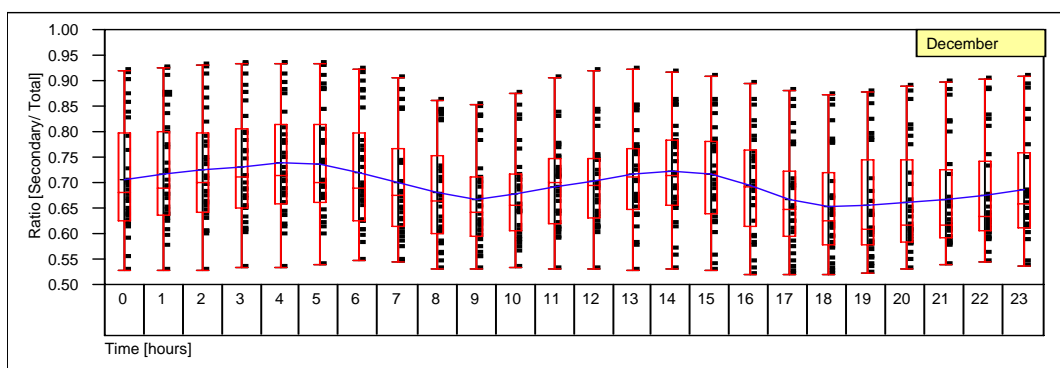
Figure 4.50. Box-plot of the Secondary to Total Formaldehyde Ratio on Hourly Concentrations at Nashville, TN, December 2003
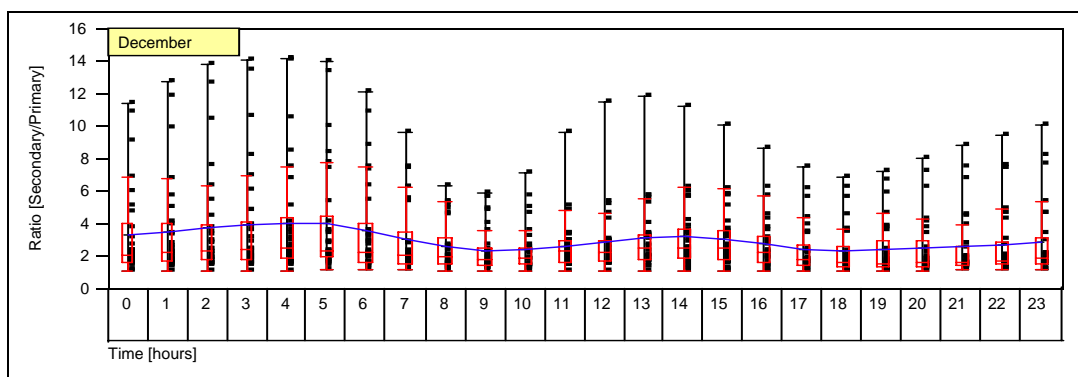
Figure 4.51. Box-plot of the Secondary to Primary Formaldehyde Ratio on Hourly Concentrations at Nashville, TN, December 2003

Finally, it may be noted that in the summer season the population was exposed almost 3

times more to daily formaldehyde concentration than in December, mainly due to

secondary formation. The annual modeled formaldehyde concentration was 2.25 ug/m^3 at

Nashville, TN, 2003, as shown in Figure 4.52 and Table 4.15. Figures 4.53, 4.54, and

4.55 compare the secondary contributions of acetaldehyde and formaldehyde for each

analyzed month. The secondary contribution to total formaldehyde was greater than

acetaldehyde in June and September, but it was lower than acetaldehyde during March

and December, mainly in December. Similarly, the secondary contribution on

formaldehyde and acetaldehyde produced more variability during December, principally

on formaldehyde. This behavior could indicate that those VOCs precursors of

formaldehyde react easier than those that generate acetaldehyde in warmer weather, but

they react slower in cold weather than those that produce secondary acetaldehyde.
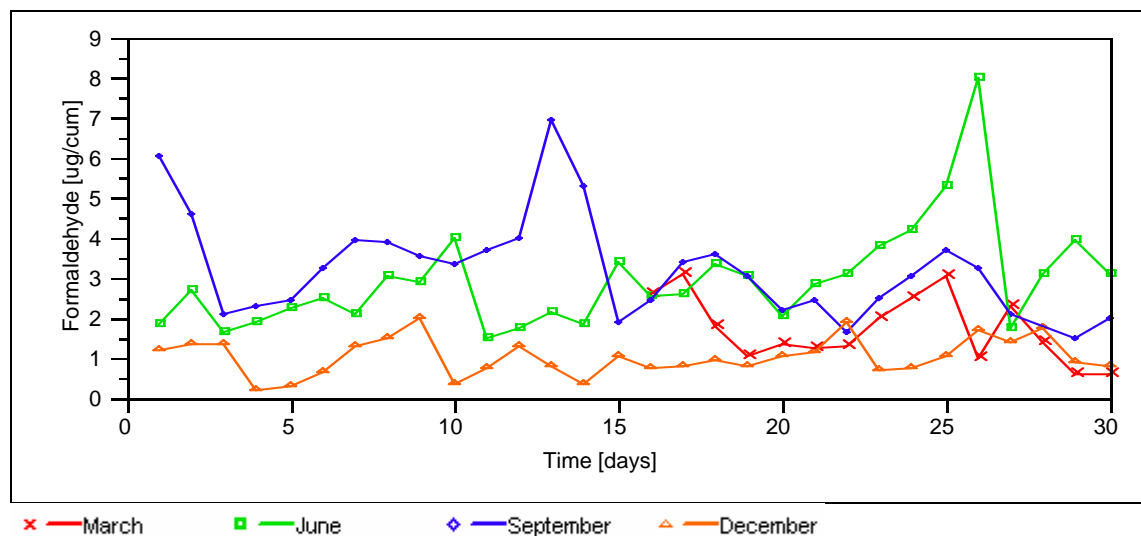
Figure 4.52. Modeled Daily Formaldehyde Concentration in Nashville, TN, 2003

Table 4-15. Modeled Formaldehyde Concentration at Nashville, Davidson County, TN, 2003

Period	Concentration [ug/m^3]
March	1.75
June	2.97
September	3.22
December	1.05
Annual	**2.25**
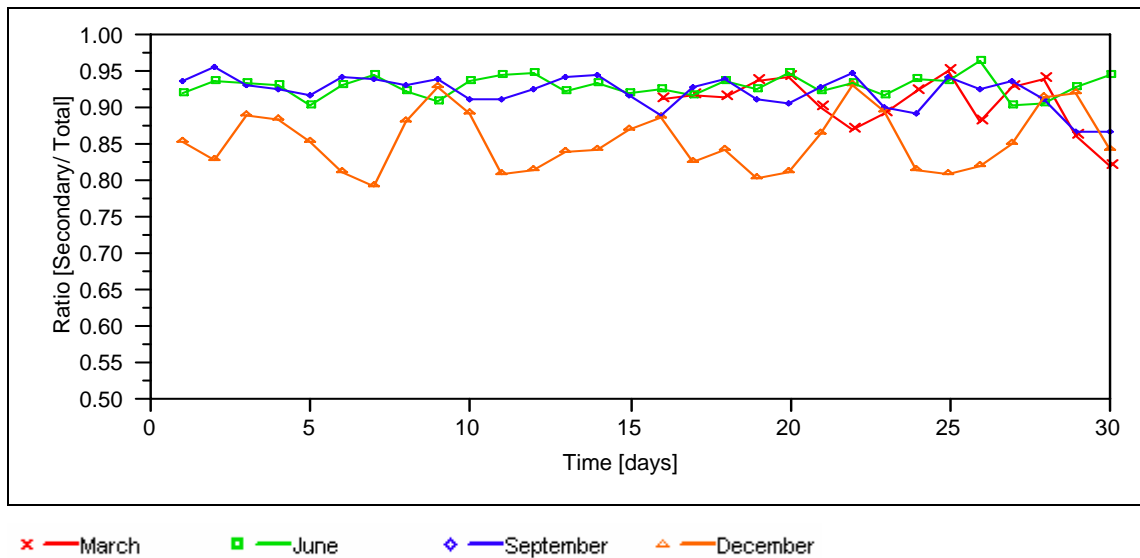
Figure 4.53. Secondary to Total Acetaldehyde Ratio on Daily Concentrations at Nashville, TN, 2003
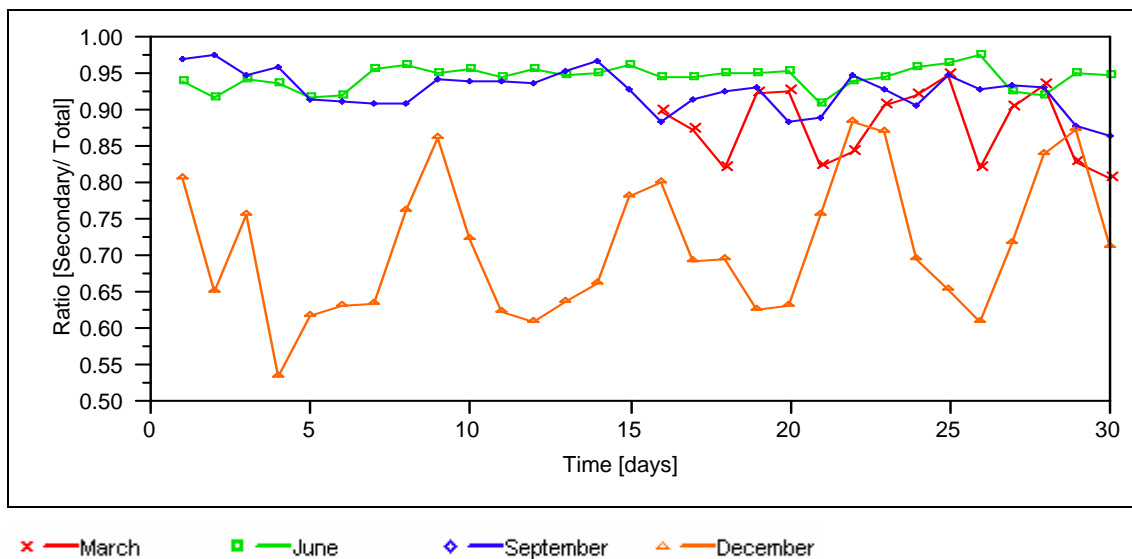
Figure 4.54. Secondary to Total Formaldehyde Ratio on Daily Concentrations at Nashville, TN, December 2003
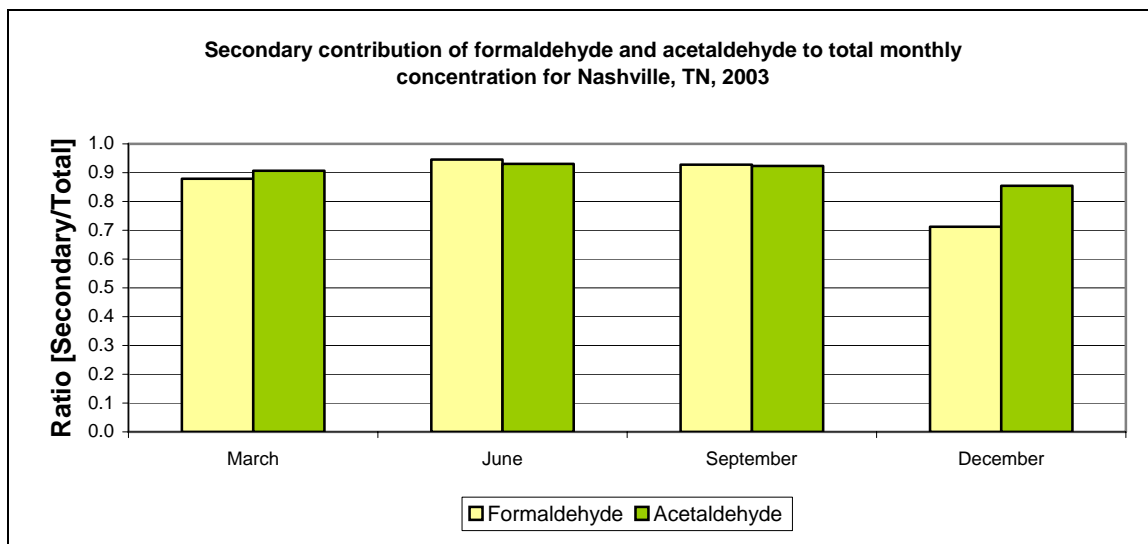
Figure 4.55. Secondary Contribution of Formaldehyde and Acetaldehyde to Total Monthly Concentration at Nashville, TN, 2003

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4.3.1.3 Acrolein

Acrolein was not well modeled by CMAQ, since it did not generate primary concentrations during March, September, and December; all of them were secondary acrolein. However, June generated 67% of secondary and 33% of primary acrolein. This could indicate that CMAQ does not simulate properly this kind of pollutant and suggests that CMAQ should be fixed to solve the problem.

Hourly acrolein concentrations at Nashville performed similar seasonal patterns for most of the 24-hour time periods for each month, as shown in Figure 4.56, where maximum concentrations occurred between 6 and 9 PM and the minimum concentrations occurred between 1 and 2 PM in March, June, and September, and around 5 AM in December. A second maximum occurred between 7 and 10 AM.

On the other hand, the daily acrolein concentrations were similar for each analyzed month, where the annual concentration was 0.03 ug/m^3 as shown in Figure 4.57. This concentration could be under estimated because of that CMAQ performance and the possible under estimated acrolein emissions for Tennessee discussed in point 4.1.
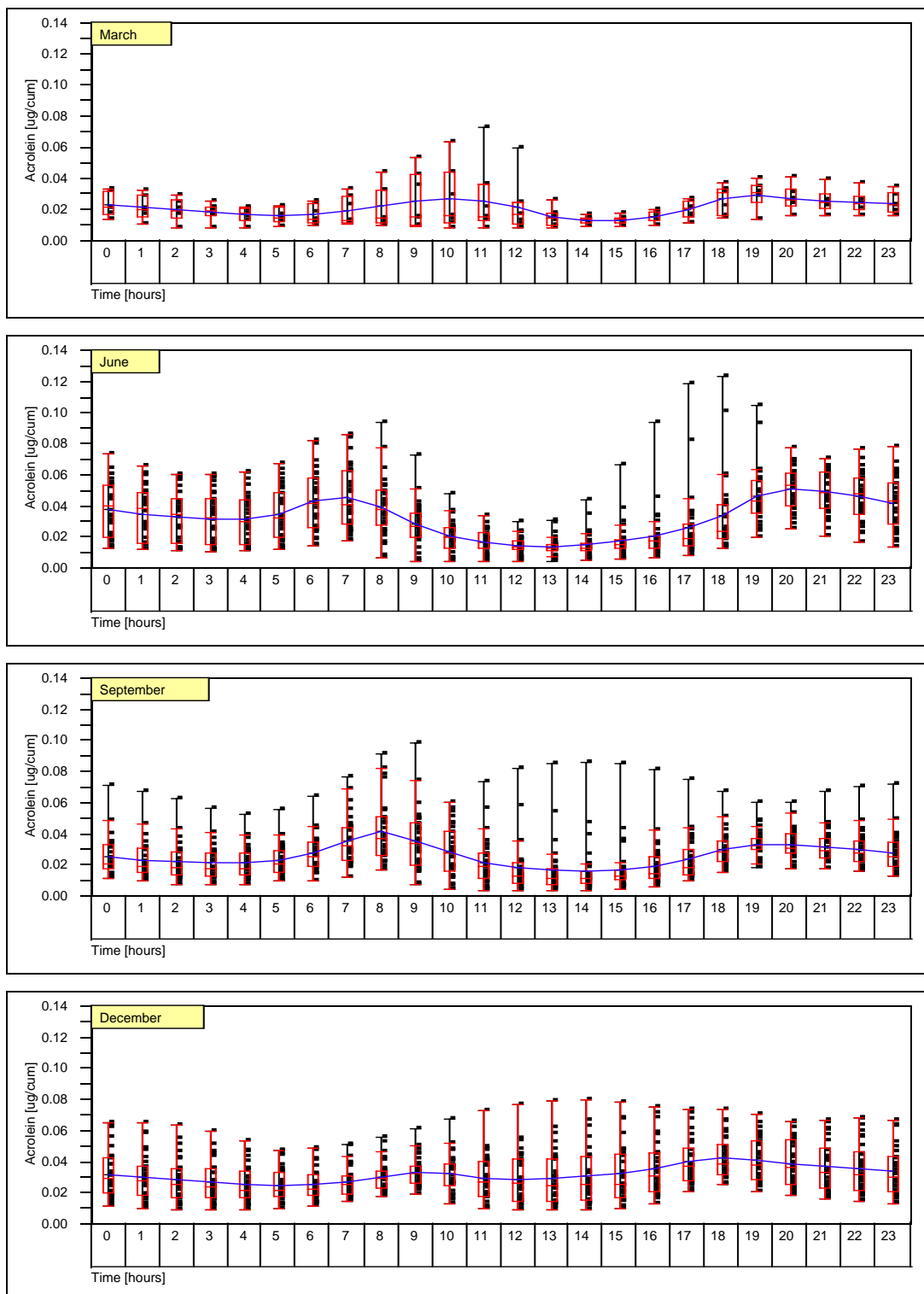
Figure 4.56. Box-Plots of the Modeled Hourly Average Acrolein Concentration at Nashville, TN, for March, June, September, and December 2003.
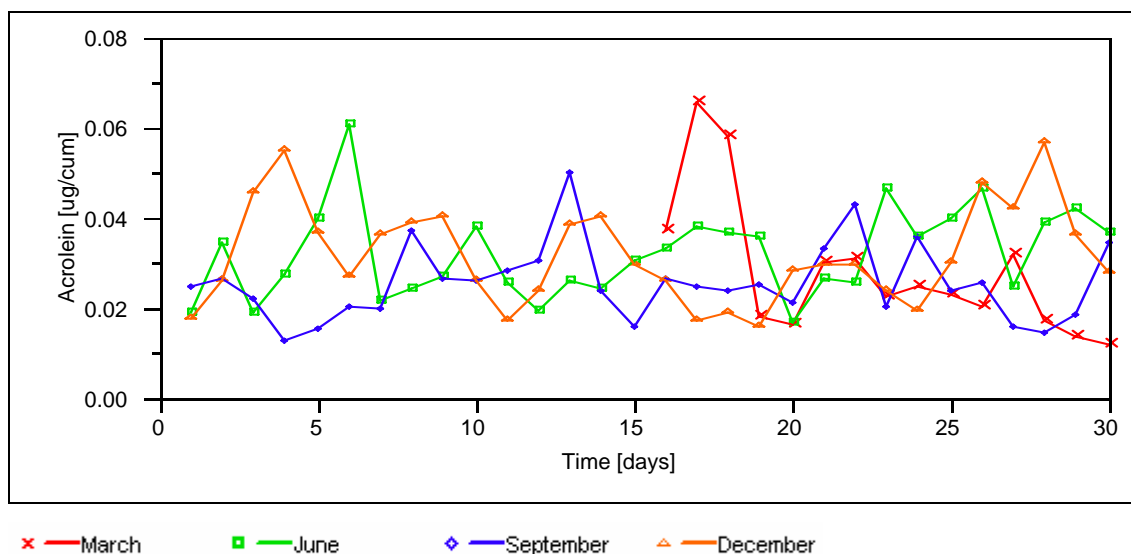
Figure 4.57. Modeled Daily Acrolein Concentration in Nashville, TN, 2003

4.3.1.4   Benzene and 1,3-Butadiene

Hourly benzene and 1,3-butadiene concentrations at Nashville produced similar seasonal patterns for the most of the 24-hour time periods for each month, as shown in Figures 4.58 and 4.59. The maximum concentrations occurred between 6 and 8 PM and the minimum concentrations occurred between 1 and 2 PM in March, June, and September, and around 5 AM in December.

A second maximum occurred between 7 and 10 AM. It may be noted that benzene and 1,3-butadiene are generated as primary pollutants, and the chemical reactions at the troposphere for this kind of hydrocarbon are normally slow but higher during the summer season, where the high temperatures speed up their overall decomposition.

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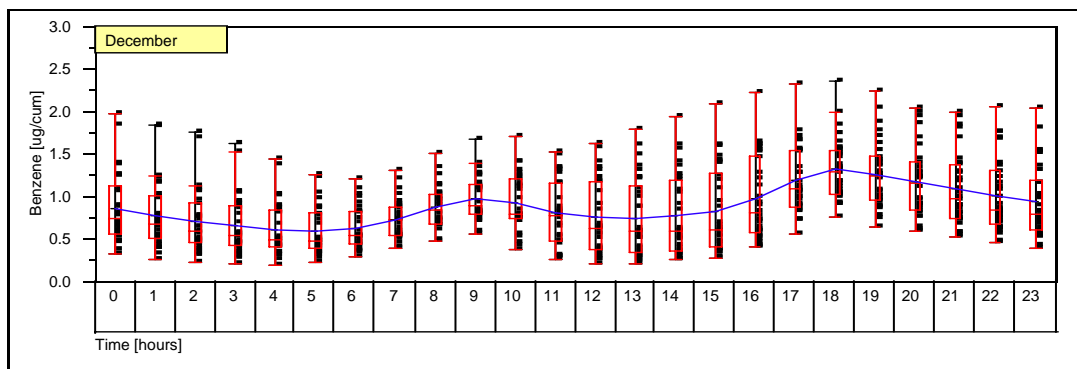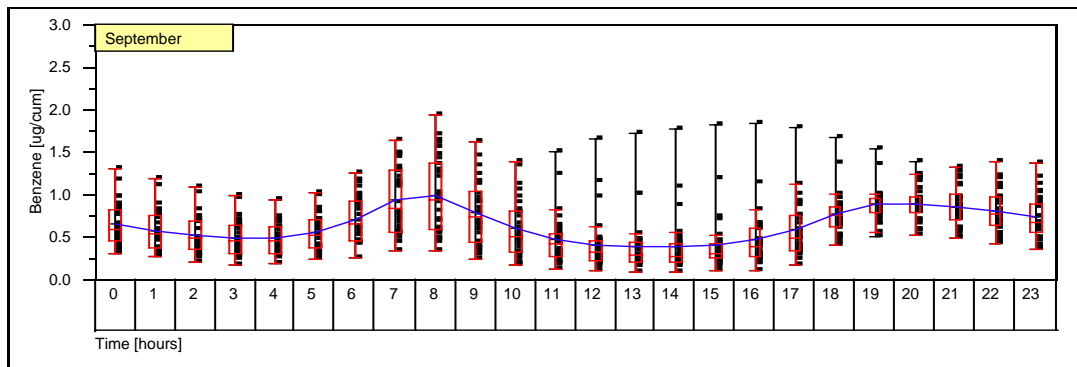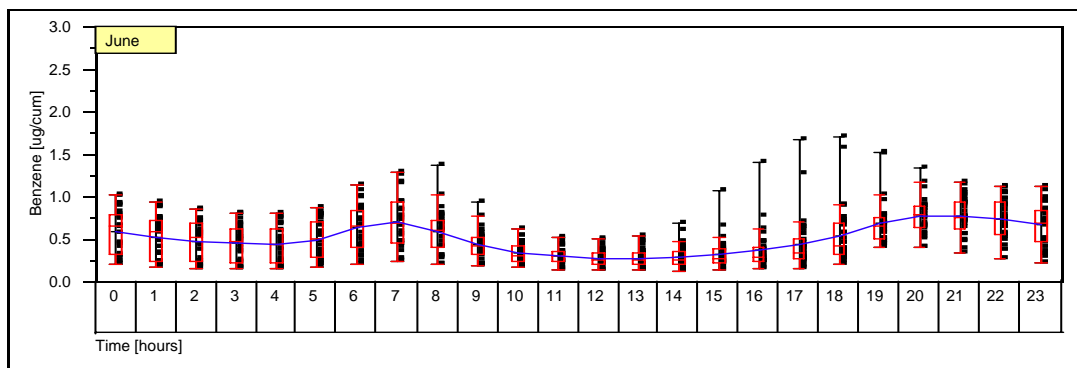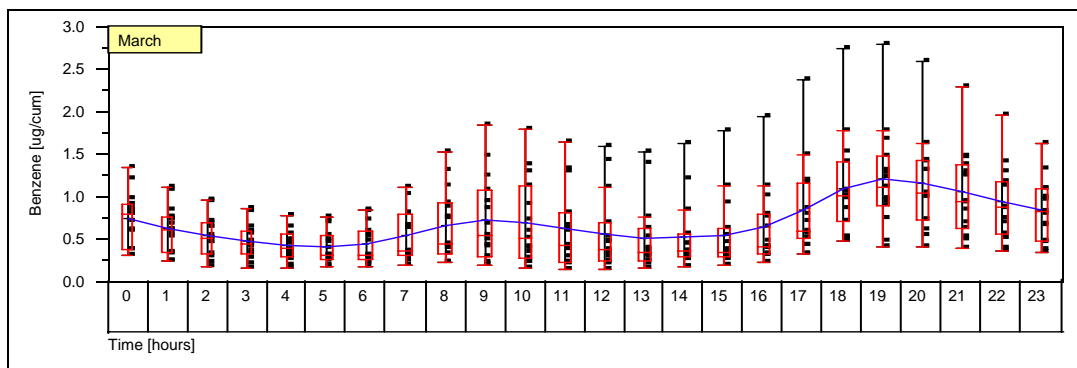
Figure 4.58. Box-Plots of the Modeled Hourly Average Benzene Concentration at Nashville, TN, for March, June, September, and December 2003.
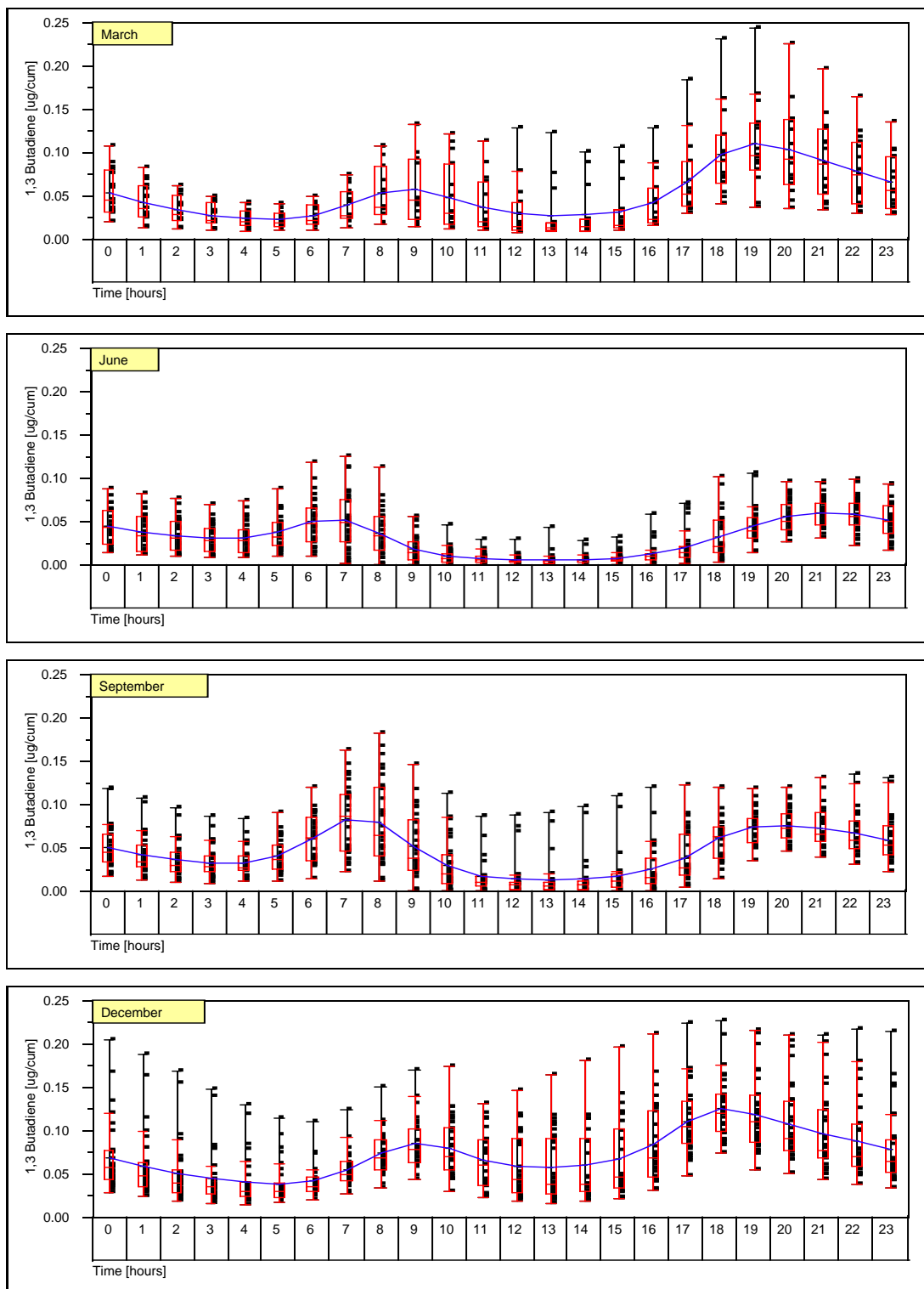
Figure 4.59. Box-Plots of the Modeled Hourly Average 1,3-Butadiene Concentration at Nashville, TN, for March, June, September, and December 2003

In fact, benzene and 1,3-butadiene are primary pollutants generated mainly from mobile sources, whose maximum emissions occur at traffic peak hours in Davidson County, i.e, from 7 AM to 7 PM, with the highest emissions at 5 PM. The main sinks of that emitted primary benzene during the summer season are the OH• radical and the afternoon dispersion.

Primary benzene emitted during morning rush-hour traffic reacted slowly with OH• during the daytime and was dispersed at around 1PM because of the wind seed, as shown in Figures 4.35 and 4.58. Those box-plots show that a certain amount of the benzene emitted at morning rush-hour traffic was decomposed and dispersed at noon (Bloss et al., 2005; Seinfeld and Pandis, 1998). At night, the benzene emitted during afternoon rush-hour traffic reacted very slowly with OH• and was slowly dispersed until the new benzene was emitted during morning traffic congestion again, as shown in Figures 4.35 and 4.58 during June and September.

This chemical mechanism is explained also for some authors (Bloss et al., 2005; Seinfeld and Pandis, 1998). On the other hand, the 1,3-butadiene emitted during morning rush-hour traffic reacted rapidly with OH• to generate organic radicals during daytime in the summer season, as well as. It also reacted with ozone and was dispersed principally at afternoon hours due to higher wind speeds, as show in Figures 4.35 and 4.59 during June and September.

Those box-plots show that most of the 1,3-butadiene emitted at morning traffic congestion was decomposed and dispersed around 1 PM. At night, the 1,3-butadiene emitted during afternoon rush-hour traffic reacted slowly with $NO_3^\bullet$ and slowly was dispersed, because of a greater atmospheric stability, until the new 1,3-butadiene was emitted during morning rush-hour traffic again, as shown in Figures 4.35 and 4.59 for June and September (Bloss et al., 2005; Seinfeld and Pandis, 1998).

During cold months, less benzene and 1,3-butadiene were decomposed due to the lack of enough $OH^\bullet$. At the same time, more benzene and 1,3-butadiene were dispersed due to the higher wind speed during December, as shown in Figures 4.35, 4.58 and 4.59 for March and December. As a result, the total benzene and 1,3-butadiene concentrations during March and December were higher than June and September. For example, benzene concentrations in December were 70% higher than the June concentration as shown in Table 4-16.

It may be noted that at daytime in the summer season the population was less exposed to benzene and 1,3-butadiene concentrations. Finally, the annual modeled benzene and 1,3-butadiene concentrations were 0.68 and 0.05 ug/m^3 respectively at Nashville, TN, 2003, as shown in Figures 4.60 and 4.61 and Table 4.16.

Table 4-16. Modeled Benzene and 1,3-Butadiene Concentrations at Nashville, Davidson County, TN, 2003

Period	Benzene [ug/m^3]	1,3-Butadiene [ug/m^3]
March	0.69	0.05
June	0.51	0.03
September	0.64	0.05
December	0.88	0.07
Annual	**0.68**	**0.05**
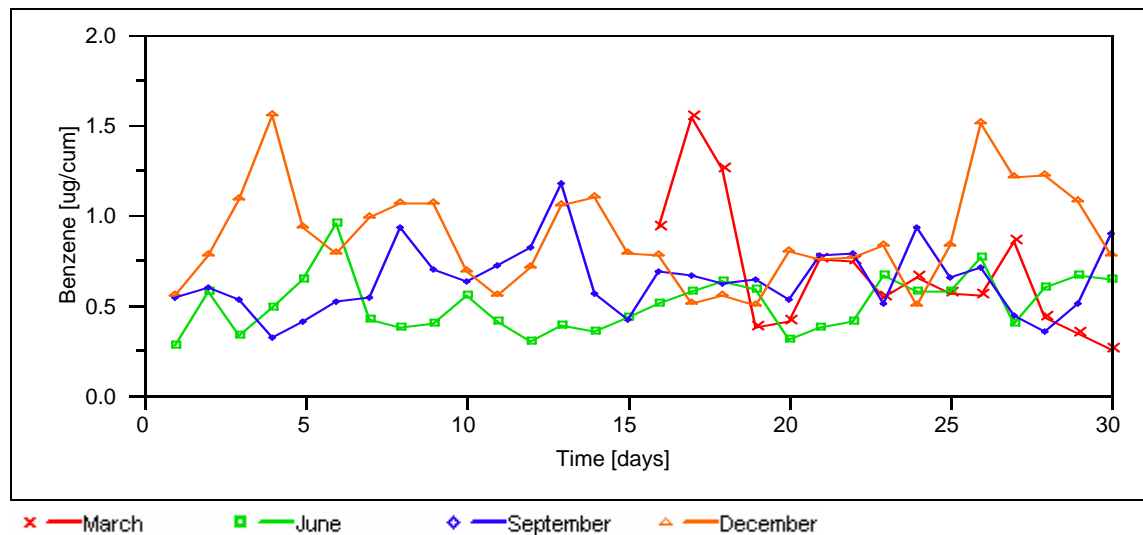
Figure 4.60. Modeled Daily Benzene Concentration in Nashville, TN, 2003
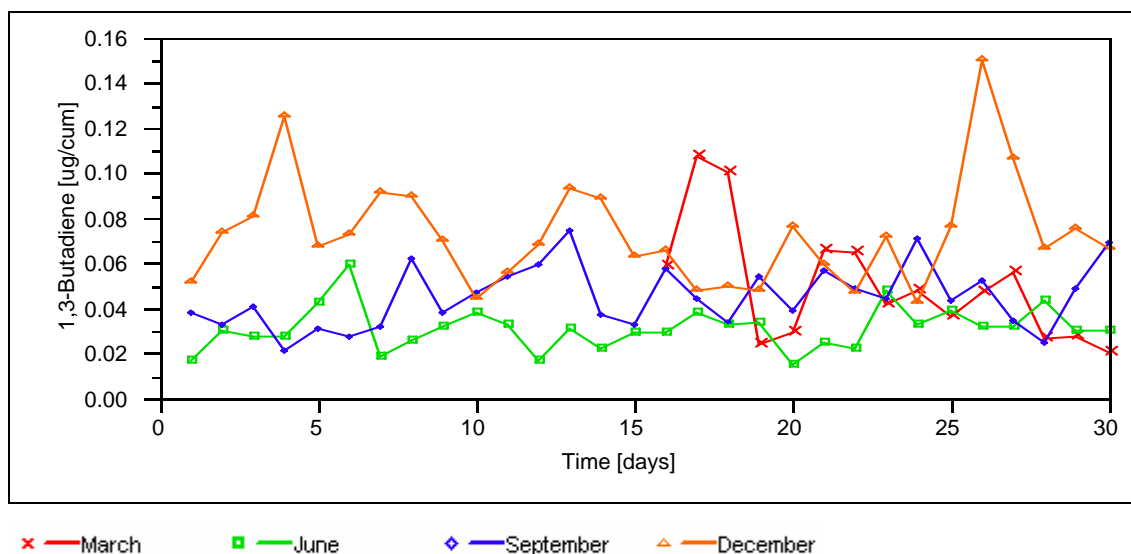
Figure 4.61. Modeled Daily 1,3-Butadiene Concentration in Nashville, TN, 2003

4.3.1.5 EC and DPM

The next analysis includes EC and DPM together because EC is the major component of

DPM (Adones et al., 2003, and Schauer, 2003). This analysis estimated the DPM by

eliminating diesel-fueled sources on the modeling domain, while EC was estimated using

all sources in the base case. As a result, hourly EC and DPM concentrations at Nashville

produced similar seasonal patterns for most of the 24-hour time periods for each month,

as shown in Figures 4.62 and 4.63. The maximum concentrations occurred between 6 and

9 PM and the minimum concentrations occurred between 1 and 3 PM. A second

maximum occurred between 6 and 10 AM. It may be noted that EC and DPM are

generated as primary pollutants. In fact, EC and DPM in nature are primary pollutants

generated mainly from diesel mobile sources, whose maximum modeled EC emissions
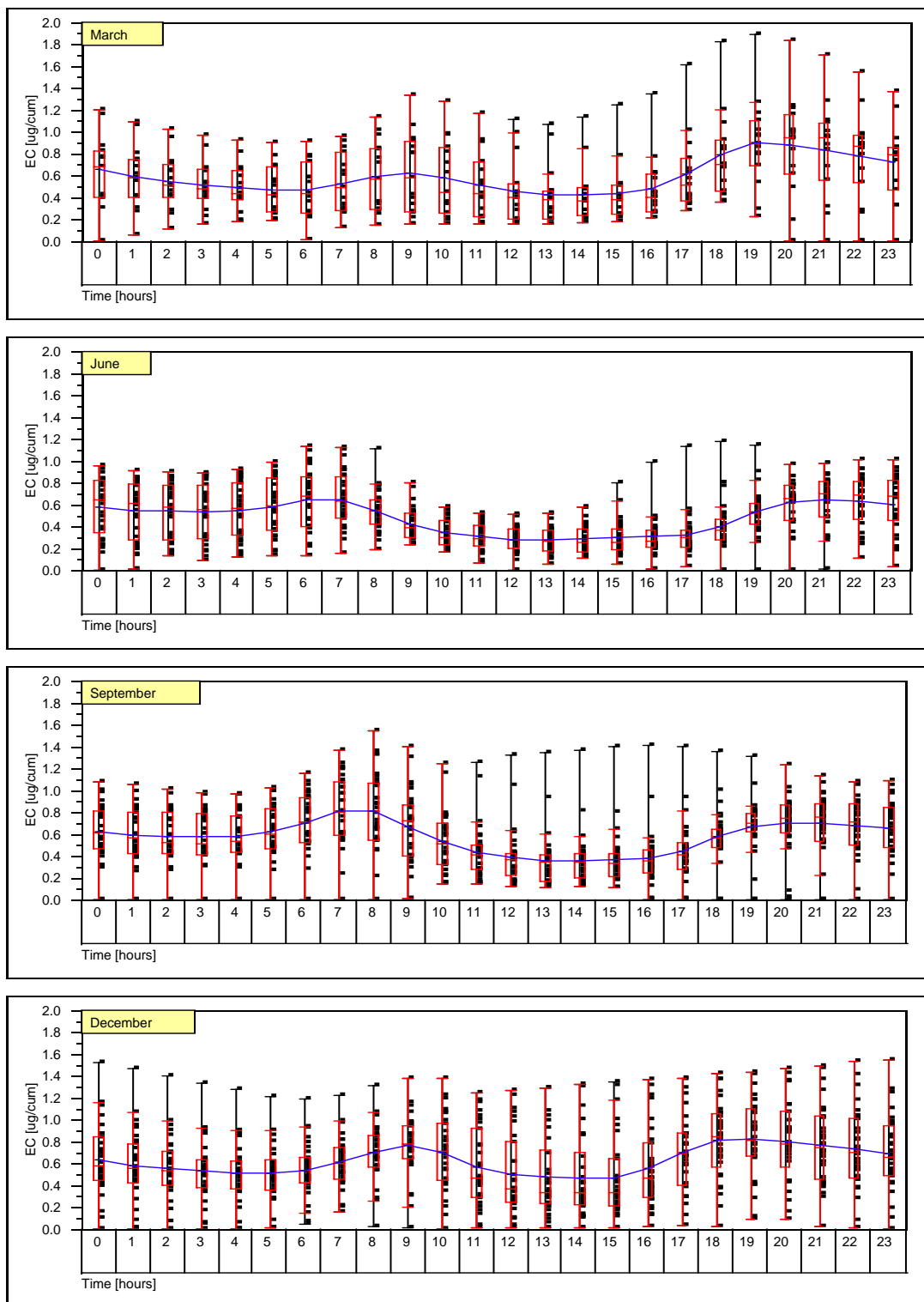
Figure 4.62. Box-Plots of the Modeled Hourly Average EC Concentration at

Nashville, TN, for March, June, September, and December 2003

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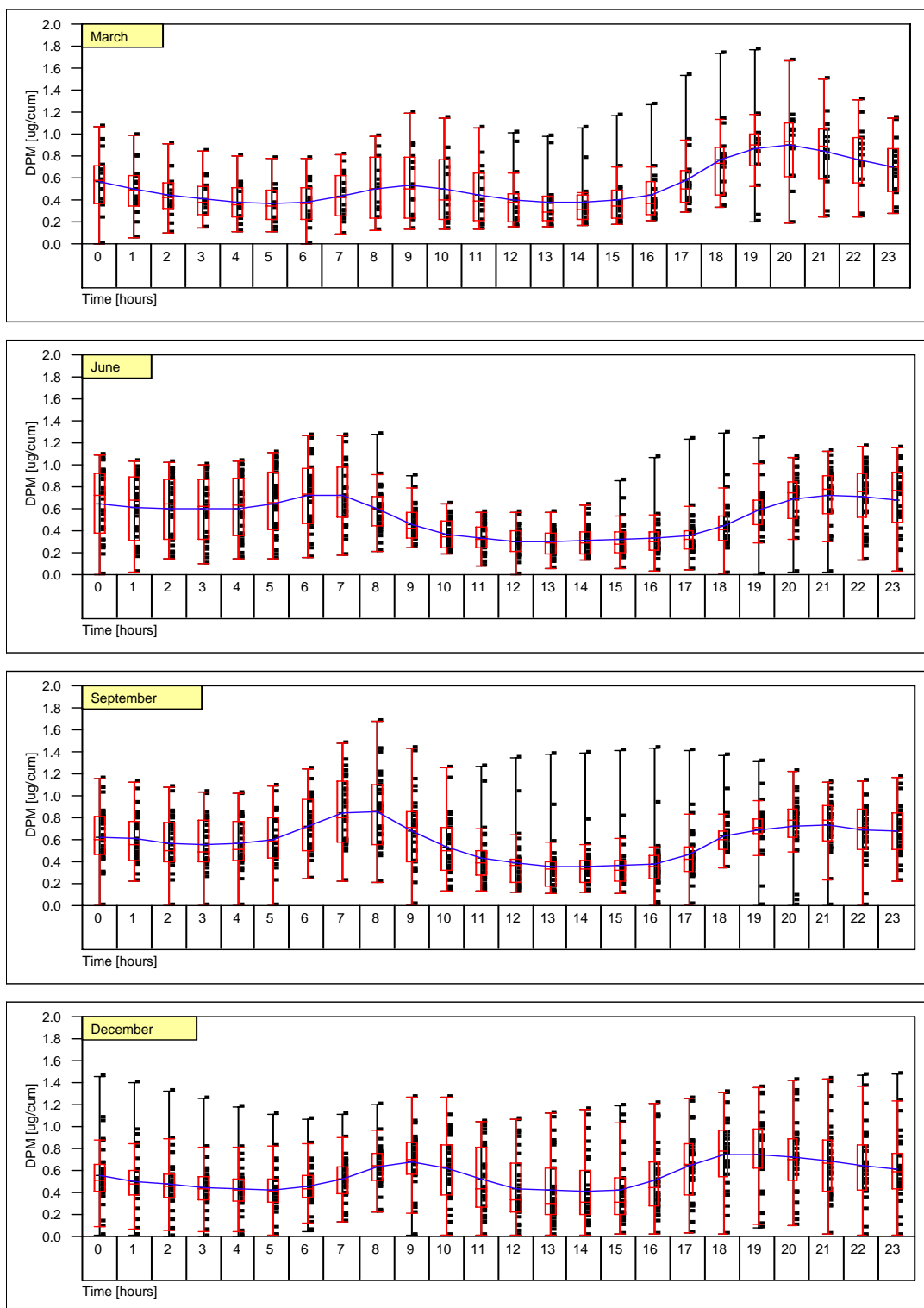
**Figure 4.63. Box-Plots of the Modeled Hourly Average DPM Concentration at**

Nashville, TN, for March, June, September, and December 2003

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occurred at traffic peak hours in Davidson County, i.e, from 7 AM and 5 PM, with the highest emissions at 5 PM. Those temporal results were obtained because SMOKE2.0 uses a default on-road temporal profile for all vehicles emissions, including diesel truck. This could be not be true, since on highways the maximum volume of trucks per hour occurs at 3 PM for the most of the days, including weekends (Miller, 2005). This more realistic temporal profile for trucks could be incorporated in SMOKE to produce better temporal hourly emissions and concentrations of DPM at places close to highways. As in Davidson County not only are there heavy-duty diesel trucks, but also there are diesel buses, diesel SUVs, and light diesel cars on the roads. The default SMOKE2.0 on-road temporal emissions profile can be considered appropriated for this research.

The main factor to dilute the emitted primary EC and DPM is the wind dispersion at afternoon hours. Figures 4.35, 4.62, and 4.63 show that an important amount of EC and DPM emitted during morning rush-hour traffic were dispersed at around 2 PM as a result of the higher wind speeds during afternoon hours. This particulate matter behavior was also described by Weber (2003) for Atlanta, GA. At the end of the day and at night, the EC and DPM emitted during afternoon rush-hour traffic dropped and dispersed slowly until the new EC and DPM were emitted during morning traffic congestion again, as shown in Figures 4.62 and 4.63. It can be noted that in general, the population of Nashville was more exposed to DPM at morning and afternoon rush-hour traffic. The monthly EC and DPM concentrations were almost similar for all months in Davidson County, principally for DPM. However, EC showed a slightly difference between hot and cold months, since the EC concentration of December was 30% higher than June.

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It could be because in December more wood is burned in homes, demonstrating the seasonal impact of wood smoke on EC concentrations published by Zheng et al., (2002). The monitored annual EC concentration was 0.64 ug/m^3 (Appendix C), while the annual modeled EC and DPM concentrations were 0.57 and 0.55 ug/m^3, respectively at Nashville, TN, 2003, as shown in Figures 4.64 and 4.65 and Table 4.17. Finally and according to Figure 4.66, it may be noted that the mean DPM contribution to the total hourly PM2.5 was higher during morning and afternoon rush-hour traffic, i.e., around 7 AM and 5 PM at Nashville. The minimum was produced around 1 PM. The highest hourly contribution was as high as 9.3% on June 13th at 8 AM. Higher hourly contribution variability was produced in June than in December, since in the summer season there is more variability on the SOA PM$_{2.5}$ formation due to the high temperature. The daily DPM contribution to the total PM$_{2.5}$ was similar for each month and was around 2.6% as shown in Figure 4.67. This figure also shows two high values produced on June 10th and 13th, which were around 5%.

Table 4-17. Modeled EC and DPM Concentrations at Nashville, Davidson County, TN, 2003

Period	EC [ug/m^3]	DPM [ug/m^3]
March	0.59	0.52
June	0.48	0.53
September	0.58	0.58
December	0.63	0.55
Annual	**0.57**	**0.55**
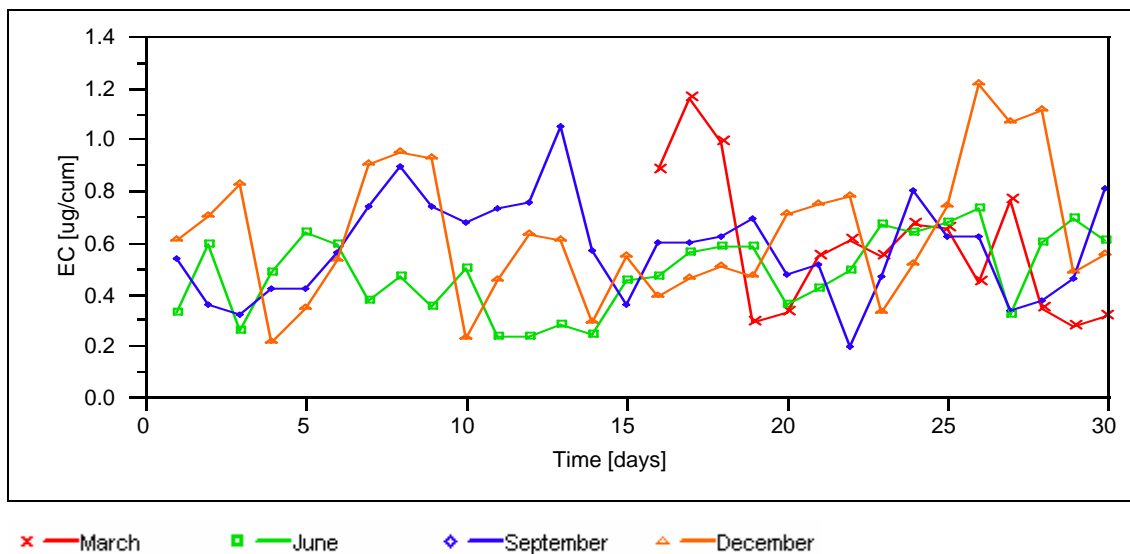
Figure 4.64. Modeled Daily EC Concentration in Nashville, TN, 2003
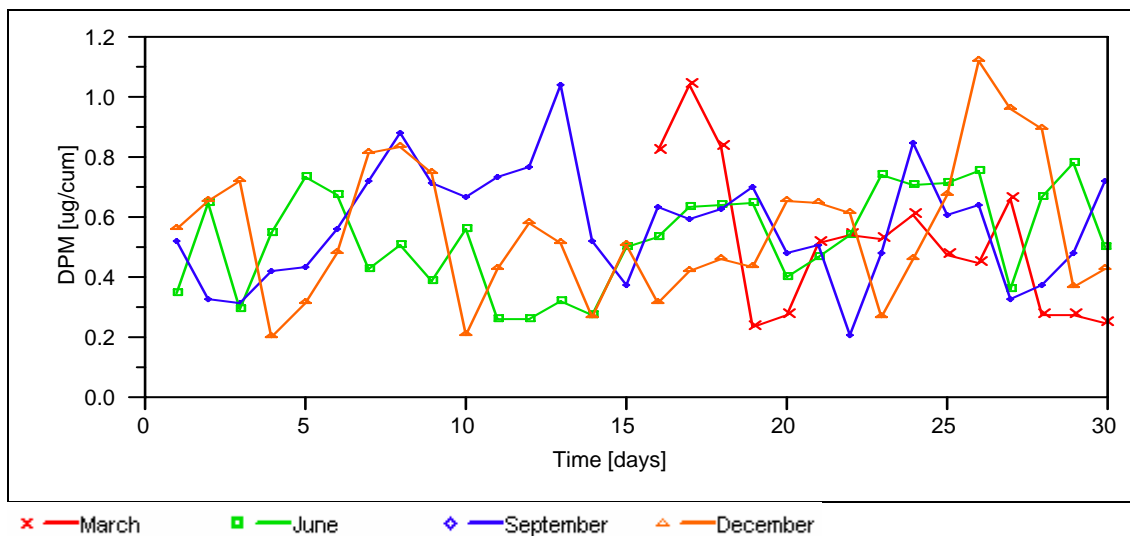
Figure 4.65. Modeled Daily DPM Concentration in Nashville, TN, 2003

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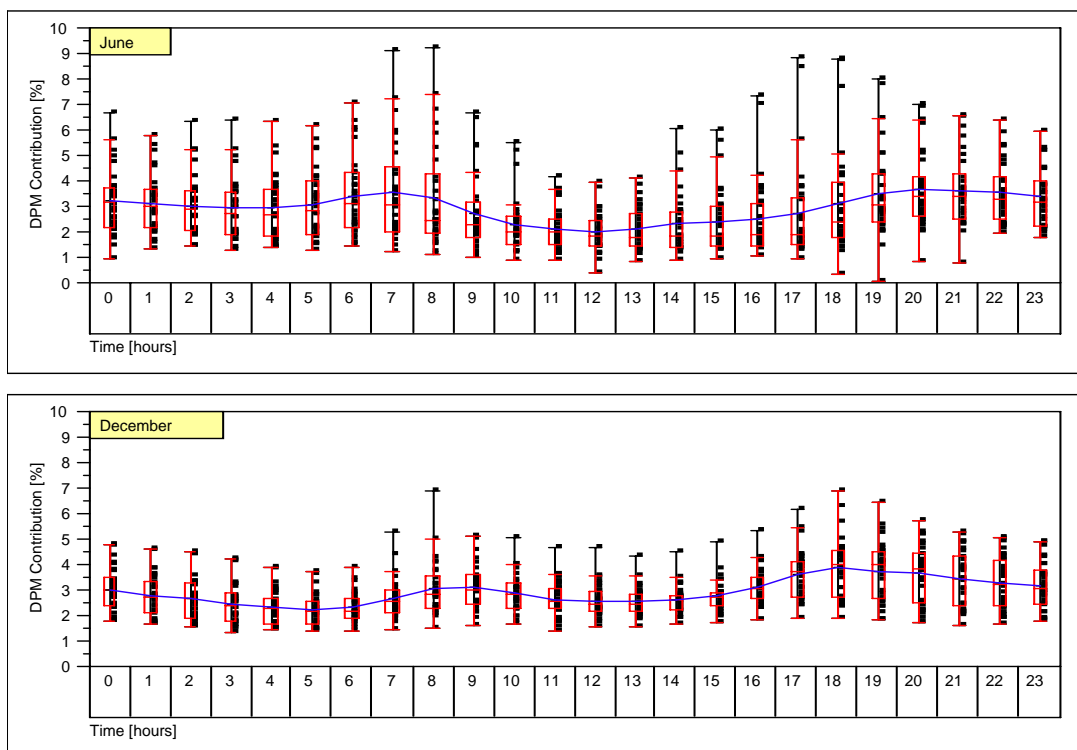
Figure 4.66. Modeled Hourly DPM Contribution to Total PM2.5 in Nashville, TN, June and December 2003
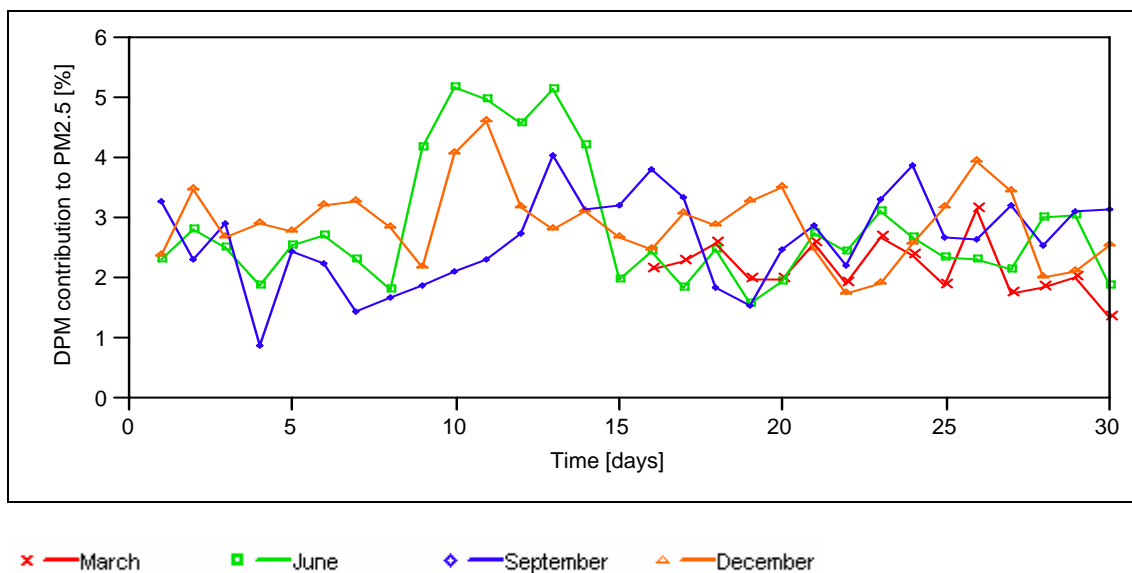
Figure 4.67. Modeled Daily DPM Contribution to Total PM2.5 in Nashville, TN, 2003

4.3.2 Spatial Distribution of Air Toxics Concentrations

The tile plots from Figures 4.68 to 4.75 show the spatial variation of acetaldehyde, formaldehyde, acrolein, benzene, 1,3-butadiene, elemental carbon, diesel particulate matter, and NOx for March, June, September, and December. It must be noted that the tile plots of monthly average concentrations were generated for 5 am GMT, which corresponds to midnight in central daylight savings time (CDT). In general, the plots show that higher concentrations occurred on Southeastern urban areas for the most of the pollutants, principally at Atlanta, GA, followed by Nashville, TN, Birmingham, AL, Raleigh, NC, and Memphis, TN. The exception was on formaldehyde during June and September (Figure 4.69), which showed high concentrations in the main part of Southeastern U.S. In addition, acrolein concentrations were clearly high in Florida as compared with neighbor states, as shown in Figure 4.70.

Acetaldehyde and formaldehyde produced higher concentrations in June and September due to the contribution of secondary formation in the Southeastern U.S. The rest of the pollutants generated higher concentrations during cold months, i.e., March and December, except DPM, which was similar for each month as shown in Figure 4.74. Those higher concentrations during those cold months were due mainly to the smaller tropospheric photochemical decomposition of those vapor HAPs and NOx, as well as, due to the seasonal impact of wood smoke on EC as shown in Figure 4.73.

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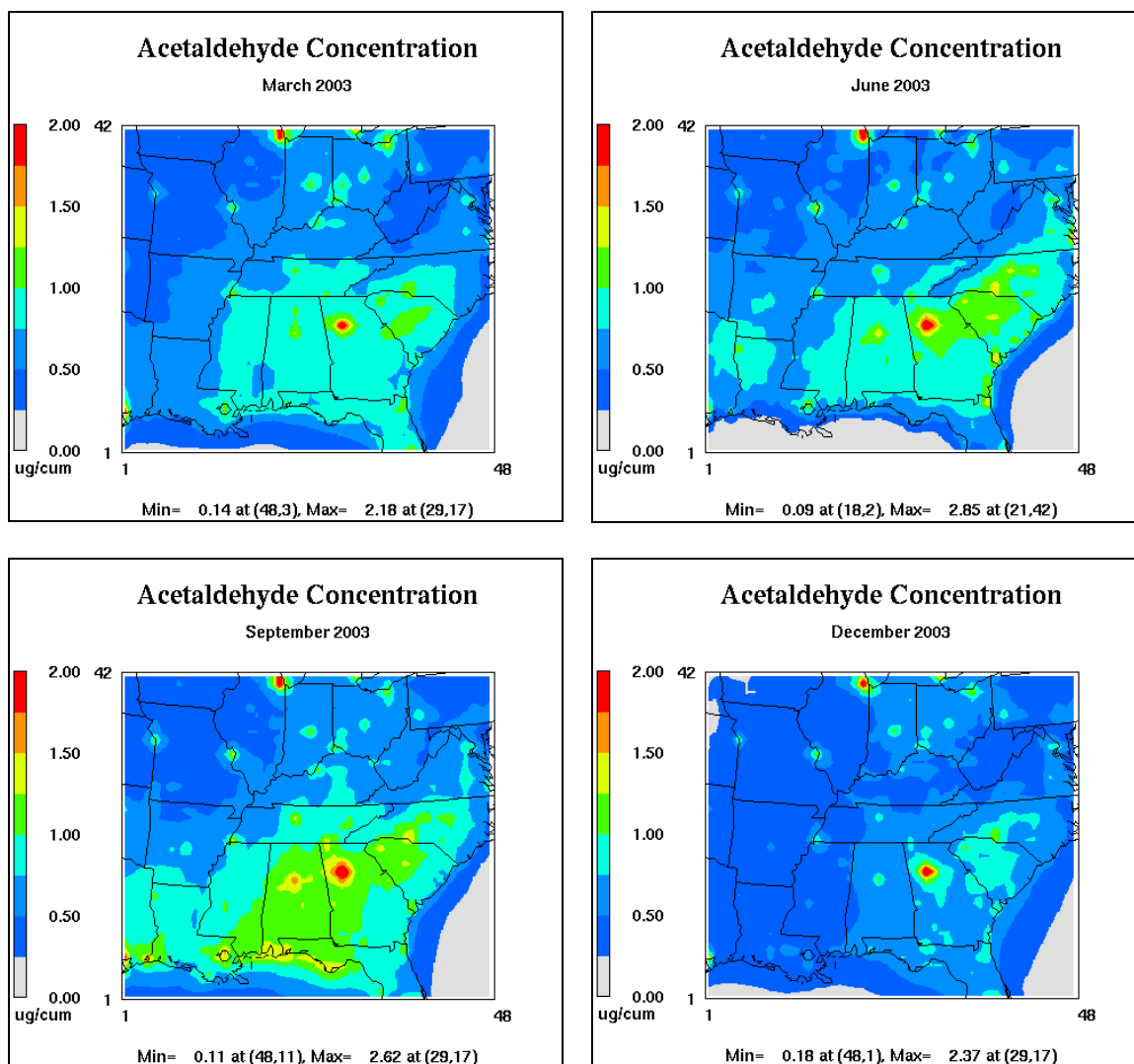
Figure 4.68. Modeled Monthly Acetaldehyde Concentration on the all 36-km Modeling Domain
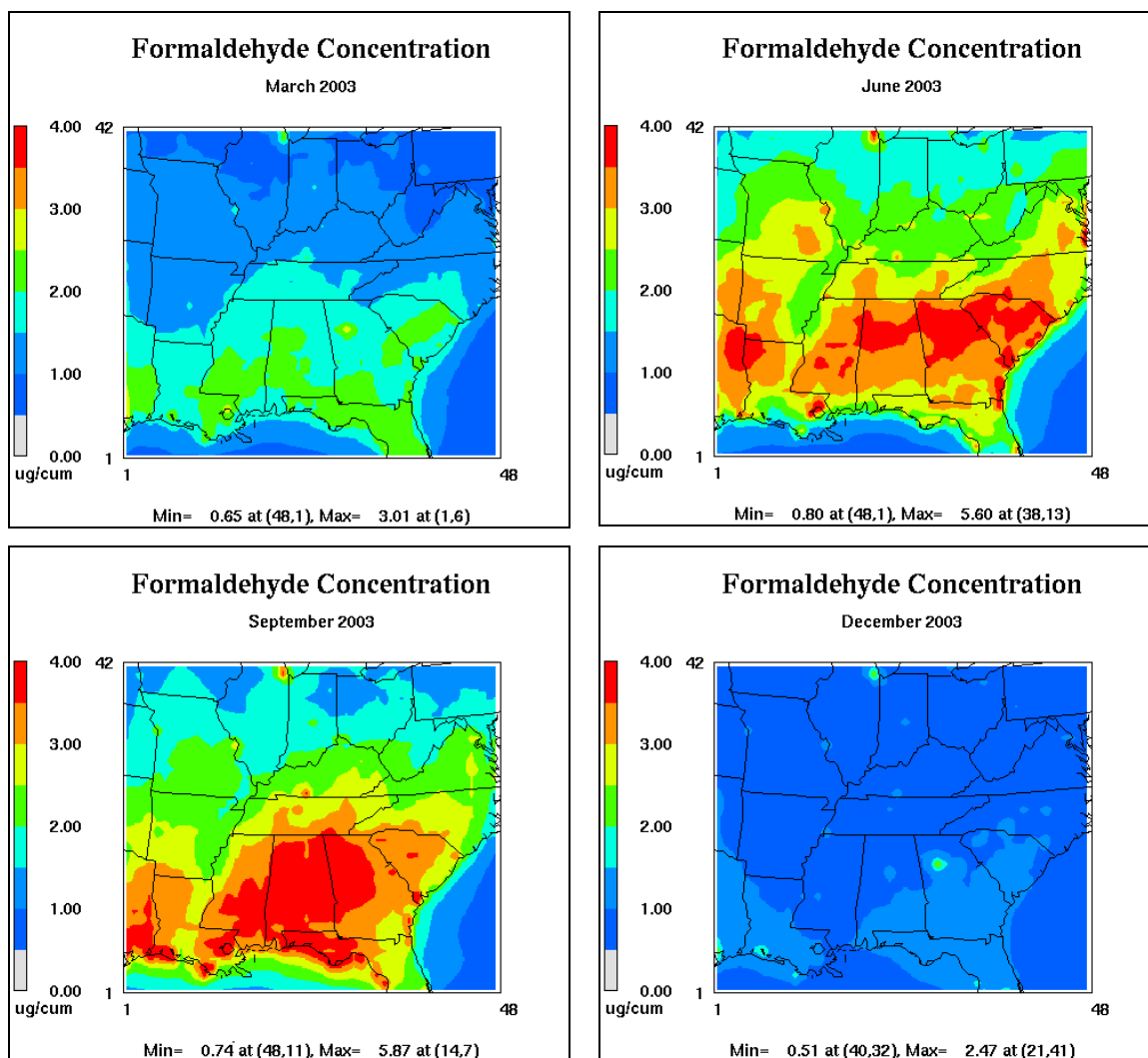
Figure 4.69. Modeled Monthly Formaldehyde Concentration on the all 36-km Modeling Domain
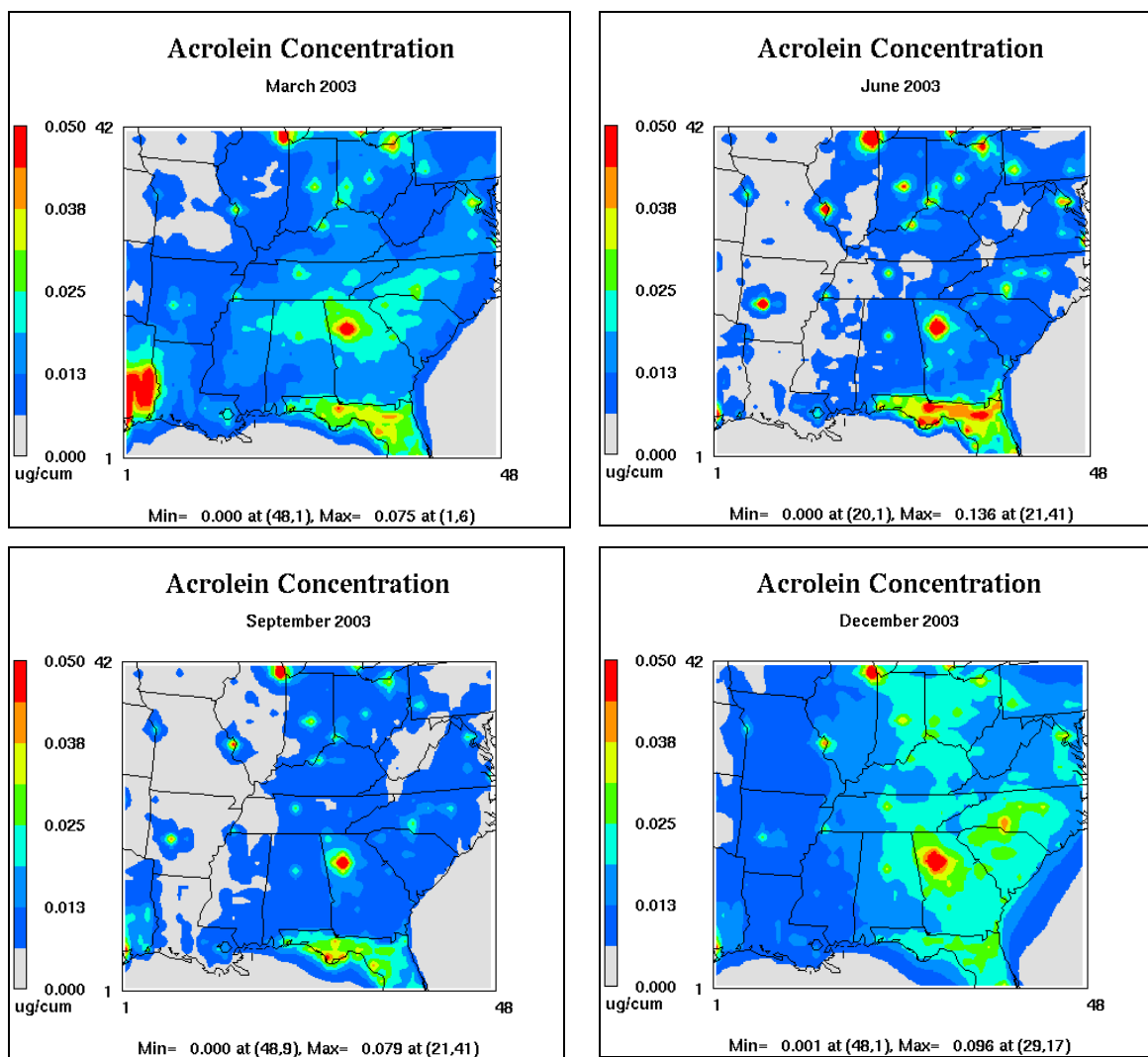
Figure 4.70. Modeled Monthly Acrolein Concentration on the all 36-km Modeling Domain
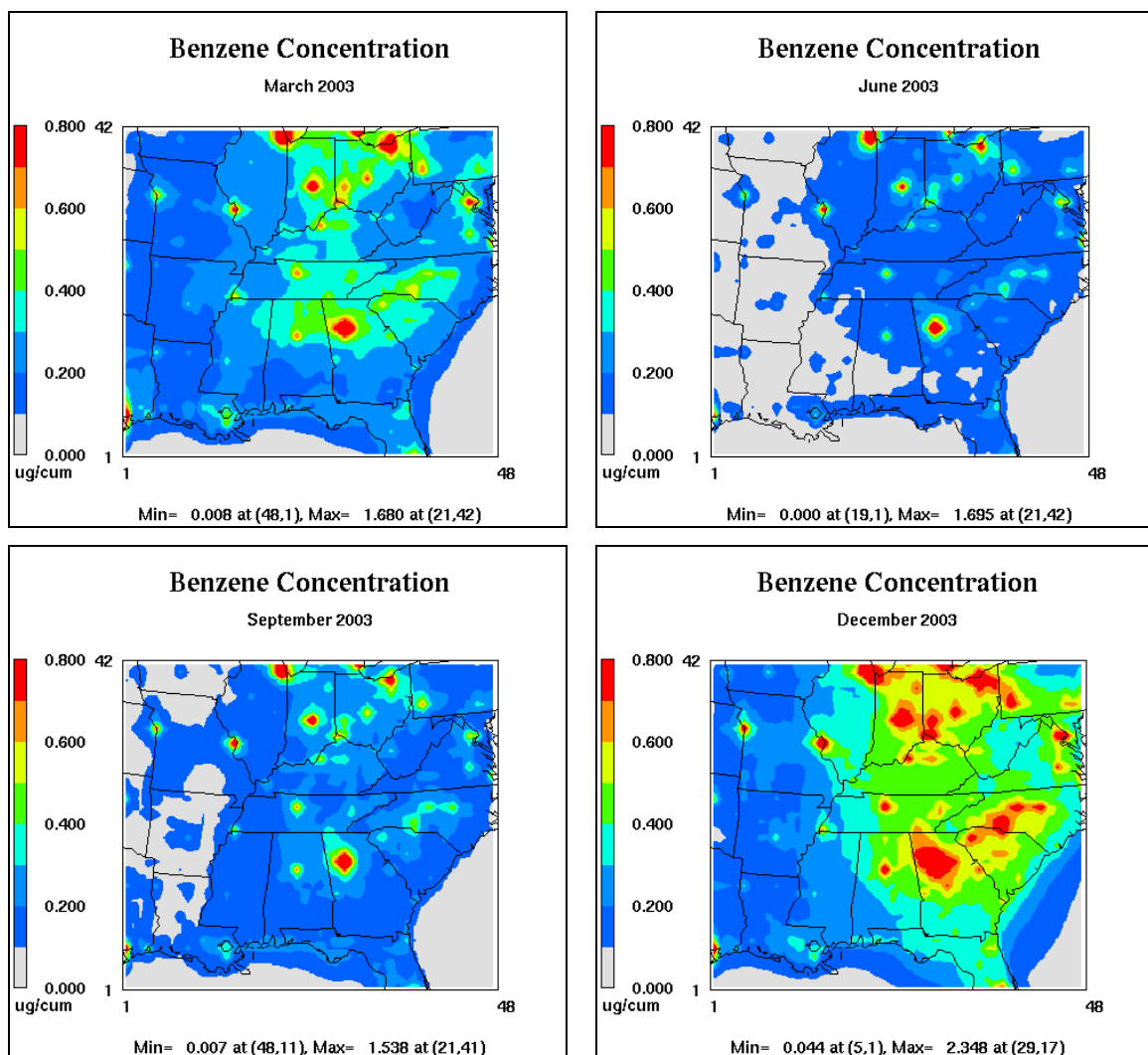
Figure 4.71. Modeled Monthly Benzene Concentration on the all 36-km Modeling Domain
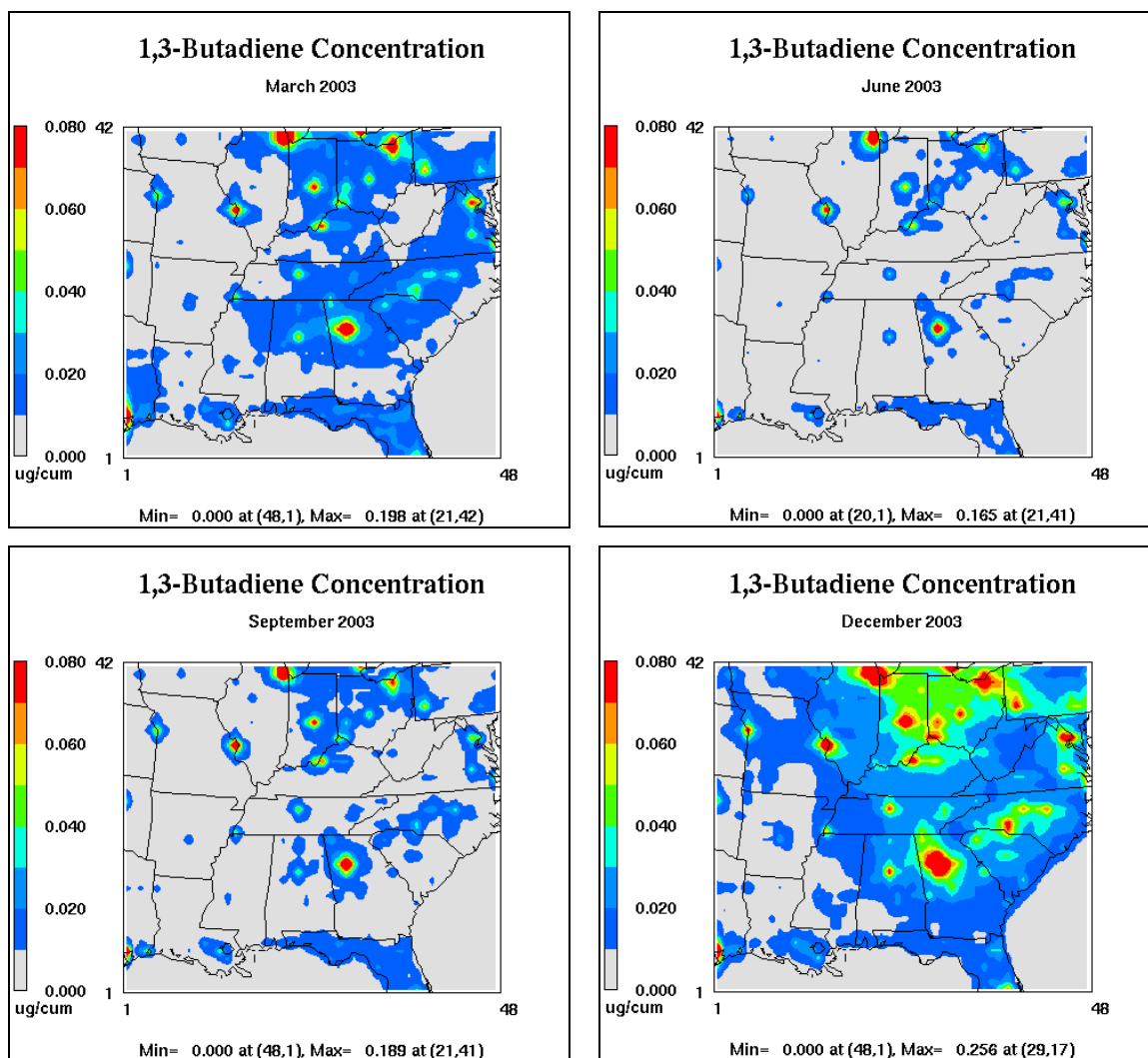
Figure 4.72. Modeled Monthly 1,3-Butadiene Concentration on the all 36-km Modeling Domain
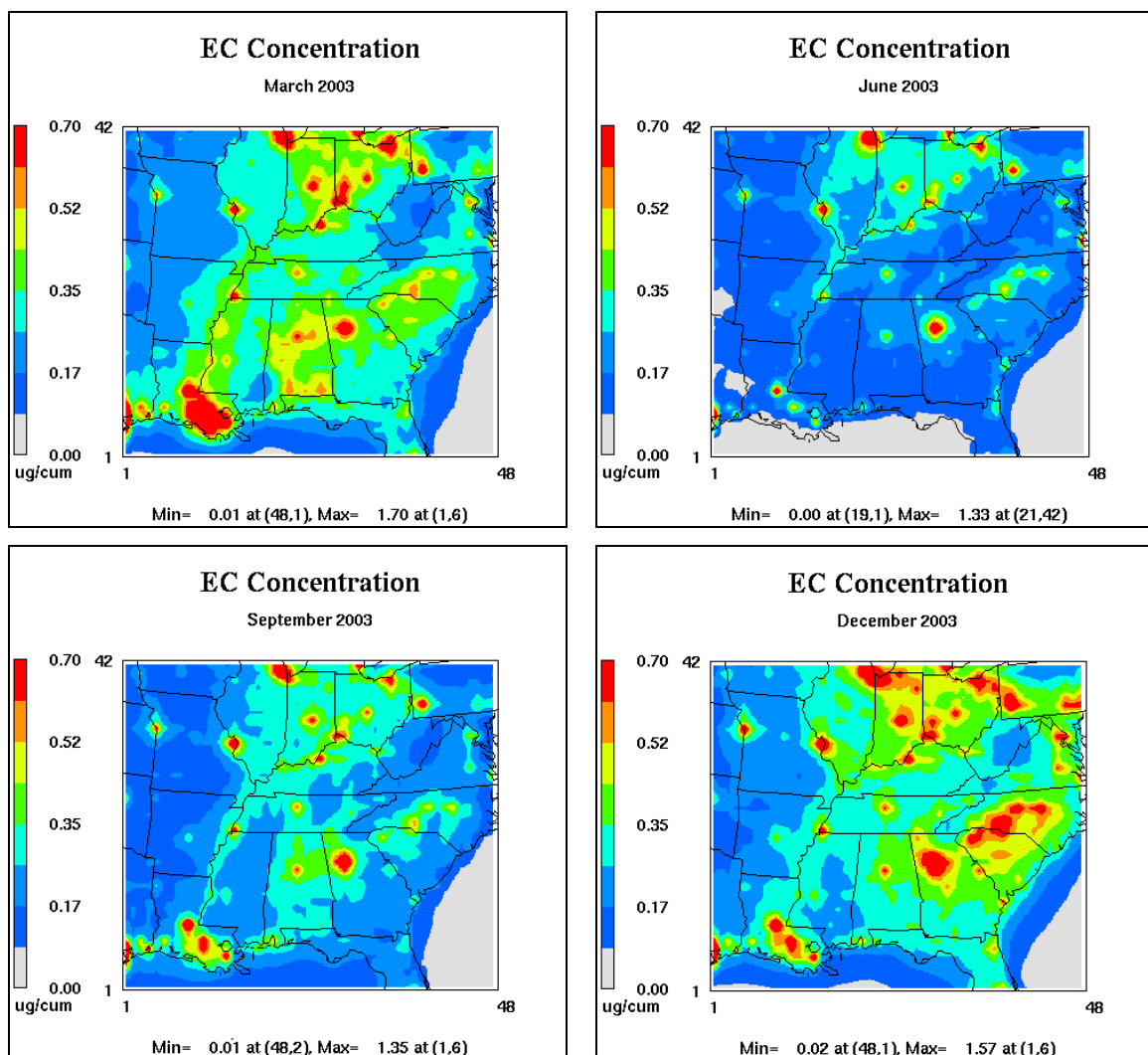
Figure 4.73. Modeled Monthly EC Concentration on the all 36-km Modeling Domain
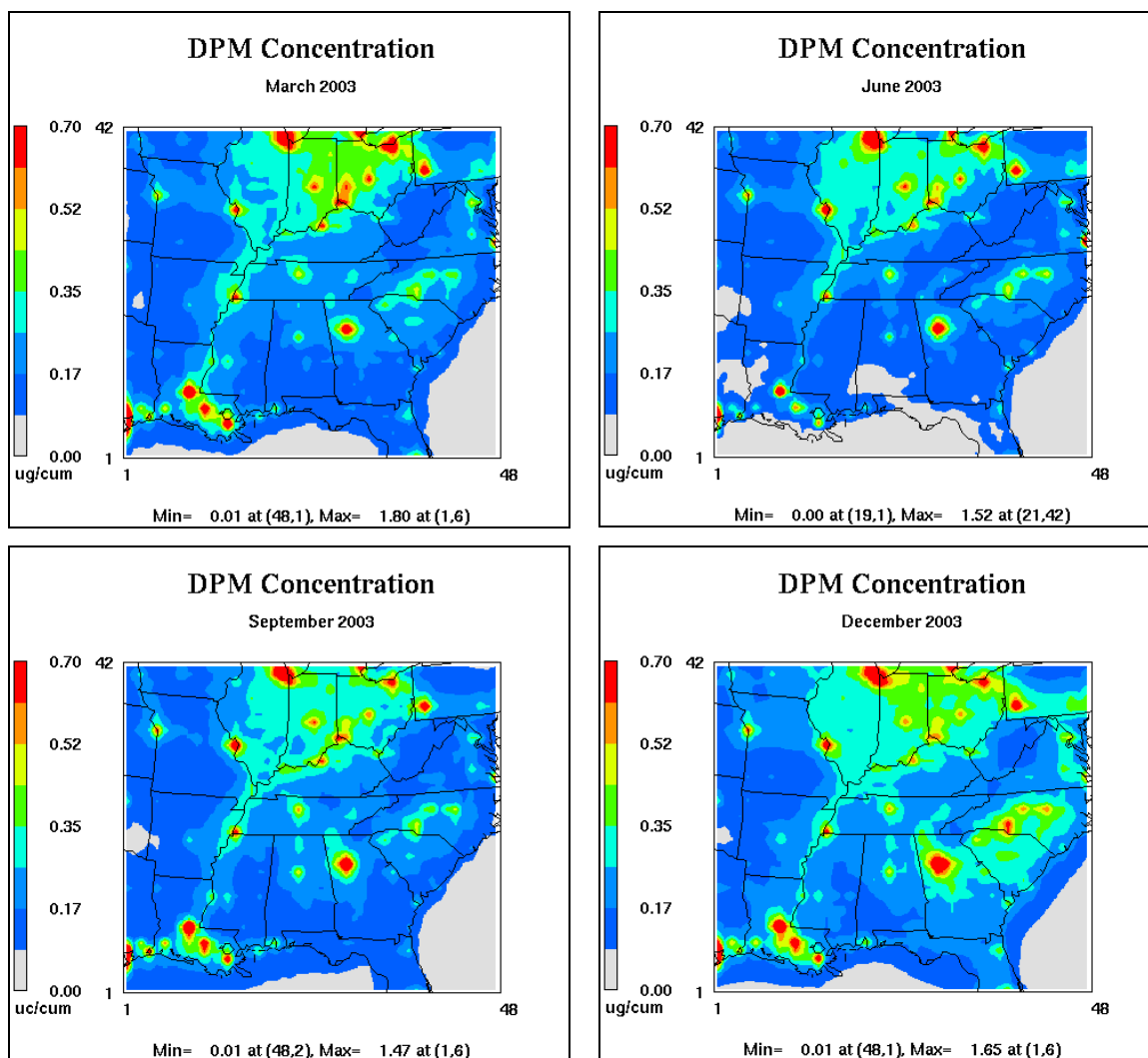
Figure 4.74. Modeled Monthly DPM Concentration on the all 36-km Modeling Domain
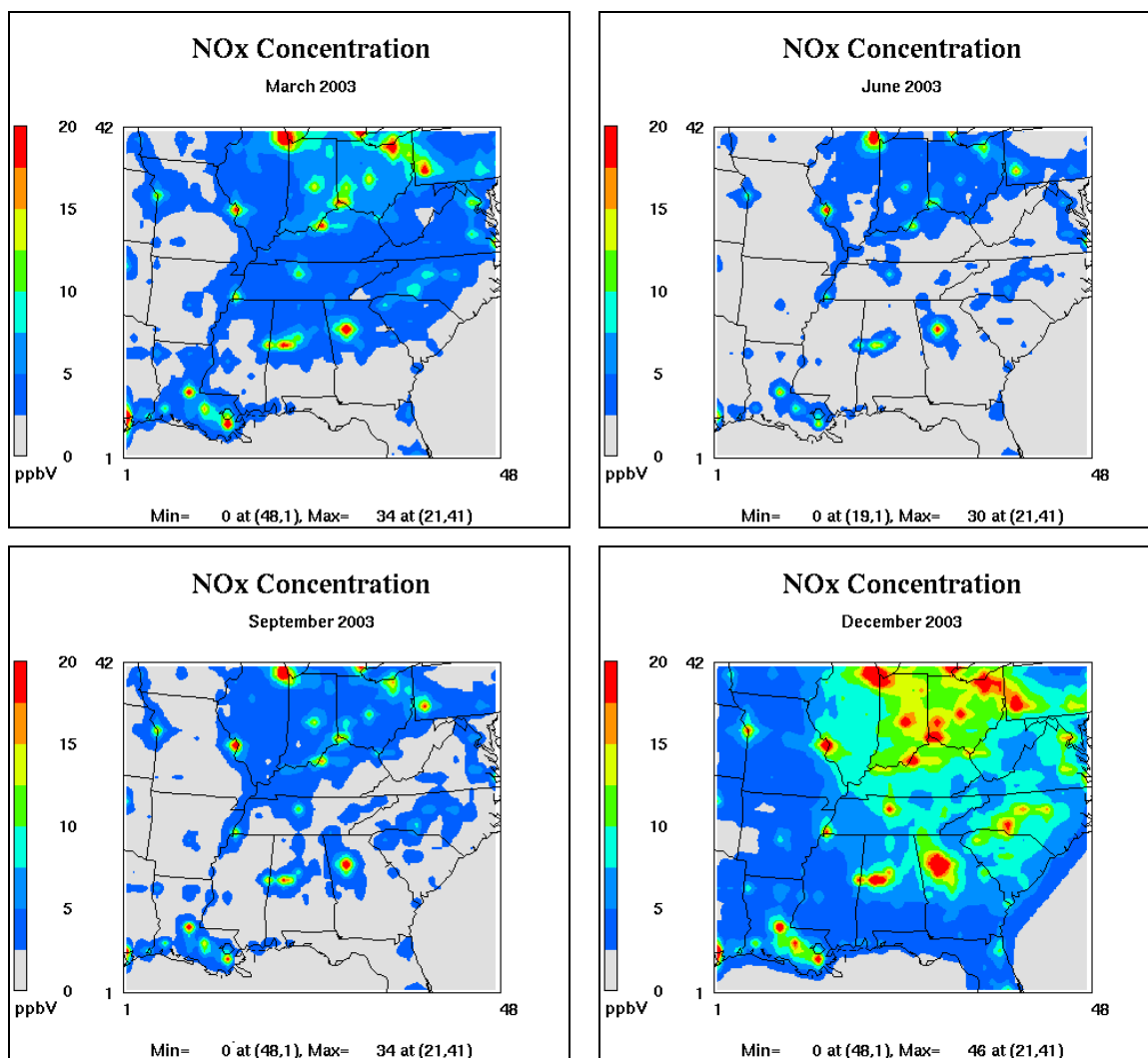
Figure 4.75. Modeled Monthly NOx Concentration on the all 36-km Modeling Domain

Finally, it can be noted that the population was more exposed to acetaldehyde and formaldehyde in June and September due mainly to the secondary contribution. On the other hand, the population was more exposed to benzene and 1,3-butadiene in March and December due to the reduced primary decomposition. For acrolein, the exposure is not clear, since CMAQ did not produce a good simulation for this HAP. Finally, the population was almost equally exposed to DPM during March, June, September, and December.

4.4 AIR TOXICS CONCENTRATIONS BY EMISSIONS SCENARIOS

The maximum reductions of daily concentrations for March, June, September, and December are shown from Figures 4-76 to 4.83 for acetaldehyde, formaldehyde, acrolein, benzene, 1,3-butadiene, EC, DPM, and NOx respectively at Nashville, TN. The annual concentrations and maximum reductions are shown in Table 4-18 and 4-19, respectively. It may be noted that the maximum reductions of daily acetaldehyde concentrations for Nashville were due to the scenario that did not consider on-road sources in the modeling domain with an average reduction of 57% and a maximum of 68% produced in December, as shown in Figure 4-76 and Table 4-19. This scenario was followed by the scenario without LDVs with an average reduction of 52%, the scenario without biogenic emissions with 50%, and the scenario for the year 2020 with 37%.
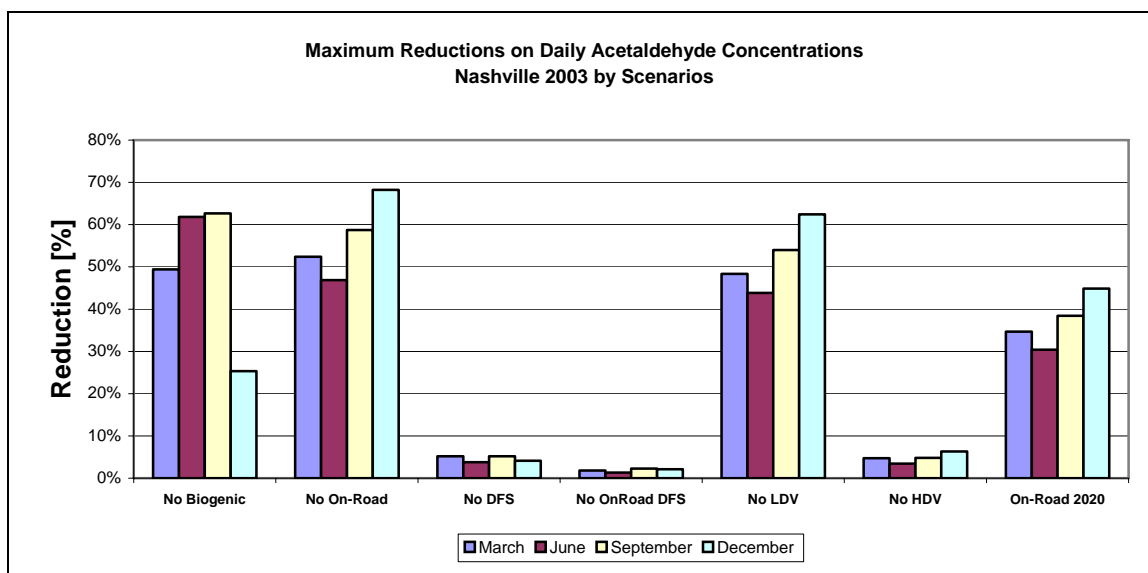
Figure 4.76. Maximum Reductions on Daily Acetaldehyde Concentrations in Nashville for March, June, September, and December 2003 by each Scenario
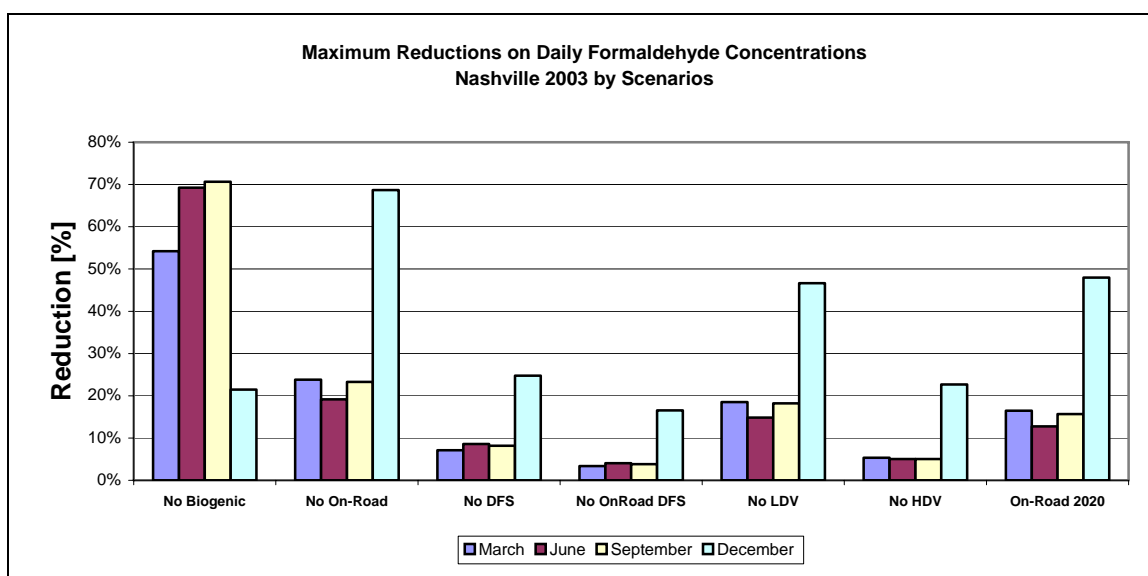
Figure 4.77. Maximum Reductions on Daily Formaldehyde Concentrations in Nashville for March, June, September, and December 2003 by each Scenario
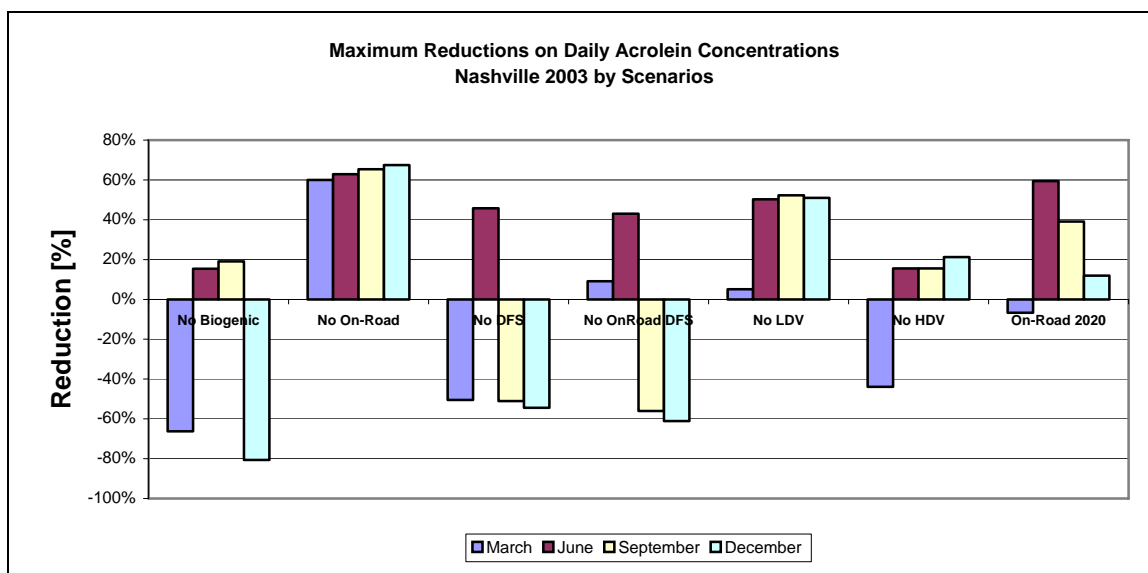
Figure 4.78. Maximum Reductions on Daily Acrolein Concentrations in Nashville

for March, June, September, and December 2003 by each Scenario
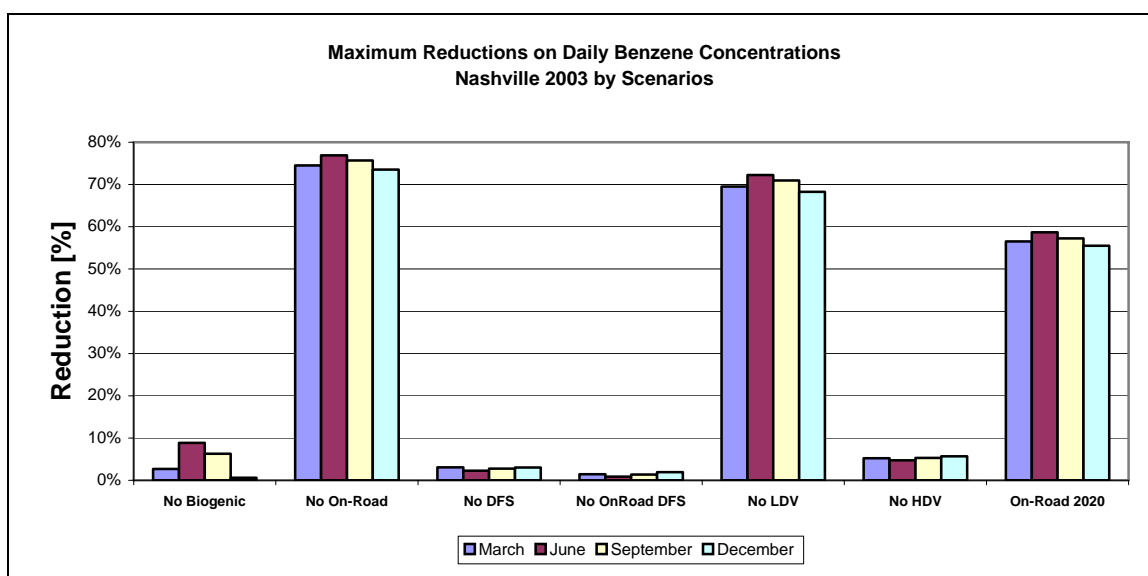
Figure 4.79. Maximum Reductions on Daily Benzene Concentrations in Nashville

for March, June, September, and December 2003 by each Scenario
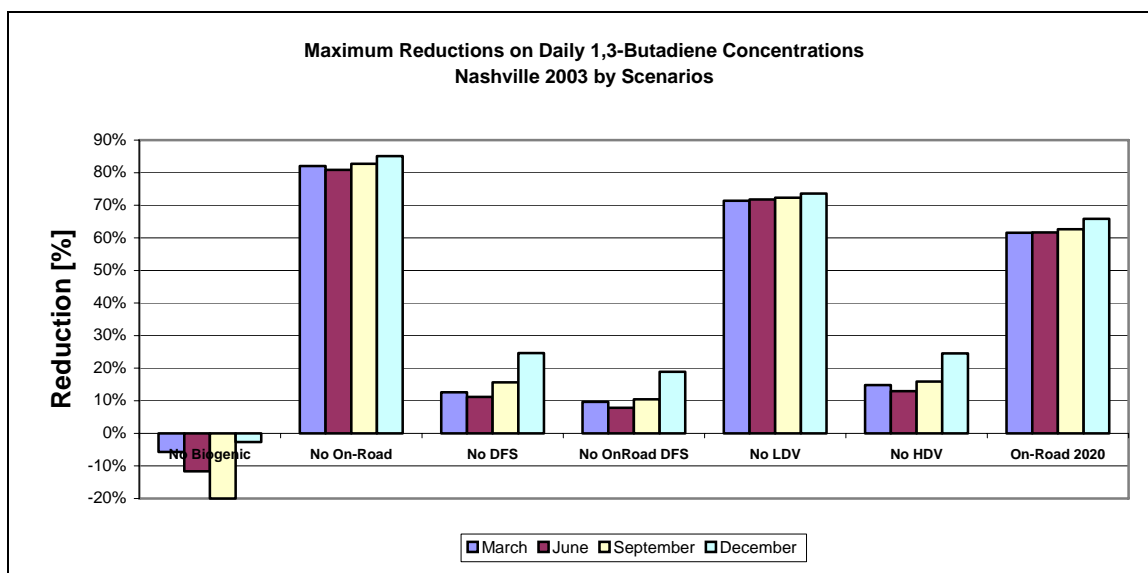
Figure 4.80. Maximum Reductions on Daily 1,3 Butadiene Concentrations in Nashville for March, June, September, and December 2003 by each Scenario
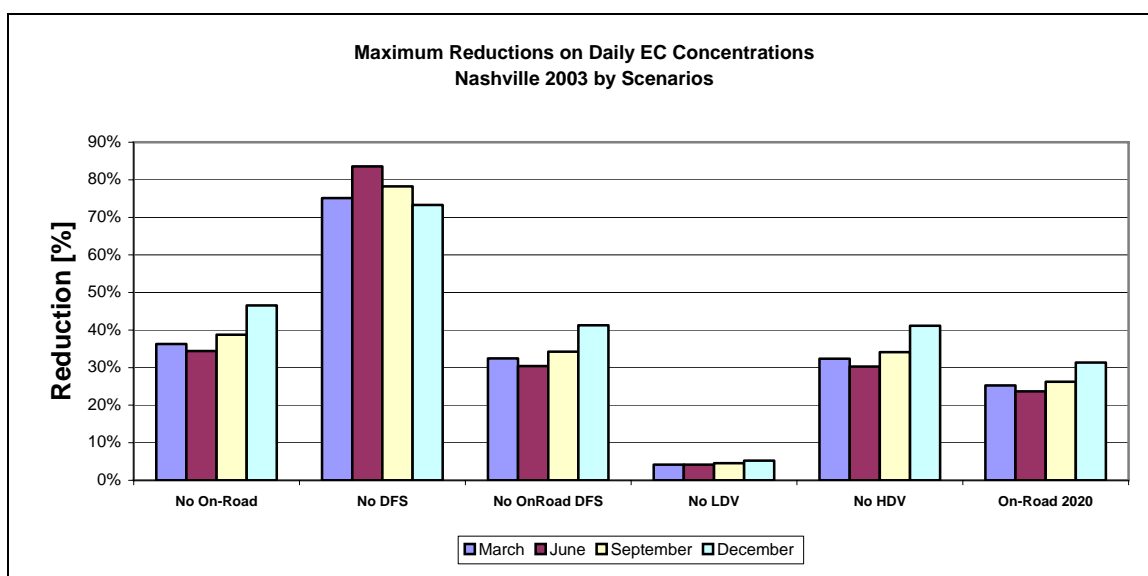
Figure 4.81. Maximum Reductions on Daily EC Concentrations in Nashville for March, June, September, and December 2003 by each Scenario
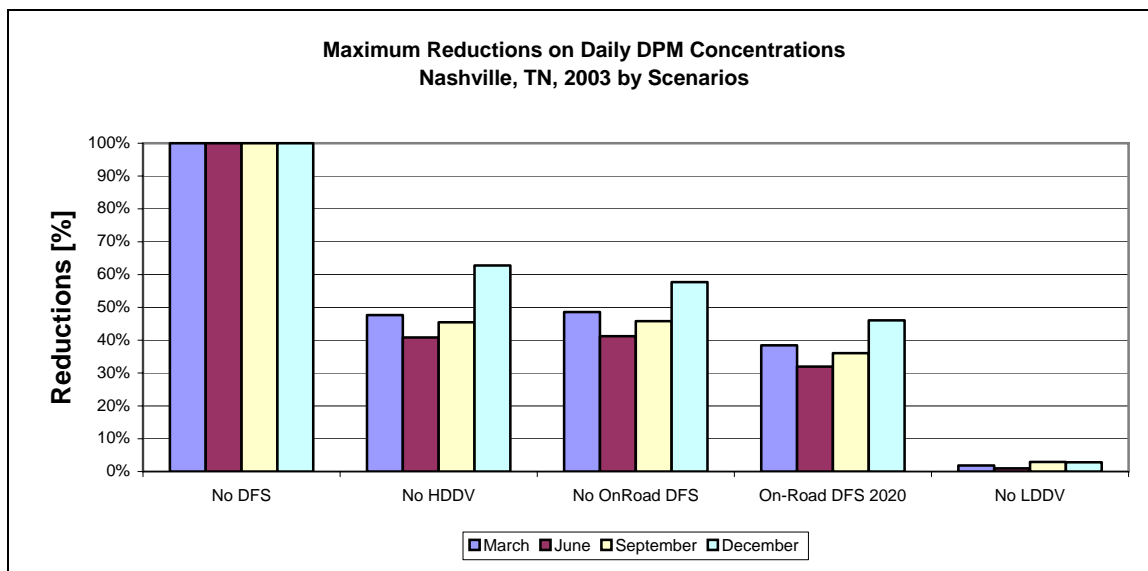
Figure 4.82. Maximum Reductions on Daily DPM Concentrations in Nashville for March, June, September, and December 2003 by each Scenario
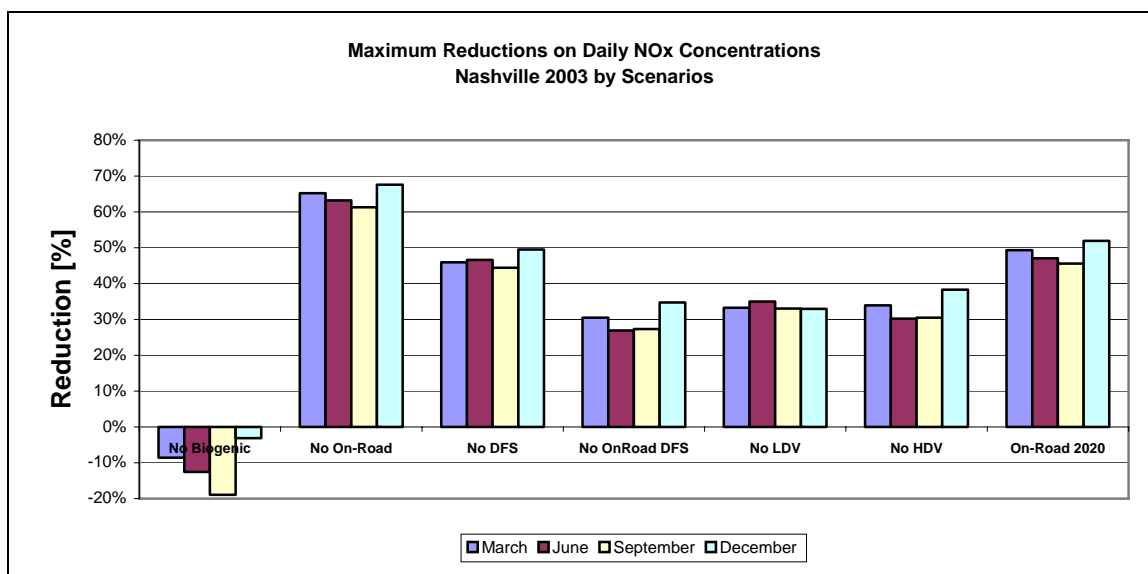
Figure 4.83. Maximum Reductions on Daily NOx Concentrations in Nashville for March, June, September, and December 2003 by each Scenario

Table 4-18. Modeled PMSATs Annual Concentrations in Nashville, 2003

Scenario	Annual Concentration [ug/m³]				
	Acetaldehyde	Benzene	Butadiene	Formaldehyde	DPM
Base Case	1.109	0.682	0.050	2.248	0.545
Non Biogenic	0.753	0.673	0.052	1.356	0.545
Non On-Road	0.670	0.192	0.010	1.895	0.316
Non DFS	1.104	0.670	0.046	2.137	-
Non OnRoad DFS	1.111	0.676	0.047	2.197	0.316
Non LDV	0.700	0.225	0.015	1.985	0.545
Non HDV	1.085	0.650	0.044	2.171	0.319
On-Road 2020	0.825	0.311	0.020	2.013	0.367

Table 4-19. Maximum Reductions on Daily Concentrations in Nashville 2003

Scenario	Acrolein	Acetaldehyde	Benzene	1,3-Butadiene	Formaldehyde	EC	DPM	NOx
No Biogenic	-28.1%	49.8%	4.6%	-10.0%	53.9%			-10.8%
No On-Road	63.9%	56.6%	75.2%	82.7%	33.7%	39.0%	48.3%	64.3%
No DFS	-27.6%	4.6%	2.8%	16.0%	12.2%	77.6%	100.0%	46.6%
No OnRoad DFS	-16.3%	1.9%	1.4%	11.7%	6.9%	34.6%	48.3%	29.9%
No LDV	39.7%	52.2%	70.2%	72.3%	24.6%	4.5%	2.2%	33.6%
No HDV	2.1%	4.8%	5.2%	17.0%	9.5%	34.5%	49.2%	33.2%
On-Road 2020	25.9%	37.1%	57.0%	62.9%	23.2%	26.6%	38.1%	48.5%

The rest of the scenarios produced a reduction lower than 5%, mainly for those scenarios without DFS and without on-road DFS, which accounted for 4.6 and 4.8% of reductions, respectively. It indicates that the gasoline LDVs were the main acetaldehyde contributors in Nashville, principally in March and December. For the scenarios without on-road sources and the year 2020, the main acetaldehyde reductions were produced in March and December due to gasoline-fueled vehicles also, showing a seasonal effect. During those cold months, the secondary acetaldehyde formation due mainly to biogenic sources was lower and the reductions were mainly on primary acetaldehyde. In fact, during June and September the reductions due to the scenario without on-road sources were only 47 and 59% respectively. Thus, for the scenario that did not consider biogenic sources, June and September produced higher reductions than March and December, with 62 and 63% respectively. Although the scenario 2020 included the on-road sources regulations only, the acetaldehyde maximum reductions were slightly significant, but not enough to achieve a strong concentration reduction compared with the hypothetic scenario without on-road sources.

It may be noted that the maximum reductions of daily formaldehyde concentrations for Nashville were due to the scenario that did not consider biogenic sources in the modeling domain, with an average reduction of 54% and a maximum of 71% produced in September, as shown in Figure 4.77 and Table 4-19. This scenario was followed by the base case scenario without on-road sources with 34%, the base case scenario without LDVs with 25%, the scenario for the year 2020 with 23%, and the base case scenario without DFS with 12%. The rest of the scenarios produced a formaldehyde reduction

lower than 10%. It may be noted a seasonal pattern on the reductions for all the scenarios, mainly between December and the rest of the months. December showed the highest formaldehyde reductions on the anthropogenic source scenarios, but generated the lowest formaldehyde reductions in the scenario without biogenic sources. This can be explained because the secondary formaldehyde is the result from photochemical reactions, especially the reaction of isoprene with the hydroxyl radical. That isoprene was generated from biogenic sources mainly during June and September. Therefore, if isoprene was not available, then less secondary formaldehyde was generated and the main reductions were due to primary formaldehyde, as shown for March, June, and September of Figure 4.77. It indicates that biogenic sources were the principal secondary formaldehyde contributors in Nashville.

The LDVs were the main formaldehyde contributors from on-road sources at this urban area, mainly in December with 47% reduction. Although the scenario 2020 included the on-road sources regulations only, the formaldehyde maximum reductions were not significant to achieve a strong air quality improvement. Nevertheless, it is expected that if a future 2020 scenario includes the non-road sources with strong fuel and technological regulations, the maximum daily formaldehyde reduction could be better at Nashville, but isoprene will continue as the main formaldehyde precursor. The maximum daily reduction of acrolein concentrations were not well produced by CMAQ as discussed on section 4.3 and shown in Figure 4.78 and Table 4-19, which is the reason why no analysis will be performed at this point.

According to Figure 4.79, most of the scenarios indicated a slightly significant seasonal variation to the maximum daily reduction of benzene concentrations in Nashville. The anthropogenic scenarios produced higher reductions in June and September, especially because during those months there were higher benzene emissions also that came from gasoline engines, as shown in Figure 4.24. The scenario that did not consider biogenic sources produced higher benzene reductions in June and September because more hydroxyl radical was available to decompose benzene. That higher hydroxyl concentration was available due to the lack of isoprene.

The highest benzene reductions occurred for the hypothetical scenario without on-road sources, which accounted on average for 75 % as shown in Figure 4.79 and Table 4-19. It was followed by the scenario that did not consider LDVs with 70 %, and the scenario for the year 2020 with 57 %. The rest of the scenarios showed benzene reductions lower than 5 %, mainly for the scenario without on-road DFS, which generated a reduction as low as 1.4 %. The benzene sources were mainly the gasoline LDVs sources. As a result, the on-road mobile sources regulations will be strong enough to reduce significantly the benzene concentrations in 2020 at Nashville.

The maximum daily reductions of 1,3 butadiene concentrations showed a behavior similar to benzene at Nashville, with the exception of the scenario that did not consider biogenic sources. The scenarios that did not include DFS, on-road DFS, and HDVs showed strong seasonal pattern reductions on 1,3-butadiene concentrations, whose higher reductions occurred during December. This higher reduction was because the

photochemical decomposition of primary 1,3-butadiene was lower in December and each scenario performed greater effect on the reductions, as shown in Figure 4.80 and Table 4-19. The highest reductions occurred for the hypothetic scenario without on-road sources, which accounted on average for 83 %, followed by the scenario without LDVs with 72 %, and the scenario for the year 2020 with 63 %. The rest of the scenarios showed reductions lower than 17 %, mainly for the scenario without on-road DFS, which generated a reduction of 12 %. Like benzene, the greatest 1,3-butadiene sources were the gasoline LDVs sources. Therefore, the on-road mobile sources regulations will be strong enough to reduce significantly the 1,3-butadiene emissions in 2020 by almost 63%.

During June, the maximum daily EC concentration reduction due to the scenario without DFS sources was as high as 84% and during December was of 73%, as shown in Figure 4.81 and Table 4-19. That maximum reduction occurred in June, because in the summer season the construction activity becomes more active in Tennessee, as well as, the impact of wood smoke becomes less important. These DFS contributions were close to the values obtained for Zheng et al., (2002) discussed in section 4.3. The rest of the scenarios showed a seasonal pattern concentration reductions, whose maximum reduction was produced in December and the minimum in June, as shown in Figure 4.82 and Table 4-19. That maximum occurred in December because more on-road sources emissions were generated during this month as shown in Figure 4.21. The maximum EC reductions due to the scenario without non-road sources were 43, 53, 44, and 32% during March, June, September, and December, respectively, which were obtained by subtracting on-road

DFS from DFS scenario. This variability explains the seasonal construction effect on EC concentrations.

On average, the maximum reductions were performed by the scenario that did not consider DFS, followed by the scenario without on-road sources, the scenario without on-road DFS, the scenario without HDVs, and the scenario to the year 2020, with 78, 39, 35, 35, and 27% respectively. The EC maximum reductions of 27% were not significant enough to achieve a strong air quality improvement at Nashville for the year 2020 compared with the hypothetic scenario without DFS. However, if a future 2020 scenario includes the non-road sources with all fuel and technological regulations, the maximum daily EC reductions could be better. As result, better DPM reduction strategies must be considered on mobile sources.

Finally, according to the Figure 4.83, the maximum daily NOx concentrations reductions showed a slight seasonality, except for the scenario without LDVs. The maximum daily NOx reductions occurred in December, followed by March, June, and September on the most of the scenarios, except the scenarios that did not consider biogenic and LDVs sources. Those maximum daily NOx reductions were produced in December, since in cold months less photochemical reactions decompose NOx and therefore more NOx is reduced if those hypothetical scenarios would occur. The scenario that did not included biogenic sources produced higher NOx concentrations than the base case scenario, since if there was not isoprene in the atmosphere less VOCs, like formaldehyde and acetaldehyde, was generated by its decomposition with ozone, hydroxyl radical, $NO_3^\bullet$,

and oxygen. Therefore, if less VOCs were generated, less NOx reacted photochemically to generate ozone. In fact, more NOx did not react during June and September, months that generate less isoprene as shown in Figure 4.83 and Table 4-19. The maximum average daily NOx reductions were produced for the scenario without on-road sources with 64 %, followed by the scenario for the year 2020 with 49 %, the scenario without DFS with 47 %, the scenario without LDVs with 37 %, the scenario that did not consider HDVs with 33 %, and the scenario without on-road DFS with an average reduction of 30% as shown in Figure 4.83 and Table 4-19. Although the scenario 2020 included the on-road sources regulations only, this scenario produced a strong NOx reduction of 49%, which was so different than the reductions produced to DPM.

4.5   INHALATION HEALTH RISK

The lifetime excess inhalation cancer risk was estimated for acetaldehyde, benzene, 1,3-butadiene, formaldehyde, and DPM assuming an additive effect risk assessment. The cancer risk for those four vapor air toxics was called 4HAPs, which was the additive result of the excess inhalation cancer risk for each of those air toxics. The risk estimates in this study were based on annual average exposures for a wide population distribution. These estimates assumed continuous exposure over an entire lifetime of 70 years to levels estimated for 2003 and did not account for expected changes in exposure over time. However, exposures to most air toxics are expected to change over time as a result of mobile and stationary source emission control programs, as analyzed in the scenario for the year 2020.

Table 4-20 and Figures 4.84 and 4.85 show the estimated inhalation cancer risk and the reductions performed for each analyzed scenario at Nashville, TN, base on the annual concentrations of Table 4-18 and the IURs of Table 2.8. The main reductions on those 4HAPs were due to the contribution of biogenic sources with 32.2%, which generated high secondary acetaldehyde and formaldehyde in the summer season.

This condition was followed for the scenario that did not consider on-road sources with a 27.5% reduction, where the main reductions were due to the air toxics contributions generated by gasoline LDVs.

Table 4-20. Inhalation Cancer Risk by Scenarios for those 4HAPs and DPM at Nashville, TN.

Scenario	4HAPS x 10^{-6}	Reduction [%]	DPM x 10^{-6}	Reduction [%]	4HAPS+DPM x 10^{-6}	Reduction [%]
Base Case	38.5		157.8		196.3	
No Biogenic	26.1	32.2%	157.8	0.0%	183.9	6.3%
No On-road	27.9	27.5%	91.4	42.1%	119.3	39.2%
No DFS	36.8	4.4%	0.0	100.0%	36.8	81.2%
No On-road DFS	37.7	2.1%	91.4	42.1%	129.1	34.2%
No LDV	29.5	23.2%	156.8	0.6%	186.3	5.1%
No HDV	37.0	3.9%	93.2	40.9%	130.2	33.7%
On-road 2020	31.0	19.4%	106.2	32.7%	137.2	30.1%
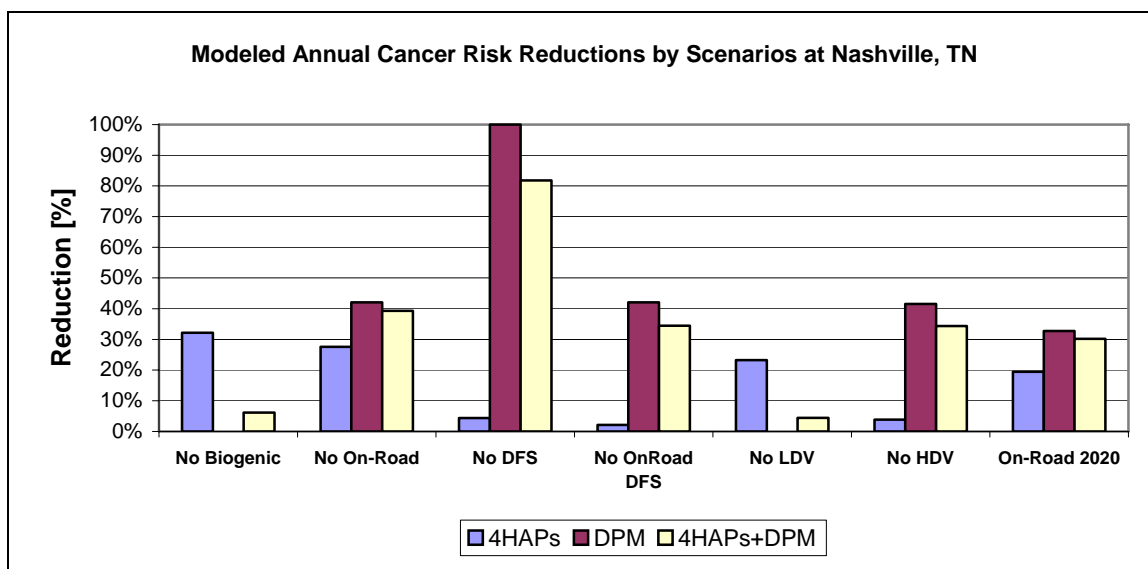
Figure 4.84. Modeled Cancer Risk Reductions by Scenarios at Nashville, TN
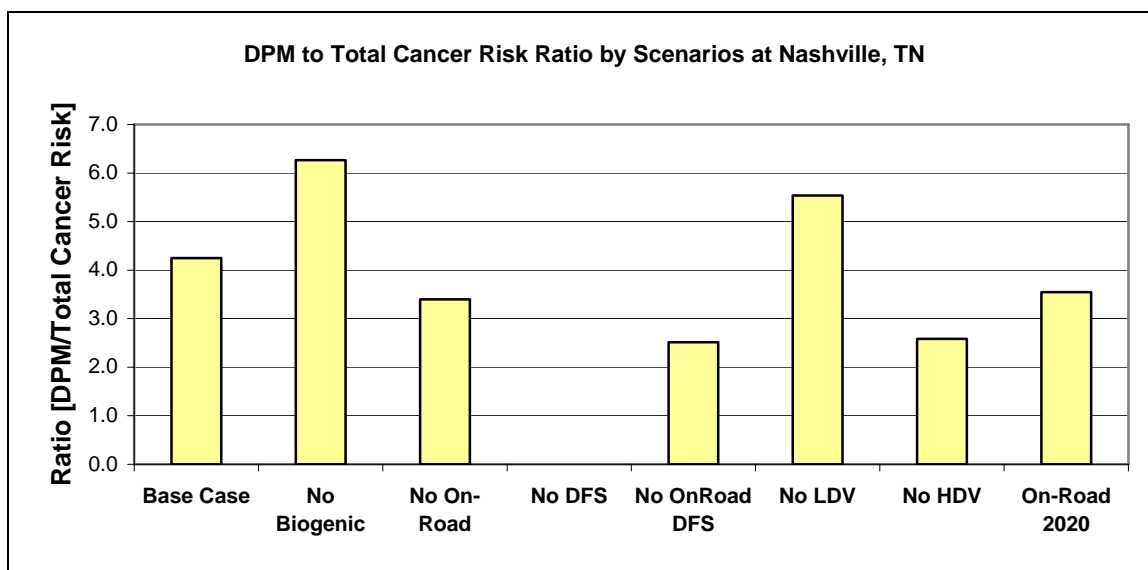
Figure 4.85. DPM to Total Cancer Risk Ratio by Scenarios at Nashville, TN

As the scenario 2020 included the on-road sources regulations only, the 4HAPs cancer risk showed a reduction as low as 19.4 %, which were not significant to achieve a strong air quality improvement at Nashville for the year 2020. If there are no on-road sources or DFS in the modeling, the cancer risk reduction is not expected to be reduced significantly also for those vapor air toxics, since major sources are important sources of acetaldehyde emission, as well as, area and non-road sources are important sources of benzene, 1,3-butadiene, and formaldehyde, as shown in Figures 4.3, 4.4, and 4.5. For that reason, better air toxics reduction strategies must be considered on the other emission sources as well, mainly on non-road and open burning sources. The rest of the scenarios showed reductions lower than 4.4 %, indicating that DFS and HDVs were not important vapor air toxics contributors at Nashville, TN.

It may be noted that DPM posed a cancer risk that was 4.1 times higher than the combined total cancer risk from all other air toxics simulated on the base case scenario. Those high cancer risk levels were due mainly to the DMP emitted from goods transportation and construction engines. This higher DPM cancer risk was also estimated by Conrad et al., (2005), who reported that DPM posed a cancer risk that was 7.5 times higher than the combined total cancer risk from all those other 33 UATs. The main reductions in DPM cancer risk were due, obviously to the contribution of DFS sources with 100.0 % reduction followed by the scenario that did not consider on-road DFS sources with 42.1% reduction, where the main reductions were due to the DPM contributions generated by HDDVs. It may be important indicate that the reduction due to a hypothetical scenario without non-road sources would be as high as 57.9%, which is the

difference between the scenario without DFS and the scenario without on-road DFS. In other words, non-road sources produced the highest contribution on ambient DPM concentrations and its associated cancer risk.

As the scenario 2020 included the on-road sources regulations only, the DPM cancer risk showed a 32.7 % reduction. If a future 2020 scenario includes the non-road sources with all fuel and technological regulations, like on-road sources, the DPM cancer risk reduction could be important but not strong enough compared with a hypothetical scenario that did not consider DFS sources. Therefore, better DPM reduction strategies must be considered on mobile sources to reduce its cancer risk in Nashville TN. The cancer risk reductions scenarios associated to 4HAPs plus DPM followed similar trends than the reductions that came from DPM. Indicating that DPM generated the higher lifetime cancer risk excess among the other air toxics, as shown in Table 4-20, and Figures 4.84 and 4.85 for Nashville, TN.

The following series of tile plots, Figures 4.86, 4.87, and 4.88, show the estimated lifetime inhalation cancer risk excess from the 4HAPs, DPM, and 4HAPs+DPM, respectively, for each analyzed scenario on the whole modeling domain. It must be noted that the tile plots were generated assuming an annual concentration base on those 4 analyzed months. In general, the plots show that no area fulfilled the EPA's cancer risk rule, since the 4HAPs, DPM, and 4HAPs+ DPM exceeded the four, one, and 5 in a million risk of cancer over a lifetime of exposure respectively. One-in-a-million is thus considered an acceptable risk of cancer for a single pollutant by the U.S. EPA.

(a)

(b)

(c)

(d)
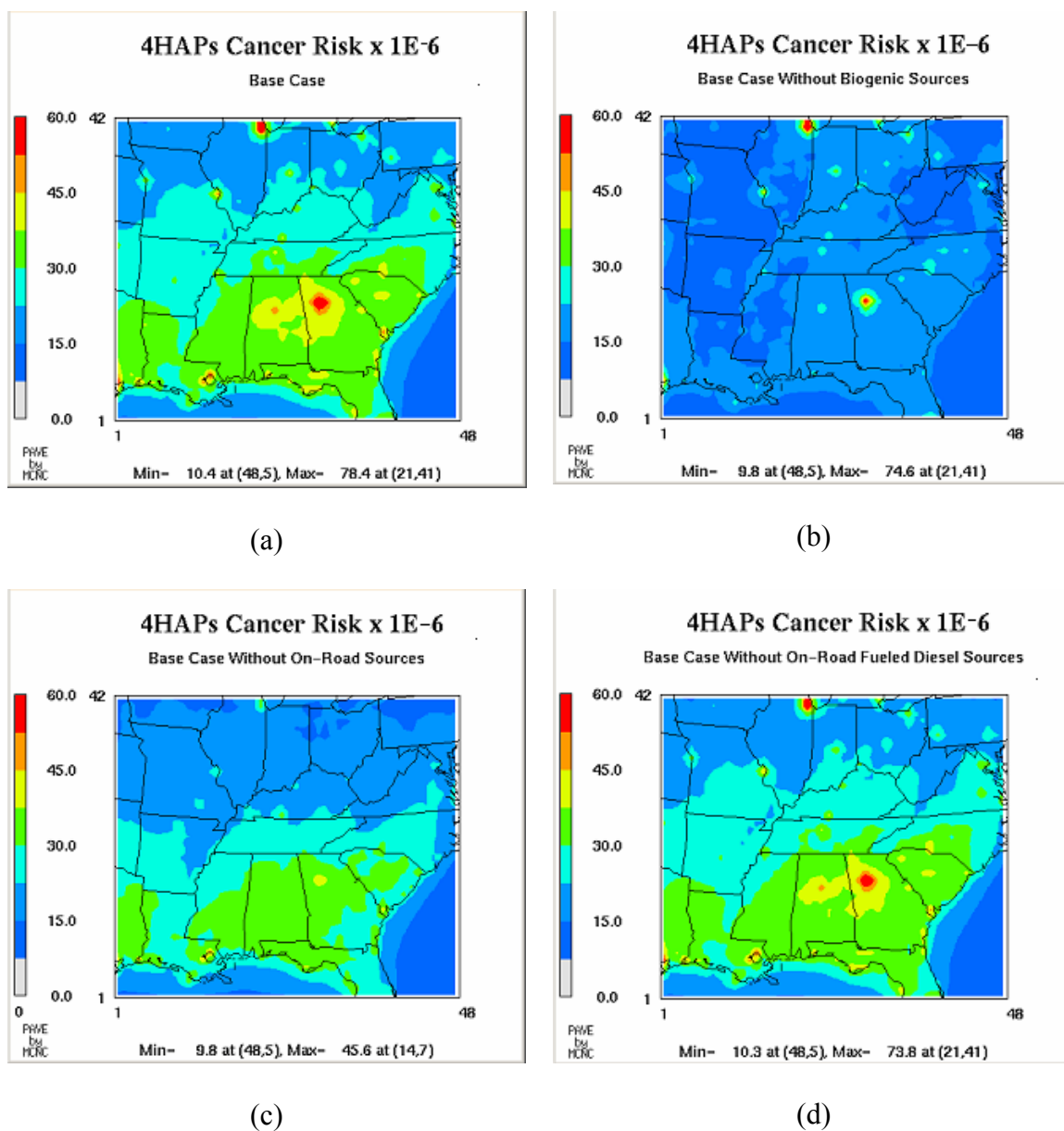
Figure 4.86. Spatial Variation of the 4HAPs Cancer Risk by Scenarios
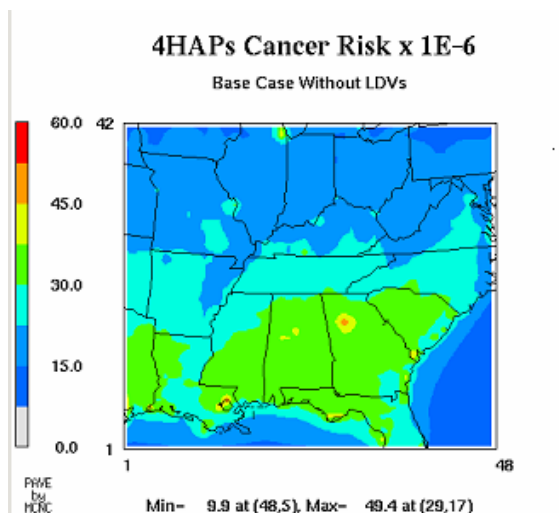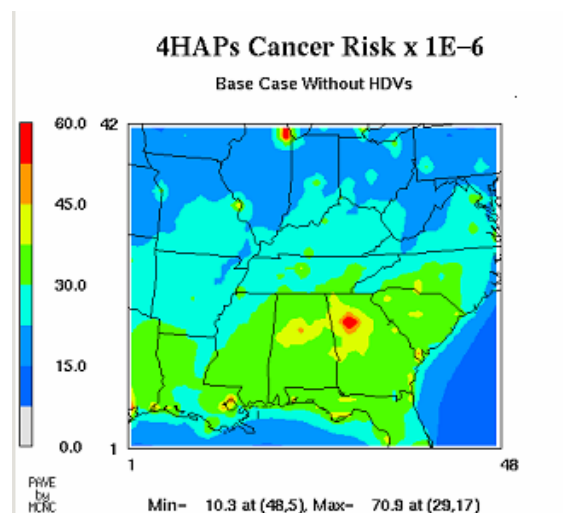
(e)   (f)
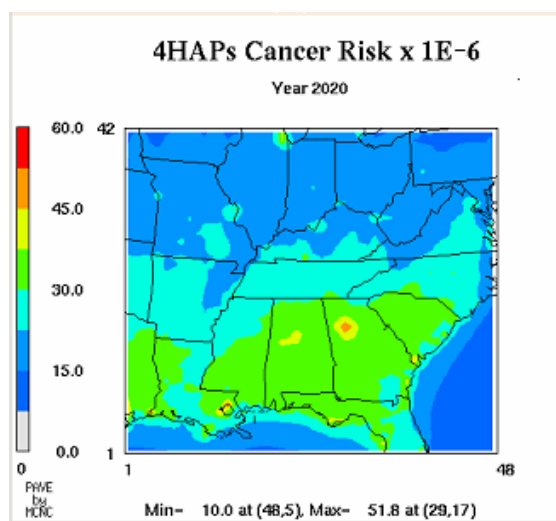
(g)

Figure 4.86. Continued
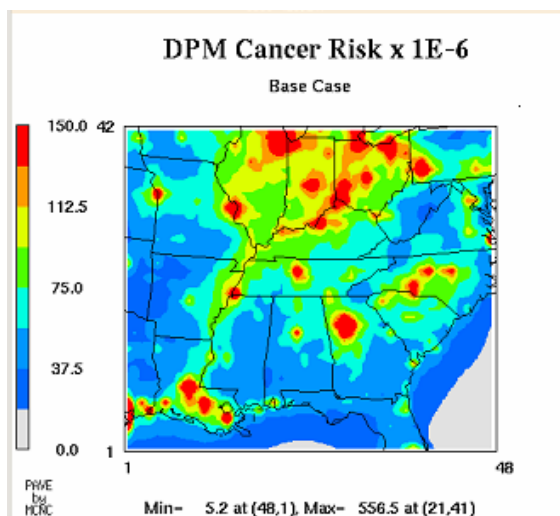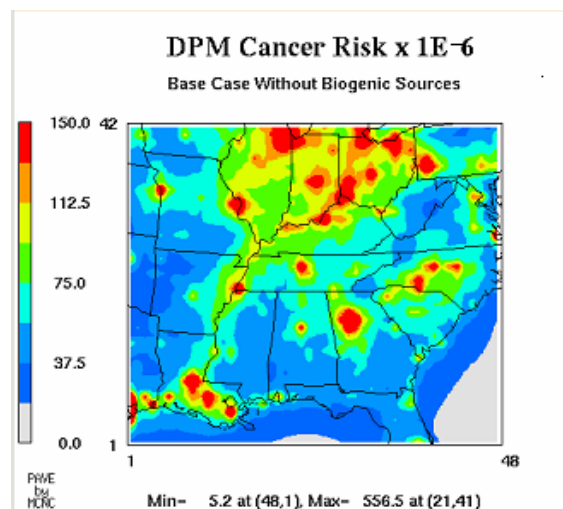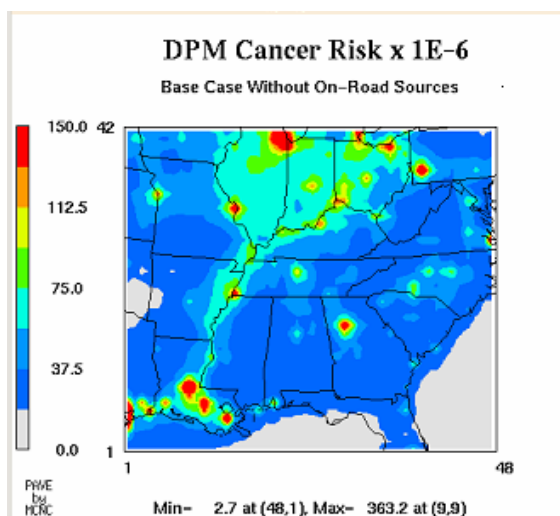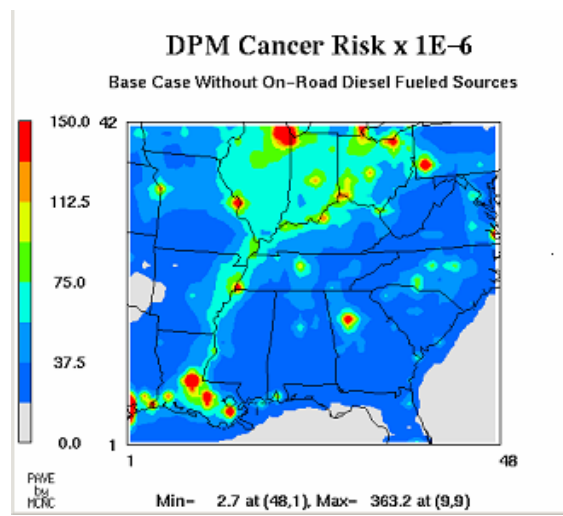
Figure 4.87. Spatial Variation of the DPM Cancer Risk by Scenarios
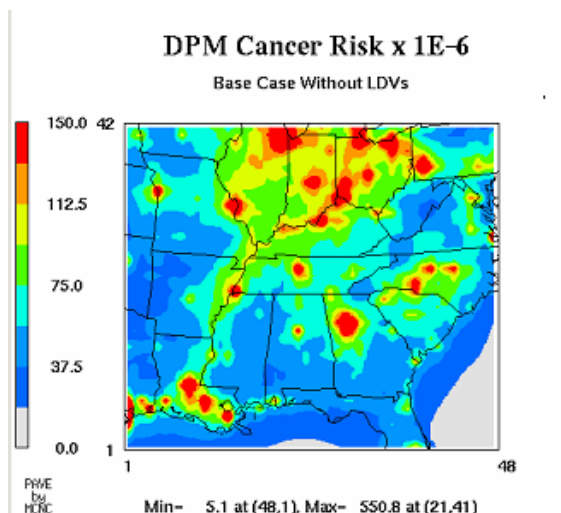
(e)
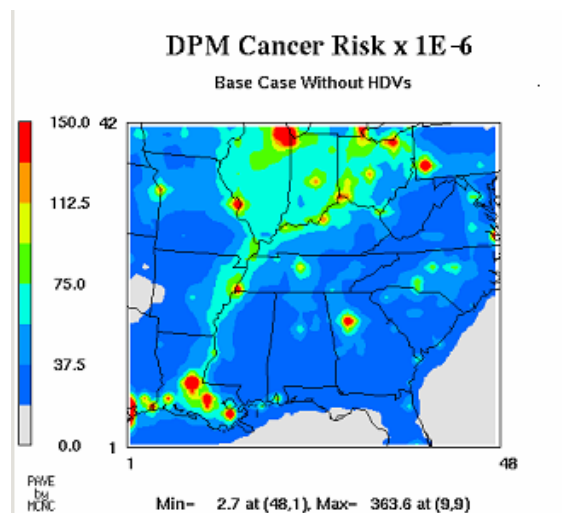
(f)
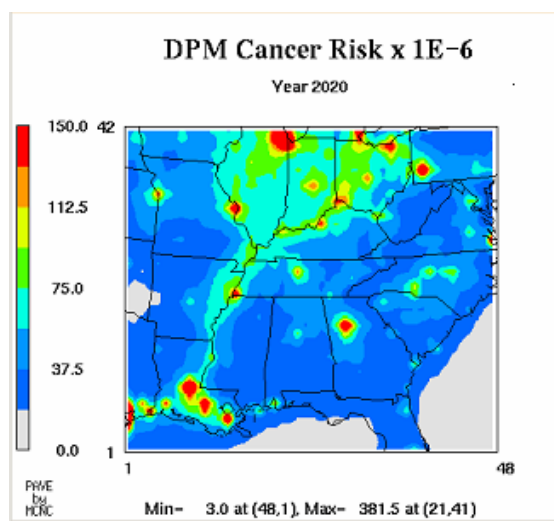
(g)

Figure 4.87. Continued

(a)

(b)

(c)

(d)
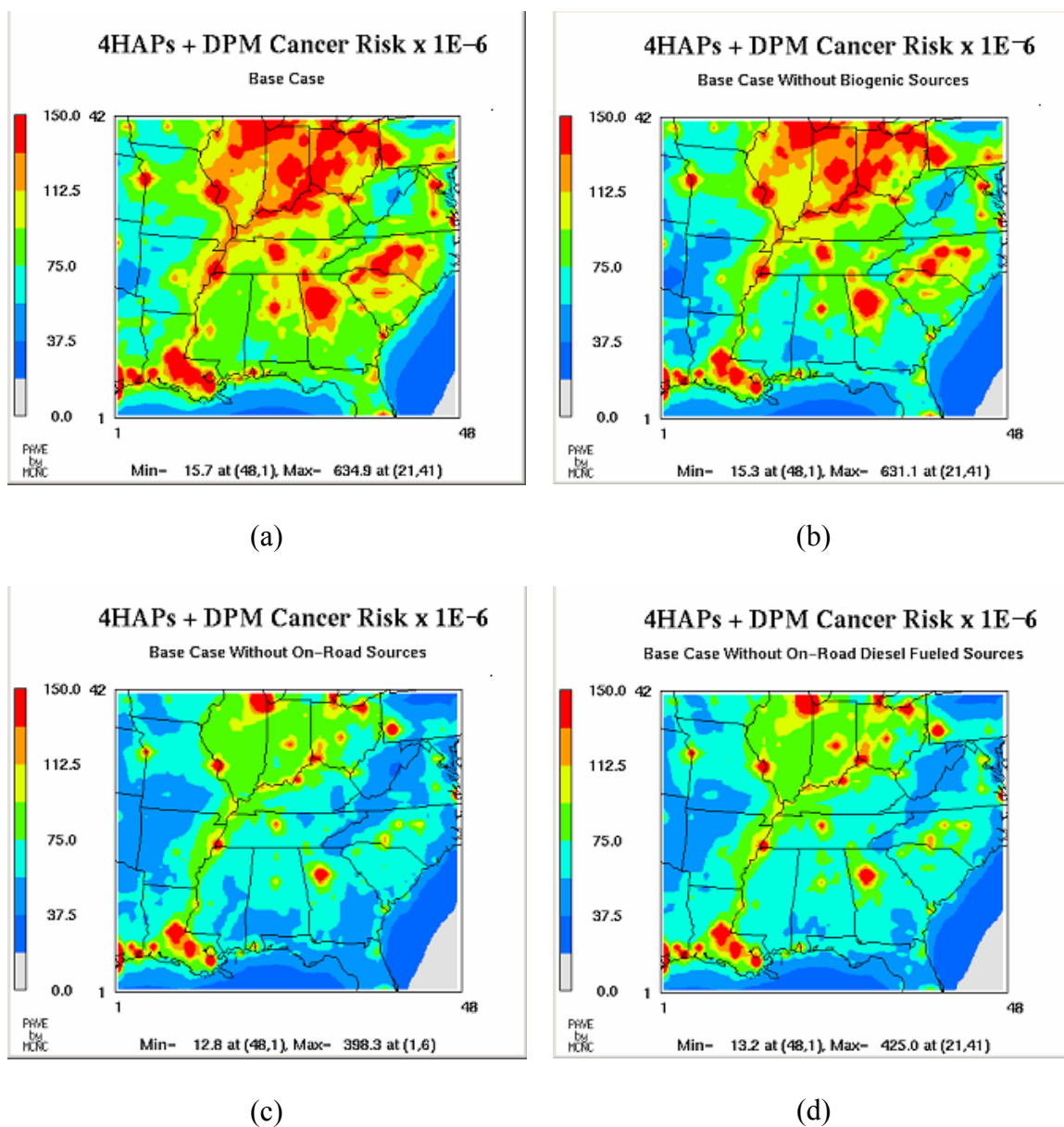
Figure 4.88. Spatial Variation of the DPM + 4HAPs Cancer Risk by Scenarios

4HAPs + DPM Cancer Risk x 1E−6
Base Case Without LDVs

Min− 15 at (48,1), Max− 592 at (21,41)

(e)

4HAPs + DPM Cancer Risk x 1E−6
Base Case Without HDVs

Min− 13.2 at (48,1), Max− 426.8 at (21,41)

(f)

4HAPs + DPM Cancer Risk x 1E−6
Year 2020

Min− 13.2 at (48,1), Max− 422.0 at (1,6)
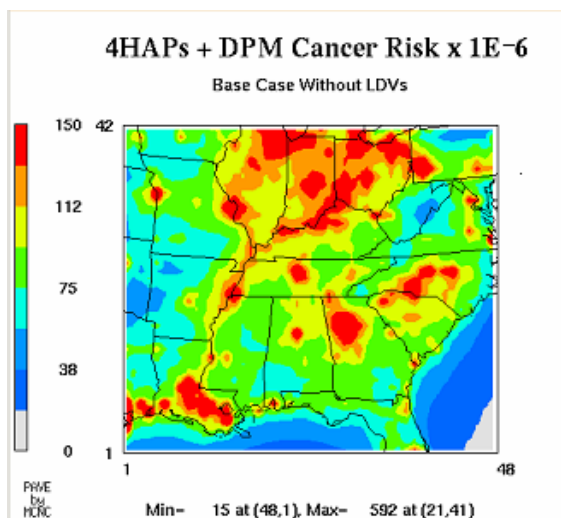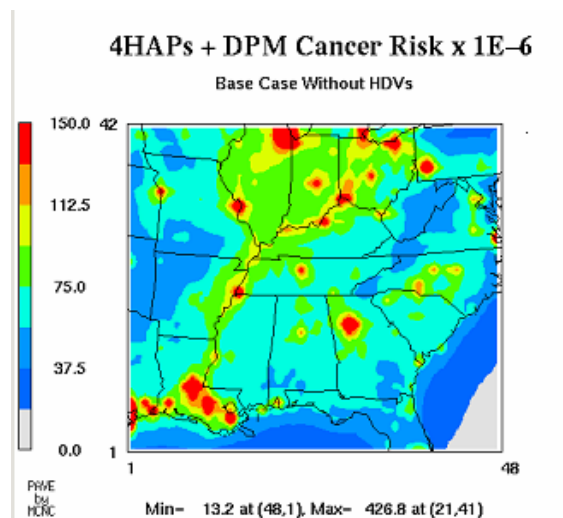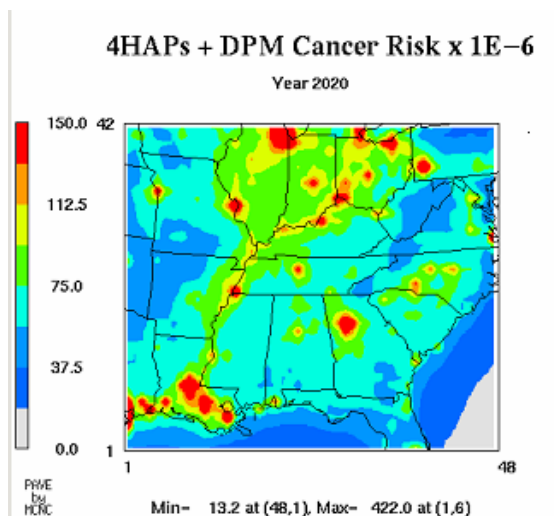
(g)

Figure 4.88. Continued

Higher cancer risk occurred on Southeastern urban areas for those 4HAPs and DPM, principally at Atlanta, GA, for the 4HAPS as shown in Figure 4.86 (a). For DPM, the higher cancer risk occurred in Atlanta, GA, followed by Nashville, TN, Birmingham, AL, Raleigh, NC, and Memphis, TN, as shown in Figure 4.87 (a).

The 4HAPs cancer risk was influenced principally for secondary acetaldehyde and formaldehyde generated in summer season by the biogenic sources effect, as shown in Figure 4.86 (b) and 4.88 (b).

The scenario for the year 2020 showed higher 4HAPs cancer risk reductions in Atlanta and Birmingham. On the other hand, the scenario that did not consider on-road DFS showed the effect of non-road DFS, as shown in Figure 4.87 (d) and 4.88 (d). In fact, the area around the Mississippi river show the impact of diesel marine engines on DPM cancer risk, which produced a cancer risk between 37.5 and 75 per million population. Finally, it was evident that the population was exposed to greater than 5 in a million level of cancer risk in the whole domain, especially due to DPM in urban areas.

The lifetime excess inhalation non-cancer hazard ratio was estimated for acetaldehyde, benzene, 1,3-butadiene, formaldehyde, and DPM. The risk estimates in this study also were based on annual average exposures for a wide population distribution. These estimates assumed continuous exposure over an entire lifetime of 70 years to levels estimated for 2003 and did not account for expected changes in exposure over time like the cancer risk estimations.

Table 4-21 shows the CMAQ modeled hazard ratio for the base case and the hazard ratio estimated from the monitored at Nashville, TN. As all hazard ratios were less than one, the total hazard index was mot estimated and the emission scenarios analysis was not considered also. In addition, it may be noted that the hazard ratio was underestimated for all those HAPs, principally for 1,3-butadiene by almost 78%. Formaldehyde was the unique PMATs that showed a hazard ratio close to 1, which was allocated in the Atlanta metropolitan area (Hazard ratio=0.975), as shown in Figure 4.89.

In opposite to the estimation of non-cancer risk base on toxicological evidence equations as shown above, the results from epidemiological functions showed significant non-cancer risk. In fact, the relative risks (RR) of CVD mortality, lung cancer mortality, and asthma hospital admissions, and the odds ratio (OR) of COPD illness were estimated base on the C-R functions to DPM described in section 2.1.5 (Pope et al., 2004a and 2002; Abbey et al., 1995; and Shepperd et al., 1999 and 2003). The RR and OR estimates in this study were based on annual average exposures for a wide population distribution.

Table 4-21. Modeled and from Monitored Data Hazard Ratio in Nashville

Compound	Hazard Ratio		Error [%]
	Modeled	From Monitored Data	
Acetaldehyde	0.1232	0.1802	31.6
Benzene	0.0227	0.0457	50.3
1,3-Butadiene	0.0252	0.1131	77.7
Formaldehyde	0.5619	0.8970	37.4
DPM	0.1090		
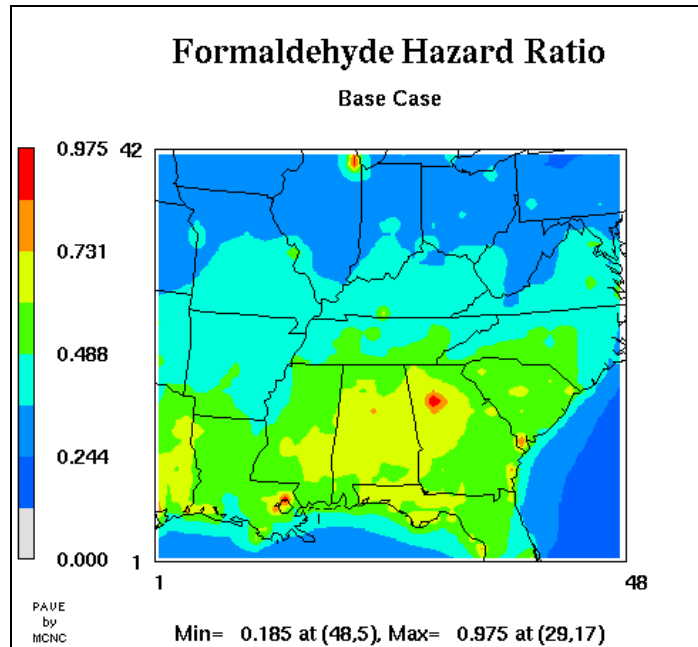
Figure 4.89. Spatial Variation of the Formaldehyde Hazard Ratio

These estimates assumed continuous long-term exposure to levels estimated for 2003 and did not account for expected changes in exposure over time. However, the exposure to DPM is expected to change over time as a result of mobile and stationary source emission control programs.

Figure 4.90 shows the estimated relative CVD mortality, lung cancer mortality, and asthma hospital admissions risk, on the other hand, Figure 4.91 shows the odds ratio of the chronic illness for COPD, and Figure 4.92 shows the risk reductions performed for each analyzed DPM scenario in Nashville, TN, for CDV and lung cancer mortality, asthma hospital admissions, and chronic illness for COPD. Although the RR and OR may seem small for the Base Case scenario, when applied to relatively large populations, the
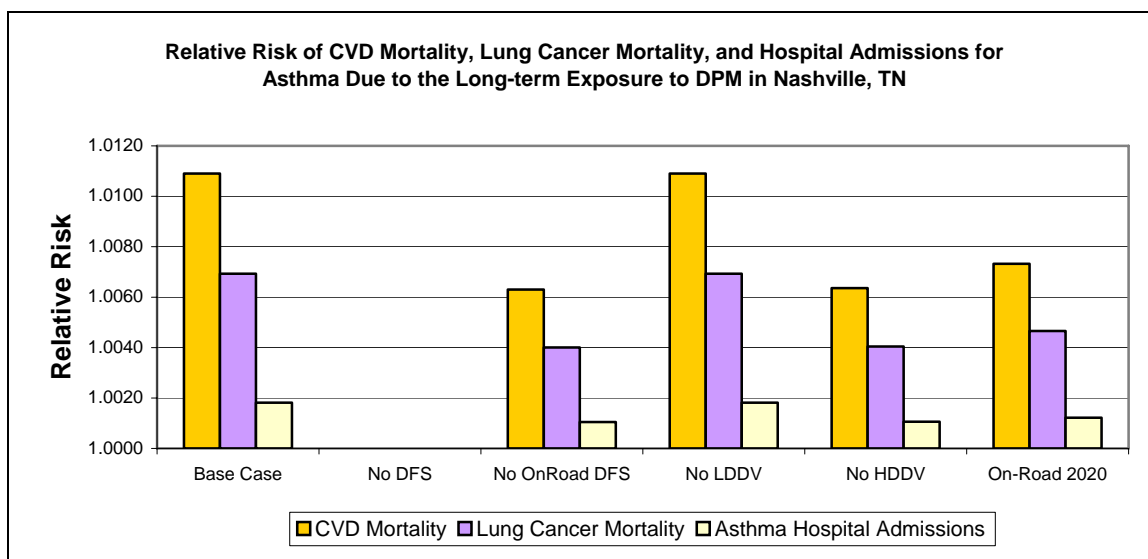
Figure 4.90. Relative Risk of CVD Mortality, Lung Cancer Mortality, and Hospital Admissions for Asthma Due to the Long-term Exposure to DPM in Nashville, TN
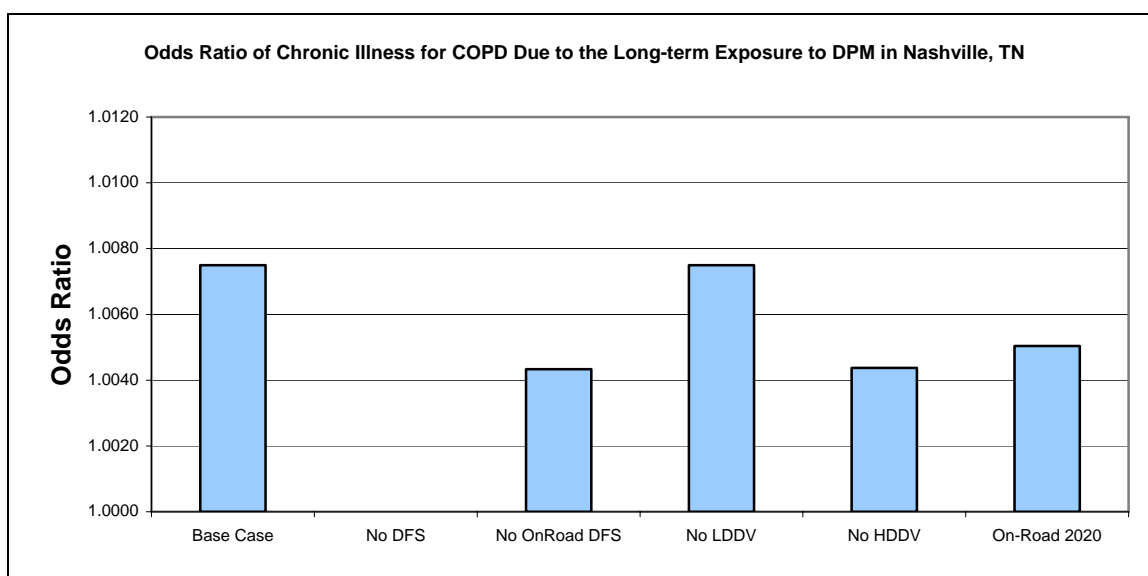
Figure 4.91. Odds Ratio of Chronic Illness for COPD Due to the Long-term Exposure to DPM in Nashville, TN
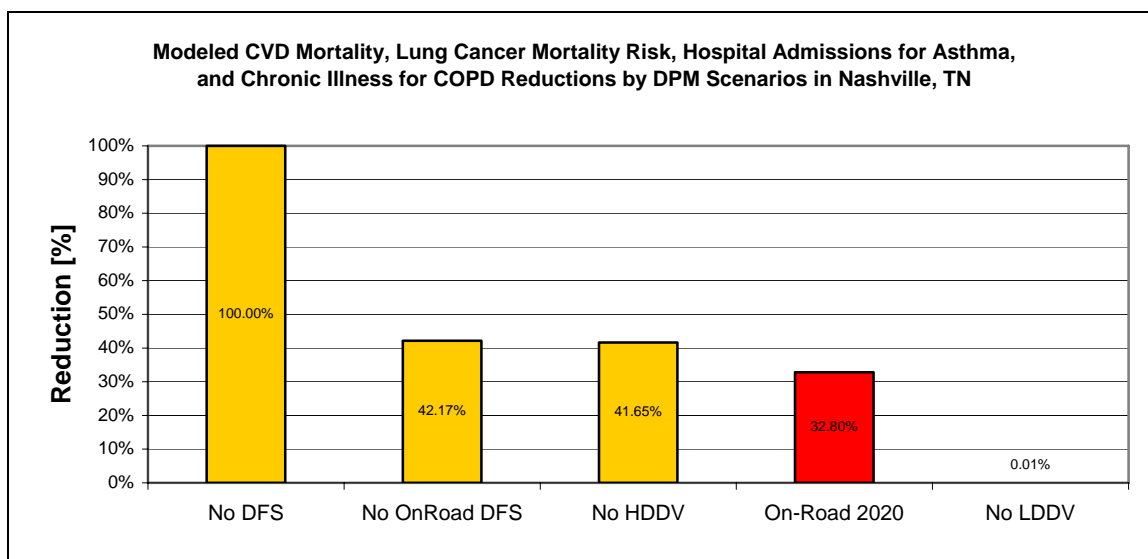
Figure 4.92. Modeled CVD Mortality, Lung Cancer Mortality Risk, Hospital Admissions for Asthma, and Chronic Illness for COPD Reductions by DPM Scenarios in Nashville, TN

health impacts can still be large. In this case, the modeled RR of CVD mortality, lung cancer mortality, and asthma hospital admissions for Nashville were 1.0109, 1.0069, and 1.0018, respectively.

In other words, 1 $\mu g/m^3$ increases in annual DPM exposure was associated with a 1.09% increase in the relative risk of CVD mortality, a 0.69% increase in the relative risk of lung cancer mortality, and 0.18% increase in the relative risk of hospital admissions for asthma in Nashville. Whereas, the modeled OR of chronic illness for COPD for Nashville was 1.0075, i.e., the odds of getting a chronic illness for COPD increase by 0.75% with each additional annual ug/m^3 of DPM.

The main reductions on DPM relative CVD mortality, lung cancer mortality, hospital admissions for asthma risk, and chronic illness for COPD odds ratio were due to the scenario that did not consider non-road DFS sources with 57.8% reduction, followed by the scenario without on-road DFS with 42.2%, where the main reductions were due to the DPM contributions generated by HDDVs. Considering that the scenario 2020 included the on-road sources regulations only, the DPM showed a 32.8 % risk reduction.

The following series of tile plots show the spatial variation of the estimated relative CVD mortality, lung cancer mortality, and asthma hospital admissions risks, and odds ratio for COPD illness due to long-term exposure to DPM for the Base Case and the scenario for the year 2020 over the whole modeling domain. Higher relative CVD mortality, lung cancer mortality, and asthma hospital admissions risks, and odds ratio for COPD illness occurred on Southeastern urban areas due to DPM, principally at Atlanta, GA, followed by Nashville, TN, Birmingham, AL, Raleigh, NC, and Memphis, TN, as shown in Figure 4.93 and 4.94 (a and c).

The scenario for the year 2020 showed some reductions, mainly on the surrounding urban areas of Atlanta, Nashville, Memphis, Birmingham, and Raleigh as shown in Figure 4.93 and 4.94 (b and d). The area around the Mississippi River shows the impact of DFS to the relative CVD mortality, lung cancer mortality, and asthma hospital admissions risks, and odds ratio for COPD illness in the Figures 4.93 and 4.94 (a and b), respectively. For Knoxville, the health risks were almost negligible for 2020.

(a)

(b)

(c)

(d)
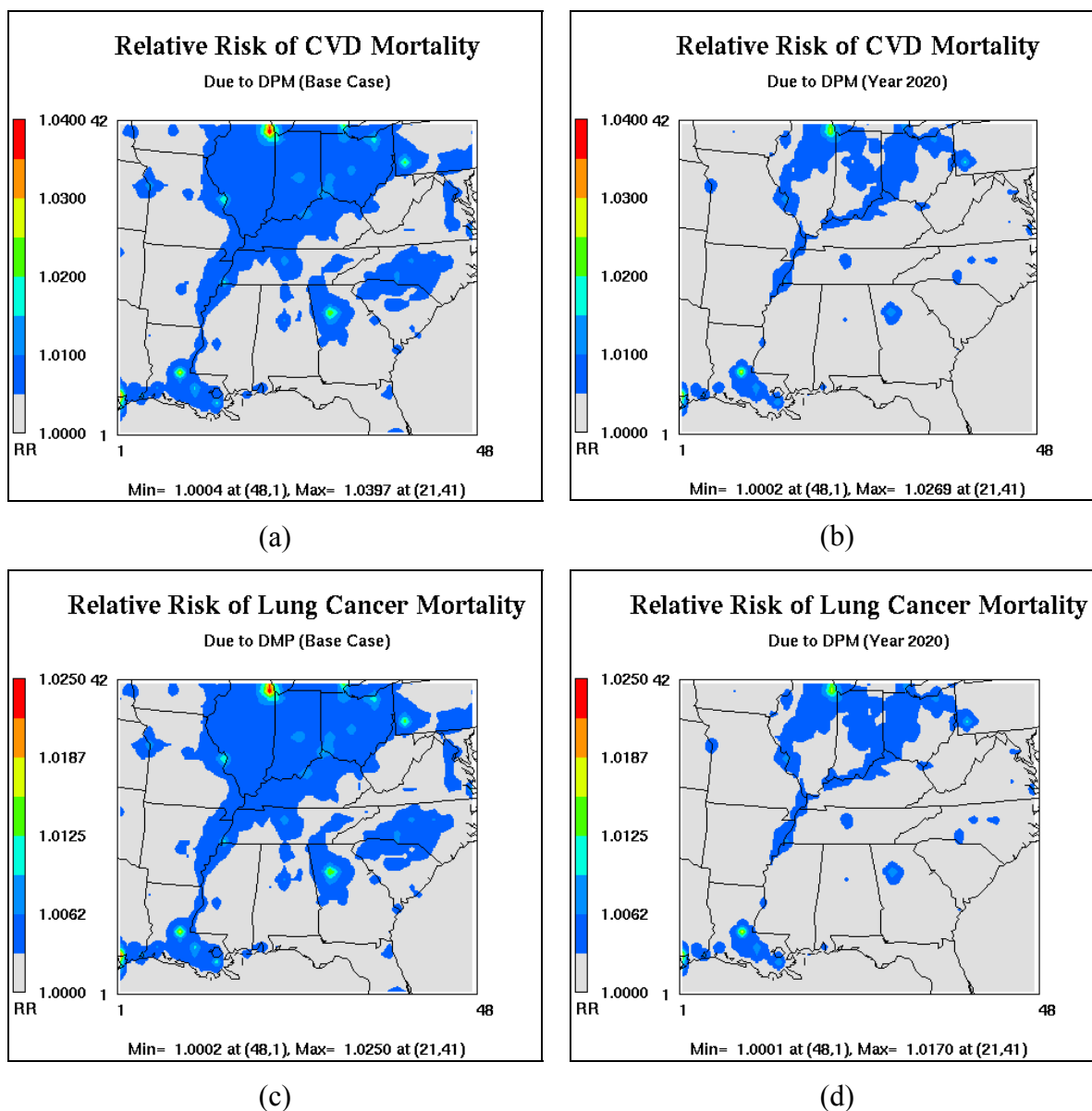
Figure 4.93. Spatial Variation of the Relative CVD and Lung Cancer Mortality Risk due to Long-term Exposure to DPM (base case and the Scenario 2020)
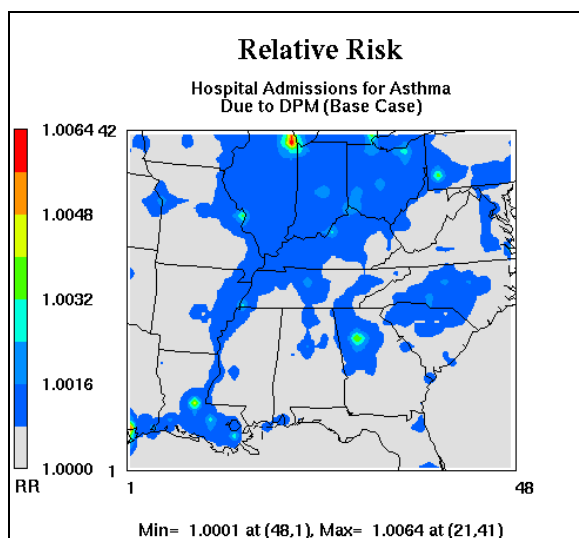
(a)
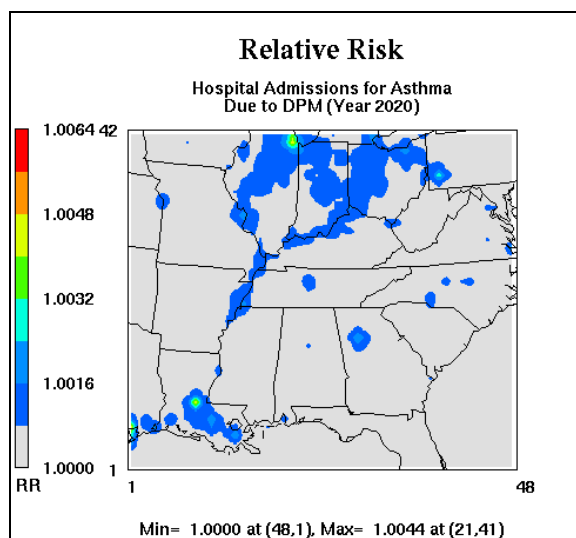
(b)
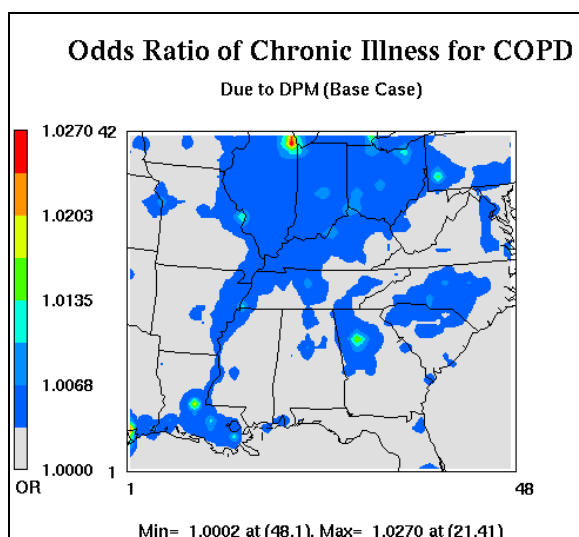
(c)
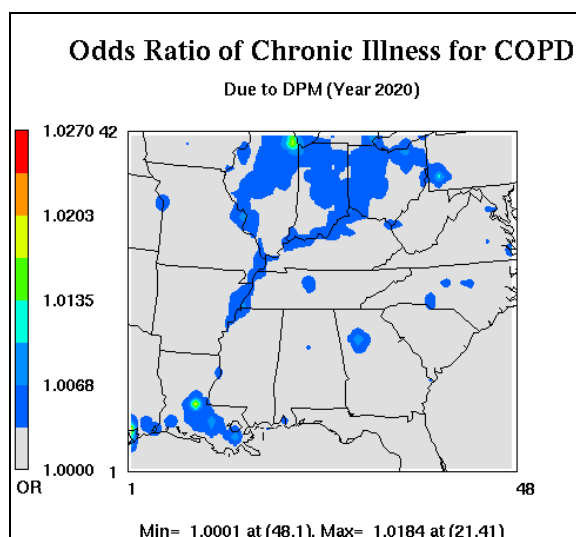
(d)

Figure 4.94. Spatial Variation of the Relative Hospital Admission for Asthma risk and Odds Ratio of Chronic Illness for COPD due to long-term Exposure to DPM (base case and the Scenario 2020)

4.6   SOURCES OF UNCERTAINTY

The sources of uncertainty in this research can be due to six components: emissions inventory, emission speciation, spatial and temporal allocation of emissions, model parameters, meteorology, and risk assessment.

The air toxics and criteria pollutants inventories are estimated base on emission factors and activities data by the U.S. EPA, using emission models, such as NMIM, NONROAD, or getting information directly from industries. Those activity data, models, and emission factors inherently are affected by uncertainty. It may be noted that the air toxics area sources inventory for 1999 was over estimated for Florida and underestimated for Tennessee, comparing with neighbor states. In fact, open burning emission does not have to depend of the states border, instead, Figures 4.11, 4.13, and 4.14 showed the wild fires in Florida did not affect its neighbor states. In addition, in this research was used different years for meteorological data and emission inventories, 2003 and 1999, respectively, which generated some uncertainties.

Air toxic emissions from all sources are typically estimated by apportioning total organic compounds and particulate matter emissions, base on specific profiles. Those emissions are processed using SMOKE 2.0. The model includes an emissions inventory element to estimate mass emissions rates, speciation, spatial allocation, and default temporal profiles. The speciation mechanism used in this research was the CB-IV, which as some

limitations. On the other hand, the default temporal profiles do not account properly for some specific sources, such as HDDVs.

Weather conditions and meteorological data sets used to model air toxics exhibit variability over time. The application of meteorological data sets from a single location to a modeling domain is also uncertain.

Uncertainties in air quality modeling are relatively smaller than emissions uncertainties. Studies of the accuracy of air quality models show that these models are more reliable for estimating longer time averaged concentrations than for estimating short term concentrations at specific locations (Asante-Duah, 2002); and the models are not reasonably reliable in estimating the magnitude of highest concentrations occurring some time, somewhere within the area, mainly with a terrain area. The chemical mechanism used was CB-IV, which has some limitations on VOCs speciation that could affect air toxics secondary formation. Finally, the model could not estimate the primary sulfate and nitrate species on DPM.

The key uncertainty in the exposure assessments for air toxics is that these are based on the worst-case assumption that all individuals are potentially exposed outdoors to air toxics at one location for 24 hours/day, 365 days/year for 70 years. The potential exposure to air toxics depends upon types of activities performed, locations of those activities, and duration of time an individual resides in an urban area.

The health risk assessment examines potential cancer risks and risks from other kinds of adverse effects. The methods used to evaluate cancer risk are designed to provide the highest possible estimate of risk associated with exposure to air toxics. The cancer factors used to estimate excess lifetime cancer risks are considered by the U.S. EPA as conservative, representing the most plausible upper bound on the risk. These are calculated in a manner that provides the largest possible slope factor at low levels of exposure, are based on cancer incidence data from the most sensitive animal test species. The methods used to evaluate other kinds of adverse effects (non-cancer effects) are based on protection of sensitive members of the population. However, for those two kinds of risk factor there is a certain uncertainty due to the synergism or antagonism effect of the chemical mixture of air toxics. The epidemiological studies are also affected by uncertainty, principally due to confounders, such as ETS, genes, etc.

4.7   SUMMARY AND OBSERVATIONS

The summary and observations for the modeling results of emissions and exposure concentrations are presented in this section.

4.7.1 Base Case Emissions Inventory

1. The major area source of acetaldehyde, acrolein, benzene, 1,3-butadiene, and formaldehyde emissions were municipal solid waste open burnings, wildfires, and

prescribed burning in Tennessee for 1999, which accounted for 55.4% of area

sources.

2. The main anthropogenic contributions of acetaldehyde, benzene, 1,3-butadiene, and

   formaldehyde emissions were due to the on-road and non-road mobile sources. While

   that the main acrolein sources were the area sources.

3. The highest HAPs emissions occurred in the Atlanta metropolitan area, Georgia,

   followed by Nashville (Davidson County), Memphis (Shelby County), Tennessee,

   and Birmingham (Jefferson County), Alabama in the Southeaster U.S. The acrolein,

   1,3-butadiene, and formaldehyde emissions were clearly higher in Florida compared

   with surrounding states.

4. Acetaldehyde emissions were highly affected by biogenic emissions in the

   Southeastern U.S., which were more significant in the summer season, June and

   September of 1999.

4.7.2 Modeling Results on Source Emissions Scenarios

5. The 2020 HAPs emissions from on-road sources were significantly lower than those

   from 1999, which resulted in a 67.6% reduction in the whole domain, and 64.1% in

   Tennessee. The reductions for acetaldehyde, acrolein, benzene, 1,3-butadiene, and

   formaldehyde in the whole domain were 66.1, 70.2, 66.7, 71.9, and 68.9 %

   respectively. Those HAPs reductions were higher than those estimated and proposed

   by the U.S. EPA from 1996 to 2020, whose proposed values were 57, 60, 57, 64 %

for acetaldehyde, benzene, 1,3-butadiene, and formaldehyde respectively in the whole nation.

6. The total criteria pollutant emissions reductions from those CNG scenarios were not significant in the modeling domain, with 15.7 % attributed to the scenario that used CNG on LDVs and 18.9 % to the scenario that used CNG on all vehicles in 1999.

7. The NMIM and MOBILE6.2 models have not incorporated functions to estimate accuratelly emissions for NH_3 and particulate matter for diesel vehicles when swishing to CNG fuel.

8. The NMIM and MOBILE6.2 models do not generate HAPs emissions to CNG vehicles; therefore, it was not possible to model those HAPs CNG scenarios on SMOKE2.0 and CMAQ.

9. The maximum daily reduction of EC emissions in Nashville occurred in June for the hypothetic scenario without DFS, which accounted for 85%. During March and September the maximum reductions were 80% and December 73%. That maximum reduction occurred in June, because in the summer season construction becomes more active in Tennessee. In winter the impact of wood smoke becomes important, demonstrating the seasonal impact of wood smoke on EC emissions.

10. The maximum EC reduction generated by the scenario without on-road sources was 43% on average, followed by the scenario without on-road diesel-fueled sources with 37%, the scenario without HDVs with 37%, the year 2020 scenario with 28%, and the scenario without LDVs with an average reduction of 6%. Better reduction strategies must be considered for mobile sources to reduce future EC emissions.

11. The maximum reductions of daily acetaldehyde emissions at Nashville were due to the scenario that did not consider on-road sources in the modeling domain with an average reduction of 70%, followed by the scenario without LDVs with 62%, the scenario for the year 2020 with 46%, and the scenario without biogenic emissions with an average reduction of 32%. The rest of the scenarios performed a reduction lower than 8%. The biogenic emissions generated a significant seasonal impact on acetaldehyde emissions.

12. The highest acrolein emissions reductions at Nashville occurred for the hypothetic scenario without on-road sources, which accounted on average for 56%, followed by the scenario without LDVs with 35%, the scenario without HDVs with 21%, the scenario for the year 2020 with 19%, the scenario without DFS with 17%, and the scenario without on-road DFS with 12%. The main emissions contribution came from open burning sources, which must be strongly controlled for the Southeastern U.S.

13. The highest benzene emissions reductions at Nashville occurred for the hypothetic scenario without on-road sources, which accounted in average for 79 %, followed by the scenario without LDVs with 74 %, and the scenario for the year 2020 with 61 %. The rest of the scenarios showed reductions lower than 6 %. The greatest benzene sources were the gasoline LDVs sources and therefore, the on-road mobile sources regulations will significantly reduce the benzene emissions in 2020.

14. The highest 1,3-butadiene emissions reductions at Nashville occurred for the hypothetic scenario without on-road sources, which accounted on average for 82 %, followed by the scenario without LDVs with 71 %, and the scenario for the year 2020 with 60 %. The rest of the scenarios showed reductions lower than 11 %. The greatest

1,3-butadiene sources were the gasoline LDVs sources like benzene and therefore, the on-road mobile sources regulations will significantly reduce the 1,3-butadiene emissions in 2020.

15. The maximum reductions of daily formaldehyde emissions at Nashville were due to the scenario that did not consider on-road sources with an average reduction of 70%, followed by the scenario for the year 2020 with 50%, the scenario without LDVs with 46%, the scenario without DFS with 31%, the scenario without HDVs emissions with 24%, and the scenario without on-road DFS with 17%. It indicated that LDVs were the main formaldehyde contributors followed by HDVs.

16. The maximum average daily NOx reductions at Nashville were performed for the scenario without on-road sources with 66 %, followed by the scenario for the year 2020 with 48 %, the scenario without DFS with 41 %, the scenario without LDVs with 35 %, the scenario that did not consider HDVs with 30 %, and the scenario without on-road DFS with an average reduction of 27%. It indicated that DFS were the main NOx contributors.

4.7.3 Base Case Modeling Performance

17. In general, the MM5 model version 7 tracked well with the monitored temperature for the analyzed 4 months, except the first 15 days of March, which were not considered in later analysis.

18. The predicted temperature had a normalized bias of –0.19 %, a normalized gross error of 0.55 %, and a linear correlation of 0.945, which were statistically very significant.

Therefore, although the modeling considered a 36-km grid size, the MM5 model performance was considered robust to run SMOKE and CMAQ scenarios analysis for HAPs and criteria pollutants.

19. The modeled annual mean concentrations compared reasonably well against the observed values, mainly for EC and formaldehyde. However, the model did not perform reasonably for acetaldehyde, benzene, and 1,3-butadiene. The possible causes were the zero concentrations in the boundary conditions, an inaccurate HAPs area emissions inventory, the inventory and meteorological data were based on different years, and a modeling grid size of 36 km was used. In fact, a perfect agreement between the grid average values with monitored measurements is not expected. Also, for a long-term health effects point of view, the annual priority MSATs concentrations is the relevant measure rather than hourly or daily concentrations.

20. The model under predicted the acetaldehyde, benzene, 1,3-butadiene, formaldehyde, and EC daily concentrations for most days of 2003. The normalized bias ranged from 13 to 57% over those four months, and the normalized gross error ranged from 28 to 57%.

21. The CMAQ base case performance was good enough to analyze the proposed emission scenarios on ground level concentrations and health risk, because the analysis approach considered the difference in mass concentrations and health risk values among the proposed emission scenarios and the base case scenario rather than the absolute mass concentrations or health risk values. This assumed that the factors that contributed to the under and over prediction of those air toxics and EC

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concentrations for March, June, September, and December contributed similarly in all the scenarios considered in this analysis, causing minimal effects on the differences among the scenarios.

4.7.4 Base Case CMAQ Modeling Results

22. Maximum PMSATs concentrations occurred between 6 and 9 PM in Nashville, which correspond to a period when the low mixing height minimizes dilution of the primary emissions.

23. The urban benzene, 1,3-butadiene, and DPM concentrations decreased rapidly outside the source or urban areas to relatively low background concentrations. This behavior was different than formaldehyde and acetaldehyde, whose ambient concentrations were high in extensive southeastern areas, especially in summer season.

24. Peoples were more expose to acetaldehyde and formaldehyde at early mornings.

25. Most of the emitted primary acetaldehyde at morning rush-hour traffic rapidly photo reacted and dispersed around 1 PM in summer season in Nashville. At night, the acetaldehyde emitted during afternoon rush-hour traffic slowly reacted and was dispersed as a result of a greater atmospheric stability.

26. Most of those VOCs acetaldehyde precursor emitted into the air by morning and afternoon rush-hour traffic in summer season were converted to secondary acetaldehyde, but at the same time, it was dispersed as a result of an unstable atmosphere by about noon in Nashville. During cold months, a reduced amount of

secondary acetaldehyde was formed through the reaction of its VOCs precursors. At the same time, less primary acetaldehyde was photo chemically decomposed and more was dispersed due to the higher wind speed during December.

27. The total acetaldehyde concentration during December was lower than June. The maximum secondary acetaldehyde contribution in December was around 85% instead of 98% in June, and the maximum mean secondary to primary hourly acetaldehyde ratio was 7 times in December instead of 40 times in June in Nashville.

28. The population of Nashville was exposed almost 24% higher daily acetaldehyde concentrations in the summer than winter season, mainly due to secondary formation. The annual modeled acetaldehyde concentration was 1.11 ug/m^3 in Nashville, TN, 2003.

29. Like acetaldehyde, most of the emitted primary formaldehyde at morning rush-hour traffic rapidly photo reacted and dispersed at noon in summer season in Nashville. At night, the primary formaldehyde emitted during afternoon rush-hour traffic slowly reacted and was dispersed as a result of a greater atmospheric stability.

30. Most of those VOCs formaldehyde precursor emitted into the air by morning and afternoon rush-hour traffic in summer season were decomposed to secondary formaldehyde, but at the same time, it was dispersed as a result of an unstable atmosphere by about noon at Nashville. During cold months, a reduced amount of secondary formaldehyde was formed through the reaction of its VOCs precursors. At the same time, less primary formaldehyde was photo chemically decomposed and more was dispersed due to the higher wind speed during December.

31. The total formaldehyde concentration during December was lower than June. The maximum secondary formaldehyde contribution in December was around 73% instead of 98% in June, and the maximum mean secondary to primary hourly formaldehyde ratio was 4 times in December instead of 70 times in June.

32. The population of Nashville was exposed almost 183% more to daily formaldehyde concentration in June than in December, mainly due to secondary formation. The annual modeled formaldehyde concentration was 2.25 ug/m^3 at Nashville, TN, 2003.

33. The secondary contribution to total formaldehyde was greater than acetaldehyde in June and September, but it was lower than acetaldehyde during March and December, mainly in December. Similarly, the secondary contribution on formaldehyde and acetaldehyde produced more variability during December, principally on formaldehyde.

34. CMAQ did not simulate properly acrolein, since it did not produce primary concentrations for March, September, and December, except June.

35. The Nashville population was more expose to benzene and 1,3-butadiene at early mornings.

36. Primary benzene and 1,3-butadiene emitted during morning rush-hour traffic reacted slowly during the daytime of summer season and were dispersed at around 1PM because of the wind speed in Nashville. At night, the benzene and 1,3-butadiene emitted during afternoon rush-hour traffic reacted very slow and were slowly dispersed until the new benzene and 1,3-butadiene were emitted during morning traffic congestion again. High concentrations of benzene and 1,3-butadiene from evening traffics sometimes persisted past midnight. The temporal variability of

benzene and 1,3-butadiene were very pronounced in the urban areas, with peak concentrations in the mornings and evenings. The peak concentrations during commute hours were consistent with a predominance of on-road mobile sources in the benzene and 1,3-butadiene emissions.

37. During cold months, less benzene and 1,3-butadiene were decomposed due to the lack of enough OH$^\bullet$. At the same time, more benzene and 1,3-butadiene were dispersed due to the higher wind speed during December. As a result, the Nashville population was 72 and 133% more expose to benzene and 1,3-butadiene concentrations, respectively, during winter than summer season.

38. The annual modeled benzene and 1,3-butadiene concentrations were 0.68 and 0.05 ug/m^3 respectively in Nashville, TN, 2003.

39. The highest benzene and 1,3-butadiene concentrations were produced in Atlanta, GA, where their concentrations were about a factor of 10 and 26 higher respectively at the Atlanta urban location relative to the rural locations.

40. The Nashville population was more expose to EC and DPM at evening, since the maximum hourly EC and DPM concentrations occurred between 6 and 9 PM and the minimum concentrations occurred between 1 and 3 PM. A second maximum occurred between 6 and 10 AM.

41. The main factor that reduced the emitted primary EC and DPM was the wind dispersion at afternoon hours. An important amount of EC and DPM emitted during morning rush-hour traffic were dispersed at around 2 PM as a result of the higher wind speed during afternoon hours. At the end of the day and at night, the EC and

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DPM emitted during afternoon rush-hour traffic dropped and dispersed slowly until the new EC and DPM were emitted during morning traffic congestion again.

42. The highest DPM concentrations were produced in Atlanta, GA. Concentrations of DPM were typically a factor of 11 higher at the Atlanta urban site than at the rural sites.

43. More significant temporal fluctuations were seen at the urban areas. The rural areas seem less significantly affected by local emissions of DPM than in the case of benzene and 1,3-butadiene. The reason was that the on-road sources contributed only by about 40% of diesel particulate emissions instead of 60 and 80% for benzene and 1,3-buadiene, respectively.

44. The urban benzene, 1,3-butadiene, and DPM concentrations decreased rapidly outside the source or urban areas to relatively low background concentrations. This behavior was different than formaldehyde and acetaldehyde, whose ambient concentrations were high in extensive southeastern areas, especially in summer season.

45. The Nashville population was exposed to DPM almost similar during each month. The EC concentrations showed slightly differences between hot and cold months, since the EC concentrations of December were 30% higher than June, demonstrating the seasonal impact of wood smoke on EC concentrations. The annual modeled EC and DPM concentrations were 0.57 and 0.55 ug/m^3 respectively at Nashville, TN, 2003.

46. The mean DPM contribution to the total hourly PM$_{2.5}$ was higher during morning and afternoon rush-hour traffic, i.e., around 7 AM and 5 PM at Nashville. The minimum

was produced around 1 PM. The daily DPM contribution to the total $PM_{2.5}$ was similar for each month and was around 2.6%.

47. Acetaldehyde and formaldehyde produced higher concentrations in June and September due to the contribution of secondary formation in the Southeastern U.S. The rest of the pollutants generated higher concentrations during cold months, i.e., March and December, except DPM, which was similar for each month. This seasonality was similar in the monitored data, principally on formaldehyde and acetaldehyde.

4.7.5 Air Toxics Concentrations by Emissions Scenarios

48. Comparing Tables 4-10 and 4-19, the maximum emission and concentration reductions are equivalent to benzene, 1,3-butadiene, EC, and NOx for each scenario in Nashville, except for biogenic sources. However, the maximum emission and concentration reductions of acetaldehyde and formaldehyde were very different. The main reason of this diference is that acetaldehyde and formaldehyde reductions are affected significantly by the secondary formation and by biogenic sources. In contrast, benzene, 1,3-butadiene, EC, and NOx don't have secondary formation in the troposphere. Therefore, running SMOKE could be enough to see the effects on the maximum concentration and health reductions of benzene, 1,3-butadiene, EC, and NOx. Finally, CMAQ-AT has to be run to see the maximum concentration reductions of acetaldehyde and formaldehyde and their health associated effects.

49. The maximum reductions of daily acetaldehyde concentrations for Nashville were due to the scenario that did not consider on-road sources in the modeling domain, with an average reduction of 57% and a maximum of 68% produced in December. This scenario was followed by the scenario without LDVs, with 52%, the scenario without biogenic emissions, with 50%, and the scenario for the year 2020, with 37%. The rest of the scenarios produced a reduction lower than 5%. It indicated that the gasoline LDVs were the main acetaldehyde contributors at Nashville, principally in March and December. During those cold months, the secondary acetaldehyde formation due mainly to biogenic sources was lower and the reductions were mainly on primary acetaldehyde. Better reduction strategies must be considered on LDVs to reduce future acetaldehyde concentrations.

50. The maximum reductions of daily formaldehyde concentrations at Nashville were due to the scenario that did not consider biogenic sources in the modeling domain, with an average reduction of 54% and a maximum of 71% produced in September. This scenario was followed by the scenario without on-road sources with 34%, the without LDVs with 25%, the scenario for the year 2020 with 23%, and the base case scenario without DFS with 12%. The rest of the scenarios produced a formaldehyde reduction lower than 10%. The LDVs were the main formaldehyde contributors from on-road sources at this urban area, mainly in December with 47% reduction.

51. If a future 2020 scenario includes non-road sources with strong regulations, the maximum daily formaldehyde reduction could be better in Nashville, but isoprene will continue as the main formaldehyde precursor.

52. The highest benzene concentration reductions occurred for the hypothetical scenario without on-road sources, which accounted on average for 75 %. It was followed by the scenario that did not consider LDVs with 70 %, and the scenario for the year 2020 with 57 %. The rest of the scenarios showed benzene reductions lower than 5 %. The greatest benzene sources were the gasoline LDVs sources. As a result, the on-road mobile sources regulations will be strong enough to reduce significantly the benzene concentrations in 2020 at Nashville.

53. The highest 1,3-butadiene concentration reductions occurred for the hypothetic scenario without on-road sources, which accounted on average for 83 %, followed by the scenario without LDVs with 72 %, and the scenario for the year 2020 with 63 %. The rest of the scenarios showed reductions lower than 17 %. Like benzene, the greatest 1,3-butadiene sources were the gasoline LDVs sources. Therefore, the on-road mobile sources regulations will be strong enough to reduce significantly the 1,3-butadiene emissions in 2020 by almost 63%.

54. The maximum daily EC concentration reductions due to the scenario without DFS sources was as high as 84% in June and during December was of 73%. That maximum reduction occurred in June due to the higher construction activity in Tennessee, as well as, the lower impact of wood smoke.

55. The maximum reductions were performed by the scenario that did not consider DFS, followed by the scenario without on-road sources, the scenario without on-road DFS, the scenario without HDVs, and the scenario to the year 2020, with 78, 39, 35, 35, and 27% respectively.

56. The maximum daily NOx reductions were produced for the scenario without on-road sources with 64 %, followed by the scenario for the year 2020 with 49 %, the scenario without DFS with 47 %, the scenario without LDVs with 37 %, the scenario that did not consider HDVs with 33 %, and the scenario without on-road DFS with an average reduction of 30%. Although the scenario 2020 included the on-road sources regulations only, this scenario produced a strong NOx reduction of 49%, which was so different than the reductions produced on DPM.

57. A great experience was gained in installing, configuring, and running the following models: MM5 version 3.7, NMIM, SMOKE version 2.0, MCIP version 2.2, CMAQ-AT, CMAQ version 4.3, and PAVE version 2.2. It was possible to perform meteorological modeling of selected months that represented an entire year using the MM5 version 3.7 on the NCAR super computer center through the bluesky machine. All these models applications represent the state-of-the-art in advanced air quality modeling and were used to generate the health risk assessment posed by the priority MSATs.

4.7.6 Air Toxics Health Effects

58. The modeled 4HAPs, DPM, and 4HAPs+ DPM exceeded four, one, and 5 in a million risk of cancer over a lifetime of exposure respectively.

59. All hazard ratios were less than one in Nashville, indicating no significant risk on non-cancer effects base on toxicological evidence functions.

60. Formaldehyde was the unique PMATs that showed a hazard ratio close to 1, which was allocated in the Atlanta metropolitan area.

61. The modeled RR of CVD mortality, lung cancer mortality, and asthma hospital admissions for Nashville were 1.0109, 1.0069, and 1.0018, respectively. In other words, 1 µg/m3 increases in annual DPM exposure was associated with a 1.09% increase in the relative risk of CVD mortality, a 0.69% increase in the relative risk of lung cancer mortality, and 0.18% increase in the relative risk of hospital admissions for asthma in Nashville.

62. The modeled OR of chronic illness for COPD for Nashville was 1.0075, i.e., the odds of getting a chronic illness for COPD increase by 0.75% with each additional annual ug/m3 of DPM.

5.0 CONCLUSIONS

The overall objective of the study was to develop a computer tool to assess the public health risk posed by the MSATs on an urban to regional area scale. The tool was developed based on different emissions scenarios and linking the major air toxics concentrations predicted by an advanced air quality model with risk factors associated to cancer and non-cancer effects. This modeling was accomplished using the U.S. EPA's third generation air quality models, CMAQ-AT and CMAQ version 4.3. To demonstrate the system's effectiveness, an analysis was conducted on acetaldehyde, benzene, 1,3-butadiene, formaldehyde, and diesel particulate matter. The region of study includes the entire Southeastern U.S. The period under consideration was for March, June, September, and December 2003. The results were analyzed for the Nashville metropolitan area, Davidson County, Tennessee.

Considering that this research on air toxics emission scenarios was based on relative analysis rather than estimates of absolute exposure concentrations and health risk values, the following conclusions were reached:

5.1 Base Case Inhalation Health Risk

1. The health risk assessment associated with the priority MSATs control will continue be one of the most important issues in the U.S. EPA's air quality rulemaking,

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principally for DPM. In this context, the proposed protocol through Models-3/CMAQ was demonstrated and can be used for decision makers in the quantitative assessment of new policies that will affect the public health and the air quality by air toxics. Eliminating emission source categories is clearly not a policy option, but rather was used to helped gain a better understanding of the total magnitude of the health effects associated with these major sources of HAPs, principally of DPM.

2. For relative basis point of view, the linking of those PMSATs emissions scenarios to the health risk effects was demonstrated through use of toxicological evidence equations for cancer and non-cancer effects, as well as the use of epidemiological C-R functions for CVD mortality, lung cancer mortality, hospital admissions for asthma risk, and chronic illness for COPD, and the use of an estimation of annual exposure concentrations based on four months that represented each season of the year 2003.

3. Higher formaldehyde and acetaldehyde exposure occurred in the summer season, while higher benzene and 1,3-butadiene concentrations occurred in the winter season. DPM did not show a strong seasonality exposure during the year 2003 in Nashville.

4. DPM posed a cancer risk that was 4.2 times higher than the combined total cancer risk from all other air toxics simulated in the base case scenario for Nashville. Those high cancer risk levels were due mainly to the DPM emitted from goods transportation and construction engines.

5. Higher cancer risk occurred on Southeastern urban areas due to DPM exposure. The highest cancer risk from DPM occurred in Atlanta, GA, followed by Nashville, TN, Birmingham, AL, Raleigh, NC, and Memphis, TN. The cancer risk from those 4HAPs was not only higher in urban areas, but also was high over rural areas of the

Southeastern U.S., mainly due to secondary formation of acetaldehyde and formaldehyde. The 4HAPs+DPM cancer risk was about a factor of 9.2 higher at the Atlanta urban location relative to the rural locations.

6. Higher relative CVD mortality, lung cancer mortality, hospital admissions for asthma risk, and chronic illness for COPD odds ratio occurred in Southeastern urban areas due to DPM long-term exposure, principally at Atlanta, GA, followed by Nashville, TN, Birmingham, AL, Raleigh, NC, and Memphis, TN.

5.2   Inhalation Health Risk by Emission Scenarios

7. The main cancer risk reductions from those 4HAPs were due to the contribution of biogenic sources with 32.2%, which generated high secondary acetaldehyde and formaldehyde in the summer season. This condition was followed for the scenario that did not consider on-road sources with a 27.5% of reduction. The main reductions were due to the air toxics contributions generated by gasoline LDVs, principally benzene and 1,3-butadiene. As the scenario 2020 included the on-road sources regulations only, the 4HAPs cancer risk showed a reduction as low as 19.4. If there are no on-road sources or DFS in the modeling, the cancer risk reduction is not expected to be reduced significantly for those vapor air toxics, because major sources are important sources of acetaldehyde emission. Area and non-road sources are important sources of benzene, 1,3-butadiene, and formaldehyde. For that reason, better air toxics reduction strategies must be considered on the other emission sources

as well, mainly on non-road and open burning sources. The rest of the scenarios showed reductions lower than 4.4 %, indicating that DFS and HDVs were not important vapor air toxics contributors in Nashville, TN.

8. The main reductions in DPM cancer risk were due to the contribution of non-road DFS sources with 57.9 % reduction, followed by the scenario that did not consider on-road DFS sources with 42.1% reduction. For the on-road DFS, the principal reductions were due to the DPM contributions generated by HDDVs rather than LDDVs. An evident positive synergism in the cancer risk reduction occurred when reducing diesel on-road and non-road source emissions simultaneously.

9. The scenario 2020 showed a DPM cancer risk, RR, and OR reduction of approximately 32.8 %.

10. For a long-term exposure to DPM, the main reductions on RR for CVD mortality, lung cancer mortality, hospital admissions for asthma, and OR for COPD chronic illness were due to the scenario that did not consider non-road DFS sources with 57.8% reduction, followed by the scenario without on-road DFS with 42.2%

11. This research provided strong evidence that reducing ambient DPM concentrations will lead to improvement in human health more than other air toxics in Nashville, indicating that better technologies and regulations must be applied to the mobile diesel engines, principally, over non-road diesel sources in Nashville.

12. Running SMOKE could enough to see the effects on the maximum concentration reductions of benzene, 1,3-butadiene, EC, and NOx and their health associated effects. Whereas, CMAQ-AT has to be run to see the maximum concentration reductions of acetaldehyde and formaldehyde and their health associated effects.

13. This approach has inherent limitations because of inability to simulate some primary

    DPM species, such as sulfate and nitrate. However, their contribution on DPM is

    negligible and did not alter the relative analysis of this research.

6.0 RECOMMENDATIONS FOR FUTURE RESEARCH

This research modeled the ambient HAPs and primary DPM concentrations for the Southeastern U.S. and Nashville Metropolitan Area through the use of an advanced photochemical air quality model and identified the major source categories that contributed to the ambient concentrations and the associated inhalation cancer risk. This research was limited in certain aspects, and the following recommendations are made for further study:

1. In general, the model performance and the source scenarios analysis depend on the inventory, the temporal profiles, the meteorological data, and the speciation used. A major amount of effort was concentrated at developing an open burning HAPs and an on-road HAPs and criteria inventory for this study. However, inventory development is based on estimates and is a continual process. Important Open Burning sources that generate HAPs, as yard waste and construction land clearing, could be included in the emissions inventory; however, the AP-42 database and its expanded EIIP documents do not have any speciated VOCs, SVOCs, metals, or PCDD/F data. A recent publication, "Emissions of organic air toxics from open burning: a comprehensive review." written by Lemieux et al., (2004) could be used. Future HAPs emission inventories may include diesel particulate matter by source categories for mobile sources, breaking down diesel emissions by construction, on-road, by vehicle weight, non-road, school buses, farm tractors, rail, barge, garbage trucks, etc. Finally, the temporal profiles that are used to distribute the average emissions to each hour may

also affect the nature of the species predicted and their effect. For example, a more realistic temporal profile for trucks could be incorporated in SMOKE to produce better temporal hourly emissions and concentrations of DPM at places close to highways.

2. Future simulations could include running fine-scale modeling on air toxics to better capture spatial and temporal variability, as well as concentration magnitudes, which could help identify and characterize the hot spots of air toxics compounds from an exposure point of view. These future simulations could simulate an entire year to see with more details the seasonal effects mainly on the coldest and hottest months of the year. These simulations could allow estimating an annual concentration more accurately.

3. More research may be needed to look at improving the model performance. This study was limited by the lack of enough monitored air toxics concentrations in the modeling domain. Modeling may be needed for other urban and rural areas to identify factors and trends that may be used to improve the model performance and its prediction of the air toxics. This research may be needed to look at improving the acrolein performance on CMAQ version 4.5, since this simulation did not produce primary acrolein concentration for March, June, and September.

4. The efficient CB-IV chemical mechanism was developed for high NOx concentrations, which occur typically for urban areas; however, for regional areas with lower NOx conditions the SAPRC99 gas phase mechanism include a more detailed representation of VOC species, the fate of NOx, and of the chemistry of

peroxyl radical species, including air toxics, which are important at low NOx levels. Therefore, future simulations could use the SAPRC99 photochemical mechanism.

5. For DPM simulations, future risk assessment research could split the contribution of diesel engines, such as solid waste trucks, buses, trucks, marine vessels, and locomotives, among others. The research could also include other areas where the diesel sulfur content and diesel vehicles market are higher than the U.S.

6. Future DPM simulations could account for the DPM sources that are apportioned between primary and secondary sulfate and nitrate aerosols. One way to do this analysis would be to redo this sensitivity study but only zero out the DPM emissions and leave the gas phase constant. As result, the gas phase contribution to SO_2, NOx, secondary sulfate, and secondary nitrate would be estimated, and therefore, the primary sulfate and nitrate concentrations on DPM.

7. Considering that Memphis showed high PMSATs concentrations and their respective health effects, it is reasonable to install a HAPs monitoring network in this urban area.

8. The next steps to improve the air quality in Tennessee and its associated health effects due to PMSATs is to implement better non-road DPM control technologies and diesel fuel regulations, as well as, implement the RVP program on gasoline fuel to low benzene, 1,3-butadiene, and other HAPs, mainly in winter season.

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APPENDICES

APPENDIX A

Daily HAPs emissions in Davidson Co. for March, June, September, and December by each scenario
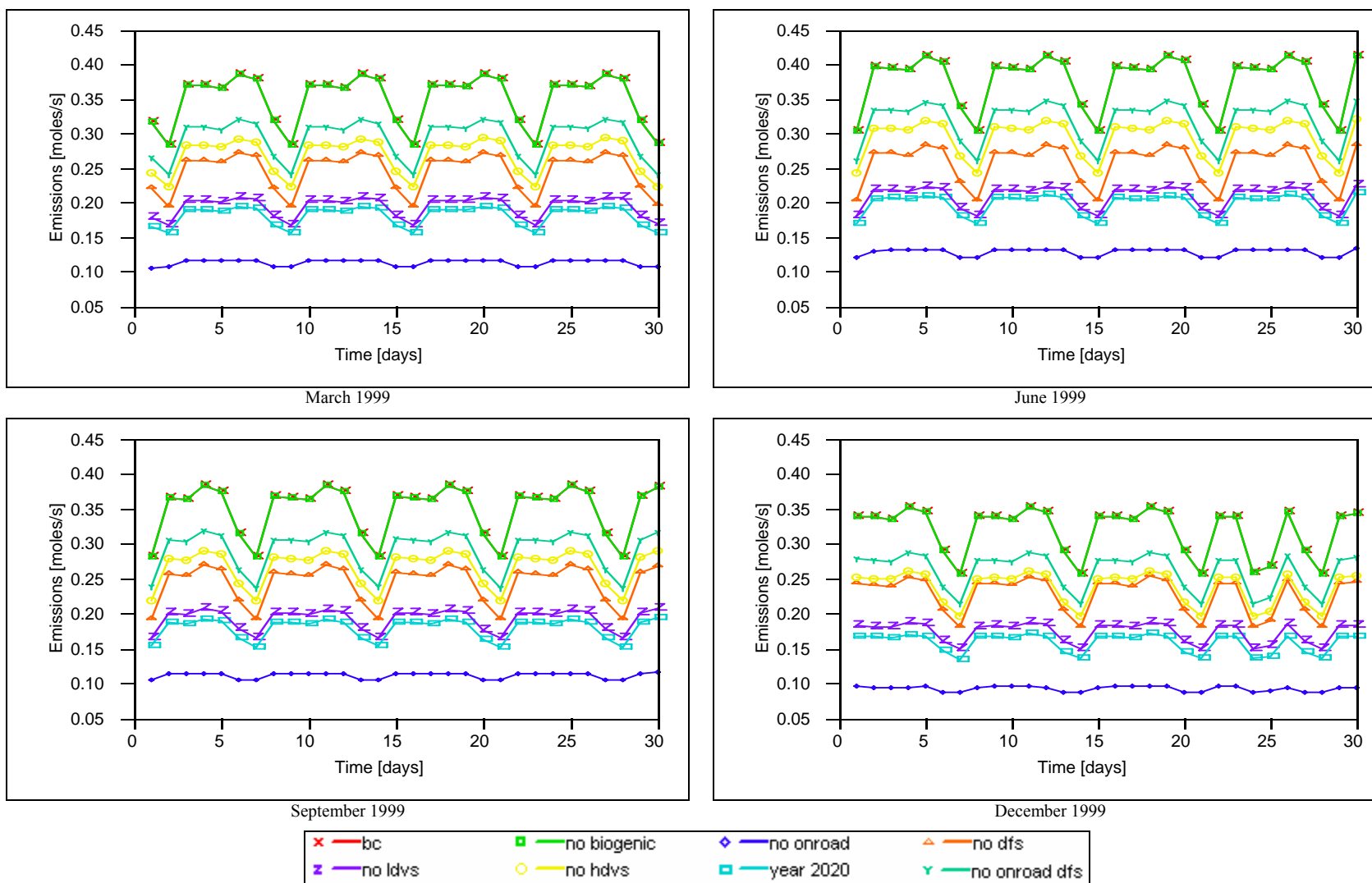
Figure A-1. Daily Formaldehyde Emissions in Davidson Co. for March, June, September, and December by Each Scenario
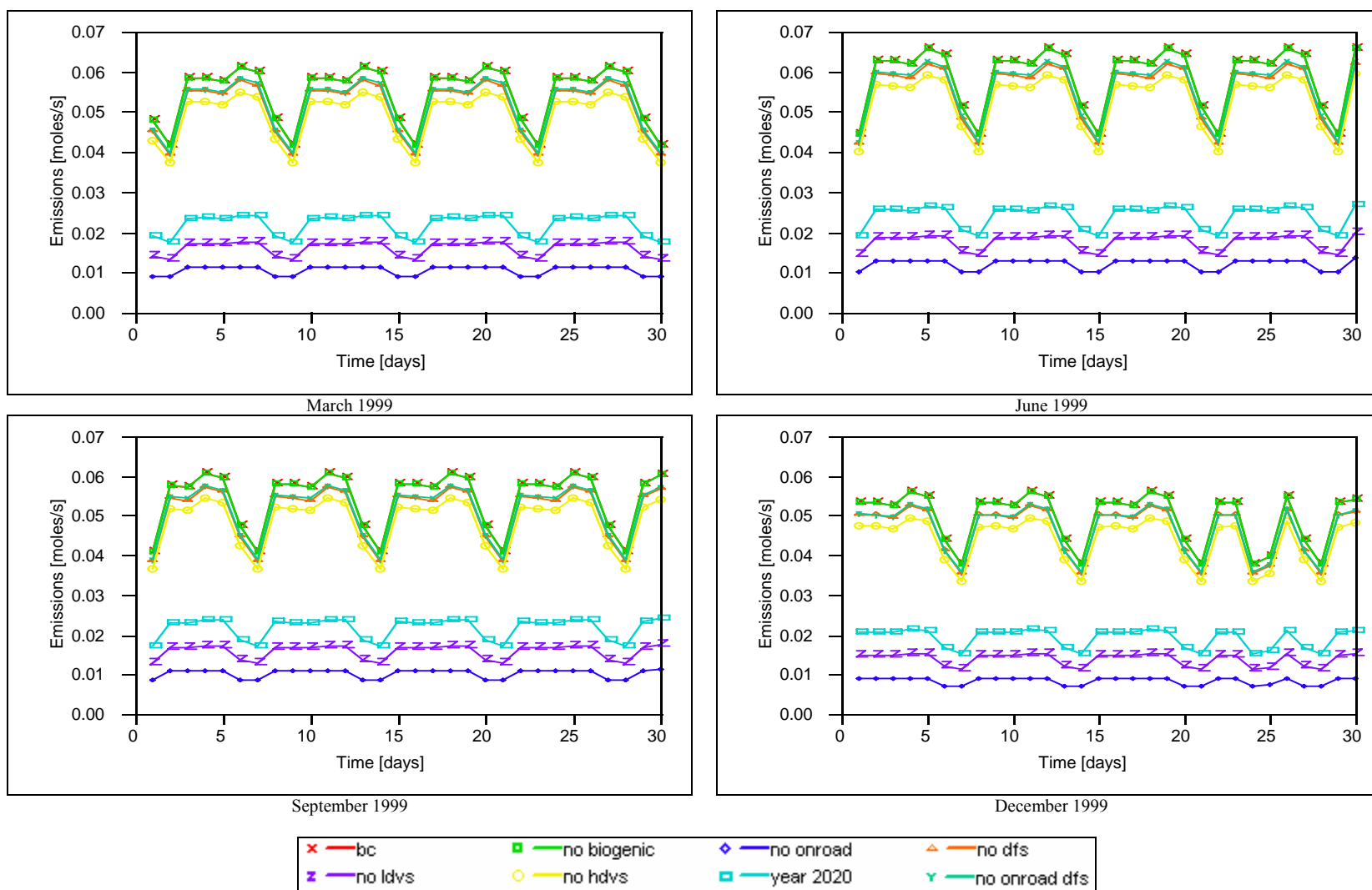
Figure A-2. Daily 1,3 Butadiene Emissions in Davidson Co. for March, June, September, and December by Each Scenario
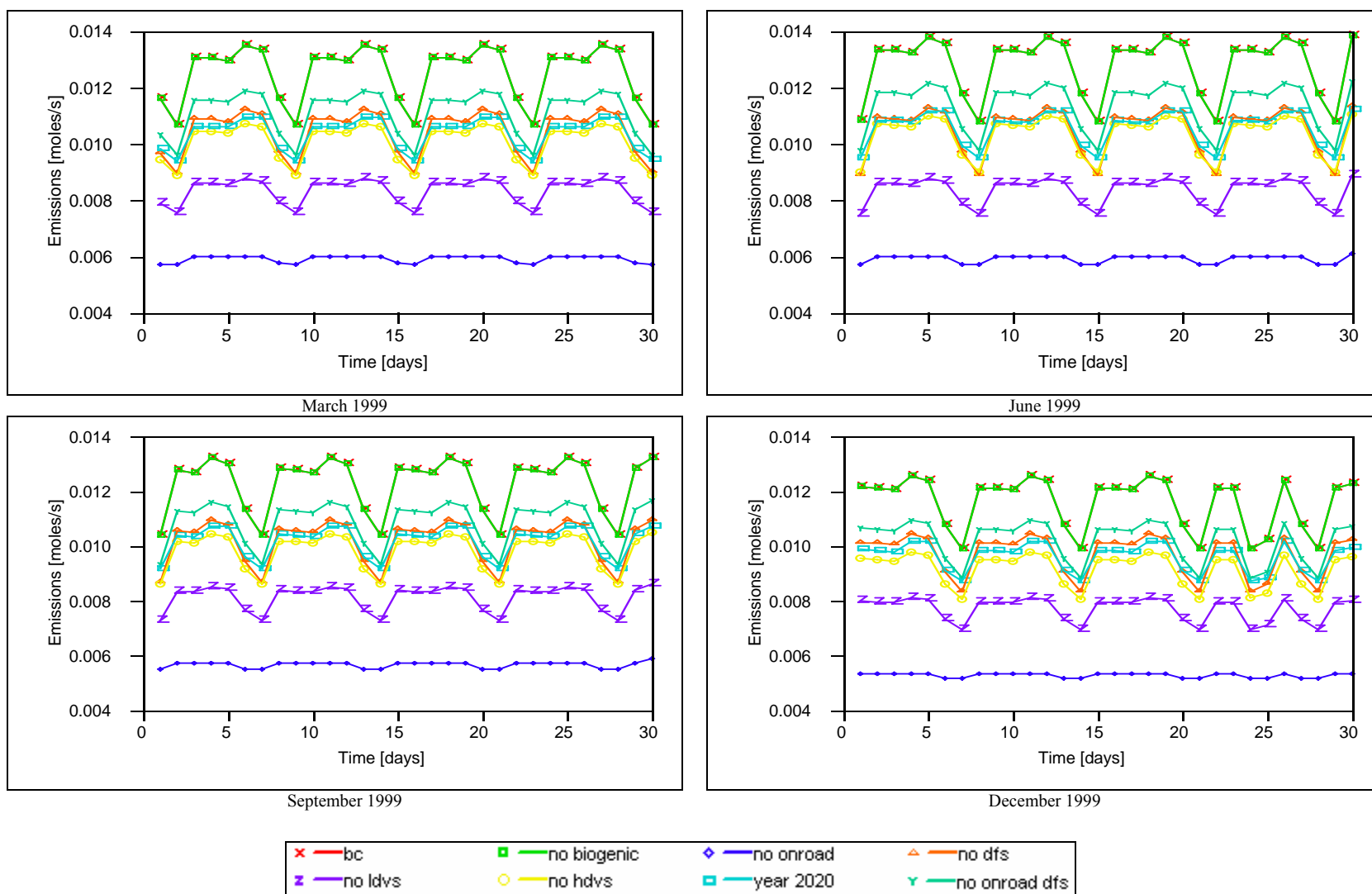
Figure A-3. Daily Acrolein Emissions in Davidson Co. for March, June, September, and December by Each Scenario
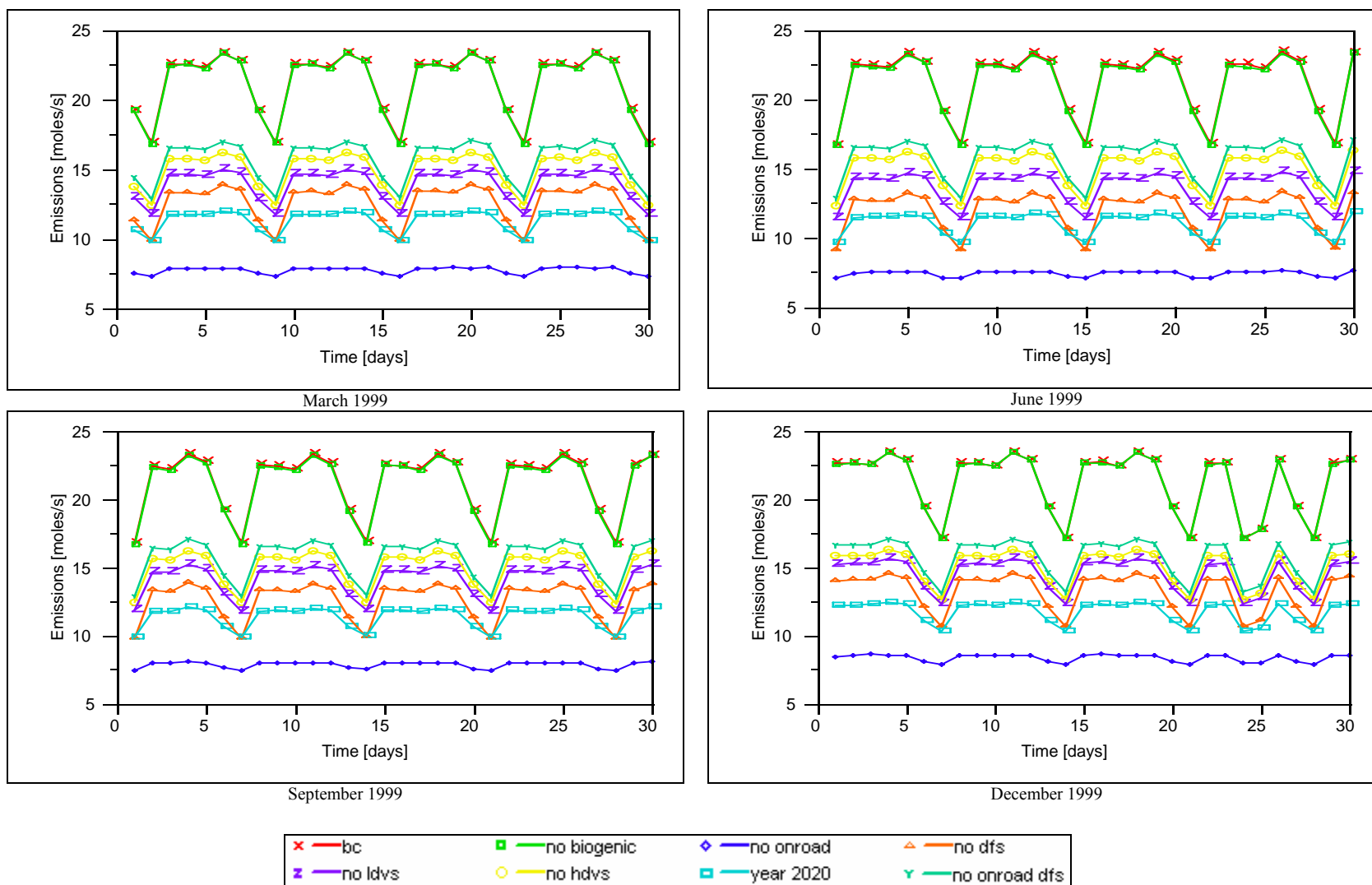
Figure A-4. Daily NOx Emissions in Davidson Co. for March, June, September, and December by Each Scenario

APPENDIX B

Time Series Plots of Hourly Modeled HAPs Average Concentrations
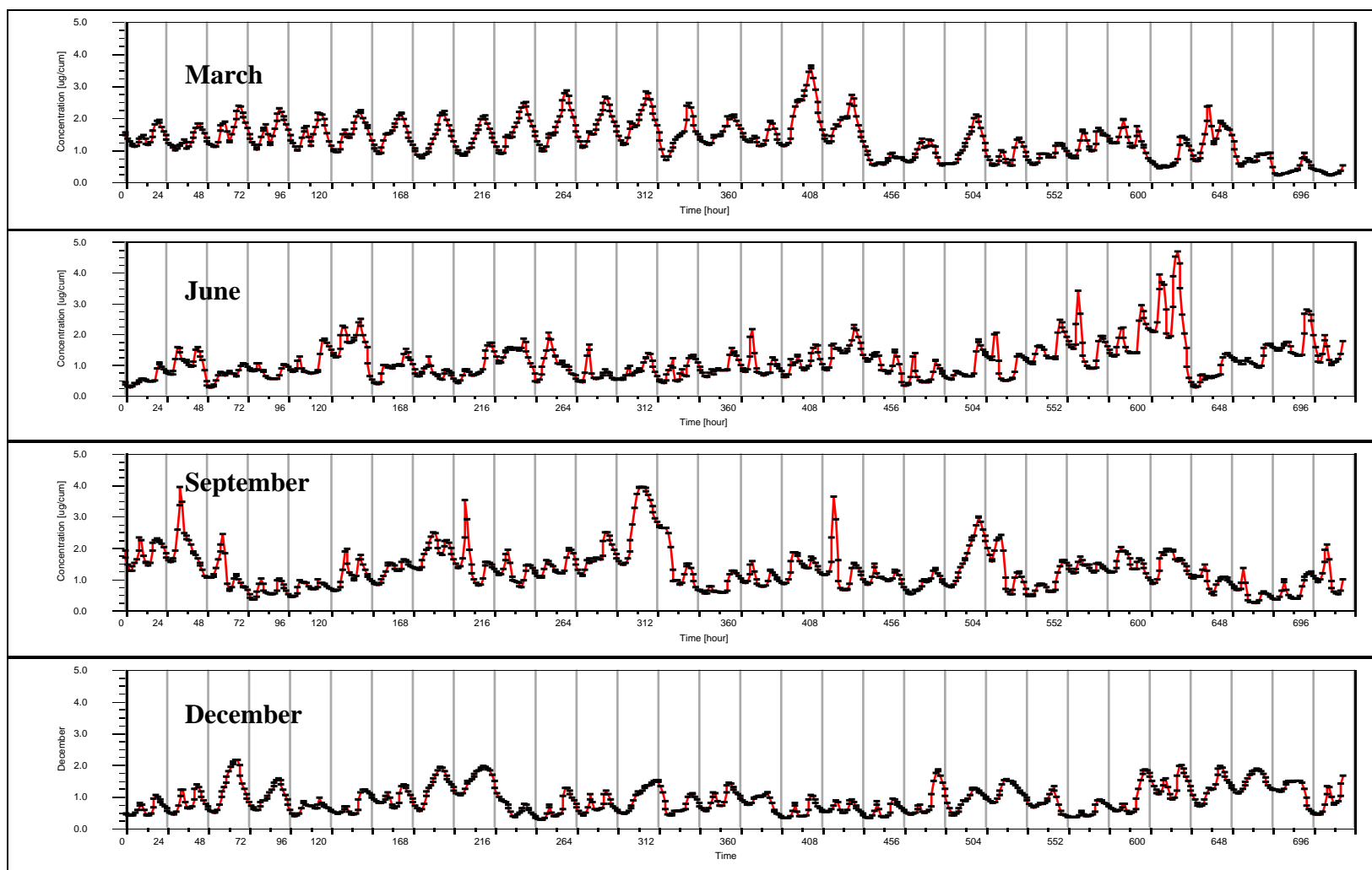
Figure B.1. Modeled Hourly Average Acetaldehyde Concentration in Nashville, March, June, September, and December 2003

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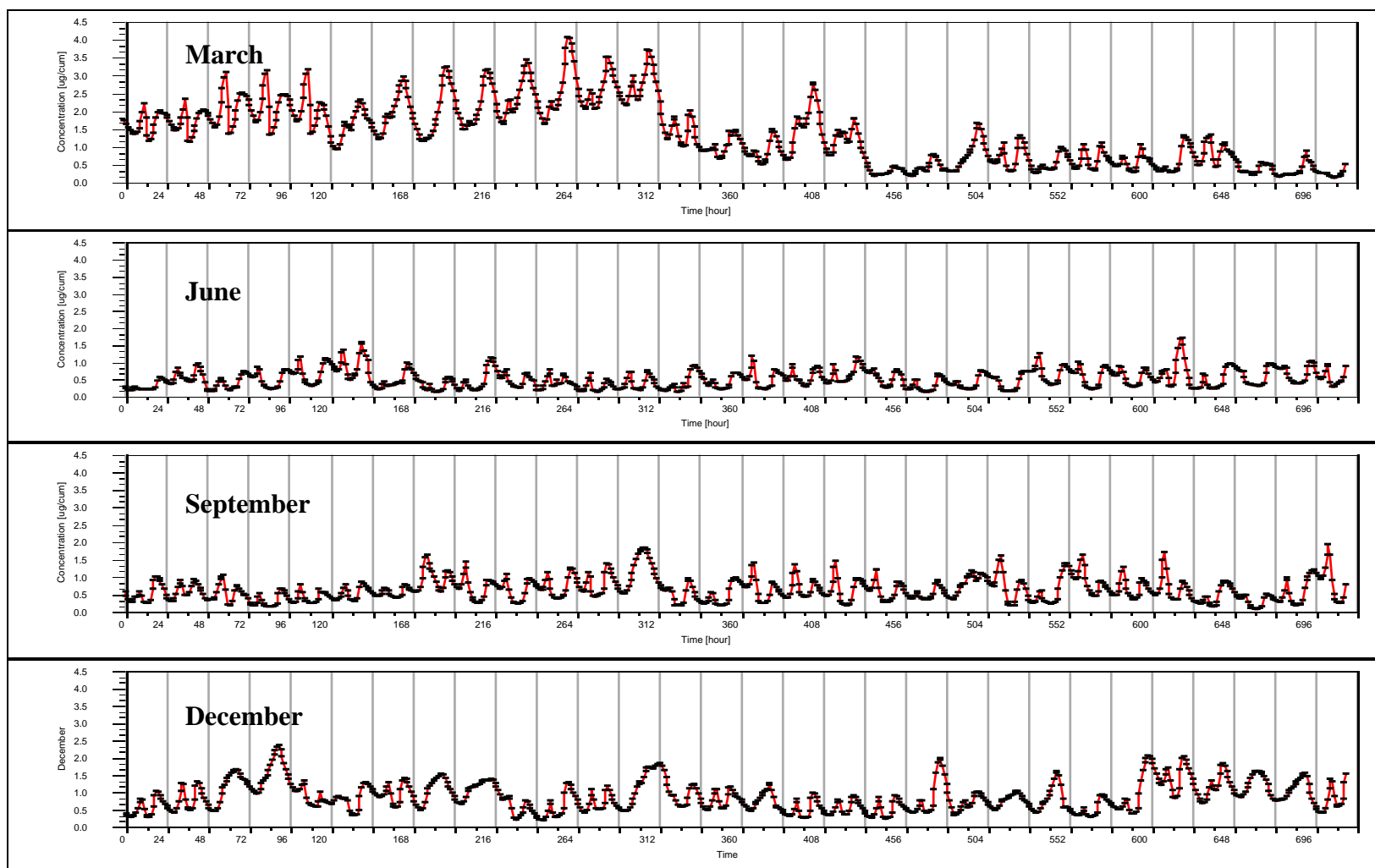
Figure B.2. Modeled Hourly Average Benzene Concentration in Nashville, March, June, September, and December 2003
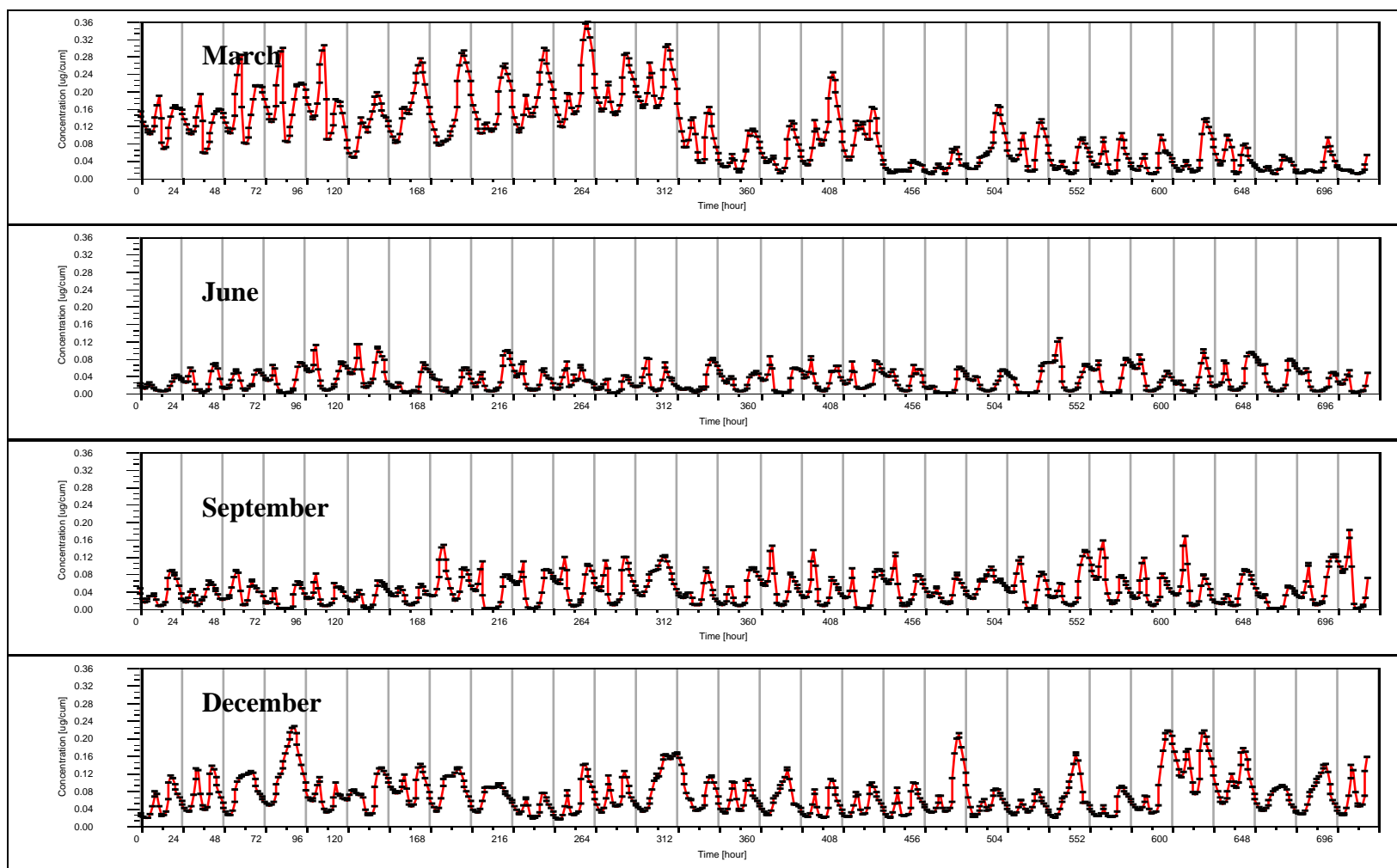
Figure B.3. Modeled Hourly Average 1,3-Butadiene Concentration in Nashville, March, June, September, and December 2003
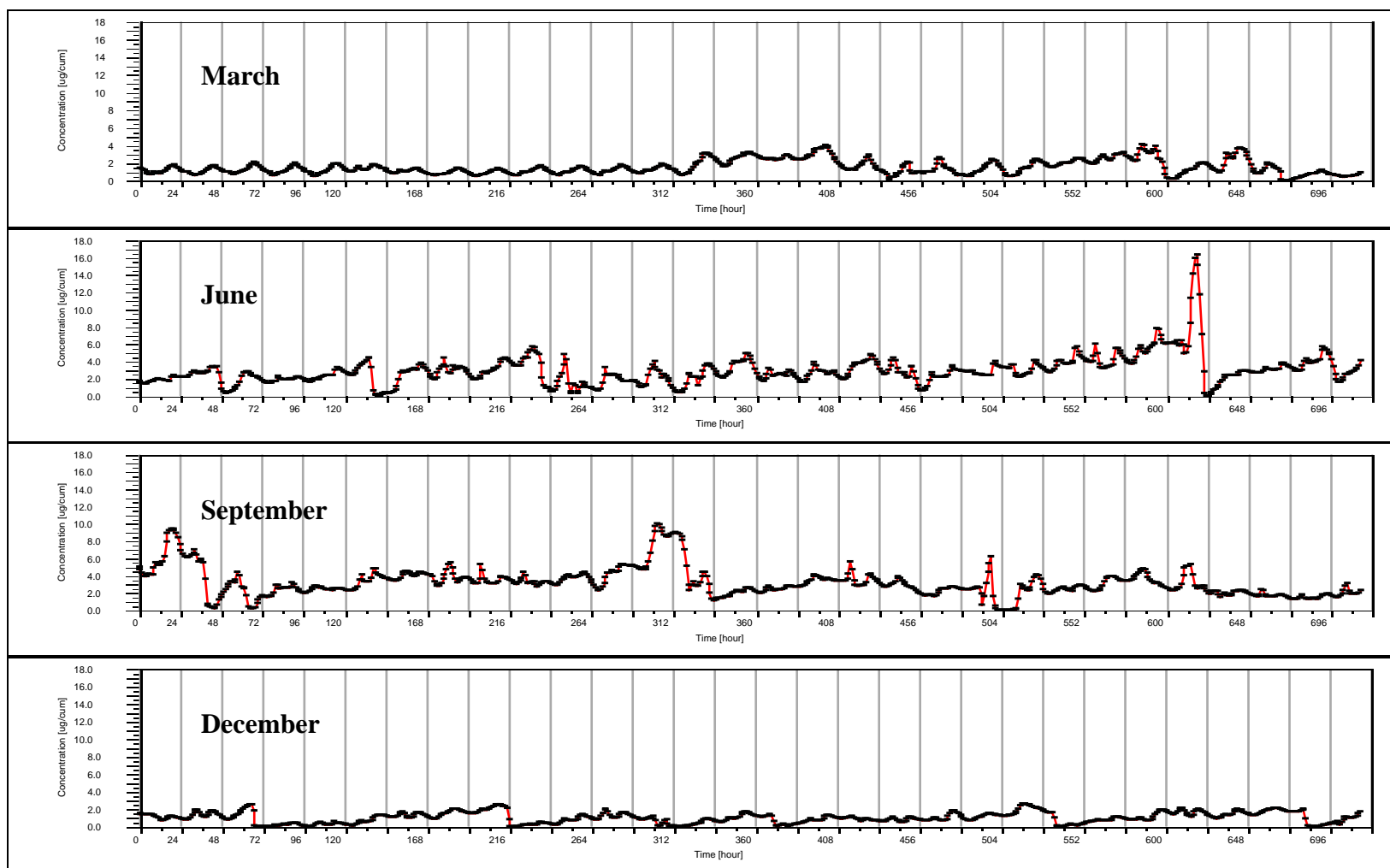
Figure B.4. Modeled Hourly Average Formaldehyde Concentration in Nashville, March, June, September, and December 2003
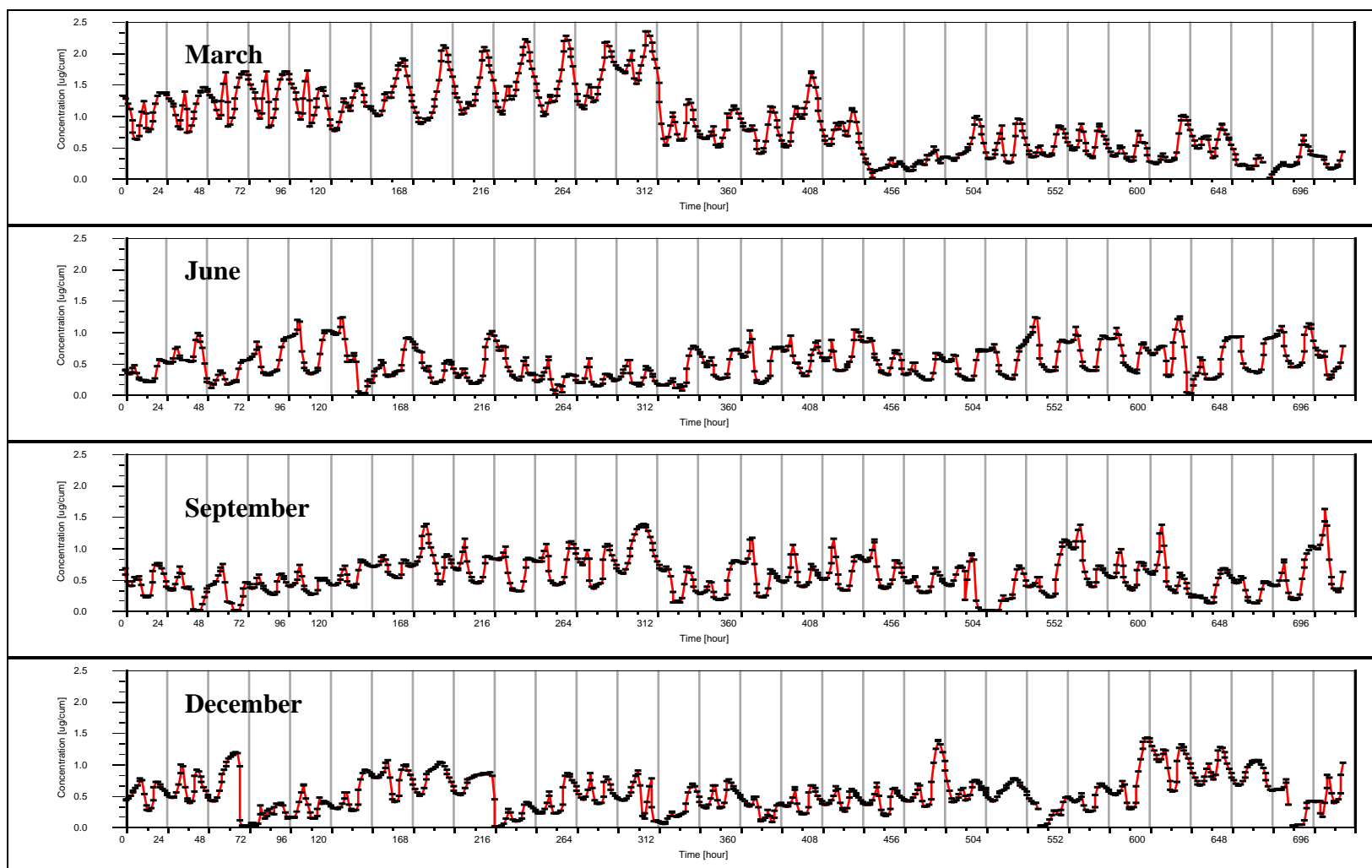
Figure B.5. Modeled Hourly Average DPM Concentration in Nashville, March, June, September, and December 2003
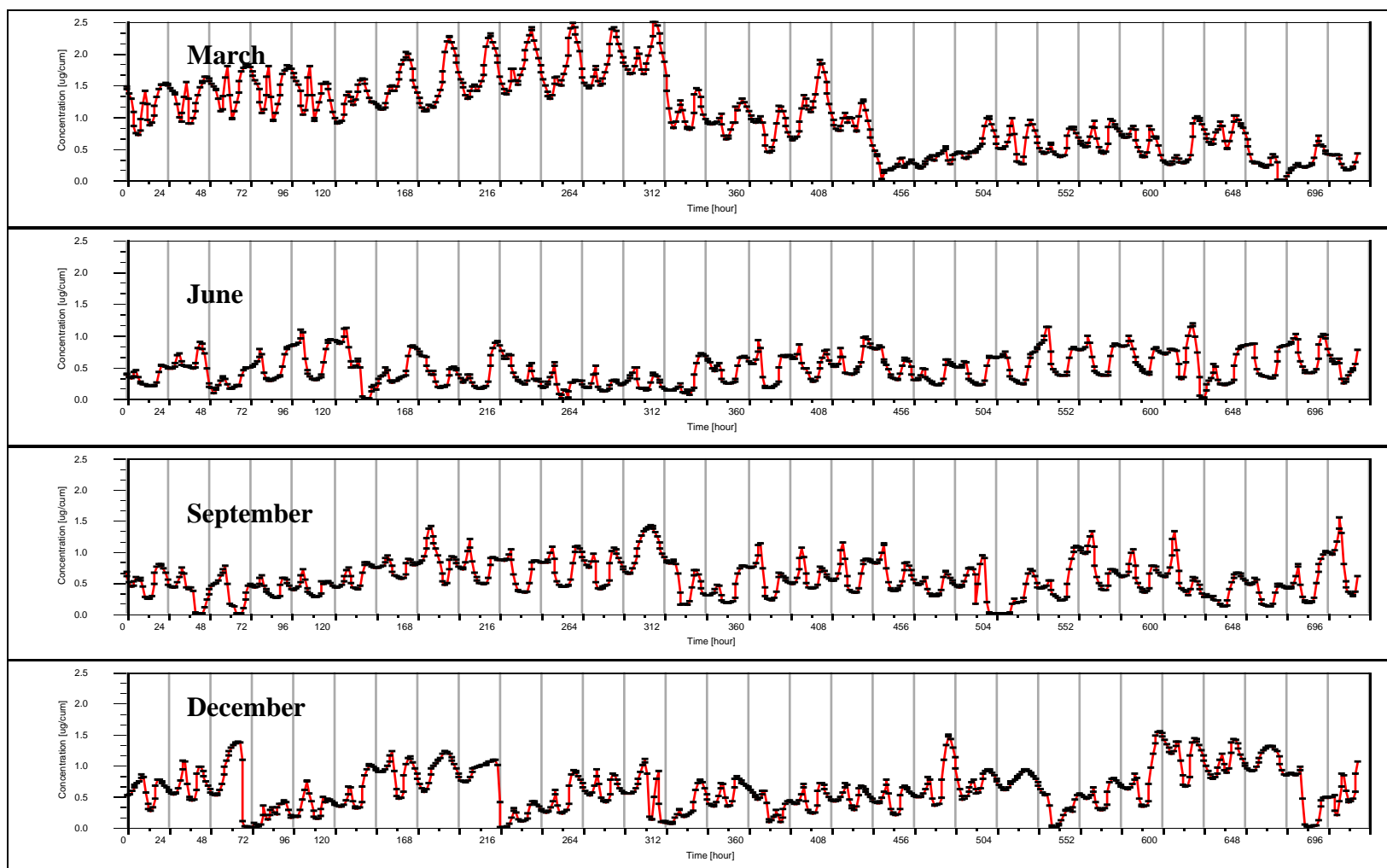
Figure B.6. Modeled Hourly Average EC Concentration in Nashville, March, June, September, and December 2003
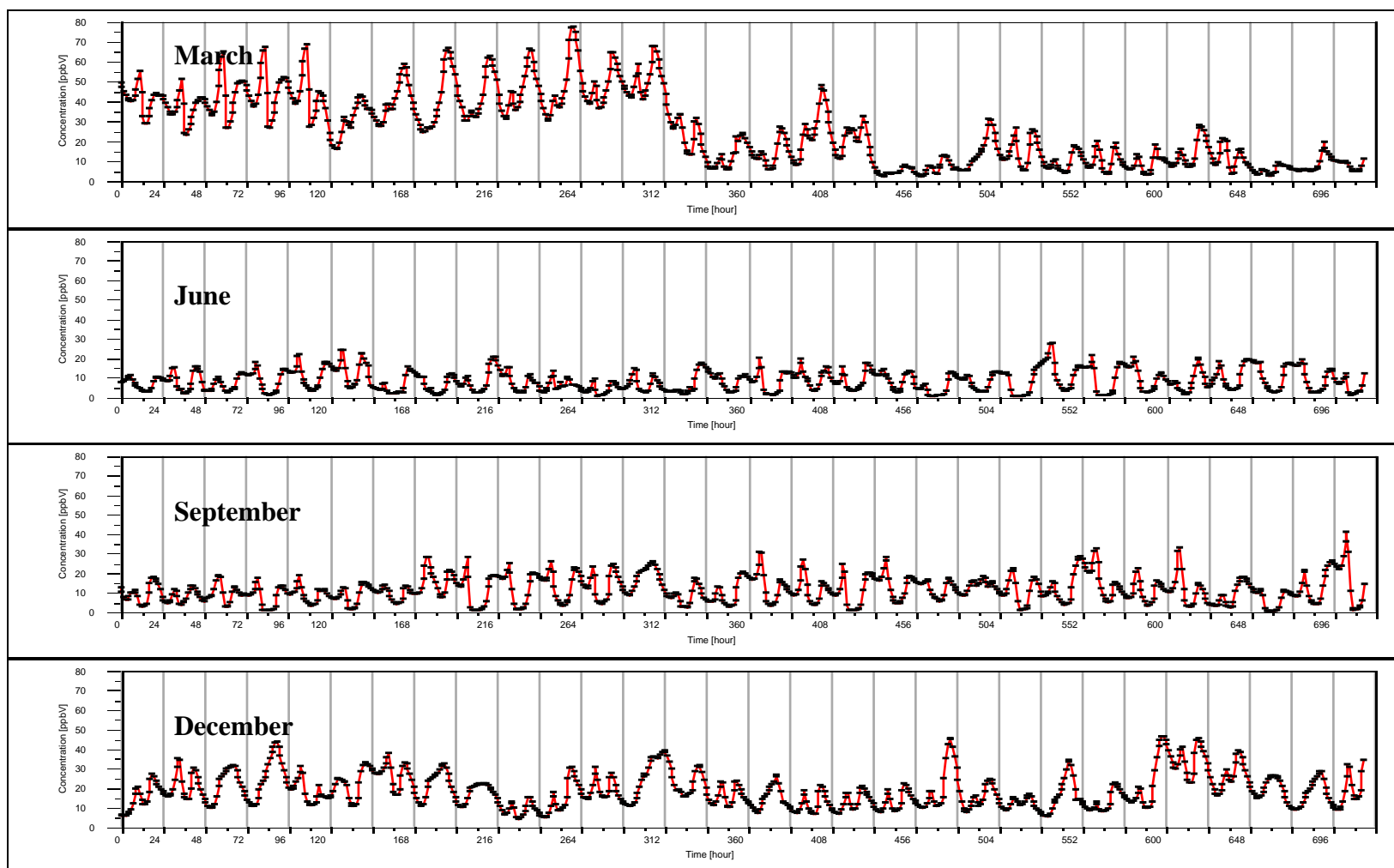
Figure B.7. Modeled Hourly Average NOx Concentration in Nashville, March, June, September, and December 2003

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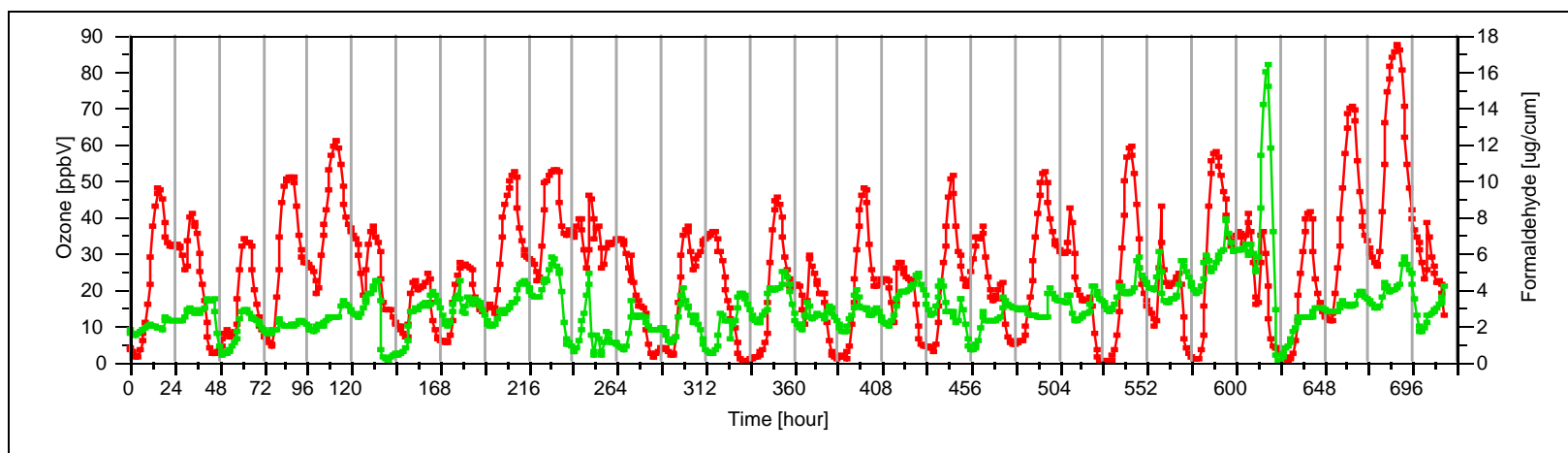
Figure B.8. Hourly Ozone and Formaldehyde Concentrations in Nashville, June 2003. (Red color: Ozone)
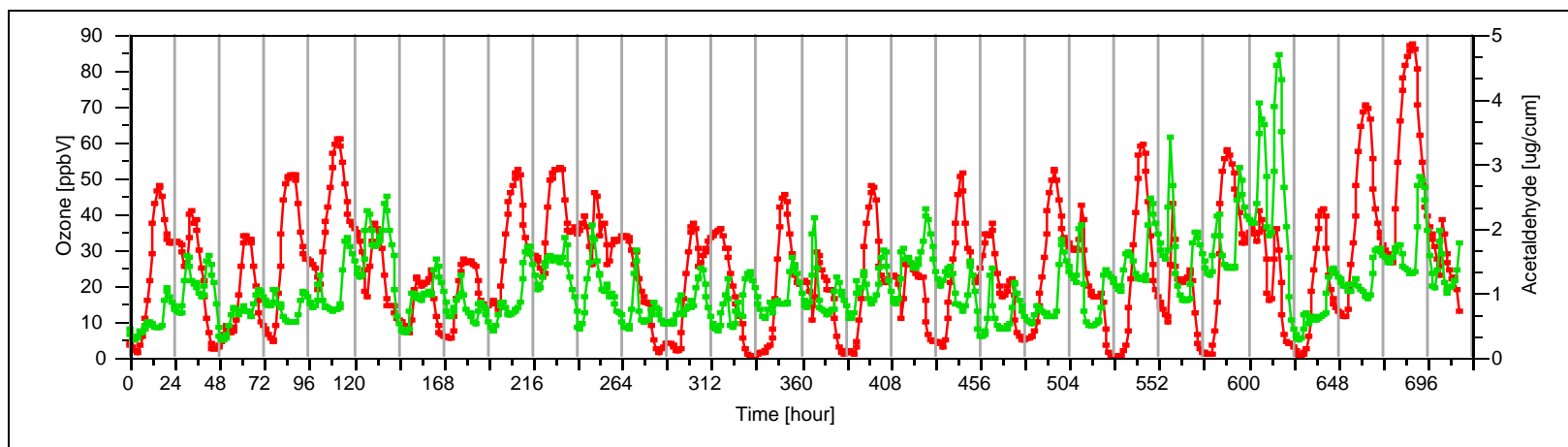
Figure B.9. Hourly Ozone and Acetaldehyde Concentrations in Nashville, June 2003. (Red color: Ozone)
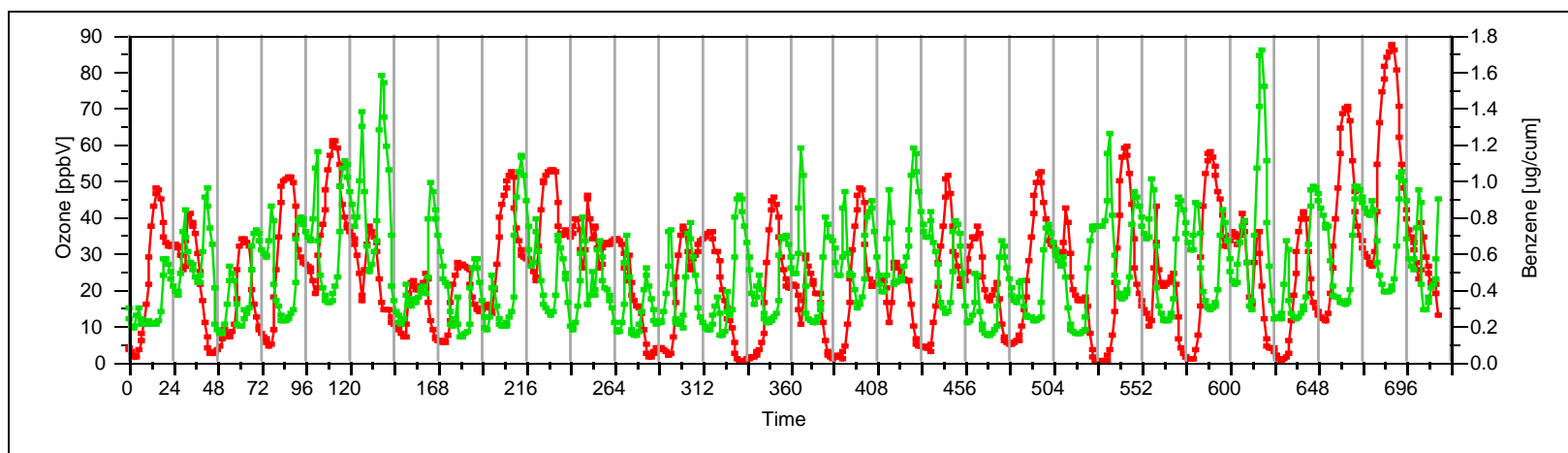
Figure B.10. Hourly Ozone and Benzene Concentrations in Nashville, June 2003. (Red color: Ozone)
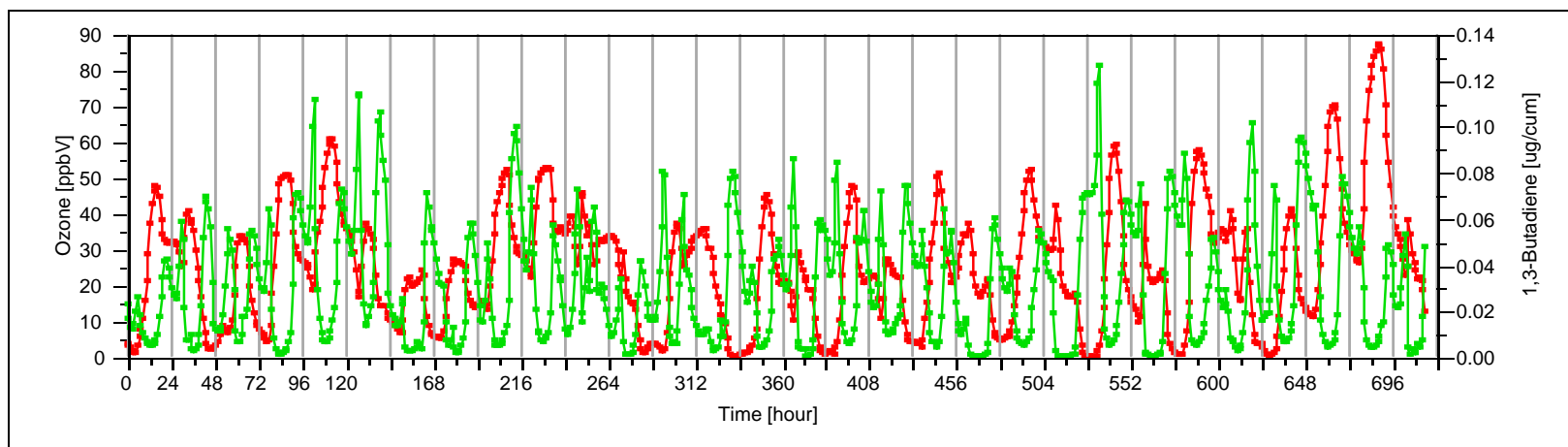
Figure B.11. Hourly Ozone and 1, 3-Butadiene Concentrations in Nashville, June 2003. (Red color: Ozone)

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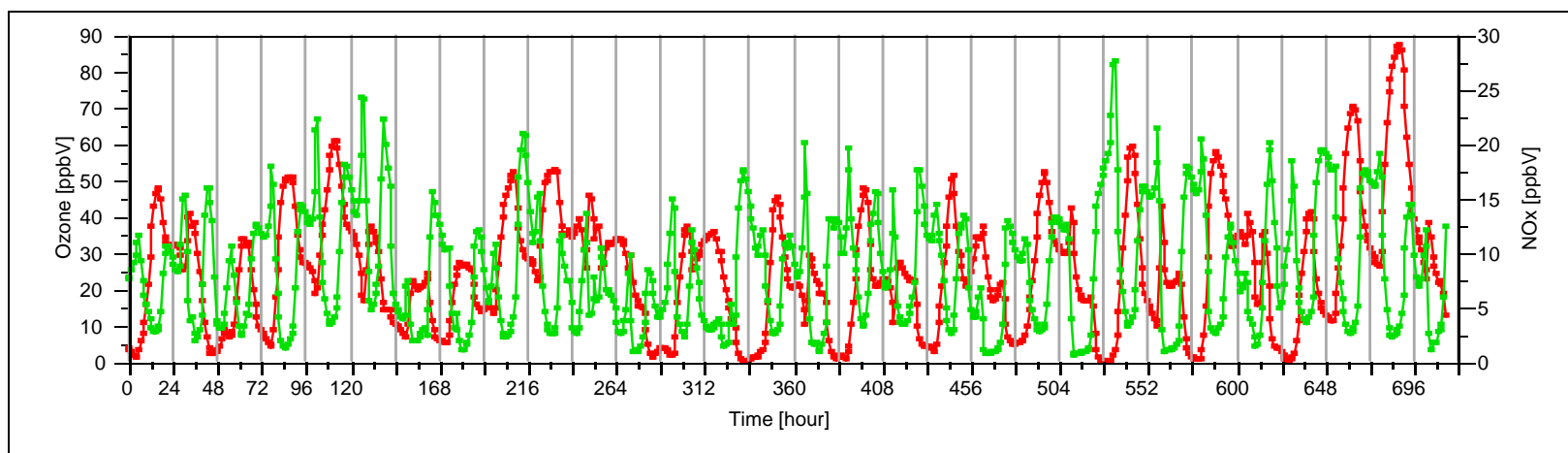
Figure B.12. Hourly Ozone and NOx Concentrations in Nashville, June 2003. (Red color: Ozone)
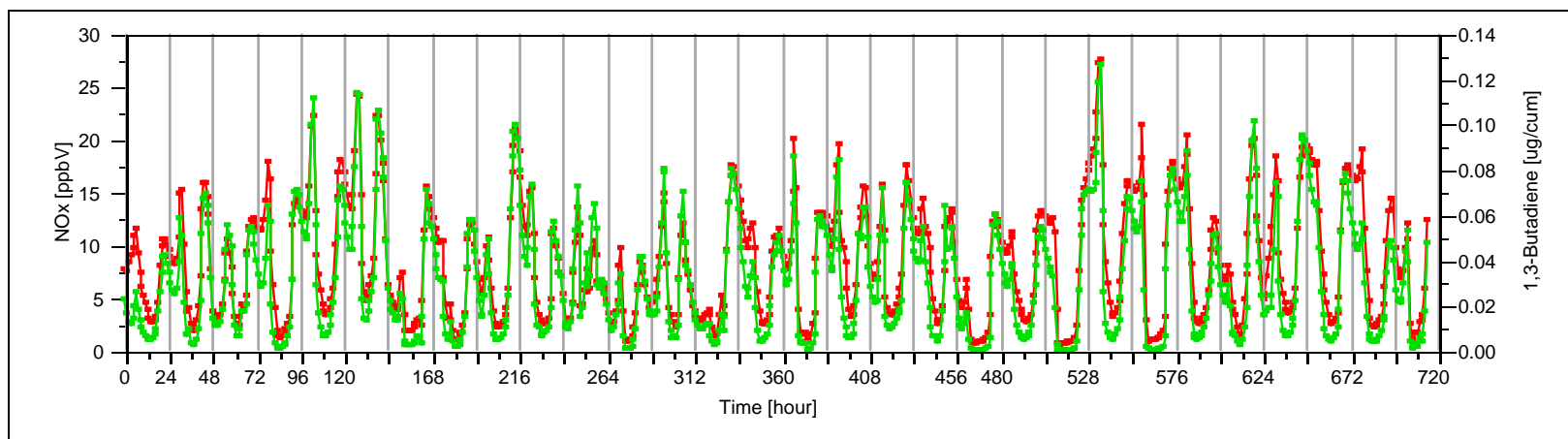
Figure B.13. Hourly NOx and 1, 3-Butadiene Concentrations in Nashville, June 2003. (Red color: NOx)

382
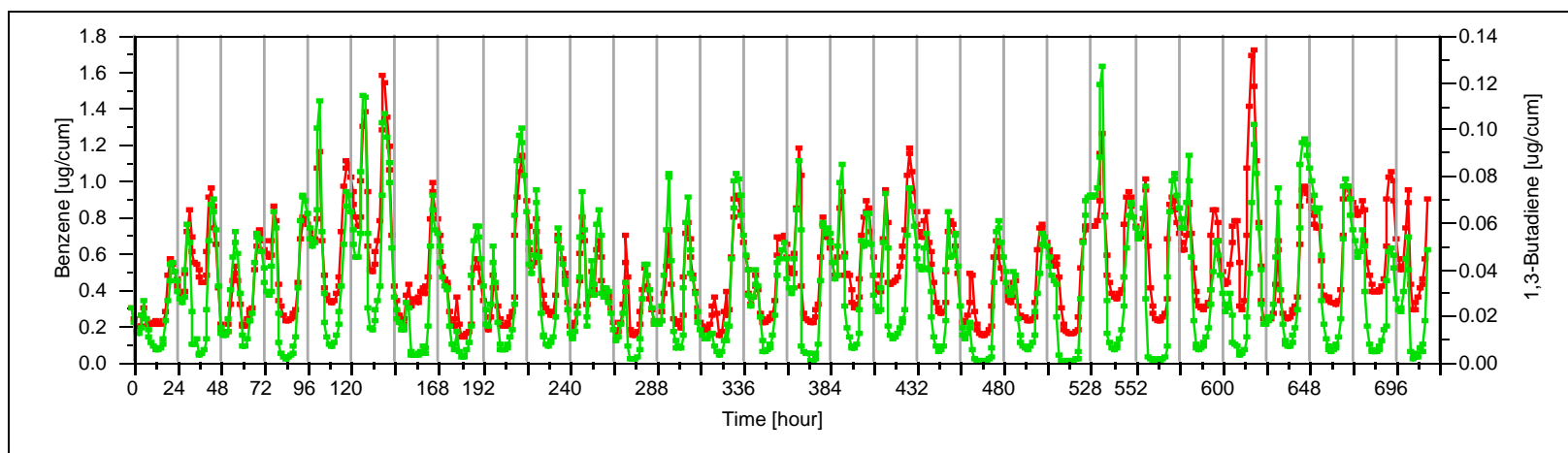
Figure B.14. Hourly Benzene and 1, 3-Butadiene Concentrations in Nashville, June 2003. (Red color: Benzene)
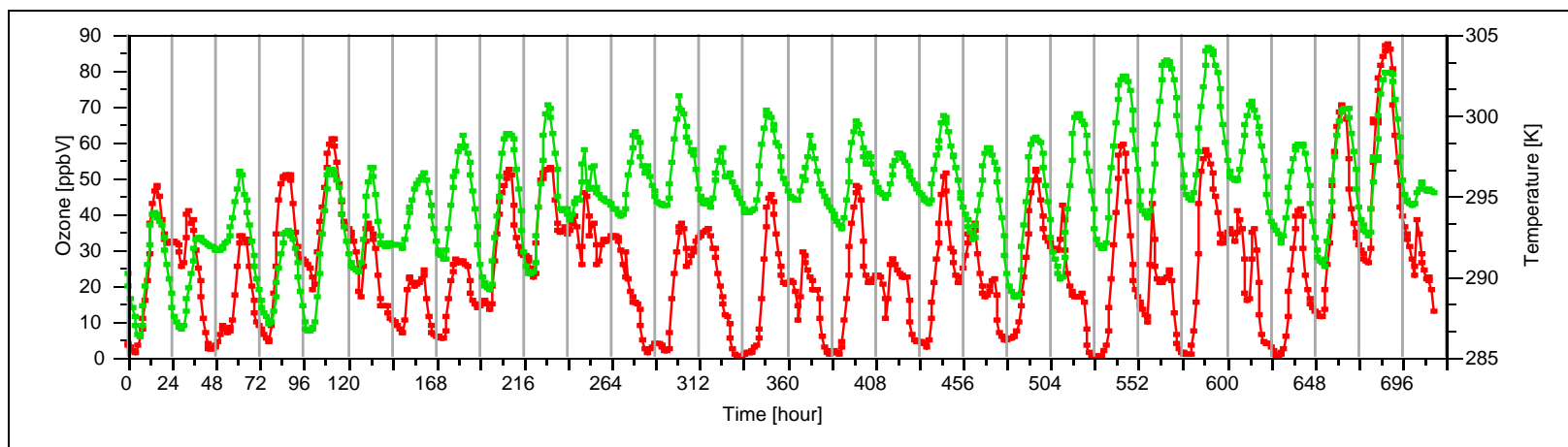
Figure B.15. Hourly Ozone Concentrations and Temperature in Nashville, June 2003. (Red color: Ozone)

383
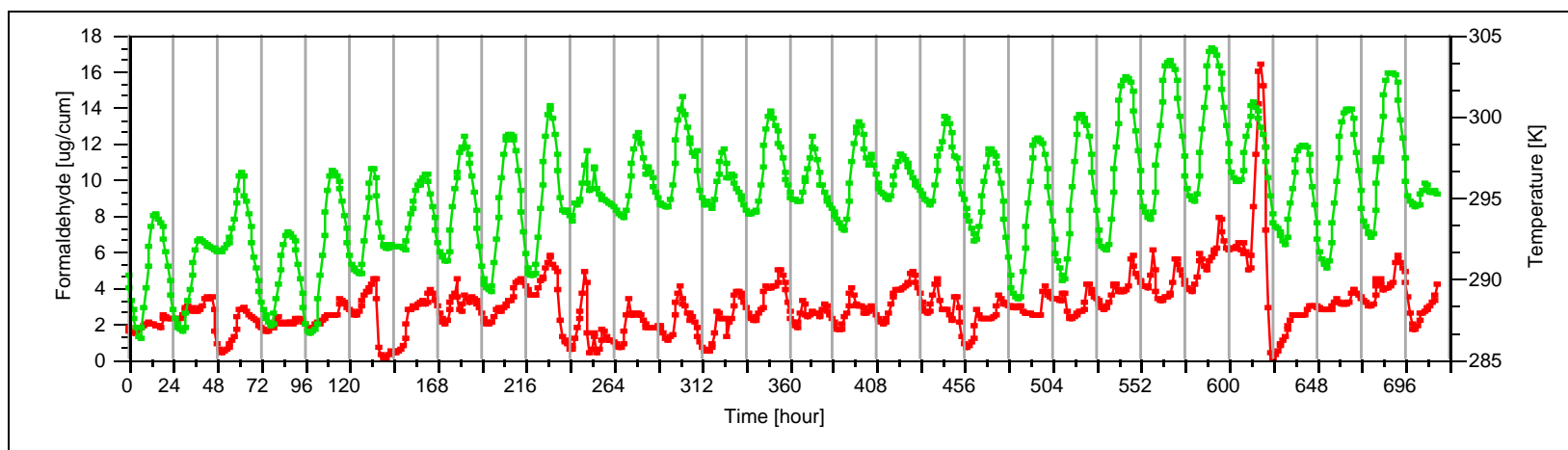
Figure B.16. Hourly Formaldehyde Concentrations and Temperature in Nashville, June 2003. (Red color: Formaldehyde)
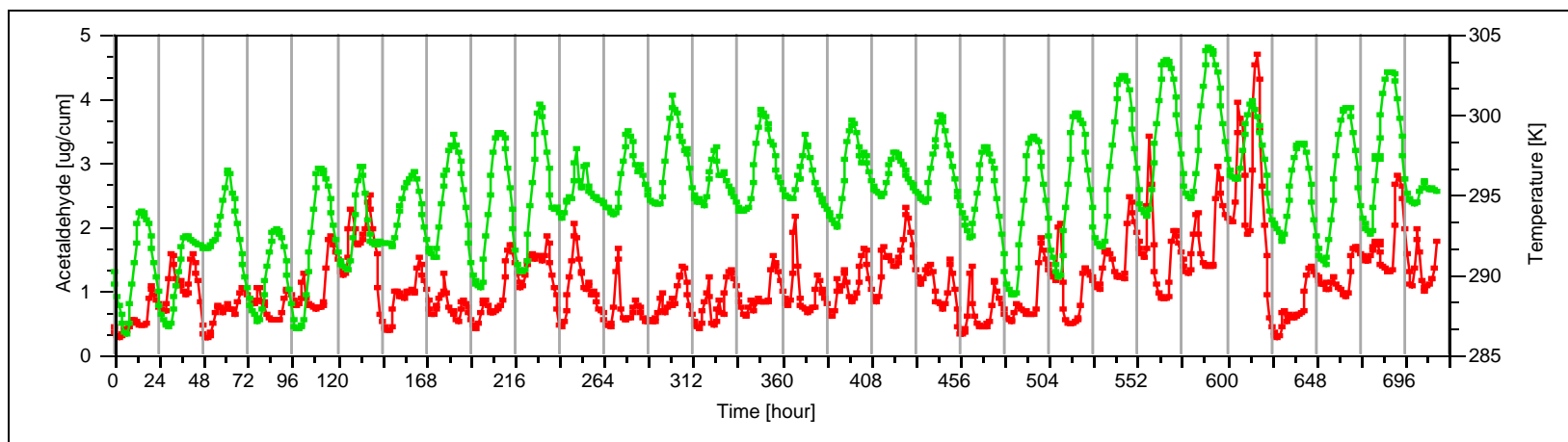
Figure B.17. Hourly Acetaldehyde Concentrations and Temperature in Nashville, June 2003. (Red color: Acetaldehyde)
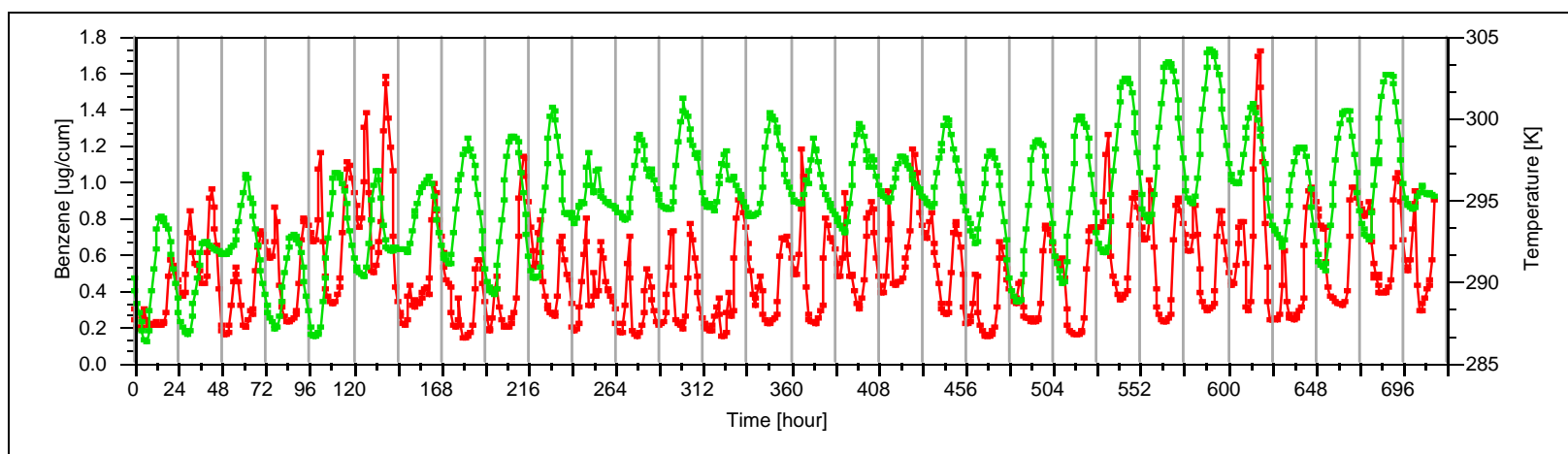
Figure B.18. Hourly Benzene Concentrations and Temperature in Nashville, June 2003. (Red color: Benzene)
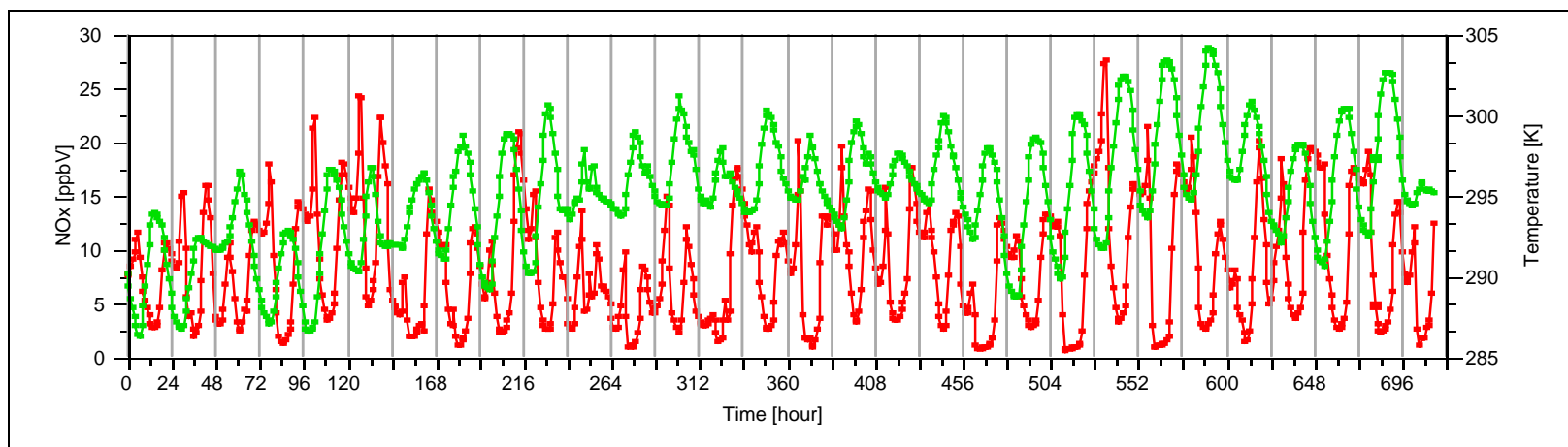
Figure B.19. Hourly NOx Concentrations and Temperature in Nashville, June 2003. (Red color: NOx)
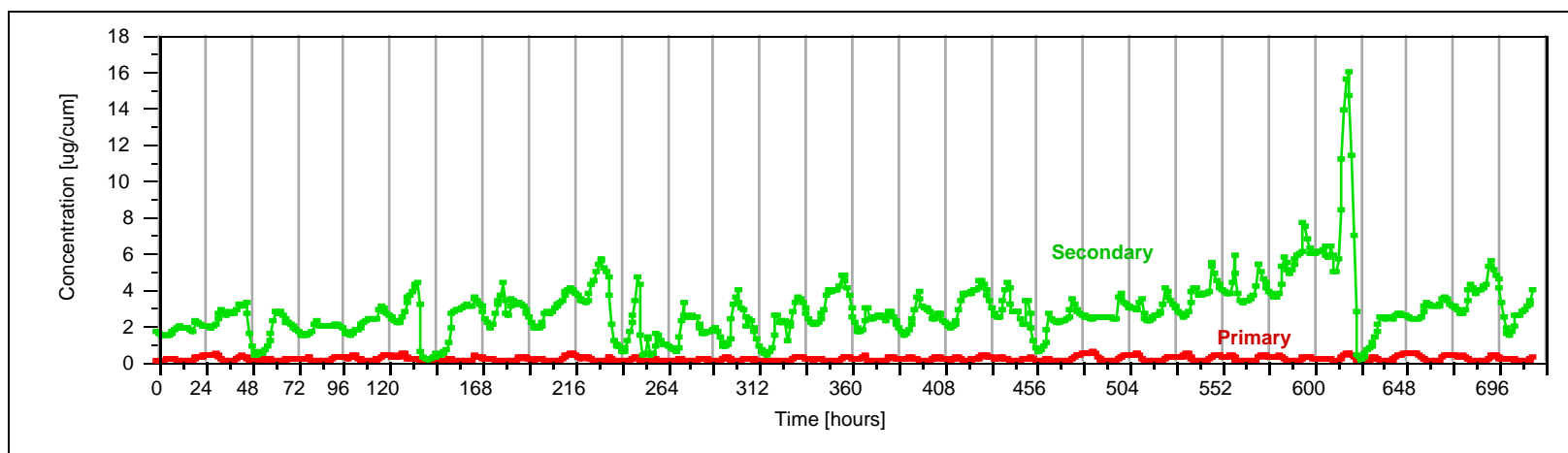
Figure B.20. Secondary and Primary Formaldehyde Daily Concentration at Nashville, TN, June 2003
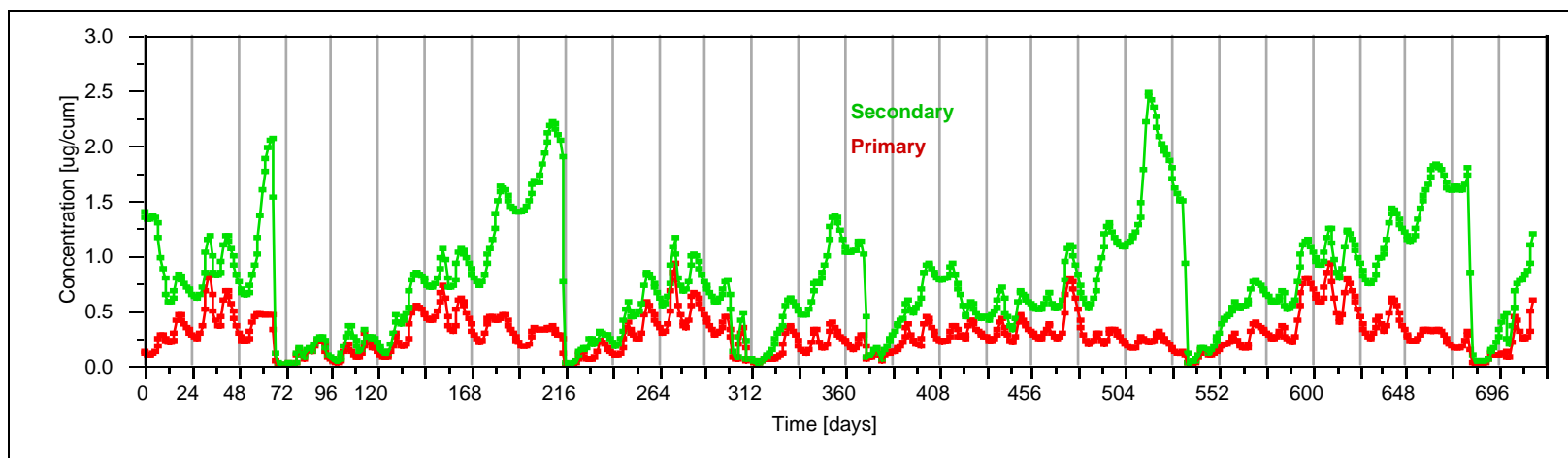
Figure B.21. Secondary and Primary Formaldehyde Daily Concentration at Nashville, TN, December 2003

386
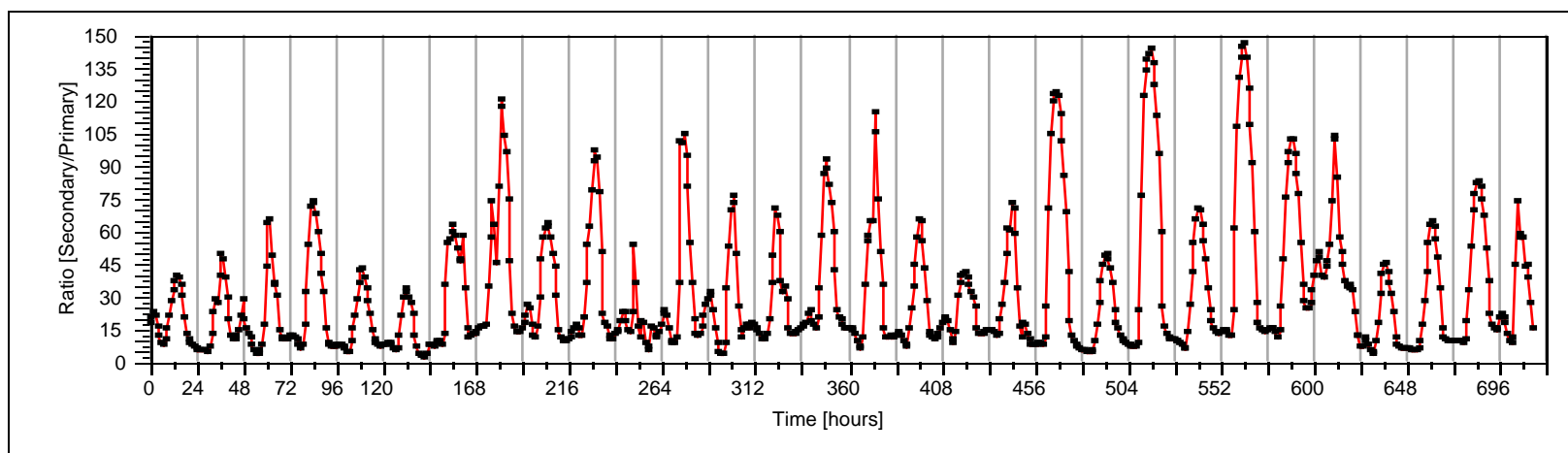
Figure B.22. Secondary to Primary Formaldehyde Ratio on Daily Concentrations at Nashville, TN, June 2003
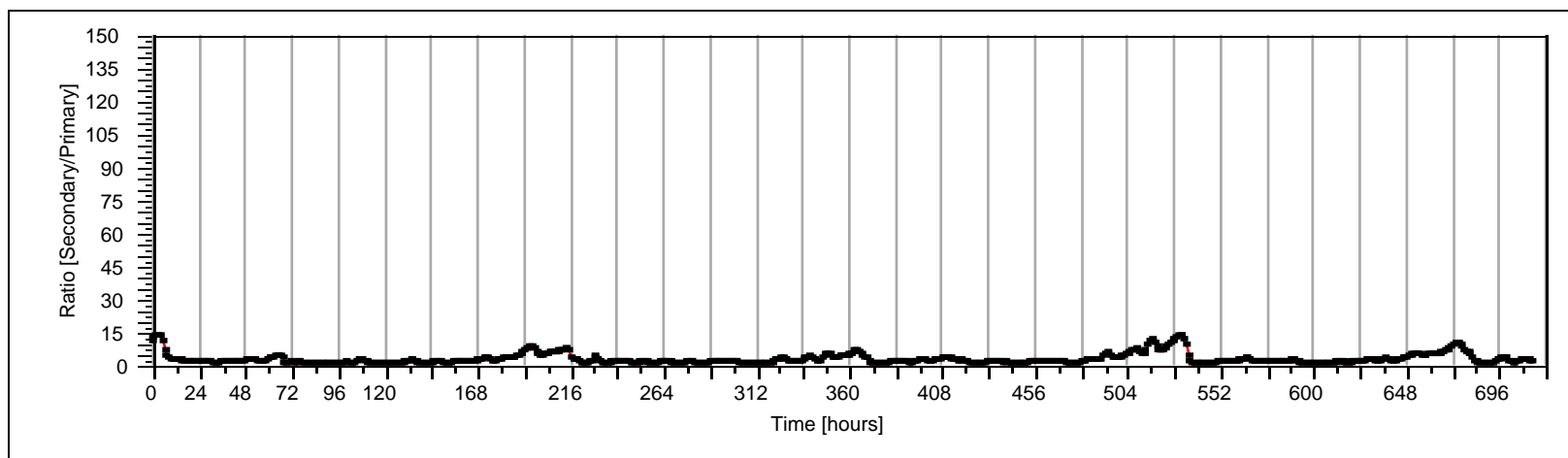
Figure B.23. Secondary to Primary Formaldehyde Ratio on Daily Concentrations at Nashville, TN, December 2003

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APPENDIX C

Monitoring HAPs Data at Nashville, Tennessee

Table C-1. EATN Monitor. Data from May 2002 to December 2002 (VOC)

SAMPLE SITE #	EATN 27501	EATN 27752	EATN 27925	EATN 28251 D1	EATN 28251 R1	EATN 28253 D2	EATN 28253 R2	EATN 28624	EATN 28715	EATN 28930	EATN 29100	EATN 29290	EATN 29674	EATN 30108	EATN 30281 D1	EATN 30281 R1	EATN 30283 D2	EATN 30283 R2	EATN 30434	EATN 30579	EATN 30826	EATN 30927	EATN 31257
SAMPLE DATE	5/14/02	5/26/02	6/7/02	6/19/02	6/19/02	6/19/02	6/19/02	7/7/02	7/13/02	7/25/02	8/6/02	8/18/02	8/30/02	10/5/02	10/17/02	10/17/02	10/17/02	10/17/02	10/29/02	11/10/02	11/22/02	12/4/02	12/16/02
ANALYSIS DATE	5/21/02	6/8/02	7/2/02	7/2/02	7/11/02	7/2/02	7/11/02	8/2/02	8/6/02	8/13/02	9/3/02	9/16/02	9/25/02	10/23/02	11/1/02	11/21/03	11/1/02	11/21/02	12/2/02	12/10/02	12/18/02	12/28/02	1/9/03
FILE NAME	L2ET025	L2FG023	N2GA020	N2GB009	N2GK006	N2GB010	N2GK007	L2HA018	L2HF009	L2HL018	L2IC015	L2IP014	L2IX015	L2JW012	L2J%016	L2KT024	L2J%017	L2KT022	L2LB009	N2LJ006	L2LR009	L2L~018	L3AH016
UNITS	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv
Acetylene	1.52	1.33	1.84	3.32	3.15	2.86	2.85	1.63	1.13	0.99	0.92	1.30	1.91	0.54	2.76	2.96	1.83	2.46	1.32	0.41	1.19	0.97	4.72
Propylene	0.58	0.81	1.16	2.19	1.77	1.71	1.30	2.02	0.57	0.68	0.58	0.78	1.00	0.55	1.62	1.88	1.01	1.32	0.68	0.30	0.03	0.75	2.16
Dichlorodifluoromethane	0.57	0.61	0.56	0.58	0.54	0.64	0.54	0.65	0.60	0.65	0.64	0.72	0.92	0.74	1.08	1.01	0.86	0.89	0.83	0.55	1.33	0.60	0.59
Chloromethane	0.54	0.62	0.70	0.64	0.55	0.67	0.51	0.69	0.56	0.62	0.59	0.76	0.92	0.51	0.72	0.86	0.54	0.59	0.55	0.58	1.05	0.52	0.50
Dichlorotetrafluoroethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.04	ND	ND	ND	ND	ND	ND	ND
Vinyl Chloride	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,3-Butadiene	ND	ND	ND	0.17	0.11	ND	0.14	0.05	0.06	0.03	0.03	0.09	0.10	ND	ND	0.31	0.13	ND	ND	ND	0.12	0.06	0.28
Bromomethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.07	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chloroethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Acetonitrile	1.50	ND	ND	ND	ND	0.79	ND	1.52	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Trichlorofluoromethane	0.26	0.26	0.24	0.25	0.29	0.25	0.22	0.30	0.29	0.30	0.28	0.31	0.41	0.35	0.46	0.46	0.38	0.40	0.36	0.27	0.60	0.30	0.30
Acrylonitrile	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

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Table C-1. Continued

SAMPLE SITE #	EATN 27501	EATN 27752	EATN 27925	EATN 28251 D1	EATN 28251 R1	EATN 28253 D2	EATN 28253 R2	EATN 28624	EATN 28715	EATN 28930	EATN 29100	EATN 29290	EATN 29674	EATN 30108	EATN 30281 D1	EATN 30281 R1	EATN 30283 D2	EATN 30283 R2	EATN 30434	EATN 30579	EATN 30826	EATN 30927	EATN 31257
SAMPLE DATE	5/14/02	5/26/02	6/7/02	6/19/02	6/19/02	6/19/02	6/19/02	7/7/02	7/13/02	7/25/02	8/6/02	8/18/02	8/30/02	10/5/02	10/17/02	10/17/02	10/17/02	10/17/02	10/29/02	11/10/02	11/22/02	12/4/02	12/16/02
ANALYSIS DATE	5/21/02	6/8/02	7/2/02	7/2/02	7/11/02	7/2/02	7/11/02	8/2/02	8/6/02	8/13/02	9/3/02	9/16/02	9/25/02	10/23/02	11/1/02	11/21/03	11/1/02	11/21/02	12/2/02	12/10/02	12/18/02	12/28/02	1/9/03
FILE NAME	L2ET025	L2FG023	N2GA020	N2GB009	N2GK006	N2GB010	N2GK007	L2HA018	L2HF009	L2HL018	L2IC015	L2IP014	L2IX015	L2JW012	L2J%016	L2KT024	L2J%017	L2KT022	L2LB009	N2LJ006	L2LR009	L2L~018	L3AH016
UNITS	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv
1,1-Dichloroethene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Methylene Chloride	0.08	0.13	ND	ND	0.05	0.16	ND	0.08	0.13	0.14	0.12	ND	0.15	ND	0.12	ND	0.10	0.27	0.16	ND	0.18	ND	0.20
Trichlorotrifluoroethane	0.20	0.24	0.19	0.16	0.15	0.15	0.13	0.30	0.14	0.20	0.19	0.19	0.23	0.14	0.16	0.18	0.10	0.18	0.09	0.10	0.25	0.14	0.17
trans - 1,2 - Dichloroethylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,1 - Dichloroethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Methyl tert-Butyl Ether	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.05	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Methyl Ethyl Ketone	ND	ND	3.28	2.63	1.32	3.01	2.24	3.33	1.13	1.55	1.42	1.01	2.26	1.08	ND	ND	ND	ND	1.69	ND	ND	1.64	0.80
Chloroprene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
cis-1,2-Dichloroethylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Bromochloromethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chloroform	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Ethyl tert-Butyl Ether	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

Table C-1. Continued

SAMPLE SITE #	EATN 27501	EATN 27752	EATN 27925	EATN 28251 D1	EATN 28251 R1	EATN 28253 D2	EATN 28253 R2	EATN 28624	EATN 28715	EATN 28930	EATN 29100	EATN 29290	EATN 29674	EATN 30108	EATN 30281 D1	EATN 30281 R1	EATN 30283 D2	EATN 30283 R2	EATN 30434	EATN 30579	EATN 30826	EATN 30927	EATN 31257
SAMPLE DATE	5/14/02	5/26/02	6/7/02	6/19/02	6/19/02	6/19/02	6/19/02	7/7/02	7/13/02	7/25/02	8/6/02	8/18/02	8/30/02	10/5/02	10/17/02	10/17/02	10/17/02	10/17/02	10/29/02	11/10/02	11/22/02	12/4/02	12/16/02
ANALYSIS DATE	5/21/02	6/8/02	7/2/02	7/2/02	7/11/02	7/2/02	7/11/02	8/2/02	8/6/02	8/13/02	9/3/02	9/16/02	9/25/02	10/23/02	11/1/02	11/21/03	11/1/02	11/21/02	12/2/02	12/10/02	12/18/02	12/28/02	1/9/03
FILE NAME	L2ET025	L2FG023	N2GA020	N2GB009	N2GK006	N2GB010	N2GK007	L2HA018	L2HF009	L2HL018	L2IC015	L2IP014	L2IX015	L2JW012	L2J%016	L2KT024	L2J%017	L2KT022	L2LB009	N2LJ006	L2LR009	L2L~018	L3AH016
UNITS	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv
1,2 - Dichloroethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,1,1 - Trichloroethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.03	ND	ND	ND	ND	ND	ND	0.03	ND	0.07	ND	ND
Benzene	0.48	0.30	0.42	0.68	0.82	0.67	0.79	0.52	0.47	0.50	0.40	0.64	0.78	0.37	1.18	1.08	0.74	0.84	0.55	0.22	0.69	0.39	1.29
Carbon Tetrachloride	0.07	0.08	ND	0.14	ND	0.16	0.14	0.10	0.08	0.07	0.06	0.12	0.12	ND	0.15	0.19	0.11	0.16	0.09	ND	0.21	0.10	0.11
tert-Amyl Methyl Ether	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,2 - Dichloropropane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Ethyl Acrylate	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Bromodichloromethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Trichloroethylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Methyl Methacrylate	ND	ND	5.65	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
cis -1,3 - Dichloropropene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Methyl Isobutyl Ketone	ND	ND	ND	0.27	ND	0.29	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

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Table C-1. Continued

SAMPLE SITE #	EATN 27501	EATN 27752	EATN 27925	EATN 28251 D1	EATN 28251 R1	EATN 28253 D2	EATN 28253 R2	EATN 28624	EATN 28715	EATN 28930	EATN 29100	EATN 29290	EATN 29674	EATN 30108	EATN 30281 D1	EATN 30281 R1	EATN 30283 D2	EATN 30283 R2	EATN 30434	EATN 30579	EATN 30826	EATN 30927	EATN 31257
SAMPLE DATE	5/14/02	5/26/02	6/7/02	6/19/02	6/19/02	6/19/02	6/19/02	7/7/02	7/13/02	7/25/02	8/6/02	8/18/02	8/30/02	10/5/02	10/17/02	10/17/02	10/17/02	10/17/02	10/29/02	11/10/02	11/22/02	12/4/02	12/16/02
ANALYSIS DATE	5/21/02	6/8/02	7/2/02	7/2/02	7/11/02	7/2/02	7/11/02	8/2/02	8/6/02	8/13/02	9/3/02	9/16/02	9/25/02	10/23/02	11/1/02	11/21/03	11/1/02	11/21/02	12/2/02	12/10/02	12/18/02	12/28/02	1/9/03
FILE NAME	L2ET025	L2FG023	N2GA020	N2GB009	N2GK006	N2GB010	N2GK007	L2HA018	L2HF009	L2HL018	L2IC015	L2IP014	L2IX015	L2JW012	L2J%016	L2KT024	L2J%017	L2KT022	L2LB009	N2LJ006	L2LR009	L2L~018	L3AH016
UNITS	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv
trans - 1,3 - Dichloropropene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.03	ND	ND	ND	ND	ND	ND	ND
1,1,2 - Trichloroethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Toluene	1.42	1.05	0.92	1.79	1.80	1.66	1.64	1.26	0.91	1.18	0.97	1.66	1.82	0.98	2.55	1.96	1.87	1.90	1.20	0.34	0.66	0.68	2.34
Dibromochloromethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,2- Dibromoethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
N-Octane	ND	0.16	ND	ND	0.13	ND	ND	0.17	0.07	0.08	ND	0.12	0.05	ND	0.19	ND	0.13	0.23	0.13	ND	ND	ND	ND
Tetrachloroethylene	0.15	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.14
Chlorobenzene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Ethylbenzene	0.21	0.12	0.13	0.23	0.29	0.24	0.24	0.26	0.15	0.17	0.18	0.29	0.28	0.12	0.38	0.47	0.35	0.42	0.24	ND	0.19	ND	0.38
m,p - Xylene	0.51	0.34	0.33	0.66	0.65	0.68	0.74	0.71	0.38	0.45	0.46	0.70	0.72	0.31	1.21	0.99	0.90	0.93	0.57	0.15	0.48	0.24	1.11
Bromoform	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Styrene	0.19	ND	ND	ND	0.08	ND	ND	0.05	ND	0.13	ND	0.16	ND	ND	1.31	1.26	1.19	1.09	0.74	ND	ND	ND	0.71

Table C-1. Continued

SAMPLE SITE #	EATN 27501	EATN 27752	EATN 27925	EATN 28251 D1	EATN 28251 R1	EATN 28253 D2	EATN 28253 R2	EATN 28624	EATN 28715	EATN 28930	EATN 29100	EATN 29290	EATN 29674	EATN 30108	EATN 30281 D1	EATN 30281 R1	EATN 30283 D2	EATN 30283 R2	EATN 30434	EATN 30579	EATN 30826	EATN 30927	EATN 31257
SAMPLE DATE	5/14/02	5/26/02	6/7/02	6/19/02	6/19/02	6/19/02	6/19/02	7/7/02	7/13/02	7/25/02	8/6/02	8/18/02	8/30/02	10/5/02	10/17/02	10/17/02	10/17/02	10/17/02	10/29/02	11/10/02	11/22/02	12/4/02	12/16/02
ANALYSIS DATE	5/21/02	6/8/02	7/2/02	7/2/02	7/11/02	7/2/02	7/11/02	8/2/02	8/6/02	8/13/02	9/3/02	9/16/02	9/25/02	10/23/02	11/1/02	11/21/03	11/1/02	11/21/02	12/2/02	12/10/02	12/18/02	12/28/02	1/9/03
FILE NAME	L2ET025	L2FG023	N2GA020	N2GB009	N2GK006	N2GB010	N2GK007	L2HA018	L2HF009	L2HL018	L2IC015	L2IP014	L2IX015	L2JW012	L2J%016	L2KT024	L2J%017	L2KT022	L2LB009	N2LJ006	L2LR009	L2L~018	L3AH016
UNITS	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv
1,1,2,2 - Tetrachloroethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
o - Xylene	0.23	0.16	0.17	0.32	0.35	0.36	0.28	0.34	0.20	0.23	0.22	0.35	0.39	0.16	0.52	0.57	0.42	0.51	0.22	ND	0.22	0.10	0.50
1,3,5-Trimethylbenzene	ND	ND	ND	0.11	0.07	0.09	0.09	0.11	0.07	ND	0.06	0.11	0.06	0.06	0.21	0.28	0.14	0.17	0.06	ND	0.07	ND	0.15
1,2,4-Trimethylbenzene	0.19	0.14	0.11	0.31	0.29	0.30	0.25	0.33	0.18	0.21	0.18	0.30	0.28	0.11	0.59	0.52	0.43	0.38	0.13	ND	0.18	0.07	0.50
m - Dichlorobenzene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chloromethylbenzene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
p - Dichlorobenzene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
o - Dichlorobenzene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,2,4-Trichlorobenzene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Hexachloro-1,3-Butadiene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

Table C-2. EATN Monitor. Data from May 2002 to December 2002 (Carb.)

SAMPLE DATE	5/14/02	5/26/02	6/7/02	6/19/02	6/19/02	6/19/02	6/19/02	7/7/02	7/13/02	7/25/02	8/6/02	8/18/02	8/30/02	9/11/02	10/5/02	10/17/02	10/17/02	10/17/02	10/17/02	10/29/02	11/10/02	11/22/02	12/4/02	12/16/02
UNITS	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv
Formaldehyde	1.62	2.33	2.61	4.92	4.92	5.03	5.04	5.50	2.11	3.84	4.76	3.44	3.23	6.27	2.16	2.02	2.02	2.03	2.03	1.16	1.73	1.78	0.95	3.73
Acetaldehyde	0.83	1.05	1.03	1.63	1.63	1.69	1.72	1.52	0.81	0.87	1.12	0.81	1.23	2.24	0.86	0.78	0.78	0.78	0.78	0.62	0.77	0.57	0.71	1.38
Acetone	1.60	0.53	0.66	0.75	0.74	1.00	0.99	0.68	0.42	0.37	0.62	0.34	0.42	0.82	0.71	2.01	2.04	2.65	2.71	0.60	0.43	0.85	0.90	1.51
Propionaldehyde	0.15	0.19	0.15	0.35	0.34	0.36	0.36	0.26	0.14	0.19	0.23	0.15	0.25	0.43	0.09	0.08	0.06	0.07	0.09	0.06	0.12	0.05	0.07	0.16
Crotonaldehyde	0.01	0.01	0.01	0.01	0.01	0.02	0.02	0.01	ND	0.01	0.01	0.01	0.01	0.02	0.005	0.01	0.01	0.01	0.01	0.004	0.003	0.005	0.01	0.02
Butyr/Isobutyraldehyde	0.23	0.18	0.20	0.15	0.14	0.16	0.16	0.12	0.14	0.13	0.14	0.14	0.16	0.26	0.10	0.13	0.13	0.14	0.13	0.08	0.09	0.09	0.09	0.17
Benzaldehyde	0.03	0.05	0.05	0.09	0.08	0.08	0.08	0.07	0.04	0.05	0.06	0.06	0.04	0.14	0.02	0.06	0.06	0.06	0.06	0.04	0.03	0.02	0.02	0.07
Isovaleraldehyde	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Valeraldehyde	0.05	0.06	0.04	0.06	0.06	0.06	0.06	0.05	0.03	0.03	0.04	0.03	0.04	0.08	0.03	0.02	0.03	0.03	0.03	0.02	0.02	0.02	0.02	0.04
Tolualdehydes	0.07	0.04	0.02	0.06	0.06	0.09	0.06	0.08	0.03	0.05	0.07	0.09	0.04	0.10	0.04	0.06	0.06	0.06	0.06	0.02	0.01	0.02	0.01	0.05
Hexaldehyde	0.05	0.06	0.03	0.08	0.09	0.12	0.08	0.06	0.05	0.06	0.06	0.06	0.05	0.08	0.03	0.04	0.04	0.04	0.03	0.02	0.02	0.02	0.03	0.04
2,5-Dimethylbenzaldehyde	ND	ND	ND	ND	ND	0.03	ND	0.04	ND	ND	0.02	ND	0.01	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

Table C-3. EATN Monitor. Data from January 2003 to June 2003 (VOC)

SAMPLE SITE #		EATN 31602	EATN 31928	EATN 32024	EATN 32215	EATN 32393	EATN 32560 D1	EATN 32560 R1	EATN 32563 D2	EATN 32563 R2	EATN 32646	EATN 32878	EATN 33107	EATN 33295 D1	EATN 33295 R1	EATN 33299 D2	EATN 33299 R2	EATN 33440	EATN 33562
SAMPLE DATE		1/15/2003	2/8/2003	2/20/2003	3/4/2003	3/16/2003	3/28/2003	3/28/2003	3/28/2003	3/28/2003	4/9/2003	4/21/2003	5/3/2003	5/15/2003	5/15/2003	5/15/2003	5/15/2003	5/27/2003	6/8/2003
ANALYSIS DATE		2/5/2003	2/27/2003	3/12/2003	3/21/2003	3/26/2003	4/15/2003	4/15/2003	4/15/2003	4/15/2003	4/29/2003	5/1/2003	5/24/2003	6/5/2003	6/6/2003	6/5/2003	6/9/2003	6/19/2003	6/27/2003
FILE NAME		N3BD022	N3B~017	N3CK019	L3CT016	N3CY018	L3DN015	L3DO010	L3DN014	L3DO009	N3D#010	N3D$011	L3EW016	N3FE011	N3FF011	N3FE012	N3FI006	N3FS008	L3F~010
UNITS	MDL	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv
Acetylene	0.08	3.13	3.63	1.28	2.22	3.24	0.80	0.70	0.82	0.71	1.51	0.87	1.68	0.96	1.19	1.13	1.35	1.93	1.08
Propylene	0.11	1.57	0.81	0.58	1.14	1.60	0.54	0.54	0.45	0.40	0.42	0.28	0.67	0.46	0.57	0.58	0.61	0.93	0.55
Dichlorodifluoromethane	0.15	0.51	0.46	0.44	0.64	0.80	0.70	0.76	0.69	0.80	0.62	0.68	0.57	0.54	0.70	0.64	0.78	0.76	0.37
Chloromethane	0.10	0.46	0.47	0.43	0.55	0.79	0.70	0.71	0.45	0.80	0.61	0.65	0.59	0.58	0.66	0.69	0.68	0.64	0.28
Dichlorotetrafluoroethane	0.11	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Vinyl Chloride	0.13	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,3-Butadiene	0.12	0.13	0.09	ND	0.08	0.14	ND	ND	ND	ND	0.02	ND	ND	0.06	0.06	0.07	0.07	0.11	ND
Bromomethane	0.13	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chloroethane	0.13	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Acetonitrile	0.43	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.87	ND	ND	ND	ND	ND	ND	ND	ND
Trichlorofluoromethane	0.07	0.25	0.25	0.18	0.30	0.30	0.37	0.39	0.33	0.40	0.28	0.32	0.25	0.22	0.29	0.27	0.34	0.27	0.17
Acrylonitrile	0.37	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,1-Dichloroethene	0.11	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Methylene Chloride	0.09	0.04	0.04	ND	0.09	0.08	ND	0.09	ND	ND	0.10	0.10	ND	0.06	0.07	0.06	0.08	0.13	ND
Trichlorotrifluoroethane	0.10	0.12	0.09	0.06	ND	0.09	0.12	0.14	0.11	0.09	0.12	0.15	0.11	0.12	0.14	0.18	0.18	0.11	0.03
trans - 1,2 - Dichloroethylene	0.12	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,1 - Dichloroethane	0.06	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Methyl tert-Butyl Ether	0.16	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.14	ND
Methyl Ethyl Ketone	0.25	2.42	ND	1.49	ND	ND	1.34	1.34	1.39	1.27	0.76	0.23	0.59	ND	ND	ND	ND	ND	ND
Chloroprene	0.07	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

Table C-3. Continued

SAMPLE SITE #		EATN 31602	EATN 31928	EATN 32024	EATN 32215	EATN 32393	EATN 32560 D1	EATN 32560 R1	EATN 32563 D2	EATN 32563 R2	EATN 32646	EATN 32878	EATN 33107	EATN 33295 D1	EATN 33295 R1	EATN 33299 D2	EATN 33299 R2	EATN 33440	EATN 33562
SAMPLE DATE		1/15/2003	2/8/2003	2/20/2003	3/4/2003	3/16/2003	3/28/2003	3/28/2003	3/28/2003	3/28/2003	4/9/2003	4/21/2003	5/3/2003	5/15/2003	5/15/2003	5/15/2003	5/15/2003	5/27/2003	6/8/2003
ANALYSIS DATE		2/5/2003	2/27/2003	3/12/2003	3/21/2003	3/26/2003	4/15/2003	4/15/2003	4/15/2003	4/15/2003	4/29/2003	5/1/2003	5/24/2003	6/5/2003	6/6/2003	6/5/2003	6/9/2003	6/19/2003	6/27/2003
FILE NAME		N3BD022	N3B~017	N3CK019	L3CT016	N3CY018	L3DN015	L3DO010	L3DN014	L3DO009	N3D#010	N3D$011	L3EW016	N3FE011	N3FF011	N3FE012	N3FI006	N3FS008	L3F~010
UNITS	MDL	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv
cis-1,2-Dichloroethylene	0.14	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Bromochloromethane	0.21	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chloroform	0.09	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Ethyl tert-Butyl Ether	0.14	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,2 - Dichloroethane	0.11	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,1,1 - Trichloroethane	0.10	ND	ND	ND	ND	ND	0.03	ND	ND	ND	0.03	0.03	0.02	0.03	0.02	0.03	0.03	0.05	ND
Benzene	0.06	0.75	0.65	0.39	0.70	1.06	0.28	0.30	0.27	0.28	0.33	0.23	0.34	0.29	0.32	0.35	0.32	0.43	0.39
Carbon Tetrachloride	0.08	0.09	0.09	0.04	0.11	0.06	0.09	0.14	0.15	0.12	0.09	0.10	0.06	0.08	0.11	0.10	0.12	0.07	0.05
tert-Amyl Methyl Ether	0.16	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,2 - Dichloropropane	0.07	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Ethyl Acrylate	0.27	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Bromodichloromethane	0.09	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Trichloroethylene	0.17	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Methyl Methacrylate	0.19	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
cis -1,3 - Dichloropropene	0.10	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Methyl Isobutyl Ketone	0.25	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.06	ND	ND	ND	ND
trans - 1,3 - Dichloropropene	0.13	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,1,2 - Trichloroethane	0.09	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Toluene	0.06	1.24	1.03	0.56	1.35	1.81	1.19	1.06	1.36	1.08	0.97	0.42	0.71	0.67	0.71	0.83	0.68	1.10	0.90
Dibromochloromethane	0.07	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

Table C-3. Continued

SAMPLE SITE #		EATN 31602	EATN 31928	EATN 32024	EATN 32215	EATN 32393	EATN 32560 D1	EATN 32560 R1	EATN 32563 D2	EATN 32563 R2	EATN 32646	EATN 32878	EATN 33107	EATN 33295 D1	EATN 33295 R1	EATN 33299 D2	EATN 33299 R2	EATN 33440	EATN 33562
SAMPLE DATE		1/15/2003	2/8/2003	2/20/2003	3/4/2003	3/16/2003	3/28/2003	3/28/2003	3/28/2003	3/28/2003	4/9/2003	4/21/2003	5/3/2003	5/15/2003	5/15/2003	5/15/2003	5/15/2003	5/27/2003	6/8/2003
ANALYSIS DATE		2/5/2003	2/27/2003	3/12/2003	3/21/2003	3/26/2003	4/15/2003	4/15/2003	4/15/2003	4/15/2003	4/29/2003	5/1/2003	5/24/2003	6/5/2003	6/6/2003	6/5/2003	6/9/2003	6/19/2003	6/27/2003
FILE NAME		N3BD022	N3B~017	N3CK019	L3CT016	N3CY018	L3DN015	L3DO010	L3DN014	L3DO009	N3D#010	N3D$011	L3EW016	N3FE011	N3FF011	N3FE012	N3FI006	N3FS008	L3F~010
UNITS	MDL	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv
1,2-Dibromoethane	0.07	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
N-Octane	0.12	0.08	ND	ND	0.09	ND	ND	ND	ND	ND	0.08	ND	ND	0.04	ND	0.04	ND	ND	ND
Tetrachloroethylene	0.10	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.12	ND	ND	0.03	0.02	0.03	0.04	0.07	0.05
Chlorobenzene	0.10	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Ethylbenzene	0.08	0.23	0.18	0.09	0.22	0.33	ND	ND	ND	ND	0.08	0.05	0.10	0.12	0.12	0.14	0.12	0.16	0.20
m,p - Xylene	0.08	0.53	0.46	0.28	0.62	0.91	0.40	0.49	0.42	0.41	0.24	0.14	0.22	0.37	0.42	0.42	0.39	0.45	0.44
Bromoform	0.07	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Styrene	0.08	0.10	ND	ND	ND	0.04	ND	ND	ND	ND	0.02	0.34	ND	0.70	0.71	0.78	0.65	0.59	0.16
1,1,2,2 - Tetrachloroethane	0.09	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
o - Xylene	0.06	0.32	0.23	0.15	0.36	0.40	ND	0.15	0.17	ND	0.08	0.07	0.13	0.13	0.14	0.13	0.12	0.19	0.20
1,3,5-Trimethylbenzene	0.05	0.09	0.05	ND	0.10	ND	ND	ND	ND	ND	0.03	ND	ND	0.06	0.05	0.06	0.05	0.07	0.08
1,2,4-Trimethylbenzene	0.09	0.25	0.20	0.12	0.33	0.23	0.12	ND	0.14	0.14	0.09	0.07	0.12	0.13	0.15	0.18	0.14	0.17	0.21
m - Dichlorobenzene	0.12	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chloromethylbenzene	0.09	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
p - Dichlorobenzene	0.15	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.02	ND	ND	0.03	ND
o - Dichlorobenzene	0.12	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,2,4-Trichlorobenzene	0.16	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Hexachloro-1,3-Butadiene	0.20	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

Table C-4. EATN Monitor. Data from July 2003 to December 2003 (VOC)

SAMPLE SITE #		EATN 33902	EATN 34710	EATN 34885	EATN 35191	EATN 35778 D1	EATN 35778 R1	EATN 35780 D2	EATN 35780 R2	EATN 35991	EATN 36153 D1	EATN 36153 R1	EATN 36155 D2	EATN 36155 R2	EATN 36429	EATN 36633	EATN 36885	EATN 37216	EATN 37358
SAMPLE DATE		6/20/2003	7/26/2003	8/9/2003	8/19/2003	9/12/2003	9/12/2003	9/12/2003	9/12/2003	9/24/2003	10/6/2003	10/6/2003	10/6/2003	10/6/2003	10/30/2003	11/11/2003	11/23/2003	12/17/2003	12/29/2003
ANALYSIS DATE		7/12/2003	8/26/2003	9/11/2003	9/17/2003	10/8/2003	10/8/2003	10/8/2003	10/8/2003	10/9/2003	11/5/2003	11/6/2003	11/5/2003	11/6/2003	11/18/2003	12/2/2003	12/13/003	1/7/2004	1/14/2004
FILE NAME		N3GK020	L3HZ013	L3IJ014	N3IP018	N3JG020	N3JH008	N3JG021	N3JH009	L3JI008	L3KE008	L3KF005	L3KE009	L3KF006	n3kr007	L3LA029	L3LL020	L4AG011	N4AM016
UNITS	MDL	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv
Acetylene	0.08	0.52	1.89	0.28	0.96	1.74	1.84	1.61	1.69	ND	2.61	2.57	2.41	2.69	1.10	1.17	0.84	1.53	1.02
Propylene	0.11	0.33	1.08	0.63	0.48	0.78	0.84	0.75	0.79	1.08	1.28	1.17	1.20	1.20	0.54	0.47	0.37	0.67	0.51
Dichlorodifluoromethane	0.15	0.54	0.60	0.58	0.68	0.82	0.86	0.81	0.82	0.61	0.62	0.60	0.61	ND	0.53	0.57	0.62	0.57	0.62
Chloromethane	0.10	0.61	0.64	0.61	0.59	0.54	0.54	0.52	0.54	0.59	0.67	0.64	0.59	ND	0.54	0.55	0.53	0.51	0.66
Dichlorotetrafluoroethane	0.11	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Vinyl Chloride	0.13	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,3-Butadiene	0.12	ND	ND	0.08	0.03	0.10	0.10	0.10	0.10	0.15	0.17	0.13	0.15	0.14	0.08	ND	ND	ND	0.04
Bromomethane	0.13	ND	ND	ND	ND	0.02	ND	ND	ND	0.02	ND	ND	0.16	0.15	0.01	ND	ND	ND	ND
Chloroethane	0.13	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Acetonitrile	0.43	7.41	7.95	4.86	8.78	0.53	0.55	0.65	0.65	ND	5.65	5.17	7.05	7.46	0.46	ND	3.17	ND	4.02
Trichlorofluoromethane	0.07	0.27	0.29	0.30	0.38	0.37	0.39	0.36	0.38	0.29	0.27	0.27	0.27	0.28	0.27	0.21	0.28	0.26	0.26
Acrylonitrile	0.37	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,1-Dichloroethene	0.11	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Methylene Chloride	0.09	0.18	0.18	0.12	0.14	0.18	0.19	0.17	0.17	0.18	0.17	ND	0.14	ND	0.08	ND	ND	ND	ND
Trichlorotrifluoroethane	0.10	0.17	0.14	0.13	0.19	0.09	0.10	0.08	0.10	0.11	0.11	0.11	0.15	0.15	0.08	0.06	0.10	ND	0.09
trans - 1,2 - Dichloroethylene	0.12	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,1 - Dichloroethane	0.06	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Methyl tert-Butyl Ether	0.16	ND	ND	0.12	0.08	0.16	0.14	0.16	0.15	0.14	0.14	0.14	0.13	0.14	ND	ND	ND	ND	ND
Methyl Ethyl Ketone	0.25	ND	0.51	1.36	1.28	0.95	1.03	0.91	0.88	0.48	0.47	0.39	0.49	0.49	0.43	0.64	1.01	ND	ND
Chloroprene	0.07	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
cis-1,2-Dichloroethylene	0.14	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Bromochloromethane	0.21	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chloroform	0.09	ND	ND	0.03	0.04	0.03	0.03	0.03	0.03	ND	ND	ND	ND	0.03	0.02	ND	ND	ND	ND
Ethyl tert-Butyl Ether	0.14	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,2 - Dichloroethane	0.11	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,1,1 - Trichloroethane	0.10	0.03	0.03	0.03	0.03	0.04	0.03	0.03	0.04	ND	ND	ND	ND	ND	0.03	ND	ND	ND	ND
Benzene	0.06	0.16	0.84	0.45	0.31	0.67	0.70	0.68	0.69	0.67	0.69	0.68	0.66	0.68	0.42	0.36	0.28	0.39	0.36
Carbon Tetrachloride	0.08	0.10	0.10	0.09	0.10	0.12	0.12	0.11	0.11	0.09	0.08	0.08	0.07	0.07	0.10	ND	0.05	0.04	0.09
tert-Amyl Methyl Ether	0.16	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,2 - Dichloropropane	0.07	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Ethyl Acrylate	0.27	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

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Table C-4. Continued

SAMPLE SITE #		EATN 33902	EATN 34710	EATN 34885	EATN 35191	EATN 35778 D1	EATN 35778 R1	EATN 35780 D2	EATN 35780 R2	EATN 35991	EATN 36153 D1	EATN 36153 R1	EATN 36155 D2	EATN 36155 R2	EATN 36429	EATN 36633	EATN 36885	EATN 37216	EATN 37358
SAMPLE DATE		6/20/2003	7/26/2003	8/9/2003	8/19/2003	9/12/2003	9/12/2003	9/12/2003	9/12/2003	9/24/2003	10/6/2003	10/6/2003	10/6/2003	10/6/2003	10/30/2003	11/11/2003	11/23/2003	12/17/2003	12/29/2003
ANALYSIS DATE		7/12/2003	8/26/2003	9/11/2003	9/17/2003	10/8/2003	10/8/2003	10/8/2003	10/8/2003	10/9/2003	11/5/2003	11/6/2003	11/5/2003	11/6/2003	11/18/2003	12/2/2003	12/13/003	1/7/2004	1/14/2004
FILE NAME		N3GK020	L3HZ013	L3IJ014	N3IP018	N3JG020	N3JH008	N3JG021	N3JH009	L3JI008	L3KE008	L3KF005	L3KE009	L3KF006	n3kr007	L3LA029	L3LL020	L4AG011	N4AM016
UNITS	MDL	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv
Bromodichloromethane	0.09	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Trichloroethylene	0.17	ND	ND	ND	ND	0.02	0.01	0.01	0.02	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.02
Methyl Methacrylate	0.19	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
cis -1,3 - Dichloropropene	0.10	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Methyl Isobutyl Ketone	0.25	ND	ND	ND	ND	0.09	0.11	ND	0.08	0.24	ND	ND	ND	ND	0.05	ND	ND	ND	ND
trans - 1,3 - Dichloropropene	0.13	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,1,2 - Trichloroethane	0.09	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Toluene	0.06	0.39	2.61	1.04	0.76	1.36	1.37	1.37	1.41	1.80	1.57	1.52	1.57	1.42	0.87	0.60	0.35	0.68	0.50
Dibromochloromethane	0.07	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,2-Dibromoethane	0.07	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
N-Octane	0.12	ND	ND	0.04	ND	ND	ND	ND	ND	ND	0.07	ND	0.07	0.07	0.02	ND	ND	ND	0.05
Tetrachloroethylene	0.10	ND	ND	ND	ND	0.05	0.05	0.05	0.05	ND	0.28	0.24	0.06	0.03	0.05	ND	ND	ND	0.05
Chlorobenzene	0.10	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Ethylbenzene	0.08	0.07	0.34	0.20	0.15	0.22	0.22	0.23	0.23	0.28	0.22	0.24	0.22	0.23	0.09	ND	0.05	ND	0.11
m,p - Xylene	0.08	0.20	0.82	0.44	0.36	0.69	0.69	0.69	0.71	0.79	0.68	0.67	0.65	0.65	0.30	0.21	0.15	0.32	0.28
Bromoform	0.07	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Styrene	0.08	0.06	ND	0.10	0.10	0.05	0.05	0.05	0.05	0.31	0.07	0.08	0.09	ND	0.05	ND	ND	0.51	0.07
1,1,2,2 - Tetrachloroethane	0.09	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
o - Xylene	0.06	0.07	0.38	0.24	0.15	0.33	0.33	0.33	0.33	0.39	0.34	0.32	0.33	0.32	0.17	0.12	ND	0.13	0.13
1,3,5-Trimethylbenzene	0.05	0.05	0.12	0.07	0.03	0.08	0.08	0.08	0.08	0.15	0.11	0.13	0.11	0.12	0.03	ND	ND	ND	0.05
1,2,4-Trimethylbenzene	0.09	0.07	0.35	0.17	0.11	0.29	0.30	0.30	0.30	0.41	0.36	0.38	0.36	0.33	0.12	0.10	0.04	0.13	0.13
m - Dichlorobenzene	0.12	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chloromethylbenzene	0.09	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
p - Dichlorobenzene	0.15	ND	ND	ND	ND	0.04	0.04	0.04	0.04	ND	ND	ND	ND	ND	0.02	ND	ND	ND	ND
o - Dichlorobenzene	0.12	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,2,4-Trichlorobenzene	0.16	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Hexachloro-1,3-Butadiene	0.20	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

Table C-5. EATN Monitor. Data from January 2003 to June 2003 (Carb.)

SAMPLE #	31604	31929	32026	32216	32395	32561 D1	32561 R1	32564 D2	32564 R2	32647	32880	33108	33296 D1	33296 R1	33300 D2	33300 R2	33442	33904
SAMPLE DATE	1/15/03	2/8/03	2/20/03	3/4/03	3/16/03	3/28/03	3/28/03	3/28/03	3/28/03	4/9/03	4/21/03	5/3/03	5/15/03	5/15/03	5/15/03	5/15/03	5/27/03	6/20/03
ANALYSIS DATE	2/13/03	3/11/03	3/13/03	4/7/03	4/25/03	5/10/03	5/10/03	5/10/03	5/10/03	5/15/03	5/30/03	6/3/03	6/25/03	6/25/03	6/25/03	6/25/03	7/8/03	7/29/03
FILE NAME	V3BL026	V3CJ021	F3CL019	F3DG009	F3DX025	F3EI013	F3EI014	F3EI011	F3EI012	F3EO007	V3E#023	F3FB020	V3FX021	V3FX022	V3FX023	V3FX024	V3GG031	V3G#022
UNITS	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv
Formaldehyde	2.122	2.195	1.008	2.882	2.845	2.589	2.587	2.633	2.628	1.073	2.072	2.470	3.072	3.102	3.151	3.154	2.985	3.512
Acetaldehyde	0.889	0.809	0.493	1.131	1.237	0.942	0.944	0.935	0.936	0.545	0.755	1.019	1.039	1.040	0.973	0.976	0.935	1.033
Acetone	1.342	1.305	0.572	1.880	1.029	0.700	0.698	0.814	0.815	1.377	0.794	0.951	0.493	0.507	0.410	0.413	0.923	0.709
Propionaldehyde	0.091	0.077	0.048	0.118	0.149	0.164	0.165	0.142	0.144	0.064	0.109	0.121	0.203	0.210	0.210	0.210	0.148	0.192
Crotonaldehyde	0.011	0.008	0.003	0.035	0.042	0.023	0.020	0.022	0.028	0.016	0.025	0.042	0.130	0.120	0.123	0.124	0.064	0.057
Butyr/Isobutyraldehyde	0.109	0.108	0.077	0.175	0.166	0.173	0.175	0.176	0.181	0.124	0.063	0.162	0.102	0.095	0.094	0.095	0.108	0.093
Benzaldehyde	0.044	0.030	0.018	0.034	0.043	0.027	0.029	0.027	0.027	0.016	0.042	0.031	0.065	0.069	0.066	0.065	0.054	0.023
Isovaleraldehyde	ND	ND	ND	0.010	0.017	ND	ND	ND	ND	0.002	0.007	ND	0.035	0.015	0.014	0.014	0.013	ND
Valeraldehyde	0.031	0.027	0.014	0.036	0.043	0.043	0.040	0.037	0.043	0.017	0.027	0.032	0.040	0.036	0.035	0.035	0.035	0.037
Tolualdehydes	0.031	0.033	0.010	0.024	0.038	0.018	0.018	0.018	0.018	0.019	0.019	0.031	0.037	0.042	0.037	0.038	0.048	0.026
Hexaldehyde	0.025	0.029	0.017	0.039	0.059	0.035	0.033	0.034	0.035	0.019	0.040	0.045	0.045	0.047	0.042	0.040	0.044	0.042
2,5-Dimethylbenzaldehyde	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

Table C-6. EATN Monitor. Data from July 2003 to December 2003 (Carb.)

SAMPLE #	34712	34886	35192	35779 D1	35779 R1	35781 D2	35781 R2	35992	36154 D1	36154 R1	36156 D2	36156 R2	36431	36634	36887	37217	37360
SAMPLE DATE	7/26/03	8/9/03	8/19/03	9/12/03	9/12/03	9/12/03	9/12/03	9/24/03	10/6/03	10/6/03	10/6/03	10/6/03	10/30/03	11/11/03	11/23/03	12/17/03	12/29/03
ANALYSIS DATE	9/3/03	9/9/03	9/23/03	10/21/03	10/21/03	10/21/03	10/21/03	11/4/03	11/12/03	11/12/03	11/12/03	11/12/03	12/11/03	12/15/03	12/31/03	1/30/04	2/5/04
FILE NAME	V3IB027	V3IH021	F3IV023	F3JT029	F3JT030	F3JT027	F3JT028	F3KC021	V3KK026	V3KK027	V3KK028	V3KK029	F3LJ023	F3LN013	V3L$021	V4A#021	V4BD017
UNITS	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv
Formaldehyde	5.622	3.785	4.636	3.962	3.990	4.009	4.022	3.755	1.958	1.967	2.020	2.010	4.826	3.813	2.922	2.076	0.847
Acetaldehyde	1.225	0.809	0.777	1.365	1.373	1.364	1.366	1.325	0.842	0.835	0.846	0.850	1.491	1.060	0.787	0.613	0.328
Acetone	0.482	0.420	0.303	0.810	0.811	0.835	0.832	1.075	1.108	1.108	1.098	1.100	1.371	0.673	0.415	3.369	0.757
Propionaldehyde	0.314	0.188	0.201	0.252	0.253	0.277	0.256	0.201	0.095	0.088	0.101	0.097	0.169	0.135	0.113	0.087	0.036
Crotonaldehyde	0.193	0.113	0.133	0.048	0.046	0.058	0.048	0.067	0.039	0.046	0.041	0.042	0.046	0.036	0.025	0.024	0.012
Butyr/Isobutyraldehyde	0.142	0.084	0.111	0.132	0.129	0.110	0.136	0.146	0.099	0.099	0.098	0.113	0.149	0.094	0.145	0.124	0.049
Benzaldehyde	0.054	0.023	0.056	0.047	0.044	0.050	0.046	0.055	0.033	0.026	0.028	0.028	0.053	0.037	0.021	0.034	0.014
Isovaleraldehyde	ND	ND	ND	ND	ND	ND	ND	ND	0.014	0.011	0.013	0.014	ND	0.013	0.005	0.004	0.004
Valeraldehyde	0.050	0.034	0.036	0.052	0.041	0.043	0.052	0.042	0.030	0.030	0.035	0.033	0.035	0.024	0.022	0.015	0.012
Tolualdehydes	0.052	0.040	0.045	0.043	0.042	0.049	0.045	0.052	0.057	0.054	0.053	0.054	0.114	0.064	0.031	0.037	0.017
Hexaldehyde	0.067	0.045	0.037	0.050	0.049	0.047	0.049	0.040	0.037	0.036	0.035	0.036	0.033	0.023	0.023	0.018	0.016
2,5-Dimethylbenzaldehyde	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

Table C-7. LOTN Monitor. Data from May 2002 to December 2002 (VOC)

SAMPLE SITE #	LOTN 27024	LOTN 27334	LOTN 27928	LOTN 28255 D1	LOTN 28255 R1	LOTN 28257 D2	LOTN 28257 R2	LOTN 28621	LOTN 28717	LOTN 29292	LOTN 29668	LOTN 29962	LOTN 30106	LOTN 30285 D1	LOTN 30581	LOTN 30828	LOTN 30925	LOTN 31255
SAMPLE DATE	4/20/2002	5/2/2002	6/7/2002	6/19/2002	6/19/2002	6/19/2002	6/19/2002	7/7/2002	7/13/2002	8/18/2002	8/30/2002	9/23/2002	10/5/2002	10/17/2002	11/10/2002	11/22/2002	12/4/2002	12/16/2002
ANALYSIS DATE	4/27/2002	5/17/2002	6/19/2002	7/2/2002	7/11/2002	7/2/2002	7/11/2002	8/2/2002	8/6/2002	9/16/2002	9/23/2002	10/17/2002	10/25/2002	11/13/2002	12/10/2002	12/18/2002	12/28/2002	1/9/2003
FILE NAME	L2DZ021	L2ER008	N2FR017	N2GB011	N2GK009	N2GB012	N2GK010	L2HA020	L2HF010	L2IP010	L2IV017	L2JQ010	L2JX021	N2KL020	N2LJ007	L2LR010	L2L~019	L3AH017
UNITS	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv
Acetylene	1.78	1.83	1.05	1.72	1.59	2.12	1.46	0.88	0.67	1.03	1.25	0.36	0.48	3.03	0.37	0.96	0.53	4.04
Propylene	2.25	1.84	0.46	1.69	1.50	1.58	1.22	0.55	0.37	0.77	0.76	0.20	0.31	1.78	0.14	0.10	0.28	1.09
Dichlorodifluoromethane	0.59	0.58	0.67	0.61	0.59	0.55	0.48	0.70	0.61	0.75	0.75	0.77	0.65	0.97	0.56	1.27	0.55	0.49
Chloromethane	0.65	0.72	0.71	0.68	0.52	0.68	0.42	0.74	0.62	0.80	0.77	0.54	0.52	0.84	0.54	1.05	0.41	0.43
Dichlorotetrafluoroethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Vinyl Chloride	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,3-Butadiene	0.04	ND	0.02	0.09	ND	ND	ND	ND	ND	ND	0.04	ND	ND	0.24	ND	0.15	ND	0.14
Bromomethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.09	ND	ND	ND	ND	ND	ND	ND
Chloroethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Acetonitrile	ND	ND	1.74	3.16	1.69	0.49	0.45	2.83	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Trichlorofluoromethane	0.25	0.24	0.31	0.26	0.30	0.21	0.21	0.32	0.31	0.32	0.30	0.41	0.34	0.41	0.26	0.61	0.29	0.27
Acrylonitrile	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,1-Dichloroethene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Methylene Chloride	0.14	0.07	0.14	ND	0.19	ND	0.06	0.13	0.09	0.09	0.11	ND	ND	0.21	ND	0.14	ND	ND
Trichlorotrifluoroethane	0.21	0.12	0.21	0.15	0.12	0.13	0.08	0.21	0.13	0.23	0.19	ND	0.05	0.13	0.05	0.33	0.13	0.15
trans - 1,2 - Dichloroethylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,1 - Dichloroethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Methyl tert-Butyl Ether	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.44	ND	ND	ND	ND
Methyl Ethyl Ketone	5.47	2.19	1.59	4.00	2.96	4.61	3.80	1.69	0.71	1.76	2.47	ND	ND	1.01	ND	ND	ND	1.25
Chloroprene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

Table C-7. Continued

SAMPLE SITE #	LOTN 27024	LOTN 27334	LOTN 27928	LOTN 28255 D1	LOTN 28255 R1	LOTN 28257 D2	LOTN 28257 R2	LOTN 28621	LOTN 28717	LOTN 29292	LOTN 29668	LOTN 29962	LOTN 30106	LOTN 30285 D1	LOTN 30581	LOTN 30828	LOTN 30925	LOTN 31255
SAMPLE DATE	4/20/2002	5/2/2002	6/7/2002	6/19/2002	6/19/2002	6/19/2002	6/19/2002	7/7/2002	7/13/2002	8/18/2002	8/30/2002	9/23/2002	10/5/2002	10/17/2002	11/10/2002	11/22/2002	12/4/2002	12/16/2002
ANALYSIS DATE	4/27/2002	5/17/2002	6/19/2002	7/2/2002	7/11/2002	7/2/2002	7/11/2002	8/2/2002	8/6/2002	9/16/2002	9/23/2002	10/17/2002	10/25/2002	11/13/2002	12/10/2002	12/18/2002	12/28/2002	1/9/2003
FILE NAME	L2DZ021	L2ER008	N2FR017	N2GB011	N2GK009	N2GB012	N2GK010	L2HA020	L2HF010	L2IP010	L2IV017	L2JQ010	L2JX021	N2KL020	N2LJ007	L2LR010	L2L~019	L3AH017
UNITS	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv
cis-1,2-Dichloroethylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Bromochloromethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chloroform	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.03	ND	ND	ND	ND
Ethyl tert-Butyl Ether	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,2-Dichloroethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,1,1-Trichloroethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.04	ND	0.02	0.03	ND	0.08	ND	ND
Benzene	0.46	0.34	0.35	0.64	0.67	0.53	0.59	0.49	0.34	0.42	0.64	0.16	0.28	0.84	0.17	0.39	0.27	0.72
Carbon Tetrachloride	0.08	0.09	0.11	0.14	0.10	0.11	ND	0.11	0.09	0.11	0.11	0.09	0.10	0.10	ND	0.24	0.09	0.09
tert-Amyl Methyl Ether	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,2-Dichloropropane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Ethyl Acrylate	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Bromodichloromethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Trichloroethylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Methyl Methacrylate	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
cis-1,3-Dichloropropene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Methyl Isobutyl Ketone	ND	ND	ND	0.27	ND	0.25	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
trans-1,3-Dichloropropene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

Table C-7. Continued

SAMPLE SITE #	LOTN 27024	LOTN 27334	LOTN 27928	LOTN 28255 D1	LOTN 28255 R1	LOTN 28257 D2	LOTN 28257 R2	LOTN 28621	LOTN 28717	LOTN 29292	LOTN 29668	LOTN 29962	LOTN 30106	LOTN 30285 D1	LOTN 30581	LOTN 30828	LOTN 30925	LOTN 31255
SAMPLE DATE	4/20/2002	5/2/2002	6/7/2002	6/19/2002	6/19/2002	6/19/2002	6/19/2002	7/7/2002	7/13/2002	8/18/2002	8/30/2002	9/23/2002	10/5/2002	10/17/2002	11/10/2002	11/22/2002	12/4/2002	12/16/2002
ANALYSIS DATE	4/27/2002	5/17/2002	6/19/2002	7/2/2002	7/11/2002	7/2/2002	7/11/2002	8/2/2002	8/6/2002	9/16/2002	9/23/2002	10/17/2002	10/25/2002	11/13/2002	12/10/2002	12/18/2002	12/28/2002	1/9/2003
FILE NAME	L2DZ021	L2ER008	N2FR017	N2GB011	N2GK009	N2GB012	N2GK010	L2HA020	L2HF010	L2IP010	L2IV017	L2JQ010	L2JX021	N2KL020	N2LJ007	L2LR010	L2L~019	L3AH017
UNITS	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv
1,1,2 - Trichloroethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Toluene	1.30	0.61	0.73	1.60	1.67	1.28	1.14	1.07	0.75	0.98	1.40	0.68	0.56	1.60	0.22	0.45	0.35	1.12
Dibromochloromethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,2-Dibromoethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
N-Octane	0.09	0.08	0.05	ND	0.11	ND	0.11	0.08	ND	ND	0.09	ND	ND	0.07	0.08	ND	ND	ND
Tetrachloroethylene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chlorobenzene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Ethylbenzene	0.17	0.09	0.09	0.25	0.25	0.21	0.20	0.19	0.12	0.17	0.18	ND	ND	0.34	ND	0.11	ND	0.20
m,p - Xylene	0.52	0.25	0.30	0.63	0.71	0.63	0.62	0.65	0.27	0.44	0.54	ND	0.28	0.78	ND	0.28	0.12	0.55
Bromoform	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Styrene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.04	ND	ND	ND	ND
1,1,2,2 - Tetrachloroethane	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
o - Xylene	0.23	0.11	0.11	0.30	0.28	0.28	0.30	0.27	0.13	0.23	0.26	ND	0.12	0.37	ND	0.15	ND	0.21
1,3,5-Trimethylbenzene	0.07	ND	ND	0.07	0.07	0.07	0.06	0.08	ND	0.08	ND	ND	0.03	0.13	ND	0.06	ND	0.07
1,2,4-Trimethylbenzene	0.21	0.09	0.06	0.22	0.40	0.24	0.30	0.23	0.13	0.21	0.20	ND	0.11	0.43	ND	0.13	ND	0.26
m - Dichlorobenzene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chloromethylbenzene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
p - Dichlorobenzene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.02	ND	ND	ND	ND
o - Dichlorobenzene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

Table C-7. Continued

SAMPLE SITE #	LOTN 27024	LOTN 27334	LOTN 27928	LOTN 28255 D1	LOTN 28255 R1	LOTN 28257 D2	LOTN 28257 R2	LOTN 28621	LOTN 28717	LOTN 29292	LOTN 29668	LOTN 29962	LOTN 30106	LOTN 30285 D1	LOTN 30581	LOTN 30828	LOTN 30925	LOTN 31255
SAMPLE DATE	4/20/2002	5/2/2002	6/7/2002	6/19/2002	6/19/2002	6/19/2002	6/19/2002	7/7/2002	7/13/2002	8/18/2002	8/30/2002	9/23/2002	10/5/2002	10/17/2002	11/10/2002	11/22/2002	12/4/2002	12/16/2002
ANALYSIS DATE	4/27/2002	5/17/2002	6/19/2002	7/2/2002	7/11/2002	7/2/2002	7/11/2002	8/2/2002	8/6/2002	9/16/2002	9/23/2002	10/17/2002	10/25/2002	11/13/2002	12/10/2002	12/18/2002	12/28/2002	1/9/2003
FILE NAME	L2DZ021	L2ER008	N2FR017	N2GB011	N2GK009	N2GB012	N2GK010	L2HA020	L2HF010	L2IP010	L2IV017	L2JQ010	L2JX021	N2KL020	N2LJ007	L2LR010	L2L~019	L3AH017
UNITS	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv
1,2,4-Trichlorobenzene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Hexachloro-1,3-Butadiene	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

Table C-8. LOTN Monitor. Data from May 2002 to December 2002 (Carb.)

SAMPLE #	27384	27751	27930	28256 D1	28256 R1	28258 D2	28258 R2	28623	28718	29293	29670	29790	29963	30107	30286 D1	30286 R1	30433	#30582	#30829	30926	31256
SAMPLE DATE	5/2/2002	5/26/2002	6/7/2002	6/19/2002	6/19/2002	6/19/2002	6/19/2002	7/7/2002	7/13/2002	8/18/2002	8/30/2002	9/11/2002	9/23/2002	10/5/2002	10/17/2002	10/17/2002	10/29/2002	11/10/2002	11/22/2002	12/4/2002	12/16/2002
ANALYSIS DATE	5/29/2002	6/11/2002	6/26/2002	7/17/2002	7/17/2002	7/17/2002	7/17/2002	7/18/2002	7/31/2002	9/5/2002	9/19/2002	10/2/2002	10/3/2002	10/25/2002	11/6/2002	11/6/2002	11/26/2002	12/21/2002	12/24/2002	1/9/2003	1/22/2003
FILE NAME	F2E#018	F2FJ019	F2FZ014	F2GP034	F2GP035	F2GP036	F2GP037	V2GQ019	F2G$025	V2ID056	V2IR040	F2JB007	F2JB046	V2JX019	V2KE028	V2KE029	V2KY016	V2LT026	V2LW049	V3AH042	V3AU025
UNITS	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv
Formaldehyde	2.52	2.72	2.76	5.57	5.55	5.42	5.43	6.39	2.50	3.90	3.68	5.47	2.95	2.57	2.37	2.34	1.61	1.92	1.64	0.95	4.28
Acetaldehyde	0.80	0.99	0.96	1.55	1.54	1.52	1.52	1.44	0.70	0.73	1.15	1.75	0.75	0.91	0.89	0.89	0.71	0.76	0.51	0.61	1.44
Acetone	0.57	0.51	0.48	1.20	1.15	1.10	1.11	0.60	0.43	0.29	0.42	0.58	1.04	0.70	1.31	1.31	0.65	0.42	0.83	1.00	1.46
Propionaldehyde	0.17	0.17	0.13	0.26	0.24	0.33	0.33	0.23	0.12	0.13	0.24	0.36	0.08	0.10	0.09	0.08	0.07	0.14	0.04	0.06	0.15
Crotonaldehyde	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	ND	0.01	0.01	0.02	0.01	0.01	0.004	0.004	0.004	0.003	0.003	0.004	0.01
Butyr/Isobutyraldehyde	0.20	0.19	0.21	0.14	0.12	0.14	0.14	0.10	0.10	0.12	0.15	0.21	0.17	0.12	0.14	0.15	0.10	0.11	0.07	0.10	0.20
Benzaldehyde	0.09	0.05	0.06	0.08	0.06	0.06	0.07	0.09	0.04	0.05	0.04	0.10	0.03	0.04	0.04	0.04	0.03	0.03	0.02	0.01	0.05
Isovaleraldehyde	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.02	ND	0.004	0.01	0.01	0.01	0.01	0.01	ND	ND	ND
Valeraldehyde	0.06	0.05	0.04	0.06	0.06	0.06	0.06	0.05	0.02	0.03	0.04	0.06	0.02	0.03	0.03	0.03	0.02	0.04	0.02	0.02	0.04
Tolualdehydes	0.02	0.03	0.04	0.05	0.05	0.05	0.05	0.06	0.02	0.07	0.04	0.08	0.02	0.03	0.06	0.06	0.02	0.01	0.02	0.01	0.04
Hexaldehyde	0.07	0.05	0.03	0.07	0.06	0.06	0.06	0.05	0.03	0.03	0.04	0.06	0.02	0.03	0.04	0.04	0.03	0.07	0.03	0.02	0.06
2,5-Dimethylbenzaldehyde	ND	ND	ND	ND	ND	ND	ND	0.04	ND	0.01	0.01	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

Table C-9. LOTN Monitor. Data from January 2003 to May 2003 (VOC)

SAMPLE SITE #		LOTN 31424	LOTN 31599	LOTN 31926	LOTN 32027	LOTN 32217	LOTN 32389	LOTN 32548 D1	LOTN 32548 R1	LOTN 32551 D2	LOTN 32551 R2	LOTN 32644	LOTN 32883	LOTN 33110	LOTN 33289 D1	LOTN 33289 R1	LOTN 33293 D2	LOTN 33293 R2	LOTN 33445
SAMPLE DATE		1/3/2003	1/15/2003	2/8/2003	2/20/2003	3/4/2003	3/16/2003	3/28/2003	3/28/2003	3/28/2003	3/28/2003	4/9/2003	4/21/2003	5/3/2003	5/15/2003	5/15/2003	5/15/2003	5/15/2003	5/27/2003
ANALYSIS DATE		1/20/2003	2/6/2003	2/27/2003	3/13/2003	3/21/2003	3/26/2003	4/15/2003	4/23/2003	4/15/2003	4/23/2003	4/29/2003	5/20/2003	5/24/2003	6/5/2003	6/7/2003	6/5/2003	6/7/2003	6/4/2003
FILE NAME		N3AT014	N3BF007	N3B~012	N3CL016	L3CT018	N3CY019	L3DO007	L3DV014	L3DO008	L3DV015	N3D#011	L3ES024	L3WE014	N3FE013	N3FF013	N3FE014	N3FF014	N3FC024
UNITS	MDL	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv
Acetylene	0.08	0.98	2.12	3.13	1.49	2.22	3.27	0.67	0.52	0.63	0.48	1.28	0.89	1.08	1.15	1.26	1.43	1.36	1.59
Propylene	0.11	0.26	0.41	0.37	0.32	1.21	1.63	0.43	0.25	0.56	0.32	0.34	0.12	0.40	0.45	0.47	0.56	0.53	0.77
Dichlorodifluoromethane	0.15	0.47	0.48	0.46	0.64	0.63	0.77	0.77	0.49	0.73	0.41	0.65	0.54	0.53	0.59	0.66	0.60	0.58	0.53
Chloromethane	0.10	0.49	0.48	0.45	0.54	0.54	0.70	0.55	0.59	0.60	0.53	0.66	0.68	0.84	0.66	0.70	0.69	0.64	0.51
Dichlorotetrafluoroethane	0.11	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.02	ND	ND	ND	ND	ND	ND
Vinyl Chloride	0.13	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,3-Butadiene	0.12	ND	ND	ND	ND	0.19	0.10	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.07	0.04	0.11
Bromomethane	0.13	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chloroethane	0.13	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Acetonitrile	0.43	ND	3.64	ND	ND	ND	5.22	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	3.18
Trichlorofluoromethane	0.07	0.29	0.24	0.22	0.26	0.28	0.30	0.33	0.26	0.39	0.25	0.31	0.30	0.24	0.28	0.30	0.28	0.27	0.25
Acrylonitrile	0.37	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,1-Dichloroethene	0.11	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Methylene Chloride	0.09	ND	0.05	ND	ND	0.20	0.09	0.08	0.09	ND	0.08	0.09	ND	0.12	0.17	0.20	0.29	0.28	0.11
Trichlorotrifluoroethane	0.10	0.10	0.11	0.09	0.07	0.08	0.04	0.11	0.11	0.14	0.09	0.13	0.09	0.06	0.12	0.13	0.11	0.11	0.11
trans - 1,2 - Dichloroethylene	0.12	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,1 - Dichloroethane	0.06	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Methyl tert-Butyl Ether	0.16	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.06	ND	ND	ND	ND	ND	0.25
Methyl Ethyl Ketone	0.25	ND	ND	ND	1.00	ND	ND	1.33	1.07	ND	0.87	0.82	2.64	19.99	2.32	2.45	12.36	11.18	4.99
Chloroprene	0.07	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
cis-1,2-Dichloroethylene	0.14	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

Table C-9. Continued

SAMPLE SITE #		LOTN 31424	LOTN 31599	LOTN 31926	LOTN 32027	LOTN 32217	LOTN 32389	LOTN 32548 D1	LOTN 32548 R1	LOTN 32551 D2	LOTN 32551 R2	LOTN 32644	LOTN 32883	LOTN 33110	LOTN 33289 D1	LOTN 33289 R1	LOTN 33293 D2	LOTN 33293 R2	LOTN 33445
SAMPLE DATE		1/3/2003	1/15/2003	2/8/2003	2/20/2003	3/4/2003	3/16/2003	3/28/2003	3/28/2003	3/28/2003	3/28/2003	4/9/2003	4/21/2003	5/3/2003	5/15/2003	5/15/2003	5/15/2003	5/15/2003	5/27/2003
ANALYSIS DATE		1/20/2003	2/6/2003	2/27/2003	3/13/2003	3/21/2003	3/26/2003	4/15/2003	4/23/2003	4/15/2003	4/23/2003	4/29/2003	5/20/2003	5/24/2003	6/5/2003	6/7/2003	6/5/2003	6/7/2003	6/4/2003
FILE NAME		N3AT014	N3BF007	N3B~012	N3CL016	L3CT018	N3CY019	L3DO007	L3DV014	L3DO008	L3DV015	N3D#011	L3ES024	L3WE014	N3FE013	N3FF013	N3FE014	N3FF014	N3FC024
UNITS	MDL	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv
Bromochloromethane	0.21	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chloroform	0.09	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.03	ND	ND	ND	ND	ND	ND
Ethyl tert-Butyl Ether	0.14	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,2 - Dichloroethane	0.11	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,1,1 - Trichloroethane	0.10	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.03	0.04	ND	0.06	0.03	0.03	0.04	0.03	0.03
Benzene	0.06	0.31	0.40	0.44	0.37	0.75	1.07	0.24	0.21	0.19	0.22	0.30	0.31	0.34	0.27	0.26	0.35	0.30	0.47
Carbon Tetrachloride	0.08	0.04	0.06	0.08	0.10	0.08	ND	0.10	0.08	0.11	0.11	0.10	0.11	0.09	0.11	0.11	0.10	0.11	0.10
tert-Amyl Methyl Ether	0.16	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,2 - Dichloropropane	0.07	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Ethyl Acrylate	0.27	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Bromodichloromethane	0.09	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Trichloroethylene	0.17	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.03	ND	0.03	ND	0.02
Methyl Methacrylate	0.19	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
cis -1,3 - Dichloropropene	0.10	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Methyl Isobutyl Ketone	0.25	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.04
trans - 1,3 - Dichloropropene	0.13	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,1,2 - Trichloroethane	0.09	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Toluene	0.06	0.35	0.54	0.43	0.32	1.35	1.88	0.45	0.39	0.36	0.45	0.48	1.33	8.58	5.66	5.73	7.65	6.64	4.24

Table C-9. Continued

SAMPLE SITE #		LOTN 31424	LOTN 31599	LOTN 31926	LOTN 32027	LOTN 32217	LOTN 32389	LOTN 32548 D1	LOTN 32548 R1	LOTN 32551 D2	LOTN 32551 R2	LOTN 32644	LOTN 32883	LOTN 33110	LOTN 33289 D1	LOTN 33289 R1	LOTN 33293 D2	LOTN 33293 R2	LOTN 33445
SAMPLE DATE		1/3/2003	1/15/2003	2/8/2003	2/20/2003	3/4/2003	3/16/2003	3/28/2003	3/28/2003	3/28/2003	3/28/2003	4/9/2003	4/21/2003	5/3/2003	5/15/2003	5/15/2003	5/15/2003	5/15/2003	5/27/2003
ANALYSIS DATE		1/20/2003	2/6/2003	2/27/2003	3/13/2003	3/21/2003	3/26/2003	4/15/2003	4/23/2003	4/15/2003	4/23/2003	4/29/2003	5/20/2003	5/24/2003	6/5/2003	6/7/2003	6/5/2003	6/7/2003	6/4/2003
FILE NAME		N3AT014	N3BF007	N3B~012	N3CL016	L3CT018	N3CY019	L3DO007	L3DV014	L3DO008	L3DV015	N3D#011	L3ES024	L3WE014	N3FE013	N3FF013	N3FE014	N3FF014	N3FC024
UNITS	MDL	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv
Dibromochloromethane	0.07	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,2-Dibromoethane	0.07	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
N-Octane	0.12	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.04	0.06
Tetrachloroethylene	0.10	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.04	ND	ND	0.02	0.03	0.02	ND	0.06
Chlorobenzene	0.10	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Ethylbenzene	0.08	ND	0.09	ND	ND	0.22	0.31	ND	0.08	ND	0.07	0.06	0.13	0.27	0.21	0.20	0.29	0.25	0.24
m,p - Xylene	0.08	0.13	0.24	0.19	0.16	0.60	0.86	0.12	0.15	0.21	0.18	0.17	0.22	0.51	0.70	0.76	0.97	0.85	0.79
Bromoform	0.07	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Styrene	0.08	ND	0.02	ND	ND	ND	ND	ND	ND	ND	ND	0.06	ND	ND	ND	ND	0.03	0.03	0.07
1,1,2,2 - Tetrachloroethane	0.09	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
o - Xylene	0.06	ND	0.13	0.09	0.08	0.32	0.41	ND	0.05	0.04	0.05	0.06	0.12	0.23	0.22	0.24	0.29	0.26	0.27
1,3,5-Trimethylbenzene	0.05	ND	ND	ND	ND	0.10	ND	ND	ND	ND	ND	0.02	ND	ND	0.05	0.04	0.06	0.06	0.10
1,2,4-Trimethylbenzene	0.09	ND	0.10	0.08	ND	0.30	0.32	ND	ND	ND	ND	0.05	0.12	0.13	0.13	0.12	0.17	0.13	0.25
m - Dichlorobenzene	0.12	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chloromethylbenzene	0.09	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
p - Dichlorobenzene	0.15	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.03
o - Dichlorobenzene	0.12	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,2,4-Trichlorobenzene	0.16	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Hexachloro-1,3-Butadiene	0.20	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

Table C-10. LOTN Monitor. Data from July 2003 to December 2003 (VOC)

SAMPLE SITE #		LOTN 34185	LOTN 34882	LOTN 35194	LOTN 35988	LOTN 36149	LOTN 36520	LOTN 36889	LOTN 37005	LOTN 37093	LOTN 37213	LOTN 37245	LOTN 37351
SAMPLE DATE		7/2/2003	8/9/2003	8/19/2003	9/24/2003	10/6/2003	11/5/2003	11/23/2003	12/5/2003	12/11/2003	12/20/2003	12/23/2003	12/26/2003
ANALYSIS DATE		8/1/2003	9/11/2003	9/18/2003	10/11/2003	10/17/2003	11/18/2003	12/16/2003	12/30/2003	12/31/2003	1/8/2004	1/8/2004	1/13/2004
FILE NAME		L3HA008	L3IJ015	N3IQ018	N3JJ015	N3JP020	N3KR018	L3LO019	L3L$014	L3L%006	L4AG020	L4AH006	N4AM010
UNITS	MDL	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv
Acetylene	0.08	0.36	0.97	0.62	2.00	2.43	0.56	0.58	2.11	2.15	2.70	0.84	3.01
Propylene	0.11	0.19	0.45	0.29	1.04	1.01	0.59	0.17	0.34	0.35	0.59	0.24	1.50
Dichlorodifluoromethane	0.15	0.33	0.64	0.69	0.69	0.67	0.47	0.64	0.58	0.61	0.63	0.65	0.64
Chloromethane	0.10	0.28	0.60	0.63	0.54	0.56	0.65	0.54	0.51	0.53	0.48	0.62	0.49
Dichlorotetrafluoroethane	0.11	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Vinyl Chloride	0.13	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,3-Butadiene	0.12	ND	0.08	ND	0.14	0.12	0.05	ND	ND	ND	ND	ND	0.19
Bromomethane	0.13	ND	ND	ND	0.03	ND	0.02	ND	ND	ND	ND	ND	ND
Chloroethane	0.13	ND	ND	ND	ND	ND	0.30	ND	ND	ND	ND	ND	ND
Acetonitrile	0.43	0.94	ND	0.87	8.46	0.28	116.85	18.77	ND	ND	ND	ND	0.39
Trichlorofluoromethane	0.07	0.17	1.19	0.34	0.36	0.35	0.62	0.27	0.25	0.24	0.25	0.29	0.26
Acrylonitrile	0.37	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,1-Dichloroethene	0.11	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Methylene Chloride	0.09	ND	0.15	0.08	0.39	0.28	0.12	ND	ND	ND	ND	ND	0.14
Trichlorotrifluoroethane	0.10	ND	0.12	0.16	0.08	0.08	0.09	0.10	0.10	0.09	ND	0.09	0.08
trans - 1,2 - Dichloroethylene	0.12	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,1 - Dichloroethane	0.06	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Methyl tert-Butyl Ether	0.16	ND	0.16	ND	0.12	0.09	ND	ND	ND	ND	ND	ND	0.07
Methyl Ethyl Ketone	0.25	0.64	1.39	1.01	0.52	0.27	ND	0.55	ND	ND	ND	ND	ND
Chloroprene	0.07	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
cis-1,2-Dichloroethylene	0.14	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Bromochloromethane	0.21	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chloroform	0.09	ND	ND	0.02	0.05	0.04	0.18	ND	ND	ND	ND	ND	0.04
Ethyl tert-Butyl Ether	0.14	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,2 - Dichloroethane	0.11	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,1,1 - Trichloroethane	0.10	ND	0.02	ND	0.03	0.03	0.03	ND	ND	ND	ND	ND	ND
Benzene	0.06	ND	0.38	0.19	0.72	0.68	0.49	0.19	0.34	0.33	0.43	0.20	0.76
Carbon Tetrachloride	0.08	0.03	0.09	0.09	0.11	0.11	0.08	0.10	0.09	0.10	0.04	0.04	0.08
tert-Amyl Methyl Ether	0.16	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,2 - Dichloropropane	0.07	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Ethyl Acrylate	0.27	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Bromodichloromethane	0.09	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Trichloroethylene	0.17	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Methyl Methacrylate	0.19	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
cis -1,3 - Dichloropropene	0.10	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Methyl Isobutyl Ketone	0.25	ND	ND	ND	ND	ND	15.06	ND	ND	ND	ND	ND	ND

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Table C-10. Continued

SAMPLE SITE #		LOTN 34185	LOTN 34882	LOTN 35194	LOTN 35988	LOTN 36149	LOTN 36520	LOTN 36889	LOTN 37005	LOTN 37093	LOTN 37213	LOTN 37245	LOTN 37351
SAMPLE DATE		7/2/2003	8/9/2003	8/19/2003	9/24/2003	10/6/2003	11/5/2003	11/23/2003	12/5/2003	12/11/2003	12/20/2003	12/23/2003	12/26/2003
ANALYSIS DATE		8/1/2003	9/11/2003	9/18/2003	10/11/2003	10/17/2003	11/18/2003	12/16/2003	12/30/2003	12/31/2003	1/8/2004	1/8/2004	1/13/2004
FILE NAME		L3HA008	L3IJ015	N3IQ018	N3JJ015	N3JP020	N3KR018	L3LO019	L3L$014	L3L%006	L4AG020	L4AH006	N4AM010
UNITS	MDL	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv
trans - 1,3 - Dichloropropene	0.13	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,1,2 - Trichloroethane	0.09	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Toluene	0.06	0.58	1.26	0.41	1.57	1.64	6.00	0.21	0.49	0.48	0.62	0.27	0.96
Dibromochloromethane	0.07	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,2-Dibromoethane	0.07	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
N-Octane	0.12	ND	0.08	ND	ND	0.03	1.67	ND	ND	ND	ND	ND	0.05
Tetrachloroethylene	0.10	ND	0.02	ND	0.05	0.06	0.03	ND	ND	ND	ND	ND	ND
Chlorobenzene	0.10	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Ethylbenzene	0.08	ND	0.16	0.06	0.24	0.16	0.47	ND	ND	ND	ND	ND	0.17
m,p - Xylene	0.08	0.13	0.42	0.18	0.83	0.69	2.27	ND	0.20	0.20	0.28	0.10	0.54
Bromoform	0.07	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Styrene	0.08	ND	0.03	ND	0.05	0.03	0.46	ND	ND	ND	ND	ND	0.07
1,1,2,2 - Tetrachloroethane	0.09	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
o - Xylene	0.06	ND	0.22	0.06	0.38	0.28	1.03	ND	0.08	0.08	0.12	ND	0.23
1,3,5-Trimethylbenzene	0.05	ND	0.06	0.02	0.08	0.06	0.81	ND	ND	ND	ND	ND	0.08
1,2,4-Trimethylbenzene	0.09	ND	0.16	0.06	0.33	0.26	2.26	ND	0.07	0.08	0.12	ND	0.23
m - Dichlorobenzene	0.12	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Chloromethylbenzene	0.09	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
p - Dichlorobenzene	0.15	ND	ND	ND	0.07	0.04	0.04	ND	ND	ND	ND	ND	0.02
o - Dichlorobenzene	0.12	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
1,2,4-Trichlorobenzene	0.16	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Hexachloro-1,3-Butadiene	0.20	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

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Table C-11. LOTN Monitor. Data from January 2003 to December 2003 (Carb.)

SAMPLE #	31425	31601	31927	32029	32218	32391	32549D1	32549R1	32552D2	32552R2	32645	32885	33111	33290D1	33290R1	33294D2	33294R2	33447	34186	34883	35195	35989	36150	36891	37006	37094	37214	37246	37352
SAMPLE DATE	1/3/03	1/15/03	2/8/03	2/20/03	3/4/03	3/16/03	3/28/03	3/28/03	3/28/03	3/28/03	4/9/03	4/21/03	5/3/09	5/15/03	5/15/03	5/15/03	5/15/03	5/27/03	7/2/03	8/9/03	8/19/03	9/24/03	10/6/03	11/23/03	12/5/03	12/11/03	12/20/03	12/23/03	12/26/03
ANALYSIS DATE	2/6/03	2/13/03	3/11/03	3/12/03	4/7/03	4/25/03	5/9/03	5/10/03	5/9/03	5/9/03	5/15/03	5/30/03	6/3/03	6/25/03	6/25/03	6/25/03	6/25/03	7/8/03	8/7/03	9/8/03	9/23/03	11/4/03	11/12/03	12/30/03	1/11/04	1/14/04	2/3/04	2/3/04	2/5/04
FILE NAME	V3BE037	V3BL027	V3CJ022	F3CL014	F3DG007	F3DX022	F3EI009	F3EI010	F3EI007	F3EI008	F3EO008	V3E#021	F3FB021	V3FX025	V3FX026	V3FX027	V3FX028	V3GG045	F3HF040	V3IH020	F3IV024	F3KC022	V3KK020	V3L$019	V4AK018	V4AN009	V4BB038	V4BB039	V4BD007
UNITS	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv	ppbv
Formaldehyde	1.196	2.026	2.414	0.909	3.040	3.399	2.862	2.867	2.911	2.882	0.970	2.447	2.956	3.547	3.574	3.278	3.268	3.644	3.746	10.18	5.360	3.610	2.058	2.941	1.217	1.850	2.532	1.987	1.346
Acetaldehyde	0.489	0.770	0.834	0.441	1.114	1.403	0.824	0.830	0.830	0.828	0.521	0.766	1.124	1.025	1.019	0.917	0.910	1.220	0.730	0.916	0.716	1.180	0.996	0.690	0.516	0.560	0.949	0.535	0.586
Acetone	0.651	1.025	1.083	0.549	2.094	1.112	0.720	0.717	0.697	0.696	1.268	0.768	0.718	0.538	0.532	0.424	0.412	0.762	0.012	0.014	0.013	0.031	0.029	0.017	1.136	1.161	1.110	0.825	0.615
Propionaldehyde	0.052	0.079	0.095	0.056	0.109	0.177	0.108	0.097	0.107	0.104	0.063	0.126	0.179	0.207	0.207	0.197	0.195	0.251	0.005	0.012	0.006	0.005	0.003	0.003	0.059	0.056	0.079	0.047	0.054
Crotonaldehyde	0.005	0.008	0.008	0.002	0.037	0.043	0.026	0.024	0.024	0.031	0.015	0.033	0.057	0.184	0.186	0.167	0.168	0.093	0.308	0.300	0.259	0.103	0.055	0.025	0.025	0.028	0.048	0.018	0.023
Butyr/Isobutyraldehyde	0.097	0.103	0.117	0.088	0.165	0.210	0.188	0.189	0.194	0.208	0.211	0.311	1.149	0.579	0.580	0.442	0.443	0.482	0.113	0.193	0.098	0.135	0.113	0.146	0.102	0.112	0.106	0.070	0.063
Benzaldehyde	0.018	0.037	0.032	0.012	0.038	0.047	0.019	0.020	0.020	0.032	0.022	0.056	0.135	0.088	0.085	0.082	0.088	0.073	0.052	0.085	0.052	0.044	0.032	0.019	0.022	0.027	0.055	0.017	0.024
Isovaleraldehyde	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.003	0.021	0.014	0.014	0.014	0.013	0.012	ND	0.029	ND	ND	0.011	0.006	0.006	0.004	0.017	0.004	0.008
Valeraldehyde	0.018	0.031	0.028	0.013	0.033	0.053	0.029	0.029	0.026	0.030	0.017	0.034	0.056	0.046	0.045	0.044	0.043	0.063	0.040	0.087	0.040	0.052	0.036	0.020	0.017	0.015	0.020	0.015	0.015
Tolualdehydes	0.012	0.021	0.022	0.009	0.040	0.035	0.016	0.018	0.020	0.017	0.014	0.014	0.038	0.041	0.040	0.039	0.031	0.052	0.050	0.086	0.064	0.040	0.027	0.033	0.023	0.036	0.050	0.024	0.027
Hexaldehyde	0.025	0.024	0.024	0.016	0.036	0.074	0.030	0.029	0.030	0.029	0.019	0.047	0.070	0.063	0.064	0.059	0.050	0.095	0.046	0.111	0.046	0.051	0.051	0.024	0.021	0.020	0.028	0.020	0.017
2,5-Dimethylbenzaldehyde	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

Table C-12. Modeled and monitored PMSATs in Nashville, 2003

Date 2003	Benzene [ug/cum]				Formaldehyde [ug/cum]				Acetaldehyde [ug/cum]				1,3 Butadiene [ug/cum]				EC [ug/cum]	
	Modeled	EATN	Modeled	LOTN	Modeled	EATN	Modeled	LOTN	Modeled	EATN	Modeled	LOTN	Modeled	EATN	Modeled	LOTN	Modeled	LOTN
4-Mar	2.132	2.232	2.132	2.391	1.240	3.533	1.240	3.727	1.580	2.034	1.580	2.003	0.177	0.177	0.177	0.419		
16-Mar	0.934	3.379	0.934	3.411	2.645	3.487	2.645	4.167	1.456	2.225	1.456	2.522	0.059	0.309	0.059	0.221	0.886	1.150
22-Mar																	0.610	0.940
28-Mar	0.442	0.901	0.442	0.685	1.410	3.198	1.410	3.531	0.862	1.690	0.862	1.489					0.343	0.460
2-Jun																	0.593	0.812
6-Jun	0.383	1.243			2.087	4.305												
8-Jun																	0.470	0.928
14-Jun																	0.241	0.505
20-Jun	0.312	0.510							0.658	1.858							0.361	0.400
26-Jun																	0.735	0.748
6-Sep																	0.565	0.440
12-Sep	0.823	2.199			4.000	4.898			1.709	2.458			0.060	0.215			0.755	1.120
18-Sep																	0.630	0.920
24-Sep	0.935	2.146	0.935	2.282	3.086	4.603	3.086	4.425	1.405	2.384	1.405	2.122	0.071	0.341	0.071	0.319	0.803	1.530
30-Sep																	0.813	0.910
5-Dec			0.942	1.084			0.301	1.491			0.661	0.928					0.348	0.360
11-Dec			0.556	1.052			0.762	2.268			0.594	1.007					0.453	0.300
17-Dec	0.518	1.243			0.807	2.545	1.051		0.547	1.102							0.462	0.330
20-Dec			0.810	1.371			0.741	3.104			0.808	1.707						
23-Dec			0.835	0.638			1.704	2.436			0.828	0.963					0.333	0.330
26-Dec			1.515	2.423				1.650			1.419	1.054			0.151	0.419		
29-Dec	1.079	1.148			0.941	1.038			1.269	0.591								
Mean	0.840	1.667	1.011	1.704	2.027	3.451	1.438	2.978	1.186	1.793	1.068	1.533	0.092	0.261	0.115	0.345	0.553	0.717
STDEV	0.558	0.889	0.516	0.957	1.138	1.246	0.913	1.070	0.439	0.652	0.389	0.589	0.057	0.077	0.058	0.095	0.197	0.361

Table C-13. Daily EC concentrations (Data from January 2003 to December 2003)

Month	Day	LOTN [ug/m^3]	Month	Day	LOTN [ug/m^3]
1	3	0.2600	7	2	0.5700
1	9	0.3900	7	8	0.4300
1	15	0.3500	7	14	0.9300
1	21	0.4400	7	20	0.7700
1	27	0.5300	7	26	0.8000
2	2	0.3300	8	1	0.6500
2	8	0.4400	8	7	0.7300
2	14	0.5600	8	13	0.4100
2	20	0.3200	8	19	0.3900
2	26	0.3800	8	25	1.0400
3	4	0.9700	8	31	0.2800
3	10	0.3700	9	6	0.4400
3	16	1.1500	9	12	1.1200
3	22	0.9400	9	18	0.9200
3	28	0.4600	9	24	1.5300
4	3	0.5000	9	30	0.9100
4	9	0.4700	10	6	1.3200
4	15	1.3700	10	18	0.7800
4	21	0.4700	10	24	0.8000
4	27	0.8100	10	30	0.6500
5	3	0.4600	11	5	0.3300
5	9	0.7000	11	11	0.5300
5	15	0.8000	11	17	0.6500
5	21	0.4200	11	23	0.3800
5	27	0.9500	11	29	0.2800
6	2	0.8100	12	5	0.3600
6	8	0.9300	12	11	0.3000
6	14	0.5100	12	17	0.3300
6	20	0.4000	12	23	0.3300
6	26	0.7500	12	29	1.0500

VITA

Luis Alonso Díaz Robles was born in Ovalle, Chile. In 1993 he received his title of Chemical engineer from the University of Santiago in Chile. In the same year he accepted the position as process engineer in UNILEVER CHILE. In 1998 he accepted the position as Instructor professor at the Catholic University of Temuco, School of Environmental Engineering. During his years as a professor, he taught Transport Phenomena, Waste Water Treatment, Heat Transfer, and Dynamic Processes Control. He performed several research studies on wastewater treatment. In summer 2002 he enrolled in the Environmental Engineering program at the University of Tennessee in Knoxville to study his PhD concentrating his studies in air quality modeling, health-effects, and statistics. In the academic year 2003-2004, he was the President of the A&WMA Student Chapter at the University of Tennessee Knoxville. In that academic period, for first time, the Student Chapter was awarded with the first place for large-size institutions in the Air & Waste Management Association's 97th Annual Conference & Exhibition, Indianapolis, Indiana. In 2005, he received the PhD in Environmental Engineering and his minor in Statistics, focusing his dissertation in the environmental decision-making tools. He will continue working in his area of interest, air quality modeling, diesel particulate matter, air toxics, and heath risk assessment, as a faculty member of the Catholic University of Temuco in Chile.